

# Assessing The Patient In Context: Determining Neurocognitive and Personality Profiles Associated With Substance Abuse

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# ALTA MIRA

## Disclosures

*Sausalito, California*



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# Background

- Substance use disorders continue to be a major health concern in the United States and Canada
- Specific causal factors and effective treatments continue to be matters of great debate.
- Co-occurring substance use/mental health disorders are responsible for a great deal of the current public health crisis, causing mental, physical and emotional damage to the patient as well as their family and friends.
- The frequent presence of comorbid psychological, social and medical disorders for some are a precursor to the substance abuse and for others are a result of it

**Important to look at substance use disorder in context - along with the psychological disorder - to inform treatment thereby maximizing successful outcomes.**



# Comprehensive Diagnostic Assessment – What?

- One of the first steps toward a proper diagnosis and successful treatment.
- Effectively informing treatment.
- Neurocognitive and personality profiles associated with substance abuse
- Psychodiagnostic profile influences the patient's cognition
- Treatment and implications for relapse.

# Comprehensive Diagnostic Assessment – Why?

Most people seeking addiction treatment have co-occurring disorders such as anxiety, depression, or trauma that need to be addressed in order to sustain long-term recovery

- ✓ TBI
- ✓ Dementia
- ✓ Stroke
- ✓ Depression
- ✓ Trauma
- ✓ Anxiety

# Comprehensive Diagnostic Assessment – How?

- Psychodiagnostic
- Personality
- Cognition
- Mood and Emotional functioning
- Trauma Assessment
- Symptom Severity
- Crisis Assessment
- Interest Assessment

# Comprehensive Diagnostic Assessment

1. Properly diagnose and treat an extensive range of illnesses and disorders that may be contributing to substance use
2. Create a medication strategy based on empirical evidence and make appropriate adjustments during course of treatment
3. Identify and work on interpersonal issues that might otherwise compromise treatment
4. Inform ongoing treatment using objective data for better treatment adherence, compliance and efficacy
5. Inform relapse prevention plan and give patient tools to reduce relapse risk and make plausible, informed referrals and recommendations
6. Present patient/referent/clinician with tangible data to better understand patient's illness and recovery

# Why Doesn't Everyone Use Assessment?

1. Expensive
2. Time
3. Complex
4. Population
5. Individual differences
6. Thought “Not the point.”



# Neuropsychological Evaluation

- Intellectual Functioning
- Academic Skills
- Attention and Concentration
- Processing Speed
- Learning and Memory
- Conceptual and Problem Solving Capacity
- Language
- Visuospatial abilities
- Sensory-Perceptual functioning
- Motor performance

# Personality Testing

- Emotional adjustment and resilience
- Personality Style
- Constructs such as:
  - Interpersonal functioning
  - Anger/Hostility
  - Cynicism
  - Depression

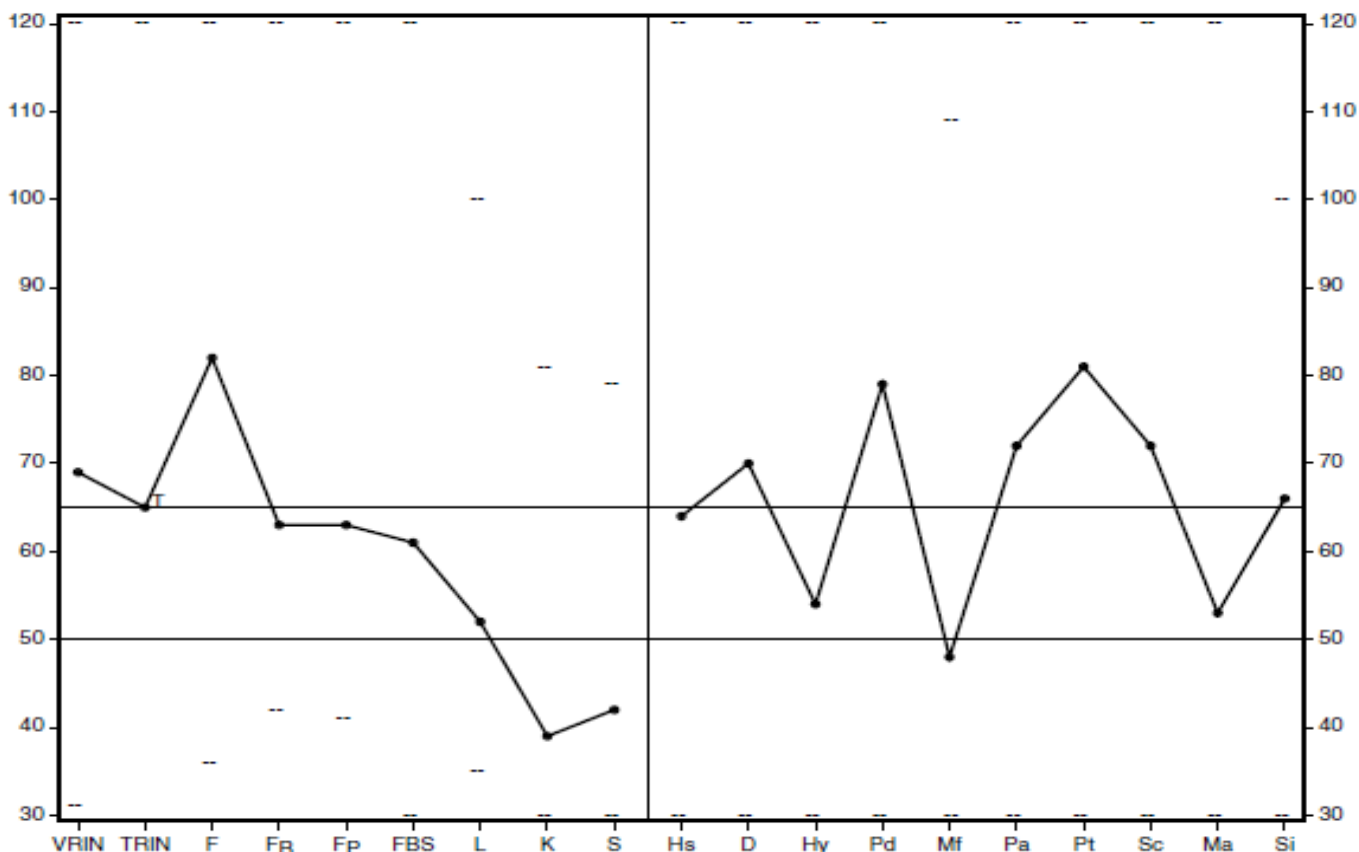
(MMPI-2, MCMI-III, SDIP-IV, Rorschach, TAT etc.)

MILLON CLINICAL MULTIAXIAL INVENTORY - III  
CONFIDENTIAL INFORMATION FOR PROFESSIONAL USE ONLY

INVALIDITY (SCALE V) = 0    INCONSISTENCY (SCALE W) = 2  
PERSONALITY CODE:    2B 1 \*\* 2A 8B 3 \* 6A + 5 6B 7 \* 8A 4 ' ' // - \*\* - \* //  
SYNDROME CODE:    - \*\* D A \* // CC \*\* - \* //  
DEMOGRAPHIC CODE:    7111/IM/M/71/W/N/19/MD/LO/-----/6/-----/

CATEGORY		SCORE		PROFILE OF BR SCORES				DIAGNOSTIC SCALES
		RAW	BR	0	60	75	85	
MODIFYING INDICES	X	103	63					DISCLOSURE
	Y	3	15					DESIRABILITY
	Z	16	68					DEBASEMENT
CLINICAL PERSONALITY PATTERNS	1	15	85					SCHIZOID
	2A	13	82					AVOIDANT
	2B	19	101					DEPRESSIVE
	3	10	78					DEPENDENT
	4	4	16					HISTRIONIC
	5	10	48					NARCISSISTIC
	6A	8	66					ANTISOCIAL
	6B	4	48					SADISTIC
	7	9	36					COMPULSIVE
	8A	3	30					NEGATIVISTIC
8B	11	80					MASOCHISTIC	
SEVERE PERSONALITY PATHOLOGY	S	7	66					SCHIZOTYPAL
	C	10	73					BORDERLINE
	P	2	30					PARANOID
CLINICAL SYNDROMES	A	7	81					ANXIETY
	H	11	73					SOMATOFORM
	N	0	0					BIPOLAR: MANIC
	D	14	82					DYSTHYMIA
	B	4	63					ALCOHOL DEPENDENCE
	T	3	60					DRUG DEPENDENCE
	R	7	65					POST-TRAUMATIC STRESS
SEVERE CLINICAL SYNDROMES	SS	14	73					THOUGHT DISORDER
	CC	17	99					MAJOR DEPRESSION
	PP	2	60					DELUSIONAL DISORDER

## MMPI-2 VALIDITY AND CLINICAL SCALES PROFILE



Raw Score:	10	11	15	5	3	16	4	10	18	13	28	23	31	25	16	31	29	20	39
K Correction:										5			4			10	10	2	
T Score (Plotted):	69	65 <sup>T</sup>	82	63	63	61	52	39	42	64	70	54	79	48	72	81	72	53	66
Response %:	98	95	98	100	100	98	100	100	100	100	98	97	96	100	95	94	96	98	100

Cannot Say (Raw): 5

F-K (Raw): 5

Welsh Code: 7<sup>-</sup>4<sup>68</sup>2<sup>0</sup>+1-3<sup>9</sup>/5: F<sup>-</sup>+L<sup>-</sup>:K#

Percent True: 55

Percent False: 45

Profile Elevation: 68.1



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**4. Shows perfectionism that interferes with task completion (e.g., is unable to complete a project because his or her own overly strict standards are not met)**

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**2-OBCMP 0 1 2 3**

Would people describe you as a perfectionist?

Do you think you are a perfectionist?

**(IF YES TO EITHER):**

How often do your high standards keep you from getting projects completed on time?

Examples?

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**5. Is reluctant to delegate tasks or to work with others unless they submit to exactly his or her way of doing things**

---

**6-OBCMP 0 1 2 3**

\* Do you end up doing a lot of jobs yourself because no one else will do it exactly the way you want it done?

Do you often take over other people's responsibilities to make sure things get done right?

**(IF YES):** Examples?

# Psychodiagnostic Testing

- Self-report measures
- Interviews (SCID 5, MINI)
- Specialty Interviews (Y-BOCS, Prodromal Questionnaire)

Inventory of symptoms

Criteria for diagnosis

Aids in looking at symptom overlap

Gives some insight into cognition

# Cognitive Deficits Due to Depression “Pseudodementia”

- Presentation with cognitive symptoms such as loss of memory and vagueness, as well as prominent slowing of movement and reduced or slowed speech that is due to depression, not dementia
- Although cognitive symptoms are seen to improve with treatment of depression...
- On follow-up, most patients develop further cognitive decline over time, and are diagnosed with an irreversible dementia



# Cognitive Deficits Due to Depression “Pseudodementia” (cont’d)

- In most cases the dementia is real and likely to persist or progress despite the treatment of depression
- Patients with dementia should still be screened and treated for depression
- Successful treatment of depression may not reverse the dementia but may improve function and quality of life
- Truly reversible causes of dementia are much more rare than previously thought
- Depression is less likely an imitator of dementia than a predictor of dementia in later life

# Differential Diagnosis: Depression vs. Dementia

Criterion	Depression	Dementia
Duration	At least 2 weeks, sometimes months	Months to years
Mood/Affect	Depressed mood, anhedonia	Disinterested, detached
Psychomotor Speed	Mental & physical slowing	Normal to mildly slow
Attention	Borderline selective attention, difficulty with complex attention	Normal to mildly abnormal until late in course
Memory	Mild impairment, memory retrieval difficulty	Abnormal, amnesic pattern in most cases
Speech/language	Decreased amount of speech	Anomic aphasia, empty or sparse content
Other cognition	Difficulty with effortful tasks	Multiple cognitive deficits
Other behavior	Indecisive, delusions and other psychiatric symptoms	Psychiatric symptoms may be present, usually less prominent
Physical symptoms	Lack of energy, sleep and appetite disturbances	Usually none until middle or late stages

# Schizophrenia - General and Specific Cognitive Deficits

- There are deficits spanning all aspects of cognition relative to healthy control.
- There is a variation in the extent of involvement across domains with evidence from multiple studies suggested that **memory** and **executive functions** are the two domains most likely to show differential impairment.
- Most common cognitive impairments found in patients with schizophrenia are those of **attention, memory and executive functions**.
- Average cognitive impairments for patients with schizophrenia in these cognitive domain reach 2SD below that of healthy control.

# General and Specific Deficits

- Rather than localized cognitive deficit, illness shows a more widespread neuropsychological dysfunction. (Buchbaum, 1990)
- Selectivity of cognitive deficits occurs against background of general impairment, with even the less sensitive tasks.(Heinrichs and Zakzanis, 1998)
- Cognitive domains of IQ, memory, language, attention and executive function-consistent trend of poor performance. (Fioravanti et al 2005)
- Confirmatory factor analysis of various cognitive functions reflected more generalized cognitive disability. (Dickinson et al 2006)
- Social Cognition also is seen to suffer and this decline has treatment implications.

# Assessment of Substance Abuse Patients

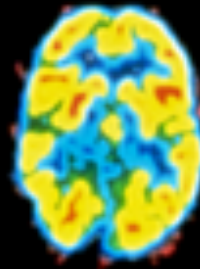
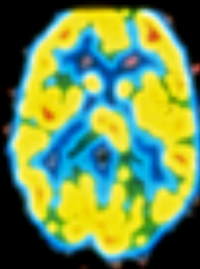
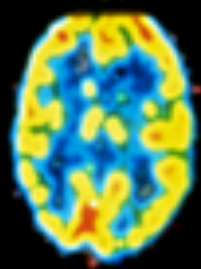
We tend to want to answer the client's questions:

“What did I do? What damage did I do to my brain?”

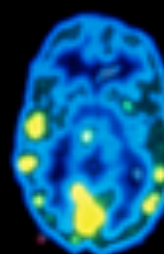
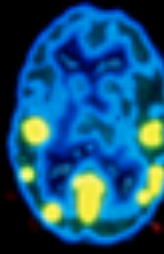
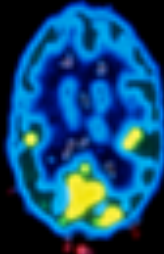
Classic Answer:

Neuropsychological Impairment due to abuse of \_\_\_\_\_

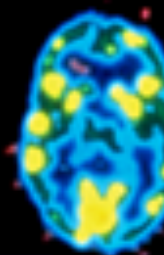
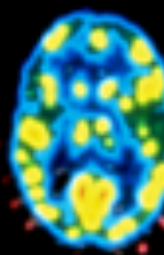
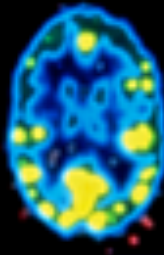
# The Brain Needs Time



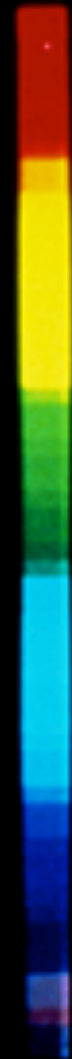
**Normal**



**Cocaine Abuser (10 Days)**



**Cocaine Abuser (100 Days)**



# Ask Questions Outside The Box

What are the effects of **ACUTE** use, **CHRONIC** use and **RESIDUAL** Use on neuropsychological performance?

Where is my client's cognition now?

Will they clear --- and when?

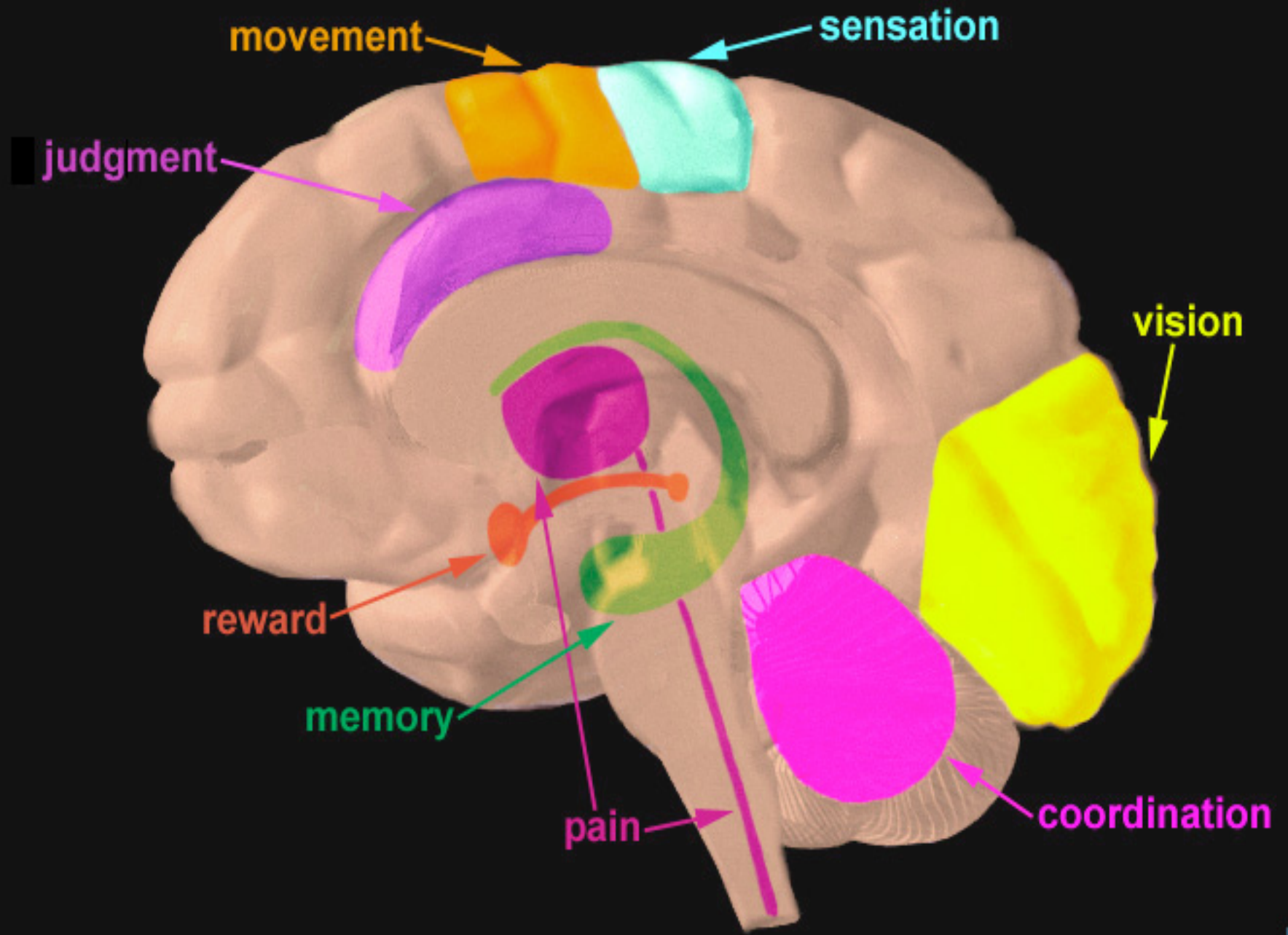
How fast can I go?

Why don't they seem to understand me? How am I not matching my treatment to their abilities?

What are they able to do? What are reasonable expectations?

# What Are We Going to Do with Them after Treatment?





movement

sensation

judgment

vision

reward

memory

pain

coordination

# Alcohol Use – Acute Effects

## Acute Effects:

- Heart rate – BP
  - Disinhibition
  - Balance instability
  - Slowed psychomotor, cognitive and sensory information processing
- 
- ✓ Depressant – CNS – looks like pattern of tranquilizer and hypnotic deficits
  - ✓ Deficits tend to correlate with intake quantity, duration and age (began)
  - ✓ Patterns are important (binge vs. heavy chronic use)
  - ✓ Positive relationship between amount and frequency of consumption and cognitive deficits

# Alcohol – Chronic Use

## Chronic Use d/o

- ✓ **Variations in medical risk factors, sex differences, family history, age**

## Deficits:

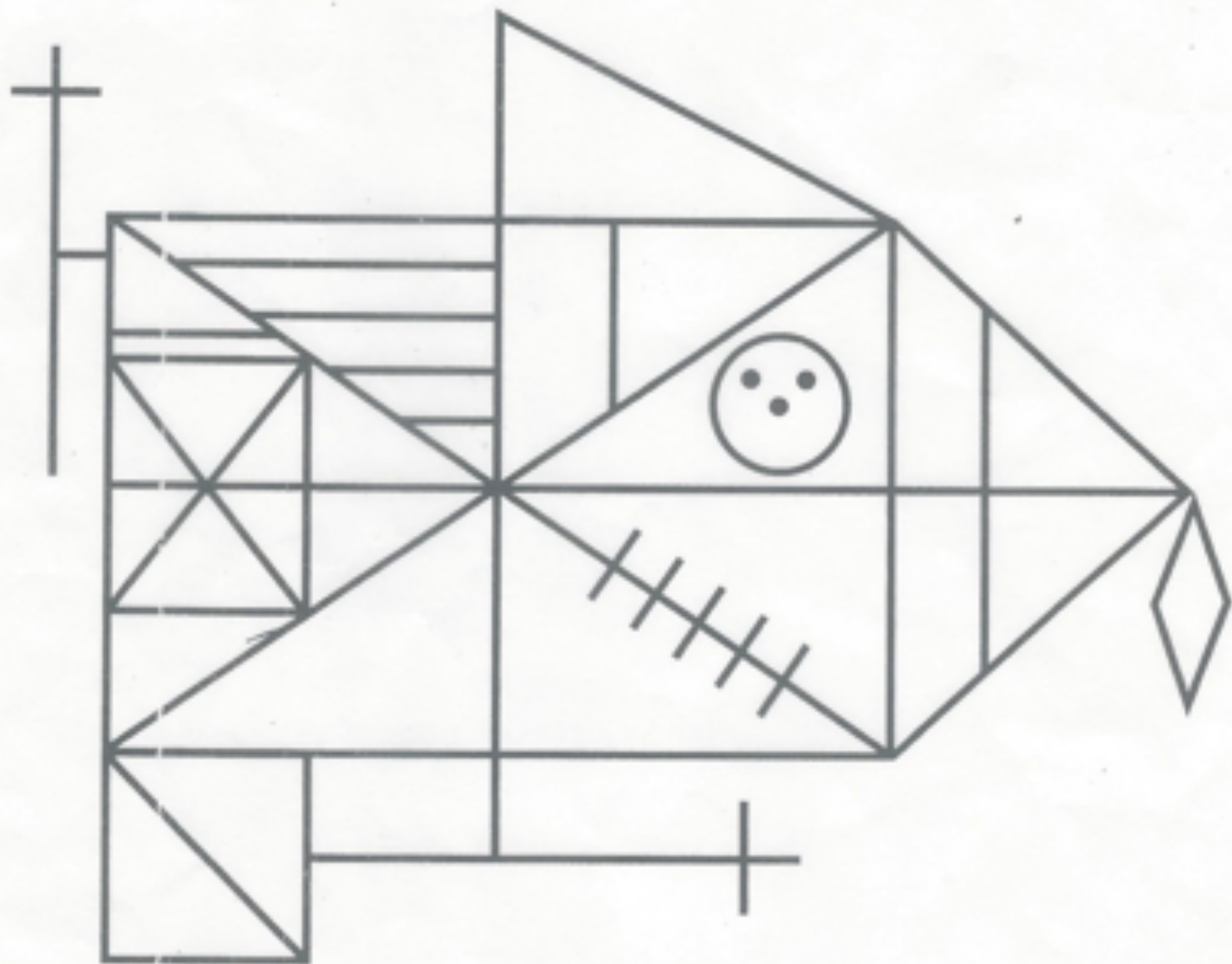
- Complex visuospatial ability
- Psychomotor speed
- Learned skills
- Short-term recall
- Subtle deficits in concept formation and mental flexibility

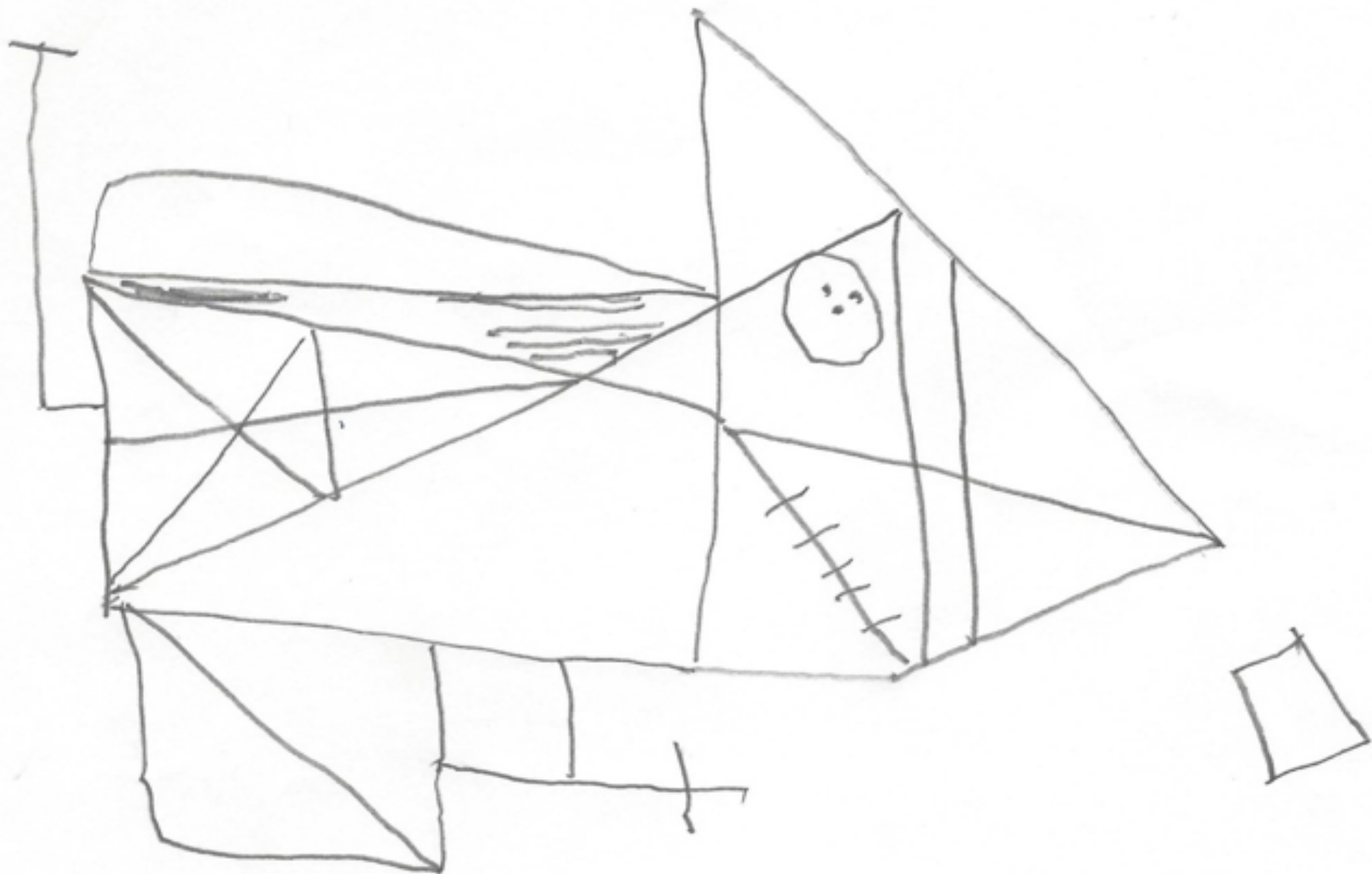
# Alcohol – Residual Effects

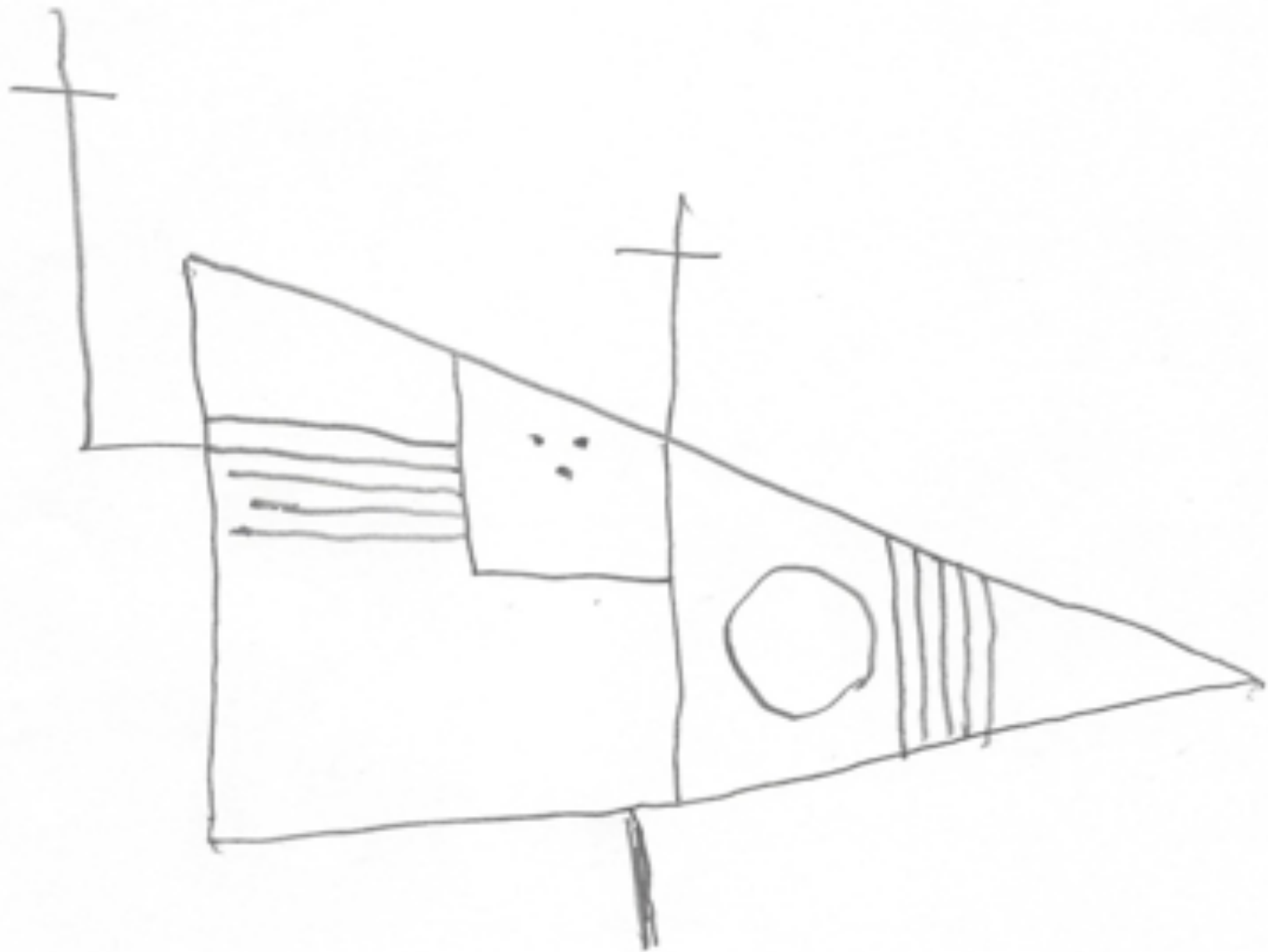
- ✓ Detox effect within 1<sup>st</sup> two weeks- all cog domains
- ✓ 4 weeks to 8 weeks improvements
- ✓ Months – most show improvement. How much is correlated with individual differences. Long-term MRIs show permanent damage.
- ✓ Age makes reversibility less obvious

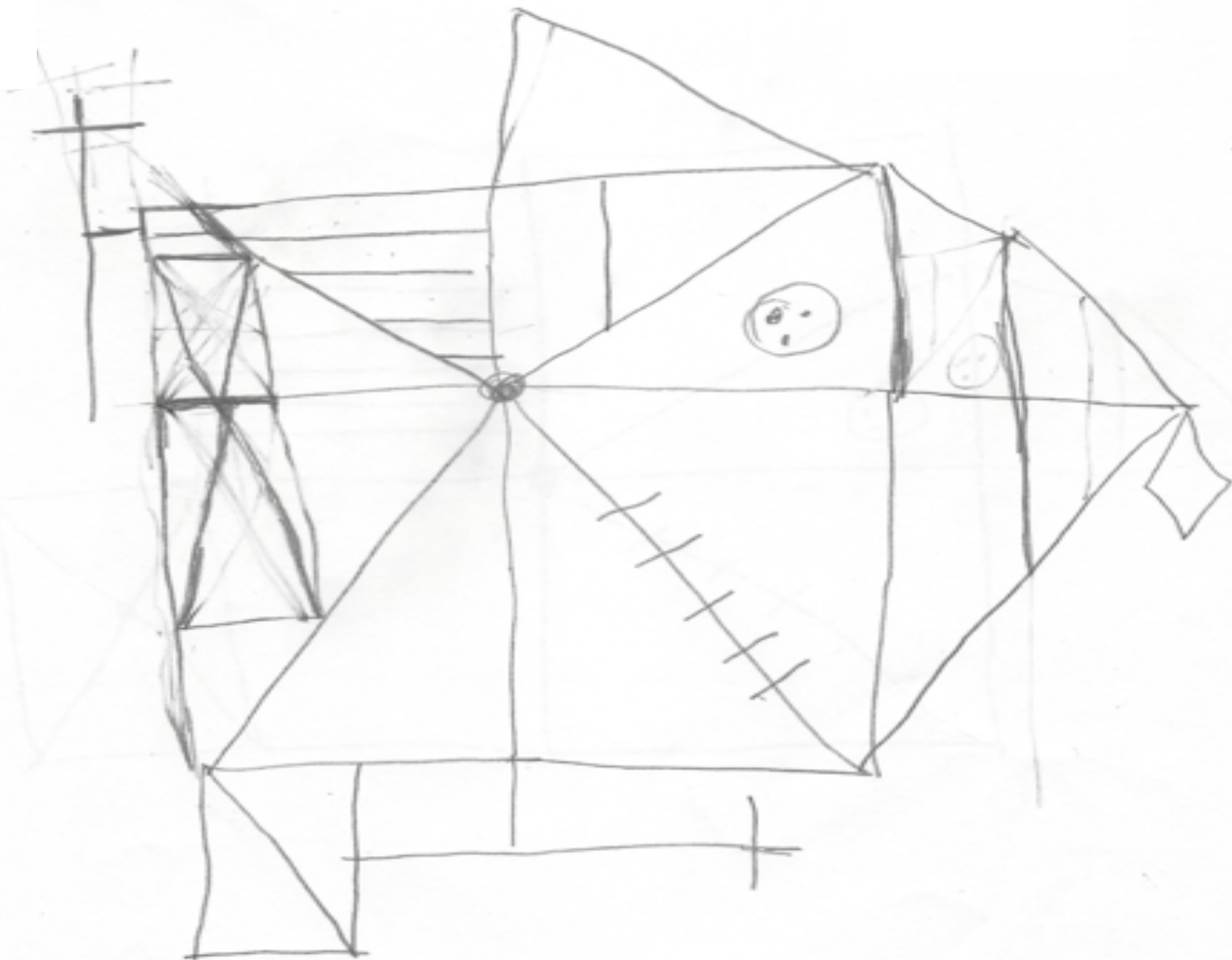
## Executive Functions

- Problem solving, decision-making, working memory, sequencing,
- Short term, explicit and declarative memory (mild to anterograde amnesia)
- Visuospatial processes
- Balance and gait, processing speed (Can last up to 1 year) (Fein & Greenstein, 2013)
- Affective Comprehension (Monnot et al., 2002)

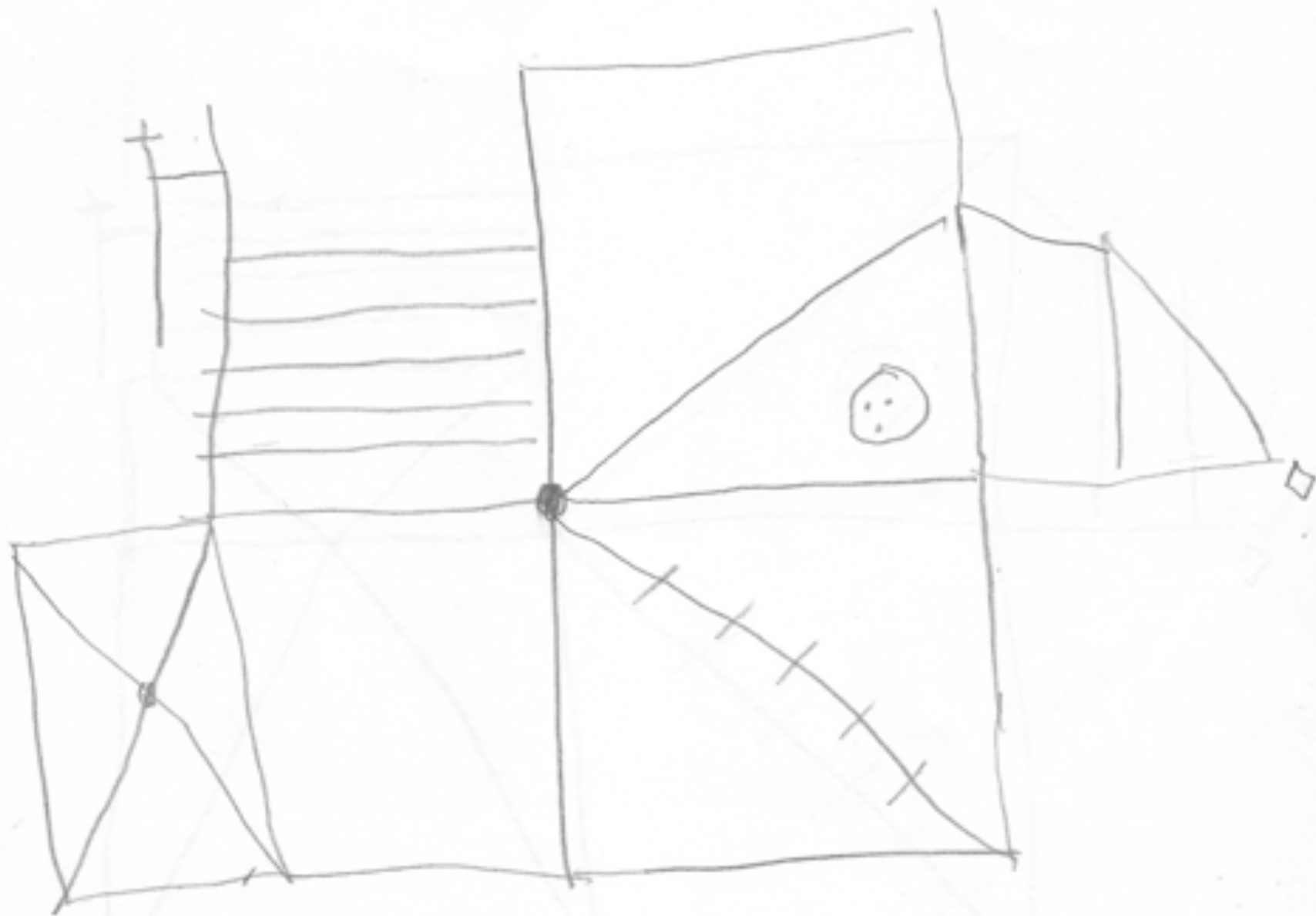












# Cocaine - Acute

- ✓ Deficits relative to amount of use, years of use and duration of abstinence
  - ✓ CT Scan atrophy – evidence of white matter lesions
  - ✓ High correlation with stroke
- 
- Disinhibition
  - Impulsivity
  - Increased HR/BP
  - Enhance learning and/or attention (NIMH, 2010)

# Addiction is a Brain Disease



**Prolonged Use  
Changes**

**the Brain**

**in  
Fundamental  
and Lasting  
Ways**

# Deficits Chronic Cocaine

- Memory and concentration
- Executive functioning (**working memory**, inhibition, set-shifting)
- Attention span and Vigilance
- Memory impairments Sp. (verbal learning and memory)(Fox et al., 2009) (Recreational users display deficits of poorer learning and recognition accuracy)
- Decreased mental flexibility (Kelley et al., 2005)
- Emotion recognition (Fernando-Serrano et al., 2010) associated with cumulative levels of exposure.
- Narrower ability to experience emotions implied in research but not validated.

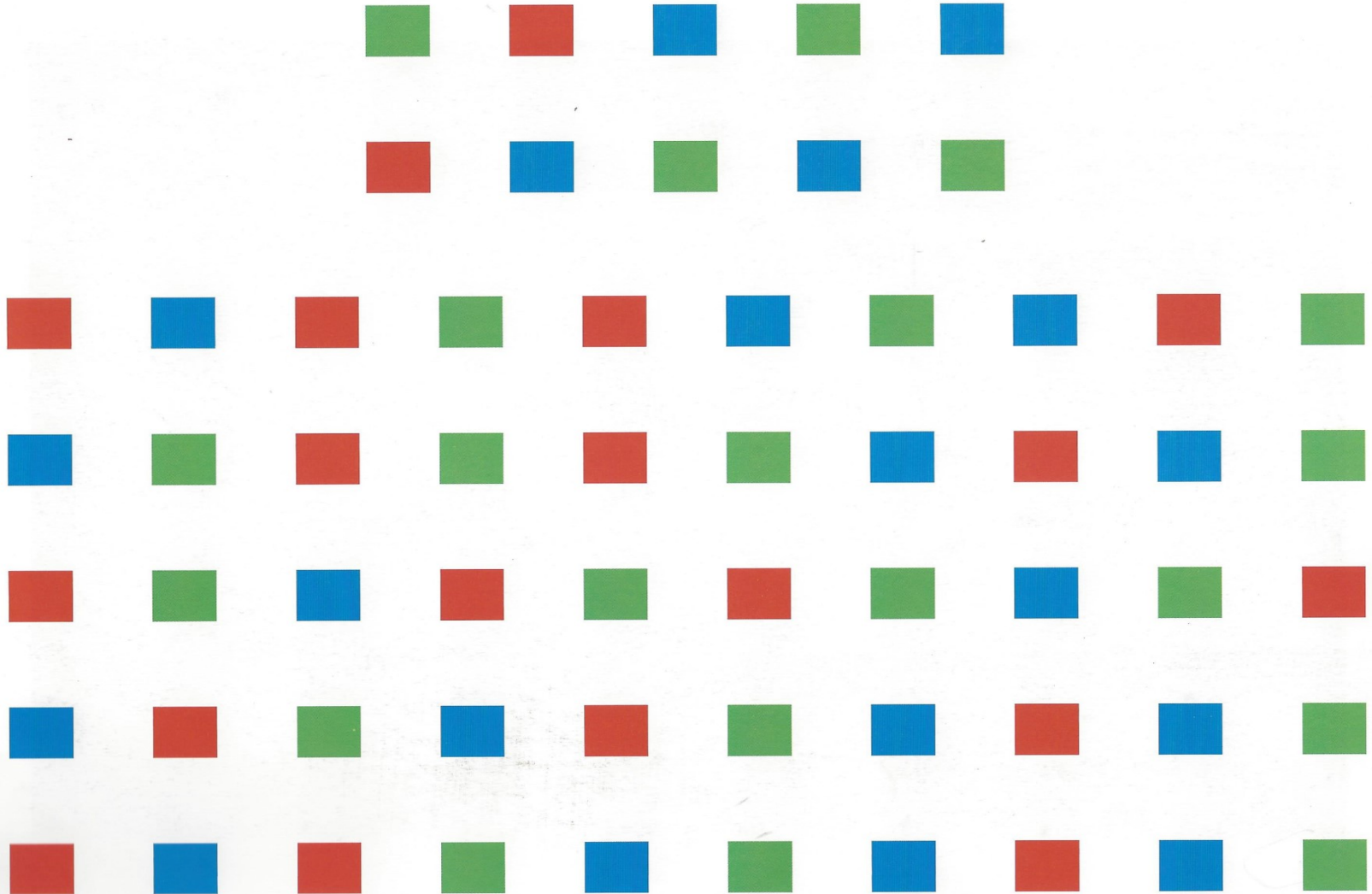
Neurocognitive effects = dose specific and persistent even after six months of abstinence

# Residual Effects of Cocaine Use

- ✓ Subtle and specific vs. General
- ✓ Executive functioning (attn. planning mental flexibility)
- ✓ Attention, reasoning, spatial planning – best indicators of treatment outcome
- ✓ Changes in affect and personality – some short lived and some permanent ASPD and BPD (associated with poorer cognitive function – frontal lobe dysfunction)
- ✓ Depression (during withdrawal – associated with cognitive deficits)

# Cocaine Phenomena

1. Heavy cocaine users may show persistent changes in brain metabolism with no detectable effects
  - Alternate neural substrates are use to perform task
  - Power and sample size of studies
2. ETOH and Cocaine – most studies found that most common
  - Short and long-term memory and visual motor functions
  - Not any worse than separate use (Pennings et al., 2002)



**red blue green red blue**

**green blue green red green**

**green red blue green blue red blue green blue green**

**red green blue green blue green red blue red green**

**red green blue green red blue green red blue red**

**blue green red blue green red blue green blue red**

**green red blue red blue green red blue red green**



**Rule:**

Name the ink color.

red blue green blue green

red blue red green red

blue green blue red blue red blue red blue red

blue green blue green red green blue red blue green

red green red blue green red green red blue green

blue green blue red green blue red green red green

green blue red blue green red blue green red blue

Rules:

1. **blue** – Name the ink color.

2. **red** – Read the word.

**blue** red **green** red **blue**

**green** **red** **green** red **blue**

**green** **blue** **green** **blue** **red** **green** red **green** red **blue**

**red** **blue** red **green** **blue** **green** **blue** **red** red **blue**

**blue** red **green** **red** red **green** **blue** **red** **blue** **red**

**blue** **green** **blue** **green** **red** red **green** red **blue** **green**

**green** **red** red **blue** **green** **blue** red **green** **green** **red**

# Opiates - Acute

- ✓ Mental and physical “sluggishness.” Can be seen in EEG slowing
- ✓ Personal neglect
- ✓ Few studies documenting cognitive effects of acute use

## Non-Drug-Abusing Patients

Reduce visual acuity and increase perceptual impairment (Zacny et al., 2011)

Slow motor performance and decreased processing speed (Zacny et al., 2011)

Focused attention impaired, while sustained and divided attention spared

Mental flexibility

## Non-dependent Users

Perceptual spatial

Psychomotor Speed

With increasing opioid tolerance and dependence,  
cognition appears to be unaffected until impaired by higher doses

# Opiates – Residual

- Mental flexibility, attention and abstract reasoning returned to baseline (non-user) levels after abstinence, however impulsivity remained at a deficit compared to control group (Pav et al, 2001)
- Overall – Long-term opiate use does not severely impair cognitive functioning

# Meth Cognitive Dysfunction and Change

- ✓ Frontal cortex, cingulate cortex and medial temporal lobe – loss of grey matter
- ✓ Approximately 50% entering treatment suffer from cerebral (cognitive) dysfunction
  - Less likely to attend continuing care
  - Less likely to be employed
  - Often mistaken as resistant or unmotivated
  - Less able to absorb information
- ✓ Stimulant addicts look like they have degenerative brain disease
- ✓ Damaging effects on nervous system

# Methamphetamine – Acute

- Aggression
- Euphoria
- Decreased anxiety
- Irritability, anxiety, hallucinations, compulsivity, paranoia (binge and heavy use) (Semple et al. 2003)
- Attention
- Memory
- Impulsivity
- Motor functioning

# Meth - Chronic

## Executive & visuospatial functioning problems include:

- Learning and Memory
- Abstraction
- Problem solving
- Cognitive Organization
- Psychomotor and Processing Speed
- Cognitive flexibility (Wisconsin Card Sorting Test, Trails B)
- Planning
- Response Inhibition (Color-Word Interference Test)
- Social Cognition

# Methamphetamine Residual

- Poorer Performance across the Board – different from other stimulants
- Long term stimulant abuse causes damage to dopamine producing cells and leads to reduced levels
- Stimulant addicts may suffer from poor attention and compromised fine motor skills
- Social Cognition
- Executive Function improves after 2 weeks (Chou et al., 2007)
- Motor skills, processing speed, verbal memory (9+ months) (Ludicello et al. 2010)
- Some cognitive deficits may persist even after longer periods – executive functioning, attention, strategic components of encoding (Ludicello et al. 2010)



# Methamphetamine Residual

- Global functional impairment in many cases
- IADLS
- Financial management
- Unemployment
- Deficits in Prospective memory – poor compliance

# Marijuana

- ✓ Positively Correlated with psychosis, depression – even with no prior history
- ✓ Other drugs (ETOH, cocaine, meth, reduced cerebral blood flow seen on frontal, limbic, cerebral regions)  
Marijuana=orbital and mesial frontal insula, temporal anterior cingulate and cerebellum
- ✓ Subjective doesn't match objective reports during acute phase – attention, well-established memories

# Marijuana - Acute Effects

- ✓ Acute hallucinatory and reactive emotional states (Brust, 2000).
- ✓ Many acute effects are dose dependent. Several resolve after habituation.

Disorientation to time

Transient memory loss

Attention (selective and sustained) impairment – although may resolve if individual habituates

Slowed visual processing

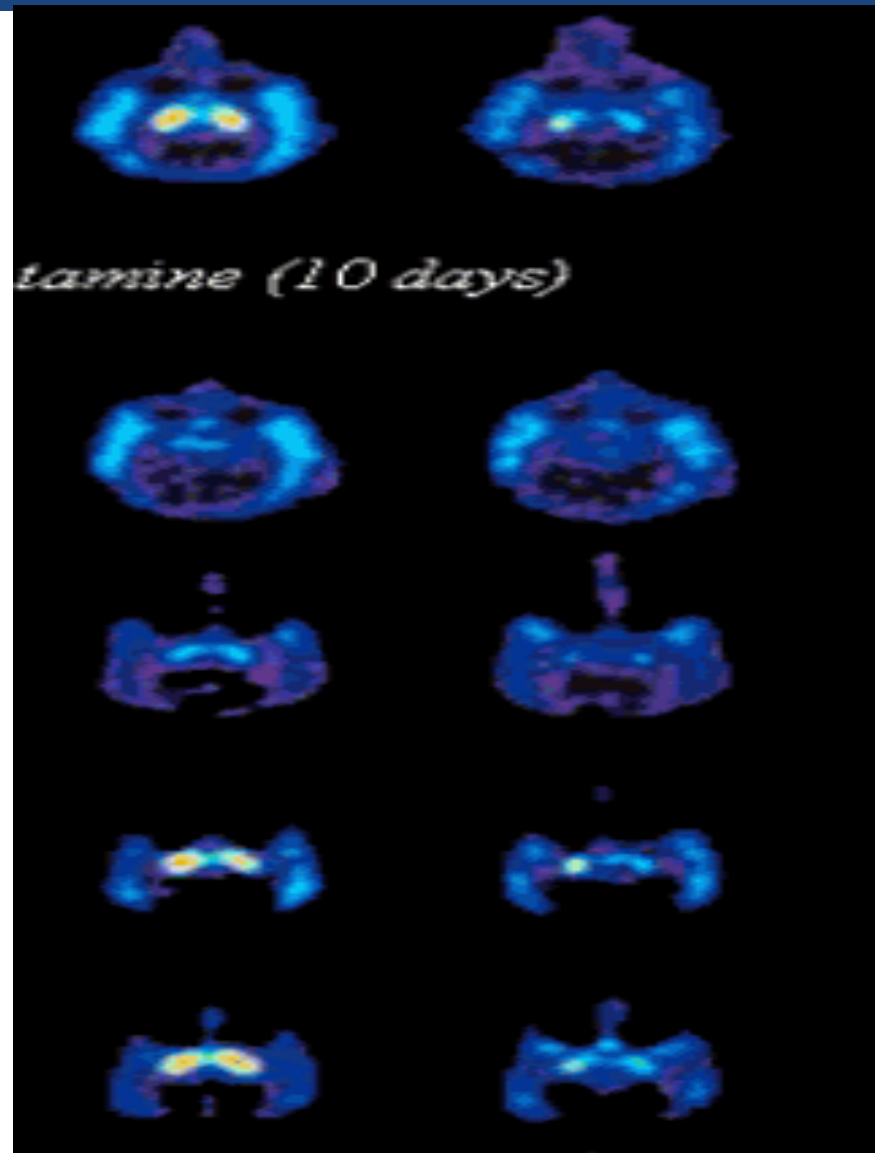
Working memory

Processing speed

# Marijuana - Long term Effects

- Research is mixed, but magnitude and persistence of impairments vary with quantity, frequency, duration of use and age of onset.
- Observed impairments were severe and long-lasting in those with more frequent and prolonged heavy use and younger age of first use (Kyprianidou, Malefaki & Papthanasopoulos, 2006). Difficult to ascertain because prior testing not available. This was true for executive functioning, processing speed.
- Research indicate that deficits could be expected to resolve in healthy adult samples (Schreiner & Dunn).
- Everyday issues reported include internally cued prospective memory, driving decrements, concentration.

# Drugs Have Long-term Consequences



# Using Assessment During Treatment

- Impulsivity and deficits in executive functions – inform modality of treatment used to treat
- Current evidence-based practices tax higher-order functions (planning, flexibility, learning, memory)
- Positive treatment outcomes rely on behavior change and implementation of new skills (Bates et al., 2013)
- Patients with cognitive deficits can be seen as having lower motivation and greater denial (Goldman, 1995)
- Patients get a feeling of “I will never get this!” contributing to noncompliance with treatment (Weinstein & Schaffer, 1993)

## Using Assessment During Treatment cont'd.

- Increase retention, cut-down on drop-outs and lower relapse rates (Smith, McCrady, 1991).
- Visual-Spatial can inform re: cognitive organization
- Attention informs scheduling
- Tracking improvements in cognition that occur with sustained abstinence can be a powerful motivational tool – and can inform the next clinician to treat.
- Patients need to know that relapse is associated with ACCELERATED negative changes to brain health and cognition (Loeber et al., 2010)

# Using Assessment for Relapse Prevention

- Substance most disrupting effects – prefrontal cortex dysfunction – problems with executive function and inhibitory response
- Memory Impairment (including prospective memory impairment – memory needed to plan and carry out future actions) (Ellis, 1996).
- Cognitive impairment may contribute to the failure to regulate substance abuse in the moment even in the face of real, negative consequences.
- Associated with reduced treatment retention and compliance, reduced self-efficacy, which in-turn predicted less successful outcomes after treatment (Bates, et al., 2006)
- Examples: AA involvement, Social Networks



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## Assessment and Relapse Prevention

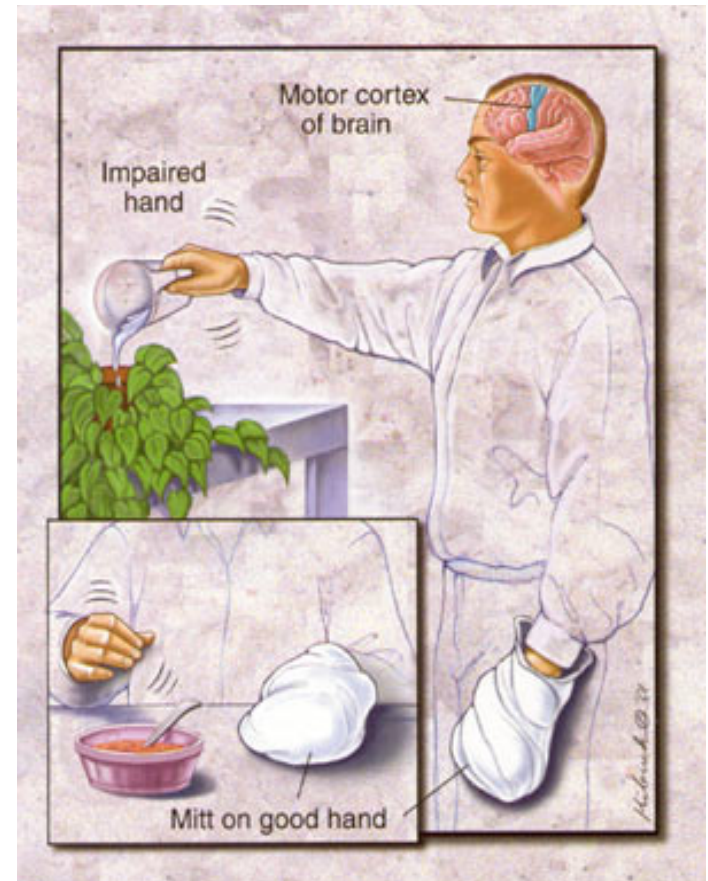


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# Cognitive Dysfunction and Change

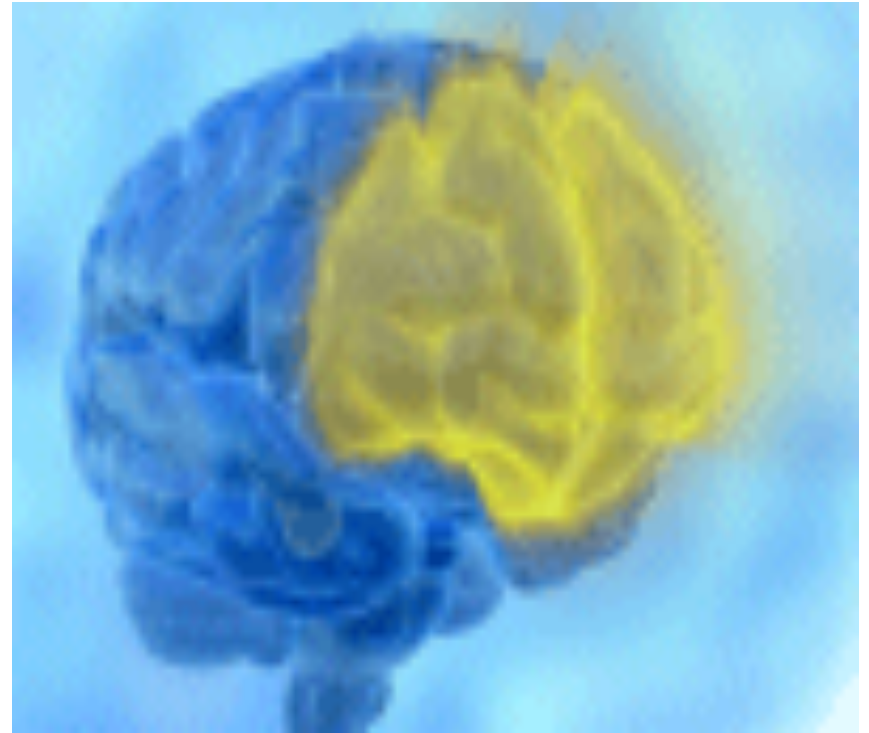
## Recovery in neuropsychological functioning

- Most of treatment is during time of greatest dysfunction
- Recovery is:
  - Time-dependent
    - Due to sustained abstinence
  - Experience-dependent
    - Active rehabilitation or repetitive behavior



# Cognitive Rehabilitation

- Repetitive recovery-oriented behaviors
- Repetitive recovery-oriented thoughts



# Suggestions

- Repetition
- Multi-modal learning
- Memory Aids
- Self-efficacy training (e.g. working memory training)
- Consultation

# Questions?



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