Specialized In-Patient Substance Use Treatment for Individuals with a TBI

What Is OASAS?

• Office of Alcoholism and Substance Abuse Services

What Is OASAS?

• One of the nation’s largest addiction services system
• More than 1,000 employees
• 60% employed in the 12 ATCs
• $1.7 billion in Medicaid, Federal Block Grant and state funds
• 1,550 prevention and treatment programs
• Treatment system serves 110,000 persons daily
• 90% in outpatient or methadone programs
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The OASAS Treatment System

- Gender: Female 25%, Male 75%
- Age Group: 18 to 24: 25%, 25 to 34: 20%, 35 to 44: 15%, 45 or older: 5%
- Race/Ethnicity: Black: 5%, Hispanic: 10%, Other: 5%, White: 80%
- Primary Substance: Alcohol: 45%, Marij/Hash: 14%, Other: 2%, Cocaine/Crack: 17%, Opiate: 22%
- Education Status: Less than high school: 5%, High School/GED or more: 95%
- Homeless: 0%

Source: OASAS, Data Warehouse

3rd Annual Addiction & TBI Conference
• TBI incidence
  - ?????????

OASAS Response
OASAS TBI Advisory Council
First meeting June 9, 2008

Website - Conferences
Specialized In-Patient Substance Use
Treatment for Individuals with a TBI

May 25, 2011

ASSESSMENT

• Screening Tool: ICD HELPS

- H Did you ever HIT your head? Were you ever HIT on the head?
- E Were you ever seen in an EMERGENCY room, by a doctor or hospitalized? For what reason?
- L Did you ever LOSE consciousness? For how long? For what reason?
- P Did you have any PROBLEMS after you were hit on the head? Headache? Dizziness? Anxiety? Depression? Difficulty concentrating? Difficulty Remembering? Difficulty with school work? Poor judgment? Poor problem solving?
- S Any other SIGNIFICANT ILLNESS? Look for hospitalizations for brain cancer, meningitis, stroke, heart attack, diabetes. Screen for domestic violence and child abuse

ICD-International Center for the Disabled: Picard, Scarisbick, Paluck, 1993

Scoring the HELPS Screening Tool

A HELPS screening is considered positive for a possible TBI when the following 3 items are identified:

1.) An event that could have caused a brain injury (yes to H, E or S), and
2.) A period of loss of consciousness or altered consciousness after the injury or another indication that the injury was severe (yes to L or E), and
3.) The presence of two or more chronic problems listed under P that were not present before the injury.

Note:
- A positive screening is not sufficient to diagnose TBI as the reason for current symptoms and difficulties - other possible causes may need to be ruled out

THE DRUG AND ALCOHOL CONNECTION TO ACQUIRED BRAIN INJURY

• Alcohol and drugs can cause brain injury directly or indirectly.
  - Alcohol is a neurotoxin, though its effect and extent of damage depends on the amount of alcohol consumption, the age and sex of the consumer, genetic vulnerability and other factors.
  - Binge drinkers may be less prone to alcohol related cognitive deficits than heavy daily users, though they are still vulnerable to alcohol intoxication related events.
ACQUIRED BRAIN INJURY

- Alcohol can cause
  - Direct brain damage (alcohol dementia, Wernicke - Korsakoff's Syndrome, and atrophy of the cerebrum and cerebellum)
  - There can be some improvement in deficits with abstinence
  - Indirect damage can be associated with
    - Falls and accidents
    - Intracerebral bleeds due to alcohol's effect on platelets and blood pressure
    - Hepatic encephalopathy due to alcohol's effect on the liver

FEMALE DRINKERS LOSE BRAIN VOLUME MORE QUICKLY THAN MEN, ACCORDING TO RESEARCHERS AT THE UNIVERSITY OF HEIDELBERG.
- PERFORMED 150 CT SCANS
- AMOUNT OF BRAIN VOLUME LOSS WAS EQUAL TO MEN, ONLY QUICKER ONSET
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3rd Annual Addiction & TBI Conference
Wernicke's Encephalopathy- Korsakoff Syndrome

It is estimated that between 10 and 24% of all cases of dementia in the United Kingdom are alcohol related. The mechanisms of alcohol related brain damage include:

- Direct neurotoxin effects of alcohol
- Direct neurotoxin effects of Acetaldehyde (an alcohol metabolite)
- Thiamine depletion
- Metabolic factors associated with intoxication
- Metabolic factors associated with withdrawal
- Cerebrovascular disease
- Hepatic encephalopathy
- Head injury related to alcohol use
Wernicke’s Encephalopathy - Korsakoff Syndrome

- Wernicke’s encephalopathy is an acute neuropsychiatric reaction to thiamine deficiency.
  - It is characterized by confusion, ataxia, nystagmus (rhythmic oscillation of the eyeballs) and ophthalmoplegia (lateral gaze paralysis).
  - Only 20% of patients with Wernicke’s are identified before death.
  - Wernicke’s encephalopathy is a medical emergency and leads to death in up to 20% of cases or goes onto Korsakoff’s syndrome in 85% of the survivors.
  - Up to 25% of the Korsakoff group will require long-term institutionalization.

- The Korsakoff syndrome includes confusion in a setting of the patient being totally awake with severely impaired conversation. The patient has impaired current and short-term memory loss and tends to invent recollections (confabulation) during conversation.
  - The onset of Korsakoff syndrome is after a Wernicke event but can be insidious.

- It is known that thiamine depletion affects at least 6 neurotransmitter systems including GABA.
- The neuropathy that can be seen with these disorders include neuronal loss, micro-hemorrhages, and gliosis (overgrowth of glial cells in the paraventricular, periaqueductal grey matter and mamillary body). There can also be variable degrees of cortical atrophy, especially of the frontal lobes.
Wernicke's Encephalopathy- Korsakoff Syndrome

- Treatment of Wernicke's is high dose parental thiamine. The ocular signs recover in days to weeks after the treatment. The ataxia responds in the first week but can take 1-2 months to resolve. Acute confusion improves in the first 1 – 2 days but can take months to totally clear.

NUTRITIONAL OPTIC NEUROPATHY
Tobacco-Alcohol Amblyopia

Decreased visual activity

Cigarette smoking exacerbates alcohol induced brain damage

- Chronic alcohol use damages the brains of alcoholics, particularly the frontal lobes which are critical for high-order cognitive functioning (problem solving, reasoning, abstractions, planning, foresight).
- Chronic cigarette use increases the severity of this brain damage.

These statements are based on measurements made on smokers, light smokers, abstinent alcoholics, and light drinkers using functional MRIs (Durazzo et al, Alcoholism Clinical and Experimental Research Dec. 2004)
ALCOHOLIC SMOKERS LOSE MORE BRAIN MASS

- The study raises the question of whether alcoholism treatment programs should also address smoking, especially since it may cause cognitive impairment as clients get older.

FETAL ALCOHOL SYNDROME

- The number one preventable disease triad of:
  - Growth deficiency
  - Mental retardation
  - Altered morphology

ACQUIRED BRAIN INJURY

- Solvents such as glue can lead to ataxia (impaired gait) and cognitive problems.
- Metabolic syndromes can also be seen especially with inhalation of substances that affect the kidney.
ACQUIRED BRAIN INJURY

- Cannabis dependence is associated with impaired attention, concentration and motivation.
  - The average volumes of both the hippocampus (top) and amygdala were reduced in the long-term cannabis users compared with controls; the average hippocampal volume was 12% less and the amygdala volume 7.1% less in the cannabis users compared with controls.

- Stimulant use can be associated with strokes, seizures and long-term memory and concentration problems.

METHAMPHETAMINE

- Journal of Neuroscience June 2004 – Dr. P. Thompson MRI research
  - Limbic system (craving, reward, emotion, mood) lost 11% of tissue
  - Hippocampus (new memories) lost 8% = to early Alzheimer's
ACQUIRED BRAIN INJURY

- Sedative effects are not well studied in the long term, though overdose can lead to respiratory compromise and oxygen deprivation.
- Oxygen deprivation can also be seen in opiate overdose.

The Route of use is an important issue as well in brain injury.
LEUKOENCEPHALOPATHY DUE TO SMOKING HEROIN

The CDC estimated that 5.3 million Americans live with disabilities due to brain injury.

67% of people in rehabilitation for brain injury have a previous history of substance abuse (Thurman, 1998).

- 50% of these people will return to using alcohol and drugs after the injury (Corrigan, 1995).

20% of persons with brain injuries who did not use alcohol or drugs prior to the injury, were vulnerable to alcohol and drug use after the injury (Corrigan, 1995).

50% of clients enrolled in OASAS Programs were affected by probable TBI (N=647) (Fenske, Gordon, Perez, Hibbard, Brandau, submitted for publication).
The Problem:

- Diagnosis is missed
- Patients with a TBI in Substance Use Treatment programs are more likely to prematurely discontinue treatment and are often characterized as non-compliant.