COGNITIVE BRAIN DEFICITS ASSOCIATED WITH ALCOHOL ABUSE: TREATMENT IMPLICATIONS

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Abstract

This is a literature review which combines new research studies findings with past studies. It focuses on three issues. The first is that alcohol abuse adversely affects the processing of information in the brain in recovering individuals and thus affects treatment outcomes. The second looks at how therapists can improve the chances of an individual having a successful recovery from alcohol abuse by implementing strategies based on knowing their patient's cognitive deficits. The third is that most brains of recovering individuals are not operating at peak efficiency in processing information and then translating it into useful behavioral changes. A case study is given and brief vignettes of patients who demonstrate these three issues. Therapeutic interventions implemented are then discussed. The goal of this paper is to help clinicians improve treatment outcomes and decrease relapse rate of those in recovery.

Key Words: Memory, perceptual motor coordination, sleep disturbances

Introduction

Alcohol is the number one drug problem in the United States yet few therapists know much about alcohol effects on the brain or how to treat alcohol abusers (Doweiko, 2006). Less than 2 percent of psychologists, for example, list substance abuse as an area of expertise. Medical school training for physicians devotes minimal training in treating alcohol/substance abuse issues yet it accounts for some 25% of primary care caseload (Jones, Knutson, & Haines, 2003). This article hopes to add to the growing awareness of cognitive issues affecting alcohol abuse treatment.

The first organ of the body that alcohol attacks is the frontal lobe of the brain, responsible for executive decision making, forethought and impulse control. Alcohol, a neurotoxin, destroys red blood cells (RBC) thus causing decreased oxygen supply to the brain (Doweiko, 2006). At the intermediate stages of alcohol abuse, cognitive deficits are found: (1) decreased problem solving, (2) decreased nonverbal abstracting skills, (3) decreased perceptual-spatial motor performance, (4) short-term memory issues and (5) depressed new learning skills (Parsons, Butters, & Nathan, 1987). Neither depression nor anxiety enter significantly into the above five deficits (Parsons et al.). Short term memory and perceptual motor coordination are the most difficult deficits to overcome over time (Arria et al., 1991). A sixth common deficit is sleep disturbances which affects learning, processing new memories, problem solving and concentrate (Brower, 2001; Kern-Hage, 2004; Winerman, 2006).

Moderate alcohol use is associated with brain atrophy (wasting away of tissue) in middle-aged adults (Ding et al., 2003) and eventually, chronic alcohol use results in cerebral cortex shrinkage (Oscar-Berman, 1992).). Glasser , Ham,& Parsons (1997) found mid-thirties alcoholics could no longer mask their deficits in verbal, problem solving or visual spatial abilities. As one grows older, permanent loss of short-term memory and loss of executive function in the frontal lobe can occur due to chronic alcoholism or the fact that thiamine is deficient (Brokate, Eling, & Hidlebrandt, 2003). "Thiamine deficiency experienced by some chronic alcoholics also produces anterograde amnesia, by creating lesions in parts of the brain known as the mammillary bodies and the medial thalamus" " (Myslinski, 1998, p. 165**)**. Alcohol also causes demyelination, a loss of myelin sheathing protecting the neurotransmission of information across the brain. Thus, neuron information transmission speed is slowed and compromised by the destruction of the myelin. (Rosenbloom, Sullivan, & Pfefferbaum, 2003).

Method

A single case study was used to illustrate the effects of alcohol abuse on cognition. Some years ago, the senior author of this paper was asked to do a neuropsychological assessment of a recently retired CEO of a large company. He was found unconscious in his home with empty bottles of liquor beside him. After admission to a hospital, a consult was generated requesting neuropsychological assessment. The consult asked two questions: (1) is he intellectually capable of

handling his financial affairs? (2) should consideration be given to revoking his driver's license as he had a recent history of auto accidents?

A review of his records indicated that he was forced to retire from his company due to alcohol interfering with his job functioning. He had been given a generous retirement package along with a superb medical workup. It indicated, among other things, that his Intelligence Quotient (I.Q.) was in the top 99.99% of adults. Also it indicated that alcohol was his drug of abuse. After waiting one month after admission to the hospital, testing began. Waiting for one month of sobriety was necessary in order to get a clearer picture of his cognitive functioning and to allow the immediate effects of alcohol on his brain to somewhat dissipate.

Results

The assessment discovered perceptual-motor coordination deficits which would likely impact his driving abilities adversely. Also, his I.Q. had fallen from about 160 to a current level of 127 (96%tile) – still very high by most standards, but much lower than his previous functioning just a few years ago. Deficits were also noted in short-term memory with long term memory basically intact.

Two recommendations, among others, were made regarding cognition. The first was that his driving was a risk to self and others due to his perceptual-motor coordination problems. The second was that in most cases, given his I.Q. was higher than that of the average lawyer, he was probably capable of handling his financial affairs *if he remained sober*.

Of further interest to note is that most people do not start out with such a high I.Q., and if they did drop some 33 I.Q. points, from 160 to 127, as he did, they would be functioning at a below average I.Q. For example, on the WAIS-III, the average I.Q. is 100 (50th percentile). If an alcoholic abuser with an average I.Q. were to drop 33 I.Q. points to a 67, he would fall into the mildly mentally retarded range, thus indicating the power of alcohol to affect the brain negatively. As one grows older, the average non-alcoholic brain has a slower recovery and replacement of new brain cells, further impacting either permanent brain deficits or slower recovery (Reitan, 1978). Another factor is that the average non-alcoholic brain starts to replace fewer brain cells after the age of 35-40 resulting in a slower rate of recovery or more opportunity for permanent loss of brain cells (Reitan). He was hospitalized three years later due to alcohol abuse. His I.Q. had now dropped to 87 (19th percentile) after waiting a month before testing him. The long-term effect of alcohol abuse combined with advancing age cannot be underestimated.

Discussion

Treating such cognitive deficits as outlined above requires a systematic, comprehensive approach integrating different strategies. An excellent holistic approach is the BioPsychoSocial model of treatment.

At the biological level, therapists need to develop a good working relationship with physicians on alcohol abusers they are treating. Medical input is needed on complications often found in newly recovering individuals such as vitamin deficiencies

and possible organ damage. Medications may be needed to be prescribed for co-morbid conditions such as Post Traumatic Stress Disorder (PTSD), bipolar disorder, depression and/or anxiety. "A period of abstinence from two to eight weeks is optimal before the patient recovering from chemical dependency is diagnosed with an independent psychiatric disorder" (Jones, Knutson, & Haines, 2003, p.1976). A few weeks of sobriety helps sort out which symptoms are related to alcohol and/or a mental health disorder. Treating both disorders at the same time optimizes recovery. Often, newly recovering individuals need a complete physical to include blood work, urine analysis, and an assessment on their patient's ability to engage in exercise.

Pharmacological, administration of thiamine at sufficient levels can improve some brain functions often associated with frontal lobe damage and may need to be discussed with a physician regarding your patient (Martin, Singleton, & Hiller-Sturmhofel, 2003). Except for short-term memory, most of the deficits can be largely compensated via new neural growth through gaining new experiences depending on the age and prior history of alcohol use, duration and quantity consumed (Arria et al., 1991; Diamond, 1997). Exercise has been shown to improve cognition many ways to include the increased flow of endorphins in the brain and improved flow of natural dopamine generated by exercise (Sutoo & Akiyama, 2003). It is important to repeat to alcohol abusers that the brain can largely recover from alcohol abuse.

Sleep disturbances, like insomnia, are common among those in recovery from Central Nervous System (CNS) depressants like alcohol. Alcohol affects sleep patterns

by interfering with the monoamine neurotransmitters, which control the body's ability to sleep peacefully (Brower, 2001). Further, sleep is important to allow the brain to rest and repair itself from all the busy activities it engages in during the day (Winerman, 2006). In addition, sleep is critical in order for effective learning to occur. Sleep allows us to process, consolidate, and retain new memories and skills (Winerman, 2006).

As an aid to sleep improvement, medications such as the benzodiazepines, which create a dependency in the body, should be avoided in the treatment of substance abusers (Friedmann et al., 2003, Jones et al, 2003). Possible addictive medications include Librium, Valium, and Xanax. A case example was a well groomed, well dressed, distinguished looking middle class older lady who was referred by her new physician to the senior author for an evaluation concerning her use of Valium for sleep. When asked how long she had been using Valium, she replied it was since the death of her husband. She was then asked how long had it been since her husband passed away. She responded some 20 years ago and that her physician had prescribed Valium for her to get through the grieving process and help her sleep. This led to the patient describing daily use of Valium for the past 20 years and her development of a network of three physicians prescribing it for her during that time period. None of the physicians knew that she was being prescribed Valium by others. It only came to light when one of the physicians retired and she began a search to replace him. Her dependency on Valium became counter-productive for treating her grief and sleep issues.

"Sleep problems may occur during active drinking, acute alcohol withdrawal, and protracted withdrawal. Although most sleep abnormalities improve over time, some problems persist for months to years after initiating abstinence" (Brower, 2001, p.121). Sleep problems can be linked to potential relapses in alcoholics, and the abstinence and the duration of the abstinence period have been shown to be no guarantee for improvement in overall sleep, so the use *of non addictive medications* and a strict regiment may be needed (Brower). The cycle of sleep can be interfered with for up to *one or two years after detoxification* (Kern-Hage, 2004). In recovery, what is very frightening to many addicts is quitting alcohol/drugs produces a dream rebound effect, that is, a dramatic increase in dreaming occurs which may include a cycle of nightmares. Telling patients that nightmares will occur and will eventually subside will help lower their anxiety levels and decrease the likelihood of relapse.

As a therapist, it is important to educate the patient about sleep issues during recovery and ensure the physician involved is aware of the patient's problems with sleep. Explain to the patient that alcohol degrades sleep and is often associated with sleep deprivation. Thus, sleep disturbances may interfere with recovery and possible lifestyle changes needed to overcome the addiction. Learning or relearning how to sleep sufficiently thus needs to be a key issue in therapy. Here it is advisable that the "first line of treatment for chemical dependency should be non-pharmacological in nature" (Jones, Knutson, & Haines, 2003, p.1971). Complicating sleep is that chronic alcohol use

decreases melatonin production, which also leads to disruptive sleep cycles (Doweiko, 2006).

At the psychological level, use of Cognitive Behavioral Therapy (CBT) interventions for patients reporting significant sleep difficulties is effective as reported by the NIDA, the National Institute of Drug Abuse (2009). Consideration should be given to teach patients about sleep and recording of sleep patterns, keeping a weekly sleep diary developing sleep hygiene rules (limiting caffeine, naps, etc.) and sleep restrictions and stimulus control of the bedroom (Holcomb, 2006).

The longer an individual stays sober, the more likely that cognition will improve. For abstract thinking deficits, it is best for the therapist to focus on *concrete steps*, frequent check in with what the patient understood, and *repetition* of suggestions to improve the likelihood of maintaining recovery. Repetition of material discussed is often indicated. In a respectful fashion, ask the patient to repeat back what they understand of what has been discussed with them. It also is very helpful for the patient to summarize salient issues. It often produces surprises as to what you, as the therapist said, and what was heard. Thus, repetition not only allows for a check on comprehension and abstract skill learning, but also solidification of what has been covered and an opportunity to correct any misunderstandings. For example, a new patient was introduced in group therapy by the senior author. The comment was made by the therapist that group therapy was an opportunity for a person to learn from someone else what works and what does not. The author then quoted Benjamin

Franklin saying that "Any fool can learn from their mistakes, a wise man learns from other people's mistakes." The new patient said words to the effect of "I like that." The therapist then asked what he liked. His response was "I like the name Ben, my best friend's name is Ben…" What the patient understood of what was said is problematic at the abstract thinking level. His understanding of the proverb seemed lacking. Only by asking what he liked did a clarification occur which then allowed for follow up. Another clear sign that abstraction skills are at a depressed level is a lack of response to humor by some substance abusers. Humor requires the ability to see connections that require abstract thinking abilities. The good news is that humor often returns quickly after sobriety has been achieved and is a clear source of strength in the growth of many a substance abuser.

Delay of gratification (impulse control) is another important component of treatment for the patient to learn/relearn. An example is to resist the urge to act out impulsively at work and verbally lash out at a coworker or supervisor. The patient must be guided to think ahead of the consequences of such actions. A strategic tactic to employ is to tell the patient they need to protect their job. Clarify what protecting their job means in concrete terms. For example, pointing out that keeping their job is necessary to provide health benefits for themselves and their children. The more concrete examples of coping with job stressors provided, the more likely they will not only keep their job, but in many cases, may learn to enjoy it more. Perhaps the most famous example of teaching impulse control skills is the Alcoholic Anonymous (AA) concept of "one day at

a time." The concept of teaching the client to deal with the present, given there probably has been frontal lobe damage, is a powerful tool on gaining control over impulsive actions that lead to spiraling negative consequences. The classic AA example is not to drink today and call your sponsor (avoid impulsive action and engage executive frontal lobe functions). All these factors combine to create a less than efficient brain and decrease one's ability to help oneself. Thus cognitive treatment strategies must be implemented if recovery is to be successful.

Additionally, what research suggests is that increasing social support systems are very effective means of improving treatment outcome (Albee, 1997: NIDA, 2009**)**. What this translates to in dealing with substance abusers is helping them to find pro-social activities that increase their contact with others. Such activities are what Dr. Glasser calls "positive addictions" in his Reality/Choice therapy (Rapport, 2004). It offers the patient the opportunity to gain support from others and positive activities that help them grow as an individual. Replacing a negative behavior, such as drinking after work from 5 PM to 8 PM (a concrete example) can be replaced with a positive behavior such as attending an Alcoholics Anonymous (AA) meeting during that same time period and/or socializing afterwards with group members. Joining AA and getting an AA sponsor, group therapy, family therapy, becoming active in hobbies, clubs and/or a church help recovery. What is needed is helping the patient develop alternative ways to deal with life issues/stressors to minimize cognitive deficits and stimulate neural growth via support groups. Reality therapy is an excellent approach to

providing alternative positive behaviors to replace negative behaviors that promote drug abuse. Others may need more basic social support systems like vocational counseling, financial assistance, childcare help, finding shelter, and legal assistance (NIDA, 2009).

The authors acknowledge that cognitive deficits are also found by abuse of other drugs besides alcohol, polysubstance abuse, trauma (often associated with intoxication) and other factors. However, it is beyond the scope of this article to review all drugs and their impact on neuropsychological functioning. What is clear is that cognitive deficits due to drug abuse contribute to relapse and decrease treatment options unless dealt with directly. What is very rewarding to both the therapist and the patient is that there is usually some quick improved cognition in only a few weeks in patients once they have begun the process of recovery. This probably accounts in part for therapists' continued motivation to work with substance abusers as they witness the rebirth of hope.

Other CNS depressants seem to have these same or similar deficits. For example, long term use of marijuana produces cognitive deficits similar to those found in individuals who abuse alcohol. The most frequently found cognitive deficits due to chronic marijuana use appear to be slower psychomotor performance, poorer perceptual motor coordination, and memory dysfunction (Martin, 2002). It may be that long-term memory impairment results from chronic marijuana use. After many years of use, this results in subtle cognitive deficits to attention, and the organization and

integration of complex information. Other CNS depressants such as barbiturates, benzodiazepines, and opioids, also have similar cognitive deficits associated with their use. More research needs to be done to seek out treatment approaches that realize cognitive deficits contribute significantly to treatment outcome.

CNS stimulates have also been implicated in cognitive disorders (Bhatia & Marsden, 1994). Pregnant women using cocaine causes prenatal cocaine exposure of infants, which directly correlates during the first two years with these infants developing increased risks of cognitive impairment (Arendt et al., 2002). Cocaine abusers have also developed neuropsychological deficits such as compromised "cognitive flexibility, visual tracking, and speed of information processing" (Smelson, Roy, Santana, & Engelhart, 1999. p. 1). Supportive findings by Taper et al (2004) found cocaine abusers suffered from deficits in executive function, nonverbal memory, and visuospatial skills. NIDA (2003) research also indicates human brain damage due to Methamphetamine abuse.

Conclusion

The frontal cortex region which is responsible for executive functioning is highly susceptible to damage by alcohol (Duke University Medical Center, 2006). Damage compromises the ability to plan ahead, inhibition (delay impulsive actions) and the ability to think out consequences of one's actions. Thus, treatment must focus immediately on frontal cortex functions that have been compromised by alcohol. The length of time that these deficits in cognition will continue after gaining sobriety is not

known because alcohol affects the executive functioning both acutely and chronically. Residual effects among people who have stopped drinking may continue for years thus limiting the individual's ability to strategize and plan effectively particularly in the areas of short-term memory and perceptual motor coordination. Treatment must then take into account not only treating the alcoholic but having spouses, children and other social contacts involved in the recovery process to help increase the patient's chances of success. Family members may need to act as frontal lobes for the patient and help re-establish executive control over impulsivity and planning ahead for consequences of immediate actions in the future.

Some of these cognitive deficits may become permanent, depending on the drug(s) abused, frequency of use, amount consumed, durations of the abuse, and age and health of the patient (Arria et al, 1991: Parsons et al, 1987). Recovery is a lifetime process. Thus, job and personal issues need revisiting and modification as time goes by and situations change for the patient.

With some exceptions, the most significant treatment tool available to clinicians is **to educate their clients** on such information as given above. Such education paves the way for other changes the client needs to make to survive the after-effects of alcohol during recovery.

Similar deficits are often found for other drugs of abuse and for polysubstance abusers on cognition. Other factors also, of course, such as poor nutrition habits, older age and low physical conditioning contribute to the development of cognitive deficits.

If these factors are improved, increased efficient cognitive functioning is possible in most cases. In future studies, issues of special populations must also be taken into consideration. For example, women react differently to alcohol abuse and tend to become dependent quicker than men and experience physiological changes different from men (Nichol, Krueger, & Iacono, 2007). These findings have implications for the treatment of women as to whether their cognitive deficits may be different in some ways from men. More research is needed to find out how to tailor treatment to cognitive deficits and if they vary by special populations.

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