DOWNLOAD PDF COGNITIVE IMPAIRMENT IN ALCOHOLICS

Chapter 1 : Alcohol use in mid-life & cognitive impairment - Gray Matter Therapy

Cognitive Impairment and Recovery From Alcoholism 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 (i.e., mental activities that involve acquiring, storing, retrieving, and using information) (1).

Even mild-to-moderate drinking can adversely affect cognitive functioning i. Persistent cognitive impairment can contribute to poor job performance in adult alcoholics, and can interfere with learning and academic achievement in adolescents with an established pattern of chronic heavy drinking 2. A small but significant proportion of the heaviest drinkers may develop devastating, irreversible brain-damage syndromes, such as Wernicke-Korsakoff syndrome, a disorder in which the patient is incapable of remembering new information for more than a few seconds 3. It stands to reason that cognitive impairment also may impede recovery from alcoholism, although evidence has not conclusively shown this to be the case. They found that impairment was not a significant predictor of poor treatment response. On the other hand, evidence does support the possibility that brain damage, whether resulting from or predating alcohol use, may contribute to the development and progression of alcoholism 5. Designing practical strategies to cope with the complex combination of alcoholism and cognitive impairment requires an understanding of the nature of cognitive functions and their interactions with structural and functional brain abnormalities. This issue of Alcohol Alert describes the nature and consequences of common alcohol-associated cognitive defects, explores the extent to which some cognitive abilities recover with abstinence, and summarizes recent research on the effects of cognitive deficits on alcoholism treatment outcome. Cognition and Alcohol Most alcoholics exhibit mild-to-moderate deficiencies in intellectual functioning 6 , along with diminished brain size and regional changes in brain-cell activity. The most prevalent alcohol-associated brain impairments affect visuospatial abilities and higher cognitive functioning 7. Visuospatial abilities include perceiving and remembering the relative locations of objects in 2- and 3-dimensional space. Examples include driving a car or assembling a piece of furniture based on instructions contained in a line drawing. Higher cognitive functioning includes the abstract-thinking capabilities needed to organize a plan, set it in motion, and change it as needed 2. Most alcoholics entering treatment perform as well as nonalcoholics on tests of overall intelligence. However, alcoholics perform poorly on neuropsychological tests that measure specific cognitive abilities 8. For example, an alcoholic who has remained abstinent after treatment may have no apparent difficulty filing office documents correctly, a task that engages multiple brain regions. However, that same person might be unable to devise a completely different filing system, a task closely associated with higher cognitive functioning. How Much Is Too Much? The link between duration and lifetime quantity of drinking and the development of cognitive problems is unclear. Some investigators have proposed that cognitive performance worsens in direct proportion to the severity and duration of alcoholism 6,9. Studies suggest that social drinkers who consume more than 21 drinks per week also fit into this category 6. Other investigators have suggested that cognitive deficits may be detectable only in those alcoholics who have been drinking regularly for 10 years or more 8, Long-term, light-to-moderate social drinkers have been found to fall into this category as well, showing cognitive deficits equivalent to those found in detoxified alcoholics 8. Tracking Structural and Functional Brain Abnormalities Structural and functional brain abnormalities generally are measured by noninvasive imaging techniques that provide a picture of the living brain with minimal risk to the individual. Structural imaging techniques, such as computed tomography and magnetic resonance imaging, are used to generate computerized pictures of living tissue. Functional imaging techniques, such as positron emission tomography and magnetic resonance spectroscopy, permit scientists to study cell activity by tracking blood flow and energy metabolism. For more information about imaging, see Alcohol Alert No. A Window on the Brain. Loss of brain volume is most noticeable in two areas: Support for these results is provided by functional imaging studies, which reveal altered brain activity throughout the cortex and cerebellum of heavy drinkers In addition, functional imaging often is sufficiently sensitive to detect these irregularities before they can be observed by structural imaging techniques, and even before major cognitive problems themselves become manifest. This suggests that

functional imaging may be particularly useful for detecting the early stages of cognitive decline Understanding the Basis of Cognitive Impairment Accurate measurement of cognitive abilities is challenging, and relating those abilities to a specific brain irregularity simply may not be possible with the current technology Discrepancies among research findings have led scientists to develop improved cognitive-measuring techniques. Using a battery of tests, Beatty and colleagues 9 have suggested that widespread i. Likewise, damaging the network of brain cells that synchronizes the overall activity of those multiple areas may produce the same cognitive impairments previously attributed to localized damage 9. Certain alcohol-related cognitive impairment is reversible with abstinence Newly detoxified adult alcoholics often exhibit mild yet significant deficits in some cognitive abilities, especially problem-solving, short-term memory, and visuospatial abilities By remaining abstinent, however, the recovering alcoholic will continue to recover brain function over a period of several months to 1 year 19 -with improvements in working memory, visuospatial functioning, and attention-accompanied by significant increases in brain volume, compared with treated alcoholics who have subsequently relapsed to drinking Rewiring Brain Networks Reversibility of alcohol-related cognitive function also may be the result of a reorganization of key brain-cell networks. Some researchers have proposed that such reorganization may contribute to the success of alcoholism treatment. Using advanced imaging techniques, Pfefferbaum and colleagues 20 examined the brain activity of cognitively impaired alcoholic participants during a series of tests designed to assess cognitive function. They found that although the alcoholic subjects had abnormal patterns of brain activation, compared with control subjects, they were able to complete the tasks equally well, suggesting that the brain systems in alcoholics can be functionally reorganized so that tasks formerly performed by alcohol-damaged brain systems are shunted to alternative brain systems. This finding-that cognitively impaired alcoholic patients use different brain pathways than unimpaired patients to achieve equivalent outcome-also was suggested in a study of patients in step treatment programs 4. Functional brain reorganization may be particularly advantageous for adolescent alcohol abusers in treatment, because their developing brains are still in the process of establishing nerve-cell networks Cognitive Function and Alcoholism Treatment The exact role that cognitive function has in alcoholism treatment success is unclear. Structural and functional imaging, as well as more specific cognitive tests, may provide scientists with the tools needed to reveal subtle relationships between alcohol-related cognitive impairment and recovery. Meanwhile, certain conclusions can be drawn from existing research that help to explain how cognitive function may influence alcoholism treatment: Cognitive deficits have been hypothesized to affect the efficacy of alcoholism treatment, although a clear association has not been established. One view finds that cognitively impaired patients may not be able to comprehend the information imparted during therapy and, thus, may not make full use of the strategies presented, thereby hampering recovery. Another view is that cognitive functioning may not directly influence treatment outcome, but may impact other factors that, in turn, contribute to treatment success Other types of non-alcohol-related brain damage also can produce symptoms resembling those associated with chronic alcoholism. Cognitive impairment is usually most severe during the first weeks of abstinence, perhaps making it difficult for some alcoholics to benefit from educational and skill-development sessions, which are important components of many treatment programs 22, For example, one study found that alcoholics tested soon after entering treatment were unable to recall treatment-related information presented in a film they had just been shown 4. As time goes by and cognitive function improves, however, patients may make better use of information presented to them in individual and group therapy, educational programs, and step programs. The new noninvasive imaging techniques that allow us to "see" how the brain operates have been a boon to the study of cognition. Through this medium, we now know that the brain is capable of "rewiring" itself. In doing so, the brain can regain some of the cognitive abilities previously diminished as a result of damage from alcohol or other diseases. First, alcohol use over a period of time, even at low levels of drinking, can produce varying degrees of cognitive damage, a problem that is of particular concern because alcohol use is so widespread. Recent evidence suggests that the adolescent brain, which is still forming important cellular connections, is more vulnerable than the adult brain to alcohol-induced damage. This is particularly troubling, given the problems associated with chronic binge drinking, which is all too common among young people. References 1

Evert, D. An overview of how alcoholism may affect the workings of the brain. Alcohol Health Res World 19 2: Executive cognitive functioning in alcohol use disorders. Recent Developments in Alcoholism: The Consequences of Alcoholism. Alcohol Health Res World 14 2: Effects of executive function impairment on change processes and substance use outcomes in step treatment. J Stud Alcohol 60 6 , Neurotoxicity and neurocognitive impairments with alcohol and drug-use disorders: Potential roles in addiction and recovery. Alcohol Clin Exp Res 25 2: Neurocognitive deficits in alcoholics and social drinkers: Alcohol Clin Exp Res 22 4: Impairments of brain and behavior: The neurological effects of alcohol. Alcohol Health Res World 21 1: Cognitive functioning in sober social drinkers: A review of the research since J Stud Alcohol 59 2: Neuropsychological deficits in sober alcoholics: Influences of chronicity and recent alcohol consumption. Alcohol Clin Exp Res 24 2: Effects of moderate alcohol consumption on the central nervous system. Alcohol Clin Exp Res 22 5: Brain CT changes in alcoholics: Effects of age and alcohol consumption. Alcohol Clin Exp Res 12 1: Brain gray and white matter volume loss accelerates with aging in chronic alcoholics: A quantitative MRI study. Alcohol Clin Exp Res 16 6: Exp Clin Psychopharmacol8 2: Alcohol and the cerebellum: Effects on balance, motor coordination, and cognition. Imaging studies of aging, neurodegenerative disease, and alcoholism. Alcohol Health Res World 19 4: Determinants of cognitive deficits in alcoholics: Clin Neuropsychologist 8 1:

Chapter 2 : Cognitive Impairment and Recovery From Alcoholism | Terry Gorski's Blog

Abstract. Impaired cognitive functioning in alcoholics is widespread during the first months of detoxification. Between half and two thirds of abstinent alcoholics exhibit cognitive impairments during this period, with residual deficits persisting for years after detoxification in some patients.

These cognitive impairments not only determine everyday management of these patients, but also impact on the efficacy of management and may compromise the abstinence prognosis. Maintenance of lasting abstinence is associated with cognitive recovery in these patients, but some impairments may persist and interfere with the good conduct and the efficacy of management. It therefore appears essential to clearly define neuropsychological management designed to identify and evaluate the type and severity of alcohol-related cognitive impairments. It is also essential to develop cognitive remediation therapy so that the patient can fully benefit from the management proposed in addiction medicine units. Alcoholism causes a multitude of social and health problems with negative impact on quality of life and secondary costs to society 1 â€” 3. Evidence suggests that numerous determinants such as environmental, individual, and genetic factors could favor evolution toward alcohol-dependence. These factors may also interact with each other. Among environmental factors, quality of the neighborhood 4 or socio-economic factors e. Individual and psychological characteristics including comorbid psychiatric disorders 7 , 8 , early life stress exposure 9 , or impulsivity 10 are also risk-factors associated with chronic alcohol consumption. In addition, family, twin, and adoption studies have highlighted that genetic factors play an important role in the pathogenesis of alcohol-dependence 11 â€” In the same way, recent studies have examined cognitive endophenotype in alcoholism. They have shown that non-alcoholic relatives of alcohol-dependent individuals performed worse on cognitive tasks specifically executive functions and presented greater impulsiveness compared to control [e. From a neurobiological perspective, alcohol-dependence is a chronic disorder, which implies the dopaminergic system. As seen in other drugs abuses, alcohol consumption acutely stimulates dopamine DA release from the major terminal area of the mesolimbic DA system, nucleus accumbens NAC. Enhanced DA transmission in the NAC plays a critical role in the positive rewarding aspects of drugs abuses and the initiation of addictive process. Chronic administration is associated with functional alterations of this important part of the brain reward system. Globally, dysregulation of the dopaminergic system caused by chronic alcohol consumption produces drug dependence reinforcement and is most likely involved in the development of drug addiction 16 â€” The harmful effects of chronic alcohol consumption on the brain and cognitive functioning have been well described in the literature over recent decades These impairments are moderate to severe but usually remain undiagnosed when they are not specifically investigated. However, detailed neuropsychological assessment or screening of these cognitive impairments appears to be fundamental to optimally adapt patient management strategies. Alcohol-Related Cognitive Impairments Neuroanatomical alterations can account for cognitive impairments affecting various functions, primarily executive functions However, there is a marked interindividual variability of the nature and severity of these impairments. More specifically, the dysexecutive syndrome can affect various processes such as working memory 24 , mental flexibility 25 , divided attention 26 , decision-making 27 , 28 , or problem-solving The characteristic profile of alteration of episodic memory in alcohol-dependent patients comprises limited learning capacities, impairments of encoding, and recollection processes, difficulties recalling the temporospatial context and deficits of autonoetic consciousness, while information storage is preserved 25 , Alteration of executive functions, particularly disorders of inhibition, flexibility, or dual-task coordination also constitute predictive factors of memory impairment 25 , In contrast, apart from obvious deficits i. Finally, visuospatial functions are also predominantly affected, as several studies have demonstrated impaired performances on visuospatial processing, memory and visual learning, visuospatial organization, and visuoconstruction tasks 31 , Three main hypotheses have been proposed in the literature to account for the characteristic cognitive profile observed in alcohol-dependent patients [see Ref. The first hypothesis is based on the pervasiveness and impaired recovery after withdrawal of visuospatial cognitive functions that are attributed to the non-dominant hemisphere. The right hemisphere would therefore

be more susceptible to the neurotoxic effects of alcohol However, this postulate has been questioned by contradictory results The second hypothesis proposes that the increased susceptibility of frontal structures would account for the cognitive profile dominated by executive deficits However, this hypothesis also appears to be too restrictive, as other cerebral structures are also involved The third hypothesis, based on neuroanatomical and neuropsychological data, reconciles the previous two hypotheses by postulating the existence of global brain damage In their meta-analysis of neuroanatomical data, the authors emphasized not only the existence of right hemisphere and frontal lobe lesions, but also lesions affecting other cerebral regions medial temporal, subcortical, and cerebellar atrophy. The neuropsychological functional deficits observed in these patients also concern several cognitive functions in both verbal and visual modalities, which constitutes an additional argument in favor of the global brain damage hypothesis. Anatomical Lesions Chronic excessive alcohol consumption induces global brain atrophy characterized by reduction of brain volume and enlargement of the ventricles and sulci The severity of brain damage depends on various factors such as the extent of alcohol consumption, age, gender, and neurological or psychiatric comorbidities The most susceptible brain structures are the neocortex in the frontal lobes, the limbic system, and the cerebellum Reduction of gray matter preferentially involves frontoparietal regions, while reduction of white matter tends to be more diffuse More precisely, Kril et al. Therefore, these results indicate an increased susceptibility of the brain in the elderly according to the model of alcohol-related premature aging of the brain Furthermore, many studies have also demonstrated functional changes with a reduction of glucose metabolism or cerebral blood flow in prefrontal regions, particularly in the medial temporal region Alcohol-related brain damage also concerns the limbic system and particularly the hippocampus, regions involved in episodic memory 41 â€” Finally, the cerebellum is also affected in these patients, with a reduction of the white matter volume in the vermis and cerebellar hemispheres A study of the connections between the cerebellum and frontal regions via the pons and thalamus also demonstrated alteration of the frontocerebellar circuit Detection and Diagnosis In this context, the detection of cognitive impairments in alcohol-dependent patients is therefore essential and should be systematic. The Montreal Cognitive Assessment MoCA Test appears to be the most appropriate screening test for detection of cognitive impairments in these patients 45 , as this tool is more sensitive than the Mini Mental State Examination MMSE for mild-to-moderate cognitive impairments The MoCA Test can be performed by medical students, medical doctors, or certified neuropsychologist. Detection of cognitive impairments can then lead to referral of the patient for neuropsychological diagnostic assessment performed by a clinical neuropsychologist. Neuropsychological assessments can last 2â€”3 h and are designed to demonstrate preservation or impairment of the most susceptible cognitive functions in this population. Clinical neuropsychologists have at their disposal a battery of tests to evaluate several cognitive domains such as memory e. In the alcohol-dependent population, the most susceptible executive processes to be evaluated are working memory, mental flexibility, inhibition, processing speed, concept formation, planning, and problem-solving capacities. Evaluation of verbal and visual memory must focus on encoding, recall, storage, learning, and recognition capacities, while assessment of visuospatial functions must focus on visuospatial organization and visuoconstruction capacities. Alcohol Consumption and Implicit Cognitive Processes The study of the implicit mechanisms involved in addictive behavior has been considerably developed over recent years. The addictive problem of alcohol-dependence results from a conflict between an urge to drink and the desire to limit alcohol intake. Dual-process models can explain this conflict by the fact that drinking behavior involves two cognitive systems: This system therefore reinforces the incentive effects between stimuli related to the addiction odors, places of consumption, or advertisements related to alcohol, for example and the addictive behavior. It mobilizes the dopaminergic system of the amygdalaâ€”striatal circuit. The underlying network involves various regions of the frontal lobe lateral inferior prefrontal, dorsolateral, ventromedial, orbitofrontal, and frontoparietal and the striatum. Finally, the insula plays a decisive role in the articulation of these two systems by translating unconscious interoceptive signals or somatic states into conscious subjective experiences desires or needs involved in the decision-making process. This system would therefore play a conflict management role between a stimulus related to addiction and a potentially associated somatic state for example withdrawal symptoms in order to guide decision-making. The interaction between the two systems

has been clearly documented in the field of alcohol-dependence According to this theory, drinking behavior is activated by automatic processes impulsive system unless the subject is able to ensure control by mobilizing executive functions reflective system. The impairment of executive functions observed in alcohol-dependent patients would therefore predispose to drinking behavior dictated by the impulsive system. More precisely, disorders of inhibition capacities and working memory play a predominant role in this dysregulation of the impulsive system by the reflective system 28 , 47 , 49 â€” 54 , thereby resulting in a vicious circle, as chronic excessive alcohol consumption induces working memory and inhibition disorders that are then responsible for dysfunction of the reflective system. Finally, alcohol consumption results exclusively from mobilization of the impulsive system that perpetuates the addictive behavior resulting in continuing deterioration of executive functions Cognitive and Brain Recovery The study of alcohol-dependent patients also constitutes a model of brain plasticity, as an increase of brain volume characterized by increased white matter and gray matter volumes and a reduction of the size of sulci and ventricles is observed right from the first months of abstinence 55 â€” The cognitive effects of this recovery consist of improvement of executive functions and verbal episodic memory 21 , 58 , 60 â€” In parallel, it has been shown that new brain regions can be recruited by recently weaned alcohol-dependent patients to compensate for alcohol-related brain damage 66 , Neuroadaptation mechanisms therefore enable patients to maintain a similar level of performance on cognitive tasks to that of control subjects. For example, alcohol-dependent patients recruit neuronal networks parallel to the frontocerebellar circuit normally used by control subjects to perform executive tasks However, although abstinence allows an improvement of cognitive functions, this is only achieved after a period of several months. A recent meta-analysis showed that, despite studies showing early cognitive recovery, a global deficit was still present several months after installation of abstinence and the cognitive profile tended to become normal only after 1 year of abstinence, while certain residual cognitive impairments may persist. For example, the presence of visuospatial function deficits may be observed after several years of abstinence, related to the decreased volume of the right parietal cortex Cognitive Impairments and Management of Alcohol Abuse Appropriate management of alcohol withdrawal is mandatory to prevent severe complications like delirium tremens or epileptic seizure. If benzodiazepines usage and appropriate rehydration are codified, the dose and duration of thiamine treatment remains unclear Nevertheless long-term abstinence is the main goal and cognitive behavioral therapy CBT and psychosocial programs are necessary. Adjuvant treatments may include: The presence of cognitive impairments therefore requires adaptation of the management of alcohol-dependent patients. CBT has been demonstrated to be effective in the management of alcohol-dependence 70 , but it is somewhat paradoxical to propose management that directly involves cerebral structures and cognitive functions altered by chronic alcohol consumption. This management approach may therefore be inappropriate or at least insufficient for a certain number of patients. The efficacy of CBT would therefore depend on the integrity of certain brain regions of interest. For example, it has been shown in schizophrenic patients that the volume of gray matter in the frontal, temporal including hippocampus , parietal, and cerebellar regions, brain regions that are also damaged in alcohol-dependent patients, is predictive of the efficacy of management Similarly, the integrity of the frontocerebellar network, a site of predilection for brain damage in alcohol-dependent patients, would play an essential role in the efficacy of CBT due to its role in executive functioning Cognitive behavioral therapy in addiction medicine also requires elaborate cognitive capacities such as episodic, semantic and procedural memories, and executive functions 23 , 73 â€” This type of management could therefore be unsuitable for patients with cognitive impairments 25 , 77 â€” Various studies have shown that alcohol-dependent patients with the most severe cognitive impairment also have the least favorable prognosis 80 â€” Cognitive impairment can also influence the expression of individual and environmental factors involved in management, such as self-efficacy, readiness to change, active participation in group therapy, or treatment compliance, as the initial cognitive impairment is predictive of poorer treatment compliance and a decreased self-efficacy. Patients with severe cognitive impairments are also less able to use their own resources during management, in which case the prognosis depends more on the role of external factors such as group therapy or the family support network Finally, Le Berre et al. Similarly, the integrity of executive functions enables patients to weigh up their decisions to reach the action stage, which can only be

implemented when decision-making capacities are preserved. Cognitive impairment therefore influences the degree of motivation of alcohol-dependent patients, an essential prerequisite to the success of management. The presence of cognitive impairments on admission and during the first months of abstinence therefore influences management at various levels by determining the efficacy of treatment and the prognosis for lasting abstinence. Addiction medicine management must therefore be adapted to alcohol-dependent patients with cognitive impairments. Cognitive Remediation Therapy In the light of these findings, it appears essential to propose management based on programs ranging from cognitive remediation to optimal use of the remaining capacities. However, very few addiction medicine units propose cognitive remediation therapy and very few studies have investigated this problem in alcohol-dependent patients. The majority of studies in the field are now relatively old 77 , 79 and no longer correspond to current methodological requirements. However, the results of studies conducted in this field are encouraging.

Chapter 3 : Practical Neurology - The Cognitive Consequences Of Alcohol Use (October)

Cognitive impairment only worsens with continued drinking and approximately 45 to 70 percent of individuals suffering from alcoholism have impaired cognitive functioning, together with deficits in problem solving, abstract thinking, concept shifting, psychomotor performance and memory.

Terry Gorski I first introduced the concept of Post Acute Withdrawal PAW in training programs that I presented starting in and the concept was originally published in the first edition of book Learning To Living Again â€" A Guide for Recovery From Alcoholism The concept of PAW was based upon a combination of clinical experience with relapse-prone alcoholics and a small but growing literature published by the National Institute of Alcohol Abuse and Alcoholism NIAAA which was suggesting long-term neuropsychological impairment in recovering alcoholics. These impairments made it difficult for alcoholics to respond to traditional addiction counseling, even when advances in the cognitive therapy field were specifically adapted to alcoholism treatment. The client got stuck in recovery was unable to progress in recovery, and became increasing frustrated. Symptoms if increased stress were obvious, but clients were unable to respond to the relaxation and stress management training which was just starting come of age. I coined the word stuck point to describe this inability to progress in recovery in spite of attempts to do so. Once the client got stuck their stress increased and a predictable pattern of early warning signs became apparent. In a future article I will present the independent research which resulted in the AWARE Questionnaire, which confirmed and refined the ability to recognize the early warning signs of relapse. The stuck point led to increased stress. The high stress, in turn, increased frustration and a morbid fear of inevitable failure. Since alcohol and drug use had be so destructive in the past, the fear of relapse activated a survival threat seemed to decrease the ability to think clearly and learn new information and skills. The relapsing people usually failed to understand what was happening and judged themselves as dumb, stupid, and unable to recover. Therapist often failed to recognize the neuropsychological basis of the symptoms, mislabeled it as denial and resistance and proceed to confront the client. The confrontation just made things worse. Other therapists decided the neurocognitive impairment was caused by unresolved family of origin problems and used deep relaxation and guided imagery to resolve trauma from previous physical or sexual abuse. This of course, was the hallmark of the codependency era. The use of confrontation, regressive hypnosis, and catharsis techniques all increased the stress of clients, lowered their self-esteem by making them feel crazy, and decreased their self-confidence. This lack of self-confidence or absence of the positive belief in their ability to successfully recover was later dubbed low efficacy by cognitive therapists such as Alan Marlatt, Dennis Donovan, and Dennis Miller. Convincing evidence began to develop that low efficacy was directly related with an increase in early relapse warning signs and the eventual failure to maintain recovery. The final trigger event was a high risk situation which activated craving in an environment that reinforced alcohol use while removing support for ongoing recovery. Alan Marlatt observed the same thing. He originally described the phenomenon of apparently irrelevant decisions, which, in essence, described the same phenomena that I described as early relapse warning signs. Both concepts were pointing at the same thing â€" symptoms caused progressive neuropsychology or neurocognitive dysfunction related to the stress of feeling stuck in a dysfunctional state and slowly becoming so dysfunctional and having no effective way to manage the growing inability to function. This produced a survival threat. We now know that high levels of stress activates the amygdala in the brain. The amygdala would shift behavioral control from slow-moving conscious decision-making to automatic use of emergency survival skills. I labeled this progressive stress-induced dysfunction as the relapse syndrome. The bottom line is this. In a compelling body of evidence exists that there is progressive brain dysfunction in alcoholic clients. This brain dysfunction is measureable and supported in the scientific literature. This evidence is summarized in the following Alcohol Alert. Even mild-to-moderate drinking can adversely affect cognitive functioning i. Persistent cognitive impairment can contribute to poor job performance in adult alcoholics, and can interfere with learning and academic achievement in adolescents with an established pattern of chronic heavy drinking 2. A small but significant proportion of the heaviest drinkers may develop devastating, irreversible brain-damage syndromes,

such as Wernicke-Korsakoff syndrome, a disorder in which the patient is incapable of remembering new information for more than a few seconds 3. It stands to reason that cognitive impairment also may impede recovery from alcoholism, although evidence has not conclusively shown this to be the case. They found that impairment was not a significant predictor of poor treatment response. On the other hand, evidence does support the possibility that brain damage, whether resulting from or predating alcohol use, may contribute to the development and progression of alcoholism 5. Designing practical strategies to cope with the complex combination of alcoholism and cognitive impairment requires an understanding of the nature of cognitive functions and their interactions with structural and functional brain abnormalities. 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Chapter 4 : Alcohol misuse and cognitive impairment in older people: An exploratory study

Certain alcohol-related cognitive impairment is reversible with abstinence (17). Newly detoxified adult alcoholics often exhibit mild yet significant deficits in some.

Nonetheless, one of the most intriguing aspects of this topic from a neurological perspective is the cognitive impact of chronic mild to moderate continuous alcohol use and binge drinking. This topic has become more important because a significant number of patients over 65 are developing cognitive decline and experts in addiction medicine are beginning to realize that cognitive function is important in management of these individuals. According to various studies, 50 to 80 percent of these individuals present with impaired cognitive function. On the contrary, a good deal of information exists regarding cognitive function and alcohol use by itself. In an article published in Neurology last year, authors concluded that regular and episodic drinking were not consistently associated with cognitive function. This certainly challenges the notion that mild to moderate drinking is healthy for your heart and brain. Ahead is a brief survey of literature on the topic. Alcohol and Cognition One of the most comprehensive studies on alcohol is a meta-analysis of papers from countries covering It describes the relationship between moderate drinking of alcohol and aspects of cognition. They also determined that cognitive testing varied between two different eras: In studies in which no ratios and standard neuropsychological testing was used in , subjects younger than 55 years old, heavy drinking of four to six drinks per day was associated with cognitive impairment and higher risk for dementia. Notably, no significant difference in cognition was observed between light to moderate drinkers and non-drinkers. Defining Cognitive Impairment in Heavy Drinkers There are four profiles of cognitive impairment in heavy drinkers1: No cognitive impairment 2. Isolated executive deficits with normal memory and global cognitive efficiency 3. Mild executive dysfunction with memory impairment and preserved global cognitive efficiency 4. Global impairment executive function, memory and impaired cognitive efficiency. These impairments can generally affect working memory, mental flexibility, attention, decision making, problem solving, processing speed, and planning. Encoding and retrieval tend to be affected most, while memory storage was normal. Visual spatial impairment is also predominantly affected, as studies have shown impaired visual spatial processing, visual learning, and visual spatial construction tasks. Ihara H et al, Group and case study of the dysexecutive syndrome in alcoholism without amnesia. Pitel AL et al, Genuine Episodic memory deficits and executive dysfunction in alcohol subjects early in abstinence. Alcohol Clinical Experimental Research. Neuropsychological review Acta Neurol Scand ; In studies with ratios of risk in patients older than 55 years of age, 87 percent used MMSE. Eighty percent of these studies took place since in multiple countries. These studies showed a decreased risk of dementia and cognitive impairment in light to moderate drinkers in older adults by 20 percent, but no significant benefit against rate of cognitive decline. It is worth noting that this analysis was subject to criticism because some of the studies it used included previous drinkers that quit, which can bias the results. Nineteen of the ratio studies excluded these former drinkers. The meta-analysis showed that most studies did not distinguish the type of alcohol used. Some studies said wine was better, whereas others found no difference between beer or spirits. Of course, another inherent criticism is that male and female drinkers were combined for analysis and it is known the two sexes have different drinking patterns, which may mask a genuine difference among types of alcohol. In this meta-analysis, 23 ratios in the worse cognitively impaired group were carefully reviewed to see why mild to moderate drinking was associated with worse cognitive impairment. Another notable aspect about the meta-analysis is that accounting for age, education, sex, and smoking produced no change in the alcohol effects. There was also no difference in outcome between longitudinal or cross sectional studies. Mental status exams were more often associated with finding better cognition in drinkers while neuropsychological tests were more often associated with no difference in cognition between drinkers and non- drinkers. Importantly, the findings suggested that moderate drinking had no impact on dementia in general, AD, or vascular dementia. Other meta-analyses have failed to find a significant benefit of alcohol use against cognitive decline, as well. As for why the MMSE test results were positive in reducing cognitive risk of decline compared to neuropsychological testing group in

light to moderate drinkers, the authors pointed out that because many studies used MMSE to increase the validity of the results, while 60 percent of the studies used additional measures of cognition, the judgement of dementia and cognitive impairment were more reliable. The authors also stated that heavy drinking i. Light to moderate drinking of two drinks per day or less in adult men and one drink or less in adult women, does not increase risk of cognitive decline, cognitive impairment, or dementia. These results were echoed by findings from another comprehensive review evaluating the same factors. Participants completed a self-administered questionnaire followed by clinical exams over future years from to four assessments. Mean alcohol consumption was calculated for each participant via questions on frequency and amount of alcohol use and which source of alcohol consumed. Investigators used cognitive testing in the middle of the study ages and repeated in , and age range Short-term memory and executive function fluency and inductive reasoning, Math, etc. At inception, roughly 7, out of 10, participants participated at least one of three cognitive assessments and constituted the analytic sample. Thirteen percent participated in the first wave of cognitive studies, This was more evident in men than women. Abstinence from drinking was associated with faster decline in global cognitive score and executive function. The number of abstinence drinkers, however, was small and included more women and non-white individuals, which may have altered the data. Alcohol consumption of 36 grams per day heavy or greater was associated with faster cognitive decline in all cognitive domains compared with consumption of 0. In women, however, compared with those drinking 0. Of note, the data were not driven by a single type of alcohol. While heavy drinking has been associated with cognitive impairments in addition to other detriments to health, there is some evidence to suggest that moderate alcohol consumption may have a positive impact. For example, one study found that low to moderate drinking reduced the risk of Myocardial Infarction 0. The main theory focuses on cerebral and cardio-pathways that play out over extended period of time. Britton A et al, Who benefits most from the cardioprotective properties of alcohol consumption: Journal Epidemiology and community health. Epidemiological considerations and mechanistic studies. Alcohol clinical experimental research ; Association of alcohol consumption with selected cardiovascular disease outcomes: A systemic review and Met-analysis. Et al, Dementia after stroke: Panza F et al, Alcohol consumption in Mild cognitive impairment and dementia: International journal of geriatric society. Et al, Reported alcohol consumption and cognitive decline: The northern Manhatten study. National Review of Neurology ; 7: The investigators looked at 28, men and women aged years old. Re-exam was done in 63 percent response rate. The cognitive testing included ten-word recall, verbal fluency, letter cancellation test to measure attention, mental speed, and concentration. They had complete cognitive data in 17, at the end of the study in Women who were non-drinkers had lower cognitive scores than light drinkers. Better cognitive scores in moderate drinkers was attenuated adjusting for social, economic, and lifestyle factors. Quantity and frequency of drinking were not associated with cognitive performance in men. First, the analytic sample was restricted to participants with cognitive measures at follow-up. Attrition was higher in participants with lower cognitive scores, non-drinkers, and frequent drinkers at baseline, which could possibly bias the results of the study. Also, self-reported alcohol measures typically underestimate actual consumption. In this study, heavy drinkers had lower participation rates and higher likelihood of cognitive impairment, suggesting that heavy drinking and cognitive function were underestimated. The other concern about this study is that the follow-up period was only three years, which may not have been a long enough time to examine trajectories of cognitive decline. Not specifically addressed in the majority of these studies was binge drinking. This topic was specifically evaluated in a study in which investigators studied Finnish Twins who provided data on alcohol consumption in questionnaires in and and were followed for 25 years. The assessments were done by telephone interviews, which have shown a strong correlation with mental assessment face to face. By the end of the follow-up, participants developed dementia, however, the cause of dementia could not be determined from the telephone interview. The study considered binge drinking as five bottles of beer or one bottle of wine on one occasion at least monthly. This was associated with a relative risk of 3. Also, blacking out during the heavy drinking periods at least twice during the previous year as reported in was associated with a with a higher risk for dementia in drinkers. The study found that binge drinking and passing out were risk factors even after controlling for alcohol consumption or after excluding from the analysis that were heavy drinkers.

Of note, the follow-up period of 25 years was considered longer than most other studies. Participants in studies such as this frequently misreport their drinking habits for 25 years due to perhaps memory problems. Of note, a large proportion of the study population was composed of abstainers 24 percent. It did not appear that abstainers were less healthy than the other study members. Interestingly, another study found that light to moderate drinkers with occasional binge drinking had a higher mortality than those light to moderate drinkers with such occasions. These data suggest that binge drinking in midlife is associated with increased risk of dementia. The Effect of Alcohol Overuse and Abstinence on the Brain When it comes to the exactly how alcohol use affects the brain, the severity of brain lesions depends on various factors including the extent of alcohol consumption, age, gender, and neuropsychological comorbidities. The limbic system, especially the hippocampus, which is involved in episodic memory, is also impaired. The cerebellum shows reduction of white matter volume in the vermis and cerebellar hemisphere, and connections between cerebellum and frontal lobe via pons and thalamus are impaired.

Chapter 5 : Cognitive Impairments in Alcohol-Dependent Subjects

The good news is that abstinence from heavy drinking can lead to improvement in cognitive impairment and in many cases return the patient to normal in three months to one year, depending on length of alcohol abuse and the age of the individual.

Alcohol misuse and cognitive impairment in older people: An exploratory study February 11, Alcohol Insight Number Research and Development Grant There may be a significant amount of undiagnosed cognitive impairment in older people aged 55 and over attending substance misuse services. Cognitive impairment screening in substance misuse services is acceptable to older service users. Cognitive impairment can make it difficult for older people to benefit fully from alcohol treatment but treatment can be modified take account of cognitive difficulties. It is possible to screen people with mild cognitive impairment or early stage dementia for alcohol problems. Older people with alcohol problems are particularly likely to experience cognitive impairment. Whilst abstinence or reduced drinking is essential to ensure the best chance of recovery of cognitive functions, cognitive impairment makes it less likely that individuals will benefit from alcohol treatment. This has led to calls for routine cognitive screening in substance misuse services Bates, Bowden, et al. It is also important that people with cognitive impairment are screened for alcohol problems. The main objectives of this study were to explore: The extent and nature of cognitive impairment in older people aged 55 and over attending substance misuse services in the UK for alcohol problems. The difficulties which substance misuse services face when working with clients with cognitive impairment and how treatment can be modified to take account of cognitive difficulties. The extent to which older people attending substance misuse services find screening with the Montreal Cognitive Assessment Nasreddine, et al. The extent to which older people with a diagnosis of mild cognitive impairment or early stage dementia can be screened for alcohol problems using standard alcohol screening tools. The extent to which NHS memory assessment services are screening their clients for alcohol problems and the challenges they face in doing so. They described cognitive difficulties ranging from transient memory problems to permanent difficulties with memory, attention and slowed thinking which was severe enough to interfere with activities of daily living. None of the participants had previously been diagnosed with cognitive impairment. They found screening with the Montreal Cognitive Assessment acceptable and the tool was quick and easy to administer with minimal training. However, one service had recently had a complete changeover of staff which meant that they felt they had insufficient experience to take part. The remaining six services were regularly encountering clients with cognitive impairment. One practitioner had a caseload of 27 people, ten of whom had obvious memory impairment or an existing diagnosis of cognitive impairment. The practitioners identified a number of challenges to working with clients with cognitive impairment including clients forgetting appointments and having difficulties retaining information from the previous session. Practitioners had developed a number of strategies to deal with these and other challenges such as writing appointments on a calendar for clients and summarising the previous session in writing. Most memory assessment services ask their service users about alcohol use, but few use standard alcohol screening questionnaires which makes it difficult for practitioners to know if the person is experiencing or at risk of experiencing problems related to their alcohol use. Although some of the participants with mild cognitive impairment or early stage dementia experienced difficulties answering questions in the alcohol screening tools, these could be overcome, for example, by using drinking diaries as aide memoires, rewording questions in more simple terms when it was evident that the individual was having difficulty understanding them or giving examples when participants were having difficulties with abstraction. Some NHS memory clinics do not conduct a full neuropsychological assessment in people with alcohol problems unless they have been abstinent for a period of time but abstinence is an unrealistic goal for some older people with alcohol problems. Implications It is important that cognitive impairment is identified in older people attending substance misuse services for alcohol problems to aid clinical decision making and so that alcohol treatment can be modified to take account of cognitive difficulties. However, this study suggests that cognitive impairment frequently goes undetected. A pilot study is required to determine whether

the potential benefits of screening outweigh potential harm such as the distress of a diagnosis. Where there is a suspicion of cognitive impairment, substance misuse practitioners are often not able to refer them to NHS memory clinics because they will not conduct a full neuropsychological assessment in people with alcohol problems unless they are abstinent which is not always realistic. Whilst it is understandable that memory clinics might not carry out assessments in individuals with alcohol problems until they have stopped drinking because some alcohol-related cognitive impairment will occur spontaneously with abstinence, this means that the full extent of the cognitive impairment will not be known in individuals who continue to drink and it is difficult to measure change in functioning over time. There is a need for debate on to what extent neuropsychological assessment should be carried out in people with chronic alcohol problems. This study has identified ways of adapting alcohol treatment to take account of cognitive difficulties. Substance misuse practitioners should be familiar with ways of working with people with cognitive impairment and this should be incorporated into their training. NHS memory assessment services should use standard alcohol screening tools to screen their clients for alcohol problems. Service managers should ensure that their staff are trained to carry out alcohol screening. Further Information The research team would like to thank the service users and practitioners who took part in the study and Alcohol Research UK for funding the study. Neurocognitive impairment associated with alcohol use disorders: Experimental and Clinical Psychopharmacology, 10 3 , Neuropsychology Review, 23 1 , Individual differences in latent neuropsychological abilities at addictions treatment entry. Psychology of Addictive Behaviors, 16 1 , A new elderly-specific screening instrument. Clinical and Experimental Research, 16, Clinical and Experimental Research, 22 Suppl. Alcohol Use Disorders Identification Test. Archives of Internal Medicine, 16 , Neurocognitive defects and their impact on substance abuse treatment. Ability of counselors to detect cognitive impairment among substance-abusing patients: An examination of diagnostic efficiency. Experimental and Clinical Psychopharmacology, 5 1 , Effect of neurocognitive status and personality functioning on length of stay in residential substance abuse treatment: Psychology of Addictive Behaviors, 8 3 , Experience-dependent neuropsychological recovery and the treatment of chronic alcoholism. Neuropsychology Review, 1 1 , Recovery of cognitive functioning in alcoholics. Computer-assisted cognitive rehabilitation with substance-abusing patients: Journal of Cognitive Rehabilitation, 21 4 , Self-report and objective measures of cognitive deficit in patients entering substance abuse treatment. Psychiatry Research, 86 2 , Validation of a new alcoholism screening instrument. The American Journal of Psychiatry, 10 , Implications of cognitive impairment for the treatment of alcoholism. Clinical and Experimental Research, 10 2 , Journal Of Psychoactive Drugs, 21 2 , Journal of the American Geriatrics Society, 53 4 , Impairments of brain and behavior: Addiction, 88 6 , Journal of Clinical Psychology, 43 3 , Neurocognitive aspects of substance abuse treatment: Theory, Research, Practice, Training, 30 2 , Neuropsychological test performance of older and younger patients with alcohol dependence. Clinical Gerontologist, 17 3 ,

Chapter 6 : right-arrow copy

The relationship between alcohol use and cognitive impairment has been notoriously difficult to disentangle. We present what is known about cognitive impairment associated with alcohol use/misuse, covering the spectrum from mild and subtle cognitive change through to severe alcohol-related brain damage, including Wernicke-Korsakoff syndrome.

While as rehabilitation specialists, we are not mental health or addiction professionals, I believe it is important for us to be aware of mental health and addiction disorders and how they impact our scope of practice. All data is available online. Baseline memory function was assed using immediate and delayed recall tasks 20 nouns. Abstract reasoning was assessed with a modified similarities test Wechsler Adult Intelligence Scale â€" Revised. In order to focus on the effects of personal history of alcohol consumption rather than current consumption participants consuming three or more drinks per day were excluded. With this consideration, findings would reflect the association of a history of heavy alcohol consumption and memory and cognitive function rather than current heavy consumption. The authors did an admirable job teasing out variables, including but not limited to formal education, race, smoking status, obesity, physical exercise, hypertension, cardiovascular disease, and head injury. The major limitation of the study was study participants self reported AUDs and people tend to underreport alcohol consumption. Also, those that engage in risky behavior are less likely to participate in survey. AUD leads to increased risk for severe cognitive impairment The authors found people with a history of AUDs developed severe memory impairment in later life with 1. While the percentages may seem low over all, AUDs more than doubled the odds of severe memory impairment. The odds for severe cognitive impairment in later life were nearly doubled in those with a history of AUDs 1. The results of this study are consistent with previous studies, even though this study had a much larger sample and had follow up over the longer period of time. How does alcohol impact the brain? Alcohol use is suspected to be related to dementia as a result of brain damage from toxic effects of alcohol, metabolic brain changes, imbalanced neurotransmitters, and nutritional deficiencies. Specifically alcohol dependence is linked to: Volume reduction in white and gray matter, especially in the frontal lobes limbic system, and cerebellum Reduced glucose metabolism in cortical and subcortical structures Neurotransmitter imbalances impacting GABA, serotonin, dopamine, and opioid receptors Head injuries Nutritional deficiencies â€" Severe thiamine deficiency may result in Wernicke encephalopathy Cardiovascular system changes including cardiomyopathy, arrhythmias, hypertension, and stroke What can you do? This study did not examine on cognitive rehabilitation treatment. The study was focused on building connections between mid-life alcohol use disorders and severe memory impairment in later life. This study offered no help in treating current patients. However, it may offer opportunities for prevention. If you or someone you know is struggling with an alcohol use disorder, seek help. Alcoholics Anonymous is an excellent place to start. Visit this page to learn more about AA meetings near you. The National Institute for Alcohol Abuse and Alcoholism reported in approximately 17 million adults had an alcohol use disorder resulting in 5. Alcohol abuse is considered the fifth leading risk for premature death and disability.

Chapter 7 : cognitive impairment | The Alcoholics Guide to Alcoholism

The research literature shows that, as a result of chronic alcohol abuse, there occur impairments in abstract reasoning, problem-solving, and perceptual-motor functioning. In an earlier study, Tamkin () obtained significant differences between alcoholics and paired neurotic inpatient controls.

With alcohol in me I communed with the Gods. The blood seemed to flow around my body better, muscles seemed to get enhanced. I was less inhibited, funnier, nicer, more humane, better company. People sought me out for advice. I was often sage-like. Especially compared to the insecure, disconnected from people, sober me. People would comment on how transformed I became with the drink, nicer, more trusting, more human, more warmed up and less distant. How I acted towards them. It made me connect with humanity, become a part of, not separate. My wife said the main reason I gave for drinking when I was in the midst of chronic alcoholism was to escape from myself â€" not to get drunk but to escape the unbearable lightness of being ME. The second, and most vital and absolutely necessary profound spiritual awakening I had was when I came into recovery via the 12 steps. With much less damage and tragedy wrought! I was not all there, a character, wired to the moon some would say. I was extrovert, but I was also insecure, uncertain, distrusting of people. People worried me so I have a whole bundle of strategies to keep them happy, at arms reach. This included my family. I was also less than whole when sober. Not properly filled in, felt like I was missing something or was protecting some indescribable weakness or deficit in me, although I was never sure want this was, this undefined sense of lacking. Always attending to, protecting some invisible psychic wound. When drinking I underwent some transformation of spirit. I connected better with my fellow human beings. I became someone I liked more than the sober me. Other people seemed to like the sober me less also and much preferred the company of the wisecracking, fun seeking drinking me. The drinking me was a shinier, more colourful me, expansive, inclusive, connecting, less manipulative. The sober me was greyer, more insular, cut off and suspicious of people. When I drank I could drink a lot and rarely had the negative impairments to speech, gait and behaviour that I saw frequently in friends and others. Alcohol did not make me drowsy or make me want to hug my friends and family. It made me slap people on the back, high five. It was not a sedative it was a major stimulant and much more. A wonderful cocktail of effects. For me it is the most brilliantly designed concoction of effect, and I have tried quite a few other drugs in my time. Not only did it give me a warm glow of absolute well being, in a way no other drug has â€" although opium was pretty good at that! It made me dream big dreams, plan my next imaginary offensive. Alcohol made me more me! Seemed to make me work better. Alcoholism gives you many heavenly feelings as it drags you to hell! The thrill was long gone by this stage. So why did alcohol have this effect. Is it only alcoholics that get this euphoric alive and kicking reaction to alcohol? And for years I got away with the physiological withdrawals and hangovers too. Alcohol made me feel bullet proof, it made me stronger. Invincible, like my internal organs were steam powered and made of metal. That I was beyond human. In this response to alcohol, in this lack of impairing effects of alcohol, in this thinking I was invincible, that the drink was my greatest ally may well have lain the seeds of my eventually destruction. In this combination of impulsive seeking and stimulative effects of alcohol was an alcohol fuelled propulsion into eventual chronic alcoholism and compulsive addictive behaviours. Alcoholism would never be something I ever had to consider because I was good, very good, at drinking!! We cite and quote directly from a very interesting article on how a family history of alcoholism contributes to impulsivity, the one psychological domain that turns up repeatedly and is supported in studies of alcoholics, addicts and those at risk genetically from these addictive disorders. Impulsivity from an early age is one variable that appears central to later addictive disorders. Beyond the risk for alcohol use disorders, familial alcoholism is also significantly associated with impulsive and externalizing behaviors Marmorstein et al. Impulsive behavior is associated with both alcohol use disorders and a family history of alcoholism FHA. One operational definition of impulsive behavior is the stop signal task SST , which measures the time needed to stop a ballistic hand movement. Beyond its potential as a reflection of baseline behavioral impulsiveness, SSRT and inhibition success but not choice reaction time are worsened by alcohol intoxication e. The principal finding from this

experiment 1 was a significant interaction between alcohol exposure and those with family history of alcoholism FHA in right prefrontal BOLD activation during motor inhibition. During clamped alcohol infusion, however, this right frontal activation in FHN was significantly reduced, while in FHP activation remained essentially unchanged. Family history of alcoholism interacts with alcohol to affect brain regions involved in behavioral inhibition. Psychopharmacology, 2 ,

Chapter 8 : Frontiers | Cognitive Impairments in Alcohol-Dependent Subjects | Psychiatry

Alcohol seemed to fortify me, make me stronger limbed, heroic, thrusting and invincible. With alcohol in me I communed with the Gods. The blood seemed to flow around my body better, muscles seemed to get enhanced.

Received Apr 27; Accepted Jun The use, distribution or reproduction in other forums is permitted, provided the original author s or licensor are credited and that the original publication in this journal is cited, in accordance with accepted academic practice. No use, distribution or reproduction is permitted which does not comply with these terms. This article has been cited by other articles in PMC. Abstract Chronic excessive alcohol consumption induces cognitive impairments mainly affecting executive functions, episodic memory, and visuospatial capacities related to multiple brain lesions. These cognitive impairments not only determine everyday management of these patients, but also impact on the efficacy of management and may compromise the abstinence prognosis. Maintenance of lasting abstinence is associated with cognitive recovery in these patients, but some impairments may persist and interfere with the good conduct and the efficacy of management. It therefore appears essential to clearly define neuropsychological management designed to identify and evaluate the type and severity of alcohol-related cognitive impairments. It is also essential to develop cognitive remediation therapy so that the patient can fully benefit from the management proposed in addiction medicine units. Evidence suggests that numerous determinants such as environmental, individual, and genetic factors could favor evolution toward alcohol-dependence. These factors may also interact with each other. Among environmental factors, quality of the neighborhood 4 or socio-economic factors e. Individual and psychological characteristics including comorbid psychiatric disorders 7 , 8 , early life stress exposure 9 , or impulsivity 10 are also risk-factors associated with chronic alcohol consumption. In addition, family, twin, and adoption studies have highlighted that genetic factors play an important role in the pathogenesis of alcohol-dependence 11 â€” In the same way, recent studies have examined cognitive endophenotype in alcoholism. They have shown that non-alcoholic relatives of alcohol-dependent individuals performed worse on cognitive tasks specifically executive functions and presented greater impulsiveness compared to control [e. From a neurobiological perspective, alcohol-dependence is a chronic disorder, which implies the dopaminergic system. As seen in other drugs abuses, alcohol consumption acutely stimulates dopamine DA release from the major terminal area of the mesolimbic DA system, nucleus accumbens NAC. Enhanced DA transmission in the NAC plays a critical role in the positive rewarding aspects of drugs abuses and the initiation of addictive process. Chronic administration is associated with functional alterations of this important part of the brain reward system. Globally, dysregulation of the dopaminergic system caused by chronic alcohol consumption produces drug dependence reinforcement and is most likely involved in the development of drug addiction 16 â€” The harmful effects of chronic alcohol consumption on the brain and cognitive functioning have been well described in the literature over recent decades These impairments are moderate to severe but usually remain undiagnosed when they are not specifically investigated. However, detailed neuropsychological assessment or screening of these cognitive impairments appears to be fundamental to optimally adapt patient management strategies. Alcohol-Related Cognitive Impairments Neuroanatomical alterations can account for cognitive impairments affecting various functions, primarily executive functions However, there is a marked interindividual variability of the nature and severity of these impairments. More specifically, the dysexecutive syndrome can affect various processes such as working memory 24 , mental flexibility 25 , divided attention 26 , decision-making 27 , 28 , or problem-solving The characteristic profile of alteration of episodic memory in alcohol-dependent patients comprises limited learning capacities, impairments of encoding, and recollection processes, difficulties recalling the temporospatial context and deficits of autonoetic consciousness, while information storage is preserved 25 , Alteration of executive functions, particularly disorders of inhibition, flexibility, or dual-task coordination also constitute predictive factors of memory impairment 25 , In contrast, apart from obvious deficits i. Finally, visuospatial functions are also predominantly affected, as several studies have demonstrated impaired performances on visuospatial processing, memory and visual learning, visuospatial organization, and

visuoconstruction tasks 31 , Three main hypotheses have been proposed in the literature to account for the characteristic cognitive profile observed in alcohol-dependent patients [see Ref. The first hypothesis is based on the pervasiveness and impaired recovery after withdrawal of visuospatial cognitive functions that are attributed to the non-dominant hemisphere. The right hemisphere would therefore be more susceptible to the neurotoxic effects of alcohol However, this postulate has been questioned by contradictory results The second hypothesis proposes that the increased susceptibility of frontal structures would account for the cognitive profile dominated by executive deficits However, this hypothesis also appears to be too restrictive, as other cerebral structures are also involved The third hypothesis, based on neuroanatomical and neuropsychological data, reconciles the previous two hypotheses by postulating the existence of global brain damage In their meta-analysis of neuroanatomical data, the authors emphasized not only the existence of right hemisphere and frontal lobe lesions, but also lesions affecting other cerebral regions medial temporal, subcortical, and cerebellar atrophy. The neuropsychological functional deficits observed in these patients also concern several cognitive functions in both verbal and visual modalities, which constitutes an additional argument in favor of the global brain damage hypothesis. Anatomical Lesions Chronic excessive alcohol consumption induces global brain atrophy characterized by reduction of brain volume and enlargement of the ventricles and sulci The severity of brain damage depends on various factors such as the extent of alcohol consumption, age, gender, and neurological or psychiatric comorbidities The most susceptible brain structures are the neocortex in the frontal lobes, the limbic system, and the cerebellum Reduction of gray matter preferentially involves frontoparietal regions, while reduction of white matter tends to be more diffuse More precisely, Kril et al. Therefore, these results indicate an increased susceptibility of the brain in the elderly according to the model of alcohol-related premature aging of the brain Furthermore, many studies have also demonstrated functional changes with a reduction of glucose metabolism or cerebral blood flow in prefrontal regions, particularly in the medial temporal region Alcohol-related brain damage also concerns the limbic system and particularly the hippocampus, regions involved in episodic memory 41 â€” Finally, the cerebellum is also affected in these patients, with a reduction of the white matter volume in the vermis and cerebellar hemispheres A study of the connections between the cerebellum and frontal regions via the pons and thalamus also demonstrated alteration of the frontocerebellar circuit Detection and Diagnosis In this context, the detection of cognitive impairments in alcohol-dependent patients is therefore essential and should be systematic. The Montreal Cognitive Assessment MoCA Test appears to be the most appropriate screening test for detection of cognitive impairments in these patients 45 , as this tool is more sensitive than the Mini Mental State Examination MMSE for mild-to-moderate cognitive impairments The MoCA Test can be performed by medical students, medical doctors, or certified neuropsychologist. Detection of cognitive impairments can then lead to referral of the patient for neuropsychological diagnostic assessment performed by a clinical neuropsychologist. Neuropsychological assessments can last 2â€”3 h and are designed to demonstrate preservation or impairment of the most susceptible cognitive functions in this population. Clinical neuropsychologists have at their disposal a battery of tests to evaluate several cognitive domains such as memory e. In the alcohol-dependent population, the most susceptible executive processes to be evaluated are working memory, mental flexibility, inhibition, processing speed, concept formation, planning, and problem-solving capacities. Evaluation of verbal and visual memory must focus on encoding, recall, storage, learning, and recognition capacities, while assessment of visuospatial functions must focus on visuospatial organization and visuoconstruction capacities. Alcohol Consumption and Implicit Cognitive Processes The study of the implicit mechanisms involved in addictive behavior has been considerably developed over recent years. The addictive problem of alcohol-dependence results from a conflict between an urge to drink and the desire to limit alcohol intake. Dual-process models can explain this conflict by the fact that drinking behavior involves two cognitive systems: This system therefore reinforces the incentive effects between stimuli related to the addiction odors, places of consumption, or advertisements related to alcohol, for example and the addictive behavior. It mobilizes the dopaminergic system of the amygdalaâ€”striatal circuit. The underlying network involves various regions of the frontal lobe lateral inferior prefrontal, dorsolateral, ventromedial, orbitofrontal, and frontoparietal and the striatum. Finally, the insula plays a decisive role in the articulation of

these two systems by translating unconscious interoceptive signals or somatic states into conscious subjective experiences desires or needs involved in the decision-making process. This system would therefore play a conflict management role between a stimulus related to addiction and a potentially associated somatic state for example withdrawal symptoms in order to guide decision-making. The interaction between the two systems has been clearly documented in the field of alcohol-dependence According to this theory, drinking behavior is activated by automatic processes impulsive system unless the subject is able to ensure control by mobilizing executive functions reflective system. The impairment of executive functions observed in alcohol-dependent patients would therefore predispose to drinking behavior dictated by the impulsive system. More precisely, disorders of inhibition capacities and working memory play a predominant role in this dysregulation of the impulsive system by the reflective system 28 , 47 , 49 â€” 54 , thereby resulting in a vicious circle, as chronic excessive alcohol consumption induces working memory and inhibition disorders that are then responsible for dysfunction of the reflective system. Finally, alcohol consumption results exclusively from mobilization of the impulsive system that perpetuates the addictive behavior resulting in continuing deterioration of executive functions Cognitive and Brain Recovery The study of alcohol-dependent patients also constitutes a model of brain plasticity, as an increase of brain volume characterized by increased white matter and gray matter volumes and a reduction of the size of sulci and ventricles is observed right from the first months of abstinence 55 â€” The cognitive effects of this recovery consist of improvement of executive functions and verbal episodic memory 21 , 58 , 60 â€” In parallel, it has been shown that new brain regions can be recruited by recently weaned alcohol-dependent patients to compensate for alcohol-related brain damage 66 , Neuroadaptation mechanisms therefore enable patients to maintain a similar level of performance on cognitive tasks to that of control subjects. For example, alcohol-dependent patients recruit neuronal networks parallel to the frontocerebellar circuit normally used by control subjects to perform executive tasks However, although abstinence allows an improvement of cognitive functions, this is only achieved after a period of several months. A recent meta-analysis showed that, despite studies showing early cognitive recovery, a global deficit was still present several months after installation of abstinence and the cognitive profile tended to become normal only after 1 year of abstinence, while certain residual cognitive impairments may persist. For example, the presence of visuospatial function deficits may be observed after several years of abstinence, related to the decreased volume of the right parietal cortex Cognitive Impairments and Management of Alcohol Abuse Appropriate management of alcohol withdrawal is mandatory to prevent severe complications like delirium tremens or epileptic seizure. If benzodiazepines usage and appropriate rehydration are codified, the dose and duration of thiamine treatment remains unclear Nevertheless long-term abstinence is the main goal and cognitive behavioral therapy CBT and psychosocial programs are necessary. Adjuvant treatments may include: The presence of cognitive impairments therefore requires adaptation of the management of alcohol-dependent patients. CBT has been demonstrated to be effective in the management of alcohol-dependence 70 , but it is somewhat paradoxical to propose management that directly involves cerebral structures and cognitive functions altered by chronic alcohol consumption. This management approach may therefore be inappropriate or at least insufficient for a certain number of patients. The efficacy of CBT would therefore depend on the integrity of certain brain regions of interest. For example, it has been shown in schizophrenic patients that the volume of gray matter in the frontal, temporal including hippocampus , parietal, and cerebellar regions, brain regions that are also damaged in alcohol-dependent patients, is predictive of the efficacy of management Similarly, the integrity of the frontocerebellar network, a site of predilection for brain damage in alcohol-dependent patients, would play an essential role in the efficacy of CBT due to its role in executive functioning Cognitive behavioral therapy in addiction medicine also requires elaborate cognitive capacities such as episodic, semantic and procedural memories, and executive functions 23 , 73 â€” This type of management could therefore be unsuitable for patients with cognitive impairments 25 , 77 â€” Various studies have shown that alcohol-dependent patients with the most severe cognitive impairment also have the least favorable prognosis 80 â€” Cognitive impairment can also influence the expression of individual and environmental factors involved in management, such as self-efficacy, readiness to change, active participation in group therapy, or treatment compliance, as the initial cognitive impairment is predictive of poorer treatment

compliance and a decreased self-efficacy. Patients with severe cognitive impairments are also less able to use their own resources during management, in which case the prognosis depends more on the role of external factors such as group therapy or the family support network Finally, Le Berre et al. Similarly, the integrity of executive functions enables patients to weigh up their decisions to reach the action stage, which can only be implemented when decision-making capacities are preserved. Cognitive impairment therefore influences the degree of motivation of alcohol-dependent patients, an essential prerequisite to the success of management. The presence of cognitive impairments on admission and during the first months of abstinence therefore influences management at various levels by determining the efficacy of treatment and the prognosis for lasting abstinence. Addiction medicine management must therefore be adapted to alcohol-dependent patients with cognitive impairments.

Chapter 9 : Cognitive Impairment and Recovery From Alcoholism Alert No:

Cognitive impairment is common in patients with advanced liver disease. It has been suggested that patients with alcoholic liver disease (ALD) have more impaired cognition than nonalcoholics. The objective of this study was to characterize any differences in cognitive functions between alcoholic cirrhotic patients and non-alcoholic cirrhotic.

Memory loss Loss of attention span Alcohol is an integral part of the American social scene. The failed experiment of Prohibition demonstrated the formidable demand Americans have for alcohol. As advertisements caution, alcohol is meant to be enjoyed responsibly; however, alcohol consumption can cause individuals to lose their ability to think responsibly. Alcohol is associated with a host of familiar cognitive changes , such as a loss of inhibitions, confused or abnormal thinking, and poor decision-making. Recreational alcohol users generally recover from its effects without any long-term problems. However, even short-term loss of control over normal mental functioning can result in legal and personal troubles that would likely not have occurred if alcohol were not involved. Alcohol consumption continues to maintain a foothold so strong in American culture that it is nearly impossible to imagine life without it. As the National Institute on Drug Abuse explains, the active ingredient in beer, wine, and liquor is ethyl alcohol, or ethanol, which is known colloquially as alcohol. The process known as fermentation produces alcohol. When yeast is fermented, sugar breaks down into carbon dioxide and alcohol. Carbon dioxide exits the process through gas bubbles and leaves behind a combination of water and ethanol. The process is so precise that if any air is present in the yeast, the result will be ethanoic acid, a chemical found in common vinegar. Alcohol is basically made from sugar and yeast, but different sources of sugar can produce different types of alcohol. For instance, the sugar from grape pulp is combined with yeast to create either red or white wine. In the case of beer , the source of the sugar is usually barley, but it can also be other grains, such as wheat or rye. To release the sugars, the grains must be malted, mashed, and boiled. Once the sugars are ready for use, yeast is added and the fermentation process begins. Different liquors are made different ways. When it comes to vodka , for example, there is a popular belief that potato is usually the source of the sugar; however, the majority of vodka is made from fruit, barley, wheat, sorghum, or corn. Alcohol products, such as wine, beer, and liquor, are made in commercial distilleries, but the process of making DIY alcohol moonshine still relies on the basics discussed above. In some parts of the world, such as Scandinavian Europe and Russia, moonshine is a popular and low-cost alternative to commercial alcohol in some locales. Moonshine is known for its potency, which can be dangerous. Moonshine can be proof, which translates into it being 75 percent alcohol. An added hazard is that it is not created in a regulated setting; therefore, there are fewer, if any, quality and safety guarantees. Although there is not a pressing public concern about rampant sales of moonshine, there are reports of Americans making DIY alcohol and finding themselves in legal hot water as a result. For instance, a year-old man was arrested in Ohio after selling moonshine out of his camper at a fair. Making moonshine may sound antiquated to modern ears, but it demonstrates the relative ease with which alcohol can be illegally made. Although some Americans still have Prohibition sympathies, the ability to home-produce alcohol serves as a reminder that it would likely be impossible to entirely remove alcohol from the American landscape. The survey made the following estimated findings with respect to alcohol consumption: In the past 30 days, This statistic includes 5. Alcohol is the most commonly abused drug in the US, with marijuana occupying second place. Despite the known dangers of excessive alcohol consumption, these statistics clearly reflect that many alcohol users engage in heavy use and binge drinking. Further, despite the well-known fact that alcohol impairs brain functioning, an estimated one in 10 Americans aged 12 or older operated a vehicle under the influence. This statistic demonstrates that only a fraction of those who drive under the influence are actually detected. It is well established in the mental health field that alcohol consumption can exacerbate an underlying mental health disorder. In the field of substance abuse treatment, an individual who simultaneously has a substance use disorder and at least one other mental health disorder is considered to have a dual diagnosis. Whether alcohol causes or simply accompanies the underlying mental health disorder is not clearly understood. However, there is general consensus that individuals who abuse alcohol and have a mental health disorder diagnosis require treatment

for both conditions. Alcohol in the Brain While it is true that alcohol can initially perk people up and even help them to socialize at a party, alcohol is a central nervous system depressant. The depressant effects of alcohol are witnessed when people who have been drinking have slurred speech and poor limb coordination that prevents them from being able to walk properly. So, how does alcohol act at the neurological level? Alcohol acts on the receptor sites for the neurotransmitters chemical messengers known as GABA, glutamate, and dopamine. With drinks, the person may feel excited or more talkative, but with more and more alcohol in their system, they become more sedated and being to lose control of their movement and may experience impaired thinking and memory. The fluctuation in BAC helps to account for why the same person can go from being the life of the party to needing help with basics, like walking. However, people play a game of roulette with themselves when they drink, especially when they drink heavily, because the effects of alcohol on the brain are uncertain both in the short-term and long-term. If other drugs are added to the alcohol consumption, the risks become more serious. Occasional Drinkers In occasional drinkers, alcohol can produce one or more short-term effects after one or more drinks. Memory impairment can begin after a few drinks, and it can become increasingly worse as the consumption increases. A high volume of alcohol consumption, especially on an empty stomach, can result in a blackout. Occasional drinkers will usually recover from a blackout without any lasting mental problems. However, there are numerous dangers associated with acute alcohol intoxication, such as engaging in reckless activities like unprotected sex, vandalism, and driving. Further, an alcohol-involved incident, such as a car accident, can cause ongoing problems as court dates will need to be attended, fines must be paid, and educational or treatment requirements will have to be met. Moderate Drinkers As the Centers for Disease Control and Prevention discusses, the Dietary Guidelines for Americans considers a moderate drinker to be a person who consumes one drink applies to women or two drinks applies to men per day. Despite extensive news coverage of medical reports that moderate drinking has positive health benefits, the guidelines advise that this is not a reason to start drinking. Moderate alcohol consumption has negative associations, such as increasing the risk of breast cancer and causing violence, falls, drownings, and car accidents. Moderate drinking does not insulate a person from the cognitive impairment associated with drinking and the dangerous consequences that can result. Heavy and Chronic Drinking Unlike an occasional or moderate drinker, a person who drinks heavily over an extended period of time may develop deficits in brain functioning that continue even if sobriety is attained. In other words, cognitive problems no longer arise from drinking alcohol but from brain damage that prior drinking caused. According to the National Institute on Alcohol Abuse and Alcoholism , most heavy long-term alcohol users will experience a mild to moderate impairment of intellectual functioning as well as diminished brain size. The most common impairments relate to the ability to think abstractly as well as the ability to perceive and remember the location of objects in two- and three-dimensional space visuospatial abilities. In addition, there are numerous brain disorders associated with chronic alcohol abuse. For example, research supports that up to 80 percent of chronic alcohol users have a thiamine deficiency, and some in this group will progress to a serious brain disorder known as Wernicke-Korsakoff syndrome WKS. Symptoms of WKS include confusion, paralysis of eye nerves, impaired muscle coordination, and persistent problems with memory and learning ability. Fortunately, certain types of cognitive impairment can be reversed through abstinence from alcohol. Studies show that those who have recently undergone medical detox exhibit mild but significant improvement in certain cognitive abilities, especially those related to visuospatial tasks, problem-solving abilities, and short-term memory. Through ongoing abstinence, over a period of several months to one year, a recovering individual will continue to improve cognitive skills, such as visuospatial abilities, working memory, and attention span. In addition, research shows that brain volume can increase with abstinence. Last updated on October 24, T