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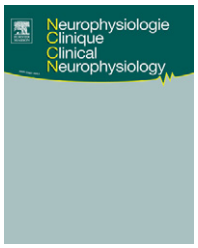

REVIEW/MISE AU POINT

How cognitive assessment through clinical neurophysiology may help optimize chronic alcoholism treatment

L'évaluation des fonctions cognitives au moyen de la neurophysiologique clinique permet d'améliorer le traitement de l'alcoolisme chronique

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Summary Alcohol dependence constitutes a serious worldwide public health problem. The last few decades have seen many pharmacological studies devoted to the improvement of alcoholism treatment. Although psychosocial treatments (e.g. individual or group therapy) have historically been the mainstay of alcoholism treatment, a successful approach for alcohol dependence consists in associating pharmacologic medications with therapy, as 40—70% of patients following only psychosocial therapy typically resume alcohol use within a year of post-detoxification treatment. Nowadays, two main pharmacological options, naltrexone and acomprosate, both approved by the US Food and Drug Administration, are available and seemingly improve on the results yielded by standard techniques employed in the management of alcoholism. However, insufficient data exist to confirm the superiority of one drug over the other, and research is ongoing to determine what type of alcohol-dependent individual benefits the most from using either medication. Available data on the application of both drugs clearly suggest different practical applications. Thus, a fundamental question remains as to how we can identify which alcoholic patients are likely to benefit from the use of naltrexone, acamprosate or both, and which are not. The aim of the present manuscript is to suggest the use of cognitive event-related potentials as an interesting way to identify subgroups of alcoholic patients displaying specific clinical symptoms and cognitive disturbances. We propose that this may help clinicians improve their treatment of alcoholic patients by focusing therapy on individual cognitive disturbances, and by adapting the pharmaceutical approach to the specific needs of the patient.

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MOTS CLÉS
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Résumé La dépendance à l'alcool représente un problème de santé publique majeur. Il n'est dès lors pas étonnant que de nombreuses études pharmacologiques aient été dévolues à la recherche d'un traitement adapté à cette pathologie. L'approche classiquement utilisée de nos jours consiste à combiner une approche psychosociale (psychothérapie individuelle ou de groupe) à un traitement médicamenteux, étant donné que 40 à 70 % des patients ne suivant qu'une thérapie rechutent dans l'année suivant leur cure de désintoxication. Actuellement, deux molécules reconnues par l'Agence américaine des médicaments, la naltrexone et l'acamprosate, semblent améliorer les résultats obtenus par rapport aux psychothérapies seules dans la prévention de la rechute de ces patients. Pourtant, une question fondamentale demeure sans réponse : il nous est toujours impossible de dire quel(s) type(s) de patients alcooliques bénéficieront le plus de l'utilisation d'une molécule par rapport à l'autre. De plus, les données recueillies, à ce jour, semblent suggérer une application « pratique » différente de la naltrexone et de l'acamprosate. Dès lors, l'objectif du présent article consiste à suggérer une méthode basée sur le recueil de potentiels évoqués cognitifs, pouvant amener les cliniciens à constituer, sur base de troubles cliniques comportementaux associés à des troubles neurocognitifs spécifiques, des sous-groupes de patients dépendants à l'alcool. La constitution de ces sous-groupes aurait comme objectif principal d'adapter la rééducation cognitive du patient à ses déficits cognitifs propres et de fournir au patient une approche médicamenteuse (naltrexone seul, acamprosate seul ou naltrexone et acamprosate combinés) adaptée à ses besoins individuels.

Introduction

Alcohol dependence is a chronic relapsing medical disorder [36], and besides its psychological and social ramifications, is essentially a brain disorder [24]. Indeed, it has been well established that, because of alcohol neurotoxicity, chronic alcoholism leads to deleterious effects on the central nervous system (CNS) and cerebellum, such as brain atrophy and/or dysfunction [60], these brain impairments being correlated with the lifetime dose of ethanol consumed [38].

As a discipline, neuropsychiatry tries to bridge the gap between neurology, on the one hand, and psychiatry, on the other, in order to achieve greater insight into the biological bases of psychiatric disorders [43]. New tools have been developed in the last few decades to investigate these brain deficits. Among these, brain imaging techniques, such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI), have allowed researchers to investigate which brain regions are involved in specific human cognitive functions. On the basis of these techniques, in addition to brain atrophy (which can largely be accounted for by white-matter loss), alcohol-related neuronal degeneration has been documented in specific regions of the cerebral cortex of alcoholics, such as the superior frontal association cortex, the hippocampus and the amygdala, which are known to be involved in many ''high-order'' psychological functions [11].

Chronic alcoholism is defined by a variety of clinical symptoms, such as a compulsive preoccupation with obtaining alcohol despite devastating impacts on social and occupational functioning, and a high vulnerability to relapse after cessation of drinking [2]. Many studies have been devoted to the identification of *cognitive candidates* that could trigger these habits. The most important are probably: the ability to inhibit or suppress mental representations loaded in working memory, which is a fundamental aspect of behavioural control and leads to general states of ''disinhibition'' or ''dyscontrol'' characterized by impulsive and exaggerated behaviour [17]; and the capacity to shift from one idea to another, as it has been shown that alcohol-related stimuli have acquired conditioned incentive properties, such that these stimuli become perceived as highly attractive, thereby ''grasping'' attention [49]. In other words, alcoholics suffer from deficits in their cognitive control mechanisms of ''inhibiting'' and ''shifting'' and these deficits are exacerbated by cognitive biases for alcohol-related stimuli [42]. This idea is at the core of a cognitive model of chronic alcoholism called ''the dual-process approach'' [14,15].

In the dual-process approach, there are two distinct processes of thinking that compete for control of our behaviour: the first one is rapid, automatic, implicit and computationally powerful, the other one is slower, explicit, controlled and dependent on central working-memory resources. In this approach, addiction is seen as the result of an imbalance between these two systems (habit/motivational vs executive). Indeed, it is suggested that, on the one hand, substance abuse is characterised by an increase in the salience of drug-related cues, so that these cues tend to ''grab the attention'' [59]; and, on the other hand, due to the neurotoxic effects of repeated drug use [39] and/or a state of vulnerability [12], addicted people lack the executive resources that are needed to modulate (i.e., to ''inhibit'') the salient and dominant response (i.e., ''drink'').

This view is based mainly on studies that were performed during the last decade. First, at the attentional level, it has been shown that repeated alcohol use is associated with a bias towards alcohol-related stimuli [16,31,40,41]. For instance, on a modified version of the Stroop task, alcoholics named the color of alcohol-related words much slower than neutral matched words, while this difference was not present in matched controls [22]. In addition, Cox et al. [13] argued, in 2002, that this kind of attentional bias