Supervisory Attentional System in Nonamnesic Alcoholic Men

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Background: Many studies have shown that recently detoxified alcoholic persons perform poorly on tasks thought to be sensitive to frontal lobe damage, supporting the hypothesis that the frontal lobes are highly vulnerable to chronic alcohol consumption. However, it appeared that most of the executive tasks used in these studies also involved nonexecutive components, and these tasks had been shown to be impaired as a result of nonfrontal lobe lesions. In this study, we examined further the “frontal lobe vulnerability” hypothesis using executive tasks, proved to be associated with frontal lobe functioning, that allowed us to distinguish the relative importance of executive and nonexecutive processes.

Method: Thirty recently detoxified asymptomatic male alcoholic inpatients and 30 control subjects were tested for planning, inhibition, rule detection, and coordination of dual task, as well as the speed of processing and nonexecutive functions (such as short-term memory storage).

Results: Alcoholics performed worse than controls in almost all tasks assessing executive functions. However, they were not slower than the controls and showed normal results for nonexecutive functions.

Conclusions: Chronic alcohol consumption seems to be associated with severe executive function deficits, which are still present after a protracted period of alcohol abstinence. These data support the idea that the cognitive deficits in recently detoxified sober alcoholic subjects are due, at least partly, to frontal lobe dysfunctioning.

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Alcoholism continues to be one of the leading public health problems in the Western World. The economic costs of alcohol dependence to society are vast. Long-term abuse of alcohol in association with nutritional deficits (thiamine deficiency) can lead to classic neurological illnesses such as the Wernicke-Korsakoff syndrome. However, during the last 2 decades, research has provided evidence of brain abnormalities in “nonamnesic” chronic alcoholic subjects, including electrophysiological, anatomical, cerebral blood flow, glucose metabolism, and a wide range of neuropsychological deficits.

Current literature postulates 3 main hypotheses concerning brain structures that may be vulnerable to the effects of prolonged alcohol abuse: the “right hemisphere abnormality,” the “generalized brain dysfunction,” and the “frontal brain vulnerability.”

The right hemisphere abnormality hypothesis states that the nondominant hemisphere is more vulnerable to alcohol’s effects. In this view, recently abstinent alcoholic subjects would show difficulties in nonverbal neuropsychological functions, supposedly related to the right hemisphere. However, several studies using dichotic listening tasks to determine whether there was any relationship between functional asymmetry and alcoholism revealed no significant effects. These data suggested that the poorer performance of alcoholic subjects on nonverbal or visuospatial tasks is a consequence of the psychometric sensitivity or unfamiliarity of these tests. The generalized brain dysfunction hypothesis asserts that long-term alcohol abuse produces neurotoxic effects throughout the brain, resulting in mild to moderate cognitive dysfunction that is global and nonspecific. Although this hypothesis is consistent with the heterogeneity of neuropsychological, neuroimaging, and neuropathological data on chronic alcoholism, it appears to be so broad that it is difficult to disprove. Lastly, the frontal brain vulnerability hypothesis suggests that the frontal lobes are greatly vulnerable to long-term consumption of alcohol. Moderate neuronal loss has been reported in the frontal cortex and in the...
cingulate gyrus in alcoholic subjects supporting this hypothesis. Functional neuroimaging studies using positron or single-photon emission computed tomography in recently detoxified alcoholic subjects have documented global cerebral metabolic or perfusion deficits that are more pronounced in the frontal regions. In addition, frontal lobe hypoperfusion is independent of brain atrophy. Pronounced in the frontal regions. Frontal brain vulnerability hypothesis by using tasks designed to assess separately nonexecutive and specific executive operations (which proved to be sensitive to frontal lobe dysfunction). The theoretical framework on which this study was based is the control-to-action model developed by Norman and Shallice. This model distinguishes 2 control-to-action mechanisms. The first, contention scheduling, is involved in routine situations in which actions are triggered automatically. The second, the Supervisory Attentional System (SAS) is needed in situations in which the routine selection of action is unsatisfactory, and they conceived it as carrying out a variety of processes allowing the genesis of plans and willed actions.

An analogy has been established between the SAS and the central executive (CE) component of the working memory model proposed by Baddeley. Working memory is a limited capacity system responsible for the temporary storage and processing of information while cognitive tasks are performed. Baddeley's model comprises 2 slave systems ensuring temporary maintenance of information, the phonologic loop, and the visuospatial sketchpad, as well as an attentional control system, the CE, the function of which is similar to that of the SAS.
According to Baddeley, an important function of the CE is to allow the performance of 2 tasks simultaneously. In the present study, we investigated the performance of recently detoxified nonamnesic male alcoholic inpatients in 3 SAS functions: planning, inhibition, and abstraction of logical rules. Additionally, the ability to manipulate stored information in working memory (a CE function that has been proven to be frequently affected in alcoholic subjects) has been assessed. We also addressed the question of how much a reduction of processing speed accounts for alcohol-related SAS/CE deficits. To determine the position of the solid circle on subsequent pages but the rules change from time to time. The subject's task was to suggest which circle will be filled on the next page and why. We counted the number and type of errors made. The first type (I) was to continue a rule after it had changed. The second type (II) was to reapply an earlier rule before the new rule is discovered. The third type (III) was a guess.

On the phonologic, semantic, and alternate verbal fluency task, subjects were given 120 seconds to generate aloud a list of words beginning with the letter /p/ (phonologic condition) but excluding proper names and variants of the same word. Then, we proceeded in the same way with a semantic category (name clothes [categorical condition]) and with 2 categories alternatively (name tools and animals [alternate condition]). The number of correct words generated was recorded. In the Trail Making Test, the subject is asked to trace a line joining in alphabetical order a series of letters distributed randomly across a sheet of paper (Trail A), and then (Trail B), a trace joining alternatively the letters of the alphabet and the numbers 1 to 20 in alphabetical or ascending order.

On the Flexibility test, the subjects were instructed to consider some common objects and the task was to generate orally as many other less common uses for each object as possible. For example, a newspaper is commonly used for reading, but it can be used to start a fire, wrap garbage, swat a fly, and others. We calculated the total number of correct infrequent uses of 3 objects.

The Stroop Interference Test comprised 4 different cards shown in a fixed sequence. The “reading condition” is to read color-words printed in black as quickly as possible. The “denomination condition” is to name color patches. The “interference condition” is to name the color of the print of a word printed in an incongruent color. The “flexibility condition” is similar to the last except that the subject is asked to read the words rather than name its color when it is underlined. The time to complete each condition and the sum of errors made on interference and flexibility conditions were recorded. To measure processing speed, the time to complete the Trail A, the color-naming part of the Stroop Interference Test, the latency time of the Tower of London test, and the time to realize part A of the Hayling task were considered.

STATISTICAL ANALYSIS

With respect to the data collected from the various neuropsychological tests, analyses of variance (ANOVAs) were performed to determine for which variables alcoholic subjects and controls differed significantly. Post hoc analyses were performed using the Newman-Keuls test. A Pearson product moment correlation analysis was also conducted to examine the relationships among demographic and clinical variables and cognitive performance in alcoholic subjects. All statistical analyses were based on 2-tailed tests of significance and were performed using SPSS 8.0.

RESULTS

With regard to the Alpha-Span Task, alcoholic subjects' (mean ± SD, 4.8 ± 0.92 words) and controls' word span (4.9 ± 0.61 words) did not differ significantly (F1, 58 = 0.49, P = .62). The scores for the direct and alphabetical recall conditions (Figure 1) were then analyzed separately using a 2-way, 2 (group) × 2 (direct and alphabetical recall) ANOVA. The analysis revealed a main effect of group (F1, 58 = 43.6, P < .001) and of condition (F1, 58 = 90.97, P < .001). A significant interaction between the group and the type of recall was also found (F1, 58 = 54.6, P < .001), with the alcoholic subjects showing a more important decrease of performance from direct to alphabetical recall than the controls, despite a similar performance in direct recall (Newman-Keuls post hoc comparisons).

On the latency time of the Hayling task (Table 2), the 2 (group) × 2 (section) ANOVA revealed a significant group effect (F1, 58 = 12.62, P = .001), a significant effect of section (F1, 58 = 116.1, P < .001) and a significant interaction between these 2 factors (F1, 58 = 12.25, P = .001). Post hoc comparisons revealed that the alcoholic subjects were significantly slower than the controls in giving an answer in section B (P < .001) but not in section A (P = .35) (Figure 2).
The section B score of the Hayling task showed that the alcoholic subjects gave more words semantically linked to the expected word (score 1) \((F_{1,58} = 113.39, P < .001)\) and fewer unrelated words (score 0) \((F_{1,58} = 113.39, P < .001)\) than the controls. Nevertheless, the alcoholic subjects did not give more expected words (score 3) \((F_{1,58} = 0.6, P = .5)\). Alcoholic subjects showed a higher overall score than the controls \((F_{1,58} = 12.44, P = .001)\).

On the Brixton test, alcoholic subjects made more errors than the controls \((F_{1,58} = 4.46, P < .001)\). The 2 groups did not differ with regard to the number of type I errors \((F_{1,58} = 1.35, P = .25)\) but the alcoholic subjects made fewer type II errors (reaplication of earlier rule) \((F_{1,58} = 23.6, P < .001)\) and more type III errors (guesses) \((F_{1,58} = 37.6, P < .001)\) than the controls.

On the verbal fluency tasks, a 2 (groups) \(\times 3\) (condition: phonologic, semantic, and alternate) ANOVA revealed a main effect of group \((F_{1,58} = 5.01, P < .001)\) and of condition \((F_{2,116} = 5.01, P < .001)\). The interaction between these 2 factors was also significant \((F_{2,116} = 7.3, P < .01}\). Post hoc analysis revealed that alcoholic subjects produced fewer words than controls only in the alternate fluency condition \((P < .001)\). On the Flexibility test, alcoholic subjects found fewer uncommon uses of objects than controls \((F_{1,58} = 4.16, P = .04)\).

On the Stroop Interference Test, the 2 (group) \(\times 3\) (condition: reading plus denomination time, interference time, and flexibility time) ANOVA revealed a main effect of group \((F_{1,58} = 6.41, P < .001)\) and of condition \((F_{2,116} = 13.3, P < .001)\). Post hoc analysis revealed that alcoholic subjects were slower than controls only on the flexibility condition \((P < .001)\). Alcoholic subjects also made more errors (sum of errors made on interference and flexibility conditions; mean \(\pm SD\), 11.4 \(\pm 6.7\)) than controls \((3.4 \pm 1.3)\) \((F_{1,58} = 7.68, P < .001)\).
On the Trail Making Test, the 2 (group) × 2 (condition: Trail A and Trail B) ANOVA revealed a main effect of group (F1,58=7.82, P=.007) and of condition (F1,58=57.9, P<.001). There was a significant group by condition interaction (F1,58=7.72, P=.007). Post hoc analysis revealed that the alcoholic subjects were slower than controls only in Trail B (P<.01).

The Tower of London test (Table 3) results showed that, on the easiest problems (3 moves), alcoholic subjects and controls did not differ for the number of moves (F1,58=0.72, P=.40), for the time needed to initiate the first move (F1,58=0.44, P=.51), or for the time to solve the problem (F1,58=0.34, P=.73). In problems requiring 5 moves, the 2 (group) × 3 (type of problem: neutral, facilitating, and misleading) ANOVA on the number of moves revealed a main effect of group (F1,58=6.02, P=.02) and showed that the alcoholic subjects took significantly more moves to solve the problems. There was also a main effect of type of problem (F2,116=6.5, P=.002). Post hoc analyses revealed that the number of moves was equivalent between the neutral and misleading problems but was greater for both than for facilitating problems. There was no significant group by type of problem interaction (F2,116=0.99, P=.37). Two-way, 2 (group) × 3 (type of problem) ANOVAs were carried out on the initiation and subsequent times. For the initiation time, the analysis showed no main effect of group (F1,58=1.3, P=.27) or type of problem (F2,116=1.9, P=.14). There was no significant group by type of problem interaction (F2,116=1.45, P=.23). For the subsequent time, significant effect of type problem (F2,116=29.9, P<.001) was observed. Either significant effect of group (F1,58=0.29, P=.59) or significant interaction between these 2 factors emerged (F2,116=0.42, P=.66).

To examine how well the alcoholic subjects and controls were able to redress their mistakes, we calculated the number of moves to solve the problem once they put the bead on the first move into a position that was not coherent with the optimal solution of the neutral and misleading problems. Alcoholic subjects moved the first bead inadequately 19 times and controls 7 times. After that, the alcoholic subjects and controls needed 10.43±2.3 and 7±1.2 (mean±SD) moves, respectively, to solve the problems. The alcoholic subjects made more moves to achieve the solution than the controls (F1,58=4.5, P<.001).

Finally, none of the correlations among age, number of years of education, number of prior cures, total lifetime intake of alcohol, number of years of heavy drinking, depression, anxiety, or those neuropsychological performances for which a significant group effect had been observed were statistically significant (all were inferior to P=.22).

This study investigated the presence of SAS/CE impairments in recently nonamnesic male alcoholic subjects. It used executive tasks inspired by Norman and Shallice’s control-to-action model and Baddeley’s working memory model. In addition, we addressed the question of: by how much does the slowing of processing speed account for SAS/CE deficits?

The analysis of the Tower of London test results shows that the alcoholic subjects were able to plan and execute efficiently the easiest problems, but, once they had begun to solve a problem incorrectly, they had more difficulty than the controls in redressing their mistake: this suggests a failure of inhibition or flexibility exploring planning ability (for a similar interpretation see Joyce and Robbins). On the inhibition test (the Hayling task), the alcoholic subjects were slower on the inhibition section, but not on the section requiring production of an automatic response. They also made more inhibition errors than the controls. The observed dissociation between the initiation and inhibition sections tackles directly the con-
tention scheduling/SAS (or automatic-controlled) distinction proposed by the control-to-action model of Norman and Shallice; deficits in alcoholic subjects are observed in the controlled process allowing the inhibition of a dominant response but not the production of an automatic response. Findings of a 1999 positron emission tomographic study showed that bilateral median frontal activation occurs during section B of the Hayling task. These data suggest that an alcoholic person’s inhibition deficits might be caused by frontal lobe abnormalities.

Other results are consistent with the existence of an inhibition deficit. In the Trail Making Test, alcoholic subjects were slower than controls on section B but not on section A. Similarly, they showed poor performance in the alternate verbal fluency task. Finally, patients spent more time to complete the flexibility condition of the Stroop Interference Test. Trail B requires inhibiting current realization strategy (1, 2, 3 . . . ) to switch between numbers and letters (1A, 2B, 3C . . . ). Performance in the alternate verbal fluency task requires, notably, that subjects inhibit one search strategy to switch to another. In the Stroop test, the flexibility condition requires the subject to switch between 2 rules alternatively, that is to inhibit the current rule. However, a puzzling result is that alcoholic subjects were slower than controls in flexibility but not in the interference condition of the Stroop test which also clearly requires inhibition. Similarly, they produced fewer words in the alternate but not the phonologic and semantic fluency tasks while inhibition process is needed for both (e.g., with regard to the phonologic fluency task, inhibiting the usual search strategy on the basis of the word meaning). These dissociations suggest the view that there exists multiple inhibitory mechanisms and/or that the different tasks require various levels of inhibitory resources. The study’s results in the rule detection test (Brixton test) show that alcoholic subjects and patients with frontal disorders responded similarly: they made more errors than controls, particularly more illogical responses (guesses).

The results of the Alpha-Span Task show that, in alcoholic subjects, the normal storage component (measured by the span size and the score of direct recall condition) remains healthy but the ability to manipulate the information stored (measured by the alphabetical recall condition) is impaired. A recent positron emission tomographic study indicated that the executive (manipulation) processes involved in a modified version of the Alpha-Span Task is distributed between frontal and parietal attentional systems. These data suggest that, in alcoholic subjects, this parietofrontal network might be disrupted.

Another interesting result concerns the contribution of a slowing down of the processing speed to SAS/EC deficits. Similar to the findings of previous studies, alcoholic subjects were not slower than controls in the color-naming part of the Stroop test, in the Trail A section, in the initiation section of the Hayling task, and in the Tower of London test. This suggests that processing speed does not constitute an important contributing factor to their executive deficits. Since many studies in the Cognitive Aging domain suggest that the speed of processing is an important mediator between age and cognitive performance, our results seem to be inconsistent with the hypothesis that alcoholic subjects show cognitive modifications similar to those observed in older nonalcoholic subjects.

Like other studies, ours failed to find any correlation between measures of drinking practice and neuropsychological performance. However, the accuracy of retrospective recall in patients known to have cognitive impairments is probably low.

The findings of the present study show that uncomplicated alcoholic subjects near the end of a period of detoxification manifest executive function deficits: these could have important clinical implications, particularly concerning relapse. Tiffany suggests that drug use behavior in addicts is largely controlled by automatic processes and, therefore, that executive functions are needed to block this and maintain abstinence. Thus, the existence of executive function deficits in alcoholic subjects, as shown in the present study, could affect the capacity to maintain abstinence. However, further studies are needed to explore this relationship.

There are 2 main limitations to the present study. First, we do not know whether cognitive deficits in alcoholic subjects recover after a longer period of abstinence. Second, it does not show whether an alcoholic subject’s executive dysfunction developed as the result of a long-term use of alcohol (a neurotoxic effect) or constituted a developmental predisposing factor to substance abuse. Further longitudinal studies should be conducted to explore both questions.

The findings of the present study showed that inhibition, planning, rule detection, and coordination of dual tasks were impaired in recently detoxified male alcoholic subjects. It seems as if these SAS/EC deficits were probably not caused by reduced processing speed. More generally, these findings are consistent with the view that the cognitive deficits in recently detoxified sober alcoholic subjects are due, at least partly, to frontal lobe dysfunction.

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