

Brain damage assessment in chronic alcoholics on early-stage recovery by Lacks' adaptation of Bender's Visual Motor Gestalt Test

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SUMMARY. It is now established that heavy alcohol intake lasting for more than 10-15 years may lead to a dramatic impairment of brain function, as in severe alcohol related pathologies, like liver cirrhosis. It is common opinion, however, that mild impairment of the cognitive functions can be demonstrated with neuropsychological tests in a very high percentage of alcoholics during the phase of detoxification, as a consequence above all of alcohol neurotoxicity. In this study, in a series of alcoholics on early stage abstinence from alcohol, brain damage was assessed by Bender's Visual Motor Gestalt Test (BVMGT) as a clinical tool for sampling visual-motor proficiency, and as a standard projective technique in the assessment of personality. According to Lacks' specific adaptation, 12 parameters (standardized "errors") were evaluated, as well as some standardized behavioral observations allowing a discrimination between patients with brain organic damage and with psychosis. The cut-off between organic and non-organic patients was set at five errors. The patients were tested on the first days of treatment, to assess the possibility of obtaining a test suitable for diagnosis and for the choice of treatment at such an early stage.

According to Lacks' adaptation and interpretation of BVMGT, our series was split into two subsets, < 5 and ≥ 5 cumulative error score: only 77 patients out of 187 were in the ≥ 5 subset of organic brain damaged patients. The heterogeneity of our total sample for organic damage was confirmed by the cumulative score, 4.24 ± 1.62 , showing a high dispersion of individual data.

According to Abbate and Ferracuti, patients affected by different diseases (organic brain damage, schizophrenia, personality disorders) could be discriminated by the percent distribution of errors. In our series of chronic alcoholics, a characteristic feature is the very high score for closure difficulty (85%) and for cohesion (73%); while closure difficulty had a high score in all subjects in Abbate and Ferracuti's study, the score of cohesion was low in all their groups.

Thus the presence of high scores only for closure difficulty and cohesion could suggest a diagnosis of alcoholism. This hypothesis, however, must be confirmed by a validation of BVMGT versus reference tests for the diagnosis of alcoholism (at present, this research is in progress in our Center).

INTRODUCTION

Chronic alcohol abuse can lead to different alcohol related pathologies: the relative risk depends on the duration of alcohol abuse (WHO, hazardous alcohol intake > 40 g/day, >25 g/day) and on the differences in the characteristics of drinking habit, including patterns of alcohol intake (daily or not) and drinking "style" (the beverages mainly preferred).

In alcoholics, brain damage and associated functional abnormalities represent one of the dreadful consequences. Organic brain lesions are commonly a consequence of alcohol abuse, although they range from episodic bouts of alcohol related neurotoxicity to irreversible alcohol dementia.

Brain lesions in alcoholics are obviously multifactorial:

- The central nervous system (CNS) may be di-

rectly damaged by ethanol and its metabolic by-products (acetaldehyde, oxygen's free radicals, etc.)¹

- fatty acid ethyl esters, generated by non oxidative metabolism of ethanol, and other non radical metabolites (i.e. alkenals etc.) can lead to an organic damage^{2,3}
- as the intake, absorption and metabolism of nutrients is often impaired, heavy drinking is often complicated by malnutrition and vitamin deficiency, especially thiamine^{4,5}.

It is now established that heavy alcohol intake lasting for more than 10-15 years may lead to a dramatic impairment of brain function, as in severe alcohol-related pathologies like liver cirrhosis.

It is commonly held, however, that a mild impairment in cognitive function can be demonstrated with neuropsychological tests in a very high percentage of alcoholics during the phase of detoxification, as a consequence above all of alcohol neurotoxicity. With the exception of alcohol neurotoxicity, in the remaining alcohol-related clinical conditions (Korsakoff Syndrome, Wernicke encephalopathy, etc.) the presence of specific neuropathological lesions in the brain is readily demonstrated by imaging techniques⁶.

In alcoholics, however, the development of neurological damage may originate both from the selective loss of neurons in some cerebral areas (i.e. mammillary bodies, thalamus, cerebellum, etc.) and from the presence of functional abnormalities and altered morphology of neurons in other areas, as in hippocampal complex and the prefrontal cortex of the brain. At present, the cellular and molecular mechanisms underlying alcohol-related brain damage are only partially understood: severe thiamine deficiency is widely accepted as a major cause of neuronal death in the mammillary bodies and thalamus, very common in alcoholics⁷. In contrast, damage to the hippocampal complex and cerebral cortical neurons is likely to be related to the neurotoxic effects of ethanol or to the consequences of alcohol withdrawal. Recent studies, both "in vitro" and "in vivo", on the effects of acute alcohol exposure, at cellular and molecular level, strongly suggested that synaptic excitation may be reduced^{7,8,9}. The abrupt removal of ethanol after chronic alcohol exposure leads to a condition of "hyperexcitability" of the central nervous system (CNS)¹⁰. This altered neuronal excitability seems to be linked to an enhanced or reduced activation of neuronal synapses in the CNS. Attention has focussed on the increased activation of excitatory neurotransmitter pathways,

probably resulting from a combination of:

- increased N-methyl-D-aspartate receptor (NMDA) activation
- decreased GABA_A receptor activation
- increased function of Voltage-Activated Calcium Channels

These represent a sort of compensatory effect stimulated by alcohol in the neurochemical regulation of the brain but, if it lasts for a long time or is more and more frequently repeated, central neurons may be destroyed via an "excito-toxic" mechanism¹¹.

Indeed, the functional integrity of some neurotransmitter systems, some synaptic receptors, some critical cerebral areas, like the hippocampus and prefrontal areas, are crucial for major cognitive functions, like perception and memory^{12,13,14,15,16,17}

In animal models, it is established that chronic alcohol administration leads to abnormal morphology and function of the hippocampal formation, with a 10-40% loss of neurons (principal cells and interneurons) depending on the duration of alcohol administration and alcohol withdrawal¹⁴. Only few data, however, have been obtained in human models, i.e.: heavy drinkers.

In this study, brain damage was assessed by Bender's Visual Motor Gestalt Test (BVMGT)^{18,19,20,21} in a series of alcoholics on early-stage alcohol abstinence. This test has been a perennial mainstay in the assessment test battery for the past half-century, used for the appraisal of intelligence, as an estimate of nonverbal IQ, as a screening technique for neuropsychological dysfunction, as a clinical tool for sampling visual-motor proficiency, and as a standard projective technique in the assessment of personality. The patient has to draw nine simple geometric figures, elaborated by Bender according to Gestalt theory.

According to Lacks' specific adaptation^{19,12} parameters (standardized "errors") were evaluated, as well as some standardized behavioural observations, allowing a discrimination between patients with organic brain damage and with psychosis. The cut-off between organic and not-organic patients was set at five errors.

MATERIALS AND METHODS

187 alcoholics, in- and out-patients of the Alcohol-Unit, Clinical Medicine Dept., "La Sapienza" University Rome, aged between 19 and 75 yrs 145 males and 42 females, with an alcohol consumption over 80 g ethanol/die for more than 3 yrs, were included in this study. The diagnosis of alcoholism was established according to the criteria of DMS-IV²². Ac-

cording to the Helsinki Convention informed consent was obtained from all subjects.

BVMGT was administered to the patients by a psychologist on a day between 0-7 abstinence day 3.5 ± 2.5 (mean \pm SD). All the patients were abstinent, and the abstinence was checked by personal interview and by the assessment of alcoemia by breath analyzer (ALCOTEST 740 DRÄGWERK, Germany). None of the subjects studied had symptoms of alcohol withdrawal syndrome, as these symptoms (if any) were suppressed by gamma-hydroxy-butyrate (GHB) treatment.

In this study, we employed the BVMGT, modified according to "Lacks Adaptation of Hutt-Briskin Scoring System", taking into account a series of 12 parameters ("errors") forming a scoring system and some standardized behavioural observations, as follows:

Scoring checklist

1. Rotation
2. Overlapping Difficulty
3. Simplification
4. Fragmentation
5. Retrogression
6. Perseveration
7. Collision Tendency
8. Impotence
9. Closure Difficulty
10. Motor Incoordination
11. Angulation Difficulty
12. Cohesion

Time greater than 15 minutes

1. Evidence of fatigue
2. Insufficient attention to stimulus
3. Extremely rapid and careless execution
4. Extreme care and deliberation
5. Dissatisfaction expressed for poorly executed drawings or repeated unsuccessful attempts correct errors
6. Poor motor coordination or hand tremor
7. Rotation
8. Apparent difficulty seeing the figures

For each subject, the evaluation of the BVMGT results (a crucial step for a correct interpretation of BVMGT results) was performed in double blind by a team of independent psychologists, without any direct relationship with the patients.

Statistical analyses were performed using SPSS for Windows™, release 6.01.

RESULTS

Simple statistics of our sample of alcoholics are reported in table 1 and Figure 1.

Figure 2 reports only the frequency of the positive score (1=error) for each Lacks error. The total sample was split into two subsets, <5 errors and >5 errors, according to Lacks, and the scores were reported for both the subsets. Indeed, the number of subjects spending more than 15 minutes on BVMGT was recorded. In the total sample, a score of 4.24 ± 1.62 (m \pm SD) was observed.

Taking into account the distribution of Lacks errors in our patients, in the total sample the highest scores were found for the following parameters:

- closure difficulties: 85.0%
- cohesion: 73.3%
- collision: 48.6%
- overlapping: 43.9%

In the subset A, the highest scores were observed for:

- closure difficulty: 79.1%
- cohesion: 73.6%
- collision or collision tendency: 38.0%
- overlapping difficulty: 29.1%

In subset B, as expected, the scores were higher, but the highest scores were observed for the same parameters, with minor differences only, as follows:

- closure difficulty: 93.5%
- cohesion: 72.7%
- overlapping difficulty: 64.9%
- collision or collision tendency: 63.6%.

2.6% of Group B subjects spent more than 15 min performing BVMGT, but none of Group A.

Table 2 summarizes the results of the statistics performed: Fisher's Exact Test, Yates corrected χ^2 , Exact Confidential Limits.

DISCUSSION

According to some psychologists, tests like BVMGT may be unsuitable in chronic alcoholics, because of incoordination, diffuse muscular tremors (mainly related to alcohol withdrawal) and lack of compliance. In our experience, on subjects in the first days of recovery from chronic alcohol intoxication, motor incoordination was observed only in 21.9%, mainly in the most seriously affected subset (group B); impotence was observed only in 22.5%, demonstrating that the feeling of inability

Table 1.

ALL SUBJECTS	BVMGT 0-4 Subset A	BVMGT >4 Subset B
AGE 19-69 yrs. 44.1 ± 11.2	19-66 yrs 44.4 ± 10.9	22-69 yrs 47.9 ± 10.6
GENDER M = 145 F = 42 N = 187	M = 81 F = 29 N = 111	M = 64 F = 13 N = 77

Table 2. Between group significant levels ($\chi^2=0.05$) with Fisher's exact test and Yates corrected chi-square.

Variables	Fisher's Exact Test	Yates corrected χ^2			Exact Confidential Limit
	P value	χ^2	OR	P value	
ROTATION	0.000054	14.44	12.66	0.0001	2.76 - 116.40
OVERLAPPING DIFFICULTY	0.000001	22.20	4.51	0.0000025	2.31 - 8.84
SIMPLIFICATION	0.00035	12.17	3.44	0.00048	1.66 - 7.24
FRAGMENTATION	0.0039	6.94	12.64	0.008	1.62 - 565.76
RETROGRESSION	0.0000001	24.86	8.41	0.0000006	3.26 - 24.14
PERSEVERATION	0.00012	14.09	3.50	0.00017	1.76 - 7.00
COLLISION TENDENCY	0.00063	10.85	2.86	0.00098	1.50 - 5.49
IMPOTENCE	0.0024	8.54	3.00	0.0034	1.39 - 6.53
CLOSURE DIFFICULTY	0.0066	6.30	3.81	0.01	1.32 - 13.39
MOTOR INCOORDINATION	0.000004	20.53	5.74	0.0000059	2.51 - 13.73
ANGULAR DIFFICULTY	0.0066	6.88	2.42	0.0086	1.23 - 4.78
COHESION	1.00 (n.s.)	0.00	0.95	0.97 (n.s.)	0.47 - 1.95

to cope with the test was only present in few subjects; in all the patients, the symptoms (if any) of alcohol withdrawal (including tremors) were suppressed by GHB treatment, if needed.

The patients were tested on the first days of treatment, to assess the possibility of obtaining a test suitable for diagnosis and choice of treatment at such an early stage.

According to Lacks' adaptation and interpretation of BVMGT, our series was split into two subsets <5 and (5 cumulative error score: only 77 patients out of 187 were in the (5 subset of organic brain damaged patients. The heterogeneity of our total sample for organic damage was confirmed by the cumulative score, 4.24 ± 1.62 , showing a high dispersion of individual data.

According to Abbate and Ferracuti, a discrimination among patients with different diseases (organic brain damage, schizophrenia, personality disorders) could be allowed by the percent distribution of errors. In our series of chronic alcoholics, a characteristic feature is the very high score for closure difficulty (85%) and for cohesion (73%); while closure difficulty had a high score in all the Abbate and Ferracuti's study, the score of cohesion was low in all their groups.

Thus, the presence of high scores only for closure difficulty and cohesion could be suggestive for a diagnosis of alcoholism. This hypothesis, however, must be confirmed by a validation of BVMGT versus reference tests for the diagnosis of alcoholism (at present, this research is in progress at our Centre).

Furthermore, cohesion and closure difficulty seem to be independent of organic damage, as the difference between the scores of the subset with <5 errors (without organic damage) and (5 errors (with organic damage) is not significant for cohesion and significant at low level (0.01, χ^2) for closure difficulty, which seems only slightly affected by organic damage in alcoholics. Highly significant differences between organic- and non-organic brain damage subsets were observed for retrogression, overlapping difficulty and motor incoordination.

According to our data, the high scores of these parameters should depend on organic brain damage more than on alcoholism. This hypothesis should also be validated versus reference procedures for the assessment of organic brain damage like positron emission tomography. Some differences may be observed between the percent distribution of scores in our probable organic damage patients and in the organic damage patients of Abbate and Ferracuti, not reported as alcoholics, suggesting that the frequency of some errors among "organic" subjects could also be influenced by chronic alcoholism.

According to this study, the BVMGT should be recommended for a tentative assessment of brain damage in chronic alcoholics in clinical settings as it is a simple, inexpensive, non invasive method, fully validated for the general population. Further confirmation of our hypothesis on BVMGT interpretation in alcoholics could be obtained from a longitudinal study of chronic alcoholics in progress in our Center, taking into account score variations for some parameters after long-term abstinence.

REFERENCES

- LIEBER CS: Biochemical and molecular basis of alcohol-induced injury to liver and other tissues. *N Engl J Med* 319:1639-1650, 1988.
- LAPOSATA EA, LANGE LG: Presence of non-oxidative ethanol metabolism by human organs commonly damaged by ethanol abuse. *Science* 231: 497-499, 1986.
- BORA PS, LANGE LG: Molecular mechanism of ethanol metabolism by human brain to fatty acid ethyl esters. *Alcohol Clin Exp Res* 17: 28-30, 1993.
- THOMSON AD, RYLE PR, SHAW GK: Ethanol, thiamine, and brain damage. *Alcohol* 18: 27-43; 1983
- VICTOR M, ADAMS RA, COLLINS GH: The Wernicke-Korsakoff Syndrome and Related Disorders Due to Alcoholism and Malnutrition. Philadelphia, FA Davis, 1989.
- CHARNESS ME: Brain lesions in Alcoholics. *Alcohol Clin Exp Res* 17: 2-11, 1993
- BERMAN MO: Severe brain dysfunction: Alcoholic Korsakoff's syndrome. *Alcohol and Health Research World* 14:120-129, 1990
- SIMSON PE, CRISWELL HE, JOHNSON KB: Ethanol inhibits NMDA-evoked electrophysiological activity in vivo. *J Pharmacol Exp Ther* 257: 225-231, 1991.
- DIDLY-MAYFIELD JE, LESLIE SW: Mechanism of inhibition of N-methyl-D-aspartate-stimulated increases in free intracellular calcium by ethanol. *J Neurochem* 56: 1536-1543, 1991.
- HOFFMAN PL, RABE CS, MOSES F, TABAKOFF B: N-methyl-D-aspartate receptors and ethanol: inhibition of calcium flux and cyclic GMP production. *J Neurochem* 52: 1937-1940, 1989.
- LOVINGER DM, WHITE G, WEIGHT FF: Ethanol inhibits NMDA-activated ion current in hippocampal neurons. *Science* 243: 1721-1724, 1989.
- MILNER B: The memory deficit in bilateral hippocampal lesions. *Psychiatr Res Rep* 11: 43-52, 1959.
- LOVINGER DM: Excitotoxicity and alcohol-related brain damage. *Alcohol Clin Exp Res* 17: 19-27, 1993.
- WALKER DW, ZORNETZER SF: Alcohol withdrawal in mice: electroencephalographic and behavioral correlates. *Electroencephalogr Clin Neurophysiol* 36: 233-244, 1974.
- SQUIRE LR: Mechanism of memory. *Science* 232: 1612-1619, 1986.
- CADETE-LEITE A, TAVARES MA, PACHECO MM: Hippocampal mossy fiber-CA3 synapses after chronic alcohol consumption and withdrawal. *Alcohol* 6: 303-310, 1989.
- PHILLIPS SC, CRAGG BG: Chronic consumption of alcohol by adult mice: effect on hippocampal cells and synapses. *Exp Neurol* 80: 218-226, 1983.
- BENDER L: A visual motor Gestalt test and its clinical use. *Res Monogr* 3, Am Orthopsychiatr Ass, New York 1938.
- LACKS P: Bender Gestalt screening for brain dysfunction. John Wiley and Sons, New York 1984.
- PIOTROWSKI C: A review of the clinical and research use of Bender-Gestalt Test, *Perceptual and Motor Skills* 1995, 81, 1272-1274.
- ABBATE L, FERRACUTI S: Validazione del metodo della Lacks per il Test Visuo Motorio della Bender in soggetti con disturbi di personalità, schizofrenia e danno organico cerebrale. *Boll Psicol Appl* 1991; 200: 23-27.
- AMERICAN PSYCHIATRIC ASSOCIATION: Diagnostic and statistical manual IVth edition, 1996