impairment, psychosis, alcoholism, or a history of head injury (Harvey, 1986). They were given tests of frontal lobe functions and sub-tests of the Weschler Adult Intelligence Scale (WAIS). Using matched normative data for Nelson's Modified Wisconsin Card Sorting Test (MWCST), the obsessinals were shown to perseverate significantly more than normals ($r = 2.80$, $P = 0.01$). Their mean percentage perseveration (50%, s.d. = 31) was greater than for patients with gross frontal lobe damage (42%, s.d. = 25). Obsessinals with or without significant perseveration were comparable on age-scaled sub-tests of the WAIS. Perseveration correlated with degree of obsessionality on the Leyton Obsessional Inventory (Spearman's $r = 0.50$, $P = 0.01$), which also correlated, negatively, with alternating category verbal fluency (ACVF) ($r = -0.62$, $P = 0.002$). This latter test and the MWCST assess cognitive set-shifting ability, a specific frontal lobe function.

Although OCD could affect cognitive tasks, such as the ACVF test, via impaired performance efficiency, it seems unlikely that a qualitatively distinctive error, such as perseveration, would occur on this basis. An overall impairment of performance would seem more likely, although this was not evident from the WAIS sub-tests. Whether or not idiopathic OCD is associated with frontal impairment therefore needs to be looked at as a specific issue. It should not be assumed that such impairment necessarily implies the presence of a known brain injury.

References


Macroytosis in alcoholics has at least four different causes, some of which are independent of the duration of drinking habit. Lindenbaum (1980) comments that alcoholics' enlarged RBC can be secondary to liver dysfunction, reticulocytosis in response to blood loss, or folate vitamin deficiency, as well as "the macrocytosis of alcoholism".

Nearly a quarter of Latcham's male subjects and a sixth of the females were not clinically diagnosed as alcohol-dependent. What differences were there, in the correlations reported, between those diagnosed as alcohol-dependent and those diagnosed as suffering from alcohol abuse?

If only 143 male subjects had GGT assays performed then the maximum number of subjects in his Table III should be the same. His data suggest that a considerable proportion of the male subjects had been abstinent for two or more weeks. This is long enough for mildly elevated GGT levels to settle to 'normal'.

The upper limit of normality for GGT of 50 i.u./litre is probably excessively high. We have recently shown (Hambridge & Jones, in preparation), that an upper limit of 40 i.u./litre is probably more clinically valid. Also, many clinicians will have experience of assessing younger, fitter alcoholics with high consumption and minimal damage – however defined.

I suggest that Latcham has not proven his case and that the measurement of GGT, RBC, and MCV remain of considerable value in assessing and managing alcoholics, when considered with the full clinical picture.

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References


Neuroleptic Malignant Syndrome or Lithium Neurotoxicity


Their criticism regarding omission of the original report by Cohen & Cohen (1974) seems to be founded on inconclusive evidence. The descriptive picture in the four patients has been found to be similar to NMS (Frankel & Spring, 1982), but there have