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Review

The relationship between alcoholic cerebellar degeneration and cognitive and emotional functioning

L.E. Fitzpatrick, M. Jackson, S.F. Crowe*

School of Psychological Science, Faculty of Science, Technology and Engineering, La Trobe University, Bundoora, Victoria 3086, Australia

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Abstract

Although it is now widely acknowledged that the cerebellum contributes to the modulation of higher-order cognitive and emotional functions, this relationship has not been extensively explored in perhaps the largest group of individuals with cerebellar damage, chronic alcoholics. Localised damage to the cerebellum has been associated with a specific constellation of deficits and has been termed the 'cerebellar cognitive affective syndrome' (CCAS) [Schmahmann, J.D., Sherman, J.C., 1998. The cerebellar cognitive affective syndrome. Brain 121, 561–579]. The CCAS describes a profile of impairments, including deficits in executive functioning and visuospatial skills, language disruption and altered personality and affective behaviour. It is conceivable that the CCAS may also develop in a subgroup of alcoholics with alcoholic cerebellar degeneration and may in part account for a proportion of the cognitive and affective deficits commonly observed with the condition. While evidence has emerged supporting such a relationship, methodological limitations and the lack of theoretically driven investigation of the contribution of cerebellar dysfunction to cognitive and emotional functioning in chronic alcoholics, preclude definitive conclusions being drawn.

Keywords: Alcohol; Cerebellum; Ataxia; Cognitive; Emotion; Cerebellar cognitive affective syndrome; CCAS

Contents

*Corresponding author. Tel.: +61 3 9479 1380; Fax: +61 3 9479 2471.
E-mail address: s.crowe@latrobe.edu.au (S.F. Crowe).

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1. Introduction

Severe chronic alcoholism has many deleterious effects on the individual and results in a range of neuropsychological and neurological abnormalities (Harper, 1998; Moselhy et al., 2001; Parsons, 1994, 1998; Torvik et al., 1982). Along with generalised cerebral atrophy, specific cerebellar degeneration occurs in a significant proportion of alcoholics and associated clinical signs, including dysmetria and ataxia, are frequently observed. Accumulating evidence now suggests that, aside from its established role in motor coordination, the cerebellum plays a modulatory role in higher-order cognitive and emotional functions. This modulation is thought to occur via the interactive cerebrocerebellar circuitry, which connects the cerebellum to associative and paralimbic cerebral areas (Heyder et al., 2004). A specific constellation of deficits has been observed in individuals with localised damage to the cerebellum which has been termed the 'cerebellar cognitive affective syndrome' (CCAS) (Schmahmann and Sherman, 1998). The CCAS describes a profile of impairments, including deficits in executive functioning and visuospatial skills, language disruption and disturbed personality and affective behaviour. This syndrome is thought to occur due to disruption of the circuitry connecting the cerebellum and other brain regions, particularly those thought to subserve cognitive and affective functions.

Despite the fact that alcoholic cerebellar degeneration is probably the most common form of cerebellar disease (Timmann-Braun and Diener, 2000), to date little research has investigated the contribution of cerebellar damage to the cognitive deficits observed in chronic alcoholics. While evidence has emerged supporting such a relationship (Hillbom et al., 1986), a number of methodological limitations, in association with the small number of previous studies that have examined the relationship, preclude definitive conclusions being drawn. One such limitation is the disparity between studies in the methods employed to measure and quantify cerebellar ataxia, which is frequently used as an index of cerebellar damage. Furthermore, important drinking variables, such as years of heavy drinking, as well as premorbid factors, such as level of education and premorbid intelligence, have not been appropriately matched between groups in some studies (e.g. Hillbom et al., 1986; Sullivan, 2003). Future investigations must more carefully consider these methodological issues and develop designs that allow for replication, so that convergent evidence may accrue.

A further limitation associated with a number of the previous studies in this area is their lack of theoretically guided neuropsychological test batteries and indeed, studies have either included a limited set of measures (Sullivan, 2003), or exhaustive batteries (Hillbom et al., 1986). It is conceivable that the CCAS may develop only in the subgroup of alcoholics with alcoholic cerebellar degeneration and may in part account for a proportion of the cognitive and affective deficits commonly observed in alcoholism more generally. Such a theoretically driven investigation of the contribution of cerebellar dysfunction to cognition in chronic alcoholics is yet to be conducted.

2. Chronic alcohol abuse

Alcohol abuse is a maladaptive pattern of alcohol use that leads to clinically significant impairment or distress and results in a failure to fulfil major role obligations, repeated use in physically dangerous situations, recurrent substance-related legal problems and/or continued use despite persistent interpersonal problems (DSM-IV-TR; American Psychiatric Association, 2000). Chronic alcohol abuse typically leads to alcohol dependence, which is manifested by tolerance and withdrawal (DSM-IV-TR; American Psychiatric Association, 2000). Chronic alcohol abuse is a major contributor to a number of adverse social, physical and mental health outcomes and the economic cost to society due to alcohol abuse and dependency is rated in the billions of dollars in Australia (Collins and Lapsley, 2002). Chronic alcohol abuse has both direct and indirect toxic effects on many organ systems and as a result is a major risk factor in the development of a number of chronic conditions, including liver and cardiovascular disease (Arria and Van Thiel, 1992; Smart and Mann, 1992).

3. Alcoholism and the brain

Another common effect of chronic alcohol abuse is alcohol-related neurologic disorder. While acute alcohol use and intoxication has well-documented deleterious effects in both alcoholic and non-alcoholic persons (Dufour and Fe Caces, 1993; Lehman et al., 1993), chronic alcohol abuse leads to a variety of long-term cerebral insults and consequent cognitive disturbances. Common alterations in brain structure and function, as well as impaired neuropsychological functioning, have been identified in alcoholics (Harper, 1998; Moselhy et al., 2001; Parsons, 1994, 1998; Torvik et al., 1982).

The Wernicke–Korsakoff syndrome (WKS) affects around 2–12 percent of alcoholics, with estimates varying across studies (Torvik, 1987; Victor et al., 1989). It is a disorder most often encountered in alcoholics that is due to a deficiency of thiamine, an essential vitamin for the normal metabolism and function of brain cells. The deficiency may result from decreased dietary intake, but also indirectly through impaired absorption, storage and utilisation of the vitamin (Langlais, 1995; Martin et al., 2003). The acute phase of the disorder (Wernicke's encephalopathy) is characterised by a triad of symptoms, including eye movement abnormalities (opthalmoplegia), confusion and poor muscular coordination (ataxia). However, not all of the classic symptoms are always demonstrable (Victor et al., 1989). The symptoms improve with the administration of thiamine (Meyer et al., 1985), although approximately 80 percent of individuals are left