Sir, We read with interest the review article in Brain by Siclari et al. (2010). Although the authors cite some relevant studies, in our opinion, they do not adequately discuss the possible causal link between alcohol ingestion and disorders of arousal.

Siclari and colleagues state ‘criteria for establishing the putative role of an underlying sleep disorder in a specific violent act have been proposed by Mahowald and coworkers (1990)’; and the authors partially reproduce, as Table 4 at page 3505, material from that earlier publication including the statement: ‘Alcohol or drug intoxication precludes the use of disorder of arousal in forensic cases’.

Our reading of the cited paper from 1990, involving several of the same authors, is subtly different: ‘To assist in the determination of the putative role of an underlying sleep disorder in a specific violent act… the act may… have been potentiated by alcohol ingestion . . .’ (Mahowald et al., 1990).

Two papers published since the review by Siclari et al. (2010) appeared in Brain are relevant to this debate. Arnedt et al. (2011) report that high doses of alcohol increase slow wave sleep in both the first part of sleep and total night-time sleep, hence increasing the propensity for alcohol-related sleepwalking in susceptible individuals; and an epidemiological study by Ohayon et al. (2012) shows that alcohol abuse/dependence confers an increased risk (odds ratio (OR) = 3.5) for frequent sleepwalking, second only to that conferred by obsessive-compulsive disorder (OR = 3.9) and sleep apnoea (OR = 3.7) (Ohayon et al., 2012).

More generally, there is a long history of clinical and forensic case reports linking alcohol intake to sleepwalking, automatism and sleep related violence. Siclari et al. (2010) reference a study by Hartmann (1983) where alcohol ingestion was shown to precipitate sleepwalking in a clinically diagnosed sleepwalker but do not acknowledge the implications of this case report. Although not quoted, a further example exists in the case of ‘R versus Lowe’, heard in the British judiciary, where alcohol dosing before sleep potentiated a confusional arousal fulfilling the ICSD-2 (International classification of sleep disorders, 2nd edn) criteria (American Academy of Sleep Medicine, 2005; Ebrahim et al., 2005; Ebrahim and Fenwick, 2008).

Our view is that factors increasing slow wave sleep and sleep fragmentation (such as sleep disordered breathing and obstructive sleep apnoea), including alcohol, can potentiate and/or trigger disorders of arousal (Roehrs and Roth, 2001; Espa et al., 2002; Andersen et al., 2007; Schenck and Mahowald, 2008; Ebrahim and Shapiro, 2010).

Based on several studies in the worldwide literature, the prevalence of alcohol as a contributor/precipitant to disorders of arousal is estimated to be in the region of 14–25% (Ohayon et al., 2000; Schenck et al., 2007; Trajanovic et al., 2007; Oudiette et al., 2009). Specifically, alcohol at all doses and levels has consistently been shown to increase slow wave sleep in the first half of sleep, when disorders of arousal are most likely to occur. But alcohol, particularly at high doses, also increases all night slow wave sleep percentage (Rundell et al., 1972; MacLean and Cairns, 1982; Block et al., 1986; Rohsrenow et al., 2010; Arnedt et al., 2011).

Alcohol may also cause obstructive sleep apnoea in asymptomatic snorers and is known to worsen sleep apnoea in patients with pre-existing obstructive sleep apnoea. Previous case reports have demonstrated the triggering of disorders of arousal by sleep disordered breathing and/or the treatment of obstructive sleep apnoea (Raschka, 1984; Pressman et al., 2007; Schenck and Mahowald, 2008).

In our opinion, an all encompassing and generalized statement ‘precluding’ a relationship between alcohol ingestion and disorders of arousal is unjustified, and may have unwarranted medico-legal
implications for defendants with a predisposition to parasomnia in response to alcohol (Bonkalo, 1974; Raschka, 1984; Moldofsky et al., 1995; Ohayon et al., 1997; Schenck and Mahowald, 1998; Ebrahim et al., 2005; Ebrahim, 2006; Andersen et al., 2007; Ebrahim and Fenwick, 2008; Ebrahim and Shapiro, 2010).

References


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