Methylphenidate role in Tourette syndrome prevalence

The earliest reports of a causative role for methylphenidate (MPH) in precipitating or exacerbating tics and Tourette syndrome (TS) cited risk estimates of 10% (1976)[1] and 53% (1977)[2]. In sixteen reports (256 cases) published between 1974 and 1977, MPH is cited as the cause of tics in more than 25% of children with attention deficit hyperactivity disorder (ADHD) and special education students[3]. In contrast, only 6% of regular classroom students have tics. A questionnaire to paediatric neurologists in the USA, to determine their usage of MPH in the treatment of ADHD, found estimates of tics in 5% (range 1–20%) of patients medicated conservatively (mean 20 mg daily). Stimulant-induced tics are dose-related, occurring mainly with larger doses.

Before the introduction of MPH for the treatment of ADHD in the 1960s, reference to TS was generally minor or not included in textbooks of neurology. Wilson (1955) has no reference to tics or TS, Ford (1960) refers to tics only as symptoms of encephalitis lethargica, and Merritt (1963) classifies TS as a psychiatric disturbance. Since the 1970s, a neurological basis for TS has been recognized, and children with ADHD are especially susceptible.

Stimulants for the treatment of hyperactive behaviour in children, first used in the USA in 1937, were not generally accepted until the 1960s. In the UK, the diagnosis of ADHD and treatment with MPH has only recently gained limited credence. In the USA, the use of MPH for ADHD has increased annually, especially in the past decade; 1.5 million children received MPH in 1995[4]. This increased use of MPH in the USA correlates with the recognition of TS by neurologists and a plethora of US reports since the 1970s. A similar increase in TS prevalence would not be expected in the UK, since use of MPH was not generally favoured.

Among aetiologies proposed for TS, MPH and other stimulants seem to play a major role in precipitating or exacerbating tics and TS in children in the USA. MPH should be avoided in patients with a personal or family history of TS, and dosage in treatment of ADHD should be conservative.

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Psychopharmacology and the human condition

Dr Charlton (November 1998 *JRSM*, pp 599–601) suggests that one reason for human psychopathology is the mismatch between stone age brains and silicon age culture. I agree with him and I believe that there are many mismatches between human nature and the modern western culture. One of the most important mismatches is the difference between the natural circadian rhythm and the sleep–wave cycle of contemporary humans.

Modern technology, including artificial light, television and telephones, permits humans to extend daytime activities into the first hours of the night. The biological night becomes delayed and humans become clamped in a long-day/short-night mode[1,2]. The nocturnal period during which modern humans rest and sleep, and are exposed to melatonin, high levels of prolactin, low core body temperature and rising levels of cortisol, is shorter at most times of the year than it would be in a natural environment. Daytime core body temperature is lower and sleep-related growth hormone secretion is greater than they would be in short days and long nights. Sleep disruption is increasingly prevalent in modern society and on many occasions people obtain less sleep than they need[1–3].

In the twentieth century the prevalence of bipolar disorder and of rapid-cycling course of that disorder increased[4,5]. It has been suggested that modern abnormal sleep–wake cycles may precipitate or exacerbate bipolar illness[2]. Abnormal rhythms of contemporary human life may also contribute to the development of delayed sleep-phase syndrome, hypernyctohemeral syndrome and various psychiatric and medical disorders. Probably, endemic psychopathology is in part related to the fact that humans have isolated themselves from the natural cycles of light and darkness that have created the endogenous rhythms of life on earth.

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