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Motor symptoms

830 The Maudsley® Prescribing Guidelines in Psychiatry CHAPTER 10 Huntington’s disease Huntington’s disease (HD) is a genetic neurodegenerative disease with an estimated prevalence of 4.88 individuals per 100,000 worldwide, and a higher incidence in Europe and North America.1 The mutant Huntington protein causes neuronal dysfunction and death through several mechanisms, resulting in a triad of motor, cognitive and neuropsychiatric symptoms.2 There are currently no disease-modifying treatments3,4 so symptomatic therapies are used to improve quality of life (Box 10.3). There are few controlled studies to guide practice in this area,3 although some direction can be drawn from published expert opinion and clinical experience. A summary of the available literature is given in this section. Readers are directed to the reports cited for details of dosage regimens and further information about tolerability. Clinicians who treat patients with HD are encouraged to publish reports of both positive and negative outcomes to increase the primary literature base. Motor symptoms Motor disturbances follow a biphasic course – an initial hyperkinetic phase with prominent chorea which tends to plateau over time, and a later hypokinetic phase characterised by bradykinesia, dystonia, balance and gait disturbance.7 With regard to chorea, the goal of treatment is not to obliterate movements but to reduce their severity to achieve better tolerability.5 Treatment pathways are available to guide management.8 First-line treatments include tetrabenazine (licensed) or SGAs (unlicensed) (Table 10.9).8 Monotherapy is preferred to prevent an increased risk of adverse effects and complicating the management of non-motor symptoms.8 Box 10.3 General principles of pharmacological symptom management in Huntington’s disease5,6 ■ ■Tailor management to the needs of the individual patient (treatment is typically initiated when symptoms become bothersome, interfering or socially stigmatising) ■ ■Check whether existing medications are causing or exacerbating symptoms before commencing new treatments ■ ■Prioritise treatment to target the most troublesome symptoms first, with consideration of comorbid features ■ ■Balance therapeutic benefit with the potential for adverse effects ■ ■Start with a low dose and titrate according to tolerability and response (patients are relatively more sensitive to cognitive and motor adverse effects which may also be difficult to distinguish from disease progression) ■ ■Regularly follow up with patients to address changes in treatment (because symptomology evolves with disease progression)

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