



Writer's cramp

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ABSTRACT

Writer's cramp is the most common form of focal, task-specific dystonia. Symptoms frequently evolve in the setting of repetitive hand movements and increased writing demands, and clinical presentations demonstrate a variety of different dystonic patterns of the upper extremity such as while writing or holding a writing utensil. However, why writer's cramp develops still remains much of a mystery. Clinical evaluation of patients with writer's cramp and various theories regarding its pathophysiology are reviewed. Treatment can be challenging and often involves a combination of pharmacologic (e.g., oral medications, botulinum toxin injections) and non-pharmacologic approaches (e.g., neurosurgical or neurostimulatory interventions, rehabilitation therapies, adaptive devices). Management strategies for writer's cramp using both of these approaches will be discussed.

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1. Introduction

Writer's cramp, a type of focal hand dystonia, manifests as an involuntary, sustained posture or contraction of the hand, fingers, and/or arm muscles during writing. Of historical interest, occupational cramps or "craft palsies" have been recognized since early 18th century reports of abnormal hand movements and postures in several occupations (Ramazzini, 1964). Writer's cramp (or "scrivener's palsy") increased in frequency in the 1830's in British Civil Service clerks among others with the introduction of new steel pen nibs that required increased pressure and grip when writing (Pearce, 2005). This review will highlight the clinical features of writer's cramp, evaluations of patients with writer's cramp, and treatment strategies.

2. Epidemiology and etiology

Writer's cramp is one of the most common forms of focal hand dystonia (Sheehy et al., 1988; Torres-Russotto and Perlmutter, 2008). Onset is usually in adulthood, around the 4th decade, and affects men slightly more than women. In an epidemiological study of dystonia in Rochester, Minnesota, the incidence of writer's cramp

was about 2.7/1,000,000 compared to other focal dystonias (24/1,000,000/year) and generalized dystonia (2/1,000,000/year) (Nutt et al., 1988). A European Collaborative group reported an incidence of writer's cramp of 14/1,000,000 population in 8 European countries (Group., 2000). However, many persons with writer's cramp do not seek medical attention and, thus, writer's cramp may be under-recognized and under-reported.

Possible triggers have been hypothesized in focal hand dystonias including writer's cramp. Prolonged, repetitive hand use is commonly reported among those afflicted (Hallett, 2006; Sheehy et al., 1988), and the concepts of over-use, over-learned, stereotyped, repetitive hand patterns and utilization form the basis of several theories regarding the pathophysiology of writer's cramp and focal hand dystonia (Byl et al., 1997; Hallett, 2006). The relationship of hand injury to the development of writer's cramp is unclear; although limb dystonia has been reported following peripheral trauma, true causation is difficult to establish. Of 91 patients with writer's cramp, 5% reported hand injuries in the 3 months preceding their symptoms (Sheehy and Marsden, 1982). Positive family histories have been reported in about 5–20% of writer's cramp patients (Martinez-Martin and Bermejo Pareja, 1985; Sheehy et al., 1988). More recent studies suggest that genetic variants may contribute to writer's cramp and musician's dystonia. Recently, an intronic variant (rs11655081) in the arylsulfatase G (ARSG) gene was shown to be associated with musician's dystonia and writer's cramp in a genome-wide association study; this genetic variant, however, was not associated with other

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focal dystonia such as cervical dystonia or blepharospasm (Lohmann et al., 2014). The ARSG gene may play a role in dystonia as it encodes proteins involved in cell signaling, protein degradation, and hormone biosynthesis. In a study of the ARSG gene in 72 writer's cramp patients and 158 musicians with dystonia, one rare missense variant, rs61999318 (p.I493T), was significantly enriched in the writer's cramp patients compared to European Americans in a large database, though no conclusive mutation in ARSG was detected (Nibbeling et al., 2015). DYT1 is an infrequent cause of hand dystonia or musician's cramp (Friedman et al., 2000; Kamm et al., 2000).

3. Clinical features and evaluation

3.1. Symptoms

Symptoms of writer's cramp are usually insidious in onset with progression over months. Patients may report tightness in the forearm or stiffness when writing or fatigue with prolonged writing. The hand, fingers, and/or arm may have involuntary flexion or extension posture during writing. There is decreased speed and cramping with prolonged writing. Tremor may accompany the abnormal writing posture in about 45% (Marsden and Sheehy, 1990). Common dystonic patterns involve flexion, extension, supination, pronation, or a combination. Predominant flexion patterns often lead to increased pressure on the writing surface, bold script, and decreased ability to loop in cursive writing, whereas with predominant extensor patterns, there may be faint script, difficulty putting the pen on the paper, or fingers/thumb lifting off of the pen.

3.2. Classification and course

Writer's cramp has been classified as simple, complex, or progressive. Simple refers to the presence of dystonia only with the task of writing; however, when the dystonia extends to other manual tasks (e.g., eating, shaving, typing, etc), these patients are classified as progressive writer's cramp. Some patients have a complex writer's cramp from initial onset with multiple manual tasks affected (Marsden and Sheehy, 1990; Sheehy and Marsden, 1982). In about 25%, writer's cramp may spread proximally or to the non-affected hand, if the patient switches to writing with the other hand. Spontaneous remissions are rare, occurring in about 5%, and more likely to occur earlier in the course (Marsden and Sheehy, 1990).

3.3. History and examination

In the evaluation of a patient with suspected writer's cramp, important historical points to consider include the following: duration and course of writing difficulty; descriptions of abnormal postures or movements; differences with type of pen, surface (e.g., blackboard), or writing style (e.g., print, cursive); sensory tricks; trial of non-dominant hand; task-specificity; other abnormal movements; history of trauma or period of intense writing; and family history of writer's cramp or movement disorders. Specific examination features to consider include observing the hand posture at rest, holding a pen, and while writing; posturing of fingers with outstretched arms; tremor at rest, action, or posture; writing in different situations (e.g., with and without adaptations or compensatory maneuvers, pens of different thicknesses, non-dominant hand); and other tasks (e.g., typing, eating).

3.3.1. Mirror dystonia

Mirror dystonia reflects involuntary movements, abnormal

postures, tremors, or jerks of the resting hand when writing with the opposite hand (typically the non-dominant and non-dystonic hand). Mirror dystonia provides insights into the pathophysiology of writer's cramp and suggests abnormal cortical activation patterns (Cox et al., 2012) (see review Cox et al., 2012). A prospective analysis of 65 patients with writer's cramp revealed that about 45% of patients had mirror dystonia (Jedynak et al., 2001). Merello et al. describe functional magnetic resonance imaging (fMRI) findings in a patient with writer's cramp and mirror dystonia, compared to a patient with simple writer's cramp without mirror dystonia and a healthy control. While a small study, the fMRI analyses revealed that the patient with writer's cramp and mirror dystonia exhibited a more widespread brain activation pattern, invoking the contralateral and ipsilateral posterior parietal cortex and putamen and ipsilateral inferior frontal gyrus, compared to the patient with writer's cramp but lacking mirror dystonia and a healthy control (Merello et al., 2006). Bilateral activation may suggest altered interhemispheric or transcallosal inhibition in the presence of mirror dystonia. Loss of interhemispheric inhibition also was found in an electrophysiological/transcranial magnetic stimulation (TMS) study comparing electromyographic (EMG) recordings of an index finger movement and surrounding thumb muscle movement during primary motor cortex stimulation in patients with writer's cramp with and without mirror dystonia and healthy controls (Beck et al., 2009). Writer's cramp patients with mirror dystonia exhibited a selective, time-dependent decrease of interhemispheric inhibition between homologous surrounding muscles hand muscles in the (premotor) phase prior to EMG onset.

Mirror dystonia may be helpful for distinguishing dystonic movements from secondary compensatory strategies (Jedynak et al., 2001). In a small retrospective study, its use was associated with greater response to botulinum toxin injections (Singer et al., 2005). Mirror dystonia also has been suggested to help select which muscles to treat with botulinum toxin injections, though further study with prospective trials and larger series may be informative.

3.3.2. Sensory tricks

Sensory tricks may provide transient improvement in writer's cramp symptoms (Ramos et al., 2014) (see review Ramos et al., 2014). Examples include different shaped pens (thicker barrel, thicker point, etc), writing with chalk or at a blackboard, adding a rubber grip holder to the pen, holding the pen in a different position, touching part of their forearm, wrist, or dystonic hand, or cooling the hand in cold water (Hauser and Burke, 2002; Loyola et al., 2013; Pohl et al., 2002; Ramos et al., 2014). Sensory tricks are frequently employed in other dystonias (e.g., over 70% of patients with cervical dystonia), but their frequency in writer's cramp is less determined, although one small study including 10 writer's cramp patients reported sensory tricks in 20% (Loyola et al., 2013). While we do not fully understand how sensory tricks work, their presence provides insights into the pathophysiology of dystonia, suggesting that they may normalize aberrant links between sensory input and motor output, decrease abnormal cortical facilitation, and inhibit overactive patterns.

3.3.3. Diagnostic studies and rating scales

In the evaluation of writer's cramp, other diagnostic studies are rarely necessary. If there are symptoms reflective of peripheral nerve problems, which could be secondary to dystonic posturing (e.g., prolonged wrist flexion and median nerve compression), EMG and nerve conduction studies can be performed. Brain and/or cervical MRI scans are usually unnecessary, unless other relevant symptoms are present. Testing for the DYT1 mutation may be considered in a patient with young onset dystonia (<26 years

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