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Tremor and Other Hyperkinetic Movements

Reviews

Focal Task-specific Dystonia—From Early Descriptions to a New, Modern Formulation

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Abstract

Background: Vivid descriptions of the phenomenology of focal task-specific dystonia (FTSD) date back to the late nineteenth century.

Methods: In this review, I summarize the natural history, phenomenology, and treatment of FTSD, focusing on nineteenth-century neurologists' descriptions of the phenomenology, etiology, treatment, and mechanism.

Results: Examining these texts through a twenty-first-century lens, the "modern" ideas of a dystonic endophenotype, disordered physiology, and dystonic metabolic networks actually appeared in these texts more than a century ago.

Discussion: By incorporating these ideas with recent investigations, I present a new conceptual model for understanding this mysterious malady.

Keywords: Dystonia, task-specific, musician, writer's cramp

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Introduction

Focal task-specific dystonia (FTSD) is a mysterious disorder that has fascinated neurologists for almost two centuries. By definition, "focal" dystonia affects one and only one body part or region, such as the upper face (eyelids or brow), lower cranial structures (lower face, jaw, tongue, pharyngeal muscles), larynx (vocal cords), arms (shoulder girdle, upper arm, hand), leg (proximal or distal leg, foot), or trunk.[1] "Task-specificity" describes an involuntary movement triggered by a specific action, such as eating, speaking, singing, or using the arm or leg in an infinite number of individual tasks. The phenomenon of task-specificity is not specific to dystonia; task-specific tremors may affect the hand (e.g., primary writing tremor)[2] the voice (e.g., isolated vocal tremor with singing),[3] or even the muscles of the lips and tongue involved in producing air flow into the mouth of a woodwind or brass instrument. However, FTSD implies an involuntary movement disorder characterized by sustained or twisting postures,[4] affecting a single body region, only when a specific task is performed (Video 1).

In this paper, I trace the evolution of understanding of FTSD from its earliest descriptions in the late nineteenth and early twentieth centuries. This rich phenomenology was largely ignored for much of the twentieth century. I then illustrate how Gowers' magnificent description of FTSD foreshadowed our modern understanding of the disorder's anatomy and physiology. I close with an attempt to integrate these features of natural history and phenomenology with modern investigations into a working model of the disorder.

Historical Background

Although Sir Charles Bell mentioned the entity of writer's cramp in 1830,[5] Samuel Sully penned the first detailed account of what he called "scrivener's palsy".[6] Sully was the first to specifically emphasize the unusual task-specific nature of the disorder, using the elegant prose of the era:

> The disease, as the name implies, shows itself outwardly in a palsy of the writing powers. The muscles cease to obey the mandate of the will….The paralyzed scrivener, though he cannot write, can amuse himself in his garden, can shoot, and can cut his

Video 1. **The Phenomenology of Focal Task-specific Dystonia is Demonstrated.** The first segment demonstrates two patients with writer's cramp. A classic pattern of involvement of flexion of the small muscles of the thumb and index finger is seen. The second patient demonstrates a more unusual pattern of extension of the third through fifth fingers. The next patient demonstrates treadmill dystonia, exercise-induced dystonia of the left foot selectively triggered by walking. No evidence of parkinsonism was present on her examination. Abductor dysphonia appears in the next patient, with breathiness and loss of vocal support triggered selectively by words with AA vowel sounds following consonants. The next six patients, all musicians, illustrate the selective involvement of a single finger by dystonia. Two patients each (banjo, flute, and violin) display task-specific dystonic contractions of one finger. The banjo players display dystonic flexion of the right index finger, delaying the timing of plucking the string. The flute players both involve the left pinky, with flexion of the fifth finger's metacarpophalyngeal joint pulling the finger off the instrumental key. The following two violinists both demonstrate a trigger of dystonic flexion of the left ring finger (with some coincident flexion of the pinky as well). Next, two musicians with prominent sensory tricks appear. The first is a traditional Irish accordion player, whose middle finger involuntarily extends off the key while playing. Application of a simple finger splint to the adjacent fourth finger allows him to "bring the third finger back into play" and to use it on the keyboard. Next, a clarinetist with a complex dystonia of the right wrist and finger serendipitously discovered that holding a pencil between his second and third fingers (something he did to facilitate marking the music while practicing), immediately and significantly improved his dystonia. The size and texture of the pencil impacted the efficacy of the trick. The final two segments demonstrate two pianists with isolated task-specific dystonia involving flexion of the index finger. The first shows flexion of the right index finger during performance of a Scarlatti sonata. Four weeks after injection of botulinum toxin into the flexor superficialis of the index finger, there is marked improvement. The last patient demonstrates a plastic brace that he built to prevent flexion of the left index finger during playing. A similar pattern of injections (performed by Dr. David Simpson) markedly improved his dystonia as well.

> meat like a Christian at the dinner-table; indeed he can do almost anything he likes, except earn his daily bread as a scribbler.

The late nineteenth century witnessed four pioneering neurologists who wrote about FTSD, using the term "functional spasm" or "occupational neurosis". These giants, Duchenne,[7] Hammond,[8] Oppenheim,[9] and Gowers,[10] used terms like "functional" or "neurosis" to infer a disturbance in nervous system function rather than a structural abnormality within the brain. They did not mean that the disorder was purely psychologically based, an inference that permeates much of the mid-twentieth-century literature on FTSD. The content of their descriptions is summarized in Table 1.

Gowers devoted a full chapter of his classic "Diseases of the nervous system" to FTSD, and his descriptions are unmatched for their elegance and clarity. These early neurologists recognized that FTSD is far more common in men than women, and that symptoms typically begin in the third or fourth decade of life. A family history of other affected members is common, as are comorbid psychiatric illnesses such as anxiety or depression. Symptoms of FTSD almost always begin insidiously, with a loss of automaticity and a need to exert increased effort to perform the task. FTSD usually worsens over time, and both Gowers and Oppenheim described the spread of dystonia to involve other tasks in the affected body part. Spontaneous remissions are uncommon, but Gowers was the first to report them.

All four authors wrote extensively about the diverse phenomenology of FTSD. In this one area, Hammond, Duchenne, and Oppenheim outshine Gowers:

> Hammond: There is a class of paralyses produced by the habitual use of a particular class of muscles in the same way for a long time. Thus we have writer's paralysis, telegrapher's paralysis, hammer paralysis, and so on….
>
> Duchenne: A fencing-master whose humerus of the sword arm rotated inwards, and whose elbow became strongly extended, as soon as he put himself 'on guard'…A turner, the flexors of whose ankle contracted as soon as the foot was placed on the treadle of the lathe…A gentleman whose head turned to the right by the actions of the rotators whenever he read….A clergyman had a mania for playing on the hautboy (Serpent)…When he played on his instrument his spasm caused him to emit sounds which amused his congregation.
>
> Oppenheim: Piano-player's cramp consists, as a rule, in abnormal muscular contractions, from which a finger or several remain lifted from the keys or are pressed upon them….Violinists' cramp may affect the bowing hand or the hand pressing upon the strings, occasionally both….An occupational neurosis of the labial muscles was seen by me in a horn-blower. As soon as he placed the mouthpiece to his lips a spasm came on in the orbicularis oris, so that the patient was unable to bring out a tune…A dancer's cramp, occurring in ballet dancers in the beginning of dancing when gliding forward upon the toes, which consists of painful tonic contractions of the thigh muscles.

Gowers, Duchenne, and Hammond noted the phenomenon of the sensory trick, describing trick devices that patients employed to temporarily improve their dystonic symptoms. However, the French neurologists Brissaud[11] and his pupils Meige and Feindel[12] were the

Table 1. Features of Focal Task-specific Dystonia Discussed by Gowers (G), Duchenne (D), Hammond (H), and Oppenheim (O) Summarized by Topic. ¶ indicates mention by the author

Category	Specific Feature	G	D	H	O
Demographic	M>F	¶		¶	
	Age distribution	¶		¶	
	Family history	¶	¶		¶
	Psychiatric comorbidity	¶			¶
Natural history	Acute presentations of dystonia	¶			
	Progression of severity	¶		¶	
	Spread to other tasks with the affected hand	¶			¶
	Spontaneous remission of symptoms	¶			
Phenomenology	Pure task-specificity of the dystonia	¶		¶	
	Sensory trick	¶			
	Sensory trick device used	¶	¶	¶	
	Other occupational neuroses (other forms of focal dystonia besides writer's cramp)	¶			¶
	Sportsman		¶		
	Proximal involvement of the affected limb		¶		
	Leg dystonia		¶		¶
	Task-specific torticollis		¶		
	Respiratory dysphonia		¶		¶
	Musicians' hand dystonia				¶
	Musicians' embouchure dystonia				¶
Etiology	Trauma as trigger of dystonia	¶			¶
	Task mechanics as causative factor in dystonia	¶		¶	¶
	Instrument as predisposition to develop dystonia	¶			
Mechanism	Central hypothesis of the cause of dystonia	¶	¶	¶	¶
	Idea of motor learning	¶			
	Idea of a motor network	¶			
	Idea of motor engrams	¶			
Treatment	Idea of prevention of dystonia	¶			
	Rest as a treatment	¶		¶	¶
	Early treatment as key to response	¶			
	Sensory motor retraining protocols	¶			¶
	Peripheral surgery on the affected limb	¶		¶	
	Limb immobilization as a proposed treatment	¶			

first to fully articulate the phenomenology of sensory gestes. In their classic monograph, "Tics and their treatment"[12] translated by Wilson, Meige and Feindel described the phenomenology of the geste: "Some patients whose inventive faculty leads them to adopt singular attributes, to execute curious gestures, to utilize elaborate apparatus…". Unfortunately they misinterpreted a common phenomenon, improvement of dystonia by initiating the geste before it has been completed, as a psychogenic sign. This phenomenon is now well established in patients with cervical dystonia and blepharospasm.[13]

Etiology

Hammond, Oppenheim, and Gowers commented on the role of inherited vs. environmental factors in FTSD. They noted that the male predilection, the frequent occurrence of similarly affected family members, and coincident psychiatric comorbidities supported a genetic contribution. Gowers however also emphasized the role of the environment, noting peripheral trauma as a possible trigger for dystonia: "Local disease or injury…may distinctly cooperate in developing the morbid state…In many recorded instances…some painful affection of a finger has preceded the onset…". He described the influence of the environment and mechanical demands of writing on writer's cramp as follows:

> The chief agent in the production of the malady is the act of writing, which has usually been excessive in degree…The occurrence of the disease is influenced less by the amount than by the manner of writing…it is the mode in which the pen is moved that chiefly determines the occurrence of the disease…the smaller the muscles employed, the greater must be the relative degree of contraction…Forming shorthand characters compels a very free style of writing, generally from the shoulder…the result is that they have an almost complete immunity from the disease.

More than a century later, studies on the role of genetics and environment in musicians' dystonia have confirmed many of Gowers' predictions. Like writer's cramp, musicians' dystonia occurs much more frequently in men than women (4 : 1).[14] Anxiety and obsessive-compulsive traits are common in affected patients, likely due to an underlying predisposition rather than a reaction to the illness.[15] A recent genome-wide association study in families with musicians' dystonia or writer's cramp revealed a risk variant at the Arylsulfatase G gene.[16]

Environmental factors also exert a powerful influence on musicians' dystonia. The disorder typically begins in the middle to late 30s, and accumulated training hours correlate with risk. Certain instruments such as piano, violin, and guitar are more commonly encountered, while others such as cello and double bass are rarely seen.[14] The mechanical requirements of the aforementioned instruments, which demand fine simultaneous activation of fingers on the keys or string, may predispose to the development of dystonia. It also appears that dystonia preferentially involves the hand engaged in the more complex motor task. For example, the right hand is affected three times as often as the left among keyboard players (piano, organ, harpsichord) and plectrum instrumentalists (guitar, banjo).[14] In these instruments, the right hand typically carries the greater technical burden. The situation is reversed in bowed instruments (violin, viola), where the left hand is much more commonly affected than the right. This makes sense, since the left (fingering) hand requires much more individual finger movement than the right (bowing) hand. Among woodwind players, where the mechanical demands of the hands to depress the instruments' keys are similar, both hands are affected equally by dystonia. The sole exception to this rule is flautists where the left hand is preferentially affected, perhaps due to the added mechanical demand of supporting the instrument with the left hand.

Gowers elegantly traced the predilection for development of writer's cramp to the use of the small intrinsic muscles of the thumb, index, and middle fingers, particularly in clerks trained to write with hard-nibbed pens. The pattern of dystonic finger movements in musicians' dystonia also appears to be related to the instrument played. The most common pattern involves flexion of the fourth and fifth fingers, particularly in the right hand of pianists or guitarists or in the left hand of violinists. Among woodwind players, a classic pattern involves the lumbricals of the third or fourth finger, extending the digit and pushing the finger off of the key. A third pattern, seen in pianists and banjo players, involves flexion of the thumb and index finger, quite similar in appearance to the most common pattern seen in patients with writer's cramp.[17]

Among patients with embouchure dystonia (FTSD of the muscles of the mouth, tongue, and jaw involved in controlling the flow of air into a brass or woodwind mouthpiece), six different dystonic phenotypes have been described: embouchure tremor, lip-pulling, lip-lock, jaw involvement, tongue involvement, and task-specific Meige.[18] The mechanical demands of the various instruments' embouchure may influence the phenotype of dystonia. High-register brass instruments such as trumpet and French horn impose high pressures on the lips and orbicularis oris muscles. The majority of embouchure tremor and lip-pulling patients play horn or trumpet, perhaps reflecting this mechanical demand. In contrast, all patients with the lip-lock phenotype play low-register brass instruments (trombone and tuba), reflecting the technical demands of these instruments to play separated and repeated notes as a musical bass line. Finally, the majority of patients who develop embouchure dystonia affecting the jaw or tongue play woodwind instruments such as oboe, clarinet, bassoon, and flute. The mechanical demands of the woodwind embouchure require the upper and lower front teeth to grasp the reed of an oboe or bassoon. Similarly, in flautists the lower jaw is fixed against the bottom part of the flute mouthpiece. In contrast brass instrumentalists do not fix their teeth or jaw to play, and they are thus less mechanically constrained. More than half of all patients seen with jaw or tongue embouchure dystonia had already experienced spread of dystonia to other oral tasks such as speaking or eating when evaluated.

Treatment

Gowers, Hammond, and Oppenheim all described treatments they employed to aid their patients with writer's cramp. Gowers described the challenge of treating the disorder:

> The disease when well developed, is one in which the prognosis is always uncertain, and often unfavorable…

He also raised the idea that prevention might be possible:

> Writer's cramp might probably be prevented to a very large extent, if not entirely…The mode of writing is a matter of education, and prevention of the disease rests therefore not with doctors but with teachers.

This idea that pedagogy or training regimen might influence the development of dystonia resonates deeply with movement disorder neurologists who treat musicians with FTSD. Whether practice habits, teaching styles, emphasis on technical exercises, or other factors influence the development of musicians' dystonia is unknown. However, anecdotal experience from select patients' histories suggests this may be true. A small minority of musicians clearly remember the onset of their first dystonic symptoms, occurring directly after an abrupt increase in practice time or a significant alteration in their instrumental set-up. Others remember dystonic symptoms beginning immediately after repeatedly practicing (sometimes for hours at a time) a particularly difficult musical passage or challenging technical exercise. Common sense dictates that musicians should avoid such triggers whenever possible.

Gowers argued that rest, defined as complete abstinence from the triggering task, was a very effective treatment:

> Treatment, to be effective, should be early. The commencing symptoms often pass away with a brief rest; a month's abstinence from writing at the onset will do more than a year's rest if the disease has continued for six months…

Unfortunately, most patients with writer's cramp present to the neurologist years after symptoms begin. Musicians may present earlier, although in clinical practice rest rarely resolves symptoms of dystonia.

Modern investigators have "rediscovered" many of the treatments that Gowers originally described. For example anticholinergic agents[19] and THC (tetrahydrocannabinol)[20] have both been shown to symptomatically improve musicians' hand dystonia. Gowers mentions both as treatments for writer's cramp:

> Injections of atropine have been strongly recommended…Indian hemp by the mouth…or inunctions of extract of belladonna….are the most useful.

Sensory-motor retraining protocols, for example training affected patients to read Braille[21] or to recognize objects by touch,[22] have been used to treat patients with writer's cramp and musicians' dystonia. These protocols involve training patients over a period of weeks or months to perform a novel task with the affected hand. Gowers suggested that training the sensory-motor apparatus of the hand was useful, and in particular he advocated exercises that avoided triggering dystonic movements:

> Gymnastic exercises are often distinctly useful, regular flexion and extension movements of the fingers and hand, for which, with advantage, a "dumb piano" may be used. They should be as different as possible from the movements that induce the spasm.

Others have attempted to reset the sensory-motor network by constraining the affected limb. Priori et al.[23] first reported improvement in musicians' dystonia by immobilizing the affected arm for 4–6 weeks. Candia et al.[24] reported similar success with a more limited restraining technique focused on individual fingers. Subsequent studies have failed to confirm these initial robust reports;[25] Gowers' observations may have predicted this as well:

> I have found no good result from rendering the arm for a time immobile by a plaster of Paris bandage.

Peripheral surgical procedures such as tenotomy, once commonly used, are no longer performed. Gowers noted this too:

> Among other means of treatment which have been recommended is tenotomy…no less than fifty tendons were successively divided…the patient at the end of the treatment was slightly improved.

Finally, Gowers mentions the use of electrical stimulation for treatment of writer's cramp; "Electricity has been extensively used, and strongly recommended in the treatment of writer's cramp." Transcranial magnetic stimulation over the motor cortex has recently been studied as a treatment for FTSD of the hand,[26] although the long-term efficacy of this approach is still unknown.

Gowers could not have predicted the development of botulinum toxin injections, the most widely used and probably most effective treatment for FTSD of the hand.[27] Nor could he have foreseen the development of stereotactic surgery. Recently, Horisawa et al. have shown that Voa (ventral-oralis-anterior) thalamotomy may also be an effective and safe approach for patients whose careers have been derailed by FTSD.[28]

Mechanism

Modern investigations over the last two decades have extensively investigated the mechanism of FTSD. Remarkably, Gowers (below), Duchenne, Hammond, and Oppenheim all posited a central localization in writer's cramp and allied disorders:

> The affection is primarily and essentially central, the result of a deranged action in the centres concerned in the act of writing…

Gowers' predictions are particularly startling; he predicted a structural correlate to explain writer's cramp:

> It has been objected to the central theory that it assumes the existence of a centre for the coordination of writing, and

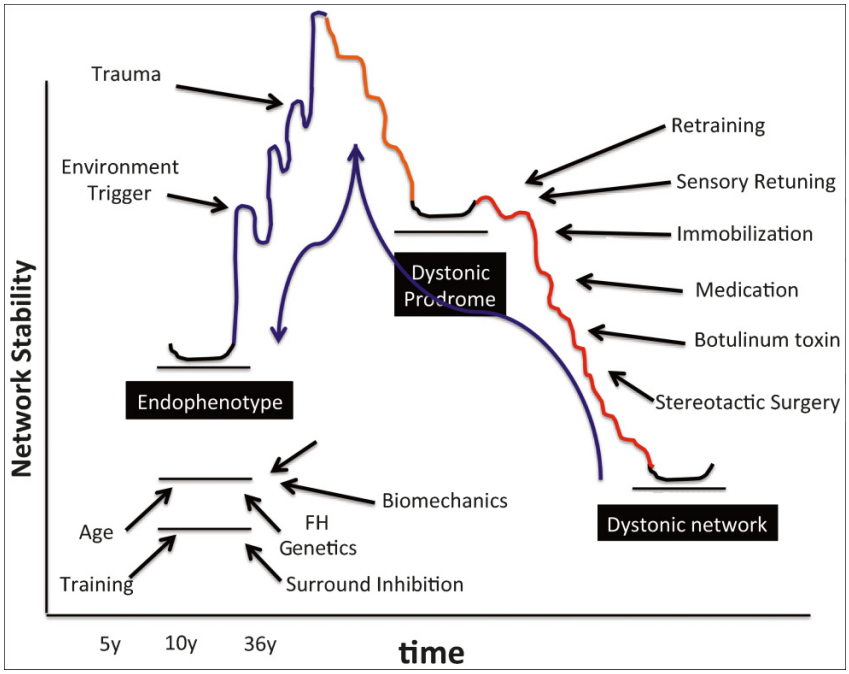

Figure 1. **A New Working Model of Focal Task-specific Dystonia.** The y-axis represents the stability of the sensory motor network. An elevation in the y-axis depicts the activation energy barrier to be overcome to achieve a new network. Time is represented on the x-axis. Starting from the left side of the graph, factors such as age, training regimen, gender, peripheral biomechanics, family history, and susceptibility to plasticity (surround inhibition) impact the stability of the baseline endophenotype network. A family history of dystonia, male gender, and late age of onset of training push the baseline endophenotype higher on the y-axis, making the network less stable and making an individual more susceptible to develop dystonia. At some point, a peripheral trigger or trauma may push the network up from left to right, over the activation energy barriers (blue hills), resulting in an eventual slide down (orange line) to a meta-stable state of a dystonic prodrome. This dystonic prodrome network is not stable, tending to degenerate (red line) and progressing to the right to a formed dystonic network (far right). Treatments such as sensory motor retraining, limb immobilization, medications, botulinum toxin injections, and stereotactic surgery are aimed at pushing the network from right to left, back up the activation energy scheme (solid blue line with arrows), and in the best-case scenario, returning the patient to a stable non-dystonic network.

> therefore of every separate action which any one part of the body can perform…The objection is invalid.

Exner proposed the existence of a cortical region selectively responsible for writing as well.[29] In a recent study using electrocorticography and functional magnetic resonance imaging, the existence of such a "graphemic motor frontal" region was confirmed.[30] Gowers also proposed a mechanism for motor learning, foreshadowing the idea of cerebral plasticity:

> The acquisition of the power of performing any action with ease means the "education" of the nervous centres concerned in it, the establishment of a tendency to the associated action of nerve cells, in perfect adjustment of varied order and degree, with the least possible effort of excitation and control.

This passage foreshadows the modern findings of central plasticity in adults during acquisition of motor engrams, such as musical skill, now an accepted phenomenon.[31] Gowers also predicted the idea of motor system networks underlying skilled motor performance:

> It must be remembered that the conception of a physiological centre does not necessarily involve that of a local limitation. Nerve cells act together that are far apart, and those that are adjacent are often independent.

Modern neuroimaging studies have demonstrated involvement of a widely distributed abnormal motor network in dystonia, involving the somatosensory, primary motor, supplementary motor, thalamic, basal ganglia and cerebellar circuitry.[32]

Finally, Gowers also conceived of a biological mechanism to explain FTSD:

> The clearest conception we can form…is that this process of lowering of resistance between nerve cells has gone too far…Although, when the same cells are excited in a different order, the resistance is normal in proportion as the order differs from that involved in the act of writing.

This passage seems to predict the modern concept of surround inhibition, the mechanism by which competing motor programs are selected and inhibited at the output level of the pallidum. Defects in surround inhibition have been demonstrated in patients with writer's cramp and musicians' dystonia.[33]

A New Formulation

To close, I present a new working conceptual model of FTSD that attempts to integrate phenomenology, physiology, and treatment (Figure 1). The stability of a motor network is represented as a level on the y-axis, with less stable networks placed higher, and more stable networks lower. A stable network can become unstable over time (the x-axis) by overcoming an activation energy barrier. Risk factors such as age, training regimen, gender, peripheral biomechanics, family history, and susceptibility to plasticity (surround inhibition) impact the stability of the baseline sensory motor network. A family history of dystonia, male gender, and late age of onset of training make an individual more susceptible to develop dystonia, demonstrated as a rise in the network's position on the y-axis. Figure 1 starts in the far lower left corner, with the endophenotype network. At some point, a peripheral trigger or trauma destabilizes the network, moving it to the right and pushing it up over the activation energy barriers (blue hills), resulting in a slide down (orange line) to a meta-stable state of a dystonic prodrome network. The dystonic prodrome network is an inherently unstable network, and it degenerates (red line) to fall down and become a stable dystonic network. Treatments such as sensory motor retraining, limb immobilization, medications, botulinum toxin injections, and stereotactic surgery push the network right to left, back up the activation energy scheme (solid blue line with arrows), and, in the best-case scenario, return the patient to a stable non-dystonic network.

Previous formulations have emphasized the dual roles of genetics and environment in the development of dystonia.[34] We believe that the present model offers several advantages in thinking about possible mechanisms of FTSD. Recent work has supported the concept that the motor and sensory systems involved in dystonia involve multiple brain regions, and that assigning the origin of dystonia to a single brain area such as the putamen or globus pallidus is probably overly simplistic. For example, recent work has determined that the cerebellum and its connections to the thalamus are integrally involved in the dystonic network,[35] and other studies have displayed abnormalities in the primary and supplementary motor area.[36] Clinical descriptions from the four nineteenth-century neurologists confirm modern clinical experience that many patients experience a progression in their dystonia severity, starting with mild, pure task-specific dystonia, progressing to more established dystonic patterns, and finally to a stable and abnormal automated pattern of skilled movement. This scenario does not necessarily imply an irreversible course however. Recent studies of deep brain stimulation support the idea that long-term stimulation might "reset" the dystonic network.[37] Further, sustained long-term improvements are occasionally observed from interventions such as botulinum toxin injections, particularly in FTSD patients. We thus thought it important to include the idea of a meta-stable "dystonic prodrome network" in the model, one which is potentially reversible if recognized early and managed appropriately.

Conclusion

In this paper, I have reviewed the content and history of nineteenth- and twentieth-century neurologists' views of FTSD. Gowers, Duchenne, Hammond, and Oppenheim defined the rich phenomenology of the disorder. The predictions of Gowers into disease mechanism and response to treatment are quite extraordinary when viewed through a twenty-first-century perspective.

Many important questions remain unresolved, including three issues of great practical importance: Can FTSD be prevented by proper instruction?; Do the available treatments for FTSD (botulinum toxin, stereotactic surgery) modulate the natural history of the disorder?; and, finally, Can FTSD be reversed? By establishing international working groups focusing on musicians' dystonia, investigators have begun to organize approaches using modern techniques of genetics, functional rating scales, and clinical trials to help answer these questions.

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