How psychogenic is dystonia? views from past to present

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In the last few centuries, there has been a constant sway between organic and psychogenic explanations for dystonia. In the current study, we investigate this history, assuming the perspective of a spectrum from organic to psychogenic, between which ideas were moving. We have focussed on (i) primary generalized dystonia, (ii) cervical dystonia, (iii) writer’s cramp and (iv) fixed dystonia related to complex regional pain syndrome. We have studied medical texts published since the 19th century and their references. Jean-Martin Charcot advocated the concept of hysteria, disorders in which, besides predisposition, environmental factors were involved in their pathogenesis. Sigmund Freud introduced psychoanalysis as an explanatory therapy for psychic disorders. Previous theories, together with the lack of an organic substrate for dystonia, made a strong case for psychogenic explanations. Consequently, many dystonia patients were told that they suffered from psychological conflicts and were treated for them. However, after the description of new hereditary cases in the 1950s, the limited efficacy of psychotherapy in torsion dystonia, the effects of surgical treatments and the lesion studies in the 1960s, more physicians became convinced of the organic nature. The culminating point was the discovery of the DYT1 gene in 1997. In the meantime, experts had already convinced the neurological community that cervical dystonia and writer’s cramp were focal dystonias, i.e. minor forms of generalized dystonia, and therefore organic disorders. In contrast, the pathophysiology of fixed dystonia related to complex regional pain syndrome remained controversial. Knowledge of this history, which played on the border between neurology and psychiatry, is instructive and reflects the difficulty in discriminating between them. Today, new insights from functional imaging and neurophysiological studies again challenge the interpretation of these disorders, while the border between psychogenic and organic has become more blurred. Abnormalities of sensorimotor integration and cortical excitability that are currently supposed to be the underlying cause of dystonia bring us back to Sherringtonian physiology. We suggest that this may lead to a common explanation of the four afflictions of which we have traced the history.

Keywords: dystonia; psychogenic; history of medicine; history of neurology

Abbreviations: CRPS = complex regional pain syndrome
Introduction

For many years, physicians have observed and discussed the remarkable signs of what we nowadays call dystonia. The introduction of the term dystonia as an abnormality of tone with coexistent hypo- and hyper-tonia goes back to 1911, when the well-known Berlin neurologist Hermann Oppenheim (1858–1919) introduced *dystonia musculorum deformans*, which was later renamed early-onset generalized torsion dystonia (Oppenheim, 1911). In 1967, Wolfgang Zeman (1921–2001) and Paul Dyken reported the presence of milder forms of dystonia in dystonia musculorum deformans families, including cases of isolated writer’s cramp (Zeman and Dyken, 1967). In 1976, David Marsden (1938–1998) proposed the term focal dystonia for blepharospasm, oromandibular dystonia, dystonic writer’s cramp and torticollis, as well as for axial dystonias, arguing that these were closely related to generalized dystonia (Marsden, 1976). Up to the present, this view has not changed. Over the years, however, there has been discussion on whether the aetiology of dystonia is either organic or psychogenic. In this paper we study the evolution of ideas with respect to dystonia, in particular whether or not it was considered an organic or psychogenic affliction. We will put the historical evolution against the background of present-day knowledge resulting from functional imaging and neurophysiological studies, and of the blurred border between organic and psychogenic.

Methods

We started our search on dystonia history using two standard books on the history of medicine (McHenry, 1969; Norman, 1991). Furthermore, we used the PubMed database by entering the term ‘dystonia’ with limitation to ‘history of medicine’. We also used medical and neurological textbooks from the 19th and 20th century written in English, French, German or Dutch (Trousseau, 1882; Gowers, 1888; Oppenheim, 1894; Lewandowsky, 1914; Bouman and Brouwer, 1930; Bumke and Foerster, 1935, 1936; Kinnier Wilson, 1940; Biemond, 1946; Vinken and Bruyn, 1968, 1970). In the tables of contents and subject indexes, we searched for dystonia, spasms(s), spasmodic contortion or contraction, torticollis, wryneck, (writer’s) cramp, scrivener’s palsy, occupational neurosis (English); dystonie, torticolis (mental), spasme clonique (du sterno-mastoïdien), spasm fonctionnel (du sterno-mastoïden), cramp fonctionnelle, crispes des écervains (French); Dystonie, Torticollis, Schreibkrampf, Funktionskrämpfe, Beschäftigungsnervose (German); dystonie, torticolis, (ver)krampe(ing) and schrijverskramp (Dutch). In addition, we searched for relevant literature in the reference lists of consulted books and papers. As many 19th and early 20th century primary textbooks refer to the work of Duchenne and Bell, we chose to discuss their descriptions in more detail. When dealing with the question whether a particular author considered a disorder psychogenic or organic, we assumed a spectrum from organic to psychogenic between which ideas of the individual authors could be placed, as far as could be derived from the text.

Definition of dystonia

The word *dystonia* was introduced in 1911 (Oppenheim, 1911). Later its meaning was changed several times. For example, Derek Denny-Brown (1901–1981) considered dystonia to be a disorder with a fixed posture or oscillation between two or more fixed postures (Denny-Brown, 1965, 1966). The modern definition is ‘a syndrome of sustained muscle contractions, frequently causing twisting and repetitive movements, or abnormal postures’ (Fahn, 1988). In this paper, we use the latter definition.

Definitions of neurosis and hysteria

In the late 18th and early 19th century, neurosis was defined as the category of clinically well-characterized nervous diseases without known pathological substrates (López Piñero, 1983; Goetz, 2006). Throughout the 19th century, this category became smaller when neuropathological substrates of several of these diseases were established (Bynum, 1985). Hystera was a subcategory within the neuroses, in which neurological signs were similar to those in patients who suffered from nervous diseases with known anatomic lesions, although somewhat different and usually more extensive. In the late 19th century, Charcot assumed that hysteria arose from a lesion of an undetermined structural or functional nature and he expected that the pathological basis would be found in due course. The neurological defect was believed to result from a combination of hereditary predisposition and an environmental, provocative factor, which usually was a physical or emotional shock (Micale, 1995). Therefore, throughout history the term psychogenic cannot always be considered equal to non-organic, in particular in the pre-Freudian period. After this period, non-organic mostly did mean psychogenic. The meanings of neurosis and hysteria changed and finally the terms were used solely in descriptions of psychiatric diseases. At present, the terms are used less often, and are no longer listed in the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2000). In this paper, where needed, we clarify the context of these words.

Results

Primary generalized dystonia

In 1871, William Hammond (1828–1900) reported on three patients ‘in which the most characteristic symptoms are an inability to retain the fingers and toes in any position in which they may be placed, and their continual motion’ (1871). He mentioned the acquired disorder ‘athetosis’ and hypothesized on a striatal lesion. Hammond’s patients may not be considered dystonic patients—although today most authors consider athetosis as part of the dystonia spectrum (Morris et al., 2002)—but it is important to mention Hammond’s coining of the term athetosis. In 1897, the Spanish physician Lluis Barraquer I Roviralta (1855–1928) reported another patient with athetosis, which later was considered the first description of generalized torsion dystonia (Barraquer, 1897; Barraquer-Bordas and Gimenez-Roldan, 1988). In 1908, the German Marcus Walter Schwalbe (1883–1927) described hysterical symptoms in the siblings Fanny, Heimann and Wulf Levin, suffering from tonic cramps, which is now recognized as early-onset generalized torsion dystonia (Figure 1) (Schwalbe, 1908; Truong and Fahn, 1988). Among the most important hysterical characteristics, there was the presence of pressure points [called ‘hysterogenic zones’ in Charcot’s work...
Dystonia was organized. In the preface of the conference book, Eldridge and Fahn wrote:

“In the past, many victims of dystonia and their families have been caused anguish and hardship over and above that caused by the disease itself owing to the frequent misdiagnosis of the symptoms as manifestations of a psychiatric ailment. We hope that the present volume will facilitate accurate diagnosis, assist practicing physicians in treating their dystonic patients, encourage them to report their observations and results, and stimulate clinical and basic research workers in efforts to elucidate the causes and eventual treatment of dystonia and related disorders. (Eldridge and Fahn, 1976).

At this symposium, Marsden emphasized the existence of sporadic torsion dystonia (Marsden et al., 1976). Fahn and Eldridge stated that psychologically based dystonia was a rare or non-existent condition (Fahn and Eldridge, 1976). However, 3 years after the symposium (1978), the ‘first case of psychogenic dystonia’ was reported (Lesser and Fahn, 1978) and in 1983, at the 35th annual meeting of the American Academy of Neurology, another five followed (Fahn et al., 1983). The first patient was a 15-year-old girl who had simulated her dystonic symptoms and signs. She was admitted after a failed suicide attempt and told that she had faked her symptoms: ‘she discarded her leg brace, and the sustained contractions in her leg and arm immediately improved’. The histories of the other five patients were not included in the publication.

In 1984, an ad hoc committee, consisting of members of the Scientific Advisory Board of the Dystonia Medical Research Foundation, re-defined dystonia as ‘a syndrome of sustained muscle contractions, frequently causing twisting and repetitive movements, or abnormal postures’ (Fahn, 1988). Four years later a classification for psychogenic dystonia followed (Table 1) (Fahn and Williams, 1988). The first locus (9q32–34 region) for idiopathic dystonia (DYT1) was found in 1989 (Ozelius et al., 1989) and 10 years later the same group identified the gene, describing a unique 3 base pair deletion in the coding region, which was responsible for almost all their cases with early-onset, but for only a few with late-onset idiopathic torsion dystonia (Ozelius et al., 1997).

Cervical dystonia

One of the earliest descriptions of cervical dystonia was given by the Swiss physician Felix Platerus, also known as Plater (1536–1614) (Steyerthal, 1906; Platter, 1963). He described a case of ‘spasmi species, in qua caput in sinistrum latus torquebatur’ [a kind of spasm in which the head was turned to the left side]. The Dutch Nicolaas Tulp, or Tulpius (1593–1674), well known from the famous Rembrandt painting The Anatomy Lesson of 1632, described dissection of the sternocleidomastoid muscle as a therapy for what he called ‘obstipi capitis’ [crooked head] in his Observationes medicae (Tulp, 1641; Tulp and von Wolzogen, 1740). However, this patient had had torticollis from childhood and the origin was probably mechanical.

The well-known Scottish surgeon-anatomist Charles Bell (1774–1842) stated that the origin of ‘spasmodic contortion of
Hungarian Jeno Kollarits (1870–1940) reported on six ‘torticollis head, later named the ‘geste antagoniste efficace’ (Meige and to correct the powerful muscle activity by simply touching the genic, which was evident from the fact that the patient was able mental’ (Brissaud, 1895). In his view, the condition was psycho-

During one of his well-known Tuesday Lessons, on 26 June 1888, Charcot presented a 63-year-old man with ‘spasme fonctionnel du sterno-mastoı ¨dien et du trape `ze’ [clonic spasm of the sternocleidomastoid and the trapezius muscles], which had been present for 8 months (Charcot, 1887). The disorder started after the patient, who was a stockbroker, had lost all his money. On 10 July 1888, he was presented again after being successfully treated with electricity. Five years later, one of Charcot’s former students, Edouard Brissaud (1852–1909), introduced the term ‘torticollis mental’ (Brissaud, 1895). In his view, the condition was psycho-

The dystonia is consistent over time or is incongruent with classical dystonia, plus at least one of the following features: – other neurological signs are present that are definitely psychogenic, e.g. false weakness, false sensory findings and self-inflicted injuries – multiple somatizations are present – an obvious psychiatric disturbance is present. In 1713 the Italian physician Bernardino Ramazzini (1633–1714) recognized intense fatigue of the hand and arm, which resulted in failure of power, as an occupational disorder in professional writers (Ramazzini, 1964). In 1844 (published posthumously), Bell most
probably described writer’s cramp when he reported on an ambiguous condition in which writing had become impossible while the arm strength remained normal (Bell, 1844). In 1861 Duchenne reported on ‘cramp des écrivains’ in which electricity was not a very successful therapy. However, he advised an ingenious prosthesis (Duchenne, 1861). He preferred the names ‘spasme fonctionnel’ and ‘paralysie musculaire fonctionnelle’ because the disorder was not restricted to cramps and could be provoked not only by writing but also by other manual actions.

En résumé, les faits et les considérations exposés précédemment démontrent, comme je l’ai dit au commencement de cette note, qu’il existe une maladie caractérisée par un spasme douloureux ou indolent (contracture, contractions clonicques, tremblements), ou par une paralysie musculaire; que ces troubles se manifestent seulement pendant l’exercice de certains mouvements volontaires ou instinctifs; enfin, qu’ils peuvent s’engager dans des régions fort diverses. (Duchenne, 1861)

[In summary, the former findings and considerations show, as I described in the beginning of this report, that there is a disease which is characterised by painful or painless spasms (contracture, jerks, tremor) or paralysis in which the signs only occur during certain (in-)voluntary actions; the involved body parts are diverse.]

Similar to the situation in cervical dystonia, the debate on aetiology started in the early 20th century. In 1914 the German Fritz Mohr (1874–1957) summarized the two conflicting theories in Lewandowsky’s Handbuch der Neurologie (Lewandowsky, 1914). Writer’s cramp was explained by some authors as a purely organic disorder, e.g. as a reflex cramp through motor nerves that was initiated by painful sensory input. The German physician Moritz Romberg (1795–1873) was mentioned as one of the early advocates [with reference to the 1853 edition of Rombergs Lehrbuch der Nervenkrankheiten des Menschen (Romberg, 1853)]. Others, including Mohr himself, believed that only people with certain personality characteristics were prone to develop the disorder, a psychological factor possibly being involved. From that view, accurate psychoanalysis would be the best therapy for patients with writer’s cramp. Kinnier Wilson’s (1878–1937) 1940 edition of Neurology described ‘writers’ cramp’ as an occupational neurosis, physiologically akin to hysteria, and assumed a cortical dysfunction (Kinnier Wilson, 1940).

Prevention by excluding people prone to develop ‘spasms’ from certain occupations was considered the best treatment.

As in other focal dystonias, Marsden advocated the organic nature of writer’s cramp (vide supra) (Marsden, 1976), which he and Sheehy further demonstrated in a report on 29 patients (Sheehy and Marsden, 1982). However, in 1983, Cottraux et al. reported on the success of behavioural therapy and biofeedback in 9 of 15 patients with writer’s cramp (Cottraux et al., 1983), and the 1985 edition of John Walton’s Brain’s Diseases of the Nervous System stated:

I find the conclusions of Sheehy and Marsden inherently implausible and unacceptable. In my experience even subtle physical signs are absent in the many simple cases that I have seen and neither focal dystonia nor any other organic disorder could in my view impair movements only when they take part in one co-ordinated act while leaving totally unaffected all other precise and complex voluntary actions involving the affected member. (Walton, 1985).

The 1993 edition stated that writer’s cramp had ‘in the past been attributed to psychological factors, but there is now good evidence that this is not so’. Interestingly, the author referred to the same single publication of Sheehy and Marsden (Sheehy and Marsden, 1982; Harding, 1993). In 1991 Rivest et al., reported for the first time on the use of botulinum toxin for writer’s cramp (Rivest et al., 1991), which is currently considered the most effective treatment.

**Fixed dystonia related to complex regional pain syndrome**

In 1864, Silas Weir Mitchell (1829–1914) described a series of American Civil War (1861–1865) victims with gunshot wounds who developed burning pain and a shiny red skin after nerve injury (Mitchel et al., 1864; Koehler and Lanska, 2004). He suspected that traumatic nerve irritation was the cause and named the condition ‘causalgia’. He recognized that patients might come into an unendurably painful hyperaesthetic state. In 1892, Charcot demonstrated another entity in two patients: ‘œdème bleu des hystériques’, a painful condition with oedema and blue discolouration of the skin, which may occur in combination with an hysterical limb contracture or paralysis (Charcot, 1892).

L’historique de cette affection n’est pas bien long. Je l’ai pour la première fois mentionnée et distinguée à propos d’un malade de ce hospice [with reference to the Leçons du Mardi from 1889], que je suis d’ailleurs à même de vous présenter de nouveau. Puis, à plusieurs reprises je l’ai observée chez des personnes de la ville, combinée tantôt avec des altérations de la sensibilité (anesthésie ou hyperesthésie), tantôt avec des troubles du mouvement (paralysies et contractures). Il s’agissait presque toujours de sujets marqués, par la présence des stigmates, au sceau de l’hystérie la mieux caractérisée. (Charcot, 1892) [This disorder has a short history. For the first time [in 1889], I reported on a patient from this hospital [Hospice de la Salpêtrière]. From then, I recognised more cases. In a number of them, I observed sensory abnormalities (anaesthesia or hyperesthesia) or movement disturbances (paralysies and contractures). Mostly, patients were extraordinary persons having characteristics which may be considered hysterical.]

In 1946, Evans renamed the latter disorder ‘reflex sympathetic dystrophy’ because he suspected involvement of spinal reflexes as well as sympathetic efferent fibres (Evans, 1946). It was different from causalgia in that it occurred in the absence of major nerve trauma. However, in 1994, the International Association for the Study of Pain introduced the name complex regional pain syndrome (CRPS) for both conditions: type 1 (reflex sympathetic dystrophy) and type 2 (causalgia) (Merskey and Bogduk, 1994). The diagnostic criteria for CRPS type 1 were: (i) the presence of an
initiating noxious event, or a cause of immobilization (not obligatory item); (ii) continuing pain, allodynia or hyperalgesia with which the pain is disproportionate to any inciting event; (iii) evidence at some time of oedema, changes in skin blood flow or abnormal sudomotor activity in the region of the pain; and (iv) no other condition that would account for the degree of pain and dysfunction. CRPS type 2 has the same characteristics, but is accompanied by nerve injury (Merskey and Bogduk, 1994).

In 1984, Marsden et al. reported on four ‘reflex sympathetic dystrophy’ patients who had dystonia, characterized by fixed, predominantly flexion, postures (Marsden et al., 1984). They believed it to be ‘a distinct clinical syndrome’. Six years later Schwartzman et al., reported on motor disturbances in 43 patients with ‘reflex sympathetic dystrophy’, in whom the most dramatic characteristic was a dystonic posture in all patients (Schwartzman and Kerrigan, 1990). The authors hypothesized on a spinal cause. In 1993, a series of 18 patients with similar characteristics was reported (Bhatia et al., 1993). However, it was remarkable that many patients met the criteria for psychogenic dystonia from 1988 (vide supra) (Table 1). They concluded that the aetiology of this disorder, psychogenic or organic, was unknown (Bhatia et al., 1993). In 2004 it was reported that many patients with features of CRPS and dystonia also had features of psychogenic dystonia (Schrag et al., 2004). In the same year it was stated that a very large proportion had a primary psychogenic disorder (Sa et al., 2004).

In a more recent paper on 110 CRPS type 1 patients with dystonia predominantly characterized by tonic flexion postures, the authors hypothesized that maladaptive plasticity with disinhibition of spinal mechanisms might be the cause (van Rijn et al., 2007).

**Discussion**

There has been a continuous vacillation between psychogenic and organic explanations for (i) primary generalized dystonia, (ii) cervical dystonia, (iii) writer’s cramp and (iv) CRPS-related fixed dystonia. Although at first sight the attributions of the terms psychogenic and organic in Table 2 seem quite obvious, it seems more realistic to assume a spectrum with two ends between which attributions were moving. Moreover, the discussion between an organic and psychogenic aetiology has not always been explicit (particularly in the 19th century). The opinions of several authors could only be derived or interpreted from their hypotheses on aetiology and their therapies.

An example is Schwabe’s description of hysterical symptoms in siblings with generalized dystonia. In the late 19th century, Charcot considered hysteria a neurosis, similar to paralysis agitans, epilepsy and chorea, which were diseases without known pathology. For paralysis agitans he expected that the lesion would be discovered (Goetz et al., 1995). Hysteria appeared as a more difficult obstacle for Charcot’s clinical-anatomic method and, when describing male traumatic neurosis, he moved towards a psychological conception of hysteria (Goetz et al., 1995). This was further elaborated by Freud and his followers (Koehler, 2003). Hysteria evolved from a disease in which an organic pathophysiology was suspected but not found, to a psychogenic disease in the late 19th and early 20th century. Recent functional imaging studies in these patients have shown specific cerebral abnormalities (Vuilleumier, 2001, 2005). From these studies it is suspected that affective or stress-related factors modulate cerebral sensorimotor representations through interactions between limbic and sensorimotor networks. It is hypothesized that primitive reflexive mechanisms of protection and alertness, which are partly independent of conscious control, are involved.

**Primary generalized dystonia**

The patients of Oppenheim made him move to the organic end of the spectrum, whereas Freud and his followers in psychoanalysis, moved in an opposite direction. The improved description of the

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**Figure 1** Developments on generalized dystonia in the 20th century. See the text for references.

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\[\text{Not for the first time but more convincing than ever before}\]
<table>
<thead>
<tr>
<th>Year</th>
<th>Author (City, Year)</th>
<th>City</th>
<th>Terminology</th>
<th>O/P</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1614</td>
<td>Platter (Steyerthal, 1906; Platter, 1963)</td>
<td>Basel (CH)</td>
<td>Spasmi species, in qua caput in sinistrum latus torquebatur</td>
<td>O</td>
<td>Case report; explained as a disorder of the muscle and surrounding tissue</td>
</tr>
<tr>
<td>1641</td>
<td>Tulp (Tulp, 1641; Tulp and von Wolzogen, 1740)</td>
<td>Amsterdam (NL)</td>
<td>Obstipi captis</td>
<td>O</td>
<td>Probably mechanic origin; dissected the involved muscle</td>
</tr>
<tr>
<td>1765</td>
<td>Lorry (Lorry, 1765; Steyerthal, 1906)</td>
<td>Paris (FR)</td>
<td>Colli singularem omnino distortionem</td>
<td>P</td>
<td>Case report in a monography on melancholia; explained as due to boredom and therefore aversion to life</td>
</tr>
<tr>
<td>1768</td>
<td>Boissier de Sauvages (1768, 1771)</td>
<td>Montpellier (FR)</td>
<td>Obstiptas spasmodica</td>
<td>O</td>
<td>Classified as partial tonic spasms, together with strabismus, tics, contractures, ankylosis, cramps and priapism</td>
</tr>
<tr>
<td>1822</td>
<td>Dupuytren (1839)</td>
<td>Paris (FR)</td>
<td>Torticolis, caput obstipum</td>
<td>O</td>
<td>Divided the sternocleidomastoid muscle</td>
</tr>
<tr>
<td>1825</td>
<td>Middlesex Hospital (Anonymous, 1825)</td>
<td>London (UK)</td>
<td>Spasmodic affection of the muscles of the neck</td>
<td>U</td>
<td>Case report</td>
</tr>
<tr>
<td>1825</td>
<td>Gilby (1825)</td>
<td>Bristol (UK)</td>
<td>Contraction of the muscles of the neck</td>
<td>O?</td>
<td>Efficaciously used electricity in the corresponding contralateral muscles</td>
</tr>
<tr>
<td>1838</td>
<td>Stromeyer (1838)</td>
<td>Hannover (DE)</td>
<td>Krampf des Kopfnickers</td>
<td>O</td>
<td>Dissected the involved muscle(s)</td>
</tr>
<tr>
<td>1844</td>
<td>Bell (1844)</td>
<td>Edinburgh (UK)</td>
<td>Spasmodic contortion of the head and neck</td>
<td>O</td>
<td>Suspected a diseased nerve</td>
</tr>
<tr>
<td>1846</td>
<td>Romberg (1846)</td>
<td>Berlin (DE)</td>
<td>Halsmuskelkrampf</td>
<td>U</td>
<td>In most cases unknown cause, sometimes due to physical strain; described that some think that it may be due to intense emotions</td>
</tr>
<tr>
<td>1861</td>
<td>Duchenne (1861)</td>
<td>Paris (FR)</td>
<td>Spasme du sternomastoïdien</td>
<td>O</td>
<td>May be cured by continuous stretch to the antagonists by means of an apparatus; no success with electricity</td>
</tr>
<tr>
<td>1867</td>
<td>Middlesex Hospital (Anonymous, 1867)</td>
<td>London (UK)</td>
<td>Spasmodic contraction of cervical muscles</td>
<td>O?</td>
<td>Case report; spinal accessory nerve was dissected, although without efficacy</td>
</tr>
<tr>
<td>1872</td>
<td>Jaccoud (1873)</td>
<td>Paris (FR)</td>
<td>Hyperkénise de l’accessoire de Willis</td>
<td>O</td>
<td>Clonic form: rare, unknown cause; tonic form: either congenital, vertebral disorder or due to pressure on sensible nerve (reflex cramp)</td>
</tr>
<tr>
<td>1873</td>
<td>Charing Cross Hospital (Poore, 1873)</td>
<td>London (UK)</td>
<td>Clonic torticolis</td>
<td>O</td>
<td>Case report; improvement by electricity together with rhythmical exercise</td>
</tr>
<tr>
<td>1888</td>
<td>Charcot (1887)</td>
<td>Paris (FR)</td>
<td>Spasme clonique du sternomastoïdien et du trapeze</td>
<td>O ≈ P</td>
<td>‘Not-organic’ variant (= hysterical = partly moral, partly physical) tends to spread from the neck to the trunk</td>
</tr>
<tr>
<td>1888</td>
<td>Gowers (1888)</td>
<td>London (UK)</td>
<td>Spasmodic wry-neck</td>
<td>O + P</td>
<td>Case report; improvement with electricity to the atrophied contralateral muscle</td>
</tr>
<tr>
<td>1889</td>
<td>Freud (Breuer and Freud, 1909)</td>
<td>Vienna (AT)</td>
<td>Genickkrämpfe</td>
<td>O</td>
<td>Case report (Frau Emmy v. N); hysteria patient who underwent hypnosis; Genickkrämpfe were not considered hysterical</td>
</tr>
<tr>
<td>1890</td>
<td>Keen (Anonymous, 1890; Keen, 1891)</td>
<td>Philadelphia (US)</td>
<td>Spasmodic wry neck</td>
<td>U</td>
<td>Divided the dorsal rami of the C1-C3 spinal nerves in a patient in whom spinal accessory nerve division was unsuccessful; some improvement</td>
</tr>
<tr>
<td>1893</td>
<td>Brissaud (1895)</td>
<td>Paris (FR)</td>
<td>Torticolis mental</td>
<td>P</td>
<td>Believed that torticolis was a tic (P), not a spasm (O); the touch that was able to correct proved the psychic nature</td>
</tr>
<tr>
<td>1894</td>
<td>Voisin (1894)</td>
<td>Paris (FR)</td>
<td>Torticolis intermittant</td>
<td>P</td>
<td>Case report; cured with suggestion during hypnosis</td>
</tr>
<tr>
<td>1894</td>
<td>Oppenheim (1894)</td>
<td>Berlin (DE)</td>
<td>Krämpfe im Bereich der Halsmuskeln</td>
<td>O ≈ P</td>
<td>Hereditary or congenital instability of kinetic centres in the cerebral cortex</td>
</tr>
</tbody>
</table>

(continued)
<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>City</th>
<th>Terminology</th>
<th>O/P</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1896</td>
<td>de Quervain (1896)</td>
<td>La-Chaux-de-Fonds (CH)</td>
<td>Torticolis spasmodique</td>
<td>O?</td>
<td>Efficaciously dissected. Involved muscles and nerves (method from Kocher); treatment effect might be due to suggestion of a cortical centre.</td>
</tr>
<tr>
<td>1900</td>
<td>Babinski (1900)</td>
<td>Paris (FR)</td>
<td>Torticolis spasmodique</td>
<td>O</td>
<td>Case report with extensor toe response.</td>
</tr>
<tr>
<td>1902</td>
<td>Meige and Feindel (1902)</td>
<td>Paris (FR)</td>
<td>Torticolis mental</td>
<td>P</td>
<td>The 'geste antagoniste efficace' is characteristic; careful and prolonged observation is needed to distinguish it from 'torticolis-spasme' (O).</td>
</tr>
<tr>
<td>1905</td>
<td>Kollarits (1905, 1908)</td>
<td>Budapest (HU)</td>
<td>Torticolis mental</td>
<td>O</td>
<td>Geste antagoniste was named 'Brissauds Handgriff'.</td>
</tr>
<tr>
<td>1907</td>
<td>Curschmann (1907)</td>
<td>Mainz (DE)</td>
<td>Spasmodischen Torticollis</td>
<td>O</td>
<td>Vestibular disorder; quinine was efficacious.</td>
</tr>
<tr>
<td>1914</td>
<td>Mohr (Lewandowsky, 1914)</td>
<td>Koblenz (DE)</td>
<td>Torticolis mental</td>
<td>O &lt; P</td>
<td>Often in neuropathic patients; organic causes must be excluded (ocular, auricular; cervical spine, or brain disease).</td>
</tr>
<tr>
<td>1914</td>
<td>New York Neurological Society (Clark, 1914)</td>
<td>New York (US)</td>
<td>Mental torticollis</td>
<td>P</td>
<td>Clark reported on the efficacy of psychotherapy; was criticized by others.</td>
</tr>
<tr>
<td>1923</td>
<td>Wartenberg (1923)</td>
<td>Freiburg im Breisgau (DE)</td>
<td>Torticollis</td>
<td>O &gt; P</td>
<td>Pathophysiological description on the influence of sensible input in extrapyramidal disorders (including the geste antagoniste).</td>
</tr>
<tr>
<td>1923</td>
<td>Cushing (McKenzie, 1924)</td>
<td>Boston (US)</td>
<td>Spasmodic torticollis</td>
<td>U</td>
<td>Performed surgery with unilateral division of the spinal accessory nerve and ventral and dorsal 1st to 3rd roots, with success.</td>
</tr>
<tr>
<td>1935</td>
<td>Yaskin (1935)</td>
<td>Pennsylvania (US)</td>
<td>Spasmodic torticollis</td>
<td>O &lt; P</td>
<td>Distinguished: psychogenic; postencephalitic; associated with an extrapyramidal disease; and progressive spasm of doubtful nature.</td>
</tr>
<tr>
<td>1938</td>
<td>Critchley (Anonymous, 1938) at the Annual Meeting of the British Medical Association</td>
<td>London (UK)</td>
<td>Spasmodic torticollis</td>
<td>O or P</td>
<td>Distinguished: neuralgic, occupational (P), spasmodic, paralytic, hysterical (P) and congenital torticollis and torticollis tic (P).</td>
</tr>
<tr>
<td>1940</td>
<td>Kinnier Wilson (1940)</td>
<td>London (UK)</td>
<td>Torticollis</td>
<td>O or P</td>
<td>103 cases; promoted surgery.</td>
</tr>
<tr>
<td>1943</td>
<td>Patterson and Little (1943)</td>
<td>Ann Arbor (US)</td>
<td>Spasmodic torticollis</td>
<td>O &gt;&gt; P</td>
<td>21 cases; psychotherapy is the treatment of choice.</td>
</tr>
<tr>
<td>1945</td>
<td>Paterson (1945)</td>
<td>Edinburgh (UK)</td>
<td>Spasmodic torticollis</td>
<td>O or P</td>
<td>43 cases; though organic in nature, the clinical picture may be influenced by psychogenic factors.</td>
</tr>
<tr>
<td>1949</td>
<td>Herz and Glaser (1949)</td>
<td>New York (US)</td>
<td>Spasmodic torticollis</td>
<td>O</td>
<td>Performed experiments in monkeys; described that torticollis arises from damage to the pretectal region, and is due to distortion of optokinetic reflexes.</td>
</tr>
<tr>
<td>1967</td>
<td>Brierley (1967)</td>
<td>Newcastle-upon-Tyne (UK)</td>
<td>Spasmodic torticollis</td>
<td>O or P</td>
<td>Psychoanalytical treatments in 60 patients; &gt;5000h; severe ego-regression in all cases.</td>
</tr>
<tr>
<td>1971</td>
<td>Mitscherlich (1971)</td>
<td>Düsseldorf (DE)</td>
<td>Spasmodic torticollis</td>
<td>P</td>
<td>Improvement with sensory feedback therapy (13 cases); also improvement in spasticity patients.</td>
</tr>
<tr>
<td>1985</td>
<td>Tsui et al. (1985)</td>
<td>Vancouver (CA)</td>
<td>Spasmodic torticollis</td>
<td>O</td>
<td>Stated that in some cases psychotherapy is indicated.</td>
</tr>
</tbody>
</table>

O = organic; P = psychogenic; O > P = majority of cases is organic; O or P = cause is organic in some cases and psychogenic in other cases; O + P = cause is a combination of organic and psychogenic factors in every case; U = unknown cause.
‘clinical entity’ in the 1940s (Herz), the new hereditary cases described in the 1950s (Zeman) and the limited efficacy of psychotherapy in torsion dystonia, as well as the effects of surgical treatments and the lesion studies in the 1960s (Eldridge, Cooper and Denny-Brown, respectively) pushed the explanatory ideas back into the organic. This culminated in Eldridge and Fahn’s 1975 statement (published in 1976). However, a new movement towards psychogenesis soon followed with the recognition of psychogenic dystonia. Meanwhile, the remaining dystonias kept their position on the organic side of the spectrum, not in the least because of the discovery of the DYT1 gene. Nevertheless, one cannot be too rigid because dystonic disorders with a genetic origin can be triggered by emotional stress (Breakefield et al., 2008).

Cervical dystonia

Bell and Duchenne probably assumed an organic cause for cervical dystonia (Table 2). Not much later Charcot, and certainly his student Brissaud, moved to the psychogenic view, in which the interpretation of observing the ‘geste antagoniste’ played an important role. At the time, such terms as ‘torticollis mental’ and ‘torticollis hystericus’ were used on both sides of the Atlantic and dealt with likewise. Psychological and surgical treatments were applied simultaneously in different patients at different places around the 1940s. After Marsden’s 1975 presentation, the aetiological ideas on cervical dystonia clearly moved away from the psychogenic to the organic side of the spectrum.

Writer’s cramp

To explain writer’s cramp, Ramazzini used such terms as ‘fatigue’ and ‘failure of power’. These should be interpreted in the humoral pathophysiological concepts of the time, i.e. animal spirits that flow through the nerves with less power than usual. One would be inclined to consider an organic aetiology here; however, we may question whether Ramazzini was concerned with this question at all. From Bell’s description a century later and also from Romberg’s work, an organic viewpoint may be recognized. Duchenne again used the term ‘functional’, which does not necessarily imply that he meant a psychogenic aetiology. A clearer distinction came about in the early 20th century, when Mohr mentioned personality characteristics and a psychological factor, and suggested psychoanalysis for treatment. An interesting position was taken by Kinnier Wilson, assuming cortical dysfunction but comparing it to hysteria. Once more, Marsden’s 1975 presentation pushed the aetiology of writer’s cramp towards the organic side, with a few exceptions in the 1980s.

Fixed dystonia related to complex regional pain syndrome

Charcot’s demonstration of two patients with ‘œdème bleu des hystériques’ occurred in a period in which he was moving towards a psychological explanation of hysteria. Marsden et al. expressed the opinion that the similarities between CRPS cases with dystonia over the world suggested its existence as a distinct clinical syndrome (Marsden et al., 1984). In contrast, Sa et al. stressed that most cases satisfied the criteria for psychogenic dystonia, and should, therefore, be considered as such (Sa et al., 2004). But these are based on expert opinion. Such statements are not like a gold standard and should, therefore, be used with caution. Moreover, it is remarkable that the reasons why CRPS-related fixed dystonia is considered psychogenic are at least partly the same as the arguments that were used in the past to explain why focal dystonia was psychogenic (Table 3): (i) the dystonia in CRPS may be considered incongruent with classical dystonia; (ii) it may be inconsistent over time; (iii) weakness, described in the majority of CRPS cases, might be interpreted as false; (iv) sensory abnormalities, which fit the diagnosis of CRPS, might be interpreted as false sensory findings; and (v) sometimes, psychiatric abnormalities are present. In recent times significant motor cortex abnormalities were found in CRPS (Schwenkreis et al., 2003; Maihofner et al., 2007; Gieteling et al., 2008).

It is clear that the discussions on the psychogenic or organic aetiology of dystonia have been emotional. In some of the periods, particularly during the 20th century, strong believers as well as non-believers may be recognized. Charcot isolated hysterical disorders from other neurologic diseases. In his view, environmental factors (‘agents provocateurs’) were involved in its pathogenesis. The rise of the psychoanalytic movement, following the work of Freud at the beginning of the 20th century, caused important disagreements between supporters of organic and psychogenic explanations. This was not specific to the interpretation of dystonia, but more generally reflected the division between biologically and psychoanalytic oriented neuropsychiatrists at the time. The success and popularity of psychoanalysis, as well as the lack of an organic substrate for dystonia, encouraged psychogenic theories. As the 20th century proceeded, knowledge in favour of a somatic origin of early-onset generalized dystonia accumulated.

**Table 3 Seven reasons why focal dystonias were regarded as psychogenic (Marsden, 1976)**

<table>
<thead>
<tr>
<th>Reason</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. The bizarre nature of the dyskinesias</td>
<td></td>
</tr>
<tr>
<td>2. Their appearance frequently only on certain actions, other motor acts employing the same muscles being carried out normally</td>
<td></td>
</tr>
<tr>
<td>3. Their relief by certain inexplicable trick actions</td>
<td></td>
</tr>
<tr>
<td>4. Their exquisite sensitivity to social and mental stress</td>
<td></td>
</tr>
<tr>
<td>5. The failure so far to find any anatomical, physiological, or biochemical abnormality in any of these conditions</td>
<td></td>
</tr>
<tr>
<td>6. The belief that such patients show overt psychiatric disturbance</td>
<td></td>
</tr>
<tr>
<td>7. A psychopathological interpretation of the significance of, for example, eye closure or neck turning</td>
<td></td>
</tr>
</tbody>
</table>
Marsden, a leading neurologist in movement disorders, convinced the neurological community in the 1970s and 1980s that both generalized and focal dystonia were somatic entities. However, psychogenic dystonia re-emerged, but as a special category. Nowadays, psychogenic dystonia is thought to be ‘common’ in specialized movement disorders clinics (Espay et al., 2006).

The recognition of the hereditary character of dystonia played an important role in attributing an organic nature in the first as well as the last decades of the 20th century. If dystonia had existed as an entity and its hereditary character was recognized previously, it would probably have been interpreted in a different way, because of the particular concepts of the late 19th century. In this period several neuroses were considered hereditary, in fact a favourite subject in the interpretation models of Charcot (Féré, 1884). Similar to contemporary psychiatrists [the French Benedikt-Augustin Morel (1809–1873) and Valentin Magnan (1835–1916), he assumed that degeneration was a constitutional factor in certain families (‘neuropathic families’) in which neuroses including hysteria, alcoholism and epilepsy could be transformed during the passage from one generation to the next. Hysteria in a parent could be inherited as epilepsy in the child (Berrios and Beer, 1995; Goetz et al., 1995). In the 20th century, following delineation of dystonia as an entity and following new discoveries in genetics, the hereditary character led to new insights. Today it is recognized that >14 genes are implicated in different monogenic dystonia syndromes, which are frequently inherited as autosomal dominant conditions with reduced penetrance. Most cases of early-onset torsion dystonia are associated with the DYT1 gene mutation (Breakfield et al., 2008). Familial occurrence of cervical dystonia or writer’s cramp has been described but appears to be rare (Defazio et al., 2007).

Medical problems nearly always unravel because of the advent of a new technology, skill or understanding of a hitherto unknown system of disease. One example is the unravelling of the electric nature of nerve action in the 18th and 19th century. Ideas on whether or not animal electricity existed and played a role in the nature of nerve conduction were put forward at the end of the 18th century by Galvani and denied by Volta (Piccolino, 1998; Koehler et al., 2009). The confirmation had to await more sophisticated sensitive measuring devices such as the galvanometer invented by Du Bois-Reymond in the 1840s, after which observation of the action potential became possible.

What will be the future ‘sophisticated sensitive measuring device’ that will finally lead to the understanding of dystonia? We believe that the increasing knowledge resulting from neurophysiological and imaging studies, combined with genetic methods, will provide the insight that the explanation of dystonia cannot just be interpreted in terms of organic or psychogenic. These modern methods may show that the interaction of genetic and environmental factors is more complex than was previously thought. When reviewing the pathophysiology of primary adult-onset focal dystonia, Defazio et al. (2007) suggest that in human focal dystonia there may be an overload of a predisposed sensory system resulting from peripheral injury or repetitive motor activity in a certain part of the body, or both, causing sensory receptive changes in the corresponding cortical brain areas and leading to abnormal regulation of inhibitory interneuronal mechanisms at the brainstem or spinal cord level (Defazio et al., 2007). There seems to be an abnormality of sensorimotor integration and cortical excitability beyond the symptomatic body part. In both generalized and focal dystonia, neurophysiological and functional imaging studies indeed point towards abnormalities in the sensorimotor circuitry, which result in a vulnerable central nervous system. Some of these phenomena have been found in asymptomatic gene carriers, as well as in representations of unaffected body parts. It is suspected that a ‘second hit’ is needed to bring the central nervous system out of balance, which leads to dystonia (Defazio et al., 2007; Breakefield et al., 2008). Musician’s dystonia is an interesting example. In a transcranial magnetic stimulation study, cortical changes were found in musicians compared to healthy controls, and these changes were more marked in those with musician’s dystonia. It is hypothesized that musician’s dystonia is a form of training-induced dystonia (Rosenkranz et al., 2005).

These suggestions, in particular the assumption of abnormal regulation of inhibitory interneuronal mechanisms at the brainstem or spinal cord level, bring us back to Sherringtonian neurophysiology as already suggested by Denny-Brown in the 1960s, when he found that damage in different anatomical structures could have the same consequence; pointing to a basic neurophysiological principle, the final common path, that had been conceived around the turn of the 19th to the 20th century by his teacher Charles Scott Sherrington (1857–1952) (Sherrington, 1906; Burke, 2007). This may still be a valid explanation of the phenomenology, if not the underlying causation of dystonia in modern terms.

Today, psychogenic dystonia is considered a disorder that results from an underlying psychiatric illness. Its diagnostic criteria have remained unchanged for decades (Table 1). In the meantime, however, the border between neurology and psychiatry has been less well defined. For example, schizophrenia (Hendler et al., 2009), autism (Mostofsky et al., 2009) and primary dystonia (Breakefield et al., 2008) are now considered neurofunctional disorders. Additionally, it has been shown recently that patients with cervical dystonia or blepharospasm have distinct neuropsychiatric and personality profiles of the anxiety spectrum (Lencer et al., 2009). Another study shows high psychiatric comorbidity in cervical dystonia, which is unlikely to be a mere consequence of chronic disease and disfigurement (Gundel et al., 2003). It is attractive to see psychogenic disorders as the consequence of functional crashes in anatomically normal brains. In these disorders, abnormalities found in neurophysiological and functional imaging studies may be interpreted as signs of organic dysfunction. We have only traced two transcranial magnetic stimulation studies on psychogenic dystonia. Interestingly, one of these found similar abnormalities in both organic and psychogenic dystonia: reduced short and long-interval intracortical inhibition and cortical silent period, and an increased cutaneous silent period (Espay et al., 2006). The other study detected difference: patients with organic dystonia had an increased response to paired associative stimulation compared to patients with psychogenic dystonia (Quartarone et al., 2009). The authors of the latter study concluded that abnormal plasticity is a hallmark of organic dystonia in contrast to psychogenic dystonia.

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If we hypothesize further, assuming abnormal regulation of inhibitory interneuronal mechanisms as mentioned above, neurophysiological and functional imaging studies may help to explain dystonia in CRPS due to peripheral injury leading to similar sensory receptive changes. Such mechanisms may also be in play in dissociation disorders, including conversion disorder (Marshall et al., 1997; Vuilleumier, 2001, 2005; Cojan et al., 2009; Seritan et al., 2009), thereby associating primary dystonia, CRPS-related fixed dystonia, and sensory and motor disorders in conversion disorder.

**Conclusions**

Opinions on whether dystonia is either organic or psychogenic continuously changed on a spectrum between the two extremes over the described period. Genetic studies, the limited efficacy of psychotherapy, the effects of surgical treatments, lesion studies and the recognition that focal dystonias may be minor forms of generalized dystonia pushed the explanatory ideas in the direction of organic. We have seen how insights were influenced by contemporary general pathophysiological concepts (humoral pathophysiology in the pre-1800 period, solid pathophysiology reflected by the clinical-anatomical method thereafter, psychological pathophysiology after about 1900, and genetic and molecular pathophysiology in recent decades), as well as by various research methods, from which we have learnt to be prudent with the interpretation of results and to reflect on epistemological mechanisms. Nevertheless, with these reservations in mind, modern neurophysiological and imaging studies may open new ways for the interpretation of dystonia. In both generalized and focal dystonia, studies point towards abnormalities in the sensorimotor circuitry, resulting in a vulnerable central nervous system. They indicate that the old distinction between psychogenic and organic is not easily applicable and perhaps should be abandoned. Similar mechanisms may be in play in CRPS-related fixed dystonia and sensory and motor disorders in conversion disorder.

Hypotheses made on the basis of neurophysiological and functional imaging studies need further testing in these groups of patients. In addition, genetic studies may provide further insight. Until more knowledge is available, we must keep in mind the lessons from history and remember 1975:

In the past, many victims of dystonia and their families have been caused anguish and hardship over and above that caused by the disease itself owing to the frequent misdiagnosis of the symptoms as manifestations of a psychiatric ailment. (Eldridge and Fahn, 1976)

Once hurt, twice shy.

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