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MINIREVIEWS

Research progress in spasmodic torticollis rehabilitation treatment

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Abstract

Spasmodic torticollis (ST) is a focal dystonia that affects adults, causing limited muscle control and impacting daily activities and quality of life. The etiology and curative methods for ST remain unclear. Botulinum toxin is widely used as a firstline treatment, but long-term usage can result in reduced tolerance and adverse effects. Rehabilitation therapy, with its minimal side effects and low potential for harm, holds significant clinical value. This article explores the effectiveness of adjunctive therapies, including exercise therapy, transcranial magnetic stimulation, shockwave therapy, neuromuscular electrical stimulation, vibration therapy, electromyographic biofeedback, and acupuncture, in the treatment of ST. The aim is to provide clinicians with additional treatment options and to discuss the efficacy of rehabilitation therapy for ST.

Key Words: Spasmodic torticollis; Rehabilitation therapy; Botulinum toxin; Exercise therapy; Adjunctive therapy; Effectiveness

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Core Tip: Spasmodic torticollis (ST) is a focal dystonia characterized by involuntary contractions of the neck muscles, significantly impacting daily activities and quality of life. Botulinum toxin is a widely used first-line treatment for ST, but long-term use can lead to reduced efficacy and potential side effects. Surgical interventions may have associated complications. Rehabilitation therapy, including exercise therapy, has potential clinical value as a low-risk treatment option. It can be used as an adjunctive therapy for ST, showing efficacy in improving clinical outcomes and reducing tolerance to botulinum toxin.

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INTRODUCTION

Spasmodic torticollis (ST), a focal dystonia affecting adults, is typified by involuntary muscle contractions in the neck. Profoundly impacting daily activities and quality of life, the etiology of ST remains as yet undefined, and there is no affirmed cure[1-4]. Recurrent botulinum toxin injections are currently the most pervasive first-line therapeutic approach for ST, but long-term usage can foster lowered patient tolerance, thus causing the alleviating effects on muscle spasms to gradually diminish and potentially result in side effects. Surgical treatments are opted for when the efficacy of botulinum toxin and oral medications fall short, but these treatments may lead to associated complications[1-8]. Consequently, rehabilitation therapy, with its minor side effects and low risk, exhibits substantial clinical value. Rehabilitation approaches described in this article, including exercise therapy, transcranial magnetic stimulation, shockwave therapy, neuromuscular electrical stimulation, vibration therapy, electromyographic biofeedback, and acupuncture, are potentially advantageous as adjunctive therapies for ST. However, their efficacies are currently underexplored. The goal of this article is to elucidate the effectiveness of these specific rehabilitation treatment regimens for ST, thereby equipping clinicians with a broader spectrum of therapeutic options.

EXERCISE THERAPY

Exercise therapy, a significant component of physical therapy, employs specific movements or professional equipment for active and/or passive exercise training to regain normal bodily functions[9]. The management of ST may involve techniques for joint mobility, soft tissue stretching, strength exercises, aerobic training, and balance training. Exercise therapy is less frequently used in isolation for alleviating muscle spasms in ST patients, but it is often combined with botulinum toxin treatment. As an adjunctive therapy for ST, it has demonstrated beneficial efficacy and could offer a therapeutic direction for clinical practitioners[10-15], as shown in Table 1. Further studies have revealed that exercise therapy can reduce the dosage of subsequent botulinum toxin injections and maintain its therapeutic effect for a longer period[13,16]. In light of the established efficacy of botulinum toxin in treating ST patients, clinicians might also consider integrating exercise therapy to improve the clinical outcome and mitigate the problem of reduced tolerance resulting from long-term botulinum toxin injections.

TRANSCRANIAL MAGNETIC STIMULATION THERAPY

Transcranial magnetic stimulation (TMS) is a non-invasive, painless brain stimulation technique, capable not only of investigating the functional roles of specific brain regions by interfering with their neurons[17] but also of relieving muscle tone disorders by modulating the excitability and plasticity of pathogenic brain areas [18]. Increasing studies have pointed to a link between abnormal muscle tone and the cerebellum[19-21]. TMS can activate Purkinje cells in the posterior cerebellum (the VIII lobule and crus II). Such activation inhibits the dentate nucleus, which delivers excitatory responses through synaptic relays in the ventrolateral thalamus to the contralateral primary motor cortex (M1), resulting in suppression of the contralateral M1 due to a decrease in dentato-thalamo-cortical excitation - this form of cortical inhibition is referred to as cerebellar inhibition[22]. TMS can reduce muscle tone and alleviate spasms by reducing the excitability of primary motor cortex through stimulating the cerebellum[23]. Many studies have shown the efficacy of TMS in treating different spasms, as shown in Table 2[24-29]. An experiment comparing TMS and botulinum toxin therapy for the treatment of ST found the latter superior; however, it is worth noting that post-botulinum toxin injection, half of the patients experienced reversible neck weakness and swallowing difficulties, while no adverse reactions were apparent after TMS treatment, indicating good safety[30]. Yang et al[31] established that the use of TMS in patients with ST results in a shortened resting period for the orbicularis oris, orbicularis oculi, and sternocleidomastoid muscles compared to the normal control group. In addition, patients with torticollis exhibit a delayed onset of the resting period for the sternocleidomastoid muscle, this substantiates that in patients with localized muscular tone disorders like ST, there is a decline in the inhibitory functions of the motor cortex and the presence of generalization, thereby affirming the efficacy of TMS in treating ST. If a patient with ST responds poorly to botulinum toxin therapy or refuses this treatment, given the non-invasive nature of TMS, we should consider its broader application to ensure patient efficacy and improve their quality of life.

Table 1 Efficacy of Botox combined with exercise therapy in neck dystonia

Ref.	Groups	Specific content of intervention	Intervention time	Evaluation indicators	Research conclusion
Hu <i>et al</i> [10]	C: Botox group; E: Botox group + exercise therapy group	Stretching exercises, active range of motion, and isometric exercises of the neck muscles	15 min/time, 5 d/wk for 6 wk	a, b, c	The spasticity and pain symptoms improved significantly
Tassor et al [11]	C: Botox group; E: Botox group + exercise therapy group	Massage, stretch, educate posture, strengthen axial muscles	For 60-90 min/time, for 2 wk	c, d, e	Remission of spasticity symptoms was similar in both groups, and pain and daily living ability were significantly improved in group E
van den Dool <i>et al</i> [12]	C: Botox + conventional PT group E: Botox + standardized PT group	Muscle stretching, passive neck activity, and training that has been found to be related to neurorehabilitation and motor learning	Family exercise: 10-15 min/time, 5-10 times/d. PT sessions twice/wk and once/wk in later stages. One year	b, c, d, f, g, h, i	Both groups were effective for spasticity and pain. Group E reduced patients' dependence on healthcare providers and reduced medical costs for this patient group
Queiroz et al[13]	C: Botox group; E: Botox + exercise therapy group	Motor stretching, passive, and active cervical loosening of the cervical spine	25 min/time, 5 d/wk for 4 wk	b, c	Group E showed significant improvement in daily living capacity and subjective pain
Stankovic et al[14]	C: Exercise therapy group, E: Botox + exercise therapy group	Increase range of motion exercise, muscle stretching, occupational and functional therapy	5 d/wk for 2 wk	c, d	Group E showed significant improvement in pain, torticollis and disability scales
Guo <i>et al</i> [15]	C: Botox group, E: Botox + rehabil- itation treatment group	Muscle pulling and relaxation training, exercise relearning training, lateral flexion movement in each horizontal axis direction and vertical axis expansion movement in passive and active modes	757 min/time, 5 d/wk, for 4 wk	c, d	In group E, the scores of physiology, physiological function, physical pain, vitality and mental health were significantly improved compared with those before treatment

"E" represents the test group; "C" represents the control group. a: The Visual Analogue Scale; b: SF-36 Quality of Life Scale; c: Toronto Western Torticollis Rating Scale; d: Torticollis Assessment scale; e: Daily Performance Scale; f: Functional Disability Questionnaire; g: Overall Clinical Impression-Disease severity Scale; h: Overall Clinical Impression-Improvement Scale; i: Cranio-cervical Dystonia Questionnaire.

Table 2 Efficacy of rTMS therapy for dystonia

Ref.	Transcranial magnetic prescription	Evaluation method	Curative effect
Bradnam <i>et al</i> [24]	Five d/wk from Monday to Friday, 10 times	a, b	Cervical dystonia symptoms, pain and quality of life
Hao et al[25]	Frequency was 10 Hz, 30 min/time for 7 d	c, d, e, f	Significantly relieved muscle tension spasm and improved quality of life
Şan <i>et al</i> [26]	Frequency 5 Hz, 15 min/time, 10 for 2 wk	c, g, h, i	Spasticity and the frequency of the seizures improved significantly
Zhao et al[27]	Frequency 1 Hz, 1/d, 6/wk and continuous treatment for 4 wk	c, f, j	Improved limb spasm, restored limb movement function, and improved daily living ability
Cheng et al[28]	The stimulation frequency was 1 Hz, each sequence with 10 pulses at 2 s intervals for a total of 1200 pulses, 1/d, 5 times/wk, and 8 wk as 1 session	c, j, k	Improved the degree of muscle tone control, alleviated muscle spasm, and improved motor dysfunction
Yuan et al[29]	The stimulation frequency was 1 Hz with 120 stimulus trains of 2 s between 10 pulses for a total of 1200 pulses. Once/d for 5 d per wk for 8 wk	c, j, k	Effectively relieved the degree of spasm, improved the motor function and daily life activities

a: The Toronto Western Torticollis Rating Scale; b: Cranio-cervical Dystonia Questionnaire; c: Modified Ashworth Scale; d: Uniform Wilson's Disease Rating Scale; e: Dystonia Scale; f: Activities of daily living; g: Penn Spasticity Frequency Scale; h: Visual analogue scale; i: Quality of Life 54; j: Fugl-Meyer motor function rating; k: Modified Barthel quantity scale.

EXTRACORPOREAL SHOCK WAVE THERAPY

Extracorporeal Shock Wave Therapy (ESWT) is a series of high-energy single-pulse mechanical waves, notable for high pressure, fast rise, short action period, and three-dimensional propagation, the speed of which increases with pressure. It can be divided into focused ESWT (fESWT) and radial ESWT (rESWT)[32]. Although the mechanism of ESWT in spasmodic treatment is still unclear, it may be related to the following aspects: (1) ESWT induces the production of nitric oxide, which increases neovascularization of muscles and tendons, thereby improving muscle stiffness; (2) ESWT may reduce the excitability of motor neurons by vibration stimulation of the tendons, thereby reducing muscle tone; (3) ESWT can reduce the amount of acetylcholine at the neuromuscular junction, causing nerve conduction disorders; and (4) ESWT can cause alternating biochemical reactions between metabolism and proliferation, affecting muscle fibrosis and rheological properties[33]. Recent research has revealed that radial shock wave therapy can effectively reduce the degree of neck muscle spasm in patients with ST, maintain neck position activity, and enhance their daily living ability[34]. Currently, the mechanism of ESWT in the treatment of spasms has been posited from perspectives such as neuromuscular junction and muscle alterations; however, there is still a lack of concrete molecular biological evidence such as signal pathways and genes[35]. Although there is still no definite plan for precise positioning and energy level/density for ESWT, its effectiveness in treating spasms has been confirmed, as shown in Table 3[36-42]. This necessitates further clinical exploration to provide more precise treatment for patients.

NEUROMUSCULAR ELECTRICAL STIMULATION

Neuromuscular Electrical Stimulation (NMES) is a non-invasive treatment technique, which when applied to spastic muscles, causes them to contract violently. To prevent muscle damage, the Golgi sensory organs are stimulated; impulses enter the spinal cord, then *via* interneurons to the corresponding anterior horn cells, reflexively inducing inhibition of the spastic muscle itself, thereby relieving spasms[43]. Studies have shown that the treatment of ST patients with NMES is significantly effective, and its short-term effect is superior to local injection of type A botulinum toxin[44]. Although research on NMES treatment for ST is currently limited, it has achieved significant efficacy in treating post-stroke spasm and spastic cerebral palsy[45-48], providing more options for clinical spasm treatment.

VIBRATION THERAPY

Vibration is a type of sinusoidal mechanical oscillation characterized by amplitude, frequency, and phase angle and is categorized into whole-body vibration training (WBV) and focal muscle vibration (FMV) based on the area of effect [49]. During WBV, the patient is positioned on the vibration platform, and repetitive perturbations are allowed to be transferred, enabling modification of the vibration's frequency, amplitude, and direction (vertical displacement or a backand-forth vertical sinusoidal oscillation)[50]. Although the operating mode of this therapy remains unclear, it appears to display its function by stimulating muscle spindles and alpha motor neurons along with short-term metabolic activities [51]. FMV can activate muscle spindles, thereby stimulating the input of Ia fibers, altering the cortical spinal pathways [52]. It can lead to the inhibition of spinal reflexes, thereby suppressing antagonistic muscle neuron circuits, diminishing the excitability of antagonistic muscles, activating the functional muscle, and mitigating symptoms of spasms[53]. Vibration therapy studies for ST patients are limited, but it has maturely evolved in improving spasticity, particularly in patients with post-stroke spasms and cerebral palsy (Table 4)[54-58]. Studies have shown that the application of vibration to head and neck muscles in ST patients resulted in an immediate and significant reduction in involuntary muscle activity [59]. Recent research indicates that following vibration therapy in patients with right torticollis, the frequency and severity of the disturbances in tension posture significantly reduced, suggesting its effectiveness in spasm treatment [60]. In clinical practice, during the early stages of treatment, patients may experience muscle tension triggered by vibratory stimulation. At this juncture, an assistant is required to help the patient maintain the original passive stretching position and angle, whilst informing the patient to relax as much as possible. The vibratory therapy is highly operational and yields minimal adverse reactions, thus it is considered to be greatly promotable in the clinical context. It aims at alleviating as much pain and spasms in patients as possible and enhancing the patient's quality of life.

ELECTROMYOGRAPHIC BIOFEEDBACK THERAPY

Electromyographic biofeedback is a method of retraining muscles by transforming the electromyographic signals in muscles into visual and auditory cues to form a new feedback system[61]. Electromyographic biofeedback therapy uses specific low-frequency pulses exciting the nerves or muscles under a preset stimulation program. Interacting with the central nervous system, endocrine system, and immune system, it consciously controls psychological processes to influence physiological processes, thereby causing muscle relaxation and reducing tension in spastic muscles[62]. With its non-invasive nature, high sensitivity, and objectivity, electromyographic biofeedback is often used in rehabilitation of muscles with weakened force or impaired control, such as damage to upper motor neurons (Table 5)[63-68], and has shown promising results. Training can improve muscle tension and activity, and enhance coordination between muscles. Studies have shown[69] that motor learning techniques focused on biofeedback can restore sensorimotor, altered body

Effectively inhibited calf muscle spasm,

function and postural control ability

improved the active range and motor function,

and improved the balance function, walking

Improved the degree of upper limb spasm,

improved the motor function and daily living

Table 3 Efficacy of shock wave treatment on post-stroke spasticity						
Ref.	Position	Prescription	Evaluation method	Curative effect		
Li et al[36]	Upper limb	5 consecutive intervals with 4 d. 6000 pulses at pressure 1.2-1.4 bar at a frequency of 18 Hz	a, b, c, d	Clearly improved upper limb flexion spasm and significantly reduced pain		
Yoldas et al [37]	Lower limbs	Twice/wk for 2 wk (4 times in total), 1500 pulses, pressure of 2 bar, frequency of 10 Hz	a, b, e, f, g	A marked reduction in plantarflexor spasticity, no significant improvement in the 6-minute walk test and activity range		
Mihai et al [38]	Lower limbs	Once/wk for 2 wk, 2000 pulses at 10 Hz and energy density of 60 mJ	a, c, h, i, j	The degree of spasticity and pain intensity were significantly reduced, improved ankle range of motion, balance and gait		
Wang et al [39]	Upper and lower limbs	Twice/wk for 4 wk, 1500-2500 pulses/site, pressure of 2.0-2.5 bar, frequency of 5-8 Hz	a, d, i	Relieved muscle tension of hemiplegic limb and improved interjoint coordination		
Li et al[40]	Lower limbs	Three times/wk (every other day) for 4 wk, 2000 pulses per site with impact intensity of 1.5 bar at $10~{\rm Hz}$	a, d, k	Improved the degree of muscle spasm in the lower limbs, and improved lower limb function and balance function		

3 times/wk for a total of 3 wk. Calf muscle: 2000

shocks, shock wave strength 3 bar, frequency 6

Hz; Achilles tendon: 2000 shocks, shock wave

Once every 3 d, twice/wk for 3 wk at 8 Hz,

pressure 1.5-2.0 bar, 1000 pulses per site

strength 2 bar, frequency 9 Hz

a: Modified Ashworth Scale; b: The modified Tardieu scale; c: Visual analog scale score; d: Fugl-Meyer Motor Function Rating Scale; e: The 6-min walk test; f: Modified Barthel Index scale; g: Measurement of ankle mobility of the affected side; h: Tinetti Assessment tool; i: Functional activity classification assessment; j: A handheld anglometer evaluated the ankle passive range of motion; k: Berg Balance scale; l: Passive and active motion of ankle dorsiflexion; m: 10 m Walking inspection; n: Time in the stand-up-walk test.

a, d, l, k, m, n

a, d, f

Table 4 Efficacy of vibration therapy on spasticity						
Ref.	Prescription	Evaluation method	Curative effect			
Bao <i>et al</i> [54]	The frequency was 36.7 Hz, and the total duration of treatment was 10 min, once/d, 5 times/wk for 3 consecutive wk	b, c, d	Effectively reduced the spasm muscle tension, improved its elasticity and hardness, improved limb movement function			
Yin <i>et al</i> [55]	Frequency 12 Hz, amplitude 4 mm, each lasting 15 min, 5 times/wk for 12 wk	e, f, g	Effectively improved ankle motion, balance function and lower limb movement ability in children with spastic diplegia			
Li et al[56]	Frequency 30 Hz, 0.5 mm amplitude once/d 2 min each time and 2 min rest interval. Four-week treatment course with a total of two courses	a, d, h, i	Reduced ankle plantar flexor spasm, improved walking ability, and improved activities of daily living			
Katusic et al[57]	Frequency 40 Hz, 20 min each time, twice/wk for 12 wk	a, j	Reduced the level of spasticity and improved the gross motor capacity in children with spastic cerebral palsy			
Cai <i>et al</i> [58]	Treatment frequency: First 12 Hz, second 18 Hz, and third 21 Hz. 3 min/time for 9 min, training for 5 d/wk and 2 d rest for 8 wk	f, j	Effectively reduced muscle tension, improved muscle strength, abnormal posture and postural control ability in spastic cerebral paralysis			

a: Modified Ashworth scale; b: Modified Tardieu scale; c: Myoton-3 muscle state detector assessment; d: Fugl-Meyer motor function rating scale; e: Scale of ankle dorsiflexion; f: Berg, balance scale; g: Gross motor function for cerebral palsy; h: Gait analysis; i: Modified Barthel index scale; j: Gross motor function (GMFM-88).

perception and motor control, and improve quality of life in patients with ST. It provides an effective supplementary regime for the treatment of ST.

OTHER TREATMENTS

Other treatments regarding affected muscle groups include acupuncture therapy, as a single target adverse stimulus, might induce immediate facilitation in spasmodic muscles. However, when considered holistically, it acts on multiple



He et al[41]

Zhao et al

[42]

Lower limbs

Upper limb

Table 5 Efficacy	of electromyogr	raphy biofeedback t	heran	v on muscle (enacticity
I able J Lilleacy	OI CICCLIOIIIYOQI	iapily biolecuback i	nciap	y on muscie :	spasiicity

Ref.	Disease name	Intervention study	Frequency and duration of the intervention	Outcome indicators	Curative effect
Gulseren et al [63]	Post-stroke spasm	E: EMG-BF + conventional treatment; C: Conventional therapy	1/d, 5/wk, 20 min/time for 3 wk	a, d, e	Improvements in joint mobility, muscle strength, muscle tone, and function
Xie et al[64]	Post-stroke spasm	E: EMG-BF + conventional treatment; C: Conventional therapy	2 times/d, 5 times/wk, 15-20 min/time for 12 wk	b, c	Improved neurological function, motor function and quality of life in patients
Wang et al[65]	Post-stroke spasm	E: EMG-BF + rehabilitation training; C: Rehabilitation training	1 time/d, 5 times/wk, 30 min/time for 8 wk	a, c, d	Effectively reduced wrist spasm in patients with early cerebral infarction
Shi <i>et al</i> [66]	Post-stroke spasm	E: EMG-BF + rehabilitation training; C: Rehabilitation training	2 times/d, 30 min/time for 8 wk	a, c	Reduced patient spasticity level and improved motor function
Xu et al[67]	Spastic cerebral palsy	E: EMG-BF + rehabilitation training; C: Rehabilitation training	20-30 min/time, once/d, 6 times/wk for 12 wk	f, j, h, i	Improved gross motor function and serological indicators in children, and improved the development quotient and self-care ability score
Zhang et al[68]	Cerebral palsy	E: EMG-BF + rehabilitation training; C: rehabilitation training	20 min/time, once/d, 5 times/wk for 12 wk	a	Significantly improved exercise capacity and quality of life

[&]quot;E" represents the test group; "C" represents the control group; EMG-BF: Electromyographic biofeedback; CPM: Continuous passive movement; a: Modified Ashworth score; b: Clinical spasm index; c: Fugl-Meyer motor function score; d: Degree of active joint activity; e: Surface EMG value; f: Gross motor function assessment table (GMFM-88); g: Daily life performance scale (ADL); h: Gesell pediatric neuropsychological development scale; i: Serological

targets in the body, which might elicit corresponding feedback and adjustment relative to the local spasm, and the longterm effect might inhibit the spasm[70]. Studies have shown that acupuncture could serve as an adjuvant therapy, helping ST patients reduce the frequency of botulinum toxin usage and alleviate muscle tension[71,72]. Multiple studies suggest that acupuncture therapy can relieve muscle spasm in ST patients, and as it has no side effects, it provides a new treatment option for ST treatment[73-76].

High-intensity laser treatment has photodynamic, photothermal, and photochemical properties, with several therapeutic effects including bio-stimulation, pain relief, anti-inflammatory effects, surface thermal effects, and muscle relaxation[77]. In one case of acute torticollis in a child caused by rotatory atlantoaxial subluxation, high-intensity laser therapy significantly alleviated muscle spasms and neck pain. This is mainly because high-power pulse emissions, believed to have photodynamic effects, can relax muscle spasms by massaging soft tissue structures, thereby stimulating deeper tissues[78]. Although there are few ST cases treated with high-intensity laser therapy, its functions of muscle relaxation and pain relief provide a new adjunct treatment for clinical practice.

OUTLOOK

The etiology of ST remains unclear, and is currently believed to be associated with various factors including genetics and the environment, and their interactions. In the epidemiology of muscle tension disorders, idiopathic isolated dystonia is the most common, with neck dystonia being the most prevalent form (with 3 to 13 cases per 100000 persons)[79]. Genetically, dystonia presents in multiple forms with inherited isolated dystonia predominately showing an autosomal dominant inheritance (such as DYT6, DYT1)[80]. DYT-THAP1 (DYT6) is a common early-onset isolated dystonia, typically manifesting in children and adolescents[81], primarily displaying as cranio-cervical dystonia, with slow or no progress[82]. DYT-TOR1A (DYT1) is the most common early-onset generalized dystonia, involving the GAG —internal deletion in TOR1A, particularly common in individuals of German-Jewish descent[83]. Early symptoms often involve the leg or later the arm, then rapidly generalize while 20% may still remain localized, usually involving writer's cramp[83].

Evidence suggests that functional changes in the dopamine signaling process may be associated with the onset of dystonia[84]. Positron emission tomography showed that the binding rate of dopamine D2 receptors and labeled ligands in the shell nucleus of dystonic patients is lower compared to normal individuals[83,85]. Moreover, two genetic defects have been discovered leading to abnormal dopamine synthesis: Deficiencies in Guanosine-triphosphate Cyclase can result in autosomal dominant inherited dopamine-responsive dystonia (such as DYT5a/GCH1 mutations, DYT5b/TH defects), while Tyrosine Hydroxylase deficiency can result in autosomal recessive inherited dopamine-responsive dystonia[86]. Advances in genetic technology have led to a rapid increase in the number of dystonia genes. The increase in gene numbers is significant as they assist in understanding the genetic causes of inherited dystonia and help link them to shared biological pathways. It appears unlikely there could be a final universal pathway to explain all types of dystonia; instead, multiple pathways can explain specific dystonia. For inherited dystonia, drugs, surgery, and the aforementioned rehabilitation treatments have limited therapeutic efficacy. Future treatments, based on pathological mechanisms, tend to lean more towards gene therapy.

CONCLUSION

The etiology and mechanism of ST are not yet clear. Currently, there are few articles on the independent use of the above rehabilitation treatments. Most are used in conjunction with botulinum toxin therapy or other rehabilitation measures. In clinical practice, when we encounter patients with complex conditions or poor symptom control, attention should also be paid to whether the patient has genetic factors or abnormal genes. In addition to the use of drugs and surgery, we should also try to combine various rehabilitation methods to solve the patient's problems as much as possible. It is hoped that in the future, gene therapy can be placed on the agenda as soon as possible to solve fundamental problems for patients.

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