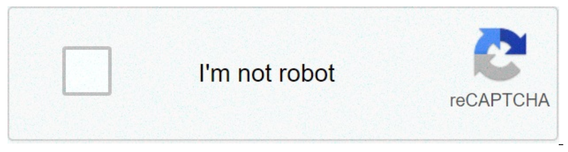

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Dystonia medical definition

Neurological movement disorder Medical conditionDystoniaA person with medication-induced dystoniaSpecialtyNeurologyComplicationsphysical disabilities (contractures, torticollis),[1] pain and fatigue[2]Causeshereditary (DYT1); birth injury; head trauma; medication; infection; toxinsDiagnostic methodgenetic testing, electromyography, blood tests, MRI or CT scanTreatmentmedication, physical therapy, botulinum toxin injection, deep brain stimulationMedicationanticholinergics, dopamine agonists Dystonia is a neurological hyperkinetic movement disorder syndrome in which sustained or repetitive muscle contractions result in twisting and repetitive movements or abnormal fixed postures.[3] The movements may resemble a tremor. Dystonia is often intensified or exacerbated by physical activity, and symptoms may progress into adjacent muscles.[4] The disorder may be hereditary or caused by other factors such as birth-related or other physical trauma, infection, poisoning (e.g., lead poisoning) or reaction to pharmaceutical drugs, particularly neuroleptics,[3] or stress. Treatment must be highly customized to the needs of the individual and may include oral medications, chemodenervation botulinum neurotoxin injections, physical therapy, or other supportive therapies, and surgical procedures such as deep brain stimulation. Classification There are multiple types of dystonia, and many diseases and conditions may cause dystonia. Dystonia is classified by: Clinical characteristics such as age of onset, body distribution, nature of the symptoms, and associated features such as additional movement disorders or neurological symptoms, and Cause (which includes changes or damage to the nervous system and inheritance).[4] Physicians use these classifications to guide diagnosis and treatment. Types Generalized Focal Segmental Psychogenic Acute dystonic reaction[5] Vegetative-vascular Generalized dystonias For example, dystonia musculorum deformans (Oppenhiem, Flatau-Sterling syndrome): [6] Normal birth history and milestones Autosomal dominant Childhood onset Starts in lower limbs and spreads upwards Also known as torsion dystonia or idiopathic torsion dystonia (old terminology "dystonia musculorum deformans"). Focal dystonias Main article: Focal dystonia These most common dystonias are typically classified as follows: Name Location Description Anismus muscles of the rectum Causes painful defecation, constipation; may be complicated by encopresis. Cervical dystonia (spasmodic torticollis) muscles of the neck Causes the head to rotate to one side, to pull down towards the chest, or back, or a combination of these postures. Blepharospasm muscles around the eyes The sufferer experiences rapid blinking of the eyes or even their forced closure causing functional blindness. Oculogyric crisis muscles of eyes and head An extreme and sustained (usually) upward deviation of the eyes often with convergence causing diplopia (double vision). It is frequently associated with backward and lateral flexion of the neck and either widely opened mouth or jaw clenching. Frequently a result of antiemetics such as the neuroleptics (e.g., prochlorperazine) or metoclopramide. Can be caused by Chlorpromazine. Oromandibular dystonia muscles of the jaw and muscles of tongue Causes distortions of the mouth and tongue. Spasmodic dysphonia/Laryngeal dystonia muscles of larynx Causes the voice to sound broken, become hoarse, sometimes reducing it to a whisper. Focal hand dystonia (also known as musician's or writer's cramp). single muscle or small group of muscles in the hand It interferes with activities such as writing or playing a musical instrument by causing involuntary muscular contractions. The condition is sometimes "task-specific," meaning that it is generally apparent during only certain activities. Focal hand dystonia is neurological in origin and is not due to normal fatigue. The loss of precise muscle control and continuous unintentional movement results in painful cramping and abnormal positioning that makes continued use of the affected body parts impossible. The combination of blepharospasmodic contractions and oromandibular dystonia is called cranial dystonia or Meige's syndrome. Segmental dystonias Segmental dystonias affect two adjoining parts of the body:[citation needed] Hemidystonia affects an arm and foot on one side of the body. Multifocal dystonia affects many different parts of the body. Generalized dystonia affects most of the body, frequently involving the legs and back. Genetic/primary Symbol OMIM Gene Locus Alt Name DYT1 128100 TOR1A 9q34 Early-onset torsion dystonia DYT2 224500 HPCA 1p35-p34.2 Autosomal recessive primary isolated dystonia DYT3 314250 TAF1 Xq13 X-linked dystonia-parkinsonism DYT4 128101 TUBB4[7] 19p13.12-13 Autosomal dominant whispering dysphonia DYT5a 128230 GCH1 14q22.1-q22.2 Autosomal dominant dopamine-responsive dystonia DYT5b 191290 TH 11p15.5 Autosomal recessive dopamine-responsive dystonia DYT6 602629 THAP1 8p11.21 Autosomal dominant dystonia with cranio-cervical predilection DYT7 602124 unknown 18p (questionable) Autosomal dominant primary focal cervical dystonia DYT8 118800 MR1 2q35 Paroxysmal nonkinesigenic dyskinesia DYT9 601042 SLC2A1 1p35-p31.3 Episodic choreoathetosis/spasticity (now known to be synonymous with DYT18) DYT10 128200 PRRT2 16p11.2-q12.1 Paroxysmal kinesigenic dyskinesia DYT11 159900 SGCE 7q21 Myoclonic dystonia DYT12 128235 ATP1A3 19q12-q13.2 Rapid onset dystonia parkinsonism and alternating hemiplegia of childhood DYT13 607671 unknown, near D1S2667[8] 1p36.32-p36.13 Autosomal dominant cranio-cervical/upper limb dystonia in one Italian family DYT14 See DYT5 DYT15 607488 unknown 18p11[9] Myoclonic dystonia not linked to SGCE mutations DYT16 612067 PRKRA 2q31.3 Autosomal recessive young onset dystonia parkinsonism DYT17 612406 unknown, near D20S107[10] 20p11.2-q13.12 Autosomal recessive dystonia in one family DYT18 612126 SLC2A1 1p35-p31.3 Paroxysmal exercise-induced dyskinesia DYT19 611031 probably PRRT2 16q13-q22.1 Episodic kinesigenic dyskinesia 2, probably synonymous with DYT10 DYT20 611147 unknown 2q31 Paroxysmal nonkinesigenic dyskinesia 2 DYT21 614588 unknown 2q14.3-q21.3 Late-onset torsion dystonia DYT24 610110 ANO3[11] 11p14.2 Autosomal dominant cranio-cervical dystonia with prominent tremor There is a group called myoclonic dystonia where some cases are hereditary and have been associated with a missense mutation in the dopamine-D2 receptor. Some of these cases have responded well to alcohol.[12][13] Other genes that have been associated with dystonia include CIZ1, GNAL, ATP1A3, and PRRT2.[14] Another report has linked THAP1 and SLC20A2 to dystonia.[15] Signs and symptoms Play media Hyperglycemia-induced involuntary movements, which, in this case, did not consist of typical hemiballismus but rather of hemichorea (dance-like movements of one side of the body; initial movements of the right arm in the video) and bilateral dystonia (slow muscle contraction in legs, chest, and right arm) in a 62-year-old Japanese woman with type 1 diabetes. Symptoms vary according to the kind of dystonia involved. In most cases, dystonia tends to lead to abnormal posturing, in particular on movement. Many sufferers have continuous pain, cramping, and relentless muscle spasms due to involuntary muscle movements. Other motor symptoms are possible including lip smacking.[16] Early symptoms may include loss of precision muscle coordination (sometimes first manifested in declining penmanship, frequent small injuries to the hands, and dropped items), cramping pain with sustained use, and trembling. Significant muscle pain and cramping may result from very minor exertions like holding a book and turning pages. It may become difficult to find a comfortable position for arms and legs with even the minor exertions associated with holding arms crossed causing significant pain similar to restless leg syndrome. Affected persons may notice trembling in the diaphragm while breathing, or the need to place hands in pockets, under legs while sitting or under pillows while sleeping to keep them still and to reduce pain. Trembling in the jaw may be felt and heard while lying down, and the constant movement to avoid pain may result in the grinding and wearing down of teeth, or symptoms similar to temporomandibular joint disorder. The voice may crack frequently or become harsh, triggering frequent throat clearing. Swallowing can become difficult and accompanied by painful cramping.[citation needed] Electrical sensors (EMG) inserted into affected muscle groups, while painful, can provide a definitive diagnosis by showing pulsating nerve signals being transmitted to the muscles even when they are at rest. The brain appears to signal portions of fibers within the affected muscle groups at a firing speed of about 10 Hz causing them to pulsate, tremble and contort. When called upon to perform an intentional activity, the muscles fatigue very quickly and some portions of the muscle groups do not respond (causing weakness) while other portions over-respond or become rigid (causing micro-tears under load). The symptoms worsen significantly with use, especially in the case of focal dystonia, and a "mirror effect" is often observed in other body parts: Use of the right hand may cause pain and cramping in that hand as well as in the other hand and legs that were not being used. Stress, anxiety, lack of sleep, sustained use and cold temperatures can worsen symptoms.[citation needed] Direct symptoms may be accompanied by secondary effects of the continuous muscle and brain activity, including disturbed sleep patterns, exhaustion, mood swings, mental stress, difficulty concentrating, blurred vision, digestive problems, and short temper. People with dystonia may also become depressed and find great difficulty adapting their activities and livelihood to a progressing disability. Side-effects from treatment and medications can also present challenges in normal activities.[citation needed] In some cases, symptoms may progress and then plateau for years, or stop progressing entirely. The progression may be delayed by treatment or adaptive lifestyle changes, while forced continued use may make symptoms progress more rapidly. In others, the symptoms may progress to total disability, making some of the more risky forms of treatment worth considering. In some cases with patients who already have dystonia, a subsequent traumatic injury or the effects of general anethesia during an unrelated surgery can cause the symptoms to progress rapidly.[citation needed] An accurate diagnosis may be difficult because of the way the disorder manifests itself. Sufferers may be diagnosed as having similar and perhaps related disorders including Parkinson's disease, essential tremor, carpal tunnel syndrome, temporomandibular joint disorder, Tourette's syndrome, conversion disorder or other neuromuscular movement disorders. It has been found that the prevalence of dystonia is high in individuals with Huntington's disease, where the most common clinical presentations are internal shoulder rotation, sustained fist clenching, knee flexion, and foot inversion.[17] Risk factors for increased dystonia in patients with Huntington's disease include long disease duration and use of antidopaminergic medication.[17] Causes Primary dystonia is suspected when the dystonia is the only sign and there is no identifiable cause or structural abnormality in the central nervous system. Researchers suspect it is caused by a pathology of the central nervous system, likely originating in those parts of the brain concerned with motor function—such as the basal ganglia and the GABA (gamma-aminobutyric acid) producing Purkinje neurons. The precise cause of primary dystonia is unknown. In many cases it may involve some genetic predisposition towards the disorder combined with environmental conditions.[citation needed] Secondary dystonia refers to dystonia brought on by some identified cause, such as head injury[citation needed], drug side effect (e.g. tardive dystonia), or neurological disease (e.g. Wilson's disease). Meningitis and encephalitis caused by viral, bacterial, and fungal infections of the brain have been associated with dystonia. The main mechanism is inflammation of the blood vessels, causing restriction of blood flow to the basal ganglia. Other mechanisms include direct nerve injury by the organism or a toxin, or autoimmune mechanisms.[18] Environmental and task-related factors are suspected to trigger the development of focal dystonias because they appear disproportionately in individuals who perform high precision hand movements such as musicians, engineers, architects, and artists.[citation needed] Chlorpromazine can also cause dystonia, which can be often misjudged as a seizure.[citation needed] Neuroleptic drugs often cause dystonia, including oculogyric crisis.[citation needed] Malfunction of the sodium-potassium pump may be a factor in some dystonias. The Na+-K+ pump has been shown to control and set the intrinsic activity mode of cerebellar Purkinje neurons.[19] This suggests that the pump might not simply be a homeostatic, "housekeeping" molecule for ionic gradients; but could be a computational element in the cerebellum and the brain.[20] Indeed, an ouabain block of Na+-K+ pumps in the cerebellum of a live mouse results in it displaying ataxia and dystonia.[21] Ataxia is observed for lower ouabain concentrations, dystonia is observed at higher ouabain concentrations. A mutation in the Na+-K+ pump (ATP1A3 gene) can cause rapid onset dystonia parkinsonism.[22] The parkinsonism aspect of this disease may be attributable to malfunctioning Na+-K+ pumps in the basal ganglia; the dystonia aspect may be attributable to malfunctioning Na+-K+ pumps in the cerebellum (that act to corrupt its input to the basal ganglia) possibly in Purkinje neurons.[19] Cerebellum issues causing dystonia is described by Filip et al. 2013: "Although dystonia has traditionally been regarded as a basal ganglia dysfunction, recent provocative evidence has emerged of cerebellar involvement in the pathophysiology of this enigmatic disease. It has been suggested that the cerebellum plays an important role in dystonia etiology, from neuroanatomical research of complex networks showing that the cerebellum is connected to a wide range of other central nervous system structures involved in movement control to animal models indicating that signs of dystonia are due to cerebellum dysfunction and completely disappear after cerebellectomy, and finally to clinical observations in secondary dystonia patients with various types of cerebellar lesions. It is proposed that dystonia is a large-scale dysfunction, involving not only cortico-basal ganglia-thalamo-cortical pathways, but the cortico-ponto-cerebello-thalamo-cortical loop as well. Even in the absence of traditional "cerebellar signs" in most dystonia patients, there are more subtle indications of cerebellar dysfunction. It is clear that as long as the cerebellum's role in dystonia genesis remains unexamined, it will be difficult to significantly improve the current standards of dystonia treatment or to provide curative treatment."[23] Treatment Reducing the types of movements that trigger or worsen dystonic symptoms provides some relief, as does reducing stress, getting plenty of rest, moderate exercise, and relaxation techniques.[citation needed] Various treatments focus on sedating brain functions or blocking nerve communications with the muscles via drugs, neuro-suppression, or denervation.[citation needed] All current treatments have negative side-effects and risks. A geste antagoniste is a physical gesture or position (such as touching one's chin) that temporarily interrupts dystonia, it is also known as a sensory trick.[24] Patients may be aware of the presence of a geste antagoniste that provides some relief.[25] Therapy for dystonia can involve prosthetics that passively simulate the stimulation.[26] Physical intervention While research in the area of effectiveness of physical therapy intervention for dystonia remains weak,[27] there is reason to believe that rehabilitation can benefit dystonia patients.[28] Physical therapy can be utilized to manage changes in balance, mobility and overall function that occur as a result of the disorder.[29] A variety of treatment strategies can be employed to address the unique needs of each individual. Potential treatment interventions include splinting,[30] therapeutic exercise, manual stretching, soft tissue and joint mobilization, postural training and bracing,[28] neuromuscular electrical stimulation, constraint-induced movement therapy, activity and environmental modification, and gait training.[29] A patient with dystonia may have significant challenges in activities of daily living (ADL), an area especially suited for treatment by occupational therapy (OT). An occupational therapist (OT) may perform needed upper extremity splinting, provide movement inhibitory techniques, train fine motor coordination, provide an assistive device, or teach alternative methods of activity performance to achieve a patient's goals for bathing, dressing, toileting, and other valued activities.[citation needed] Recent research has investigated further into the role of physiotherapy in the treatment of dystonia. A recent study showed that reducing psychological stress, in conjunction with exercise, is beneficial for reducing truncal dystonia in patients with Parkinson's Disease.[31] Another study emphasized progressive relaxation, isometric muscle endurance, dynamic strength, coordination, balance, and body perception, seeing significant improvements to patients' quality of life after 4 weeks.[32] Since the root of the problem is neurological, doctors have explored sensorimotor retraining activities to enable the brain to "rewire" itself and eliminate dystonic movements. The work of several doctors such as Nancy Byl and Joaquin Farias has shown that sensorimotor retraining activities and proprioceptive stimulation can induce neuroplasticity, making it possible for patients to recover substantial function that was lost due to Cervical Dystonia, hand dystonia, blepharospasm, oromandibular dystonia, dysphonia and musicians' dystonia.[33][34][35][36][37] Some focal dystonias have been proven treatable through movement retraining in the Taubman approach, particularly in the case of musicians. However other focal dystonias may not respond and may even be made worse by this treatment.[citation needed] Due to the rare and variable nature of dystonia, research investigating the effectiveness of these treatments is limited. There is no gold standard for physiotherapy rehabilitation.[31] To date, focal cervical dystonia has received the most research attention;[29] however, study designs are poorly controlled and limited to small sample sizes.[27] Medication Different medications are tried in an effort to find a combination that is effective for a specific person. Not all people respond well to the same medications. Medications that have had positive results in some include: diphenhydramine, benzatropine and atropine. anti-Parkinsons agents (such as ropinirole and bromocriptine), and muscle relaxants (such as diazepam).[citation needed] Anticholinergics Medications such as anticholinergics (benztropine), which act as inhibitors of the neurotransmitter acetylcholine, may provide some relief. In the case of an acute dystonic reaction, diphenhydramine is sometimes used (though this drug is well known as an antihistamine, in this context it is being used primarily for its anticholinergic role).[citation needed]. See also Procyclidine. Another which in many has shown total control of symptoms, alone or taken with other medications,often clonazepam, is gabapentin. Noting, some are generic sensitive, as with other medications, and either have to sample test each one for a successful result, or take the original version.[citation needed] Baclofen A baclofen pump has been used to treat patients of all ages exhibiting muscle spasticity along with dystonia. The pump delivers baclofen via a catheter to the thecal space surrounding the spinal cord. The pump itself is placed in the abdomen. It can be refilled periodically by access through the skin. Baclofen can also be taken in tablet form[38] Botulinum toxin injection Botulinum toxin injections into affected muscles have proved quite successful in providing some relief for around 3–6 months, depending on the kind of dystonia. Botox or Dysport injections have the advantage of ready availability (the same form is used for cosmetic surgery) and the effects are not permanent. There is a risk of temporary paralysis of the muscles being injected or the leaking of the toxin into adjacent muscle groups, causing weakness or paralysis in them. The injections must be repeated, as the effects wear off and around 15% of recipients develop immunity to the toxin. There is a Type A and a Type B toxin approved for treatment of dystonia; often, those that develop resistance to Type A may be able to use Type B.[39] Muscle relaxants Clonazepam, a benzodiazepine, is also sometimes prescribed. However, for most, their effects are limited and side-effects like mental confusion, sedation, mood swings, and short-term memory loss occur.[citation needed] Parkinsonian drugs Dopamine agonists: One type of dystonia, dopamine-responsive dystonia, can be completely treated with regular doses of L-DOPA in a form such as Sinemet (carbidopa/levodopa). Although this does not remove the condition, it does alleviate the symptoms most of the time. (In contrast, dopamine antagonists can sometimes cause dystonia.)[citation needed] Ketogenic diet One complex case study found that a ketogenic type diet may have been helpful in reducing symptoms associated with alternating hemiplegia of childhood (AHC) of a young child. However, as the researchers noted, their results could have been corollary in nature and not due to the diet itself, though future research is warranted.[40] Surgery Schematic representation of a patient with cervical dystonia, with deep brain stimulation (DBS) electrodes implanted in the internal globus pallidus (GPi) Surgery, such as the denervation of selected muscles, may also provide some relief; however, the destruction of nerves in the limbs or brain is not reversible and should be considered only in the most extreme cases. Recently, the procedure of deep brain stimulation (DBS) has proven successful in a number of cases of severe generalised dystonia.[41] DBS as treatment for medication-refractory dystonia, on the other hand, may increase the risk of suicide in patients. However, reference data of patients without DBS therapy are lacking.[42] History The Italian Bernardino Ramazzini provided one of the first descriptions of task-specific dystonia in 1713 in a book of occupational diseases, The Morbis Artificum.[43] In chapter II of this book's Supplementum, Ramazzini noted that "Scribes and Notaries" may develop "incessant movement of the hand, always in the same direction ... the continuous and almost tonic strain on the muscles... that results in failure of power in the right hand". A report from the British Civil Service also contained an early description of writer's cramp. In 1864, Solly coined the term "scrivener's palsy" for this affliction. These historical reports usually attributed the etiology of the motor abnormalities to overuse. Then, dystonia were reported in detail in 1911, when Hermann Oppenheim,[44] Edward Flatau and Wladyslaw Sterling described some Jewish children affected by a syndrome that was retrospectively considered to represent familial cases of DYT1 dystonia. Some decades later, in 1975, the first international conference on dystonia was held in New York. It was then recognized that, in addition to severe generalized forms, the dystonia phenotype also encompasses poorly-progressive focal and segmental cases with onset in adulthood, such as blepharospasm, torticollis and writer's cramp. These forms were previously considered independent disorders and were mainly classified among neuroses. A modern definition of dystonia was worded some years later, in 1984. During the following years it became evident that dystonia syndromes are numerous and diversified, new terminological descriptors (e.g., dystonia plus, heredodegenerative dystonias, etc.) and additional classification schemes were introduced. The clinical complexity of dystonia was then fully recognized.[45] See also Extrapyramidal symptoms Hypertonia Sydenham's chorea Ulegyria (brain condition with dystonia symptoms)

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