

Transient Global Amnesia, Movement Disorders and Gait Disorders

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Transient Global Amnesia

TGA is a somewhat rare clinical syndrome, often associated with increased social stress, severe pain, cold-water immersion, severe exertion or sex, and characterized by:

- short-term memory impairment with repetitive questions,
- severely impaired access to long-term memory no other neurological symptoms, and
- an otherwise normal neurological examination and no immediate identifiable cause.

The timeframe ranges from about an hour (15% of cases) up to a week. Rarely some deficits last 4 weeks. Average is about 7 hours. The recurrence rate is about 10%. *Acute Confusional Migraine* is thought to be a similar syndrome in children.

As a clinical syndrome, the final common pathway is likely temporary impairment of the hippocampal memory circuits.

There are several common associations and possible causes:

- migraine – particularly in younger adults and those involved with competitive sports – likely the major association
- petit mal seizures
- although less common, CVAs and TIAs

There is some association with increased venous back-pressure and reflux of the internal jugular.

There are case reports of TGA associated with: exercise/subclavian steal, heavy lifting with PFO (presumed embolic CVA), drugs (OCPs, propafenone, heparin, Viagra), vasospasm from an angiogram, early encephalitis, CNS AV fistula, cerebral thrombophlebitis, aortic dissection, acute MI.

One interesting study looked at risks (physical exertion, emotional stress, sexual intercourse or immersion in cold water) and there was a high association with total number of risks and occurrences of TGA.¹

Workup:

- CT AND MRI: in one study, 10% had

structural brain lesions. Maybe CT angio (though dissection rare)

- ADMIT for observation and possible EEG and echo with bubble test.

Movement Disorders: 'Not everything that shakes is epilepsy'

EXTRAPYRAMIDAL REACTIONS are found from antipsychotic drugs, and other phenothiazines such as Compazine and Reglan, and treated with anticholinergic effects of antihistamines (diphenhydramine, e.g., Benadryl) 12.5-50 mg PO, + benzotropine (e.g., Cogentin) 1-2 mg IM or PO.

EXTRAPYRAMIDAL SYMPTOM CLASSIFICATION BY TIME ONSET:

- EARLY (acute: hours-days): acute dystonic reactions, acute akathisia
- INTERMEDIATE (acute: days-weeks): parkinsonism, akathisia
- LATE (chronic: months-years): tardive dyskinesias

DYSTONIC REACTIONS are briefly sustained or fixed abnormal postures including torticollis, oculogyric crisis (sustained deviation of the eyes and neck), facial grimacing, opisthotonos (spinal spasms) or laryngeal spasm. There is often a sense of panic.

TARDIVE DYSKINESIA (not usually classified as extrapyramidal) is involuntary buccolinguomasticatory movements including lip-smacking, tongue protrusion, grimacing, chewing. It may cause occasional loss of motor control causing patient to fall. It may involve sustained postures, initially of the face, eyes, lips and tongue, but sometimes also of the fingers, toes, or trunk.

AKATHISIA is a sense of motor restlessness; the patient is often pacing, shifting weight from foot to foot, has "restless legs" and often a sense of claustrophobia. Akathisia is common from Reglan and Compazine. Benadryl and Cogentin seem to be only mildly effective, some argue worse than useless; benzodiazepines such as Versed seem better. Inderal has been touted but the author

has found it useless.

PAROXYSMAL NONEPILEPTIFORM MOVEMENT DISORDERS

Generalized seizure-like movements may come from syncope from cardiac or postural causes, hypoglycemia or hyperventilation. Toxic and metabolic causes of seizure-like movements include hypoxia, DTs, porphyria, the tonic spasm of tetanus, rabies, and strychnine poisoning, black widow spider bites and Centruroides scorpion stings. Pseudoseizures, subarachnoid hemorrhage, CVA, and migraine may all trigger seizure-like movements.

PAROXYSMAL KINESIGENIC DYSKINESIA (PKD) AND PAROXYSMAL NON-KINESIGENIC DYSKINESIA (PNKD) are sudden, transient attacks of chorea, athetosis, dystonia, ballismus, or any combination of these abnormal movements involving muscle groups of the arms, legs, trunk, face, and/or neck. Kinesigenic dyskinesias are those brought on by attempts at movement, but non-kinesigenic dyskinesia starts spontaneously. These are mostly chronic conditions with no specific ED management but worth reviewing.

- ESSENTIAL TREMOR is considered the most common neurologic movement disorder, with involuntary, rhythmic tremor of a body part, most typically the hands and arms.
- MYOCLONUS refers to sudden, brief, shock-like movements. These movements may be "positive" or "negative." Positive myoclonus results in contraction of a muscle or multiple muscles. Myoclonus is normal during the hypnogogic and hypnopompic states (just as drifting off to sleep or awakening) and may also be seen in narcolepsy and sleep apnea or hypercarbia from any cause. Persistent myoclonus also may be seen in a bewildering variety of neurological conditions.
- In ASTERIXIS, or negative myoclonus, there is a brief loss of muscle tone and then the tightening (contraction) of other muscles; this results in a flapping-type motion, and is classic for liver disease.
- CHOREA is an irregular, rapid, uncontrolled, involuntary, excessive movement that seems to flow randomly from one part of the body to another.
- ATHETOSIS is a slower writhing and twisting movement.
- CHOREOATHETOSIS is a movement of intermediate speed, between the quick, flitting movements of chorea and the slower,

Definition

"Transient global amnesia (TGA) has been defined as the presence of an impairment of short-term memory, retrograde amnesia and repetitive queries, without any other neurological signs or symptoms. The precise pathophysiology of TGA is unclear, although thromboembolic, epileptic, migrainous and metabolic mechanisms have been suggested."

[Moccia F, Aramini A, Montobbio P, Altomonte F, Greco G. Transient global amnesia: disease or syndrome? Ital J Neurol Sci 1996; 17:211-4.]

1. Agosti C, Akkawi NM, Borroni B, Padovani A. Recurrence in transient global amnesia: a retrospective study. Eur J Neurol 2006;13:986-9.

writhing movements of athetosis. Chorea and athetosis are generally associated with a variety of degenerative neurological conditions.

- **HEMIBALLISMUS** or **BALLISMUS** is a violent, flinging, flailing movement. It is generally considered a severe form of chorea. Acutely it may be caused by destruction of the contralateral subthalamic nucleus. The violence of these movements may cause death. However, movements gradually can be suppressed or interrupted by voluntary action, and in 6 to 10 weeks, may stop.
- **RESTLESS LEGS SYNDROME:** The desire to move the legs in association with unusual or uncomfortable sensations (paresthesias/dysesthesias) deep within the legs, usually in the calves; may be a *forme fruste* (minor case of) akathisia or:
- **SEROTONIN SYNDROME:** altered mental status (akathisia, agitation, confusion, incoordination, later mania or coma) with autonomic instability (hyperthermia, tachypnea, diarrhea, mydriasis, diaphoresis, shivering, tachycardia) and neuromuscular abnormalities (nystagmus, myoclonus, rigidity, tremor, hyperreflexia, clonus, seizures). Serotonin syndrome is associated with the SSRI and SNRI antidepressants including Prozac, or the herbal St. Johnswort; often associated with a precipitant such as dextromethorphan, Demerol or cocaine. Patients may have sustained choreoathetosis. Benzodiazepines are the acute treatment of choice.
- Tics are intermittent, repeated, stereotyped movements or sounds that may occur in an infrequent or almost continuous manner. Tics may be “simple,” such as a cough, grunt, facial twitch, or shoulder shrug, or “complex,” such as a word, phrase, or a stereotyped sequence of movements.
- **TOURETTE’S SYNDROME** is an inherited disorder characterized by sudden, involuntary, repetitive muscle movements (motor tics) and vocalizations (vocal tics).

PARKINSON’S SYNDROME, from damage to the substantia nigra, includes the following classic motor symptoms: tremor; bradykinesia; rigidity and freezing in place; stooped, shuffling gait; decreased arm swing when walking; difficulty arising from a chair; micrographia (small handwriting); lack of facial expression.

Five classes of drugs are used to treat PD:

- **DOPAMINERGIC AGENTS:** Levodopa (precursor to dopamine); carbidopa is included in the standard oral formulation to increase the effectiveness of levodopa and minimize nausea and vomiting. The most troubling adverse effect from long-term levodopa use are dyskinesias as described above.
- **DOPAMINE AGONISTS:** Apomorphine (Apokyn®), Bromocriptine (Parlodel®), Pramipexole (Mirapex®), Ropinirole (Requip®); agonists produce more edema and psychosis than levodopa. Subcutaneously injected apomorphine is a “rescue” with “off” episodes; onset 10 minutes, lasts ~90 minutes.

- **COMT INHIBITORS:** Entacapone (Comtan®), Tolcapone (Tasmar®)
- **MAO-B INHIBITORS:** Rasagiline as Azilect®, Selegiline as Eldepryl® and Zelapar™
- **ANTICHOLINERGICS:** Trihexyphenidyl (Artane®), Benztropine (Cogentin)
- **AMANTADINE** (Symmetrel®)

Gait Disorders

Among the neurological causes of gait disturbances are chronic, progressive neurological disorders such as Parkinson’s disease, muscular dystrophy, multiple sclerosis, amyotrophic lateral sclerosis (ALS, also known as Lou Gehrig’s disease), post-polio syndrome and Huntington’s chorea. Impaired gait can also indicate Alzheimer’s disease or other dementias. Other neurological causes include:

- **CERVICAL SPINAL STENOSIS.** A narrowing of the spinal canal in the neck that can cause pressure on the brain in the back of the skull. This may result in a spastic or ataxic gait.
- **CEREBRAL PALSY.** Brain damage typically occurring before birth and resulting in muscular impairment. This may result in a variety of gait disturbances.
- **STROKE** often results in a hemiplegic gait.
- **DIZZINESS, vertigo, Meniere’s disease, cerebellar dysfunction** or decreased sensation in the feet. Patients with any of these tend to have a wide gait, which means putting the feet more outside the body’s midline than usual.
- **PERIPHERAL NEUROPATHY.** Diabetic neuropathy may cause a disabling deformity known as Charcot foot. Neuropathies can also be due to trauma or alcoholism.
- **NERVE IMPINGEMENT** in the lower back or buttocks. Sciatica from a herniated disc, DJD, spinal stenosis or piriformis syndrome can cause weakness leading to a gait disturbance.
- **COMPLEX REGIONAL PAIN SYNDROME.** Disorder formerly known as reflex sympathetic dystrophy syndrome and causalgia. It usually occurs after a trauma to an arm or a leg and is characterized by burning or aching pain along with inflammation, skin discoloration, altered temperature, abnormal sweating and hypersensitivity of the affected area.
- **CHARCOT-MARIE-TOOTH DISEASE (CMT).** A common inherited condition, also known as hereditary motor and sensory neuropathy (HMSN) and peroneal muscular atrophy, CMT impairs peripheral nerves and may result in foot drop, high-stepping gait or deformities of the foot.

Gaits from structural or neurological causes:

- **ATAXIC GAIT.** Staggering, unsteady and uncoordinated; typically from CNS problems. A variation is the **TABETIC** gait, a high-stepping ataxic gait where the feet slap the ground.
- **TOE-WALKING GAIT.** A variation is the **EQUINE GAIT**, a high-stepping toe-walking gait.
- **STEPPAGE GAIT.** Seen with foot drop, where the foot appears to hang limp at the ankle. The foot is lifted high so the toes do not drag on the ground; the toes touch ground first.

The hip and knee are bent to clear the toes from the ground.

Gait disturbances typically structural only:

- **LIMP.** A jerky, uneven gait that may be caused by pain, weakness or deformity. Antalgic gait, a type of limp, is the most common gait disturbance. It is caused by pain and compensates for that pain by keeping weight off of a painful part as much as possible.
- **SPASTIC GAIT.** A stiff gait where the toes catch and drag, the legs are held together and the hips and knees are kept in a slightly bent position.
- **HEMIPLEGIC GAIT.** This is characteristic of paralysis or weakness in one leg and is common after a stroke. The patient swings the paralyzed leg around to bring the foot in front. This gait avoids placing weight on the affected leg.
- **SENILE GAIT.** Usually seen in the elderly. It is associated with a stooped posture, with knees and hips bent. Arm swinging is lessened and there is stiffness in turning. Steps are small and broad-based.
- **WADDLING GAIT.** The feet are held wide apart and the patient walks somewhat like a duck. This is a common gait disturbance in late pregnancy.

Gait disturbances typically neurological only:

- **FESTINATING GAIT.** The patient walks on the toes as if being pushed. Steps start slowly and increase in speed. Often, the patient cannot stop until grasping or running into something.
- **PARKINSON’S GAIT.** This is the festinating gait characteristic of Parkinson’s disease. Steps are short and shuffling, with feet scrapping the ground. They start slow and build up speed. The patient’s upper body is bent forward, head down, and arms, elbows, hips and knees are bent.
- **MAGNETIC GAIT.** AKA glue-footed gait. The patient seems to have difficulty taking the first step, as though the feet had been glued to the ground. Once the first step is made, subsequent steps are small and shuffling.
- **DOUBLE-STEP GAIT.** Alternating steps are made of different length or rate. The stride of one side does not match the other.
- **HELICOPOD GAIT.** The patient swings one or both feet in a half circle with each step.
- **SCISSOR GAIT.** The legs cross in walking. The left leg moves too far to the right and the right leg moves too far to the left.
- **TRENDELENBURG GAIT.** People with a lesion of the superior gluteal nerve have weakness of abducting the thigh at the hip. This type of gait may also be seen in L5 radiculopathy and after poliomyelitis, but is then usually seen in combination with foot drop.