

Blepharospasm and Tardive Dyskinesia

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Blepharospasm is a focal dystonia. Dystonia is a disease affecting the motor system of the brain that leads to involuntary movements and postures. In its focal forms, it affects only one part of the body. In blepharospasm, the dystonia affects the eyelid closing muscles. Blepharospasm is characterized by frequent blinking or sustained closure of the eyelids. In its purest form, involvement of the eyelid closing muscles is the only symptom. Some patients with blepharospasm will have other focal dystonias involving nearby or distant muscles. Commonly, other muscles of the face will be involved, giving rise to abnormal movements of the eyebrows, forehead, or lips. The dystonia can even spread to involve muscles that open and close the jaw and the tongue muscles. When the dystonia affects more widespread facial muscles, then the disorder is known as cranial dystonia or Meige syndrome.

Tardive dyskinesia is a movement disorder that results from taking certain drugs, particularly a class of drugs called neuroleptics. These drugs influence the chemicals in the brain. One of the chemicals affected is dopamine, which seems to play an important role in control of movement. The prolonged use of these drugs may result in some alteration in the motor system that leads to the production of involuntary movement. Since these movements are produced as a late effect of taking these drugs, the dyskinesia is called tardive, which means late. Once these movements occur, they can be quite long-lasting and possibly permanent. Tardive dyskinesias can affect any muscle in the body, but they very commonly affect cranial nerve muscles. The muscles affected tend to be the tongue, the jaw closing muscles, and the muscles around the mouth, but eyelid closing muscles can also be affected. When eyelid closing muscles are affected, there can be blinking or sustained closure of the eyelids, which could have the appearance of blepharospasm. Movements of the tongue and lips are particularly prominent in tardive dyskinesia. These movements can also occur in rhythmic repetitive trains.

Because both blepharospasm and tardive dyskinesia can cause blinking or sustained closure of the eyelids, their appearance can be similar. However, tardive dyskinesia would only infrequently involve the muscles of eye closure. Therefore, unless the focal dystonia in the patient with blepharospasm has spread to involve the rest of the face, it ordinarily would not be difficult on clinical grounds to separate patients with blepharospasm and tardive dyskinesia.

The etiologies of blepharospasm (or dystonia) and tardive dyskinesia are unknown. There are increasing hints of important genetic factors in blepharospasm. Certainly, familial generalized dystonia has a genetic origin. Evidence is accumulating that there is a higher than expected incidence of focal dystonias in some families, which indicates a genetic basis. An additional factor that seems important in the production of dystonia is trauma. Many patients have a history of irritation of the eye that precedes the development of blepharospasm. Neither genetic factors nor trauma seem to play a role in tardive dyskinesia as far as can be determined at the present time. By definition, tardive dyskinesia is caused by drug therapy. Since both movement disorders have some similarities of appearance, and the causes of both are unknown, it is possible that they are physiologically similar in some ways, but this would have to be considered speculation.

Lastly, both blepharospasm and tardive dyskinesia are difficult to treat. There is no systemic medication that works well for either condition. If patients with tardive dyskinesia have prolonged eyelid closure that makes it difficult for them to see, then it might well be appropriate to treat them with botulinum toxin, just as it is in patients who have blepharospasm.