

Future therapies in blepharospasm and related movement disorders

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There are currently few treatments which are truly effective for blepharospasm (BEB). No curative treatments exist. The introduction of botulinum toxin in the symptomatic treatment of BEB has revolutionized its management and greatly improved the quality of life of patients but has important limitations. It fails outright to improve some patients from the very beginning, it loses efficacy in some others with the passage of time and requires periodic, frequent, applications in those patients, the majority, in which it is effective. Other medications known to be effective such as the anticholinergics, baclophen or clonazepam are much less so than botulinum toxin. It is therefore not surprising that new therapies are constantly being tested or in the process of development to treat BEB as well as other related focal dystonias. Since BEB is clinically and probably etiologically heterogeneous and of unknown cause in most instances, the type of treatments proposed are quite variable ranging from oral administrations of drugs which modify different neurotransmitter systems in the brain to electrical stimulation or immobilization of the affected muscles to surgical lesion or inactivation of different basal ganglia nuclei.

In my presentation I shall review drugs with potential to improve BEB which are currently under investigation, non-drug strategies that have recently been proposed to treat dystonias such as forced immobilization and transcranial magnetic stimulation and the application of the new surgical strategies such as deep brain stimulation. In the field of movement disorders new "futuristic" treatments are being investigated in the laboratory and promise to be of great help. These techniques include stem cell therapy and gene therapy. The use of transgenic animal models is allowing us to test treatments that may normalize the biological abnormalities known to occur in some of these disorders such as Parkinson disease, Huntington disease or the hereditary dystonias. Only if we learn more about the biological and molecular substrate of BEB will we be able to apply such phenomenal array of new and exciting therapies to investigate on the cause of BEB and develop effective symptomatic and preventive treatments for this disorder.

Presented at:
20th Annual International Conference and Scientific Symposium of the Benign Essential Blepharospasm Research Foundation
Houston, TX
August 23-25, 2002

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