Plasticity and Laryngeal Dystonia

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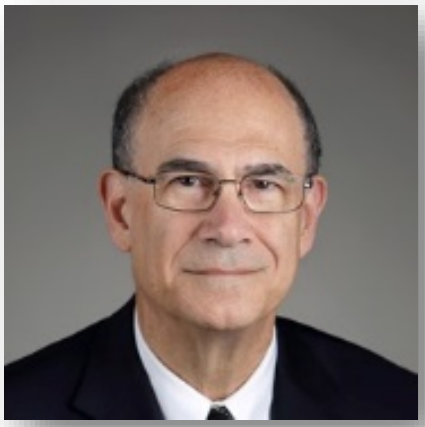

Plasticity is the capacity to change. Neuroplasticity is the capacity of the brain to change. The brain is actually highly plastic; not only *can* it change, *it is constantly* changing. Whenever a person learns something new, a new fact or a new motor skill, the brain has changed. If a person develops laryngeal dystonia (LD), also referred to as spasmodic dysphonia (SD), the brain must have changed in some way so that abnormal vocal cord movement will happen.

The basic science of neuroplasticity is being actively investigated, and much is being learned about the underlying physiology. For example, the synapses between nerve cells can be modified to convey messages either more strongly or more weakly. Additionally, nerve processes can grow or shrink. The location in the brain where the alterations occur for different types of plastic changes is also being investigated. For learning a new fact, for example, there are changes in the part of the brain called the hippocampus. The site or sites of changes in relation to LD are not fully understood but are being investigated.

Plasticity processes in dystonia are not fully normal. In some experiments, learning new skills are slower than they should be. In other experiments, plasticity actually seems excessive and changes can go beyond expected limits. Dystonia might well be produced when plastic changes are more than a brain area can ordinarily tolerate.

So if LD results from an unwanted plastic change, might it be possible to reverse that plastic change? The brain still has its neuroplasticity, why not just go back the other way and return to normal? In other words, if there was the right training exercise, could LD be treated? This idea has been studied in several different ways mainly with focal hand dystonia, particularly in patients with writer's cramp and musician's dystonia. These studies have had mixed success, and the idea is still being studied. Some therapists feel strongly that it works and have been promoting its use.

While there is certainly some reason that such approaches can work, at least with hand dystonia there are some alternate explanations for improvement with training. For example, since focal hand dystonia is often task specific, if the "task" is changed, then the dystonia is not apparent.

A pianist with musician's dystonia can get "better" just by altering technique – but in fact the original dystonia still remains with the original technique. Of course, this is a perfectly acceptable solution!

Alternative ways of speaking might well also help. For example, everyone with LD should be able to whisper without difficulty (since it does not involve the vocal cords). Some persons are able to sing even though they cannot speak normally. This uses an alternate method of vocal production. Rarely, it can also happen that there will be dysphonia for singing, but with normal talking. If there can be a plasticity changing therapy with hand dystonia, might there be something similar for LD? No such therapy has been developed yet (as far as I know).

Another way of making a plastic change is with brain stimulation, such as transcranial magnetic stimulation (TMS). TMS has been FDA approved for the treatment of depression. There is some evidence that TMS might be beneficial in some types of dystonia, but the experimental results so far are limited. Dr. Teresa Kimberley at Massachusetts General Hospital Institute of Health Professions is currently studying TMS for laryngeal dystonia.

It was hoped when botulinum toxin therapy was introduced, that correcting the speech pattern with the injection might lead to a favorable plastic change that would outlast the weakness caused by the injection. Unfortunately, that doesn't seem to be the case, and points out that even if a favorable plastic change is induced, it is also critical to maintain that change.

In summary, at the heart of LD, there is an unfavorable plastic change in the brain motor networks. It may well be possible to reverse that change, but a robust method is not yet available. Likely we will have to learn more about the pathophysiological changes before we can fully reverse them.

ABOUT MARK HALLETT, MD

Dr. Hallett obtained his A.B. and M.D. at Harvard University, had his internship in Medicine at the Peter Bent Brigham Hospital and his Neurology training at Massachusetts General Hospital. He had fellowships in neurophysiology at the NIH and in the Department of Neurology, Institute of Psychiatry in London, where he worked with C. David Marsden. Before coming to NIH in 1984, Dr. Hallett was the Chief of the Clinical Neurophysiology Laboratory at the Brigham and Women's Hospital in Boston and progressed to Associate Professor of Neurology at Harvard Medical School. He is currently Chief of the Medical Neurology Branch and Chief of its Human Motor Control Section. He is now Past-President of the International Federation of Clinical Neurophysiology. He has been President of the International Parkinson and Movement Disorder Society and Vice-President of the American Academy of Neurology. He served as Editor in Chief of Clinical Neurophysiology. Among many awards, in 2012 he became an Honorary Member of the American Neurological Association, and in 2014 won the Lifetime Achievement Award of the American Association of Neuromuscular and Electrodiagnostic Medicine. In 2017 he received the degree of Doctor of Medicine Honoris Causa from the University of Hamburg, and in 2018 was made an Honorary Member of the European Academy of Neurology. His research activities focus on the physiology of human voluntary movement and its pathophysiology in disordered voluntary movement and involuntary movement.