Dystonia, and particularly focal and segmental dystonia, is traditionally seen as an involuntary movement, caused by the lesion of basal ganglia, most probably putamen. This view of focal and segmental dystonia led to the development of current treatment strategies, which use botulinum toxin (BTX). BTX is injected according to the clinical manifestation of dystonia, according to its muscular pattern and sometimes on the basis of polymyographic finding. This pathophysiological, diagnostic and treatment concept is reasonable and useful, and it is usually successful regarding the therapeutically outcome, which is permanent.

On the other hand, some different insights into the pathophysiology of dystonia have been published in recent years. These concepts have not seen dystonia as a motor disorder or simple involuntary movement. They found dystonia to be rather “unconscious” than “involuntary” movement, which is caused by the defective chain of normal “motor routines” and “motor subroutines” in the motor program. Then the movement, which is normally performed unconsciously and automatically, is performed according to this defective program. However, the motor performance itself remains normal. This concept of dystonic movement deserves a slightly different therapeutically approach. BTX, when used, should be directed into those muscles, which prime and start the dystonic movement, and also into their antagonists. Then the possible feedback effect, mediated through Ia afferents, can involve central mechanism of dystonia, and might induce long-lasting remission.