

A MODERN PERSPECTIVE ON SOME OF THE MOST HIGHLY CITED JNNP PAPERS OF ALL TIME

EMG analysis of stereotyped voluntary movements in man

Mark Hallett



Mark Hallett from NIH describes how understanding of motor control principles and opening a window on movement pathophysiology begins with a detailed analysis of the patterns of motor output

Movement is the principal output of the brain and the way that an organism interacts with the environment. One of the major goals of neuroscience is to understand how the brain makes movement; and one of the goals of neurology is to understand how the processes fall apart in different movement disorders. A good way to start the analysis of this set of problems is to analyse movement itself, from the bottom up. There are many degrees of freedom of the body and movements could be accomplished in many different ways. As central nervous systems have evolved they have certainly taken certain directions which simplify, and hopefully optimise, how movements are actually made. Measuring the EMG of muscles involved in the movement is a direct reflection of the motor commands. This paper looks at the EMG patterns in several types of normal voluntary movements and can be viewed as an introduction to analyses of how these patterns are normally made and can be used as a measure for characterising movement problems.

This paper was my first in the field of motor control. I was a membrane biophysicist and got interested in motor control by interacting with Edward Evarts, Ragnar Granit and Mahlon

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EMG ANALYSIS OF STEREOTYPED VOLUNTARY MOVEMENTS IN MAN¹

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DeLong when I was a fellow at the National Institutes of Health (NIH). During my neurology residency that followed, neurology residents were encouraged to do research and Bhagwan Shahani convinced me to do this in the physiology laboratory that was supervised by himself and Robert Young. I wanted to follow-up a paper by Ronald Angel that we had read in a journal club at NIH,² and to use this information to start studying movement disorders.

In the work leading to this paper, I investigated normal slow and fast flexion movements at a single joint, the elbow. There had been some previous papers on the subject, beginning, of course, in the 'old' German literature, but the findings had not been consistent. Slow movements could just be continuous activity of the agonist muscle. The pattern of the fast, or ballistic, movement was stereotyped in the subjects and showed a triphasic pattern of sequential agonist, antagonist and agonist activity. These activities were in bursts and generally no more than 100 ms duration. When the antagonist muscle was active prior to a fast movement, it silenced with a pause before the agonist burst. There were also some experiments which added evidence to the idea that these patterns were centrally programmed, rather than resulting from spinal reflexes. One important bit of evidence was that all these patterns were present in a patient with a pan-sensory neuropathy.

I also began the work on movement disorders by doing similar studies in patients with cerebellar ataxia which led to a companion paper in *JNNP* that immediately followed the normal physiology study.³ This showed that the patterns were distorted in these patients. Prolonged EMG bursts appeared to underlie dysmetria and the failure of the antagonist silence prior to agonist activity

was suggested to be a factor in dysdiadochokinesia. Analysis of other movement disorders followed over the next years. Another observation along these lines, also highly cited, was that bradykinesia in patients with Parkinson's disease is characterised by repetitive running of the triphasic pattern with low amplitude.⁴ This type of work, showing elemental movement patterns in disease, from my laboratory and others was largely completed by the late 90's.⁵ These studies showed that slowness and incoordination from different pathologies might well look different. The collective work gives both a clinical neurophysiological tool for differential diagnosis and direction for further studies.

While these studies have clearly been valuable, they are indeed only the beginning of understanding the pathophysiology. We know what needs to be explained. More still needs to be learnt about how the CNS command is crafted to create these patterns and how it becomes distorted in disease. The way the motor cortex produces the basic triphasic pattern command is still not known. And, of course, there are also other elemental principles of motor control such as primitive movement modules,⁶ how to combine elemental movements into more complex movements, surround inhibition⁷ and motor learning.

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