# A kinematic analysis of finger tapping in dystonia

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**Objective:** To perform a computerised analysis of finger tapping in patients with dystonia and healthy controls in order to develop a better understanding of the dynamics of repetitive actions in dystonia.

**Background:** The electromyographic features of dystonia—abnormally long EMG bursts, agonist-antagonist co-contraction, overflow activity in remote muscles—are well recognised. Kinematic studies reveal slower, less precise and more variable movements. Analysis of bradykinesia in dystonia has revealed no decremental tendency. However, a detailed exploration of its characteristics has not yet been performed.

**Methods:** 30 patients with dystonia—21 with idiopathic focal dystonia (15 cervical, 6 focal hand), 5 with generalised genetic dystonia and 4 with secondary dystonia—and 23 age- and sex-matched controls were compared. A simple task involving repetitive finger tapping was assessed while subjects wore electromagnetic sensors secured to index finger and thumb. Subjects were advised to tap "as fast and as big as possible" for two trials, each 15 seconds long. Precise position and orientation data, in six degrees of freedom, were recorded from each sensor. A high sampling rate permitted 'real time' analysis of movement. Separable components (such as rhythm, speed and amplitude) were derived from a comparison of the *x*, *y* and *z* coordinates of each sensor. These components were extracted from the data using a custom script written in MATLAB (The MathWorks Inc., Natick, Massachusetts, USA). Data from the two trials was then averaged and analysed for statistical significance by Mann-Whitney *U* test using SPSS.

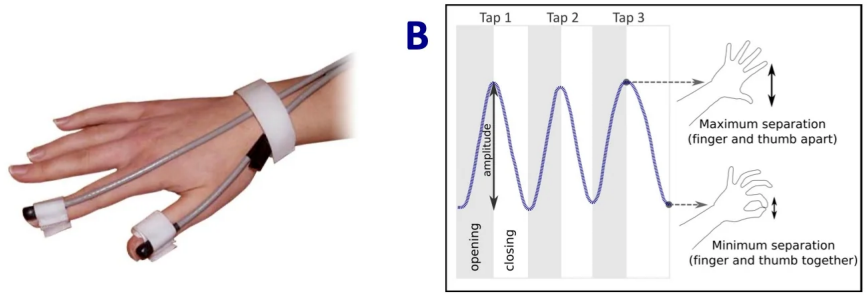


**Figure 1:** A: Two electromagnetic sensors attached to a subject's hand; B: Representational diagram of positional separation data, showing opening and closing phases of three tapping cycles.

**Results:** In the dystonia group frequency ($p = 0.008$) and maximum opening deceleration ($p = 0.001$) were reduced, and the percentage of halts was increased ($p = 0.012$). The product of amplitude and frequency gives the excursion of the movement sensors per unit time, a measure of the average speed of movement during the 15s task. This was lower in the dystonia group ($p = 0.005$). There were no significant group differences in decremental tendency or rhythmicity.

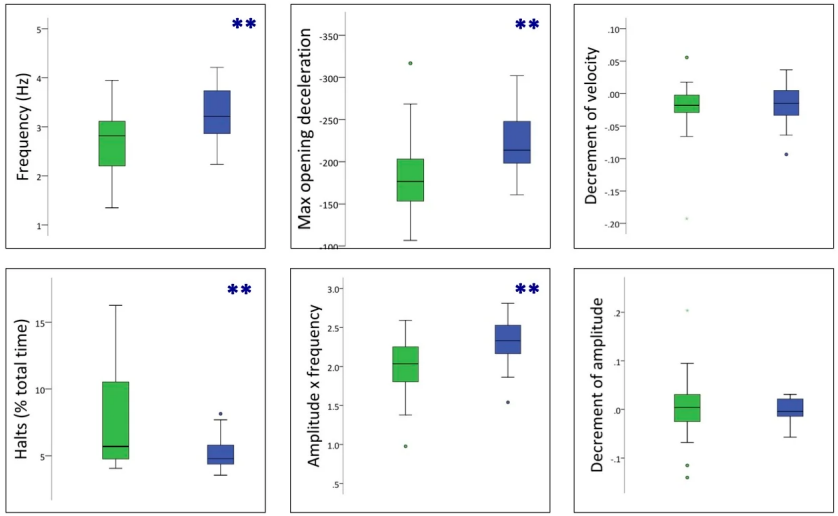


**Figure 2:** Boxplots demonstrating kinematic measures for dystonia (green) and healthy control (blue) groups. Box = interquartile range (IQR); whiskers = highest and lowest values within 1.5 x IQR; band inside box = median. Significant results ($p < 0.05$) denoted by **.

**Conclusions:** The deficits in sequential movement observed in dystonia have a different character to those of Parkinson's disease. Subjects with dystonia produce less frequent repetitive actions, with a greater tendency to halt mid-task. Overall speed of movement, as captured by the product of amplitude and frequency, is reduced in dystonia. This accords with previous kinematic research. But dystonic slowness does not significantly affect amplitude, and the decremental tendency and loss of rhythmicity of parkinsonian bradykinesia is not present. Our findings are probably explained by altered agonist-antagonist muscle activation in dystonia, which is interfering with the sequencing and initiation of patterned movements.

References:
1. Berardelli A, Rothwell JC, Hallett M, Thompson PD, Manfredi M, Marsden CD. The pathophysiology of primary dystonia. Brain 1998; 121: 1195 - 1212.
2. Haggstrom L, Darveniza P, Tisch S. Mild parkinsonian features in dystonia: literature review, mechanisms and clinical perspectives. Parkinsonism Relat Disord 2017; 35:1–7.