Objective Quantification of Wrist and Finger Spasticity: An Alternative to Current Clinical Measurements: A Commentary

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hyper-excitability of the stretch reflex…' [2]. While dystonia/spasms refer to spontaneous contractions that are not induced by muscle stretch [1]. Contractures and spasticity are common following brain injury and the incidence depends on a number of factors including (but not limited to) diagnosis, part of the body, lesion severity and amount and type of training post injury. At least 52% of stroke [3], 66% of spinal cord injured [4] and 56% of multiple sclerosis [5] patients develop at least one contracture (depending on contracture definition) and at least 17% of stroke [6], 67% of spinal cord injury [7] and 84% of multiple sclerosis [8] patients develop spasticity. These can be a significant burden to patients causing pain, falls and limiting the ability of the patient to perform activities of daily living [3-6]. Although spasticity and contracture are very different phenomena and require different medical management, they are often difficult to distinguish and separate. Using the main current clinical measurements of spasticity - the Ashworth (and Modified Ashworth) and Tardieu scales - it is difficult to separate contracture from spasticity. Despite this, these clinical measures are easy to use, widespread and low cost and hence remain in the clinical setting throughout the world.

Due to the problems associated with the clinical measurement and subsequent quantification of spasticity devices have been constructed to objectively quantify reflex and non-reflex stiffness over the wrist/fingers [9,10], knee [11] and ankle [12-16]. Although these devices have taken positive steps towards the quantification of the different elements of stiffness to be clinically routine they must fulfil a number of requirements without sacrificing the accuracy of the measurements. These need to be accepted by clinical staff, cost effective, portable (and small), time efficient, easy to understand and easy to use. A recently developed device called the Neuroflexor™, has been created and used to measure the stiffness components of the wrist and fingers, and could fulfil these requirements [9,17,18]. The Neuroflexor™ has been used to quantify the elastic, viscous and reflex components of stiffness about the wrist/fingers and can separate these components without the use of EMG electrodes ([17] for picture of the device). The arm is rested on the device such that the elbow is at 90 degrees and the shoulder at approximately 45 degrees. The forearm is pronated and the fingers are placed on a force plate with metacarpal heads placed on a line on the force plate, the distal edges of the malleoli of the wrist are placed in line with the fulcrum and the arm and fingers are strapped into the machine to ensure minimal movement. The machine parameters and basic measurements (such as height and weight) are programmed into the computer. Following this, five slow stretches (5 deg/s) and 10 fast stretches (236 deg/s) are applied. The computer/algorithm immediately calculates the force of the elastic, viscous and reflex components of stiffness providing a real-time view of the amount of stiffness. If the patient has spontaneous contractions, trials can be removed as required. Due to its simplicity of use a device such as this is an attractive option for the measurement of the stiffness components in the clinic. As there is a standardisation of the velocity and placement of the hand it is possible to measure the hand with the same settings over time by the same or different clinician. This removes the subjectivity that occurs when using the current clinical measures (which are based on feel).

The algorithm uses force values from both stretches to calculate the stiffness components (and described in detail in [9]). The force profile of the slow stretch is used to calculate the elastic component. This

measures the stiffness of the tendons, muscles and joint capsules etc. As there is non-linear stiffness at the end of the stretch, the force of the elastic component is measured 1 second following the end of the slow stretch. The viscosity component is calculated using the fast stretch. This describes the force created by the 'sliding muscle fibres' [9]. Firstly, the inertia of the hand must be calculated which is the mass of the hand (0.6 × body mass) multiplied by the acceleration. In addition to this, the mass of the platform is also included in the model and the angles the hand and platform make relative to the gravitational force are considered. The viscosity of the muscle is largest while the hand is accelerating to the required velocity. The initial acceleration force will be comprised of the inertia of the hand and the viscosity of the muscle. The inertia of the hand is subtracted from the total force and the initial viscous force component remains. This provides the basis to calculate the late viscous force component which is 20% of the force of the initial viscous force component. The reflex force component is calculated at the end of the extension stretch. This is the force created by the reflex following the extension stretch. The reflex component is the residual of the total force at the end of the stretch subtracted by the elastic component and late viscous component.

The simple algorithm is therefore able to calculate the viscous, elastic and reflex stiffness using the force profile during (and following) the slow and fast stretches. Due to its ease of use it would be possible to use clinically. Additionally, it will be able to assess the efficacy of treatments for stiffness and spasticity and allow clinicians to ascertain the 'real' effect of their treatments. Although the Neuroflexor™ is a promising advancement in the measurement of the components of stiffness there are some potential draw backs to its use. The variability of the measurement is quite large [17] and therefore it is difficult to observe a 'real change' in elastic, viscous or reflex stiffness. When botulinum toxin was applied to the flexor carpi radialis, flexor carpi ulnaris, flexor digitorum profundus and/or the flexor digitorum superficialis only seven patients showed a reduction in reflex stiffness beyond the variability of the measurement (although 17 patients showed a reduction in reflex stiffness) [18]. Although this may appear to invalidate the NeuroFlexor™ it should be noted that the amount of botulinum toxin injected to the target muscles was low (≤ 100 units) in 14 patients and as botulinum toxin has a dose dependent reduction in reflex stiffness it may have been too low to observe measureable reductions in reflex stiffness. Additionally, the patients were not naïve to botulinum toxin which reduces its effect. Although the major muscles for causing reflex stiffness were treated with botulinum toxin, as there are a number of muscles that cross the wrist joint it is likely that all of these will contribute in some way to the stiffness over the joint. When the authors applied ischemia to the upper limb (that causes a reduction in the reflex excitability) the reflex stiffness was reduced [9] which, similarly to the reduction in stiffness observed following botulinum toxin administration, indicates that the device can distinguish between stiffness components. However, due to the lack of a 'gold standard' for measuring stiffness and spasticity it is difficult to ascertain the accuracy of the device.

Other possible issues with the device are that patients must have at least 50 degrees of range of wrist extension/flexion [18]. This automatically excludes the patients with the worst contractures of the wrist and fingers and therefore might limit the clinical use of the device. The algorithm calculates the reflex stiffness as the residual of the total stiffness (i.e. the reflex stiffness is calculated once the viscous and elastic components have been subtracted from the total stiffness) and therefore any errors of measurement in the preceding force components will manifest as an error of the reflex stiffness component. The lack of EMG electrodes means that it is not possible to observe the pre-contraction status of the patients. Large spontaneous contractions can be visually observed and subsequently removed however, smaller non-stretch dependent spontaneous contractions [1] (which are very different from the viscous, elastic and neural stiffness described by the algorithm) may be present but not visually apparent. These spontaneous contractions will be difficult to detect and could cause