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Evaluation of Spasticity in Children With Cerebral Palsy Using Ashworth and Tardieu Scales Compared With Laboratory Measures

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Abstract

The content validity of the Tardieu Scale and the Ashworth Scale was assessed in 27 independently ambulant children with cerebral palsy (gender: 17 males, 10 females; age: 5–9 years; Gross Motor Function Classification: level I and II). Ashworth and Tardieu Scale scores and laboratory measures of spasticity and contracture were collected from the plantarflexor muscles by 2 examiners who were blinded to the results. The Tardieu Scale was more effective than the Ashworth Scale in identifying the presence of spasticity (88.9%, kappa = 0.73; P = .000), the presence of contracture (77.8%, kappa = 0.503; P = .008) and the severity of contracture (r = 0.49; P = .009). However, neither scale was able to identify the severity of spasticity. The Tardieu Scale can provide useful information in children with cerebral palsy because it differentiates spasticity from contracture. However, a more comprehensive clinical method of testing neural and non-neural contributions to impairments and function is needed.

Keywords

spasticity, contracture, cerebral palsy, Ashworth Scale, Tardieu Scale

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Cerebral palsy (CP) is a heterogeneous collection of nonprogressive motor disorders of the developing brain that can occur in the antenatal or postnatal period up to the age of 2 years.[1] The most common type of cerebral palsy, in terms of motor control impairment, is spastic cerebral palsy, which affects a large proportion of this population.[2] Spasticity is described as a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as 1 component of the upper motor neuron syndrome.[3] Clinically, it is typically assessed by evaluating the resistance to imposed passive movement when the limb is moved through the full available range of movement.[4] However, the force opposing the movement reflects not only reflex-mediated neuronal activity, but also the passive mechanical properties of the musculotendinous unit and its related connective tissues.[5] This can be of particular importance, given the findings of a recent study which reported that the passive stiffness of the calf muscle is almost 3 times as great in ambulant children with cerebral palsy compared with typically developing children.[6] Irrespective of which element is the principal opponent to movement, both neural and non-neural factors need to be considered when assessing the resistance to passive movement in children with cerebral palsy.

Currently, the original or modified Ashworth and Tardieu Scales appear to be the most common tools used in clinical practice to detect and quantify spasticity in children with cerebral palsy.[7] However, not only is reliability of both scales reported to be low in such children,[7,8] but the validity of the Ashworth Scale is also unclear and has not been studied in these children.[9] There is concern that this scale cannot be a precise measure of spasticity because the method of testing does not comply with the accepted definition of spasticity; that is, it does not take into account the velocity of limb movement and muscle stretch, nor does it distinguish between neural and non-

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neural causes of resistance to passive movement.[4] The Tardieu Scale appears to be more closely linked to the accepted definition of spasticity, as it takes into account the velocity involved in assessment of resistance to passive movement at both slow and fast speeds.[10] However, this scale has limitations[11] and more studies are needed to clarify its validity.[10] Limitations to research on this topic include the lack of an ideal, or gold standard, against which to make comparison.

Several recent studies have recommended using a combination of electromyography (EMG) and biomechanical measurements as a more accurate method of evaluating spasticity.[4,12,13] Recently, Patrick and Ada[14] have used this combination to evaluate the Tardieu Scale as a measure of spasticity after stroke, testing its content validity and comparing it with the Ashworth Scale. They concluded that, in an adult stroke population, the Tardieu Scale was more accurate than the Ashworth Scale, because it differentiated spasticity from contracture. In the only study specific to children with cerebral palsy, the validity and test–retest reliability of the Tardieu Scale were examined.[15] The scale demonstrated a significant ability to detect change in spasticity following botulinum toxin type A injection into the lower limb adductor muscles with good test–retest reliability.

In practice, the gastrocnemius is the muscle most often treated for spasticity in cerebral palsy.[16] The validity of the Tardieu Scale in testing the ankle plantarflexors (gastrocnemius and soleus) of children with spastic cerebral palsy has not, however, been tested. The purpose of this study was to investigate and compare the content validity of the Tardieu Scale and the Ashworth Scale in children with cerebral palsy, comparing them with a combination of EMG and biomechanical measurements of spasticity and contracture. The specific research questions were: (a) Does the Tardieu Scale identify the presence of spasticity more effectively than the Ashworth Scale in children with cerebral palsy? (b) Does the Tardieu Scale reflect the severity of spasticity more effectively than the Ashworth Scale in children with cerebral palsy? and (c) Does the Tardieu Scale identify the presence and reflect the severity of contracture in children with cerebral palsy?

Methods

Study Participants

Participants were recruited through the Physical Disability Service of The Children's Hospital at Westmead, Australia. A sequential sample of convenience was chosen, consisting of 27 independently ambulant children with cerebral palsy, and including 17 males and 10 females. Their average age was 7 (standard deviation [SD], 1.9) years, height 1.2 (SD, 0.12) m, and their average body mass was 25.1 (SD, 6.7) kg. Participants were consecutively selected based on inclusion and exclusion criteria, and their willingness to participate.

Inclusion criteria for children with cerebral palsy were: (a) diagnosed with spastic cerebral palsy affecting 1 or both lower limbs and (b) able to walk independently on level ground according to the Gross Motor Function Classification System[17] levels I and II. Exclusion criteria were: (a) cognitive problems that could hinder communication or cooperation; (b) severe affective or psychiatric impairments; (c) any unrelated musculoskeletal impairment or past surgical intervention (eg, arthrodesis, tendon lengthening dorsal rhizotomy) that might interfere with ankle joint movement; and (d) receiving systemic anti-spasticity medications or had received phenol and/or botulinum toxin type A injections in the calf muscles within the previous 5 months.

Study Design

A cross-sectional, analytical study was undertaken, in which the 2 clinical scales were compared against a reference standard derived through laboratory measures of spasticity and contracture. The 2 examiners were physiotherapists with at least 5 years' experience in pediatric rehabilitation and in the assessment of contractures and spasticity. One examiner (JL) assessed the ankle plantarflexors of each participant using the Ashworth Scale and the Tardieu Scale. The other examiner (AA) assessed plantarflexor contracture and spasticity of each participant using the laboratory measures. The 2 examiners were blinded to the results of the other measures during the measurement session. The order of the measures was randomized, and all testing procedures were completed in a single session.

The study was approved by the Human Research Ethics committees of the Children's Hospital at Westmead and the University of Sydney. Informed consent was obtained from each child's parent/guardian.

Outcome Measures

Outcomes were collected using 2 clinical scales, the Ashworth Scale and the Tardieu Scale and a laboratory measurement. For all measures, each participant was examined lying supine on a couch in a relaxed position with the legs extended. The foot of the affected (in case of hemiplegia) or more affected (in case of diplegia) was tested.

Ashworth Scale. The Ashworth Scale consists of a 5-point rating, ranging from 0 to 4, which measures resistance to passive movement.[18] For this study, spasticity was quantified using the criteria for grades 0 to 4 outlined by Ashworth[18] and was determined to be present in ankle plantarflexors if the score was ≥ 1.

Tardieu Scale. The Tardieu Scale[8] accounts for the velocity-dependent nature of spasticity by passively stretching the muscles at different speeds. It is a 6-point rating scale for describing the quality of muscle reaction (X). The point in the joint range in which a ''catch'' or clonus was felt during a quick stretch of the muscle at a velocity denoted as V3 and defined as fast as possible determined the angle of muscle response (R1); the angle of full range of motion (R2) was equivalent to the passive range of motion when the muscle is stretched at a speed denoted as V1 and defined as slow as possible. These 2 angles were measured using a hand-held goniometer relative to the neutral position or resting anatomic position of the ankle joint. The difference between the angle of muscle response (R1) and the angle of full range of motion (R2) was taken to indicate the relative contribution of spasticity versus contracture. A large difference between the angle of muscle response (R1) and the angle of full range of motion (R2) indicates more spasticity whereas a small difference indicates more contracture.[19]

Spasticity was quantified according to the quality of muscle reaction (X) for grades 0 to 5 during the fast as possible stretch (V3) and considered present if the muscle reaction was ≥ 2. Contracture was

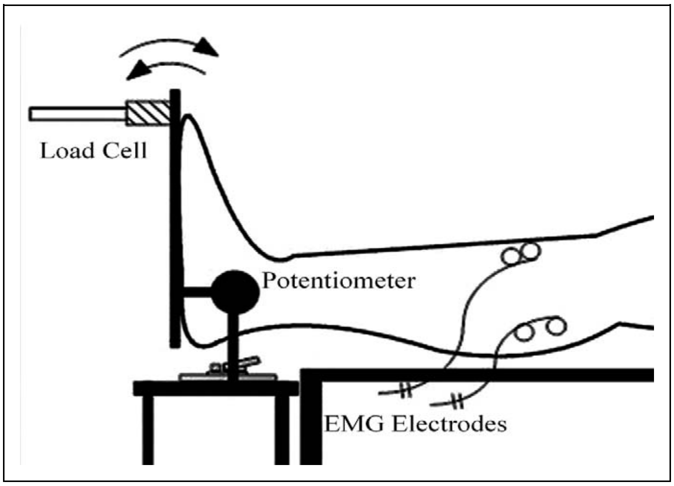

Figure 1. A schematic of the experimental set-up. Three signals were collected: a potentiometer measured ankle joint angle, a load cell measured applied torque, and electrodes measured muscle activity.

defined as present if the angle of full range of motion (R2) during the slow stretch (V1) was less than 10 degrees of ankle dorsiflexion.

Laboratory Measures. The laboratory measures were collected using a specially constructed ankle measurement device,[20] consisting of a footplate hinged to a support bracket for the lower leg, with a rotary potentiometer (Model 157, RS Australia, Sydney) aligned with the ankle, to measure angular displacement (Figure 1). The footplate and axis of rotation were matched to the dimensions of the child. A 450N load cell (XTRAN S1W, Applied Measurement Australia Pty, Oakleigh Vic., Australia) was attached perpendicular to the footplate to measure resistance to movement. A handle was attached to permit manual oscillation of the foot plate. Applied torque values were calculated from the product of applied force and the perpendicular distance from the point of application of the force to the axis of rotation of the footplate.

Each participant's foot was placed in the ankle apparatus and positioned, by visual approximation, such that the point midway between the lateral and medial malleolus in the sagittal plane was aligned with the axis of rotation of the device. The foot was secured with Velcro straps, the knee was placed in an extended position, and light pressure was applied by the researcher's hand over the thigh near the knee to ensure that knee position was maintained. The calf was free of contact and clear of all surfaces and structures. Joint displacements into dorsiflexion were taken as positive and those in the plantarflexion direction as negative. An ankle angle of 90 degrees (plantigrade) was considered to be the neutral position and defined as zero.

Electromyographic activity of the soleus, medial gastrocnemius, and tibialis anterior muscles was recorded simultaneously with ankle rotation, using a telemetric 16 channel electromyography unit (Telemyo 2400R G2 system, Noraxon, Arizona) with a sampling rate of 3000 Hz. We used disposable, self-adhesive silver/silver chloride bipolar surface electrodes (Kendall Medi-Trace Mini 130 Foam ECG Electrodes, Neurotronics, Randwick, NSW, Australia), placing pairs of electrodes parallel to the muscle fiber direction with minimal inter-electrode distance. Before electrode placement, we cleaned the skin with isopropyl alcohol. The location of electrodes was based on contemporary recommendations.[21] Electromyography acquisition enabled monitoring of muscle activity during the test. All children were instructed to keep their legs relaxed and to avoid assisting or resisting the motion during the sinusoidal rotation. The same experimental set-up was used for measurement of both contracture and spasticity.

Maximum passive joint excursion was measured. Participants were asked to relax with the affected limb positioned in the frame, and the electromyography recording was displayed on the computer screen to ensure that participants remained relaxed. The ankle was slowly moved from full plantarflexion to full available dorsiflexion. The angle at 4.6 Nm of applied torque, half of the torque value used in adults,[14] was recorded. Contracture was operationally defined for the laboratory measure as an angle less than 10 degrees of dorsiflexion at 4.6 Nm.

Stretch-induced muscle activity was measured. Participants were asked to relax with the foot positioned in the frame and, before stretching, the electromyography recording was displayed on the computer screen to ensure that muscles were electronically silent. The examiner then moved the ankle passively through the range at 2 velocities (fast and slow).

Spasticity was determined as present if, on visual examination, a clear burst of electromyographic activity was time locked to the maximum velocity of ramp stretch in at least 3 trials. To quantify the severity of the spasticity using the laboratory measure, electromyographic activity root mean square values were calculated and normalized against maximum isometric voluntary contraction. The maximum isometric voluntary contraction was calculated from the best of 3 maximum efforts to push the foot against the static footplate for a duration of 2 seconds.

Statistical Analysis

There were 3 discrete yes/no measures of spasticity based on the criteria defined for the Ashworth Scale, Tardieu Scale, and laboratory measurement. There were 2 discrete yes/no measures of contracture based on the laboratory definition (Angle <10 degrees at 4.6 Nm) and Tardieu Scale definition (the angle of full range of motion [R2] during the slow as possible velocity stretch [V1] was less than 10 degrees of ankle dorsiflexion).

All statistical analysis was carried out using SPSS version 16. The percentage exact agreement (PEA) between laboratory and clinical measures was calculated to examine the ability of the Ashworth and Tardieu Scales to identify the presence of spasticity and/or contracture. The significance of the differences in percentage exact agreement was determined using Fisher's exact P-value of the chi-square statistic to account for small numbers in some cells. Kappa (K) is interpretable as a measure of agreement beyond that due solely to chance. Zero represents random agreement, and 1.00 represents perfect agreement.[22] K statistics were categorized as poor agreement when lower than 0.20, as fair between 0.21 and 0.40, as moderate between 0.40 and 0.60, as good between 0.61 and 0.80, and as very good agreement above 0.80.[23]

To examine the ability of both scales to reflect the severity of spasticity, Pearson product-moment correlation coefficient was used. Critical values for significance were set with $P < .05$. To examine the ability of the Tardieu Scale to reflect the severity of contracture, the interval measures of contracture were correlated with the interval laboratory measures of spasticity and contracture, using the Pearson correlation coefficient.

Results

The results of the laboratory measure indicated that 21 of 27 participants had spasticity of the ankle plantarflexors; the

Table 1. Number of Participants out of Total of 27 Participants With Spasticity and Contracture as Detected by the Different Measurements

Method of measurement	Spasticity	Contracture
Laboratory measurements	21 of 27	17 of 27
Ashworth Scale score	26 of 27	-
Tardieu Scale score	18 of 27	19 of 27

Table 2. The Results of Percentage Exact Agreement (PEA) and Pearson Product-Moment Correlation Coefficient (*$P < .05$) Between Each Clinical Scale and Laboratory Measurement for Spasticity and Contracture

	Spasticity		Contracture	
Comparison	PEA	Pearson correlation	PEA	Pearson correlation
Tardieu Scale vs. Lab Measurement	88.9% k = 0.73 P = .000*	r = −0.17 P =.4	77.8% k = 0.503 P = .008*	r = 0.49 P = .009*
Ashworth Scale vs. Lab Measurement	81.5% k = 0.24 P = .057	r = 0.09 P =.7	-	-

Ashworth Scale identified 26 of 27 with spasticity, whereas the Tardieu Scale identified 18 of 27 (Table 1). Therefore, the Ashworth Scale gave 5 false positive and the Tardieu Scale 3 false negative results.

In identifying the presence or absence of spasticity (Table 2), the percentage exact agreement between the Tardieu Scale and the laboratory measure of spasticity in the ankle plantarflexors was 88.9%, showing a significantly good agreement ($K = 0.73$; $P = .000$). Whereas, the percentage exact agreement between the Ashworth Scale and the laboratory measure was 81.5%, showing a nonsignificant fair agreement ($K = 0.24$) of this scale to correctly identify spasticity ($P = .057$).

The relationships between clinical and laboratory measures (Table 2) were used to identify the severity of spasticity. There was no significant relationship between the Ashworth Scale and the peak stretch-induced electromyographic activity (r = 0.09; $P = .7$). There was also no significant relationship between peak stretch-induced electromyographic activity and the quality of muscle reaction (X) (r = −0.17; $P = .4$) nor of the difference between the angle of full range of motion (R2) and the angle of muscle response (R1; R2-R1 value: dynamic component of spasticity) using the Tardieu Scale (r = 0.26; $P = .3$). However, there was a significant negative relationship between the angle of muscle response (R1) and peak stretch-induced electromyographic activity (r = −0.48; $P = .03$). That is, an early catch angle was accompanied by higher electromyographic activity.

In identifying the presence or absence of contracture (Table 2), there was 77.8% exact agreement between the Tardieu Scale and the laboratory measure of contracture ($K = 0.503$; $P = .008$). In terms of the severity of contracture, ie, the relationship between Tardieu Scale and laboratory measures of contracture, there was a significant relationship between the angle of full available passive dorsiflexion range of movement (R2) during the slow velocity stretch (V1) using both the Tardieu Scale and the laboratory measure of contracture (r = 0.49; $P = .009$).

Discussion

Our study was informed by the work of Patrick and Ada[14] whose investigation of the validity of the Ashworth and Tardieu Scales in detecting and differentiating spasticity and contracture in adults following stroke indicated that the Tardieu Scale was better able to differentiate elements of movement restriction caused by neural and non-neural phenomena. We believe it is important to replicate this work in children with cerebral palsy as both the pathological processes and the potential compliance of these children to testing might be expected to differ from an adult population.

Spasticity (velocity-dependent stretch reflex activity) in the plantarflexors was present in most of the children during the fast velocity stretch. Our results showed that although the Tardieu Scale was better than the Ashworth Scale in identifying the presence of spasticity, it failed to identify spasticity 11% of the time when compared with laboratory measures of stretch-induced electromyographic activity. In contrast, the presence of spasticity was overestimated by the Ashworth Scale 19% of the time. Only 20% of the overestimate could be accounted for by the presence of contracture. The lack of agreement between the Ashworth Scale and laboratory measures of spasticity in the current study was similar to the results obtained in the Patrick and Ada study[14] which revealed that the Ashworth Scale is unable to distinguish between neurological and non-neurological components of resistance to passive movement, because it does not take into account the speed of muscle stretch. Our study lends further support to this finding, with more than 62% of our study population showing contracture in the calf muscles.

Our study showed that neither scale was able to determine the severity of spasticity; a finding which is in line with previous studies of the Ashworth Scale.[13,14,24] However, while the previous investigation of the Tardieu Scale in adult stroke survivors[14] found a moderate relationship between the grade of the quality of muscle reaction (X) during the fast velocity stretch (V3) and peak stretch-induced electromyographic activity (r = 0.62; $P = .01$), our results did not concur with this. This can be because of our sample, which did not include children with more than level 2 on the Gross Motor Function Classification Scale. Although no significant relationship between peak stretch-induced electromyographic activity and the grade of the quality of muscle reaction (X) was found, the angle of muscle response (R1) was negatively correlated with the peak stretch-induced electromyographic activity suggesting that recording the angle of muscle response (R1) can give insights into spasticity severity. It is, therefore, important for clinicians to record

the angle of muscle response (R1) as well as the quality of muscle reaction (X).

The limitations of the clinical scales in quantifying spasticity are well documented.[13,14,24-26] However the advent of instrumented devices such as the 1 used in this study and others such as the Montreal Spasticity Measure[25] which quantify reflex responses does not necessarily resolve all issues. There is ongoing debate as to which physiological parameter is most appropriate to measure.[27] Measurement of the gain[28-30] and threshold of the reflex responses[25,26] have both been used. The findings that both stretch reflex threshold and gain increase with velocity of stretch suggest that both are relevant to the definition of spasticity.[26,28] Calota and colleagues[25,26] argue that threshold is the conceptual unit of measure of spasticity because it can be altered by descending pathways, including cortico-spinal systems with or without a significant enhancement of reflex gain. They report measurement of threshold as a more representative measure for subjects with moderate to high spasticity.[26] This view is based on data collected in the upper extremities of adults after stroke. Whether threshold is the most appropriate measure in children with cerebral palsy or in the lower limbs warrants further investigation.

Our results show that the Tardieu Scale is able to identify the presence and severity of contracture, enhancing its clinical usefulness. However, like the Ashworth Scale, it grades spasticity according to the resistance to passive movement,[4] a finding that can be confounded by changes in non-neurally mediated muscle stiffness. It is, therefore, important to establish the relative contribution to function of increased muscle stiffness and contracture as well as spasticity. Our previous work[6] has shown significant changes in the passive muscle stiffness in children with cerebral palsy, suggesting that passive muscle stiffness should be measured in these children.

In conclusion, our study noted the limitations of both the Tardieu and Ashworth Scales in children with cerebral palsy. The Tardieu Scale appears to be preferable because it can differentiate between spasticity and contracture. However, given that spasticity, contracture, and increased passive muscle stiffness are all complex phenomena coexisting in cerebral palsy and affecting functional motor performance, a more comprehensive method of testing these phenomena is necessary for clinical practice. A simple instrumented system such as the 1 used in this study could be developed for clinical use and would enable objective testing of both neural and non-neural (muscle) contributions to impairment and to functional limitations.

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Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the authorship and/or publication of this article.

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Contributors

Adel A. A. Alhusaini, Catherine M. Dean, and Jack Crosbie are designated as first authors as they contributing equally to this work, Roberta B. Shepherd and Jenny Lewis are designated senior investigators who provided support and mentorship necessary for the success of the work.

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