



DOES THE DUNCAN-ELY TEST PREDICT ABNORMAL ACTIVITY OF THE RECTUS FEMORIS IN STROKE SURVIVORS WITH A STIFF KNEE GAIT?

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Objective: To determine the diagnostic value of the Duncan-Ely test in predicting abnormal rectus femoris activity during gait in stroke survivors walking with a stiff knee gait.

Design: Cross-sectional diagnostic study.

Subjects: A total of 95 patients with chronic stroke.

Methods: During physical examination, the Duncan-Ely test was performed and scored. Surface electromyography of the rectus femoris was then recorded during dynamic gait. To determine the diagnostic value, the results of the Duncan-Ely test and surface electromyography recordings (gold standard) were compared.

Results: The Duncan-Ely test had a sensitivity of 73%, a specificity of 29%, a positive predictive value of 60%, and a negative predictive value of 42%. The area under the curve was 0.488 (95% CI 0.355–0.621, $p = 0.862$), showing that the Duncan-Ely test is not better than random guessing.

Conclusion: The Duncan-Ely test has no predictive value for determining abnormal activity of the rectus femoris during gait. Using this test can lead to incorrect identification of abnormal rectus femoris activity, which might hamper the selection of optimal treatment options. We recommend stopping use of the Duncan-Ely test to predict rectus femoris overactivity during swing, and instead use surface electromyography.

Key words: stroke; stiff knee gait; rectus femoris; spasticity; Duncan-Ely test; diagnostic.

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Stiff knee gait (SKG) is commonly observed in individuals with spastic paresis as a result of an upper motor neurone lesion, such as cerebral palsy, multiple sclerosis, traumatic brain injury, or stroke. SKG is characterized by a diminished knee flexion during swing and can result in problems with foot clearance. Although the exact mechanisms remain unclear and appear to be multifactorial, abnormal activity of the rectus femoris during the swing phase is a frequently mentioned cause of SKG (1–4).

LAY ABSTRACT

After stroke, some patients have difficulty bending their knee when they swing their leg forwards. This can be the result of the rectus femoris muscle being too active. It is important to know whether this is the case. If the rectus femoris is too active, we can select a treatment option that aims to reduce its activity. Healthcare professionals often use the Duncan-Ely test, to test for overactivity of the rectus femoris. However, it is not known whether this test is reliable. This study therefore compared the Duncan-Ely test with a measurement in which the activity of the rectus femoris was recorded during walking. No correlation was found between the score of the Duncan-Ely test and the activity of the rectus femoris during walking. We conclude that healthcare professionals should no longer use the Duncan-Ely test to assess overactivity of the rectus femoris, but should replace it with surface electromyography.

Different treatment options for SKG (5) are used in clinical care, and mainly focus on influencing the abnormal activity of, or the force produced by, the rectus femoris. These options include chemodenervation of the rectus femoris (6, 7) and a rectus femoris transfer (8, 9). Chemodenervation is a technique in which a pharmacological compound, such as botulinum toxin, is used to paralyse a muscle or a group of muscles (10). The indication for chemodenervation or rectus femoris transfer treatment is often based on abnormal activity of the rectus femoris in the pre-swing or swing phase of the gait cycle.

In clinical practice, there are 2 options available for establishing abnormal rectus femoris activity: surface electromyography (sEMG) and the Duncan-Ely test. The generally accepted gold standard is sEMG of the rectus femoris during dynamic gait analysis (11). However, sEMG measurements require expensive measurement equipment and specific expertise, which limits its applicability in daily clinical practice. The second option is the Duncan-Ely test (12, 13), which is part of a routine clinical examination of muscle tone. This test is performed with the patient lying in a prone position and the examiner passively flexing the knee rapidly. This clinical test does not require any measurement equipment and is easy to perform, making it suitable for daily clinical practice. However, whether

the Duncan-Ely test is a useful test to predict abnormal rectus femoris activity during the swing phase of gait is dependent on how the outcomes of this test relate to the gold standard. Using a test with a limited diagnostic value can lead to the incorrect selection of patients and treatment options for SKG. To the best of our knowledge, comparison of the Duncan-Ely test with sEMG has not been performed in a group of stroke survivors.

The aim of this study was therefore to determine the diagnostic value of the Duncan-Ely test to assess how accurately the test can predict abnormal rectus femoris activity during gait in stroke survivors with a SKG. In this light, this study can also contribute to the discussion as to whether a clinical test to establish abnormal activity or spasticity performed on the bench in a static (relaxed) position can provide information about how muscles act in dynamic situations.

METHODS

Design and participants

Participants were recruited from Roessingh, Centre for Rehabilitation in Enschede, The Netherlands. Inclusion criteria were: chronic stroke survivors (at least 6 months post-stroke), age over 18 years, able to walk independently with or without walking aids, diminished peak knee flexion in swing ($>45^\circ$) (14) as established by video observation and written informed consent.

Exclusion criteria were: botulinum toxin injection in the rectus femoris in the 5 months prior to inclusion in the study, rectus femoris transfer surgery, length of the rectus femoris $<65^\circ$, presence of joint range of motion (ROM) limitation that impedes walking, or neurological problems other than stroke. The study was designed as a cross-sectional trial with a single measurement and is a part of the randomized controlled trial in which the efficacy of a botulinum toxin injection into the rectus femoris was investigated (trial NL2052 (NTR2169)), and is approved by the medical ethics committee (MEC Twente).

Procedure

During physical examination, the length of the rectus femoris was measured. This is the normal length test of the rectus femoris, in which the examiner checked for a fixed contracture of the rectus. The patient was lying in a prone position and the examiner passively flexed the knee slowly until hip flexion appeared. The knee joint was flexed until hip flexion appeared and the knee angle at this time-point was measured with a goniometer. The Duncan-Ely test was subsequently performed twice in a standardized manner (12, 13). While the patient was lying in a prone position in a relaxed state, the examiner passively flexed the knee fast (at a speed similar to the limb falling under gravity) over the total length of the rectus femoris. The test was considered positive if the examiner perceived resistance (perceived resistance) and/or the patient flexed the ipsilateral hip (occurrence of hip flexion) (12, 13). The perceived resistance was scored using the modified Ashworth scale (MAS). The occurrence of hip flexion was visually inspected and manually checked by the examiner. The lowest measured score of the

MAS was noted in case of doubt, which is in line with the recommendation of Fleuren et al. (15). The test was positive if the score on the MAS was ≥ 1 and/or hip flexion occurred. It was scored negative when no increase in muscle tone was felt and hip flexion did not occur. Two well-trained examiners with substantial experience in physical assessments evaluated all participants prior to the gait analysis to ensure that they were blinded for the results of the sEMG evaluation.

Gait analysis

To obtain EMG of the rectus femoris during walking, participants completed 4 walking trials on a 10-m walkway at comfortable walking speed. During these walking trials, sEMG of the rectus femoris and vastus lateralis of the affected leg was measured. The gait pattern and sEMG were synchronously recorded using the Flamenco system (TMSi, Oldenzaal, The Netherlands), using 2 cameras (Basler scout high resolution scA1300–32gc GigE camera, 50 frame per s, Ahrensburg, Germany) in the sagittal and the frontal plane.

Participants were allowed to walk with an ankle-foot orthosis or walking aid if they used these in their daily life. The first 2 and last 2 strides of the 10-m walkway were discarded for sEMG analysis to exclude variations in gait speed caused by initiating and terminating gait.

EMG analysis

The electrodes (Kendall ECG H93SG, 42×24 mm Covidien, Mansfield, MA, USA) were placed on the rectus femoris and vastus lateralis according to the Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles (SENIAM) recommendations (16). SEMG was recorded with the Mobita sEMG device (TMSi, Oldenzaal, The Netherlands), with a bandwidth of 0–400 Hz and a 24-bit resolution. SEMG was recorded with a frequency of 1,000 Hz and band-pass filtered at 20–400 Hz. The noise level of this system was determined as $10 \mu\text{V}$, which was subtracted from the band-pass filtered signal. Subsequently, the sEMG signals were full-wave rectified and filtered with a second-order Butterworth 10-Hz low-pass filter with phase correction in order to create the sEMG linear envelope for each gait phase.

Detection of abnormal activity of the rectus femoris

A custom-made computerized algorithm was used to detect abnormal activity of the rectus femoris and to discriminate it from cross-talk activity of the vastus intermedius. The algorithm is based on the normal sEMG pattern of the rectus femoris and vastus lateralis (4). It compares the area under the curve (AUC) of the root-mean-square value (RMS) of the rectus femoris and vastus lateralis muscle activity. Rectus femoris activity was labelled abnormal when activity was seen in the second part of initial swing and/or in mid-swing. In case the AUC of the RMS in initial swing and/or mid-swing of the vastus lateralis was equal or higher compared with the rectus femoris activity, the sEMG signal obtained over the rectus femoris was considered to be the result of cross-talk from the vastus intermedius (17).

A stride could be scored as normal activity of the rectus, abnormal rectus femoris activity or cross-talk activity. For each patient a sample of 10 strides was randomly selected from the 4 trials, and scored according to the predefined categories. Of the 10 strides the dominating type of rectus femoris activation

Table I. Patients' characteristics

Characteristics	
Number of participants, <i>n</i>	94
Affected side (left/right)	45/49
Time after stroke, years, mean (SD) [Range]	6.9 (5.9) [2–21]
Men/women	65/29
Age, years, mean (SD)	57.0 (12.6)
Use of ankle-foot orthosis (yes/no)	36/58
Use of assistive device (yes/no)	34/60

SD: standard deviation.

allocated the patient in 1 of the 3 categories. In case there was no clear dominating pattern, the patient was scored as undefined.

Statistical analysis

To determine the diagnostic value of the Duncan-Ely test, sensitivity, specificity, positive and negative predictive value were calculated. This was done separately for the perceived resistance and for the occurrence of ipsilateral hip flexion. The receiver operating characteristic (ROC) curve and the area under the curve (AUC), which relates the outcome of the sEMG measurement and the Duncan-Ely test, were calculated using IBM SPSS Statistics version 19.0 for Windows (IBM Inc. Chicago, IL, USA).

RESULTS

Participants

A total of 95 stroke survivors participated in this study. For patient characteristics, see Table I. One patient was excluded from the study because no sEMG was recorded, due to technical problems.

Results categorization of sEMG activity rectus femoris

Based on the sEMG signal, 31 participants were categorized as having normal rectus femoris activity and 45 participants as having abnormal rectus femoris activity. Twelve participants were categorized as cross-talk activity and 6 participants as undefined. Because the individuals in the last 2 groups could not be categorized as having normal or abnormal rectus femoris activity, these 18 participants were not included in results of the analysis.

Table II. Perceived resistance during the Duncan-Ely test

	sEMG abnormal	sEMG normal	
Duncan-Ely ≥ 1 (perceived resistance)	33 (true positives)	22 (false positives)	Positive predictive value: 60% (33/55)
Duncan-Ely = 0 (perceived resistance)	12 (false negatives)	9 (true negatives)	Negative predictive value: 42% (9/21)
	Sensitivity: 73% (33/45)	Specificity: 29% (9/31)	76

sEMG: surface electromyography.

Results test characteristics of the Duncan-Ely test: perceived resistance.

Based on the Duncan-Ely test, 70 participants scored positive on the perceived resistance and 24 scored negative. As stated above, based on sEMG results, 76 individuals could be classified as having normal or abnormal activity. These 76 individuals were included in the analysis. The results for perceived resistance are shown in Table II.

The ROC curve is shown in Fig. 1. The area under the curve is 0.488 ($p=0.862$, 95% CI 0.355–0.621).

Results test characteristics of the Duncan-Ely test: occurrence of hip flexion.

During the Duncan-Ely test, there were 18 participants in whom hip flexion occurred and 68 in whom no hip flexion occurred. From 8 participants, the absence or presence of hip flexion during the Duncan-Ely test was not written down. These 8 participants were not included in the calculation. Of the remaining 86 participants, 68 could be classified as having normal or abnormal rectus femoris overactivity based on sEMG. These 68 participants were included in the analysis. The results

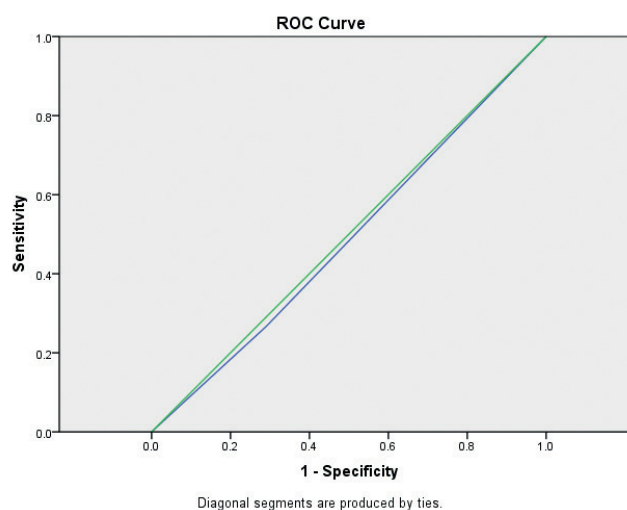


Fig. 1. Receiver operating characteristics (ROC) curve for perceived resistance during the Duncan-Ely test. Green diagonal reference line: indicates a worthless test. Blue: measured line.

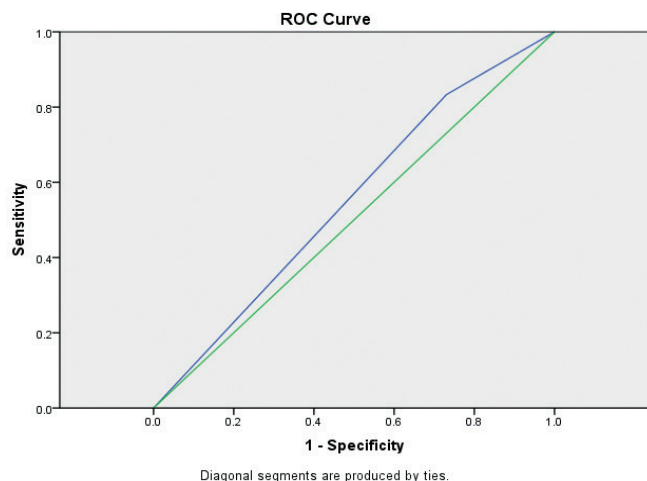


Fig. 2. Receiver operating characteristics (ROC) curve for occurrence of hip flexion during the Duncan-Ely test. Green diagonal reference line: indicates a worthless test. Blue: measured line.

for the occurrence of hip flexion are shown in Table III. The results of the ROC curve are shown in Fig. 2. The AUC is 0.551, $p=0.480$ (95% CI 0.408–0.695).

DISCUSSION

This study investigated the diagnostic value of the Duncan-Ely test for predicting abnormal activity of the rectus femoris during gait of stroke survivors with SKG. Test characteristics were calculated to investigate whether abnormal sEMG activity of the rectus femoris during the swing phase (gold standard) corresponds with a positive score on the Duncan-Ely test (diagnostic test). For perceived resistance during the Duncan-Ely test, this study showed a positive predictive value of 60.0% and a sensitivity of 73.3%. Looking at the occurrence of hip flexion during the Duncan-Ely test, the positive predictive value and sensitivity are considerably lower at, respectively, 50.0% and 16.7%. Based on the calculated tables it can be concluded that the Duncan-Ely test has no value in predicting abnormal muscle activity of rectus femoris during the swing phase of stroke survivors walking with SKG. This is also reflected in the ROC curve, which showed no correlation ($AUC=0.488$; $p=0.480$) between perceived resistance during the Duncan-Ely test and abnormal sEMG of the rectus femoris. There was also no correlation ($AUC=0.551$; $p=0.480$) bet-

ween the measurements of only the occurrence of hip flexion and the sEMG measurements. The AUC showed that the Duncan-Ely test is no better than random guessing.

The limited correlation between the Duncan-Ely test and the sEMG analysis might be explained by several factors. During the Duncan-Ely test, both the rectus femoris and the vastus intermedius, vastus lateralis and vastus medialis are stretched. Therefore, the Duncan-Ely test might not only test the velocity-dependent activity of the rectus femoris, but might also assess the velocity-dependent activity of these other muscles. It might therefore be difficult to distinguish between the rectus femoris and vastus intermedius, vastus lateralis and vastus medialis during the Duncan-Ely test.

That other muscles might be involved in a positive Duncan-Ely has been reported previously by Perry et al. (18). They concluded that the

Duncan-Ely test is not a specific indicator for the rectus femoris tightness or spasticity. In addition to an electromyographic response in the rectus femoris, the test can also provoke an electromyographic response in the iliacus in subjects with cerebral palsy. The fact that up to 3 muscles could be involved in a positive test might imply that the test is not able to distinguish between velocity-dependent activity of the rectus femoris, vastus intermedius, vastus lateralis and vastus medialis and iliacus. This could have reflected on all studied outcome parameters and might contribute to the limited correlation between the Duncan-Ely test and the sEMG of the rectus femoris. Furthermore, the fact that other muscles also result in a positive Duncan-Ely test could be one of the reasons that after a chemodenervation of the rectus femoris or rectus femoris release, the Duncan-Ely test is still positive in some participants (6, 19–22). Future research could focus on these points by investigating whether the correlation between the Duncan-Ely test and overactivity of any of the knee extensors, as measured by sEMG, is more sound.

Another explanation for the limited correlation between the sEMG and the Duncan-Ely test could be the fact that the Duncan-Ely test score is based on a subjective evaluation by the examiner. The score on the perceived resistance and the occurrence of hip flexion could be dependent on which examiner performs the

Table III. Occurrence of hip flexion during the Duncan-Ely test

	sEMG abnormal	sEMG normal	
Duncan-Ely ≥ 1 (Perceived resistance)	7 (true positives)	7 (false positives)	Positive predictive value: 50% (7/14)
Duncan-Ely = 0 (Perceived resistance)	35 (false negatives)	19 (true negatives)	Negative predictive value: 35% (19/54)
	Sensitivity: 16% (7/42)	Specificity: 37% (7/26)	68

sEMG: surface electromyography.

test and interprets the results differently. Furthermore the occurrence of hip flexion is difficult to observe, and rating is not standardized. In addition, the angular velocity with which the Duncan-Ely test should be performed is not standardized nor controlled. This could influence the score on the Duncan-Ely test, because it aims to measure the velocity-dependent response to passive movement. In case the angular velocity is higher, one might hypothesize that the response is also larger. Based on the findings of Lee et al. (23) we tried to standardize the angular velocity at a speed similar to or faster than the speed of the limb falling under gravity. Although we tried to standardize the angular velocity, speed it is not controlled, and can be different between examiners and patients, potentially leading to different scores in similar cases.

Furthermore, there may be a discrepancy between the knee angular velocity and knee range of motion during walking, and the knee angular velocity and knee range of motion applied during the Duncan-Ely test (24). In case the knee angular velocity and range of motion during the Duncan-Ely test is much higher than the knee angular velocity during walking, the velocity-dependent resistance of the rectus femoris during the Duncan-Ely test will also be higher. This discrepancy between the knee angular velocity executed on the bench and the knee angular velocity during functional walking can contribute to the reported differences between the results of the Duncan-Ely test and the results of the sEMG.

Besides these apparent shortcomings of the Duncan-Ely test itself, it is also disputable whether a clinical test to establish overactivity or spasticity performed on the bench in a static (relaxed) position can provide information about how muscles act in dynamic situations. In other words, it is debatable to which degree the results of a passive measurement of an impairment relates to the dynamic functional activity, such as walking (25–27). There are more studies supporting this notion. Dietz & Sinkjaer (28) suggest that there is a disparity between clinical assessment findings and how spasticity manifests during walking. In addition to the discussion about static vs dynamic test situations, there is also an influence of posture on the static test conditions. Yelnik et al. (29) and Perry et al. (30) reported that the spastic response varies greatly according to the position of sitting or standing. This is also the case when comparing the activity patterns of knee extensors and flexors during sitting and lying in different patient populations (31–33). In addition, the study of Lamontagne et al. (34) showed that spasticity at rest only weakly predicts spasticity during the stance phase of gait, which emphasizes the need for a locomotor-specific measure of spasticity. Finally, Non-

nekes et al. (35) stated that the presence or absence of spasticity observed during clinical examination often does not translate to muscle activity or overactivity observed by instrumented analysis of the gait, and suggested that sEMG is necessary to detect or confirm muscular overactivity during gait.

The finding that the Duncan-Ely test has no diagnostic value for predicting overactivity of the rectus femoris in stroke survivors walking with SKG has implications for clinical care as well as scientific research. In both fields, abnormal activity of the rectus femoris is usually quantified using the Duncan-Ely test. Because of its limited diagnostic value, using this test can lead to the selection of incorrect treatment options for SKG. Furthermore, using the Duncan-Ely test to establish rectus femoris activity might lead to the false identification of individuals with and without rectus femoris activity during walking. This, in turn, could lead to heterogeneous study population, which might have contributed to the variable results that have been published about the effect of treating abnormal rectus femoris activity (6, 7, 20, 36–39).

This study had some limitations. First, some assumptions were made in determining abnormal activity based on the sEMG. The algorithm contained assumptions that can give room for discussion. One example of this is the noise level value. To test the robustness of these assumptions, additional analyses were performed in which the noise level was changed to 8 and 12 μV . Varying the noise level did not change the conclusions of this study. Furthermore, 18 participants were excluded from the analysis, due to cross-talk activity or undefined sEMG. Exclusion of these individuals could have influenced the current results. We therefore performed a sensitivity analysis based on investigating whether our conclusion would change if we had included these 18 participants. This sensitivity analysis did not change the conclusion. When all 18 patients were included and categorized as normal activity rectus femoris sEMG, the AUC of perceived resistance was 0.511 ($p=0.856$). When all 18 patients were included and categorized as having abnormal activity rectus femoris, the AUC of perceived resistance was 0.474 ($p=0.682$).

Future research could investigate whether the findings of this study can be replicated when the signals from the rectus femoris are obtained using fine-wire EMG. Compared with sEMG, fine-wire is not susceptible for cross-talk activity from the vastus intermedius, and is therefore more precise.

Furthermore, this study raises additional information concerning the diagnostic performance of clinical tests for spasticity of other muscles that relate test results in a static position to the muscle activity in a dynamic

functional activity, such as walking. This might also be true for other clinical tests, which could be the subject of future research.

In conclusion, this study showed that the Duncan-Ely test has no diagnostic value for predicting abnormal activity of the rectus femoris in stroke survivors with SKG. Factors that may contribute to this conclusion are that the score on the Duncan-Ely test is a subjective assessment, that a static test is used to assess a problem occurring in dynamic situations, and that the Duncan-Ely test might also assess other knee extensors and hip flexors. We recommend stopping use of the Duncan-Ely test for this purpose, but instead using sEMG. Furthermore, this study confirms the notion that it is disputable whether a clinical test that aims to establish overactivity or spasticity performed on the bench in a static position can provide information about how muscles act in dynamic situations.

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REFERENCES

- Riley PO, Kerrigan DC. Torque action of two-joint muscles in the swing period of stiff-legged gait: a forward dynamic model analysis. *J Biomech* 1998; 31: 835–840.
- Piazza SJ, Delp SL. The influence of muscles on knee flexion during the swing phase of gait. *J Biomech* 1996; 29: 723–733.
- Goldberg SR, Anderson FC, Pandy MG, Delp SL. Muscles that influence knee flexion velocity in double support: implications for stiff-knee gait. *J Biomech* 2004; 37: 1189–1196.
- Perry J. *Gait analysis: normal and pathological function*. Thorofare, NJ: Slack, 1992.
- Tenniglo MJB, Buurke JH, Prinsen EC, Kottink AIR, Nene AV, Rietman JS. Influence of functional electrical stimulation of the hamstrings on knee kinematics in stroke survivors walking with stiff knee gait. *J Rehabil Med* 2018; 50: 719–724.
- Stoquart GG, Detrembleur C, Palumbo S, Deltombe T, Lejeune TM. Effect of botulinum toxin injection in the rectus femoris on stiff-knee gait in people with stroke: a prospective observational study. *Arch Phys Med Rehab* 2008; 89: 56–61.
- Tenniglo MJ, Nederhand MJ, Prinsen EC, Nene AV, Rietman JS, Buurke JH. Effect of chemodenervation of the rectus femoris muscle in adults with a stiff knee gait due to spastic paresis: a systematic review with a meta-analysis in patients with stroke. *Arch Phys Med Rehab* 2014; 95: 576–587.
- Perry J. Distal rectus femoris transfer. *Develop Med Child Neurol* 1987; 29: 153–158.
- Gage JR, Perry J, Hicks RR, Koop S, Werntz JR. Rectus femoris transfer to improve knee function of children with cerebral palsy. *Develop Med Child Neurol* 1987; 29: 159–166.
- Millodot M. *Dictionary of Optometry and Visual Science*, 7th ed, Edinburgh; New York, Butterworth-Heinemann Elsevier. 2009
- Drefus LC, Clarke S, Resnik K, Koltsov J, Dodwell ER, Scher DM. The Root-Ely modified test of rectus femoris spasticity has reliability in individuals with cerebral palsy. *HSS J* 2018; 14: 143–147.
- Marks MC, Alexander J, Sutherland DH, Chambers HG. Clinical utility of the Duncan-Ely test for rectus femoris dysfunction during the swing phase of gait. *Develop Med Child Neurol* 2003; 45: 763–768.
- Bleck EE. *Orthopaedic management in cerebral palsy*. Clinics in developmental medicine Vol No 99/100. London: Mac Keith Press, 1987.
- Sutherland DH, Davids JR. Common gait abnormalities of the knee in cerebral palsy. *Clin Orthop Relat Res* 1993; 288: 139–147.
- Fleuren JF, Voerman GE, Erren-Wolters CV, Snoek GJ, Rietman JS, Hermens HJ, et al. Stop using the Ashworth Scale for the assessment of spasticity. *J Neurol Neurosurg Psychiatry* 2010; 81: 46–52.
- Hermens HJ, Freriks B, Merletti R, et al. *European Recommendations for Surface ElectroMyoGraphy*. Enschede: Roessingh Research and Development, 1999.
- Byrne CA, Lyons GM, Donnelly AE, O'Keeffe DT, Hermens H, Nene A. Rectus femoris surface myoelectric signal cross-talk during static contractions. *J Electromyogr Kinesiol* 2005; 15: 564–575.
- Perry J, Hoffer MM, Antonelli D, Plut J, Lewis G, Greenberg R. Electromyography before and after surgery for hip deformity in children with cerebral palsy. A comparison of clinical and electromyographic findings. *J Bone Joint Surg Am* 1976; 58: 201–208.
- Tok F, Balaban B, Yasar E, Alaca R, Tan AK. The effects of onabotulinum toxin A injection into rectus femoris muscle in hemiplegic stroke patients with stiff-knee gait. *Am J Phys Med Rehabil* 2012; 91: 321–326.
- Chantraine F, Detrembleur C, Lejeune TM. Effect of the rectus femoris motor branch block on post-stroke stiff-legged gait. *Acta Neurol Belg* 2005; 105: 171–177.
- Ellington MD, Scott AC, Linton J, Sullivan E, Barnes D. Rectus femoris transfer versus rectus intramuscular lengthening for the treatment of stiff knee gait in children with cerebral palsy. *J Pediatr Orthop* 2018; 38: e213–e218.
- Gross R, Robertson J, Leboeuf F, Hamel O, Brochard S, Perrouin-Verbe B. Neurotomy of the rectus femoris nerve: short-term effectiveness for spastic stiff knee gait: clinical assessment and quantitative gait analysis. *Gait Posture* 2017; 52: 251–257.
- Lee SY, Sung KH, Chung CY, Lee KM, Kwon SS, Kim TG, et al. Reliability and validity of the Duncan-Ely test for assessing rectus femoris spasticity in patients with cerebral palsy. *Dev Med Child Neurol* 2015; 57: 963–968.
- Banky M, Ryan HK, Clark R, Olver J, Williams G. Do clinical tests of spasticity accurately reflect muscle function during walking: a systematic review. *Brain Inj* 2017; 31: 440–455.
- Ada L, Vattanasilp W, O'Dwyer NJ, Crosbie J. Does spasticity contribute to walking dysfunction after stroke? *J Neurol Neurosurg Psychiatry* 1998; 64: 628–635.
- Graham HK. *Pendulum test in cerebral palsy*. *Lancet* 2000; 355: 2184.
- Brashear A, Elovic E. *Spasticity: diagnosis and management*. Demos Medical, New York: Springer Publishing Co.; 2011.
- Dietz V, Sinkjaer T. Spastic movement disorder: impaired reflex function and altered muscle mechanics. *Lancet Neurol* 2007; 6: 725–733.
- Yelnik A, Albert T, Bonan I, Laffont I. A clinical guide to assess the role of lower limb extensor overactivity in hemiplegic gait disorders. *Stroke* 1999; 30: 580–585.

30. Perry J, Waters RL, Perrin T. Electromyographic analysis of equinovarus following stroke. *Clin Orthop Relat Res* 1978; 131: 47–53.
31. Fleuren JF, Nederhand MJ, Hermens HJ. Influence of posture and muscle length on stretch reflex activity in poststroke patients with spasticity. *Arch Phys Med Rehabil* 2006; 87: 981–988.
32. Kakebeeke TH, Lechner H, Baumberger M, Denoth J, Michel D, Knecht H. The importance of posture on the isokinetic assessment of spasticity. *Spinal Cord* 2002; 40: 236–243.
33. Vodovnik L, Bowman BR, Bajd T. Dynamics of spastic knee joint. *Med Biol Eng Comput* 1984; 22: 63–69.
34. Lamontagne A, Malouin F, Richards CL. Locomotor-specific measure of spasticity of plantarflexor muscles after stroke. *Arch Phys Med Rehabil* 2001; 82: 1696–1704.
35. Nonnekes J, Benda N, van Duijnhoven H, et al. Management of gait impairments in chronic unilateral upper motor neuron lesions: a review. *JAMA Neurol* 2018; 75: 751–758.
36. Kay RM, Rethlefsen SA, Kelly JP, Wren TA. Predictive value of the Duncan-Ely test in distal rectus femoris transfer. *J Pediatr Orthop* 2004; 24: 59–62.
37. Muthusamy K, Seidl AJ, Friesen RM, Carollo JJ, Pan Z, Chang FM. Rectus femoris transfer in children with cerebral palsy: evaluation of transfer site and preoperative indicators. *J Pediatr Orthop* 2008; 28: 674–678.
38. Hemo Y, Aiona MD, Pierce RA, Dorociak R, Sussman MD. Comparison of rectus femoris transposition with traditional transfer for treatment of stiff knee gait in patients with cerebral palsy. *J Child Orthop* 2007; 1: 37–41.
39. Loftrod B, Terjesen T. Results of treatment when orthopaedic surgeons follow gait-analysis recommendations in children with CP. *Dev Med Child Neurol* 2008; 50: 503–509.