Background and Purpose. Spasticity (hypertonicity) is a frequent problem that can develop after stroke and can lead to a number of secondary complications, such as contractures and pain. Consequently, many rehabilitation resources are used in treating the condition and its secondary complications. At present, the clinical assessment of spasticity incorporates descriptive scales of resistance to passive movement, but the use of a neurophysiological measure of muscle activity levels has been advocated. This case report focuses on the diagnosis of spasticity through the use of a neurophysiological measure.

Case Descriptions. Two individuals who required botulinum toxin treatment for poststroke spasticity were assessed over a course of 20 weeks with both clinical (Modified Ashworth Scale) and neurophysiological (surface electromyography recording of levels of muscle activity) measures of spasticity. Additionally, arm function, arm movement, and pain were measured. The individuals’ responses to treatment with botulinum toxin and overall recovery after stroke are described.

Outcomes. There were discrepancies between the clinical and the neurophysiological measures of spasticity. The clinical measure of spasticity was not effective in consistently identifying the presence of spasticity and, therefore, also was ineffective in documenting the individuals’ responses to treatment. The neurophysiological measure was able to identify when muscle activity levels had been reduced, but a reduction in muscle activity levels did not always correspond with a reduction in Modified Ashworth Scale scores.

Discussion. The accurate identification of spasticity is important not only for assessment but also for the selection of appropriate treatments after stroke.
Stroke is a leading cause of adult disability, with a third of people who have survived acute stroke being left with moderate or severe levels of disability. The recovery of the affected upper extremity after stroke is particularly concerning, with an estimated 40% of patients not regaining functional use of this limb. The known consequences of stroke include muscle weakness, loss of dexterity, and spasticity (hypertonicity).

Spasticity is a neurological impairment that frequently occurs after stroke and is believed to contribute not only to a loss of function but also to the development of joint contractures and pain. Spasticity is defined as “disordered sensorimotor control, resulting from an upper motor neuron lesion, presenting as intermittent or sustained involuntary activation of muscles.” Rehabilitation resources can be invested in identifying spasticity, attempting to prevent its development, and treating the condition once it is established. Pharmacological methods are used routinely to treat established spasticity, and intervention is recommended when spasticity causes functional problems. Given the effects attributed to spasticity, effective management might involve initiating treatment at the first signs of spasticity as opposed to waiting until either functional problems or secondary complications have developed. Achieving this goal would require a method for accurately identifying spasticity. Physical therapists are highly involved in the management of poststroke spasticity and are increasingly becoming involved in the decision-making processes underlying the use of pharmacological agents, such as botulinum toxin A. Thus, the accurate assessment of spasticity is a key role for any neurological physical therapist; that is, there is a need for a way in which to identify the presence of (or rule out) abnormal muscle activation.

The assessment of spasticity in clinical practice usually is accomplished with descriptive scales for evaluating the resistance to passive movement at a nominal or ordinal level of measurement; one of the most frequently used scales is the Modified Ashworth Scale (MAS). The MAS has been criticized—both its reliability and its validity having been challenged—because it is unable to distinguish between neurological and mechanical contributions to the stiffness levels observed during passive movement. It is, therefore, an indirect measure of spasticity because it cannot establish the presence of (or rule out) abnormal muscle activation. Nevertheless, it is unlikely that scales such as the MAS will cease to be used by clinicians for the foreseeable future. It has been advised that the assessment of spasticity should include neurophysiological measurements of muscle activity levels, which would provide a direct measure of spasticity, according to the current understanding of the phenomenon.

This case report focuses on the diagnosis of spasticity through the use of a neurophysiological measure. The report describes 2 individuals who developed poststroke spasticity and who subsequently were given botulinum toxin type A (BoNT-A) as part of the management of their spasticity. A neurophysiological method of spasticity assessment was used alongside the more traditional MAS. The information gained through the use of this neurological assessment tool, both in the initial identification of upper-limb spasticity and in the subsequent evaluation of the response to treatment with BoNT-A, is discussed. Repeat measurements of upper-limb function, arm movement, and pain for both individuals allow for further discussion of how the information gained through an accurate diagnosis of spasticity can be applied in clinical practice.

**Measures**

Spasticity, passive and active ranges of motion in the elbow and wrist joints, arm function, and pain were first assessed at 34 days after stroke for one of the individuals and at 18 days after stroke for the other individual. Repeat measurements were obtained at 4, 8, 12, and 20 weeks after the initial assessment.

Spasticity was assessed at the elbow and wrist with a neurophysiological measure and the MAS. Surface electromyography (EMG) electrodes were initially placed on the biceps...
and triceps muscles, the long wrist and finger flexor muscles, and the long wrist and finger extensor muscles. The SENIAM (surface electromyography for the noninvasive assessment of muscles) recommendations were followed for EMG electrode placement and, when available, for the positioning of the sensors over the muscles.\(^1\)

For the elbow measurement, an electrogoniometer\(^2\) was positioned across the elbow joint (medial aspect). The individual was sitting with the shoulder abducted to 90 degrees (or as close to this position as could be achieved without pain) and in external rotation so that the thumb was uppermost. The assessor then manually moved the elbow from maximal flexion to maximal extension. This passive movement was made initially at a low velocity and then was repeated at a high velocity.

For the wrist measurement, the electrogoniometer was placed across the wrist joint (medial aspect). The arm was repositioned with the shoulder in the neutral position, the elbow at 90 degrees, and the forearm fully supported and parallel to the floor, mid pronation/supination, so that the palm was facing medially with the thumb uppermost. The same technique of manual displacement from maximal flexion to maximal extension at low and high velocities was used.

The displacement (ie, passive range of motion) and EMG data were simultaneously recorded at 1,000 Hz at each testing point and saved for subsequent analysis. All data were obtained using the DataLINK data acquisition system.\(^3\) The MAS measurements were obtained during the fast passive movements of the elbow and wrist.

The active range of motion was that achieved by the individual from the starting position—maximal extension for elbow flexion, maximal extension for elbow extension, and from neutral for the wrist. These active movements were made with the limb supported; that is, the individual did not have to maintain the limb against gravity. Angular displacement and EMG activity\(^1\) were recorded during these movements. The active range of motion of the elbow was directly comparable to the passive range of motion achieved by the assessor, as the active range of motion began at the same position as the passive range of motion. For the wrist, the active range of motion began at a neutral position; therefore, the value that was comparable to the value for the passive range of motion (from maximal flexion to maximal extension) was the total for active flexion and active extension combined.

Arm function was assessed with the Action Research Arm Test, a test of arm function with established reliability and validity for people with stroke.\(^15\) Pain was rated by the individual with a 5-point verbal descriptor scale: no pain, mild pain, moderate pain, severe pain, or pain could not be worse.

### Data Analysis

Raw EMG data were notch filtered (50 Hz) and then smoothed with root-mean-square methods (50-millisecond window width). For the analysis of spasticity, the average integrated EMG activity (referenced to angular displacement) was calculated during both low-velocity and high-velocity passive stretches by use of Mathcad (version 12.1).\(^2\)

At baseline, Mr A had no strong clinical signs of flexor muscle spasticity (as assessed with the MAS) at either the elbow or the wrist (Table), but the neurophysiological measure revealed a velocity-dependent increase in muscle activity at both the elbow and the wrist, although the increase was more marked at the wrist (Fig. 1). Mr A had no active movement or arm function at the baseline assessment, nor did he report any pain in his arm (Table).

Four weeks later, Mr A had developed clinical signs of spasticity, that is, increased resistance to passive movement (as recorded with MAS scores) and decreased range of motion, at both the elbow and the wrist (Table). The neurophysiological assessment demonstrated that the level of spasticity remained similar to that seen at the baseline. Figure 2 shows the neurophysiological spasticity at Mr A’s elbow and wrist; almost immediately after the limb was moved into extension, the flexor muscle ac-
Activity levels began to increase, and this activity continued throughout the rest of the stretch. This increase in activity was present in the slow- and fast-velocity passive stretches for both the elbow and the wrist. In addition, the level of activity at the wrist joint was greater with the high-velocity movement, indicating a velocity-dependent component to the spasticity at this joint. Mr A still did not have any active movement or arm function, but he had now begun to experience pain in the upper extremity (Table).

**Intervention.** Subsequent to the measurements obtained 4 weeks after the baseline, a clinical decision was made to administer to Mr A botulinum toxin type A treatment was administered 5 days after the week 4 measurements were obtained. Botulinum toxin type A was chosen because of the focal nature of its action, as Mr A complained primarily about pain and stiffness in the upper extremity. The muscles selected for the injections were the biceps brachii, brachialis, brachioradialis, flexor digitorum superficialis, and flexor digitorum profundus muscles; Mr A received a total dose of 400 U of BoNT-A (Botox®) distributed among these muscles 5 days after the week 4 measurements were obtained. During the week after the BoNT-A injections were given, Mr A was discharged from inpatient care and returned home with support. Outpatient physical therapy and occupational therapy were arranged but did not commence until 3 weeks after discharge.

**Outcome.** The MAS scores for Mr A remained high throughout the follow-up, never being less than 3, indicating that clinical signs of spasticity remained at both the wrist and the elbow. In contrast, the neurophysiological measure of spasticity at...
the elbow suggested a decrease in spasticity (Fig. 3A); the response to the passive stretch was of a much smaller magnitude than the response observed 4 weeks earlier, before the BoNT-A injections were given. The levels of muscle activity in the elbow spasticity assessments continued to be low for the remainder of the observation period, although by the final measure (week 20 after the baseline), there was some increase in muscle activity levels during the fast stretch. After injection with BoNT-A, the neurophysiological responses at the wrist remained similar to those observed before the intervention (Fig. 3B); the neurophysiological measure indicated a decrease only at the final (week 20) assessment. Therefore, although both the clinical

Figure 2.
(A) Electromyography (EMG) activity in the biceps muscle during slow and fast passive stretching into extension of the elbow for Mr A at 4 weeks after the baseline (before botulinum toxin type A injections). (B) EMG activity in the long wrist flexor muscles during slow and fast passive stretching into extension of the wrist for Mr A at 4 weeks after the baseline (before botulinum toxin type A injections).

Figure 3.
(A) Electromyography (EMG) activity in the biceps muscle during slow and fast passive stretching into extension of the elbow for Mr A at 8 weeks after the baseline (after botulinum toxin type A injections). (B) EMG activity in the long wrist flexor muscles during slow and fast passive stretching into extension of the wrist for Mr A at 8 weeks after the baseline (after botulinum toxin type A injections).
and the neurophysiological measures indicated signs of spasticity at the wrist, there was inconsistency between the clinical findings and the neurophysiological observations for the elbow.

Mr A did not regain any active movement or functional capabilities in the upper extremity during the observation period. The limitations in the range of motion at the elbow remained, possibly indicating adaptive shortening. Mr A continued to experience pain in the upper extremity, which was at its highest level at the week 8 assessment and was still present, albeit at a lower level, at the final (week 20) assessment (approximately 6 months after the stroke).

Mr B

Examination. Mr B was a 54-year-old man who had been admitted to a hospital with a first-instance unilateral stroke with right-side hemiplegia. Clinically, the stroke had been classified as total anterior circulation syndrome, and a computerized tomography scan showed a left parietal infarct. Mr B worked full time as a prison warden before his stroke and lived with his wife and their 5-year-old son.

The baseline assessments for Mr B took place 18 days after the stroke. The measurements are shown in the Table, with the exception of the neurophysiological spasticity assessments, which are shown in Figure 4. Mr B had indications of high levels of spasticity on both the clinical scale (MAS score of 3 at both the elbow and the wrist) and the neurophysiological measure (Fig. 4). The increase in muscle activity levels at the wrist was clearly demonstrated by the neurophysiological measure (Fig. 5A), which revealed a steady increase in the activity of the flexor muscles as the wrist was moved toward extension. Mr B had active movement only in the elbow flexor muscles, and he had no arm function at the baseline assessment.

Intervention. The clinical signs of spasticity resulted in a decision to treat the spasticity early with pharmacological interventions, initially baclofen and then later BoNT-A because the therapy team believed that there had been an insufficient response to the baclofen. Botulinum toxin type A was administered 38 days after admission to the hospital (20 days after the baseline assessment); the elbow flexor muscles were injected with 200 U of BoNT-A (Botox) divided among the biceps, brachialis, and brachioradialis muscles. The BoNT-A injections were given 1 week before the week 4 assessment (which took place approximately 6 weeks after the stroke). Mr B remained an inpatient throughout the course of the observation period but was scheduled for discharge to his home shortly after the final assessment.

Outcome. The clinical signs of spasticity for Mr B, as measured with the MAS, remained high immediately after the BoNT-A injections were given but subsequently lessened; by the final assessment (week 20 after the baseline), no clinical evidence of spasticity was seen at the wrist, and only minimal evidence was seen at the elbow. The neurophysiological measure revealed a sharp decrease in muscle activity levels at both the elbow and the wrist after the BoNT-A injections were given (in contrast to the clinical MAS measure at week 4), and these levels remained low throughout the follow-up. The neurophysiological measure (Fig. 5B) showed that although the wrist flexor muscles still responded to the fast stretch, the magnitude of the response was smaller than that observed at the baseline, and the response of the same muscles to the slow stretch was limited primarily to the end range of motion into extension. Although there was some increase in muscle activity levels at the week 8 assessment for the wrist, the levels decreased at later assessments (Fig. 4).

Mr B experienced a return of active movement at both the elbow and the wrist in both the flexor and the extensor directions at week 4, and this movement continued to improve throughout the follow-up. By week 12, Mr B could actively move his elbow and wrist through the same range as the assessor could move them passively. Arm function recov-
ery began slightly later than active movement recovery, but Mr B had made good progress by the final assessment, with an Action Research Arm Test score of 20. This score indicated that he had both proximal recovery and distal recovery and was able to lift his hand onto a table and grasp an object placed on the table. The passive range of motion at both joints remained stable, indicating no development of contractures. Although Mr B experienced moderate pain in the upper limb at the week 4 assessment, this pain did not linger, and he did not complain of pain from week 12 onward.

**Discussion**

This case report documented the progress of 2 individuals who had stroke and who developed spasticity in their upper limbs early after the onset of a stroke. Both individuals had experienced severe disabilities as a consequence of the stroke, with no recovery of upper-extremity function in the first few weeks after the stroke. Their prognosis for further functional recovery of the upper extremity, therefore, was limited.16,17 Despite early treatment with BoNT-A, 1 of the 2 individuals continued to demonstrate clinical signs related to spasticity and associated problems of contractures and pain. The other individual did not, and he progressed enough to regain some upper-limb function.

**Discrepancy Between Clinical and Neurophysiological Measures of Spasticity**

There were inconsistencies between the 2 measures of spasticity, that is, the MAS and the neurophysiological measure. The MAS testing took place simultaneously with the fast passive stretch; this strategy would result in the starting position and velocity of movement during the procedure being the same for both the neurophysiological measure and MAS testing. The starting position for MAS testing was sitting rather than supine,18 but the procedures used in the fast stretch closely followed the protocols described for the elbow by Bohannon and Smith9 in all other aspects. We do not believe that testing in the sitting position would have adversely affected the reliability of the MAS, as the starting position was standardized throughout as recommended by Pandyan et al.11

The MAS failed to consistently detect increased levels of muscle activity indicative of spasticity in Mr A at the baseline assessment and was not able to detect decreased levels of muscle activity after the injection of BoNT-A into the elbow flexor muscles in Mr A. These inconsistencies could be attributable, in part, to the different constructs of the 2 measures. The MAS is a measure of resistance to passive movement, whereas the neurophysiological measure quantifies levels of muscle activity. Thus, the neurophysiological measure, unlike the MAS, is a direct measure of spasticity, as defined by the Support Programme for Assembly of Database for Spasticity Measurement consortium.5 Although muscle activity can contribute to an increase in the resistance to passive movement, this relationship is confounded by a variety of other biomechanical factors.5 For example, for Mr A, the high residual stiffness levels documented by MAS testing at the elbow, despite the

![Figure 5.](https://academic.oup.com/ptj/article-abstract/89/7/688/2747289)
reduction in the levels of muscle activity, may have been a reflection of adaptive shortening that had already taken place.

The method for assessing the response to passive stretching involved the use of a handheld tool and therefore would have allowed some variations in the velocity at which the limb was moved at different assessment sessions. It has been acknowledged that muscle activity levels could be affected by the velocity of the passive stretch, but controlling for this effect would necessitate the use of a motorized assessment system. The use of a handheld tool for spasticity assessment offers greater flexibility in the assessment process. Although this method can result in some variations between assessment sessions, the velocity of movement can be recorded and examined subsequently by the assessor. Manual testing of this nature is more feasible for adoption by clinicians because it can be used at a patient’s bedside and, therefore, can be used for patients with severe impairments after stroke, such as the 2 individuals described here, who may not be able to tolerate a motorized assessment system.

Examination of the mean velocities at which the limbs were moved showed good consistency for the slow movement, with mean velocities ranging from 3°/s to 13°/s for Mr A and from 8°/s to 17°/s for Mr B. There were more variations in the velocities of the fast movement, but in all instances, there was a distinct difference between the slow and the fast passive movements at each assessment. A primary concern with these variations in velocities would be underestimation of the presence of spasticity as a result of not detecting a velocity-dependent component if the limb was moved too slowly. The mean velocities of the fast passive movement for Mr A ranged from 27°/s to 58°/s at the wrist and from 22°/s to 104°/s at the elbow. The mean velocities of the fast movement were much lower at week 4 (32°/s) and week 20 (22°/s) than at the other assessment points. Interestingly, although it might be expected that movement at a lower velocity would result in less EMG muscle activity being recorded, these 2 assessment points actually had higher levels of EMG muscle activity seen in Mr A’s elbow flexor muscles during the observation period (Fig. 1). It is possible, though, that even higher levels of muscle activity could have been recorded had the limb been moved at a higher velocity at these assessment points.

For Mr B, the mean velocities of the fast movement ranged from 73°/s to 122°/s for the wrist and from 51°/s to 108°/s for the elbow. It is acknowledged that large variations in the velocities at which a limb is moved between assessment points is undesirable. However, given that in both individuals the highest levels of muscle activity were observed on the occasions when the velocity of perturbation was lower than usual, we believe that the presence of spasticity was not missed in these cases.

It is important that clinicians using handheld techniques to measure muscle activity levels also record the velocities at which the limb was moved, when possible. We could not eliminate differences in the amplitudes of the signals from the surface EMG electrodes at consecutive assessments, but we reduced them by adopting the standardized EMG procedures described by SENIAM. Clinicians interpreting the results of spasticity measurements should consider these factors, which may cause differences in measurements. In its present form, the neurophysiological measure of spasticity is of primary value as a diagnostic tool, identifying the presence of abnormally increased muscle activity levels. Because of the known errors in measurements that occur with this tool, it should be used with caution for clinical measurements if identifying subtle changes in muscle activity levels over time is the goal. Marked trends, however, can be observed, and these trends can contribute to a clinician’s understanding of an individual’s spasticity.

**Early Identification of Spasticity and Potential Prevention of Secondary Complications of Spasticity**

Although the neurophysiological measure revealed signs of spasticity for Mr A at the baseline, he was not treated with BoNT-A until clinical signs of spasticity had also developed. This finding raises the question of whether treatment with BoNT-A should have been used prophylactically to prevent the posturing that is associated with abnormal muscle activity and that could have contributed to adaptive shortening. The management of poststroke spasticity with BoNT-A treatment is one of several strategies that can be used by a rehabilitation team. Regardless of the treatment selected, however, the need for accurate assessment and monitoring of the effectiveness of the chosen treatment remains paramount.

**Treatment Strategies for Patients After Injections With Botulinum Toxin Type A**

The focus of this case report was to examine how the adoption of a neurophysiological measure of spasticity can assist in diagnostic processes for clinicians and thus direct treatment strategies. However, it is important to acknowledge that the contrasting outcomes for the 2 individuals described in this case report could have been influenced by their different rehabilitation programs. Both patients received specialized stroke rehabilitation, but discharge from
inpatient care occurred much earlier for Mr A. After he was given injections of BoNT-A, Mr A received only 1 week of therapy before being discharged from the hospital, and there was a gap of 3 weeks until outpatient therapy began. Once therapy was re-instituted, it took place on a weekly basis, as opposed to the daily treatments that Mr A had been receiving as an inpatient. In contrast, Mr B remained an inpatient for 6 months after the stroke and thus continued to receive an intensive therapy program throughout the follow-up, including the period immediately after the BoNT-A injections. Guidelines for the use of BoNT-A clearly indicate that a program of therapy is requisite after injections, and it is possible that the additional functional benefit observed in Mr B was attributable to his concomitant intensive therapy program.

Repeatead Cycles of Botulinum Toxin Type A Treatment

The case of Mr A highlights the problem of using a confounded measure, such as the MAS, to quantify the effectiveness of spasticity treatment: it is possible for people who respond to treatment to be inappropriately identified as nonresponders. The continued signs of spasticity at the wrist with both the clinical and the neurophysiological spasticity measures could indicate that Mr A did not respond to the BoNT-A treatment. However, this possibility seems unlikely given the response observed at the elbow with the neurophysiological measure. Other reasons for the continued signs of wrist spasticity could be either inadequate dosage or injections into the wrong muscle (ie, in Mr A, only the finger flexor muscles and not the wrist flexor muscles were injected). Currently, it is advised that there be 12 weeks between cycles of treatment with BoNT-A; thus, in the event of insufficient dosage or incorrect selection of muscles for injection, there is an increased likelihood of secondary complications becoming established before a clinician can repeat the injection. However, some preliminary data have suggested that it may be possible to re-inject BoNT-A within this 12-week time frame. Monitoring with a neurophysiological measure could assist in the selection of patients for whom such a treatment approach may be appropriate. For example, it might have been considered that the treatment dosage was insufficient for Mr A given the sustained clinical signs of spasticity after the BoNT-A injections were given and thus that re-injection should be considered. However, an analysis of the neurophysiological measure would show that this option would be inappropriate at the elbow, where BoNT-A had successfully shut down the flexor muscles, but might be worth considering for the wrist.

The 3 key issues outlined by this case report are as follows. There are discrepancies between the spasticity measure of the MAS and the neurophysiological measure of increased muscle activity. The MAS may lack sensitivity and, therefore, is an inadequate tool for identifying spasticity and monitoring its response to treatment. The neurophysiological measure of spasticity described in this case report is a bedside tool that can be adopted by clinicians to aid in identifying and monitoring the presence of spasticity.

All authors provided concept/idea/project design. Ms Cousins, Dr Rimington, and Dr Pandyan provided writing. Ms Cousins provided data collection. Ms Cousins, Dr Roffe, and Dr Pandyan provided data analysis. Dr Roffe, Dr Rimington, and Dr Pandyan provided project management. Dr Pandyan provided fund procurement and facilities/equipment. Dr Roffe provided patients and institutional liaison. Dr Ward, Dr Roffe, Dr Rimington, and Dr Pandyan provided consultation (including review of manuscript before submission).

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