A Case of Finger and Wrist Flexor Spasticity Following a Stroke

Case Report

A 63-year-old white male suffered a cerebral vascular accident nine months ago which left him aphasic and with a right hemiplegia. His aphasia has improved to a mild speech disorder and his gait has become fully normal. The major disability for which he seeks consultation is his right hand disability.

Hand placement is excellent with normal active control of the right shoulder and elbow. There is marked spasticity of the finger and wrist flexors. He is able to flex actively the fingers and wrist, but is not able to release. The finger and wrist extensors are flail, so the hand assumes a flexor pattern when he attempts to use it (Fig. 1). Sensory exam is almost fully normal with excellent position sense and two point discrimination of 3-4 mm at the fingertips.

Discussion

Before discussing the specific treatment of this patient, the pathophysiology involved should be understood. By definition, peripheral dysfunction in stroke is secondary to brain injury. This brain injury is not simply the loss of innervation of specific muscles or muscle groups. The central nervous system in man has evolved as a series of centers of higher control. The most central or primitive centers in the brain stem allow gross motor action, while the higher centers provide finer sophistication and coordination of fine motor function. The highest centers in the cerebral cortex provide modulation or fine-tuning. In stroke, one sees unmasking of the more primitive levels of motor function. This leads to motor imbalance between agonist and antagonist muscles and muscle groups, with subsequent loss of fine motor control. These disorders of muscle balance usually appear in combination and are referred to as synergy patterns or, simply, synergy. Hence, the problem is not real spasticity or hypertonicity, but alternation in the agonist/antagonist muscle balance with relative hypertonicity.

The goal of surgical therapy in stroke is the achievement of agonist/antagonist motor balance. Types of procedures are limited and specific. Muscle-tendon unit lengthening procedures decrease hypertonicity by decreasing the stretch reflex. These procedures are the most reliable and reproducible. Specific in-phase tendon transfers are applicable when an appropriate in-phase muscle is available. Out-of-phase tendon transfers are very difficult, as these patients do not possess the capacity for retraining.

Timing is extremely important. Following a stroke, the time delay in recovery is important and prognostic. Those patients who regain function within the first few days or make rapid recovery are more likely to regain function approaching normal. The greater the delay in recovery or the slower the rate of recovery, the less complete will be the final expected function. Most patients attain their final recovery within three months, but we often see recovery up to eight or nine months following the stroke. For this reason, surgery is rarely undertaken before six months and only in patients who have reached a plateau in recovery with intensive hand/occupational therapy.

Hand placement is crucial. The patients must be able to place the hand where it is needed to function. Frequently, elbow and shoulder muscle-tendon procedures must be performed at the same time or prior to hand surgery.

Sensory examination is the final prerequisite to surgery. With decreasing sensation, position sense and awareness, the patient is less likely to use the hand, even if balanced. Fingertip two-point discrimination has been an excellent predictor of hand utilization. Patients will usually use the hand independently if they possess up to 5-7 mm of two-point discrimination. With 7-12 mm, they will use the hand as an assist; whereas one cannot expect much

(continued on page 93)
Fig. 1: Typical flexor pattern of a patient with hemiplegia.

Fig. 2: Position in which the fingers and wrist will be immobilized post-operatively.

(continued on page 96)
functional use in hands where two-point discrimination is greater than 12 mm.

This patient’s problem is relative hypertonicity of the finger and wrist flexors with no function in the antagonist extensors. Placement and sensation are almost normal, so it is likely that the patient will use the hand independently.

The flexor spasticity can be approached with Z-lengthening of the flexor carpi radialis and flexor carpi ulnaris tendons, and fractional muscle lengthening of the flexor digitorum profundus, flexor digitorum superficialis, and flexor pollicis longus. Out-of-phase transfer of the flexor carpi ulnaris to the extensor carpi radialis longus can be performed to treat the lack of extension. There is an increased risk of overpull with the subsequent development of a hyperextension deformity at the wrist, however, so a more predictable result could be obtained by performing an extensor tenodesis of the wrist extensors. This can be usually accomplished by stapling the tendons of the extensor carpi radialis longus and extensor carpi ulnaris to the radius and ulna respectively with small barbed staples, while the wrist is held at 45° of extension. The hand is splinted at this position for four to six weeks (Fig. 2). The patient is then started in hand/occupational therapy and is gradually weaned from the splint over a prolonged period of time, when he eventually wears it only at night. The extensor tenodesis will gradually stretch so that the final expected wrist position is 0 to 10° of extension (Fig. 3).
Conclusion

The spasticity/hypertonicity that develops following stroke or brain injury is due to agonist/antagonist motor imbalance. The goal of surgery is to restore this motor balance in the extremities where there is volitional agonist control and motor imbalance. In this patient motor balance can be obtained by weakening the agonist flexor muscle group (fractional muscle lengthening) and static control of the antagonist extensor group (extensor tenodesis). If successful, motor imbalance will be somewhat restored, and functional capacity will be greatly increased.

Bibliography


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