7 Symptomatic and Functional After-Effects of the Syndrome of Spastic Paresis

Nathaniel H. Mayer and Alberto Esquenazi

Chiefly, the bones serve as struts, while the muscles and ligaments act as ties – cross-braces.
– D'Arcy Wentworth Thompson, On Growth and Form, 1917.

- An upper motor neuron syndrome that leads to spastic paresis can result in important symptomatic and functional consequences.
- In the upper limb, these consequences include skin and nerve problems, problems of access to the hand, elbow crease and axilla, grasp and release dysfunction, and problems of reaching.
- Symptomatic and functional consequences in the lower limb include deformities that impact skin and footwear, inadequate limb clearance, improper foot loading, abnormal limb advancement, and inadequate single limb support during locomotion.
- A thorough understanding of these disabling consequences is necessary for planning appropriate treatment.

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P. Raghavan (ed.), *Spasticity and Muscle Stiffness*,
https://doi.org/10.1007/978-3-030-96900-4_7

Introduction

This chapter describes important after-effects of the upper motor neuron syndrome which leads to spastic paresis that impedes function and causes deformity and disability [1]. Spastic paresis is characterized by involuntary phenomena that position limb segments in undesired and maladaptive configurations. These include both neural changes causing involuntary muscle overactivity including spastic dystonia, spastic co-contraction, increased flexor reflex afferent activity, and associated reactions, and non-neural changes in the muscle including muscle shortening, stiffness, and contracture [2, 3]. See Chaps. 1, 2, 3, 4, 5, and 6 for explanation of the pathophysiology of these phenomena. Spastic paresis is also characterized by agonist muscle weakness and lack of selective control of movement needed to generate voluntary movement. The neural and non-neural changes superimpose creating a brew of consequential after-effects that lead to skin, nerve, and joint problems, and dysfunction in movement, mobility, and activities of daily life.

Functional Consequences in the Upper Limb

Skin and Nerve Problems

Skin Problems of the Hand, at the Elbow Crease, and in the Axilla

A common presentation in individuals with spastic paresis is a clenched fist with involuntary flexion of the fingers and thumb. The wrist is typically flexed but may be hyperextended. A clenched fist that cannot be unclenched easily leads to maceration, erosion, detritus, skin erythema and breakdown, malodor, and potentially, infection (see Figs. 7.1, 7.2, 7.3, 7.4, 7.5). Severe shortening and/or spastic dystonia involving the flexor digitorum superficialis (FDS) and flexor digitorum profundus

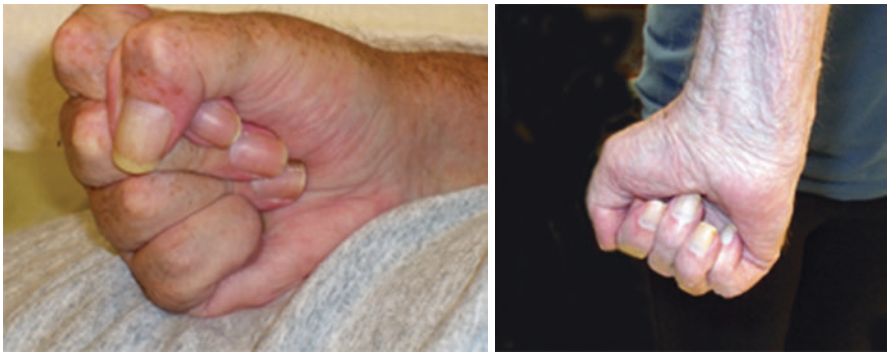

Fig. 7.1 Muscle overactivity or shortening involving the flexor digitorum superficialis (FDS) muscle results in a largely "FDS type hand" (left image), whereas overactivity or shortening involving the flexor digitorum profundus (FDP) muscle leads to an "FDP type hand" (right image). A persistently clenched fist leads to moisture retention, tissue maceration, malodor and skin breakdown

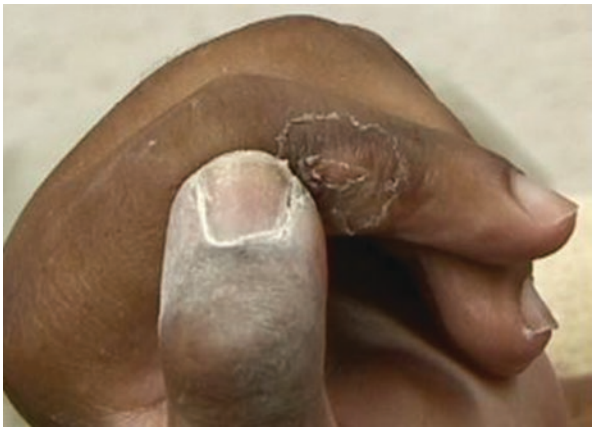

Fig. 7.2 Skin shearing and erosion due to persistent overactivity or shortening involving the flexor pollicis longus (FPL) muscle in an "FPL type thumb" is seen on the middle phalanx of the index finger

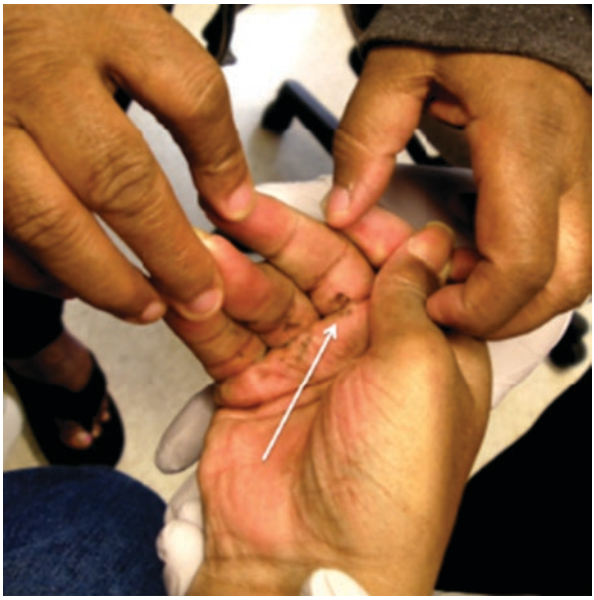

Fig. 7.3 Detritus has collected in the proximal crease of the long finger in this patient with a clenched fist

(FDP) muscles result in an "FDS hand" and an "FDP hand," respectively, as seen in Fig. 7.1. It is not unusual, however, to have mixed findings with some fingers of the FDS type and others of the FDP type. The description of the FDS hand and FDP hand can guide the selection of muscles for injection treatments. Fists that have been clenched for a long time are prone to contracture. Serial casting or serial splinting is a consideration in order to open the hand and provide access for air circulation, moisture evaporation, and elimination of malodor (see Fig. 7.6) [4]. See Chaps. 12 and 13 for local injection treatments if the muscles are not contractured. Surgical interventions such as an "STP" transfer (superficialis to profundus transfer) are a consideration when the patient is chronically affected and not expected to have

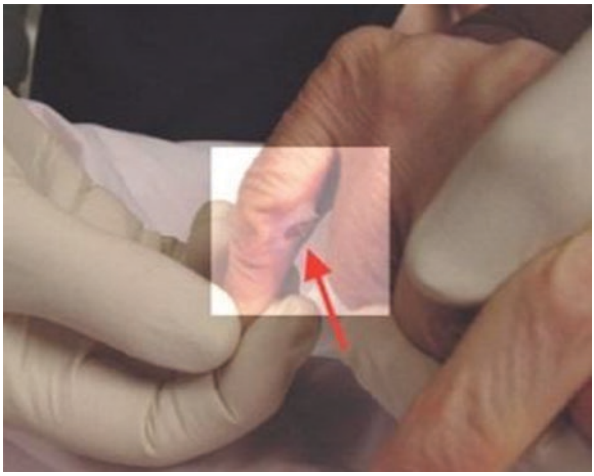

Fig. 7.4 Frank skin ulceration developed on the inner aspect of the little finger in this patient with a clenched fist

Fig. 7.5 Muscle overactivity or shortening of the flexor digitorum profundus muscle can lead to the fingernails digging into the palmar skin causing indentation and laceration

neurological recovery of voluntary finger movements [5]. An aeration splint (see Fig. 7.7) to keep the fingers away from the palm and allow air circulation through the hand is an alternative when surgery is not feasible. When volitional finger flexor movement is seen in the presence of flexor contracture, fractional lengthening may be a surgical consideration.

A flexed elbow with skin overlap can lead to moisture accumulation, irritation, erythema, and skin breakdown (see Fig. 7.8). Skin irritation and erythema can be seen in the elbow crease in the image on the right side of Fig. 7.8. The image on the left side of Fig. 7.9 shows overlapping skin of another patient with a chronically flexed elbow. Persistent flexion posturing with skin overlap led to severe ulceration and skin breakdown, seen in the image on the right side of

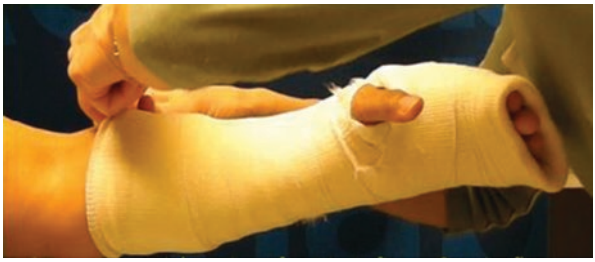

Fig. 7.6 Fists that have been clenched for a long time are prone to developing contractures. This patient is undergoing composite serial casting to stretch out the combined contracture of wrist and finger flexors with the goal of opening the hand to provide access for air circulation, moisture evaporation, and elimination of malodor

Fig. 7.7 An aeration splint has been fabricated by an occupational therapist to keep the fingers away from the palm and allow air circulation through the hand. Unlike a resting hand splint which makes full palmar contact, an aeration splint creates a tunnel to allow air to circulate across the palm

Fig. 7.9. It is important to educate families to observe skin condition when they perform passive range of motion exercises and bring the aforementioned signs to medical attention. Treatment involves releasing the shortened muscles and/or mitigating muscle overactivity prior to instituting a serial casting program. Reducing tension facilitates patient tolerance to stretching by the casting process. After completion of the casting course, a maintenance resting splint is usually provided.

An adducted shoulder can result in persistent skin overlap leading to skin irritation, maceration, and breakdown (see Figs. 7.10 and 7.11). Shortening and/or overactivity in the pectoralis major (PM), teres major (TM), latissimus dorsi (LD), and subscapularis muscles may be involved. Local treatment of skin lesions along with

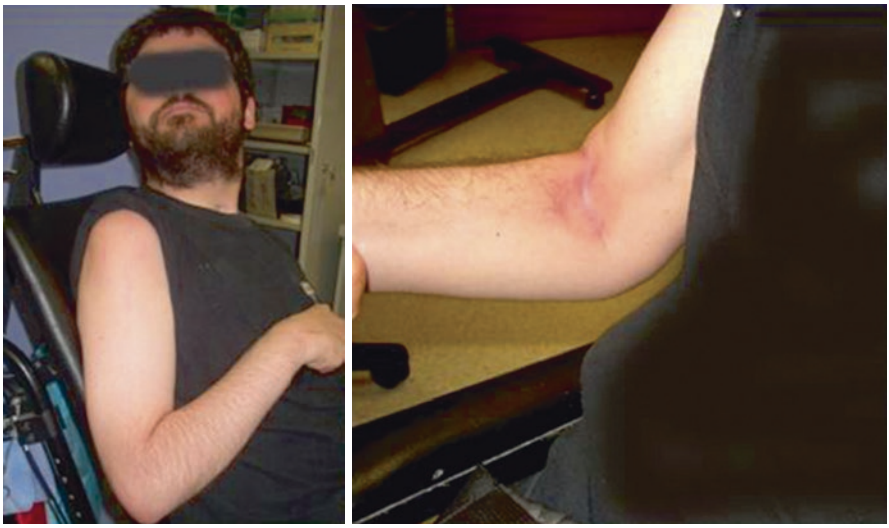

Fig. 7.8 A chronically flexed elbow seen in the left image has led to skin irritation and erythema seen in the elbow crease on the right image

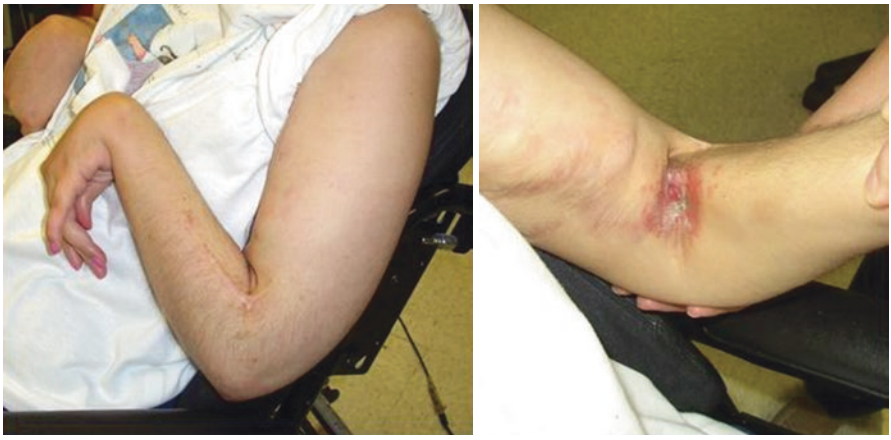

Fig. 7.9 The image on the left side shows overlapping skin in another patient with a chronically flexed elbow. Persistent flexion posturing with skin overlap led to severe skin breakdown in the elbow crease seen on the right image. In addition to the intrinsic value of doing range of motion exercises, it is important to teach families and caregivers to observe skin condition

release of muscle tension followed by vigorous passive range of motion exercises are treatment considerations. Serial casting is not a treatment consideration in this region. Ranging can be conveniently carried out when patients are showered but we also recommend that passive shoulder abduction be performed many times a day because the natural resting position of the shoulder is one of arm contact with the

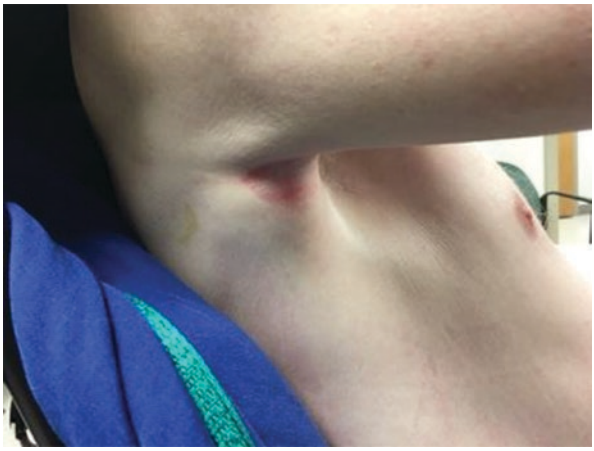

Fig. 7.10 An adducted shoulder can result in persistent skin overlap leading to skin irritation, maceration, and breakdown. Note the area of erythema at the confluence of the adductor tendons

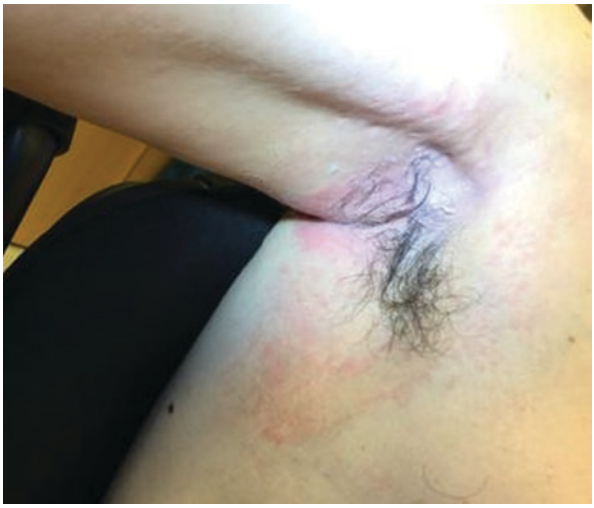

Fig. 7.11 Skin irritation due to moisture retention in the axilla is seen in this image. A persistently adducted shoulder can result from involuntary muscle overactivity or shortening of the pectoralis major, teres major, latissimus dorsi, and/or subscapularis muscles

trunk, i.e., skin overlap. When a patient is not able to actively abduct the shoulder, the ordinary rest position closes off the axilla from aeration.

Episodic Disruptive Symptoms

Sustained clonus involving the upper and lower limbs can be functionally disruptive to activities of daily living, transfers, and gait. The trigger for such episodic behavior is often unclear but some patients note that contact with a cold floor when they first get out of bed may trigger the clonus. Others recall nociceptive input or passive stretch as potential triggers. Focal chemodenervation is sometimes effective in mitigating diffuse clonus if one can identify one or more muscles that are the first to be clonic. Dantrolene sodium may help because it has been found to be effective in reducing or abolishing clonus [6, 7]. In the postsynaptic muscle membrane, the

release of calcium ions, mediated by ryanodine receptors, is an essential step in muscle contraction [8]. Dantrolene depresses excitation-contraction coupling in skeletal muscles by binding to ryanodine receptor 1, thereby preventing the release of calcium ions from storage sites in the sarcoplasmic reticulum. Because tonic contractions release more calcium ions than can be inhibited by dantrolene binding, the drug is more effective in reducing low frequency (5–8 Hz) oscillations of phasic clonus than the tonic involuntary contractions that have tetanic frequencies.

Episodic flexor spasms are usually disruptive in the lower extremities, a finding that is more often seen in spinal cord pathologies [9]. Multiple spinal levels are often involved, accounting for multi-segmental involvement that typically includes muscles responsible for ankle dorsiflexion, knee flexion, and hip flexion, and may include the lower abdominal muscles as well. Oral anti-spastic agents such as baclofen or tizanidine may be used while an intrathecal baclofen system is considered for unbridled flexor spasms [10].

Associated Reactions

Physical or voluntary activity occurring in one part of the body may be accompanied by involuntary activity in another, the latter being termed an associated reaction. Associated reactions were first described by Walshe in 1923 as released postural reactions deprived of voluntary (cortical) control [11]. The involuntary movements of an affected arm during transfers and ambulation exemplify associated reactions of the upper limb. Yawning and coughing are included as associated reactions because they are considered to be semi-voluntary movements. Walshe's own words clarify the concept of associated reactions as follows:

> "… all voluntary purposive movements are accompanied by an appropriate postural adjustment of the rest of the skeletal musculature, and that in forceful movements this adjustment or adaptation is necessarily bilateral and widespread. Although carried out under voluntary control, postural adaptation is a function of reflex mechanisms situated in the brain-stem, which are not put out of action by the lesion which produces hemiplegia and abolishes voluntary control of the musculature on the affected side of the body. In these circumstances, we should still expect postural reactions to occur when forceful voluntary motor activities are carried out by the musculature of the sound half of the body. Now, however, deprived of cortical control, they would occur in exaggerated intensity and deprived of that fineness of adaptation which that control ensures. It was concluded that the associated movements of hemiplegia are phenomena of this order, appearing in the muscles of the affected side on certain voluntary movements of the normal limbs, or on such semi-voluntary movements as yawning."

Bakheit and Sawyer state that associated reactions occur in 80% of stroke patients with spastic hemiplegia and often interfere with balance and safe mobility [12]. Figures 7.12, 7.13, and 7.14 show the sequential development of activity in the biceps in a patient standing up from a chair. Figure 7.12 reveals some dystonic EMG activity of the biceps and other flexor muscles "at rest" when sitting on a chair. Figures 7.13 and 7.14 reveal increased activity in the biceps during an associated reaction as the patient completes the sequence of standing up. Associated reactions have the potential to occur frequently throughout the day and are important contributors to abnormal postures. Local injection of involved muscles is a treatment consideration.

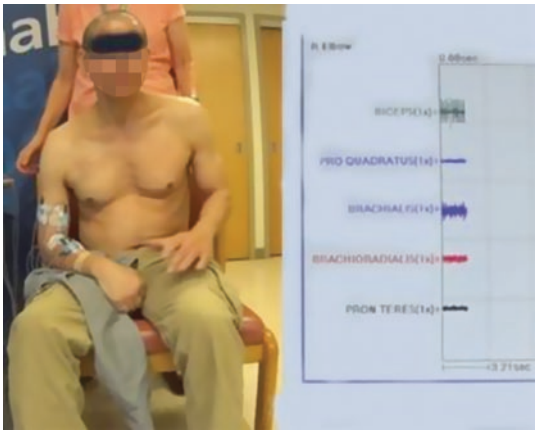

Fig. 7.12 Figures 7.12 through 7.14 show sequential development of an associated reaction during the act of standing up from a chair as reflected in biceps activity in the top trace of the EMG recording on the right side of the image. The patient, sitting quietly on a chair, has low-grade dystonic EMG activity in the biceps (green trace). Note that images were reduced in size to fit together

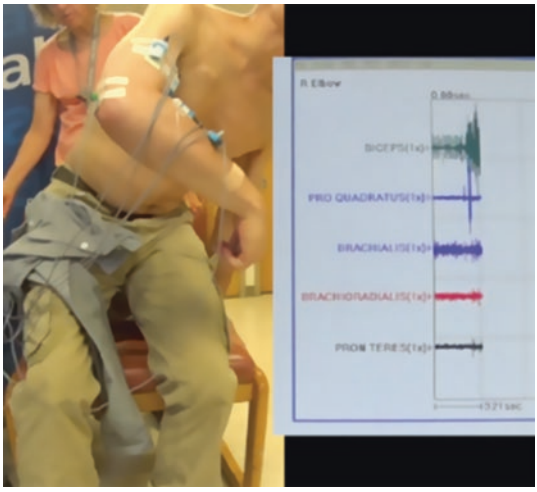

Fig. 7.13 The patient is well on his way toward standing up in this image and the EMG activity in the biceps in the top trace is seen developing more intensely than at baseline when sitting

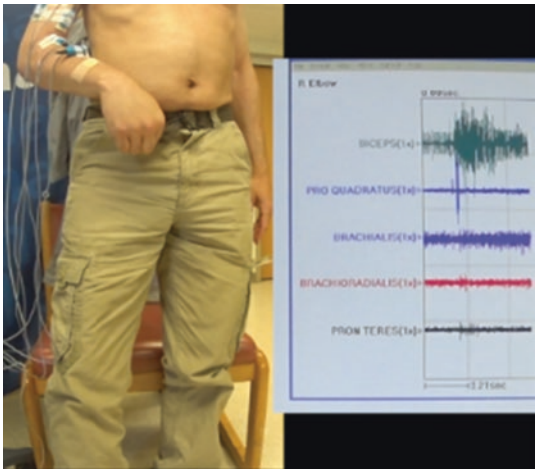

Fig. 7.14 As the person completes standing up, full activation of the biceps can be appreciated on the right side of this image in the top trace. Associated reactions occur commonly during everyday activities and are important contributors to abnormal postures

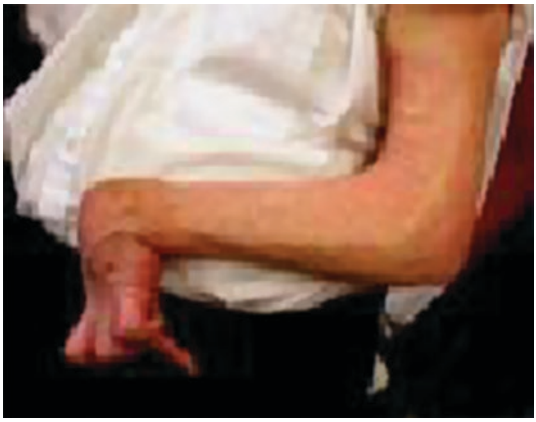

Fig. 7.15 This is a patient with left spastic hemiparesis who developed an ulnar nerve neuropathy due to chronic stretch injury around a persistently flexed elbow. A chronically flexed wrist with subluxation also led to compression of the median nerve in the carpal tunnel. Physical tissue injury is a potential after-effect in patients with spastic paresis

Physical Compression and Injury

Upper limb deformity causes a net imbalance of forces in a given direction and can result in peripheral nerve compression and injury. Figure 7.15 illustrates a patient with left spastic hemiparesis who developed an ulnar neuropathy due to chronic stretch injury around a persistently flexed elbow. A chronically flexed wrist with subluxation also led to compression of the median nerve in the carpal tunnel. Surgical intervention may be necessary in such cases. Serial casting is less successful in cases with prolonged deformity. Trauma is occasionally observed due to elbow flexor spasms that cause the hand to strike the neck, face, and chest. Some patients complain of being awakened at night by elbow flexor spasms that strike their chest or chin. Oral anti-spastic agents such as baclofen or tizanidine may be tried. Focal chemodenervation is also a consideration.

Problems of Access

Access to the Hand, Fingers, and Thumb

Access to the hand is important for object acquisition. Access can be blocked by several types of abnormal hand posturing due to shortening, stiffness, contracture, or overactivation affecting the FDS (see Fig. 7.16), FDP, and the intrinsic muscles of the hand. In addition, the thumb-in-palm configuration may involve the FPL, the flexor pollicis brevis (FPB), the adductor pollicis (AP), and the first dorsal interosseous (FDI) muscles. Mixed FDS and FDP findings for different fingers are not uncommon (see Fig. 7.17). One muscle group may also mask the effect of another. For example, the effect of finger intrinsics can be masked by the stronger contraction of the finger and thumb extrinsics. For example, examination of Fig. 7.17 reveals an "FPL type posture" of the thumb but one cannot rule out involvement of the FPB or, for that

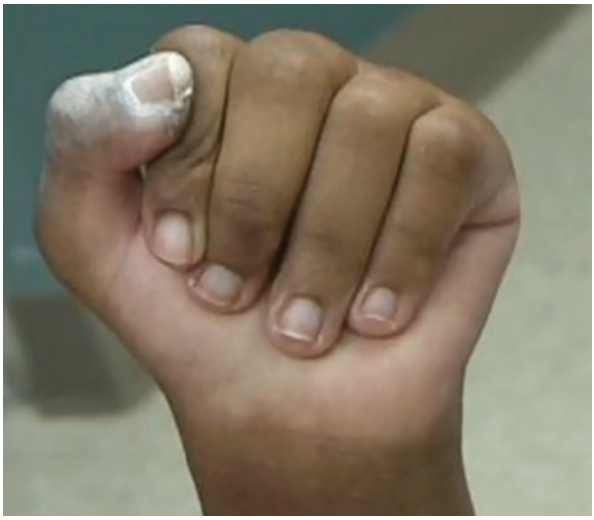

Fig. 7.16 Image of an "FDS type hand" with an "FPL type thumb-in-palm". Access to the hand is blocked by involuntary muscle overactivity or shortening affecting these muscles. *FDS* flexor digitorum superficialis, *FPL* flexor pollicis longus

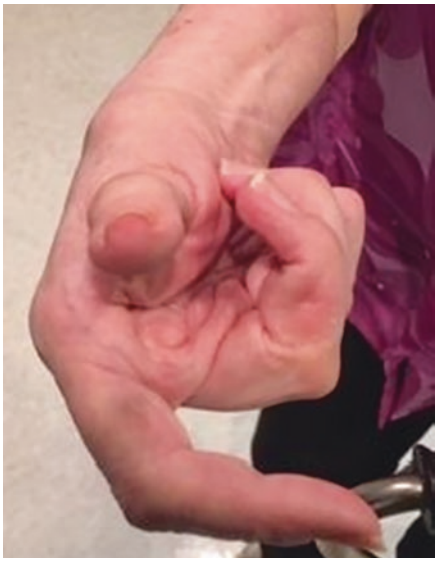

Fig. 7.17 Varied hand configurations can be seen. This figure illustrates mixed involvement of the flexor digitorum superficialis and flexor digitorum profundus muscles of different fingers. Analysis of the various finger configurations will inform the selection of muscles for treatment

matter, the intrinsics of the fingers. Dynamic polyEMG of hand muscles may be useful if there is suspicion of a combined extrinsic/intrinsic hand [13]. When extrinsic finger flexors are weakened by chemodenervation or surgical lengthening, unmasking of overactive intrinsics can result in an intrinsic plus hand (see Fig. 7.18). [An intrinsic plus hand is not common in patients with spastic paresis in our experience.]

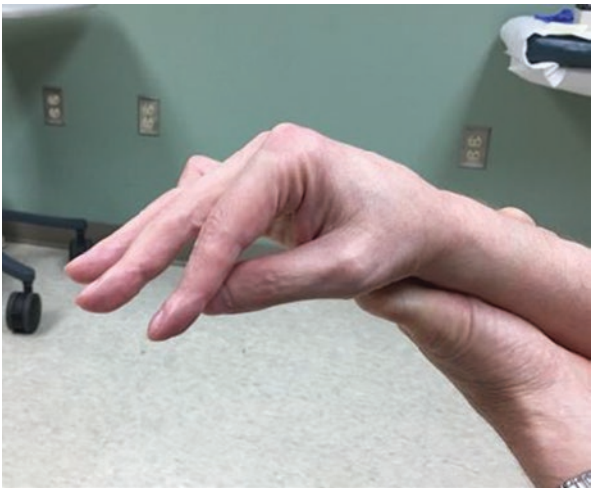

Fig. 7.18 This image shows an intrinsic plus hand involving the fingers and the thumb. The narrowed web space inlet impairs access to the hand, affecting "hand as a holder" function

Impaired access affects "hand as a holder" function. A clenched fist typically involves flexed fingers and a thumb-in-palm deformity and is a common clinical configuration in patients with spastic hemiplegia after a stroke. Sources of flexor muscle overactivity include spastic dystonia, spastic co-contraction, and associated reactions. Changes in the rheologic properties of muscle tissue including shortening, stiffness, and contracture contribute as well. An additional key finding is marked paresis or frank absence of voluntary finger extension. Nevertheless, an object can be inserted, pushed, or wriggled into a clenched fist for the functional purpose of holding. We term this motor behavior "hand as a holder" function. Treatment that reduces the degree of clenching of the fingers may allow insertion of larger sized objects into the affected hand, thus increasing its functional capacity.

The difficulty experienced by persons with a clenched fist and thumb-in-palm configurations is based on impaired or absent volitional activation of extensors and involuntary rheologic stiffness of flexor tissues along with flexor muscle overactivity. To improve access to the clenched fist, the cause must be treated and the contracture may be mitigated with serial casting or splinting [14]. Improving access to the hand by easing the intensity of a clenched fist helps make the "hand as a holder" function feasible.

Access to the Elbow Crease

Chronic elbow flexion driven by spastic dystonia, flexor spasm, and/or associated reactions often results in progressive skin overlap at the elbow, making access for washing the elbow crease difficult, leading to maceration, skin irritation and breakdown. Figure 7.19 illustrates the resting flexion posture of the elbow as well as the large degree of lost range of elbow extension. The goal of treatment is to improve

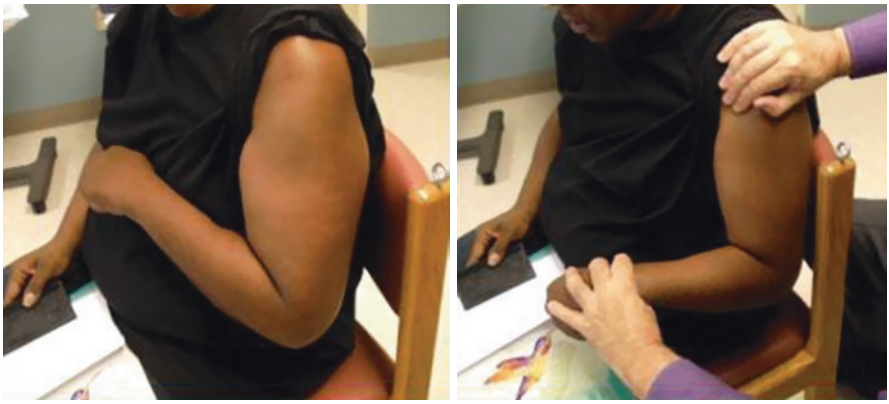

Fig. 7.19 Access for washing the elbow is made difficult by chronic elbow flexion, driven by spastic dystonia, flexor spasm, and/or associated reactions, as well as changes in the rheologic properties of muscle tissue including shortening, stiffness and contracture, leading to progressive, persistent skin overlap at the elbow. Dressing function may be affected as well

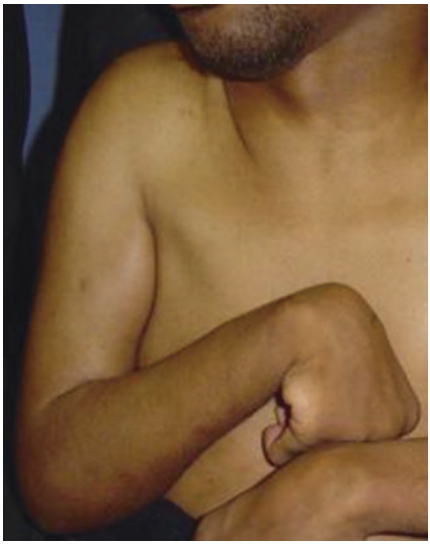

Fig. 7.20 Involuntary shoulder adduction/internal rotation often blocks access to the axilla, making washing and deodorant application difficult. Muscles that may be involved include the pectoralis major, teres major, latissimus dorsi, and subscapularis

the range of motion to a point where air has access to the elbow crease at all times. Local injection to reduce the tension in the elbow flexors followed by serial casting or surgical lengthening are treatment considerations.

Access to the Axilla

Shortening and/or involuntary shoulder adduction/internal rotation often block access to the axilla, making washing and deodorant application difficult (Fig. 7.20).

Muscles that may be involved include the PM, TM, LD, and subscapularis. Phenol injection is a consideration because the PM, TM, and LD muscles are innervated by motor nerves and the clinician does not have to worry about sensory dysesthesia induced by phenol. Reasons to use phenol are the cost of the drug and saving on the amount of neurotoxin that needs to be expended for large proximal arm muscles when there may be many distal arm muscles and leg muscles that also require neurotoxin injections in the same patient. However, phenol is a neurolytic agent which can cause inflammation and fibrosis, making it difficult (after a number of injections) to find the motor nerve with electrical stimulation for future treatments [15].

Grasp and Release Dysfunction

Problems of hand opening are typically related to the triad of muscle shortening, weakness of extrinsic finger extensors, and overactivity of extrinsic finger flexors. In the polio era, paralysis of finger extensors never led to a clenched fist deformity because the finger flexors were never subjected to involuntary neural overactivity. However, spasticity by itself is also insufficient to lead to a clenched fist. Rather, other neural phenomena such as co-contraction and associated reactions occurring many times during the day along with non-neural phenomena such as muscle shortening, stiffness, and contracture due to lack of mobility summate leading to the grasp and release dysfunction.

Inadequate Hand Opening Preceding Grasp

Normally, the reach phase prior to grasp is characterized by active finger extension [16]. The fingers and thumb extend and the hand opens in proportion to the size and configurational properties of the target object. The left side of Fig. 7.21 reveals the

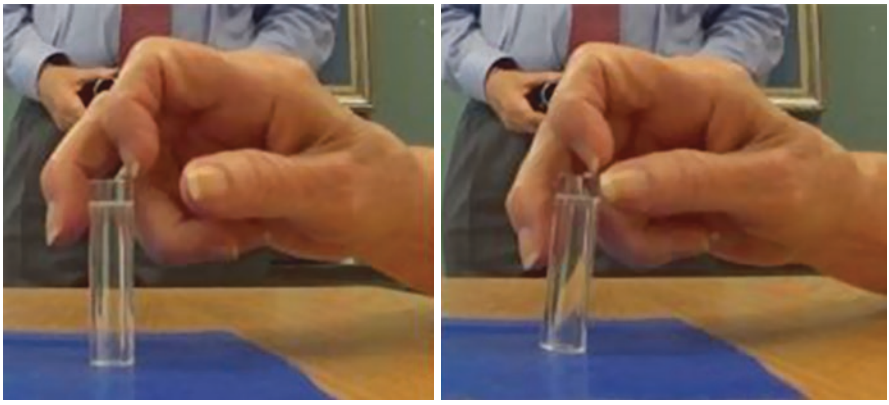

Fig. 7.21 During the pre-grasp phase of reaching, the fingers and thumb normally extend as the hand opens in proportion to the size and configuration of a target object. In this patient, the left image reveals an excessively flexed index finger during pre-grasp. The right image reveals that the index finger cannot participate in grasp of the cylinder, due to being excessively curled and past the locus of contact with the object

hand of a patient approaching a small 0.5″ diameter cylinder. Note that the index finger is excessively curled prior to grasp such that the cylinder cannot be grasped as seen on the right image of Fig. 7.21. In fact, the patient tipped the cylinder over when contact was made with the fingers and thumb (see Fig. 7.22). The motor behavior that knocked the cylinder over is analogous to the problem of lack of adequate clearance commonly seen in the lower limb of hemiparetic patients (see later in the chapter). In this case, the patient chose to retrieve the fallen cylinder using her thumb and long finger as seen in Fig. 7.23. The patient finally grasped and

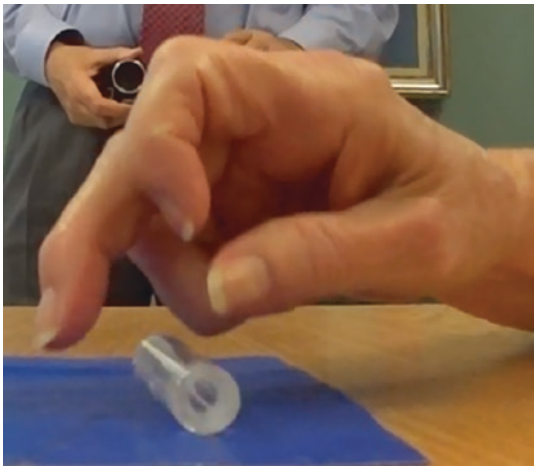

Fig. 7.22 The patient tipped the cylinder when initial contact was made with the hand

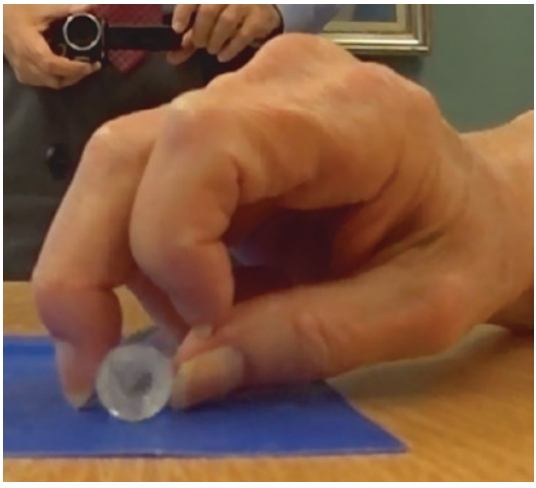

Fig. 7.23 The patient chose to retrieve the fallen cylinder using her thumb and long finger

lifted the cylinder, but the grasp appears tenuous and dysfunctional as seen in Fig. 7.24. The thumb nail rather than the thumb pad provides a partial counterforce to contact pressure with the flexed distal interphalangeal joint (DIP) joint of the long finger. The index finger is curled and makes no contact. Somewhat hidden in the photo is the middle phalanx of the ring finger which provides the other counterforce to the contact pressure of the long finger pressing on the cylinder. Objects of larger size cannot be picked up at all, as seen in Fig. 7.25.

It is clear from the figures that the FDP of the index finger is involved but the clinician has to infer the cause. Decisions about whether muscle shortening and/or overactivity are driving excessive thumb flexion and ring finger proximal interphalangeal (PIP) joint flexion need to be made. Treatment with neurotoxin is often effective in mitigating muscle overactivity but selecting muscles relevant to the

Fig. 7.24 The patient finally grasped and lifted the cylinder, but the grasp appears tenuous because the long finger compresses the cylinder against the flexed middle phalanx of the ring finger, the thumb tip rather than the pad is in contact with the cylinder, and the index finger remains excessively curled "above the fray"

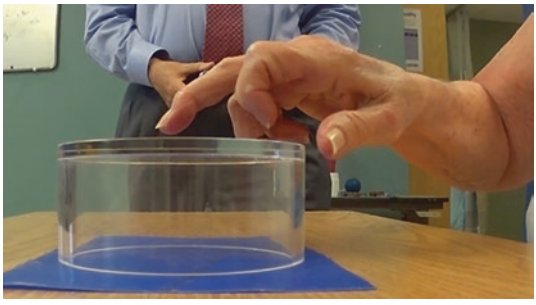

Fig. 7.25 The patient was unable to grasp a 4.5 inch diameter cylinder. Impaired access to the hand makes it difficult to pick up large diameter objects

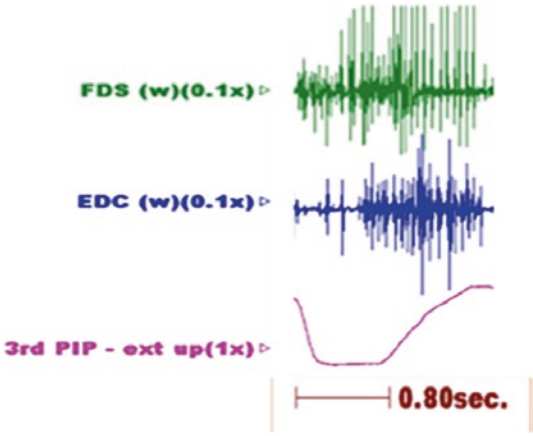

Fig. 7.26 This polyEMG record (image enlarged) reveals spastic co-contraction of the flexor digitorum superficialis (FDS, top EMG trace) during attempted finger extension by the extensor digitorum communis (EDC, middle EMG trace) that slows down finger extension of the 3rd proximal interphalangeal (PIP) joint. A patient's inability to relax involuntary overactivity of finger flexors prevents object release in many patients. Difficulty releasing is usually greater for larger sized objects than smaller ones

clinical problem is key to decision-making so as to not cause weakness. When injecting neurotoxin, consider electrical stimulation to isolate parts of the FDS and FDP muscles controlling the relevant fingers.

Inadequate Hand Opening During Release

The normal release of a handheld object is characterized by relaxation of the finger and thumb flexors followed immediately by active extension of the fingers and thumb. Patients with spastic paresis often have difficulty releasing a grasped object. Figure 7.26 reveals slowed finger extension after prior flexion due to persistent spastic co-contraction of the FDS. Difficulty releasing is usually greater for larger sized objects than for smaller ones, presumably because the larger object stretches the shortened flexors more than smaller objects. When finger extensors are very weak, the patient may let the object slip out of the hand by gravity. Weak finger extension may be aided by a spring-loaded orthosis that works as an extension assist [17].

Hyperextended Wrist and Finger Flexor Tenodesis

A hyperextended wrist produces a clenched fist by the mechanism of finger flexor tenodesis [18]. Figure 7.27 illustrates the tenodesis effect of wrist angle on finger configuration. When the wrist is flexed, the rheologic tension in the finger flexors is reduced because the finger flexors are slack. When the wrist is extended, the rheologic tension in the finger flexors is increased due to stretch of the finger flexors across the wrist. Even when there is no neural input to the finger flexors, the fingers will flex toward the palm because of rheologic tension in the finger flexors when the wrist extends. Shortening of the finger flexors, the development of stiffness, and contractures due to disuse can further exacerbate the tenodesis effect. The finger

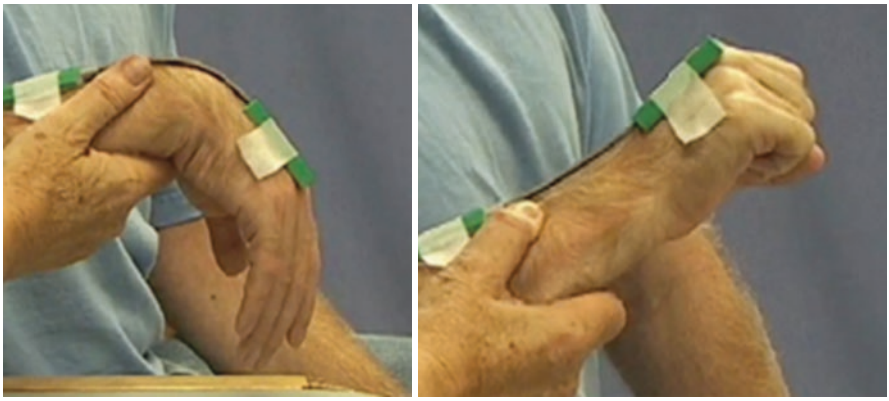

Fig. 7.27 A hyperextended wrist produces a clenched fist by the mechanism of finger flexor tenodesis. This image illustrates the tenodesis effect of wrist angle. When the wrist is flexed, rheologic tension of the finger flexors is reduced because the finger flexors are not stretched. When the wrist is extended, rheologic finger flexor tension increases due to stretch of the finger flexors across the wrist, which is exacerbated by muscle shortening, stiffness, and contracture

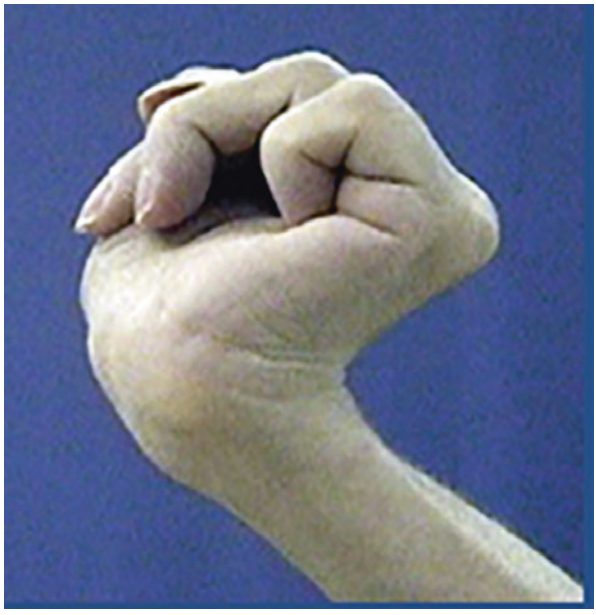

Fig. 7.28 This figure illustrates a patient with cerebral palsy whose hyperextended wrist resulted in tenodesis-driven finger flexion. Initial treatment must be aimed at restoring wrist flexion before treatment of the finger flexors

flexors may or may not be neurally overactive in such cases and, unless clinical examination is highly suggestive of spastic dystonia or a dynamic EMG study is available to ascertain FDS/FDP overactivity, weakening the finger flexors further may not be worthwhile. Figure 7.28 illustrates a patient with cerebral palsy whose hyperextended wrist resulted in tenodesis-driven finger flexion. Shortening and/or overactivity of the wrist and finger extensors may be responsible for the excessive

finger flexion in this case and must be treated first to restore range of motion at the wrist.

Joint and Muscle Contractures

Contracture is often a factor that makes for difficulty with hand opening. A clinician needs to distinguish between two types of contracture: contracture of joint capsules and fixed shortening of muscle tissue. Tendons do not shorten. In actuality, other tissues such as connective tissue, vascular tissues, and nerve tissue are also subject to contracture by unremitting joint configurations. It is for this reason that a surgeon, for example, will not lengthen contractured elbow flexors all the way because strain damage to arteries, nerves, and other soft tissue is likely to occur if the joint is pulled into extension completely [19]. Lengthening is performed for "half the distance to the goal line" with the rest of the contracture worked out post-operatively by serial casting or splinting. We note that joint contractures of the fingers are unrelated to wrist position, whereas finger flexor muscle contractures are tightened by wrist extension and loosened by wrist flexion with the range of motion worsening or improving accordingly due to the tenodesis effect. The examiner keeps this in mind when distinguishing between joint and muscle contracture, noting that combined contracture of joints and muscles may frequently occur. The distinction between joint and muscle contractures is important for both conservative and surgical treatment. Neurotoxin injections do not help joint contractures. The association between immobility, accumulation and aggregation of extracellular hyaluronan in muscle, development of muscle stiffness, and contracture can explain the prevalence of finger flexion contractures [20]. Also see Chaps. 5 and 6 for non-neural changes in muscles, and Chaps. 8 and 13 for treatment of muscle shortening with pharmacologic release using hyaluronidase injections.

The Intrinsic plus Hand

When extrinsic finger flexors and extensors are very weak, the presence of voluntary intrinsic muscle activation results in an intrinsic plus hand. This is a useful hand for grasp and release but it is not a powerful hand. In addition, the web space inlet is relatively narrow so that grasp of larger sized objects may be limited.

An intrinsic plus hand (see Fig. 7.29) is sometimes seen after surgical lengthening of the extrinsic finger flexors. Figure 7.30 shows a patient who had a markedly flexed elbow, flexed wrist, clenched fist, and thumb-in-palm. Elbow flexors, wrist flexors, and extrinsic finger flexors were surgically lengthened. However, post surgery, the opposite deformities developed, namely, an extended elbow (patient couldn't flex the elbow actively), an extended, radially deviated wrist (patient could not flex the wrist actively), intrinsic plus fingers, and an intrinsic thumb. The patient could hold small objects that were placed in her hand, but she could not bring them to her body because she lacked elbow flexion. Consequently, she did not use the hand or upper limb very much. However, dressing the limb became easier because she could pull her sleeve up her straight arm more easily than up the markedly flexed elbow that she had prior to surgery. The reason for development of the opposite deformities after surgery was that prior to surgery, she actually had substantial

Fig. 7.29 Image of an intrinsic plus hand that developed after surgical lengthening of the extrinsic finger flexors for a clenched fist

Fig. 7.30 This figure shows a patient who had a markedly flexed elbow, flexed wrist, clenched fist, and thumb-in-palm that were treated with surgical lengthening. Post surgery the patient developed opposite deformities with elbow extension, wrist extension, and an intrinsic plus hand

triceps muscle activity, wrist extensor activity, and intrinsic muscle activity. However, the neural activity of these muscle groups was masked by the much stronger activity of elbow flexors, wrist flexors, and extrinsic finger flexors. It was not anticipated that opposite deformities would occur. Nevertheless, this case serves as a cautionary tale as clinical postures reflect a net balance of forces around a joint. Weakening one muscle set of dominant forces can lead to releasing the effect of

active antagonist muscles, resulting in opposite deformities. In this regard, dynamic polyEMG of agonists and antagonists around a joint may provide useful pre-operative information. Clinical postures may also represent non-neural changes in muscle that should be treated first prior to considering surgical interventions that may not lead to functional benefit.

The Flexed Thumb

The thumb is flexed by the FPL (Fig. 7.31) and FPB muscles (Fig. 7.32). The FPL is an extrinsic muscle and is subject to tenodesis tightening by wrist extension. Testing of Ashworth scores without maintaining a consistent wrist position can confuse the picture regarding which muscle is causing the thumb to flex (this is true for the finger flexors as well). Flexion of the interphalangeal (IP) joint of the thumb reflects an overactive FPL. Weakness of the extensor pollicis longus (EPL) muscle plays a negative role as well. Flexion of the IP joint of the thumb narrows the web space inlet, affecting "hand as a holder" function. An overactive FPB results in flexed metacarpophalangeal (MP) and carpometacarpal (CMC) joints, the IP joint typically being extended. The thumb ends up well into the palm, reducing access, and consequently compromising holding function (Fig. 7.32). Overactivity or shortening of the AP and FDI muscles adds to the problem.

Distinguishing between the "FPL type" deformity and the "FPB type" deformity which also involves the AP and FDI is imperative for the selection of the appropriate muscles for treatment. Thumb muscles, like other muscles elsewhere, are subject to non-neural rheologic changes, i.e., muscle shortening, stiffness, and contracture and need appropriate early treatment. Serial casting or splinting may be needed.

Fig. 7.31 This patient had a flexed thumb of the ''FPL type''. The thumb with a flexed interphalangeal joint has a narrow web space inlet, affecting "hand as a holder" function. *FPL* flexor pollicis longus

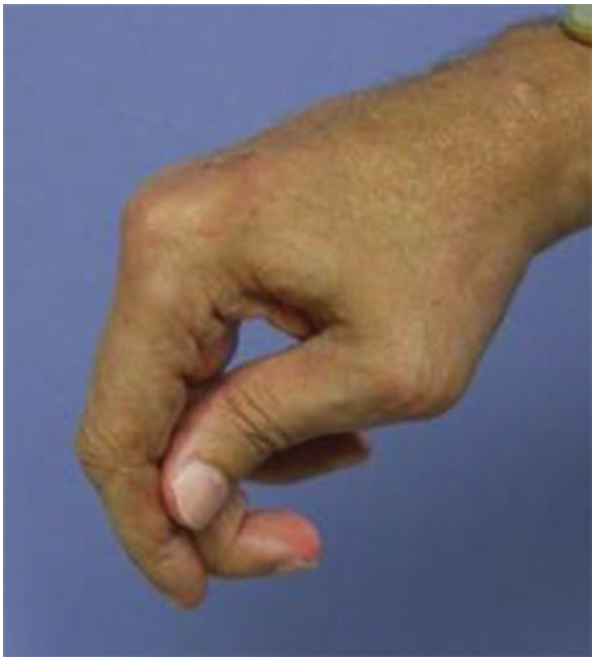

Fig. 7.32 Image of an "FPB type" thumb. An overactive flexor pollicis brevis (FPB) results in flexed metacarpophalangeal and carpometacarpal joints, the interphalangeal joint typically remains extended. The thumb ends up well into the palm, reducing access, and consequently compromising holding function

Neurotoxin injections of thumb flexors may be able to mitigate muscle overactivity but weakness of thumb extensors does not allow the thumb to be a post against which the fingers can compress an object for holding. If this happens, the use of a thumb spica or other type of orthosis for posting the thumb can go a long way toward restoring the function of hand grasp.

Reach Dysfunction

Reaching, as a motor behavior, involves the whole limb. Individuals may have some degree of two-way volitional movement of a given joint when tested in isolation but individuals with spastic paresis may or may not have such movements available for whole limb reaching. However, not all reaching movements are the same because depending on the intent and direction of the reach, the specific muscle ensembles involved are different. Hence, "reaching for a cup" is different from "reaching for a pen," which is different from "reaching for a ball on the right side of the body" [21]. In addition to single joint movements, traditional manual muscle strength testing is also not likely to be predictive of whole limb behavior. Instead, direct testing of tasks that require whole limb actions is recommended, such as reaching to touch a target with the fist or reaching to pick up an object with the hand. The target and the object may be placed sequentially on the left, center, and right side of the individual to allow the observer to make inferences about scapula, shoulder, elbow, forearm, wrist, finger, and thumb movements during whole limb behavior. For the clinical

purpose of evaluating whole limb motor behavior in spastic paresis, it is helpful to ask the following questions: (1) Is weakness of one or more components of whole limb movement impeding a specific task? (2) Is co-contraction of muscles across a joint restraining the movement or movement components of a given task? Or (3) are the movement responses the same (or very similar) and stereotypical regardless of the task? See Chaps. 2 and 3 for a full description and functional assessments in spastic movement disorder.

Deficits in reaching behavior may occur due to a primary problem at the scapula, shoulder, elbow, and/or forearm, and secondarily at the wrist, fingers and thumb. Impairment of the latter segments mainly affects grasp and release. Since the purpose of a reaching movement is to place the hand at a given location in space, limitation of movement at one or more joints can affect it.

Inadequate Scapular Rotation

Forward reaching of the arm requires protraction of the scapula, shoulder flexion, and elbow extension. Hand placement across the midline to the contralateral side requires the most protraction of the scapula, and shoulder adduction. Clinical testing of scapular motion can best be observed by asking the patient to place the hand contralaterally on a tabletop or on the opposite knee.

An important agonist of scapular motion is the serratus anterior, the prime protractor of the scapula which enables scapulothoracic motion and forward movement of the arm. Weakness or stiffness in the serratus anterior impairs reaching. The pectoralis minor and major also contribute to protraction of the scapula. The retractors of the scapula are primarily the rhomboids and middle trapezius. During forward reach with the unaffected left arm, note appropriate protraction of the scapula on the left image of Fig. 7.33. The image on the right reveals a retracted scapula on the affected right side. Treatment considerations may include aggressive stretching of muscles retracting the scapula as well as injection treatment to assist with lengthening these muscles. When the scapula does not move during reaching, i.e., when there is no retraction or protraction, a clinician must decide whether this is due to weakness or stiffness in the agonists and/or antagonists, and the net balance of the agonist and antagonist muscles.

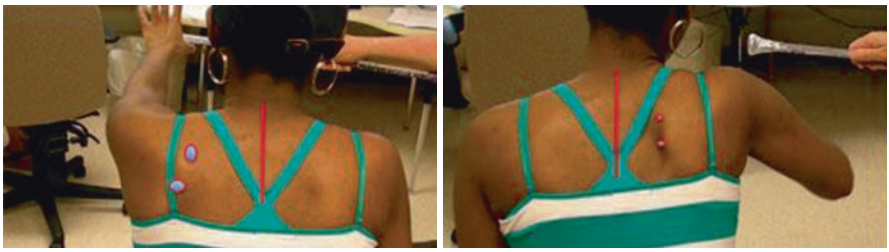

Fig. 7.33 During forward reach of the arm by a patient with right hemiparesis, the left image shows protraction of the scapula on the unaffected side while the right image reveals a retracted scapula on the affected right side which impedes forward reaching

Inadequate Shoulder Movement

Reaching in front of the body requires shoulder flexion and elbow extension against gravity. Therefore, the muscles involved must necessarily activate anti-gravity muscles to support the weight of the arm against gravity and also control the direction of limb placement in forward locations. Shoulder flexion generates upward and forward motion of the arm and may be accompanied by shoulder internal rotation and adduction for hand placement on the contralateral side of the body, or by external rotation and abduction for hand placement away from the body. Clinical testing of shoulder motion can be performed by hand placement at various locations on a tabletop (see placement of objects recommended for the Frenchay Arm Test in Chap. 3). Support of the forearm on a table eliminates gravity to facilitate examination of the directional control of movement. Shoulder external rotation is tested by asking the patient to bring the hand behind the neck or by demonstrating the prethrowing motion of a football quarterback. Shoulder internal rotation, important for toileting, can be tested by bringing the hand behind the low back. When patients with spastic paresis have a fair degree of recovery, voluntary alternating movements of the shoulder, namely, flexion/extension, abduction/adduction, external/internal rotation movements may reveal potential temporal asymmetries such as slower flexion phase compared to the extension phase, slower abduction compared with adduction, and slower external rotation compared with internal rotation (or vice versa). Temporal asymmetry can point to restraint by co-contracting antagonists or to stiffness that may be palpable by the clinician. It might also reflect weakness of specific agonist muscles rather than antagonist restraint (see Step 5 of the Five-Step Assessment in Chap. 3).

Because the shoulder joint is a shallow ball-and-socket joint with three degrees of freedom, it enables hand placement in the workspace around the body. In spastic paresis, weak descending signals to synergistically acting shoulder muscles result in impairment of hand placement. This can be tested clinically by placing targets to the left, right, and center of the body on a tabletop. When a patient lifts the arm to reach toward a target, two operational problems are present: (1) support of the arm against gravity and (2) placement of the hand in a specific location in the workspace. When descending signals are weak, lifting the arm against gravity becomes strenuous and one can observe all kinds of effortful attempts by the patient to lift and hold the arm against gravity. Effortful behavior tends to activate many compensatory muscles farther away from the more localized muscle ensemble that is activated normally. Such activation may interfere with hand placement because the same muscles may have dual anti-gravity and placement roles. The first goal is to enable arm support against gravity by strengthening muscles that support the weight of the arm. The second goal is restoration of directional control by performing exercises that enable reaching targets in the workspace.

Shortening or co-contraction of shoulder adductors and extensors often leads to restraint of voluntary movement. Figure 7.34 reveals a young woman who has difficulty placing her hand behind her neck. The movement ordinarily requires shoulder abduction by the deltoids and external rotation by the rotator cuff muscles. Shortened or overactive internal rotators of the shoulder including the PM, LD, TM, and

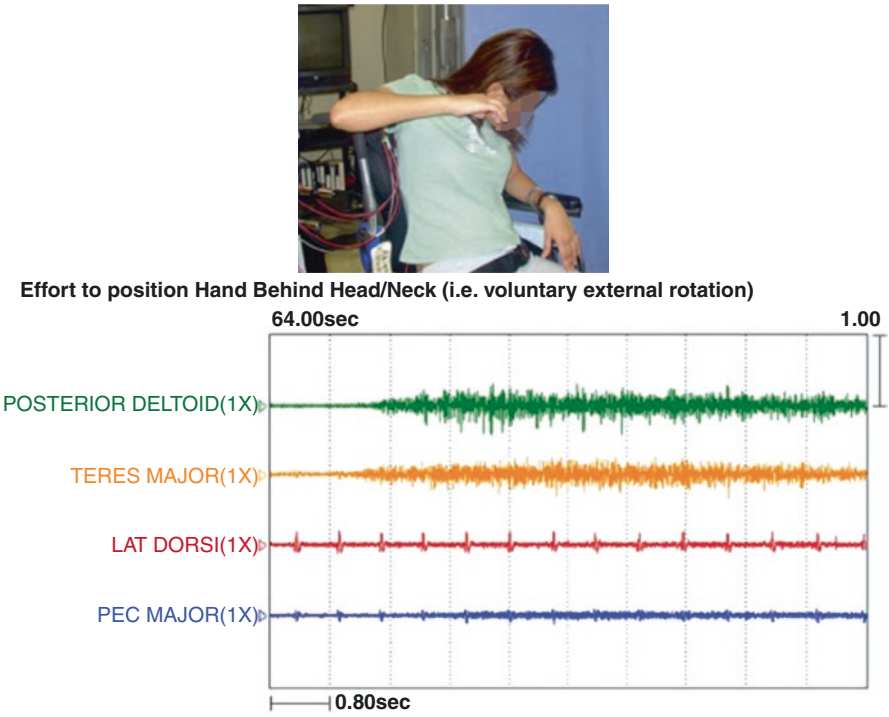

Fig. 7.34 Co-contraction of shoulder adductors often leads to restraint of voluntary movement. This figure reveals a young woman who has difficulty placing her hand behind her neck. The poly-EMG record reveals co-contraction of teres major, an adductor and internal rotator of the shoulder. Latissimus dorsi was not active and pectoralis major was only minimally active. *LAT DORSI* latissimus dorsi, *PEC MAJOR* pectoralis major

subscapularis can stymie this task. In this case, the figure reveals co-contraction of the TM and posterior deltoid (PD) muscles. There was no activation of LD or PM and we do not know whether there was co-contraction of the subscapularis (not recorded) muscle. Weak signals to the external rotators may also be contributory although activation of the PD, an abductor and external rotator of the shoulder, was good. The limitation of dynamic EMG is that it does not reflect the degree of force being generated which must be inferred by the examiner. On the other hand, if one were to treat shortening or overactivity, a good first approach would be to inject the TM based on noted activation of this internal rotator with no activation of other internal rotators (LD and PM). Admittedly, dynamic EMG is not a commonly available tool. Hence, clinical examination including observation and palpation of muscles and tendons must necessarily be relied upon to make inferences regarding the appropriate muscles to select for treatment [22]. It is important to keep in mind that spastic paresis results in variable agonist and antagonist muscle weakness and restraint.

Temporary diagnostic nerve blocks may also be helpful in identifying involuntarily overactive shoulder muscles that restrain movement or cause dysfunctional

posturing. Figure 7.35 reveals a patient with stroke who complained of persistent posterior posturing of her shoulder when she walked. Clinical examination revealed a stiff LD by palpation of the muscle and its taut tendon in the axilla. A diagnostic lidocaine block of the thoracodorsal nerve quickly eliminated the posturing, and it was inferred that no other extensors were involved. Phenol application to the thoracodorsal nerve (an entirely motor nerve without sensory component) was performed for long-term treatment of the problem.

More than one shoulder extensor may be co-contracting. Figure 7.36 reveals the polyEMG record of a patient with spastic paresis who had great difficulty performing shoulder flexion. Although he could initiate some flexion at the shoulder, the movement flattened out. The agonist muscle, anterior deltoid, showed a good EMG recruitment pattern. However, co-contraction of the TM and long head of triceps, a two joint muscle that extends the shoulder when the elbow is fixed by flexors, negated the effect of activation of the anterior deltoid in flexing the shoulder. Some activation of the LD, another shoulder extensor, can be seen as well. Treatment of multiple muscles should be considered.

Weakness in the shoulder external rotators and abductors favors the development of stiffness and contracture in the opposing shoulder internal rotators and adductors. Precursors of rheologic change in these muscles include immobility due to severity of the spastic paresis as well as neural phenomena including spastic dystonia, spastic co-contraction, flexor reflex afferent activity, and associated reactions, which bias the shoulder joint toward internal rotation and adduction. The weak descending signals to the external rotators/abductors are insufficient to redress the bias. Two of the internal rotators/adductors are also shoulder extensors (specifically, the LD and TM) so that forward motion of the arm, necessary for reaching, may be significantly affected by extensor shortening and stiffness. Unlike elbow, forearm, wrist, and finger

Fig. 7.35 This figure reveals a patient with stroke who complained of persistent posterior posturing of her shoulder when she walked. Clinical examination suggested an overactive latissimus dorsi by palpation, and a diagnostic lidocaine block of the thoracodorsal nerve quickly eliminated the posturing. Temporary nerve blocks may be useful in predicting the outcome of longer-term treatments

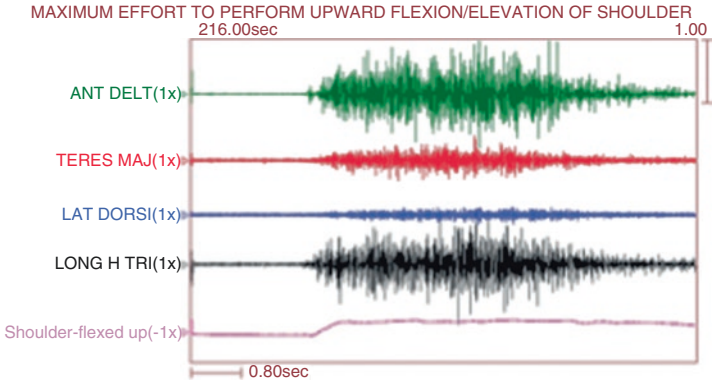

Fig. 7.36 More than one shoulder extensor may be co-contracting. This figure reveals the poly-EMG record of a patient with spastic paresis who had great difficulty performing shoulder flexion. The record reveals co-contraction of multiple shoulder extensors including the teres major and long head of triceps with some activation of the latissimus dorsi as well. *ANT DELT* anterior deltoid, *TERES MAJ* teres major, *LAT DORSI* latissimus dorsi, *LONG H TRI* long head of triceps

contractures, shoulder contracture is not amenable to serial casting. Prevention of such contractures requires positioning the shoulder in external rotation starting early on, and facilitation of shoulder external rotation movements [23, 24]. Surgical intervention is a consideration, especially when skin problems in the axilla are persistent.

Inadequate Elbow Movement

Elbow extension lengthens the upper limb during reaching and elbow flexion shortens it when bringing the hand to the body. A combination of scapular protraction, shoulder flexion, and elbow extension are key movements that place the hand in various spatial locations away from the body.

Paresis of the elbow extensors (triceps and anconeus) results in an impairment of elbow extension during reaching. The patient in Figs. 7.37 and 7.38 illustrates impaired length of reaching in the left paretic upper limb compared with the right. The elbow flexors were not co-contracting or stiff on clinical palpation. Shoulder flexion, tested separately, was good. Incomplete elbow extension was attributed to weak activation of the triceps.

In patients with spastic paresis, it is common for shortening and/or spastic co-contraction of elbow flexors to restrain elbow extension [25]. The result is a strained, slow, and effortful movement which may result in incomplete extension (see Fig. 7.39). The underlying dynamic EMG is seen in Fig. 7.40. Triceps and anconeus are active during the extension phase while biceps has the most activity in the flexion phase. However, antagonist activity in biceps, brachialis, brachioradialis, and pronator teres during the extension phase restrains active elbow extension. Restraint can be seen in the prolonged extension phase compared with the smooth, rapid flexion phase of the movement. Release of the restraining muscles, particularly of the brachioradialis and pronator teres, should be considered.

Fig. 7.37 Normal elbow extension during full arm reach

Fig. 7.38 In this figure, paresis of elbow extensors (triceps and anconeus) results in an impairment of elbow extension during reaching. The elbow flexors were not co-contracting and the shoulder flexors, tested separately, were not weak either

A flexion contracture blocks full reaching of the upper limb. Figure 7.41 reveals the impact of a flexion contracture of the elbow in a patient with good neurological recovery from right hemiparesis but with a residual elbow flexion contracture. The patient had to shorten his step length because elbow flexion limited his ability to extend the walker forward during limb advancement. Ambulation speed was necessarily slow and his chief complaint was "I walk slowly."

Elbow extensor overactivity and/or contracture restrain active elbow flexion with a major effect on feeding if the patient is otherwise able to do so. Release of the muscle and serial casting are considerations as treatment. Extensor contracture is sometimes seen after surgical release of a severe elbow flexion contracture. The extensor contracture occurs when it is not recognized that extensor overactivity is

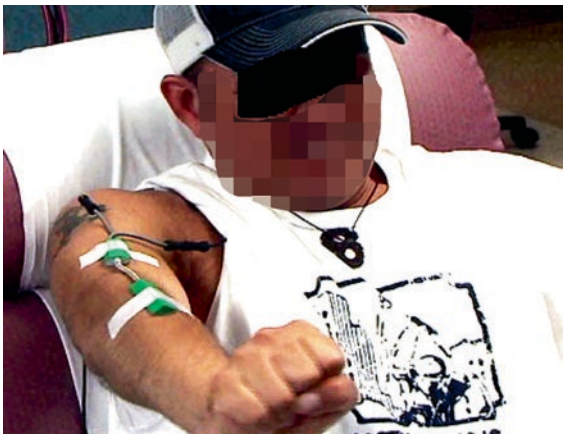

Fig. 7.39 It is common for spastic co-contraction of elbow flexors to restrain elbow extension, resulting in a strained, slow effort to achieve full elbow extension, or more commonly, to achieve only partial elbow extension

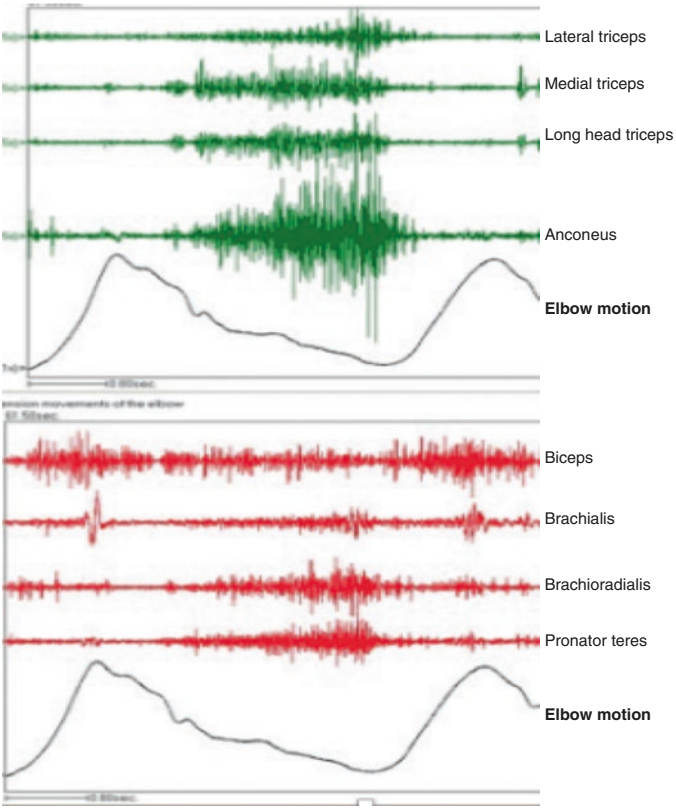

Fig. 7.40 The underlying enlarged dynamic EMG image of the patient in Fig. 7.39 during elbow extension includes agonist activity in the triceps and anconeus muscles (top 4 green traces) during the extension phase along with antagonist activity in the biceps, brachialis, brachioradialis, and pronator teres muscles (bottom 4 red traces), acting to restrain elbow extension. Spastic co-contraction acts like a brake while simultaneously accelerating

Fig. 7.41 A flexion contracture blocks full reaching of the upper limb. This figure shows a patient with spastic paresis who had to shorten his step length because his elbow flexion contracture limited his ability to extend the walker during limb advancement

present at the time of elbow flexion posturing. Surgical release of flexors changes the postoperative balance of forces that will, in some cases, favor elbow extension. In spastic paresis, it is the net balance of forces across a joint that determines clinical posturing. Clinicians should not assume that severe posturing of a joint in one direction means that there is no muscle activation in the other direction.

Inadequate Forearm Orientation of the Hand

The palm-down (overhand) orientation of the hand occurs due to forearm pronation while the palm-up (underhand) orientation requires forearm supination.

Reaching overhand to pick up an object such as a ball involves active pronation (unless the forearm is already pronated). In spastic paresis, a pronated forearm configuration is seen more often than a supinated one (see Fig. 7.42). Shortening and/or involuntary overactivity of the pronator teres is a major contributor to restraint of supination during reaching but the pronator quadratus needs to be considered as well (see Fig. 7.43). Pronator muscle stiffness and contracture frequently develop when volitional supination is weak. Serial casting can be performed for contracture.

Weakness of pronators is associated with involuntary overactivity and/or stiffness of muscles that supinate (biceps, supinator) and can result in a forearm with a

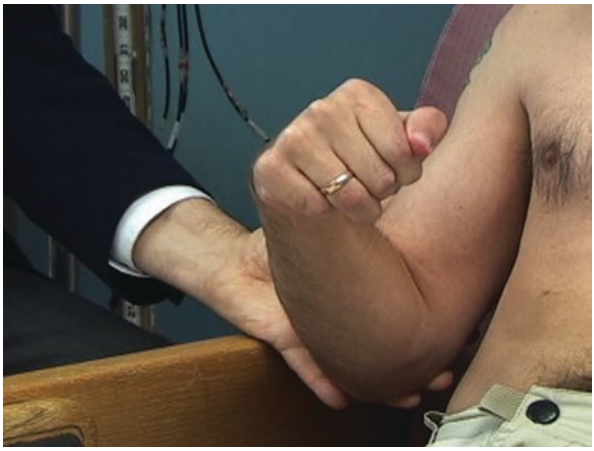

Fig. 7.42 In spastic paresis, a pronated forearm configuration is seen more often than a supinated one. This figure reveals a patient who is attempting voluntary forearm supination

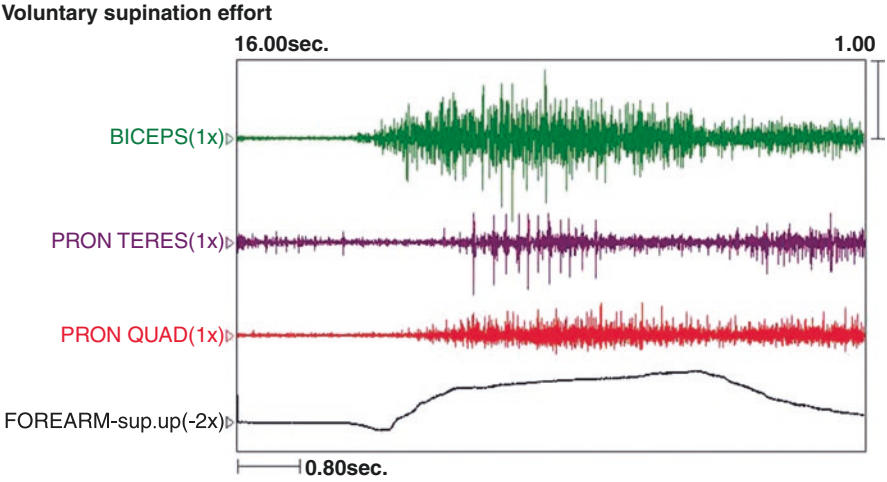

Fig. 7.43 Although the biceps muscle (supinator when the elbow is flexed) is active during supination, so are the pronator teres and pronator quadratus muscles in this polyEMG record corresponding to the patient in the figure above. *PRON TERES* pronator teres, *PRON QUAD* pronator quadratus, *sup* supination

resting supinated configuration. The biceps, in particular, supinates the forearm when the elbow is flexed. A fully extended elbow is supinated by the supinator, not the biceps. If some degree of active pronation is noted clinically, release of the supinating muscles may increase voluntary pronation.

Inadequate Vernier Adjustments of the Wrist

In addition to forearm rotation, the wrist contributes to the orientation of the fingers and thumb to acquire an object. For example, reaching for a small object such as a ball on a table requires wrist, finger, and thumb extension as part of reaching.

Extension of these limb segments typically occurs during the reach phase prior to contact with the object and informs how the hand makes contact and articulates with an object.

The wrist provides vernier (fine adjustment) movements for the reaching arm. This is illustrated in Fig. 7.44 which reveals the flexion adjustment of the wrist required to insert the hand into a receptacle with an upper boundary wall, such as the large box in the figure. A patient who cannot flex the wrist will not be able to make this adjustment as seen in Fig. 7.45.

A more common type of wrist restraint during whole limb movements is co-contraction of wrist flexors that restrain wrist extension. The left image of Fig. 7.46 reveals normal extension of the wrist prior to throwing a ball. The right image reveals a wrist that extends to neutral but is restrained from further extension by spastic co-contraction of the wrist flexors. Co-contraction is a normal phenomenon which refers to simultaneous contraction of the agonist and antagonist muscles by supraspinal drive to stabilize a joint. The co-contracting antagonist muscles are not activated by a stretch reflex but are triggered by their sensitivity to stretch, particularly if the muscle is shortened (see Fig. 7.47). However, it is not easy to

Fig. 7.44 The wrist provides fine adjustment (vernier) for the reaching arm. This figure illustrates flexion adjustment at the wrist required to retrieve a cookie from a large metal box

Fig. 7.45 The patient in this figure could not flex his wrist when he put his hand into the metal box to retrieve a cookie

Fig. 7.46 Wrist restraint during whole limb movements may result from co-contraction of wrist flexors that restrain wrist extension. The left image of this figure reveals wrist extension in the unaffected upper limb prior to throwing a ball. The image on the right side reveals a wrist that extends to neutral but is restrained from further extension by spastic co-contraction of the wrist flexors as illustrated in the next figure

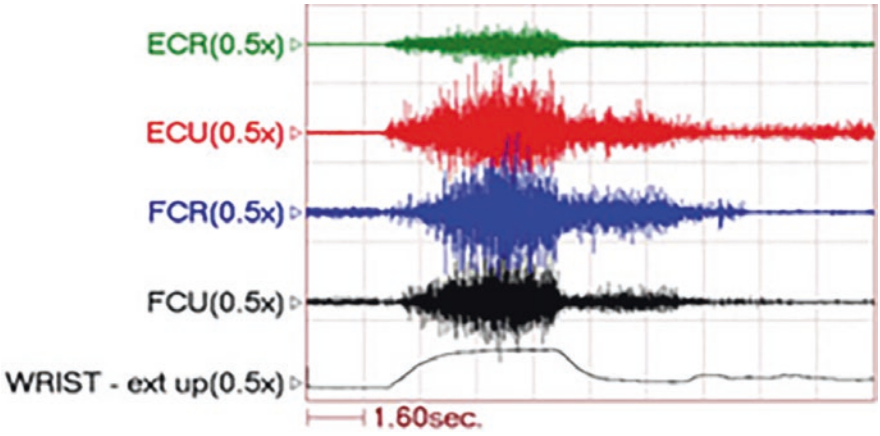

Fig. 7.47 Voluntary wrist extension in a patient with co-contraction of antagonist muscles FCR and FCU accompanying agonist muscles ECR and ECU. Co-contracting antagonist muscles are not initially activated by a stretch reflex but are triggered by their sensitivity to stretch (particularly if the muscle is shortened) and reduce the effect of agonist activation on the movement, hence the term *spastic* co-contraction. *ECR* extensor carpi radialis, *ECU* extensor carpi ulnaris, *FCR* flexor carpi radialis, *FCU* flexor carpi ulnaris, *ext* extension

differentiate whether weakness of the agonist, muscle shortening of the antagonist, or overactivity in the antagonist is the problem. PolyEMG can identify antagonist muscle activity that starts simultaneously with agonist activity, establishing the presence of antagonist co-contraction. Once the movement is underway, superimposed spastic activity due to stretch may or may not contribute.

Functional Consequences in the Lower Limb

The three main functional goals of ambulation are: (1) to move from one place to another, (2) to move safely, and (3) to move efficiently. Gait is characterized by periods of limb loading and unloading in a cyclic and complex pattern. In order to analyze gait, an understanding of basic terminology is needed to identify the components and events of the gait cycle. Considering one limb at a time, the gait cycle has two basic components: a stance phase during which the limb is in contact with the ground, and a swing phase during which the limb is off the ground. During the stance phase, the foot is on the ground for about 60% of a whole symmetrical gait cycle, and the swing phase is approximately 40% of the gait cycle.

The objectives of the stance phase are proper positioning of the limb for stability at initial contact, maintaining a normal loading response, single limb support and forward progression of the body over the stance phase limb, and preparing the lower extremity for the subsequent swing phase. During the swing phase, the objectives are to enable limb clearance and advancement.

Pathological gait can be classified as stance and swing phase abnormalities. For example, observation of hemiparetic gait reveals an overall loss of symmetry with decreased stance time and increased swing time on the affected side, and increased stance time on the unaffected side. A shorter step length on the affected side and increased double support time preserves stability, because more time in double support and more weight-bearing time on the sound limb results in less time spent weight-bearing by the affected limb [26]. Reciprocal arm motion is typically absent or diminished on the affected side, the arm is typically positioned in shoulder adduction and elbow flexion, though it may be flaccid in the early stages of neurological recovery. The affected lower limb appears stiff-legged, showing an extensor synergy pattern with hip extension, adduction, and internal rotation, reduced knee flexion, and plantarflexion/inversion of the foot/ankle (equinovarus). Because extensor posturing lengthens the limb, patients may have difficulty achieving adequate clearance during the swing phase, leading to compensatory maneuvers such as hip hiking (elevation of the hip), circumduction, lateral trunk bending away from the side of the affected swing limb, and less commonly, contralateral vaulting. Initiation of swing phase is delayed, prolonged, or effortful, and is usually associated with a stiff knee and ankle equinus [27].

During stance phase on the affected side, ankle dorsiflexion is decreased at initial contact, and also during the stance and swing phases. Knee hyperextension in the stance phase is seen in most patients, although in some cases, excessive knee flexion is observed. During the swing phase on the affected side, knee flexion is reduced, initiation of hip flexion is delayed, and compensation occurs by means of hip hiking or limb circumduction.

Skin and Footwear Problems

Striatal Toe

Observation of barefoot walking reveals extension of the great toe during the stance and swing phases of gait (Fig. 7.48). When wearing shoes, the patient may report pain at the first metatarsal head, commonly on the tip of the great toe (Fig. 7.49). Shortening or overactivity of the extensor hallucis longus is the mechanism for this problem. However, weakness of the flexor hallucis longus may also contribute. Frequently, patients will complain of pressure on the tip of the big toe resulting from

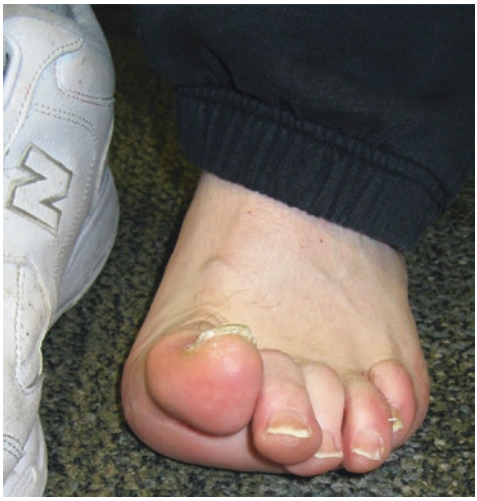

Fig. 7.48 Image of a striatal toe. Observation of barefoot walking reveals that the great toe is held extended during the stance and swing phases of gait

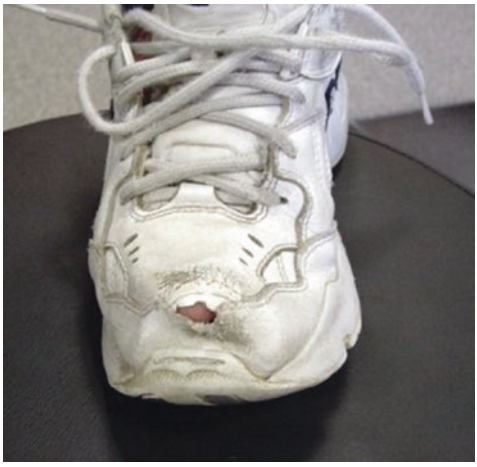

Fig. 7.49 When wearing shoes, the patient with a striatal toe may report pain at the first metatarsal head, commonly on the tip of the great toe

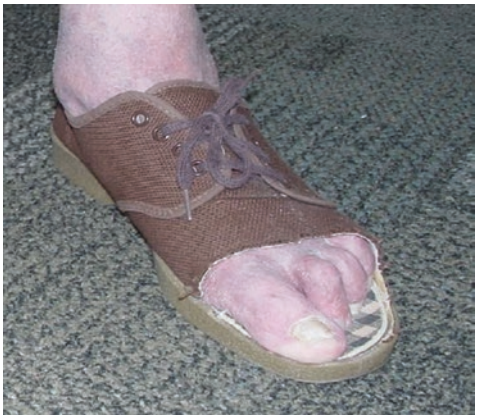

Fig. 7.50 This patient with spastic paresis had painful toe flexion. Observation of barefoot walking revealed the toes to be flexed during the stance phase of gait, producing pain as a symptom that interfered with weightbearing

contact with the top of the shoe's toe box. Ankle equinovarus posture may accompany this deformity, and if present, evaluation of the tibialis anterior, tibialis posterior, gastrocnemius, soleus, and long toe flexors should be performed. Relieving pressure on the tip of the toe inside the shoe can increase comfort.

Painful Toe Flexion

Observation of barefoot walking reveals the toes to be flexed during the stance phase producing pain that interferes with weightbearing (Fig. 7.50). When wearing shoes, the patient may report pain on the dorsal aspect of the toes due to pressure from the shoe's toe box. Pain during standing may impair weightbearing, and interfere with transfers and walking. Shortening or overactivity of the flexor digitorum longus is the cause of this problem. Patients may complain of pressure on the tip of the toes. Evaluation of the long toe flexors should be performed. Pressure relieving toe pads inside the shoe can increase comfort [28].

Problems of Limb Clearance

Stiff Knee with and without Equinovarus

In patients with a stiff knee, the knee is maintained in extension throughout the swing phase. Even when the foot and ankle are adequately positioned, limb clearance can be inadequate due to an inability to bend the swing phase limb. The foot can drag, even if the ankle has adequate dorsiflexion. Compensatory mechanisms involving the trunk, ipsilateral hip (e.g., circumduction), and contralateral limb (e.g., vaulting or early heel rise) may be used by the patient to increase the likelihood of safe ambulation.

Out-of-phase activation of the rectus femoris (RF) during swing phase can be a major contributor to the stiff knee gait, because the RF crosses both the hip and knee joints and can restrict knee flexion. Out-of-phase activation of the vasti (medialis, lateralis, and intermedius) muscles during the swing phase can also contribute to a stiff knee gait. At the hip, a weak iliopsoas muscle can reduce hip flexion in

the swing phase, further impairing knee flexion. Shortening or overactivity of the gluteus maximus and hamstring muscles during the swing phase may restrain hip flexion, potentially causing a stiff knee gait.

If a stiff knee is present in the absence of an ankle deformity, a diagnostic lidocaine block of the motor branch of the femoral nerve to the rectus femoris can be performed to help differentiate the contribution of the knee extensors versus the hip extensor muscles and guide the treatment decisions.

Inadequate Hip Flexion

Limited hip flexion is a common finding in individuals with spastic paresis that has functional implications for limb clearance. Inadequate hip flexion may occur due to hip muscle weakness from prolonged sitting, leading to muscle shortening of the hip flexors or overactivity of hip extensor muscles (e.g., gluteus maximus). During walking, a common penalty is reduced step length due to limited hip flexion and an inability to put the affected limb in front of the unaffected limb during the swing phase of gait. Hip adduction may result from shortening and/or overactivity of the adductor longus, adductor brevis, sartorius, and in some cases, from weakness of the gluteus medius.

Problems of Foot Loading

Loading on the Lateral Border of the Foot

Equinovarus foot deformity is the most frequently seen abnormality in the lower limb in individuals with spastic paresis. The foot and ankle are inverted, pointed downward (plantarflexed), and the toes may be curled as well.

The patient frequently complains of pain on the lateral border of the foot during weightbearing (Fig. 7.51). During loading, contact with the ground occurs first with the forefoot, weight is borne primarily on the anterior and lateral border of the foot and may frequently be concentrated in the area under the fifth metatarsal. Limited dorsiflexion during midstance prevents forward progression of the tibia over the stationary foot leading to increased pressure over the metatarsals, promoting lateral ankle instability and potentially causing knee hyperextension. Compensatory hip flexion may occur to maintain ambulation. The presence of an inadequate base of support may result in instability of the whole body and correction of the problem is essential, even for persons with limited ambulation capacity.

Muscles that potentially contribute to the equinovarus deformity include the gastrocnemius, soleus, tibialis posterior, tibialis anterior, long toe flexors, extensor hallucis longus, and peroneus longus.

Forefoot First Loading

Foot equinus deformity is frequently seen during walking in individuals with spastic paresis. The foot and ankle are plantarflexed prior to initial contact. During loading, the patient first touches the ground with the forefoot and contact with the ground may be primarily or exclusively on the forefoot (Fig. 7.52).

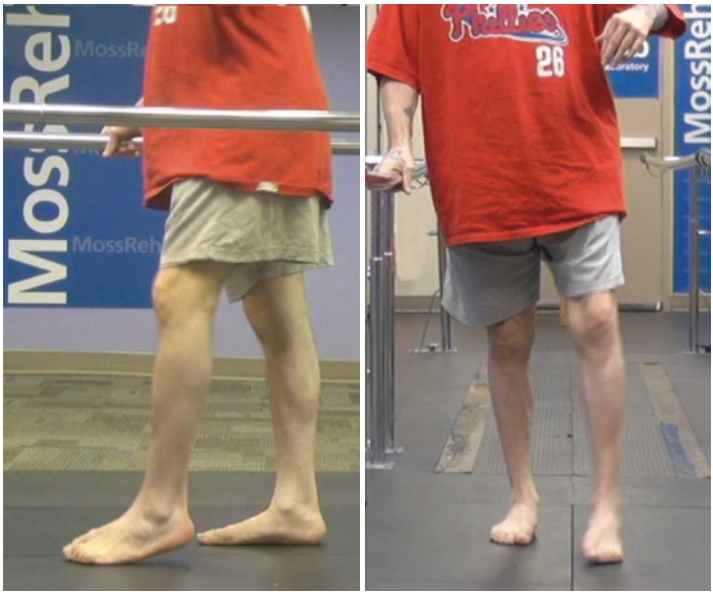

Fig. 7.51 Equinovarus foot is the abnormal posture most frequently seen in the lower limb in individuals with spastic paresis. The foot and ankle are inverted, plantar flexed and the toes may be curled as well. The patient frequently complains of pain on the lateral border of the foot during weightbearing

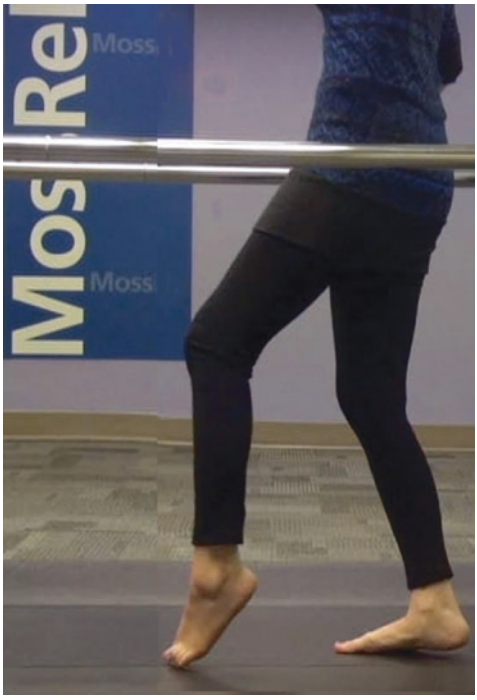

Fig. 7.52 Image of "forefoot first loading." The foot and ankle are plantarflexed prior to initial contact. The patient touches the ground first with the forefoot during loading, and in some patients, contact with the ground may be exclusively or primarily on the forefoot during the subsequent stance phase

Limited dorsiflexion during midstance prevents forward progression of the tibia over the stationary foot leading to increased pressure over the metatarsals. Compensatory hip flexion, and depending on the degree of equinus, knee flexion or extension may occur to accommodate the deformity to maintain ambulation. The presence of equinus may interfere with limb clearance during the swing phase and require contralateral early heel rise (vaulting) or increased hip and knee flexion (steppage). Muscles that potentially contribute to this deformity include the gastrocnemius, soleus, and tibialis anterior.

Problems of Limb Advancement

Insufficient Hip Flexion

Lack of hip flexion not only affects walking by interfering with limb advancement but may also impact floor clearance by the foot. Weakness of hip flexor muscles (e.g., iliacus, psoas, pectineus, and rectus femoris), and in some cases, overactive hip extensors (gluteus maximus and hamstrings) may be the source of lack of hip flexion. Reduced swing phase hip flexion may also promote a more extended knee (akin to a stiff knee gait) because restraint of hip flexion during swing phase reduces limb acceleration needed to generate the pendular motion of the knee.

Impaired hip flexion can also become a significant positioning issue during sitting (Fig. 7.53). In addition to hip extensor tone/spasticity, one should also consider heterotopic ossification, hip dislocation, undiagnosed fractures and other sources of painful stimuli, because pain can increase overactivity of the hip extensors and knee flexors (see Chap. 9).

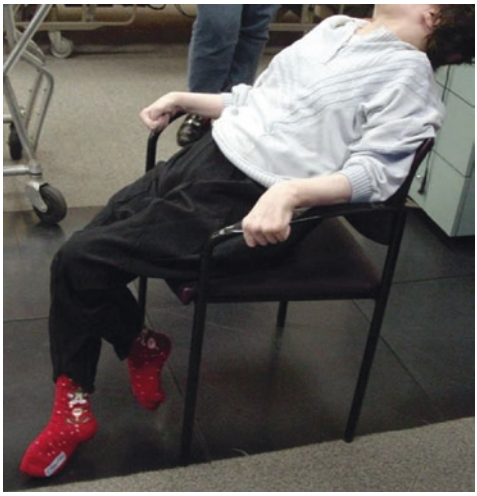

Fig. 7.53 As seen in this image, insufficient hip flexion can cause a significant positioning issue during sitting. In addition to shortening of hip extensors and hip extensor overactivity, one should also consider heterotopic ossification, hip dislocation, undiagnosed fractures and other sources of painful stimuli, because pain can increase overactivity of the hip extensors and knee flexors

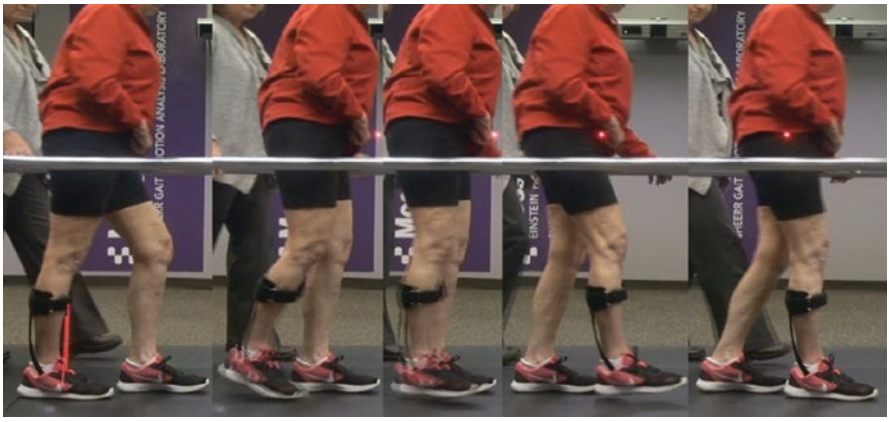

Fig. 7.54 Image of a patient with a stiff knee gait. The knee is maintained in a largely or completely extended position throughout the swing phase and the moment of inertia of the lower limb is increased, further impairing hip flexion

Stiff Knee (Inadequate Knee Flexion in Swing Phase)

In this gait deviation, the knee is maintained in a largely or completely extended position throughout the swing phase and the moment of inertia of the lower limb is increased, further impairing hip flexion (Fig. 7.54). In the context of spastic paresis, a stiff knee gait results from a dynamic deformity created by muscle shortening or contraction and external moments rather than a structural deformity of the knee joint. The resulting lack of adequate limb clearance due to reduced hip and knee flexion can result in a foot drag, even if the ankle has adequate dorsiflexion. Compensatory mechanisms in the trunk, ipsilateral hip (e.g., circumduction), and contralateral limb (e.g., vaulting or early heel rise) may be present, leading to increased effort during walking.

Out-of-phase activation of the RF during the swing phase can be a major contributor to the stiff knee pattern because the RF crosses both the hip and knee joint and can restrict knee flexion. Out-of-phase activation of the vasti muscles in the swing phase can also contribute to this gait deviation. At the hip, overactivity of the gluteus maximus and hamstrings during the swing phase may restrain hip flexion, resulting in a stiff knee gait. A weak iliopsoas can also reduce hip flexion in the swing phase, further impairing knee flexion. Ankle equinus can contribute to knee hyperextension in the stance phase by preventing forward progression of the tibia and delaying knee flexion. In general, when a concomitant ankle deformity is observed, the ankle should be addressed first, as this may ameliorate the stiff knee gait pattern.

Toe Drag

During the swing phase, five different mechanisms facilitate limb clearance so that the limb can be advanced. These include: (1) swing limb hip flexion, (2) swing limb knee flexion, (3) swing limb ankle dorsiflexion, (4) stance limb

Fig. 7.55 Image of a patient with toe drag. In its most frequent presentation, toe drag is present during the early swing phase. It can also be seen at the end of the swing phase or throughout swing. When it is present throughout swing, toe drag impairs limb clearance and restrains limb advancement. When it occurs in the early swing phase, the penalty is limited limb advancement

extension control (controlled forward progression of the tibia), and (5) stance limb control of pelvic tilt. If toe drag occurs, it is because of the loss of three or more of the five identified mechanisms, possibilities for compensation having been lost. In its most frequent presentation, toe drag manifests during the early swing phase (Fig. 7.55). It can also be evident at the end of the swing phase or throughout swing. When it is present throughout swing, toe drag impairs limb clearance and restrains limb advancement. When it occurs in early swing phase, the penalty is limited limb advancement [29].

Inadequate Knee Extension in Terminal Swing

Limited knee extension is a common physical finding that has functional implications for walking (Fig. 7.56). It may occur due to contracture from prolonged sitting or overactivity of knee flexor muscles (e.g., hamstrings and gastrocnemius). When severe, heterotopic ossification, knee dislocation, and undiagnosed fractures should be ruled out. During walking, a common penalty is reduced step length due to limited knee extension. Hip flexion deformities may also contribute to knee flexion posturing when the patient lies supine. Hip flexion deformities can restrain hip acceleration during the swing phase, reducing the inertial forward displacement of the tibia. Hamstring and gastrocnemius stretching should increase knee extension in this phase of walking [30].

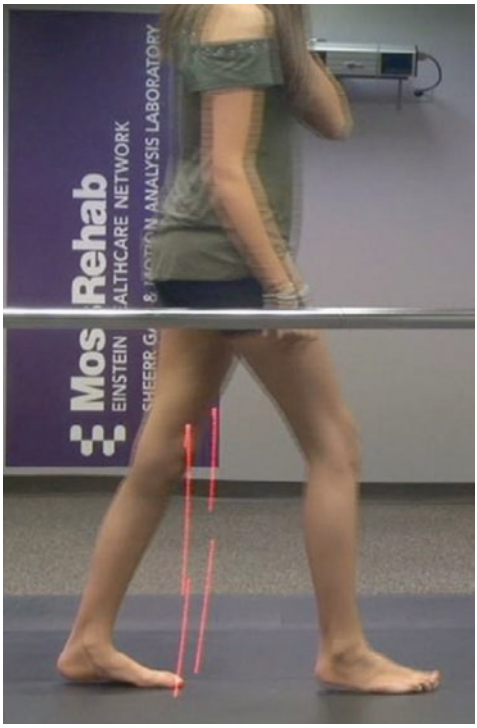

Fig. 7.56 Limited knee extension of the right lower limb at the very end of terminal swing (virtually at initial contact), with resulting limited limb advancement and a short step length

Problems of Single Limb Support

Knee Flexion in Stance

The flexed knee deformity may refer to the flexed posture of the knee in both the stance and swing phases. Not only does a flexed knee deformity impair limb stability and contralateral limb clearance, but the lack of knee extension in terminal swing also limits limb advancement. This gait pattern is often associated with hamstring muscle shortening, overactivity, or contracture. Other factors that contribute to this dysfunction include shortening or overactivity of the gastrocnemius in the stance phase and of the iliopsoas in the swing phase.

Weak Knee Extensors

Profound weakness of the knee extensors, hip extensors, or ankle plantar flexors may also lead to knee flexion in the stance phase with marked limb instability that forces the patient to rely on upper limb support (Fig. 7.57). The patient's sense of instability shortens the contralateral step length and substantially reduces walking velocity and tolerance [31]. Bracing may be the only alternative because the lack of motor control may prevent strengthening. Bracing the knee to prevent flexion appears to be the most sensible intervention, but this unfortunately also limits knee flexion during the swing phase and interferes with limb clearance. Newer

Fig. 7.57 Image of weak knee extensors. Profound weakness of knee extensors, hip extensors, or ankle plantar flexors may also lead to knee flexion in the stance phase with marked limb instability that forces the patient to rely on upper limb support. The sense of instability shortens the contralateral step length and substantially reduces walking velocity and tolerance

devices that provide support only during the stance phase would work best, but these devices tend to be heavier and more expensive. The use of a limited motion ankle foot orthosis with a dorsiflexion stop and rear entry design may be a suitable solution for this problem (Fig. 7.58).

Painful Toe Flexors During Loading and Weightbearing

When the toe flexors are painful, observation of barefoot walking may reveal that the lesser toes and/ or hallux are held in flexion during the phases of terminal stance and terminal swing (Fig. 7.59). The patient will complain of pain at the tip of the toes, demonstrate prolonged limb loading, and reduced stance phase duration. When wearing shoes, the patient may report pain at the tip of the toes and pressure from the shoe upper. Shortening or overactivity of the flexor digitorum longus and flexor hallucis longus muscles is the principal mechanism for this problem. However, weakness of the extensor muscles may also contribute. Toe flexion has a deleterious effect on late stance propulsion and can reduce limb clearance and advancement

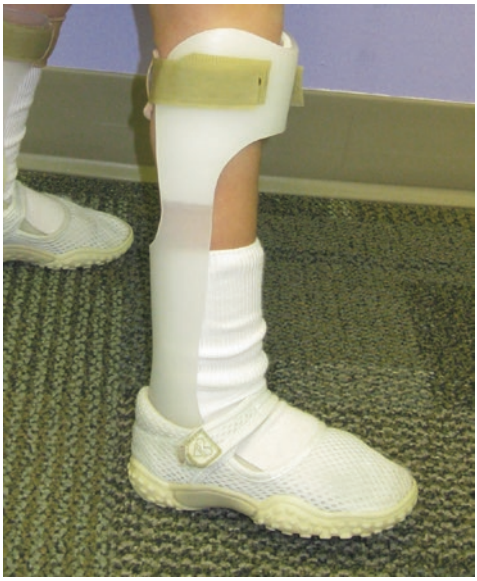

Fig. 7.58 Bracing may be the only alternative for weak knee extensors because lack of motor control may prevent extensor strengthening. Bracing the knee to prevent flexion appears to be the most sensible intervention, but it also limits knee flexion during the swing phase and interferes with limb clearance. As depicted in this figure, the use of a limited motion ankle foot orthosis with a dorsiflexion stop and rear entry design may be a suitable solution for weak knee extensors

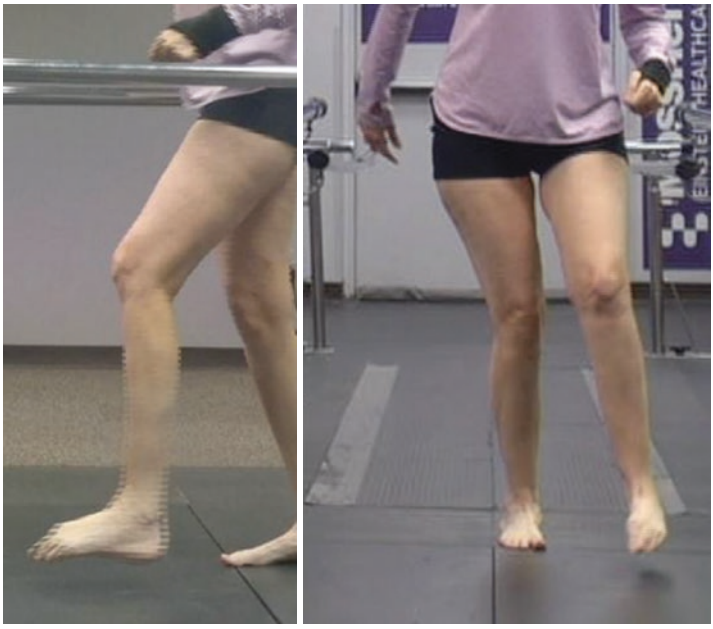

Fig. 7.59 Painful toe flexors during loading and weightbearing. Observation of barefoot walking reveals that the lesser toes and/ or hallux are held in flexion during the phases of terminal stance and terminal swing. The patient will complain of pain at the tip of the toes, demonstrate prolongation of the loading response during the period of double support, and reduced stance duration because of shortened single limb support

during swing. Ankle equinus posture may accompany this deformity, and if present, evaluation of the gastrocnemius and soleus muscles should be performed [32].

Conclusions

This chapter outlines the abnormal limb postures that affect the upper and lower limbs as a result of long-standing spastic paresis, and attempts to describe the various muscles that contribute to these postures. We have also reviewed the functional consequences of these abnormal postures to set reasonable goals for treatment.

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