20 Walking Recovery and Rehabilitation after Stroke

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Motor control deficits are a common manifestation of and a major contributor to walking disability after stroke. As a result, limitations in mobility related to walking are evident in 75% of individuals who sustain a stroke each year (1). During the first six weeks post-stroke, the majority of stroke survivors will regain some ability to walk; however, 40% will have severe motor impairment that restricts functional walking to household ambulation. For those with mild to moderate motor impairment, independent walking ability is likely, but of those who achieve physical independence in walking, 60% will be limited in community ambulation (2). Furthermore, motor impairments that limit walking ability contribute to balance deficits after stroke. Individuals who have mild to moderate motor impairment and are functional ambulators after stroke have a 73% incidence of falls within the first six months post-stroke (3, 4). This culminates in a fourfold increase in falls risk after stroke and among those who fall, a tenfold increase in hip fracture.

The greatest predictor of community ambulation and participation in home and community mobility is walking speed (5). Perry et al. conducted a cross-sectional study of individuals with gait impairments after chronic stroke and classified them into one of six functional walking categories based on the degree of community ambulation and social interaction (5). The most significant difference between groups was preferred walking speed, with mean speeds ranging from slower than 0.4 meters per second (m/s) for household walkers, 0.4–0.8 m/s for limited community walkers, and faster than 0.8 m/s for unlimited community walkers. As expected, the higher the amount of community ambulation ability, the more likely one was to participate in social activities such as family outings, grocery shopping, going to church, etc. However, achieving the break point of faster than 0.8 m/s (1.8 mph) in walking speed only represents 60% of what is normal for most healthy adults (6). Thus, the functional impact of walking impairment after stroke is significant. Activity outcome measures of walking function such as the Barthel Index (BI) (7) or the Functional Independence Measure (FIM) (8) are poor indicators of community mobility and participation after stroke since the majority of stroke survivors can achieve high BI or FIM scores (i.e., independent walking 10 meters or less) without any consideration of walking speed.

The impact of motor control impairments and walking disability extends beyond the acute and subacute phases to the chronic phase post-stroke. Post-stroke, lower-extremity, motor control deficits contribute to specific lower-extremity primary and secondary motor impairments and activity restrictions that impact balance ability and walking skills and increases the risk of secondary complications related to

impaired walking ability such as an increased risk of falls and hip fracture, and, ultimately, mobility limitations that restrict participation across the lifespan for an individual post-stroke. Walking recovery and rehabilitation after stroke includes therapeutic strategies that are designed to address the complicated and multivariate predictors of walking function. The overall purpose of this chapter is to describe all of the following:

1. Biomechanical features of post-stroke walking impairment
2. Primary and secondary motor impairments and recovery factors that contribute to activity restrictions in the acute and chronic phases post-stroke
3. Effects of stroke severity (i.e., severe compared to mild-moderate sensorimotor impairment) and secondary impairments on walking outcomes
4. Rehabilitation interventions for post-stroke walking recovery that are currently being used in practice to address the sensorimotor sequelae of stroke

BIOMECHANICS OF POST-STROKE GAIT

Stroke results in a constellation of sensorimotor impairments such as weakness, impaired selective motor control, spasticity, and proprioceptive deficits that interfere with the invariant features of normal adult gait. There is a wide range of walking ability after stroke that depends on the severity of these sensorimotor impairments. After stroke, there are obvious gait deviations that can be observed during observational gait analysis. Biomechanical gait characteristics such as spatiotemporal features (i.e., interlimb symmetry), kinematics (i.e., joint motion relationships within and between limbs), and kinetics (i.e., forces required for stance and forward progression) can be quantified in an appropriately instrumented gait lab. This section describes the biomechanical features of post-stroke gait since biomechanical analysis is an essential element for understanding the mechanisms of walking recovery and responsiveness to specific walking rehabilitation interventions.

Changes in Temporal Gait Characteristics

While asymmetry is a dominant characteristic of hemiparetic walking, the heterogeneity of the hemiparetic population has made it difficult to develop an integrative understanding of how spatiotemporal asymmetry is related to the underlying impairments. There are a multitude of variables that have been used to quantify spatiotemporal asymmetry, and inconsistent reporting of important variables has limited our understanding of the basis of asymmetry. For example, temporal asymmetry is often reported, while spatial asymmetry is not (or vice versa), with few studies reporting both variables in detail. Additionally, the general speed dependence of spatiotemporal characteristics must also be considered when interpreting hemiparetic spatiotemporal data (9) because most persons with hemiparesis walk slowly with an altered stride length and/or cadence. Despite all of the complications that have limited a more detailed understanding of hemiparetic spatiotemporal asymmetry, some general relationships are beginning to emerge.

For example, walking speed is determined by a combination of cadence and stride length (distance between consecutive foot placements of the same foot in the direction of progression). While both are reduced in hemiparetic walking when compared to non-impaired persons walking at their self-selected speed (10), neither appears to be consistently different when compared to non-impaired persons walking at the same speed (9). This is likely explained by the mechanical constraints of walking and the required symmetry imposed by the definition of stride length and cadence. Hemiparetic subjects differ most dramatically from non-impaired individuals walking at the same speed in the spatiotemporal measures that indicate interlimb asymmetry.

There are two main temporal asymmetries in hemiparetic walking. First, it has been consistently reported that persons with hemiparesis spend significantly more time in stance phase on the non-paretic leg than on the paretic leg (10, 11). Note that this also requires that they spend significantly more time in swing phase on the paretic leg than on the non-paretic leg. Second, it has been somewhat less consistently reported that the double support phase preceding paretic leg foot-off (which we will define as paretic pre-swing) is longer in duration than is the initial paretic leg double support phase (which we will define as paretic weight acceptance). Olney et al. found an average increase in duration for 32 persons (25% vs. 20% of the gait cycle) (12). Similarly, de Quervain et al. also found that the paretic pre-swing phase was prolonged for 18 persons (13). However, Goldie et al. reported no significant differences in the duration of the two double support phases in a group of 42 persons (10).

The main spatial asymmetry of hemiparetic walking is characterized by differences in the paretic and non-paretic step length (i.e., paretic step length is the distance in the direction of progression that the paretic foot is placed in front of the non-paretic leg, and vice versa for non-paretic step length). While many studies have reported that on average the paretic step length is longer than the non-paretic step length, these studies typically find substantial variability and that some persons with hemiparesis exhibit substantial asymmetry in the opposite direction. Until recently there has been no mechanistic explanation advanced to explain this variability.

In a recent study, Balasubramanian et al. found that step length asymmetry was related to propulsive force generation during hemiparetic walking (14). Propulsive force

generation was quantified from the forward directed component of the horizontal ground reaction forces (GRFs) by the time integral of the propulsive GRF (positive area under the GRF versus time curve), since it is that impulse that acts to accelerate the body forward. Paretic propulsion in particular is defined as the proportion of the total propulsive impulse produced by the paretic leg (i.e., impulse of paretic leg divided by sum of the impulses of the paretic and non-paretic legs). Paretic step ratio is used to quantify step-length asymmetry and is similarly defined as step length of the paretic leg divided by sum of the step lengths of the paretic and non-paretic legs. Specifically, these authors found that the subjects that generated the least paretic propulsion walked with relatively longer paretic steps, suggesting that increased propulsion by the non-paretic leg may be one mechanism contributing to production of a longer paretic step (Figure 20-1). Further, those with more severe hemiparesis (those dependent on abnormal flexor and extensor synergies) walk with the longest paretic steps relative to non-paretic (Figure 20-2). However, these results indicated that asymmetrical step lengths do not necessarily limit the self-selected walking speed (i.e., some subjects with longer paretic steps walk faster), likely because of other compensatory generation of propulsion by the non-paretic leg.

Changes in Kinematic Parameters

The kinematics of hemiparetic walking are altered in both legs relative to healthy walking, with the changes being necessarily consistent with the slower speed, slower cadence, and greater asymmetry of hemiparetic walking. Paretic hip motion becomes more abnormal during pre-swing as self-selected walking speed declines (13). De Quervain et al. found that of 12 subjects studied post-stroke, seven with severe hemiparesis did not begin hip flexion until toe-off, five more did not begin hip flexion until the last third of pre-swing, and in the subjects with moderate hemiparesis, beginning hip flexion occurred at the midpoint of pre-swing (13). In contrast, the timing of non-paretic hip flexion was normal in even the slowest walkers. Ankle kinematics may also be important for the performance of paretic pre-swing since peak plantarflexion is less than in normal walking, even when the ankle is in a more plantarflexed (i.e., equinus) position throughout much of stance (11). Nine of twelve subjects with severe hemiparesis were found to have little or no plantarflexion in pre-swing (13).

Two studies performed detailed analyses in persons very early in the recovery phase (13, 15). Using the kinematic data, both studies classified subgroups of hemiparetic subjects with similar characteristics. Based on visual identification of common patterns of knee and ankle motion coupling, De Quervain et al. classified 18 subjects into four groups (13). Mulroy et al. used a nonhierarchical cluster analysis based on spatiotemporal

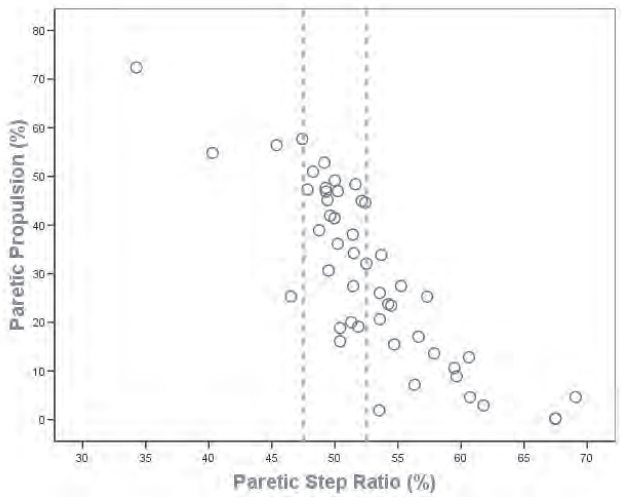

FIGURE 20-1

There is a strong relationship between step length asymmetry and paretic propulsion ($r = -0.83$, $p = 0.001$). Subjects with a longer paretic step generate less propulsion with their paretic leg. Modified from Balasubramanian et al. (14).

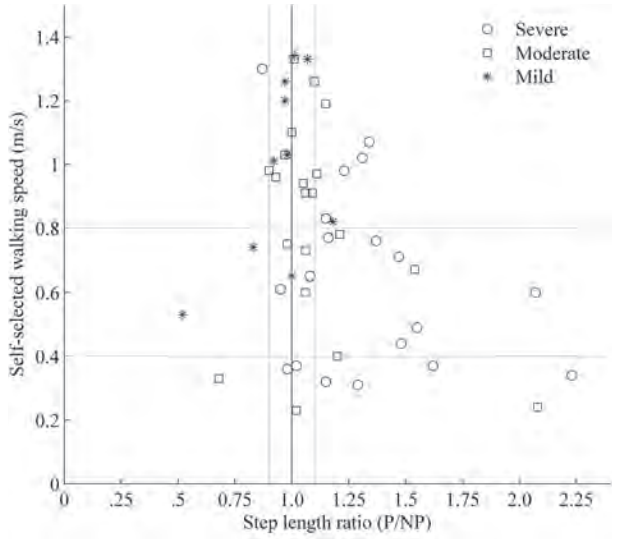

FIGURE 20-2

Relationship between step length asymmetry, walking speed, and hemiparetic severity. The solid vertical line indicates symmetric steps (SLR), vertical dashed lines indicate the SLR subdivisions at SLR = 0.9 and SLR = 1.1. Horizontal dashed lines indicate subdivisions of walking speeds (<0.4m/s, house hold walkers; 0.4–0.8m/s, limited community walkers; >0.8m/s, community walkers). Note that subjects with different SLR walk at all levels of walking speeds, yet the majority of those with severe hemiparesis walk asymmetrically at SLR greater than 1.1. From Balasubramanian et al. (14).

characteristics and peak sagittal plane joint angles for each gait cycle phase and classified 47 subjects into four groups (15).

Subjects with slow walking speeds (i.e., severe hemiparesis) demonstrated more variable kinematics; however, similar kinematic patterns are described in the two studies. The "extension thrust" pattern described by De Quervain et al. was very similar to the "extended" pattern described by Mulroy et al. with increased ankle plantarflexion and knee hyperextension throughout stance, inadequate ankle dorsiflexion in pre-swing, and inadequate knee flexion in swing. Similarly, the "buckling-knee" pattern described by De Quervain et al. was similar to the "flexed" pattern described by Mulroy et al. with greatly increased ankle dorsiflexion and knee flexion throughout stance, decreased thigh extension in pre-swing, and decreased ankle dorsiflexion in swing. De Quervain et al. also described a "stiff-knee" pattern in which knee angle remained constant throughout stance at an angle of 20°–30° of flexion in combination with decreased ankle dorsiflexion. Mulroy et al. did not identify any subjects who exhibited a similar "stiff-knee" pattern in their study. Neither study identified a group of subjects with a fixed equinus deformity at this early stage of recovery, although others have reported it to be a major impairment during the later phases of recovery (16).

The kinematics of subjects who exhibit moderate to fast walking speeds (i.e., moderate to mild severity) tend to be more similar to those of nondisabled walkers, although some who are classified as the "extended" or "flexed" groups by Mulroy et al. are able to walk at these higher speeds. Mulroy et al. classified subjects into a "moderate" and "fast" group within this range, with the main difference being increased knee flexion in stance and decreased thigh extension and plantarflexion in pre-swing for the "moderate" group as compared to the "fast" group. De Quervain et al. appear to have combined these two groups into a single group, with intermediate characteristics.

The kinematics of the non-paretic leg differ from normal in a much more consistent fashion than did those of the paretic leg. Hip angles seem generally consistent with nondisabled walking at slower speeds with a shorter stride (13, 17). Those walking slowly tended to show the biggest differences at the ankle, where dorsiflexion in late stance is increased and the ankle plantarflexion in pre-swing is decreased or absent.

Changes in Kinetic Parameters

Generally, the ground reaction forces (GRFs) in hemiparetic walking are infrequently reported. Vertical forces have been investigated predominantly to determine symmetry of weight bearing. Three different patterns of vertical GRF are typically observed in the paretic leg: double-peaked with early and late peaks (similar to normal), one single peak in midstance, and a plateau with no discernable peak (18). Kim and Eng investigated 28 hemiparetic subjects of varying ambulatory ability and found that all but three had reduced vertical GRFs in the paretic leg and that symmetric weight distribution between the legs was related to increased temporal symmetry but not spatial symmetry during walking (19).

A recent study used the propulsion force as a performance measure to quantify the contribution of the paretic leg to hemiparetic walking (20). Lacking such a performance measure has been a significant barrier to understanding paretic leg motor performance during walking. Measures have typically focused on bilateral gait-related outcomes such as walking speed, over-ground ambulatory capacity, endurance, and balance. The biomechanical and mechanistic contributions of the paretic leg to such measures are not unique. For example, the same gait speed can be achieved with many levels of coordination of the paretic leg if the non-paretic leg provides different levels of compensatory output. Previously, estimates based on mechanical work calculations had suggested that the paretic leg does 30%–40% of the total mechanical work over the gait cycle, regardless of hemiparetic severity (17). However, these work estimates could be flawed because they may be dominated by the support of body weight and not a unique contribution of each leg to forward propulsion, this being a critical component of walking performance.

The purpose of the Bowden et al. study was to establish a quantifiable link between hemiparetic severity and paretic leg contribution to propulsion during walking using a measure based on the Anterior-Posterior (A-P) GRF. In particular, the measure defined the proportion of the summed propulsion from both legs generated by the paretic leg (PP). Total body forward propulsion by a leg is calculated by integrating over time (e.g., stance) the forward propulsion produced by that leg. PP is the ratio (in percent) of the total body forward propulsion produced by the paretic leg to the summed forward propulsion by both legs. Forty-seven participants with chronic hemiparesis walked at self-selected speeds. Spatiotemporal parameters and GRFs were collected. A-P GRF measures were correlated with both walking speed and hemiparetic severity (Figure 20-3). The percentage of propulsion generated by the paretic leg (PP) was found to be 16%, 36%, and 49% for those with severe, moderate, and mild hemiparesis (as assessed by Brunnstrom stages (21), respectively (Figure 20-4). Thus, PP provided a quantitative measure of the coordinated output of the paretic leg that indicates that the motor performance of the paretic leg can be assessed independently and corresponds to the severity of hemiparesis.

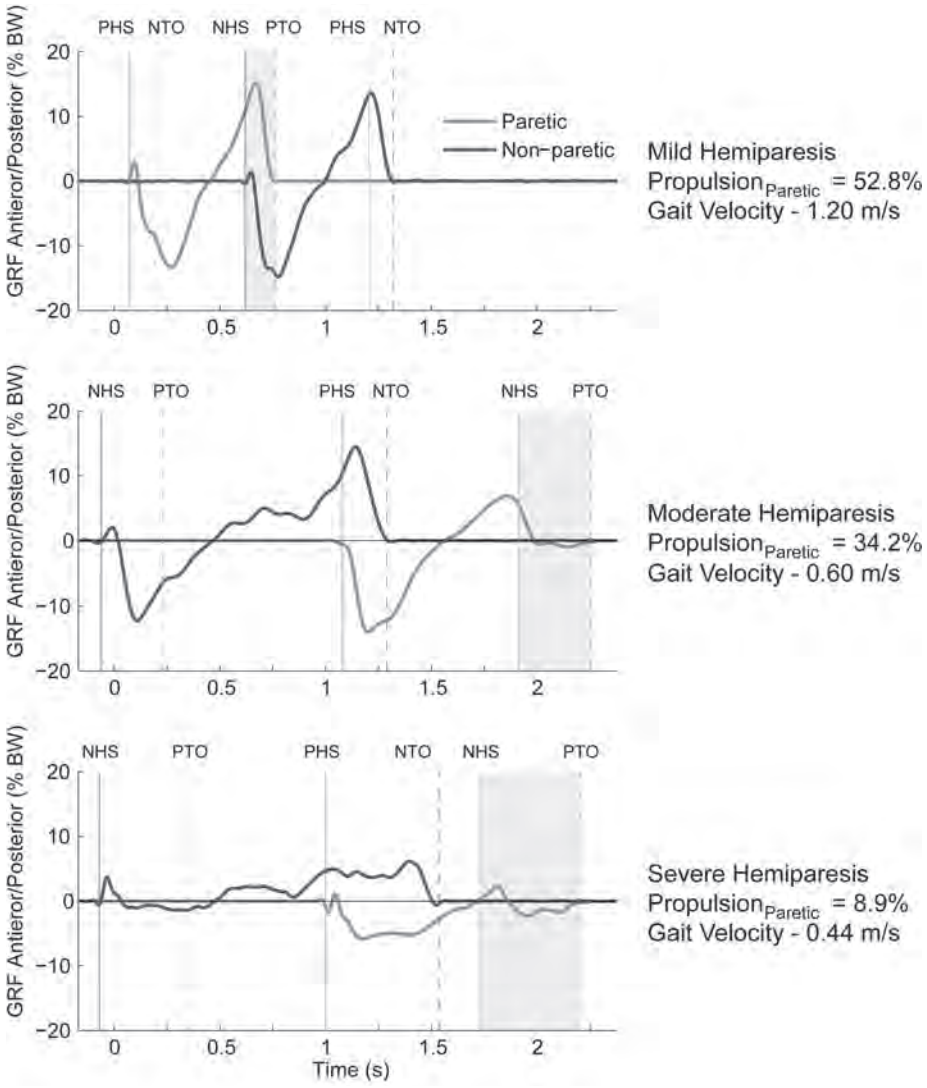

FIGURE 20-3

A-P GRFs for three subjects of differing hemiparetic severity. Positive values represent propulsion, and the positive area under the curve is the propulsive impulse. PHS indicates paretic heel strike; NTO, non-paretic toe off; NHS, non-paretic heel strike; PTO, paretic toe off. Increased hemiparetic severity was associated with decreased PP and decreases in self-selected walking speed. Modified from Bowden et al. (20).

Subsequently, additional studies used propulsion-based measures with success to explain step length asymmetry (14) and the consequences of abnormal electromyographic (EMG) timing (22) to assess the contribution of the paretic leg to hemiparetic walking. Forward body propulsion by a leg at any instant is defined as the force that acts on the body to accelerate it forward (represented by the red vector at hip in Figure 20-5a), which results from the anteriorly directed, forward ground reaction force (red vector parallel to ground in Figure 20-5a) acting on the leg at that instant.

PP is sensitive to differences in motor control not captured by multiconstruct measures such as gait speed, and therefore it may be an effective tool for distinguishing functional compensation by the non-paretic leg from physiological restitution of paretic leg performance. Quantifying forward propulsion provides great insight into overall paretic leg performance because it measures both the active generation of propulsive forces by muscles (e.g., plantarflexors) and the indirect contribution of other muscle activity (e.g., hip and knee flexors) through their influence on the walking mechanics. For example, in terminal stance, or pre-swing, of normal walking, the heel rises and the forefoot acts as a "rocker" that allows body forward progression (Figure 20-4a). An inverted pendulum representation of walking illustrates this contribution

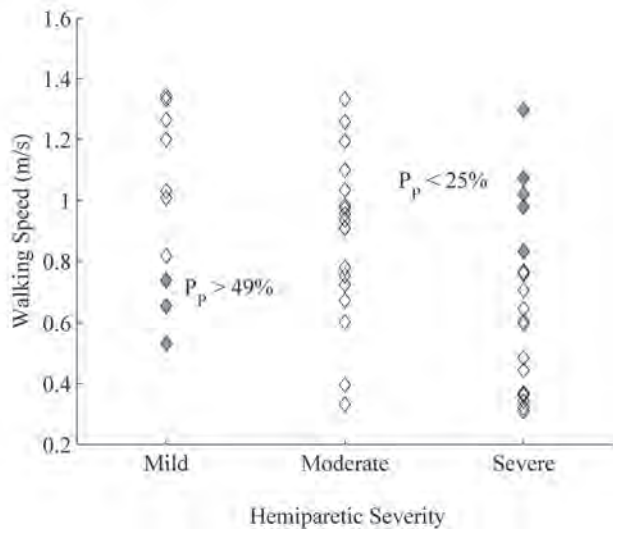

FIGURE 20-4

Individual walking speed data show that there is substantial variability in the weak relationship between walking speed and severity. Note, however, that PP helps explain much of this variation, as the five participants with severe hemiparesis who achieve faster walking speeds (>0.8 m/s) all demonstrate substantial decreases in PP, while the three participants with mild hemiparesis who walked >0.8 m/s have normal PP (>49%). From Bowden et al. (20).

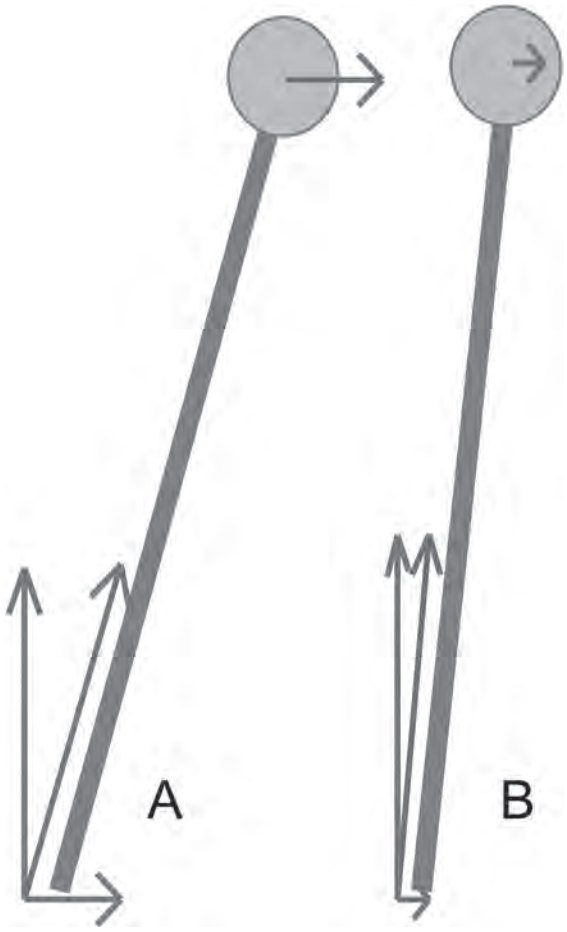

FIGURE 20-5

Illustration of the potential contribution of mechanics to the propulsive ground reaction force for two leg configurations in pre-swing. Reduced hip extension would result in decreased contribution to forward propulsion from walking mechanics.

to propulsion because the resultant ground reaction force acts along the axis of the leg (blue vector), which results in an anteriorly directed component (red vector parallel to ground) of a particular magnitude in direct relation to those walking mechanics. Thus, reduced propulsion will occur as a result of walking mechanics per se should early initiation of leg flexion prevent the foot from moving sufficiently posteriorly relative to the pelvis (i.e., should the hip not extend sufficiently; Figure 20-4b).

As would be expected during slow hemiparetic walking, joint moments and powers are greatly reduced when compared to non-disabled subjects walking at their self-selected speed. However, the differences are much reduced when compared with walking at a matched speed. In fact, it is remarkable the extent to which the paretic leg moment and power profiles are similar in pattern but reduced in amplitude in comparison to the non-paretic legs, which are mostly similar to those of matched speed control subjects (17). Thus, the main power generation bursts in hemiparetic walking are the same as that in non-disabled walking. There are the power generation burst associated with the ankle plantarflexor moment in pre-swing (commonly referred to as A2, as by Olney et al.) (17), the hip extension moment during initial double support (H1), and the hip flexion moment during pre-swing (H3). The A2 positive power burst associated with the ankle plantarflexor moment in pre-swing is usually greatly reduced on the paretic side. H3 has also been found to be reduced in some subjects, especially those who walk most slowly, although it has been hypothesized for some subjects that H3 compensates for the absence of ankle plantarflexor power.

Ankle joint plantarflexor power (A2) was barely evident in the paretic leg in a group of 10 household walkers (17). In addition, when ankle moments during gait were compared with maximum isometric moments, it was suggested that the walking speed of 10 of 17 hemiparetic subjects was limited by plantarflexor weakness (although the four fastest walkers in that group appeared to compensate by increasing hip flexor moment) (23). Ankle moments are also substantially reduced in hemiparetic persons in comparison to slow walking controls, with household walkers having reduced moments compared to limited community walkers (17). In contrast to the paretic leg, the non-paretic leg A2 power generation is

equal to or greater than speed matched control subjects, as the A2 power bursts supply about half of the positive work generated by the joint moments (24).

Increased pre-swing hip flexor moments and powers would appear to be able to compensate for plantarflexor weakness and produce faster walking speeds in some faster walkers (25). Hip flexor moments and powers seem to be reduced for slow walkers and increased for fast walkers with respect to healthy controls (11, 17). Since hip flexion abnormalities during pre-swing differentiated the functional walking groups, De Quervain et al. concluded that "insufficient power and inappropriate initiation of flexion of the hip must be assumed to be the cause, as no activity was noted in the extensor muscles" (13). While the H3 burst may be greater in the non-paretic leg in some subjects, this non-paretic burst does not seem to be used to produce substantial amounts of compensatory positive work as does the A2 burst.

The other potential mechanism for increased positive power generation is the hip extensor burst in initial double support (H1). While H1 has a positive relationship with speed across different subjects (17) and a positive relationship with change in speed after rehabilitation (24), it produces less overall work than the other two bursts. Interestingly, the non-paretic leg H1 appears to produce some compensation as it is increased in slow walking subjects (17).

There is also an important non-sagittal plane power burst at the hip that is seen in persons with hemiparesis; a study found the hip abductor moment is prolonged in hemiparetic walking in more than half of the subjects (19). Since it is seen most often in the faster walking subjects, Kim and Eng suggest that it is a compensatory mechanism for poor dorsiflexion and knee flexion during swing (19). This finding challenges the notion that treatment approaches should be directed toward restoring "normal" kinematic and kinetic patterns of walking if the goal is to achieve optimal functional performance rather than an aesthetic one.

Thus, kinetic measures such as ground reaction forces and joint moments can provide much insight into the impaired mechanics of walking after stroke. Propulsion is a crucial task during walking, and new biomechanical measures based on the anteriorly directed ground reaction force provide a measure to quantify the contribution of the paretic leg to walking and offer great potential for distinguishing compensation from recovery in the paretic leg performance. Furthermore, the ankle plantarflexors during push off, the hip flexors during pull off, and the hip extensors during weight acceptance have been identified as the primary sources of power generation during normal and hemiparetic walking, with ankle plantarflexor power generation being particularly impaired in hemiparetic walking.

EARLY POST-STROKE PATTERNS OF RECOVERY

Immediately following stroke, the most common feature of motor impairment is either paresis or paralysis, partial reduction or complete loss of the ability to generate force in the muscles of the extremities contralateral to the stroke (26). At this time point post-stroke, the primary contributors to contralateral paresis are disruption of the motor neural pathways leading to reduced motor recruitment (27) and disuse atrophy (28). Consequently, in the early post-stroke phase the characteristic asymmetrical gait results primarily from inadequate muscle force generation (29).

Abnormal Motor Unit Recruitment and Muscle Weakness

Paresis in the lower extremity can impair both the ability to advance the limb for swing and to support body weight during stance (6, 11, 19). Support of body weight can be compromised by inadequate activation of the ankle plantar flexors, knee extensors, or hip extensors (30). Paresis of the ankle dorsiflexors may result in poor foot clearance in swing (29). If the hip flexors also are weak, inability to compensate by lifting the limb results in a toe drag and limited step length.

Alterations in electromyographic (EMG) timing and intensity during walking result from both reduced activation and from compensations for loss of force (15, 29). Few studies have described muscle activity patterns in the early post-stroke phase (13, 15, 29, 31). The impact of reduced muscle activation and resultant weakness on the characteristic patterns of walking after stroke was identified in 47 individuals at admission to acute rehabilitation and again at six months post-stroke (15). Four distinct gait patterns were discerned. Those subjects in the two groups with greater walking impairment walked at just 10% to 11% of normal velocity, and lower-extremity muscle strength ranged from 17% to 36% of normal with weakness most severe at the ankle. In both of the slower groups, marked paresis of the ankle plantar flexor muscles removed the normal restraint of the tibia in stance. The pattern of walking compensation adopted initially was reflected in the knee position in mid-stance. The relative strength of the proximal muscles in the paretic leg was the factor that determined which compensatory pattern was adopted. In those individuals with greater strength in the knee extensors than in the hip extensors, the knee collapsed into flexion with the demand shifted to the quadriceps (Flexed group). In contrast, subjects with stronger hip extensors and weaker quadriceps thrust into hyperextension at the knee (Extended group) relying on passive stability at the knee and increased activation of the hip extensors for stance control and progression of body weight.

TABLE 20-1
Mean EMG Intensity during Walking at Admission to Rehabilitation and Six months after Stroke

	EMG Intensity (% MAX MMT)				
	Able-bodied	Stroke – Admission		Stroke – Six Months	
		Slow	*Fast*	*Slow*	*Fast*
SMEMB	20	12 ± 11	18 ± 8	17 ± 10	21 ± 9
BF long	13	11 ± 9	13 ± 10	16 ± 14	17 ± 8
GMAX	18	17 ± 9	23 ± 9	21 ± 11	20 ± 8
VI	13	15 ± 9	29 ± 15	24 ± 16*	29 ± 17*
SOL	35	17 ± 13	30 ± 20	27 ± 19*†	36 ± 15*†
PB	20	5 ± 12	10 ± 8	7 ± 9*†	18 ± 17*†
ADL	14	9 ± 8	12 ± 7	15 ± 7*†	26 ± 11*†
RF	15	8 ± 7	12 ± 12	14 ± 10†	17 ± 11†
AT	25	10 ± 6	26 ± 18	20 ± 11*	28 ± 16*

SMEMB. Semimembranosus; BF long, long head of biceps femoris; GMAX, gluteus maximus; VI, vastus intermedius; SOL, soleus; PB, peroneus brevis; ADL, adductor longus; RF, rectus femoris; AT, anterior tibilalis. * = significantly greater in Fast than in Slow; † = significantly greater at six months than at admission.

In the two faster groups (Fast and Moderate), walking velocity was 44% and 21% of normal, respectively. Ankle plantar flexion strength was less than normal (53% and 38% of normal), but not as markedly reduced as in the two slower groups (Flexed and Extended). This greater residual plantar flexion strength resulted in only mildly excessive knee flexion in stance for the Moderate group and normal extension in the group with the least walking impairment (Fast). Moreover, activation intensity was *greater than normal* at admission for the two faster groups in vastus intermedius and gluteus maximus, indicating increased proximal activity in stance as a substitution for reduced distal control (Table 20-1). The significant impact of reduced activation of the calf muscles on walking function in the acute post-stroke phase was confirmed by Lamontagne and colleagues, who documented that gastrocnemius EMG intensity during gait explained a full 52% of the variance in walking speed in the early post-stroke phase (29).

The swing-phase movement pattern in the acute phase was characterized by the amount of dorsiflexion achieved in mid-swing (15). The decreased activation of anterior tibialis in both of the two slower groups produced inadequate dorsiflexion in mid-swing (Table 20-1). While volitional ankle dorsiflexion strength also was less than normal in the two faster groups, the muscle response was adequate to achieve at least a neutral ankle in swing.

In addition to reduced EMG intensity, alterations in the timing of muscle activity have been identified during walking (13, 15, 31). In our study of walking recovery (15), at the early post-stroke assessment, onsets of muscle activity were delayed in the two groups with the slowest velocities for all hip and knee extensors that have a typical onset in mid- or terminal swing (gluteus maximus, gluteus medius, semimembranosus, biceps femoris, and vastus intermedius). This finding is indicative of a reduced need for limb deceleration in swing because of the subjects' very slow velocities. In contrast, EMG onsets for the faster subjects were premature in vastus intermedius and adductor longus, indicating that these subjects had the capacity to activate the muscles, but the decreased muscle force output required an early onset to achieve the desired swing-phase motions. Muscle activity was prolonged with cessations in mid-stance for the hamstrings and in terminal stance for gluteus maximus and vastus intermedius for subjects in all groups, indicating need for increased proximal stability in stance to substitute for the reduced plantar flexor activity distally. Rectus femoris activity was prolonged through swing for both fast and slow velocity groups, which may indicate either spasticity or the increased need for hip flexor force in swing. Because of its insertion below the knee, however, prolonged rectus femoris activity results in inhibition of the necessary knee flexion required for foot clearance and limb advancement. These results are consistent with those of den Otter and colleagues, who identified premature

onset of gastrocnemius and prolonged activity through stance in biceps femoris and rectus femoris (31, 32). As they utilized surface electrodes to record EMG activity, the rectus femoris recording likely also reflects activity from the underlying vasti.

CHRONIC POST-STROKE PATTERNS OF RECOVERY

As time following stroke progresses, muscle strength, activation, and walking ability begin to improve (15, 33, 34). Incomplete recovery and development of additional impairments, however, can contribute to continued gait dysfunction (11, 13, 19, 35). Spasticity or contracture of the ankle plantar flexors can cause excessive plantar flexion and toe drag in swing and impede forward progression during stance (29, 35). Knee extensor or hamstring spasticity can inhibit limb advancement in swing (36). In the chronic post-stroke phase, disuse muscular atrophy compounds the initial neurologic injury, and consequently, muscle weakness remains prevalent despite the functional recovery during the acute phase (37). Decreases in activity level contribute to reduced cardiovascular fitness and endurance across the chronic phase post-stroke (see Chapter 23 for more information).

Abnormal Motor Unit Recruitment

In the study of walking recovery at six months post-stroke, consistent with improvement in walking speed, lower-extremity muscles, particularly distal ones, demonstrated increased activation intensity during walking compared to the early post stroke phase (15). Improved walking speed was associated most strongly with increased activation in soleus and adductor longus. Anterior tibialis intensity increased at the six-month assessment for subjects in the slow groups, while it remained unchanged for those in the faster groups at levels similar to those exhibited by able-bodied subjects. Subjects in the two faster groups exhibited a greater-than-normal intensity in both vastus intermedius and adductor longus in this later assessment. Those in the slow groups had greater than normal intensity only in vastus intermedius (Table 20-1).

Timing of muscle activity also improves with recovery but remains altered compared to normal phasing for most individuals (15, 31, 32, 38). The hip and knee extensors, which initially displayed a delayed onset late in terminal swing, began earlier in swing as recovery progressed at six months. Onsets were premature compared to normal timing for semimembranosus, vastus intermedius, and soleus. These premature onsets can interfere with the flexion necessary for limb advancement in swing. The swing-phase muscles—adductor longus, rectus femoris, and anterior tibialis—also displayed earlier onsets at six months post stroke, resulting in premature activation.

The swing-phase movement pattern at six months was more influenced by the magnitude of knee flexion than ankle dorsiflexion, consistent with the improved anterior tibialis activity exhibited by subjects with slower initial walking speed (15). Those subjects who retained the extended pattern developed a stiff-legged gait with severely decreased knee flexion in swing. All four subject groups (e.g., fast, moderate; flexed, extended) demonstrated excessively prolonged activity of rectus femoris throughout swing, which would inhibit knee flexion. Only the intensity of adductor longus activity (a hip flexor in gait) differentiated those who displayed markedly reduced knee flexion in swing. It appears that individuals with sufficient activity in adductor longus were able to overcome the knee extension force from excessive activity of rectus femoris by generating a strong hip flexion force from adductor longus, resulting in thigh flexion and secondary knee flexion. Thus, restoration of walking function in the sub-acute post-stroke phase is accomplished both by restitution of distal muscle activation and greater than normal proximal muscle activity. (See Chapter 16 for similar discussion.)

Muscle Weakness

Although muscle strength improves from the acute to the chronic post-stroke phase (after six months), for most individuals, strength remains less than normal in the muscles of the lower-extremity contralateral to the side of the stroke (15, 33, 39). Ankle plantar flexion and dorsiflexion weakness still dominate in the slowest individuals. Neckel and colleagues documented greater than normal knee extension strength in a group of individuals who were at least one year post stroke (39). They postulated that knee extensor muscles may have been strengthened from increased activity during walking as part of a compensatory strategy. This is consistent with the finding of a greater-than-normal intensity of vastus intermedius activity (15).

Musculoskeletal models of walking have identified the function of muscle activity in normal walking and predicted the impact of paresis in individual muscles for subjects with pathology (40, 41). In walking at self-selected speed by able-bodied adults, a model of walking demonstrated that force production by plantar flexors in terminal stance and pre-swing is critical to trunk forward progression, swing initiation, and power generation (41). This is consistent with the robust finding of reduced ankle joint power that is strongly related to walking velocity after stroke (12, 42, 43).

Compensatory strategies to substitute for distal muscle weakness have been identified in the proximal musculature of the paretic leg as well as in muscles of

the non-paretic limb (30, 40, 44, 45). Increased distal muscle co-activation in the non-paretic leg during the double-limb support phases was documented as a compensatory mechanism in subjects with more severe motor impairments (29, 45). Musculoskeletal modeling demonstrated that an individual with severe plantar flexion weakness and the flexed-knee post-stroke walking pattern would exhibit increased contributions from the paretic hip and knee extensor muscles in single-limb stance as an alternate source of stability confirming the experimental EMG evidence (30, 40). Later in stance phase, the model also identified that increased hip flexor activity can compensate for plantarflexor weakness and produce faster walking speeds (40). (See Chapter 16 for more information on the relationship between proximal and distal power strategies during walking.)

Abnormal Muscle Synergies

Clinical observations of volitional movement patterns in both the upper and lower extremities following stroke have been described as abnormal muscle synergies that are stereotypical combinations of secondary, unwanted motions that accompany the primary desired motion (21, 26, 39). During attempts of isolated, volitional joint movement, lower-extremity abnormal synergistic movement combinations have been described as massed extension of the entire limb with adduction and internal rotation and massed flexion with abduction and external rotation. Measurement of secondary torques during isolated, isometric contractions in subjects with stroke, however, did not confirm the patterns observed during attempted dynamic movements (39). Individuals who have impaired selective movement control can utilize these mass extension and flexion patterns of the lower extremity for stance and limb advancement during walking. The extent of dependence on the abnormal synergy patterns and the strength of the patterns during walking vary greatly among individuals such that the subjects that depend on abnormal movement patterns for walking have greater functional impairment (46). The mass extension pattern does not have the normal graduated increases in muscle activation necessary for controlled knee flexion in initial double-limb support or ankle dorsiflexion in single-limb stance. The flexion pattern can limit stride length when knee extension in terminal swing is incomplete with continued hip and knee flexion. Excessive, premature ankle plantar flexion and hip extension in late swing often accompany attempted knee extension, which also impedes limb advancement and reduces stride length.

Spasticity

In his classic descriptive study of recovery after stroke, Twitchell described the timing and pattern of the onset of spasticity (26). He noted that spasticity began between 2 and 20 days post-stroke and was most common for the lower extremity in the ankle plantar flexors. Clinical measures of spasticity are related to both the intensity and duration of EMG activity elicited during passive stretching of the muscle (47).

In a study of walking recovery after stroke, the duration of EMG responses to a manual quick stretch was recorded as an indicator of spasticity at an average of 21 days post-stroke and again at six months post-stroke Table 20-2 (15). A normal EMG response to quick stretch lasts for less than 0.10 seconds. At both time periods adductor longus was the muscle that most frequently exhibited a prolonged response to quick stretch (greater than 3.0 seconds in over 80% of subjects). This excessive stretch sensitivity, however, was not related to increased functional deficits in walking. In fact, greater adductor longus activity

TABLE 20-2
Duration of EMG Activity Following Rapid Stretching at Admission to Rehabilitation and Six Months after Stroke

	Admission					Six Months				
	% < 1 Sec	% 1–3 Sec	% > 3 Sec	Mean Sec	Median Sec	% < 1 Sec	% 1–3 Sec	% > 3 Sec	Mean Sec	Median Sec
Gluteus Maximus	90	6	4	0.44	0.12	90	8	2	0.33	0.10
Semimemb	45	15	40	2.03	1.14	32	19	49	2.49	2.96
Biceps Femoris	63	10	27	1.5	0.32	59	8	33	1.65	0.26
Adductor Longus	11	6	83	3.77	4.24	8	6	86	3.80	4.20
Rectus Femoris	65	14	21	1.25	0.36	53	10	37	1.94	0.84
Vastus Intermedius	76	10	14	0.98	0.24	74	13	13	0.97	0.38
Soleus	60	13	27	1.42	0.16	79	3	18	1.01	0.10
Anterior Tibialis	94	2	4	0.28	0.06	89	8	3	0.35	0.04

during gait was associated with greater (more normal) swing-phase knee flexion despite prolonged activation in rectus femoris. One-third to one-half of subjects exhibited prolonged responses to quick stretch in the hamstrings (semimembranosus and biceps femoris long head), rectus femoris, and soleus. Though less frequent, the impact of severe spasticity on walking in these muscles was greater than that of the adductor longus.

Spasticity of the hamstrings can limit knee extension in terminal swing and inhibit thigh flexion in initial swing with secondary decrease in peak swing knee flexion (36). Rectus femoris spasticity is a common contributor to reduced knee flexion in swing (36), and soleus spasticity can contribute to equinovarus, which can impair forward progression of body weight over the ankle in stance and impact foot clearance in swing (35). Both semimembranosus and rectus femoris demonstrated increased spasticity at the six-month test compared to the earlier evaluation, which corresponded with greater impairment in swing-phase knee flexion.

Although anterior tibialis does not often demonstrate increased response to passive stretch, excessive or continuous activity during walking is a common contributor to excessive subtalar joint varus (48). Although spasticity is not a strong determinant of walking function for the majority of individuals following stroke (49, 50), for those who do exhibit obstructive spasticity, both surgical interventions and anti-spasticity medication can produce increases in walking speed and reduce gait deviations at both the ankle and knee joints (35, 36, 51). (See Chapter 25 for more information.)

Joint Contractures

After stroke, longstanding weakness in a muscle group or spasticity in the antagonist group can result in increased passive stiffness and joint contractures (29, 47, 52). In the lower extremity the ankle plantar flexion contractures are the most common site for restrictions of joint motion, although inversion contractures are also common, and flexion contractures at the knee and hip joints develop occasionally, particularly in individuals with limited standing and walking function (35, 48, 53). Adequate passive joint mobility is necessary to obtain a stable passive alignment during the stance phase of gait.

Using a mechanical modeling simulation, Kagaya and colleagues (54) confirmed the clinical impression of the lower-extremity postures that produce maximal passive stability during stance, which comprised 5° of dorsiflexion at the ankle, 0° flexion at the knee, and 15° of extension at the hip (54, 55). The model predicted significant increases in muscle demand for postures at or greater than 6° of plantar flexion and 20° of flexion at the knee or hip joints. In a subsequent mechanical modeling simulation hip or knee flexion contractures of no greater than 15° or ankle plantar flexion contractures of less than 0° were required to maintain positive step length and forward movement of the center of gravity (54, 56). These theoretical models indicate that, while moderate to severe contractures are destabilizing, mild contractures may be accommodated, and in fact, mild ankle plantar flexion tightness (0°–5° of dorsiflexion) can augment support for weak calf muscles in stance (6, 45).

Proprioception

The influence of impaired joint position sense has proved to be more of a "yes/no" threshold rather than a scalar determinant of gait quality (5, 53). No significant difference in proprioceptive ability was identified between poor and good hemiparetic walkers (5, 19). Proprioception loss in the lower extremity tends to be more severe distally.

Absence or impaired proprioception in the ankle joint can be controlled with an ankle-foot orthosis that limits available motion at the ankle. If lack of proprioception extends to the knee joint, a knee-ankle-foot orthosis (KAFO) may be required to stabilize the knee in stance. If proprioception at the knee is impaired, but not absent, an unlocked KAFO may provide sufficient sensory input, but if joint position sense is absent at the knee, a locked KAFO is usually required for stability in stance. This is important for safety during walking since absent proprioception in the knee is related to the risk of frequent falls (57). The weight of the orthosis and increased energy cost of walking with an extended knee in swing typically result in KAFO use for household or exercise use only. If proprioception loss extends to the hip, potential for functional ambulation is typically limited. (See Chapter 31 for more detailed description of indicators for orthotic prescription.)

POST-STROKE WALKING REHABILITATION

Customary walking speed has been identified as the primary indicator of overall functional locomotor ability after stroke (5, 19). Moreover, improvements in walking speed categories following rehabilitation have been related to significant improvements in quality of life, confirming the impact that reduced walking speed has on functional mobility (58). Thus, the primary goals of walking rehabilitation programs are to develop walking ability at speeds and distances that are functionally significant for community ambulation. (See Chapter 16 for further discussion of walking speed as a global indicator of locomotor ability after stroke.)

In addition to gait training, walking rehabilitation programs address the various residual motor impairments identified as significant predictors of walking speed after stroke. Physical contributors to walking

deficits post-stroke include selective motor control (i.e., synergy stage), muscle strength and balance (19, 49, 57, 60–65). It is beyond the scope of this chapter to address all physical rehabilitation intervention that may contribute to walking recovery. Rather, we focus on the main predictors of walking recovery and best-practice strategies based on current evidence as guides for the clinician in developing effective walking rehabilitation programs for their patients with stroke.

Predictors of Walking Recovery and Responsiveness to Rehabilitation Interventions

Improvement in walking after stroke is accomplished by a combination of natural recovery and rehabilitation intervention (66). Pre-morbid factors as well as stroke-related variables can impact an individual's ability to respond to intervention. Both older age and greater initial severity of stroke have been found to be predictors of poor outcome following rehabilitation (67, 68). This finding may give the erroneous impression that older individuals or those with severe stroke do not make significant or functionally useful gains in rehabilitation. In individuals with more severe involvement, functional improvement occurs more gradually and the magnitude of improvement is not as large as that seen in subjects with moderate or mild stroke (69). Moreover, the mechanisms underlying functional improvement may differ depending on initial impairment level.

Degree of Neurologic Damage and Multiple Comorbidities. There are numerous covariates associated with motor recovery and responsiveness to therapy such as the pathophysiologic consequences and degree of neurologic damage directly related to the stroke as well as the personal and environmental factors associated with the individual. Cramer (70) discusses the impact that clinical variables such as stroke characteristics (e.g., location, volume, hemorrhagic or ischemic injury), time post-stroke, age, post-stroke depression, and other comorbidities have on recovery potential. With advances in imaging techniques such as functional magnetic resonance imaging and diffusion tensor imaging, the relationship between damage to neurologic structures, motor severity, and functional recovery potential are being elucidated (71).

In particular, the degree of pyramidal tract damage has been associated with both stroke severity and recovery potential for the upper extremity (72, 73). Case-series evidence for similar relationships in lower-extremity and walking recovery are emerging in adults and children with stroke at birth (55, 74). For both upper- and lower-extremity function, motor impairment severity measures such as the Fugl-Meyer motor assessment appear to be correlated with the degree of pyramidal tract damage. In other words, lower Fugl-Meyer motor scores indicate more synergistic movement patterns that are associated with a greater degree of pyramidal tract damage and less motor recovery potential. Thus, the clinical observations related to lower-extremity Fugl-Meyer (LEFM) scores and walking speed may be clinical predictors of both functional walking recovery and post-stroke brain damage. More importantly, therapeutic strategies for walking recovery most beneficial for individuals with mild to moderate stroke may differ compared to those with more severe stroke. (See Chapter 17 for more information on physiological and clinical predictors of recovery after stroke.)

Motor Severity. Recovery of walking speed is most strongly linked to the magnitude of initial motor impairment, with less recovery in those individuals with the greatest initial severity (63). The strength of this relationship increases up to the first month post-stroke (21, 75). Prediction of further recovery after the first two to six months is less clear. The total score from the lower-extremity motor portion of the Fugl-Meyer test has been identified as a moderate predictor of both walking speed and stride length in patients after a stroke (63, 76). Walking speed was positively correlated with the Brunnstrom motor stage (measure of selective motor control) in the proximal limb, but not with the motor stages of the ankle or foot (64). The authors concluded that the ability to compensate for poor distal motor control determined the individual's walking speed.

Lower-extremity muscle strength in the paretic limb is strongly correlated with maximum walking speed post stroke (19, 49, 57, 60, 65, 77). Specific muscle groups that demonstrated the strongest relationship with walking velocity varied greatly between studies depending on the number of muscles investigated, the parameter used to quantify strength (hand dynamometer force, isometric or isokinetic torques), and the method of documenting gait velocity (comfortable or fast speeds, distance walked, with or without assistive devices and orthoses).

Studies that compared multiple muscle groups most frequently identified strength in the hip flexor (25, 61, 78) and ankle plantar flexor (19, 65) muscle groups as the strongest predictor of walking speed post stroke, although strength in the knee extensor (61, 62) hip extensor (60), and ankle dorsiflexors (57, 78) muscle groups were also identified as significantly related to gait speed. The dominance of ankle plantar flexion strength in the studies of multiple muscle groups is consistent with the relationship of calf strength and initial severity of the stroke (79). Moreover, the contribution of the hip flexors and ankle plantar flexors to maximizing walking speed has been related to their large bursts of power generation late in the terminal stance and pre-swing phases of the gait cycle (12, 17, 19, 23, 25).

Kosak and Reding investigated the effectiveness of gait training using a body-weight supported treadmill (BWST) in stroke subjects who required moderate assistance to ambulate prior to training (80). In a post-hoc analysis, they found locomotor outcomes varied based on the initial severity of neurologic impairments; training using the BWST was more effective for individuals who required moderate assistance to ambulate initially and presented with combined motor, sensory, and visual impairments. However, it should be noted that this study only investigated stroke patients with severe locomotor impairments and was not a study that compared different locomotor impairment severity levels.

In contrast, Sullivan and colleagues specifically investigated the effect of locomotor severity in chronic stroke (81). Neurologic motor impairment, functional locomotor ability, and locomotor severity were analyzed as potential factors that might contribute to treatment effectiveness. They demonstrated that initial walking speed was independently correlated to change in overground walking speed as a result of training with BWST. Lower-extremity motor impairment severity as measured by the LEFM score was correlated highly with the magnitude of walking speed improvement.

The mechanisms behind the functional recovery in walking stimulated by the gait training using a BWST after stroke are likely dependent on the individual's initial motor severity (82). The increase in free walking velocity after six weeks of training with BWST and strengthening exercises in 20 individuals who were six months to five years post stroke was related more strongly to initial LEFM score ($r = 0.65$) and isometric ankle plantar flexion torque ($r = 0.57$) than to initial walking velocity ($r = 0.42$). This finding indicates that those subjects with more preserved motor function in the paretic limb, particularly distally, have a better capacity for restoration of walking function, and it is also consistent with the finding that relatively fast walking speeds can be achieved after stroke with abnormal kinematic patterns through compensatory strategies (20, 83).

Subjects with higher initial LEFM scores (above 25) increased gait speed by improving the terminal stance mechanics of the paretic limb at both the ankle and hip. The increase in the plantar flexion power in terminal stance was associated with improved pre-swing hip flexion power, which contributes to increases in both cadence and stride length. In fact, soleus was the only muscle with a significant correlation between increased activation level and increased pre-swing *hip flexion power* ($r = 0.54$). The hip flexion power was likely augmented by the improved elastic recoil of the ankle plantar flexors assisting with swing initiation of the limb. Most of those individuals with higher LEFM scores also had stronger ankle plantar flexors (more preserved distal function) and responded to the intervention with increased activation intensity of the soleus to accomplish these improved mechanics (increased by 15% of maximum). This represents restitution of walking function (Figure 20-6).

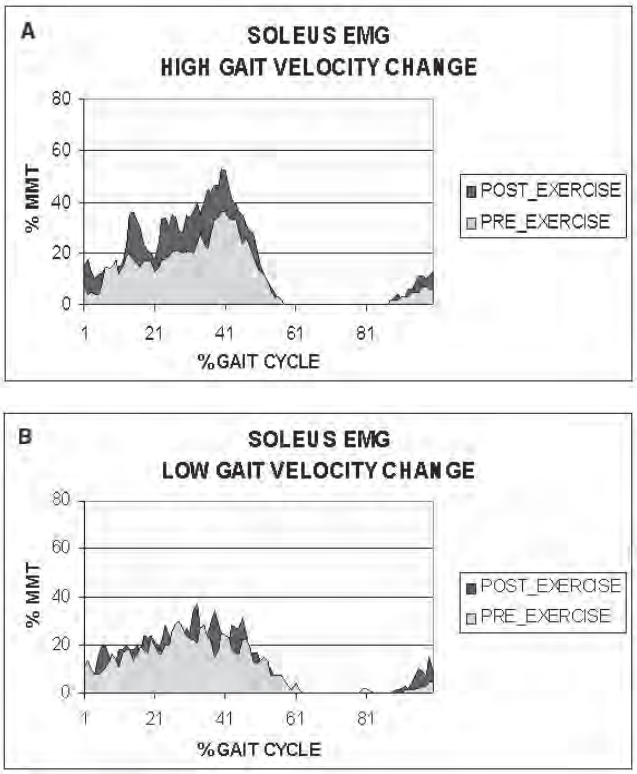

FIGURE 20-6

Change in soleus EMG during walking for subjects who demonstrated high gait velocity (top) and low walk gait velocity (bottom) increases with treadmill training with body-weight support.

In contrast, for those subjects with lower initial LEFM scores (and weaker plantarflexion strength), there was no correlation between change in ankle plantar flexion power and improved walking velocity (82, 84). Those subjects with low LEFM scores and a few individuals with higher LEFM scores but poor distal function improved paretic limb mechanics by other mechanisms (not related to increased soleus) including improved activity of proximal muscles (gluteus maximus, semimembranosus, and gluteus medius) leading to better knee and hip extension in stance, but no change in ankle plantar flexion power (85). Hip extension power absorption in terminal stance and hip flexion power generation in pre-swing, however, were related to improved velocity for those with low LEFM scores, indicating that these subjects relied on proximal muscle control of the paretic limb and increased swing-phase power of the non-paretic limb. This same proximal compensation pattern was documented in subjects without active ankle control from amputation at the ankle

joint (Symes level) (86) and for those with reduced ankle power from high-heeled shoes (87).

The proximal muscles demonstrating increased activation in subjects with poor distal control are consistent with predictions of a simulation model developed by Jonkers and colleagues (44), who demonstrated that gluteus maximus, medial hamstrings, and gluteus medius would all contribute to hip extension and heel-off in single-limb stance. Thus, individuals with less severe initial motor involvement from the stroke demonstrated a greater capacity to restore distal control and walking function with intervention, and those with more severe distal motor impairment made more modest improvements by increased proximal and contralateral activity.

Standing Balance. Standing balance also has shown strong correlation with walking speed after stroke, particularly for individuals with more severe impairments (49, 84, 88). Patterson and colleagues (49) identified that the strongest predictor of both short- (30 feet) and long-distance (6 minute) walking speed in a cross-sectional analysis was the Berg Balance Score for subjects who walked at or below the median speed (49). In contrast, peak VO2 was the strongest predictor for the faster subjects. Standing balance and lower-extremity strength are strongly related after stroke, and this relationship is particularly strong for those with greater impairments (62).

Improvement in standing balance has been found to be correlated with increased walking speed after basic rehabilitation and muscle strengthening interventions, particularly for individuals with more severe impairments (62, 84). For subjects with less severe initial impairments, however, Pohl and colleagues documented that improvements in lower-extremity motor function and cardiovascular fitness were most strongly associated with increased walking speed (84). This suggests a hierarchy of functional substrates for walking with the ability to control balance during single-limb stance as a requisite skill for even slow walking and greater muscle strength, selective motor control, and fitness as determinants of faster walking speeds (49, 62).

Walking Rehabilitation Interventions

Determining the appropriate and most effective interventions to use for walking recovery is one of the primary roles of the physical therapist during the acute, subacute, and chronic phases post stroke. Evidence is building that innovations in post-stroke walking rehabilitation such as resisted lower-extremity strengthening, task-specific training, and aerobic training are more effective than conventional neurophysiologic approaches used by physical therapists (89–91). Recent innovations in therapeutic approaches to walking rehabilitation derive from understanding the biomechanical and neurologic mechanisms that result in walking impairment after stroke.

As previously discussed in this chapter, post-stroke severity caused by primary motor impairments such as degree of lower-extremity weakness and selective motor control are significant predictors that contribute to the biomechanics of post-stroke gait and responsiveness to rehabilitation interventions. In addition, other secondary impairments such as balance dysfunction and deconditioning also contribute to post-stroke walking disability. Current evidence from rehabilitation research suggests that lower-extremity strength training, task-specific training such as body-weight supported treadmill training, and aerobic conditioning are interventions that contribute to gait recovery and increased community ambulation for individuals with walking disability after stroke (Figure 20-7).

Strength Training. Resisted strength training for the upper or lower limb after stroke has been questioned by therapists in the past because of the concern that resistance increases muscle spasticity and abnormal movement patterns. However, evidence is building that strength training leads to improvements in both lower-limb muscle strength and gait endurance post-stroke (91) without increasing spasticity (92, 93). The effectiveness of muscle strengthening to improve walking speed and endurance appears to relate to whether the strength training program is directed at single muscles or groups of muscles that are typically activated during the stance and swing phases of gait. For example, muscle strengthening for a single muscle group after stroke increases muscle strength but results in little or no improvement in walking speed or endurance (94). In contrast, strength training of multiple LE muscle groups is associated with modest functional changes in walking distance or improved balance and/or sit-to-stand ability but not increased walking speed (95–98). This finding is consistent with a recent study that used a resisted LE cycling task specifically designed to strengthen extensor and flexor groups with torque demands biased to emphasize the stance and swing phases of gait. The moderately high-intensity cycling program for 20–30 minutes of resisted cycling, two days a week for 6 six weeks, resulted in significant increases in post-intervention LE flexor torque production and walking endurance as measured by the distance walked in six minutes, but did not increase walking speed.

For individuals after stroke with moderate to mild walking impairment (i.e., baseline walking speeds approximately 0.30–0.80 m/s), strength programs that incorporate functional weight-bearing activities or dynamic high-intensity resistance training resulted in increases in strength of LE flexors and extensors that produced increases in walking speed, as well. Yang et al. used a program of progressive LE strengthening using functional

FIGURE 20-7

Rehabilitation interventions of treadmill training with body-weight support (top right), resisted lower-extremity cycling (top left), therapeutic progressive resistive exercise example (bottom left), and upper-extremity ergometry (bottom right).

weight bearing activities for exercise such as step climbing and single-limb heel raises to realize gains in LE strength and walking speed (99). Patten et al. found that a dynamic resistance training program of eccentric muscle strengthening using isokinetic exercises (15 sessions over five weeks) followed by clinic-based gait training (nine sessions over three weeks) resulted in increases in both gait speed and LE torque production (100). Increases in speed were not evident for the group that received concentric strength training with gait training. Together these findings suggest that functional task strengthening or strength training programs that emphasize the dynamic muscle control needed to support the kinematics, kinetics, and muscle activation of more normal gait are more effective strength training programs for walking recovery post stroke.

While it is evident that resisted LE strength training after stroke does not cause harm, there remains a relatively small body of literature that clearly elucidates the overall benefits or limitations of strength training. The recent Canadian Stroke Network (91) systematic review of LE strength training and mobility post stroke concludes that there is strong evidence that strength training improves distance walked after stroke but only moderate evidence that benefits extend to improved performance in other activities of daily living. Furthermore, our understanding of the appropriate dosing (i.e., intensity, frequency, duration) for significant strength gains in this group of patients with neurologic as well as peripheral mechanisms of weakness are not well understood. This lack of understanding is further complicated by lack of studies that have examined how principles of exercise that apply to healthy young and older adults apply to individuals with stroke. For example, a recent study that investigated the combined benefits of a rehabilitation program that alternated therapy days of LE strength training with high-intensity treadmill training with body weight support found evidence of overtraining (i.e., impaired strengthening) in the group that received successive days of LE muscle exercise with the group that received a program in which UE and LE exercise were provided on alternate days (101). Clearly, more investigation into maximizing the effects of resisted exercise post stroke is needed.

Task-Specific Training. Task-specific training is the repetitive practice of a task that is specific to the intended outcome. Treadmill training is a therapeutic modality that allows the individual with stroke to actively engage in repetitive stepping. Walking on a treadmill is an example of task-specific gait training that appears to be critical to the achievement of improved walking speed and endurance (95, 102–104). Treadmill training with body-weight support (TM-BWS) is an adaptation of treadmill walking that involves fitting the patient in a harness and suspending them over a treadmill with a portion of the patient's body weight reduced so that a physical therapist, with help from another therapist or aide if needed, can assist the patient to step on the moving treadmill belt. Visintin et al. demonstrated that treadmill training with 40% body weight support (BWS) provided in early training that was progressively decreased over training sessions resulted in better walking outcomes for individuals in the acute and subacute phases post stroke than treadmill training without BWS (105).

In a control-comparison RCT of 80 individuals with chronic stroke who had severe to moderate walking impairment (average gait velocity: severe, 0.25 ± 0.12 m/s; moderate, 0.71 ± 0.12 m/s), Sullivan et al. demonstrated a statistically significant, and clinically meaningful, increase in both walking speed and distance that was maintained at a six-month follow-up for *any* of the exercise groups that received TM-BWS compared to the group that received resisted cycling (101). In this study, there were four exercise groups that received exercise four days per week for six weeks. Three of the groups received TM-BWS combined with another exercise such that the program included two days per week of TM-BWS alternated with two days per week of upper-extremity ergometry, progressive resistance LE exercise, or resisted cycling. The fourth group received two days per week of resisted cycling alternated with two days per week of upper-extremity ergometry. Each group was controlled for time in the exercise session (one hour per session) and intensity (moderately high). Moderate- to high-intensity TM-BWS sessions included 20 minutes of cumulative walking time on the treadmill at speeds that ranged from 1.8 to 2.3 mph. In addition to providing specific dose parameters and intervention protocol specifications for the exercise programs, this study demonstrated the overarching benefit of an exercise program that included TM-BWS for making long-term changes (six-month follow-up) in both walking speed and distance in individuals with chronic stroke.

Several recent systematic reviews have suggested that the collective findings on treadmill training or TM-BWS effectiveness compared to other gait interventions remain inconclusive (91, 106, 107). However, there is strong evidence that task-specific functional training performed at high intensity may be a critical factor that modulates gait training effectiveness (89, 108, 109). With task-specific gait therapies such as treadmill training or TM-BWS, responsiveness to this particular intervention is related to the training parameters used. Recent evidence consistently demonstrates that treadmill training at higher speeds (i.e., higher intensity), with or without body-weight support, is more effective for improving gait speed post stroke than training at slower speeds (81, 110, 111). These reports of differences in therapeutic effectiveness related to the type and dosing of the exercise program illustrate the

importance of exercise prescription. Exercise prescription is a major contributor to therapy outcomes and most likely contributes to the inconclusive findings of systematic reviews that have not included trials in which training intensity is controlled.

Why are higher intensity, task-specific gait interventions more likely to achieve functionally significant changes in walking outcomes after stroke? We propose that walking rehabilitation that combines *specificity* of training intrinsic to walking with the *intensity* of walking at challenging speeds requires that the individual post stroke respond to and develop the gait dynamics and muscle activation patterns associated with more normal gait parameters. Thus, changes in walking recovery and response to appropriately selected and dosed walking interventions are related to the residual damage to the nervous system post stroke and the response of the nervous system to interventions that provide an appropriate dynamic stimulus to the nervous system to drive recovery. Long-term changes in performance are consistently achieved when the conditions of task practice are similar to the task and conditions in which long-term changes in performance are expected (112). For individuals post stroke, walking is a highly practiced functional task in which the learner has to reacquire the motor abilities associated with gait function; thus, improvements in walking outcomes post stroke appear to be more likely with task-specific interventions like treadmill walking with or without BWS. (See Chapter 17 for similar discussion.)

Aerobic Training. Deconditioning after stroke is a chronic health condition that is evident across the acute, subacute, and chronic phases post stroke (see Chapter 23 for more information). Aerobic training can be safely incorporated into rehabilitation exercise programs after stroke. Physical activity and exercise post stroke are highly recommended to address deconditioning and to reduce secondary risks for the post-stroke survivor (113). In addition, valuable evidence-based sources are now available for the rehabilitation specialist that provide heart rate and blood pressure guidelines to individually monitor the patient during moderately high exercise programs (for example, see: http://www.apta.org/AM/Template.cfm?Section=PFSP_Pocket_Guides&Template=/MembersOnly.cfm&ContentID=42129). While cardiovascular fitness is essential for good health after stroke, there is building evidence that specific types of aerobic programs are also associated with improved walking outcomes for the stroke survivor.

Pooled data from systematic reviews and well-designed RCTs reveal that walking outcomes such as walking speed and distance, functional tasks such as stair climbing, and increased community mobility and participation are positively impacted by aerobic training for patients with subacute and chronic stroke (91, 106, 114). Across these studies large-muscle activities such as walking, treadmill, stationary cycle, combined arm-leg ergometry, arm ergometry, and seated steppers were used. Exercise intensity among studies in which walking outcomes were improved ranged from 3 to 19 weeks, 20–40 minutes, three to five days per week at 50%–80% of heart rate reserve. Pooled data show that aerobic exercise had a small, statistically significant effect on walking speed 0.26 m/s (95% CI: 0.05–0.48, $p = 0.008$) and a moderate, statistically significant effect on walking distance 0.30 meters (95% CI: 0.06–0.55, $p = 0.008$).

Aerobic exercise training specifically designed to improve gait and gait-related activities is associated with greater clinically and statistically significant gains in walking outcomes (see systematic review by Van de port (115). Activities such as cycling, water-based exercise, and gait-oriented cardiovascular training resulted in greater post-intervention changes in walking speed 0.45 m/s (95% CI: 0.27–0.63) and distance 0.62 meters (95% CI: 0.30–0.95). Across the studies reviewed the most common exercise prescription (i.e., frequency, intensity, duration) averaged three times per week for 60 minutes across four weeks (ranges: 8–90 minutes; three to five time per week; 4–19 weeks). Exercise intensity in terms of percentage of heart rate maximum, heart rate reserve, or rate of perceived exertion was not reported.

The wide confidence intervals are most likely associated with the wide variety of exercise methods and prescriptions that ranged across the various trials. Once again, well-designed aerobic exercise rehabilitation clinical trials are needed to better understand the exercise method and aerobic training parameters that result in clinically significant walking recovery in individuals post stroke. However, efficacy for physical activity and aerobic training to improve walking outcomes after stroke is evident.

RESEARCH FRONTIERS

Future directions in walking rehabilitation therapies will most likely include the use of technology to enhance current walking rehabilitation interventions. Currently, technological advances in walking neurorehabilitation that are in either experimental (pre-clinical) or early phase I clinical trials are in the areas of lower-limb assisted robotics, implantable electrodes for neuromuscular function, and electrical stimulation.

Lower-limb assisted robotics are in development that will provide machine-assisted stepping since the physical demands during treadmill training are high for the therapist. Examples include both motorized (116) and gravity-balanced (117) exoskeletons that serve as gait-assisted orthotics during walking on a treadmill with a supportive harness and body-weight support. A recent

clinical trial that examined walking outcomes in patients with chronic stroke demonstrated that therapist-assisted stepping was more effective than robotic-assisted stepping with a motorized exoskeleton (118). However, more study is needed to determine the optimal training paradigms that integrate the expertise of the therapy clinician with the most effective mechanical assistance.

Advances in technology and reduced device size have allowed neuromuscular electrical stimulation (NMES) to be used functionally as a neuroprosthesis. There is strong evidence that NMES, particularly peroneal nerve stimulation, in conjunction with gait training is an effective walking rehabilitation strategy (91). Surface electrode stimulation devices are readily available for clinical use; however, future research endeavors will most likely include the development of implantable electrode devices that will increase the effectiveness of NMES and increase ease of use and comfort for the patient (119). A recent study by Kottink et al. demonstrates initial evidence for the feasibility of NMES with implantable electrodes as an adjunct to walking rehabilitation therapy (120).

CONCLUSION

This chapter summarizes the temporal gait characteristics and kinematic and kinetic features associated with post-stroke gait dysfunction. Stroke severity affects the dynamic strategies used for walking after stroke. Stroke severity as measured by clinical assessments of LE motor impairment assessment (i.e., selective movement control, strength, spasticity) or functional walking ability (i.e., walking speed or distance) is also used as an indicator of stroke walking impairment severity and are associated with predictions of walking outcomes and disability after stroke. The capacity to recover movement control as indicated by changes in force production, increased movement selectivity, and appropriate motor unit activation and recruitment during walking appears to be related to the degree of corticospinal tract damage and the methods used during walking rehabilitation.

Walking recovery after stroke appears to be associated with rehabilitation strategies that incorporate strengthening of functional muscle groups with power and activation patterns that are more similar to those used in gait. Task-specific interventions such as treadmill walking with or without BWS are examples of effective rehabilitation strategies that incorporate the demands of gait into repetitive stepping and task-specific practice most similar to that of normal gait demands. Furthermore, aerobic training in gait-related activities or LE cycling tasks appear to be effective interventions to increase cardiovascular fitness associated with reduced activity levels after stroke. Collectively, these types of rehabilitation interventions improve walking outcomes and lead to enhanced participation and quality of life for individuals with stroke.

References

1. Duncan PW, Zorowitz R, Bates B, et al. Management of adult stroke rehabilitation care: a clinical practice guideline. *Stroke* 2005; 36(9):e100–143.
2. Jorgensen HS, Nakayama H, Raaschou HO, Olsen TS. Recovery of walking function in stroke patients: the Copenhagen Stroke Study. *Arch Phys Med Rehabil.* 1995; 76(1):27–32.
3. Forster A, Young J. Incidence and consequences of falls due to stroke: a systematic inquiry. *BMJ.* 1995; 311(6997):83–86.
4. Keenan MA, Perry J, Jordan C. Factors affecting balance and ambulation following stroke. *Clin Orthop.* 1984(182):165–171.
5. Perry J, Garrett M, Gronley JK, Mulroy SJ. Classification of walking handicap in the stroke population. *Stroke* Jun 1995; 26(6):982–989.
6. Perry J. *Gait analysis: Normal and pathologic function*. Thorofare, NJ: Slack, Inc., 1992.
7. Mahoney FI, Barthel DW. Functional evaluation: The Barthel Index. *Md State Med J.* 1965; 41:61–65.
8. Hamilton BB, Laughlin JA, Fiedler RC, Granger CV. Interrater reliability of the 7-level Functional Independence Measure (FIM). *Scandinavian Journal of Rehabilitation Medicine* 1994; 26(3):115–119.
9. Chen G, Patten C, Kothari DH, Zajac FE. Gait deviations associated with post-stroke hemiparesis: improvement during treadmill walking using weight support, speed, support stiffness, and handrail hold. *Gait Posture* 2005; 22(1):57–62.
10. Goldie PA, Matyas TA, Evans OM. Gait after stroke: initial deficit and changes in temporal patterns for each gait phase. *Arch Phys Med Rehabil.* 2001; 82(8): 1057–1065.
11. Olney SR, CJ. Hemiparetic gain following stroke: Part I: Characteristics. *Gait & Posture.* 1996; 4:136–148.
12. Olney SJ, Griffin MP, McBride ID. Temporal, kinematic, and kinetic variables related to gait speed in subjects with hemiplegia: a regression approach. *Phys Ther.* 1994; 74(9):872–885.
13. De Quervain IA, Simon SR, Leurgans S, et al. Gait pattern in the early recovery period after stroke. *Journal of Bone & Joint Surgery - American Volume* 1996; 78(10): 1506–1514.
14. Balasubramanian CK, Bowden MG, Neptune RR, Kautz SA. Relationship between step length asymmetry and walking performance in subjects with chronic hemiparesis. *Arch Phys Med Rehabil.* 2007; 88(1):43–49.
15. Mulroy S, Gronley J, Weiss W, et al. Use of cluster analysis for gait pattern classification of patients in the early and late recovery phases following stroke. *Gait & Posture* 2003; 18(1):114–25.
16. Waters HJ, Perkin JM. Study of stroke patients in a single general practice. *Br Med J (Clin Res Ed).* 1982; 284(6318):791–793.
17. Olney SJ, Griffin MP, Monga TN, McBride ID. Work and power in gait of stroke patients. *Arch Phys Med Rehabil.* 1991; 72:309.
18. Wong AM, Pei YC, Hong WH, et al. Foot contact pattern analysis in hemiplegic stroke patients: an implication for neurologic status determination. *Arch Phys Med Rehabil.* 2004; 85(10):1625–1630.
19. Kim CM, Eng JJ. The relationship of lower-extremity muscle torque to locomotor performance in people with stroke. *Physical Therapy* 2003; 83(1):49–57.
20. Bowden MG, Balasubramanian CK, Neptune RR, Kautz SA. Anterior-posterior ground reaction forces as a measure of paretic leg contribution in hemiparetic walking. *Stroke* 2006; 37(3):872–876.
21. Brunnstrom S. *Movement therapy in hemiplegia*. New York: Harper & Row, 1970.
22. Turns LJ, Neptune RR, Kautz SA. Relationships between muscle activity and anteroposterior ground reaction forces in hemiparetic walking. *Arch Phys Med Rehabil.* 2007; 88(9):1127–1135.
23. Nadeau S, Gravel D, Arsenault AB, Bourbonnais D. Plantarflexor weakness as a limiting factor of gait speed in stroke subjects and the compensating role of hip flexors. *Clin. Biomech.* 1999; 14(2):125–135.
24. Parvataneni K, Olney SJ, Brouwer B. Changes in muscle group work associated with changes in gait speed of persons with stroke. *Clin Biomech.* 2007; 22(7):813–820.
25. Nadeau S, Arsenault AB, Gravel D, Bourbonnais D. Analysis of the clinical factors determining natural and maximal gait speeds in adults with a stroke. *Am.J.Phys.Med. Rehabil.* 1999; 78(2):123–130.
26. Twitchell TE. The restoration of motor function following hemiplegia in man. *Brain* 1951; 74:443–480.

27. Clark DJ, Condliffe EG, Patten C. Activation impairment alters muscle torque-velocity in the knee extensors of persons with post-stroke hemiparesis. *Clinical Neurophysiology* 2006; 117(10):2328–2337.
28. Andrews K, Brocklehurst JC, Richards B, Laycock PJ. The rate of recovery from stroke—and its measurement. *Int Rehabil Med.* 1981; 3(3):155–161.
29. Lamontagne A, Malouin F, Richards CL, Dumas F. Mechanisms of disturbed motor control in ankle weakness during gait after stroke. *Gait & Posture* 2002; 15:244.
30. Higginson JS, Zajac FE, Neptune RR, et al. Muscle contributions to support during gait in an individual with post-stroke hemiparesis. *Journal of Biomechanics* 2006; 39(10):1769.
31. Den Otter AR, Geurts ACH, Mulder TH, Duysens J. Gait recovery is not associatted with changes in the temporal patterning of muscle activity during treadmill walking in patients with post-stroke hemiparesis. *Clinical Neurophysiology* 2006; 117:4.
32. Den Otter AR, Geurts AC, Mulder T, Duysens J. Abnormalities in the temporal patterning of lower-extremity muscle activity in hemiparetic gait. *Gait & Posture* 2007; 25(3):343.
33. Andrews AW, Bohannon RW. Short-term recovery of limb muscle strength after acute stroke. *Arch Phys Med Rehabil.* 2003; 84:125.
34. Jorgensen L, Jacobsen BK. Changes in muscle mass, fat mass, and bone mineral content in the legs after stroke: a 1 year prospective study. *Bone* 2001; 28 (6):655.
35. Pinzur MS, Sherman R, Dimonte-Levine P, et al. Adult-onset hemiplegia: changes in gait after muscle-balancing procedures to correct the equinus deformity. *Journal of Bone and Joint Surgery* 1986; 68A(8):1249.
36. Kerrigan DC, Gronley JK, Perry J. Stiff-legged gait in spastic paralysis: a study of quadriceps and hamstring activity. *American Journal of Physical Medicine* 1991; 70(6):294.
37. Hachisuka K, Umezu Y, Ogata H. Disuse muscle atrophy of lower limbs in hemiplegic patients. *Arch Phys Med Rehabil.* 1997; 78(1):13–18.
38. Kautz SA, Duncan PW, Perera S, et al. Coordination of hemiparetic locomotion after stroke rehabilitation. *Neurorehabilitation and Neural Repair.* 2005; 19(3):250.
39. Neckel N, Pelliccio M, Nichols D, Hidler J. Quantification of functional weakness and abnormal synery patterns in the lower limb of individuals with chronic stroke. *J Neuroengineering Rehabil.* 2006; 3(17):1.
40. Goldberg EJ, Neptune RR. Compensatory strategies during normal walking in response to muscle weakness and increased hip joint stiffness. *Gait & Posture* 2007; 25:360.
41. Zajac FE, Neptune RR, Kautz SA. Biomechanics and muscle coordination of human walking part II: Lessons from dynamical simulations and clinical implications. *Gait & Posture* 2003; 17:1.
42. Nadeau S, Gravel D, Arsenault AB, Bourbonnais D. Plantarflexor weakness as a limiting factor of gait speed in stroke subjects and the compensating role of hip flexors. *Clin Biomech.* 1999; 14(2):125–135.
43. Richards CL, Malouin F, Bravo G, et al. The role of technology in task-oriented training in persons with subacute stroke: a randomized controlled trial. *Neurorehabilitation & Neural Repair* 2004; 18(4):199–211.
44. Jonkers I, Stewart C, Spaepen A. The study of muscle action during single support and swing phase of gait: clinical relevance of forward simulation techniques. *Gait & Posture* 2003; 17:97.
45. Lamontagne A, Richards CL, Malouin F. Coactiviation during gait as an adaptive behavior after stroke. *Journal of Electromyography and Kinesiology* 2000; 10:407.
46. Chen G, Patten C, Kothari DH, Zajac FE. Gait differences between individuals with post-stroke hemiparesis and non-disabled controls at matched speeds. *Gait & Posture* 2005; 22:51.
47. Cooper A, Musa IM, van Deursen R, Wiles CM. Electromyography characterization of stretch responses in hemiparetic stroke patients and their relationship with the Modified Ashworth scale. *Clin Rehabil.* 2005; 19(7):760.
48. Perry J, Waters RL, Perrin T. Electromyographic analysis of equinovarus following stroke. *Clinical Orthopaedics and Related Research* 1978; 131:47.
49. Patterson SL, Forrester LW, Rodgers MM, et al. Determinants of walking function after stroke: differences by deficit severity. *Arch Phys Med Rehabil.* 2007; 88(1): 115–119.
50. Sommerfeld DK, Eek EU, Svensson A, et al. Spasticity after stroke: Its occurence and association with motor impairments and activity limitations. *Stroke* 2004; 35:134.
51. Francisco GE, Boake C. Improvement in walking speed in poststroke spastic hemiplegia after intrathecal baclofen therapy: a preliminary study. *Arch Phys Med Rehabil.* 2003; 84(8):1194.
52. Chung SG, van Rey E, Zhiqiang B, et al. Biomechanic changes in passive properties of hemiplegic ankles with spastic hypertonia. *Arch Phys Med Rehabil.* 2004; 85(1638):1646.
53. Keenan MA, Ure K, Smith CW, Jordan C. Hamstring release for knee flexion contracture in spastic adults. *Clinical Orthopaedics and Related Research* 1988; 236:221.
54. Kagaya H, Sharma M, Kobetic R, Marsolais EB. Ankle, knee, and hip moments during standing with and without joint contractures: simulation study for functional electrical stimulation. *Am J Phys Med Rehabil.* 1998; 77:49–54.
55. Dobkin BH, Firestine A, West M, et al. Ankle dorsiflexion as an fMRI paradigm to assay motor control for walking during rehabilitation. *Neuroimage* 2004; 23:370–381.
56. Kagaya H, Ito S, Iwani T, Obinata G, Shimada Y. A computer simulation of human walking in persons with joint contractures. *Tohoku Journal of Experimental Medicine* 2003; 200(1):31.
57. Soyuer F, Ozturk A. The effect of spasticity, sense, and waking aids in falls of people after chronic stroke. *Disability and Rehabilitation* 2007; 29(9):679.
58. Schmid A, Duncan PW, Studenski S, et al. Improvements in Speed-Based Gait Classifications Are Meaningful. *Stroke* 2007; 38(7):2096–2100.
59. Lin PY, Yang YR, Chen SJ, Wang RY. The relationship between ankle impairments and gait velocity and symmetry in people with stroke. *Arch Phys Med Rehabil.* 2006; 87(4):562.
60. Bohannon RW. Strength of lower limb related to gait velocity and cadence in stroke patients. *Physiother Can.* 1986; 38:204–206.
61. Hsu A, Tang P, Jan M. Analysis of impairments influencing gait velocity and asymmetry of hemiplegic patients after mild to moderate stroke. *Arch Phys Med Rehabil.* 2003; 84:1185.
62. Suzuki K, Imada G, Iwaya T, et al. Determinants and predictors of the maximum walking speed during computer-assisted gait training in hemiparetic stroke patients. *Arch Phys Med Rehabil.* 1999; 80:179.
63. Chae J, Johnston M, Hekyung K, Zorowitz R. Admission motor impairment as a predictor of physical disability after stroke rehabilitation. *Am J Phys Med Rehabil.* 1995; 74:218.
64. Chen CL, Chen HC, Tang SFT, et al. Gait performance with compensatory adaptations in stroke patients with different degrees of motor recovery. *Am J Phys Med Rehabil.* 2003; 82:925.
65. Bohannon RW. Selected determinants of ambulatory capacity in patients with hemiplegia. *Clinical Rehabilitation.* 1989; 3:47–53.
66. Richards CL, Olney S. Hemiparetic gait following stroke: Part II Recovery and physical therapy. *Gait and Posture* 1996; 4:149–162.
67. Kwakkel G, Kollen B, Lindeman E. Understanding the pattern of functional recovery after stroke: Facts and theories. *Restorative Neurology and Neuroscience* 2004; 22:281.
68. Lofgren B, Nyberg L, Gustafson Y. Rehabilitation of stroke patients who are older and severely affected: Short- and long term perspectives. *Top Stroke Rehab.* 2000; 6(4):20
69. Mayo NE. Epidemiology and recovery. *Physical Medicine and Rehabilitation*; *State of the Art Reviews* 1993; 7:1.
70. Cramer SCM. Functional Imaging in Stroke Recovery. *Stroke* 2004; 35(11): 2695–2698.
71. Stinear CM, Barber PA, Smale PR, et al. Functional potential in chronic stroke patients depends on corticospinal tract integrity. *Brain* 007; 130(Pt 1):170–180.
72. Binkofski F, Seitz RJ, Arnold S, et al. Thalamic metbolism and corticospinal tract integrity determine motor recovery in stroke. *Annals of Neurology* 1996; 39(4):460–470.
73. Feydy A, Carlier R, Roby-Brami A, et al. Longitudinal study of motor recovery after stroke: recruitment and focusing of brain activation. *Stroke* 2002; 33(6):1610–1617.
74. Phillips JP, Sullivan KJ, Burtner PA, et al. Ankle dorsiflexion fMRI in children with cerebral palsy undergoing intensive body-weight-supported treadmill training: a pilot study. *Developmental Medicine & Child Neurology* 2007; 49(1):39–44.
75. Duncan PW, Goldstein LB, Matchar D, et al. Measurement of motor recovery after stroke: Outcome assessment and sample size requirements. *Stroke* 1992; 23:1084.
76. Brandstater M, deBruin H, Gowland C, Clark BM. Hemiplegic gait: analysis of temporal variables. *Arch Phys Med Rehabil.* 1983; 64:583.
77. Bohannon RW. Relavence of muscle strength to gait performance in patients with neurologic disability. *J Neuro Rehab.* 1989; 3:97–100.
78. Lin SI. Motor function and joint position sense in relation to gait performance in chronic stroke patients. *Arch Phys Med Rehabil.* 2005; 86(2):197.
79. Hendricks HT, Pasman JW, van Limbeek J, Zwarts MJ. Motor evoked potential of the lower extremity in predicting motor recovery and ambulation after stroke: a cohort study. *Arch Phys Med Rehabil.* 2003; 84:1373.
80. Kosak MC, Reding MJ. Comparison of partial body weight-supported treadmill gait training versus aggressive bracing assisted walking post stroke. *Neurorehabilitation & Neural Repair.* 2000; 14(1):13–19.
81. Sullivan KJ, Knowlton BJ, Dobkin BH. Step training with body weight support: effect of treadmill speed and practice paradigms on poststroke locomotor recovery. *Arch Phys Med Rehabil.* 2002; 83(5):683–691.
82. Klassen TD, Mulroy SJ, Sullivan KJ. Gait parameters associated with responsiveness to a task-specific and/or strengthening program in individuals post-stroke. *J Neurol Phys Ther.* 2006; 29:4.
83. Kim CM, Eng JJ. Magnitude and pattern of 3D kinematic and kinetic gait profiles in persons with stroke: relationship to walking speed. *Gait & Posture* 2004; 20:140.
84. Pohl PS, Perera S, Duncan PW, et al. Gains in distance walking in a 3-month follow-up poststroke: What changes? *Neurorehabilitation and Neural Repair* 2004; 18:30.
85. Sullivan KJ, Klassen T, Mulroy S. Combined task-specific training and strengthening effects on locomotor recovery post-stroke: a case study. *Journal of Neurologic Physical Therapy* 2006; 30(3):130–141.
86. Dillon MP, Barker TM. Preservation of residual foot length in partial foot amputation: a biomechanical analysis. *Foot and Ankle International* 2006; 27 (2):110.
87. Esmail A, Walsh S, Walhein G, Gitter A. Kinetics of high-heeled gait. *J Am Podiatr Med Assoc.* 2003; 93:27.
88. Kollen B, van de Port I, Lindeman E, et al. Predicting improvement in gait after stroke: A longitudinal prospective study. *Stroke* 2005; 36:2676.
89. Kwakkel GP, van Peppen RMPT, Wagenaar RCP, et al. Effects of Augmented Exercise Therapy Time After Stroke: A Meta-Analysis. *Stroke* 2004; 35(11):2529–2536.
90. Van Peppen RPS, Kwakkel G, Wood-Dauphinee S, et al. The impact of physical therapy on functional outcomes after stroke: what's the evidence? *Clinical Rehabilitation* 2004; 18(8):833–862.
91. Foley NT, Bhogal, S. Mobility and the lower extremity. In: Teasell RFN, Salter K, Bhogal S, Jutai J, Speechley M., eds. *Evidence-Based Review of Stroke Rehabilitation*. Canadian Stroke Network; 2007:1–105.
92. Brown DA, Kautz SA. Increased workload enhances force output during pedaling exercise in persons with poststroke hemiplegia. *Stroke* 1998; 29(3):598–606.

93. Patten CL, J; Brown, HE. weakness and strength training in persons with poststroke hemiplegia: Rationale, method, and efficacy. *Journal of Rehabilitation Research & Development*. 2004; 41(3A):293–312.
94. Sharp SA, Brouwer BJ. Isokinetic strength training of the hemiparetic knee: effects on function and spasticity. *Arch Phys Med Rehabil.* 1997; 78(11):1231–1236.
95. Dean CMR, Malouin, F. Task-related circuit training improves performance of locomotor tasks in chronic stroke: a randomized, controlled pilot trial. *Arch Phys Med Rehabil.* 2000; 81(4):409–417.
96. Ouellette MM, LeBrasseur NK, Bean JF, et al. High-intensity resistance training improves muscle strength, self-reported function, and disability in long-term stroke survivors. *Stroke* 2004; 35(6):1404–1409.
97. Teixeira-Salmela LF, Olney SJ, Nadeau S, Brouwer B. Muscle strengthening and physical conditioning to reduce impairment and disability in chronic stroke survivors. *Arch.Phys. Med.Rehabil.* 1999; 80(10):1211–1218.
98. Weiss A, Suzuki T, Bean J, Fielding RA. High intensity strength training improves strength and functional performance after stroke. *American Journal of Physical Medicine & Rehabilitation* 79(4):369–376.
99. Yang Y-R, Wang R-Y, Lin K-H, et al. Task-oriented progressive resistance strength training improves muscle strength and functional performance in individuals with stroke. *Clinical Rehabilitation* 2006; 20(10):860–870.
100. Patten C, Dozono J, Jonkers I. Gait speed improves significantly following dynamic high-intensity resistance training in persons poststroke. *Stroke* 2007; 38(2): 466–467.
101. Sullivan KJ, Brown DA, Klassen T, et al. Effects of task-specific locomotor and strength training in adults who were ambulatory after stroke: results of the STEPS randomized clinical trial. *Physical Therapy* 2007; 87(12):1580–1602; discussion 1603–1587.
102. Ada L, Dean CM, Hall JM, et al. A treadmill and overground walking program improves walking in persons residing in the community after stroke: a placebo-controlled, randomized trial. *Archives of Physical Medicine & Rehabilitation* 2003; 84(10): 1486–1491.
103. Richards CL, Malouin F, Wood-Dauphinee S, et al. Task-specific physical therapy for optimization of gait recovery in acute stroke patients. *Arch Phys Med Rehabil.* 1993; 74(6):612–620.
104. Salbach NM, Mayo NE, Wood-Dauphinee S, et al. A task-orientated intervention enhances walking distance and speed in the first year post stroke: a randomized controlled trial. *Clinical Rehabilitation* 2004; 18(5):509–519.
105. Visintin M, Barbeau H, Korner-Bitensky N, Mayo NE. A new approach to retrain gait in stroke patients through body weight support and treadmill stimulation. *Stroke* 1998; 29(6):1122–1128.
106. Brosseau L WG, Hillel F, et al. Ottawa panel evidence-based clinical practice guidelines for post-stroke rehabilitation. *Topics in Stroke Rehabilitation* 2006; 13(2):1–26.
107. Moseley AM, Stark A, Cameron ID, Pollock A. Treadmill training and body weight support for walking after stroke. *Cochrane Database of Systematic Reviews* 2005 (4):CD002840.
108. Foley NT, Bhogal, Speechley, M. The efficacy of stroke rehabilitation. In: Teasell RFN, Salter K, Bhogal S, Jutai J, Speechley M., eds. *Evidence-Based Review of Stroke Rehabilitation*. Canadian Stroke Network; 2007:1–53.
109. Langhorne P, Wagenaar R, Partridge C. Physiotherapy after stroke: more is better? *Physiother Res Int*. 1996; 1(2):75–88.
110. Pohl MM. Speed-dependent treadmill training in ambulatory hemiparetic stroke patients: a randomized controlled trial. *Stroke* 2002; 33(2):553–558.
111. Lamontagne A, Fung J. Faster is better: implications for speed-intensive gait training after stroke. *Stroke* 2004; 35(11):2543–2548.
112. Schmidt RAL, *Motor Control and Motor Learning: A Behavioral Emphasis*. 4th ed. Champaign, IL: Human Kinetics, 2005.
113. Gordon NF, Gulanick M, Costa F, et al. Physical activity and exercise recommendations for stroke survivors: an American Heart Association scientific statement from the Council on Clinical Cardiology, Subcommittee on Exercise, Cardiac Rehabilitation, and Prevention; the Council on Cardiovascular Nursing; the Council on Nutrition, Physical Activity, and Metabolism; and the Stroke Council. Circulation 2004; 109(16):2031–2041.
114. Duncan P, Studenski S, Richards L, et al. Randomized Clinical Trial of Therapeutic Exercise in Subacute Stroke. *Stroke* 2003; 34(9):2173–2180.
115. Van de port 2007
116. Mayr A, Kofler M, Quirbach E, et al. Prospective, blinded, randomized crossover study of gait rehabilitation in stroke patients using the lokomat gait orthosis. *Neurorehabil Neural Repair.* 2007; 21(4):307–314.
117. Agrawal SK, Banala SK, Fattah A, et al. assessment of motion of a swing leg and gait rehabilitation with a gravity balancing exoskeleton. *Neural Systems and Rehabilitation Engineering, IEEE* 2007; 15(3):410–420.
118. Hornby TG, Campbell DD, Kahn JH, et al. Enhanced gait-related improvements after therapist- versus robotic-assisted locomotor training in subjects with chronic stroke: a randomized controlled study. *Stroke* 2008; 39(6):1786–1792.
119. Anke IR, Kottink AIR. The orthotic effect of functional electrical stimulation on the improvement of walking in stroke patients with a dropped foot: a systematic review. *Artificial Organs* 2004; 28(6):577–586.
120. Kottink AIR, Hermens HJ, Nene AV, et al. Therapeutic effect of an implantable peroneal nerve stimulator in subjects with chronic stroke and footdrop: a randomized controlled trial. *Phys Ther.* 2008; 88(4):437–448.