Review articles

Hemiparetic gait following stroke. Part I: Characteristics

Sandra J. Olney*[a], Carol Richards[b]

[a]*School of Rehabilitation Therapy, Queen's University, Kingston, Ontario K7L 3N6, Canada*
[b]*Physiotherapy Department, Faculty of Medicine, Laval University, Quebec City, Province of Quebec G1K 7P4, Canada*

Abstract

The biomechanical patterns that characterize the gait of persons who have sustained a stroke are reviewed. Reduced walking speed and longer stance phases, greater on the unaffected side, are reported. Variations in joint excursions include several deviations at initial contact and reduced excursions during swing. Electromyographic patterns have provided a classification method. Joint moment reports were variable, but included high hip flexor moments in late stance, positively related to speed. The muscle groups of the unaffected side performed about 60% of the work of walking across speeds. Commonly occurring gait deviations resulting from particular impairments or adaptations to impairments are identified.

Keywords: Stroke; Cerebrovascular disorders; Gait; Locomotion

1. Introduction

Only 23–37% of persons who have sustained a stroke are able to walk independently during the first week [12], but there is general agreement that 50–80% of survivors can walk unaided at 3 weeks or at discharge [3], and by 6 months this figure may be as high as 85% [2]. Clearly, retraining of walking is a major goal in a rehabilitation program for persons with stroke. Consequently, their disturbed gait has been the object of study throughout the decades of advancement in methods of gait analysis. The purpose of Part I of this review is to identify the pathological patterns that characterize the gait of persons who have sustained a stroke. The purpose of Part II is to review locomotor recovery after stroke and the physical therapy approaches employed.

The performance of gait is directed to the accomplishment of four related tasks: maintaining the balance of the heavy trunk, arms and head on two ball and socket joints; maintaining support of the limb segments during stance phase; clearing the floor with the swinging foot during swing phase; supplying enough energy to the body system with each stride to cause it to move forward, and, preferably, accomplishing this using energy conservation measures. The accomplishment of these seemingly simple tasks may present substantial challenges to an impaired movement system.

The pathophysiological basis of stroke is damage to motor cells and pathways of the central nervous system caused by haemorrhage or thrombus affecting the arterial supply of the brain, usually of one side, typically occurring with little or no warning. The two immediate impairments of most significance to gait performance are diminished strength, or the inability to generate voluntary muscle contractions of normal magnitude in any muscle groups, and inappropriately timed or inappropriately graded muscle activity. After a few weeks, there may also be two further impairments: spasticity and changes in the mechanical properties of the muscle causing abnormal extensibility of muscle groups [4]. Both active and passive restraints are involved in the clinical term 'tone', which is frequently used to describe the sense of increased resistance to lengthening of the muscles.

Decreased 'strength' refers to several measurable factors, and may be expressed as decreased ability to generate a moment of force about a joint, a deficiency

* Corresponding author. Tel.: +1 613 545 6102; fax: +1 613 545 6776; e-mail: OLNEYS@QUCDN.QUEENSU.CA.

in muscle power [5], or, when taken over time, as diminished work accomplished by the muscles. The electromyographic activity (EMG) of the affected side is generally lower than that of the unaffected side, and this factor is compounded by an unusually low force to EMG ratio [6], which results in overall decreased force or moment output. There may be several causes of the loss of strength [7] including: decreased capacity to activate the motor units, a reduced number of functioning motor units [8], and reduced motor unit firing rates [6]. Other factors that could explain the apparent decrease in strength involve the performance of the antagonist muscle groups. Although a study of the interrelationships between static measures of strength and measures of spasticity have not supported a major role for spasticity of an opposing muscle in limiting voluntary activation [9], the role of spasticity during dynamic activities has not been adequately assessed. Another factor that can explain the apparent decrease in strength of a muscle group is increased stiffness of the opposing group. In one study, structural changes in spastic ankle plantarflexor muscles accounted for the appearance of weak dorsiflexors, as excessive activation of the dorsiflexors was required to overcome the excessive passive resistance of the plantarflexors [4]. Active restraint by opposing muscles may also be implicated, and inappropriate co-contractions have been reported in the gait of subjects with hemiparesis [10,11].

2. Spatio-temporal characteristics

The average walking speed reported for subjects with stroke is lower than values for able-bodied subjects, with the values reflecting the severity of the stroke, the time since its occurrence and the age of the subjects. In 17 studies reporting spatio-temporal characteristics, the average speeds ranged from 0.23 m/s (S.D. = 0.11 m/s) [3] to 0.73 m/s (S.D. = 0.38 m/s) [12]. Consistent with speed decreases, both stride length and cadence are lower than values for able-bodied subjects. Nakamura and colleagues [13] reported the relationship between cadence and speed to be linear up to a speed of about 0.33 m/s and a cadence of about 90 steps/min, with further gains primarily attributable to increases in stride length.

Three differences in the proportions of stance and swing have been reported (Fig. 1). First, the stance phase of both the affected and unaffected sides is longer in duration and occupies a greater proportion of the full gait cycle in subjects with stroke than in the able-bodied walking at normal speeds. Second, the stance phase is both longer and occupies a greater proportion of the gait cycle on the unaffected side than on the affected side. However, if stroke data are compared to that of the able-bodied walking at similar speeds, the proportion of stance on the unaffected side varies little from the able-

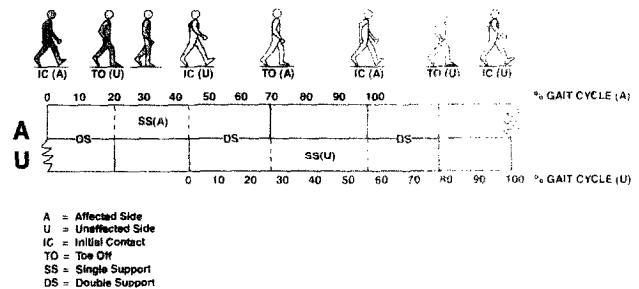## TEMPORAL EVENTS IN STROKE HEMIPARESIS

Fig. 1. Temporal events in persons with hemiparesis [5] following stroke.

bodied [14], and is significantly shorter on the affected side. The third difference is that a greater proportion of the gait cycle is spent in double support than that of able-bodied walking at normal speeds. However, when compared with able-bodied subjects walking at comparable speeds, the total double support of hemiparetic subjects is significantly lower (49% compared to 53%) [14] than that of the able-bodied. The total double support can be viewed in two parts. The portion of double support occurring during late stance of the affected foot (when the unaffected foot is spatially ahead of the affected limb) has been reported to be about 5% greater than the alternate, which at first seems counterintuitive as the stance phase is significantly longer on the unaffected side (e.g. 77% compared to 71% in one study [15]). However, foot contact of the unaffected limb occurs, on average, at 45% of the gait cycle of the affected limb, whereas the affected limb does not make contact until about 55% of the cycle of the unaffected side (Fig. 1).

3. Kinematic characteristics

Kinematic characteristics include linear and angular positions, their displacements and the time derivatives, notably the linear and angular velocities and accelerations. Of particular interest are the angles between body segments, referred to as joint angles, and their relationships to the events of the gait cycle. The positions of body segments in space and their excursions have received less attention. It is difficult to make direct quantitative comparisons across studies due to differences in reporting. While there are expected variations attributable to the data collection and analysis systems, the major differences are in selection of the variables to report. In most cases, the variables selected are those most relevant to the perceived clinical problem, and

these fall into two groups: the kinematics of the joint occurring at a particular instant of the gait cycle, (such as initial contact or mid stance), and the maximum or minimum values during a phase or sub-phase (such as the maximum knee flexion in swing).

The descriptions of variations in joint kinematic patterns are summarized by Burdett and colleagues [3], who assessed the gait of 19 subjects walking without and with orthoses. Their subjects' natural speed was low (0.27 m/s, S.D. 0.17 m/s). The authors concluded that subjects' major kinematic differences from the able-bodied were: (1) decreased hip flexion at initial contact, increased hip flexion at toe off, and decreased hip flexion during mid swing; (2) more knee flexion at initial contact and less knee flexion at toe off and mid swing; and (3) more ankle plantarflexion at initial contact and mid swing and less ankle plantarflexion at toe off.

Other authors have reported some or all of the following differences: decreased knee flexion and increased ankle plantarflexion at initial contact and during swing phase, with the magnitude of the deficit generally related to speed of walking [11,14,16–18]. In studies in which the kinematics of both sides have been reported, the maximum knee flexion during swing phase is in the order of 15–20 degrees greater on the unaffected side [5,11].

The characteristics of a group of 30 ambulatory subjects with stroke [5], have been divided into three groups

of 10 according to gait speed: fast (mean 0.63 m/s, S.D. 0.08 m/s), medium (mean 0.41 m/s, S.D. 0.08 m/s), and slow (mean 0.25 m/s, S.D. 0.05 m/s). Table 1 provides a data base of means and standard deviations of important kinematic and kinetic variables for the three groups and the whole study. The mean and standard deviations of the joint angle profiles are shown in Fig. 2a. The mean profiles for each group shown in Fig. 2b show a clear continuum with speed increases, though the large standard deviation evident in Fig. 2a is evidence of substantial inter-subject variability. In general, all phases of normal joint excursion patterns were present in the average profiles, and the timings and the amplitudes of the excursions deviated from the normal profiles in proportion to differences from normal speed. The average hip profiles did not show much difference in maximum hip flexion between speed groups, nor much difference from normal. In contrast, the hip extended an average of 10 degrees beyond neutral in late stance in the fastest group, about 2 degrees in the medium group, and was a few degrees short of neutral in the slowest group. The unaffected limb showed similar trends, though extension was greater than that of the affected side and the average always exceeded neutral.

Other authors have noted the serious reduction in maximum hip extension, when compared with the able-bodied. In one report, the average hip extension was 14 degrees less on the affected side in a group of subjects

Table 1
Joint range, power and work variables according to side affected and walking speed[a]

Variable	Fast speed		Medium speed		Slow speed		Total	
	Affected mean (S.D.)	Unaffected mean (S.D.)	Affected mean (S.D.)	Unaffected mean (S.D.)	Affected mean (S.D.)	Unaffected mean (S.D.)	Affected mean (S.D.)	Unaffected mean (S.D.)
Kinematic, degrees								
Max dorsi	13 (4)	16 (4)	11 (8)	17 (6)	11 (6)	18 (5)	12 (6)	17 (5)*
Max plant	−9 (3)	−10 (5)	−7 (8)	−7 (5)	−7 (8)	−4 (5)	−8 (7)	−7 (6)
Max knee stance	17 (4)	16 (5)	16 (10)	17 (9)	23 (11)	16 (5)	19 (9)	17 (7)
Max knee swing	46 (10)	60 (6)	38 (12)	59 (7)	34 (9)	54 (8)	39 (12)	58 (8)*
Max hip flex	18 (5)	22 (6)	22 (8)	25 (7)	22 (6)	23 (7)	21 (7)	23 (6)
Max hip ext	− 13 (6)	−10 (6)	−4 (11)	−9 (6)	2 (8)	−5 (10)	−5 (10)	−8 (7)
Power, W/kg × 10⁻²								
Max ankle	101 (43)	183 (104)	61 (47)	105 (47)	16 (9)	80 (35)	60 (51)	125 (82)*
Max knee	24 (14)	20 (9)	17 (12)	24 (11)	16 (10)	13 (6)	19 (13)	19 (10)
Max hip	66 (37)	65 (37)	29 (20)	46 (13)	18 (10)	30 (10)	38 (32)	48 (27)
Work, J/kg × 10⁻²								
Positive ankle	13 (5)	25 (11)	11 (5)	16 (6)	4 (3)	13 (5)	9 (6)	18 (9)*
Negative ankle	11 (4)	14 (4)	9 (5)	12 (6)	7 (5)	13 (6)	9 (5)	13 (5)*
Positive knee	5 (3)	4 (2)	4 (3)	5 (3)	5 (3)	4 (2)	5 (3)	5 (2)
Negative knee	17 (9)	26 (13)	10 (7)	19 (9)	6 (4)	19 (9)	11 (8)	21 (11)*
Positive hip	14 (4)	17 (9)	8 (3)	17 (9)	8 (6)	10 (3)	10 (6)	15 (8)*
Negative hip	9 (6)	7 (5)	6 (5)	8 (5)	2 (1)	9 (5)	6 (5)	8 (5)*
Total positive	32 (8)	46 (14)	23 (7)	38 (12)	18 (8)	28 (6)	25 (10)	38 (14)*
Total negative	38 (15)	48 (18)	24 (9)	40 (13)	14 (8)	40 (12)	25 (15)	43 (15)*

[a]Student t-test for total group.
*P < 0.05, corrected for number of comparisons.

a

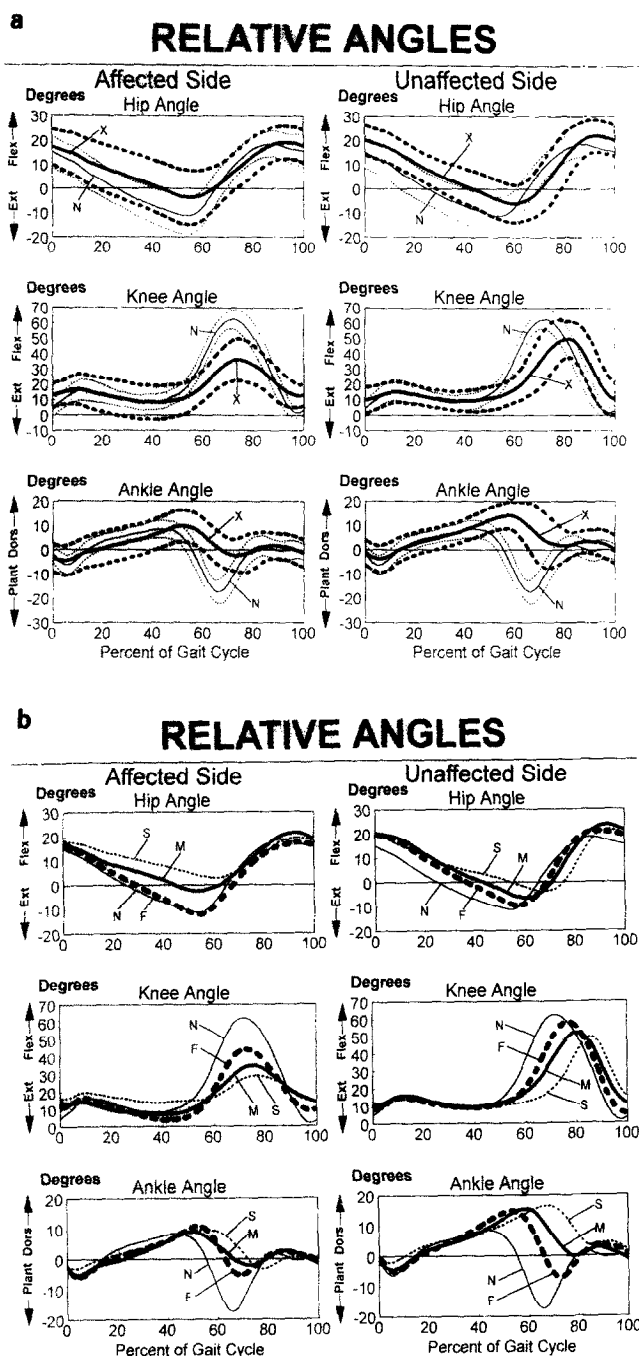## RELATIVE ANGLES

b

RELATIVE ANGLES

Fig. 2. (a) Mean and standard deviation of relative joint angles for 30 subjects with hemiparesis [5] (X) shown with mean and standard deviation of profiles of normal subjects (N) walking at slow speed, from Winter [25]. (b) The mean profiles of relative joint angles for slowest (S) subjects with hemiparesis (mean 0.25 m/s, S.D. 0.05 m/s), medium speed (M) subjects (mean 0.41, S.D. 0.08 m/s) and fastest (F) subjects (mean 0.63 m/s, S.D. 0.08 m/s) [5] shown with profile of normal subjects walking at slow speed (N), from Winter [25].

walking at a mean of 0.46 m/s, when compared with able-bodied subjects walking at comparable speeds [14]. Some studies report decreased hip flexion at initial contact [3] while others report none [5]. This difference is likely attributable to the slower speeds of walking reported in the former.

In the three-group study [5], the maximum knee flexion of the affected limb during swing phase ranged from an average of 46 degrees for the fast group and 38 degrees for the slow group, and that of the unaffected side was near 60 degrees for the fast and medium groups and only 54 degrees for the slowest. Not surprisingly, the knee flexion phases in stance showed the most disturbance both in pattern and amplitude. The slowest group made initial contact of the affected foot with the knee in 15 degrees of flexion, on average, and, after a flexion phase of only a few degrees, remained near 15 degrees until swing phase began. The fastest group made initial contact with the knee in about 10 degrees of flexion, performed a flexion phase of about 5 degrees and proceeded smoothly into extension in late stance, reaching a maximum of a few degrees less than neutral, which is within normal values for the able-bodied. The unaffected limb showed initial contact at 10 degrees of knee flexion for all groups, stance-phase flexion of about 5 degrees, and maximum extension about 10 degrees short of neutral.

The ankle of the affected limb of all groups after foot flat achieved several degrees of plantarflexion, on average, and thereafter increased smoothly to a maximum of 11–13 degrees of dorsiflexion in late stance. The average ankle profiles of the affected limb, including the plantarflexion excursion, varied a small amount across speeds, with the slowest group showing about 3 degrees less plantarflexion excursion during push off than the other two groups. On the unaffected side, the ankle made contact in a neutral position in all groups, and reached a maximum dorsiflexion angle of 16–18 degrees in all groups. The net effect was that the average plantarflexion excursion during push off varied a few degrees between each group, affording the faster groups considerably greater opportunity for power generation on the unaffected side.

Wagenaar and Beek [19] focused on the transverse rotations of the trunk and their relationship to speed. Phase differences between pelvic and thoracic components of trunk rotation and the total range of trunk rotation were almost linearly related to walking speed. However, the total range of pelvic and thoracic rotations were not speed dependent, suggesting that the phase differences in trunk rotation and not the total range of pelvic and thoracic rotation are responsible for the increase in total range of trunk rotation. From the velocity range of 0.75 m/s to 1 m/s onwards, pelvic rotation is the primary factor lengthening the stride.

4. Electromyographic characteristics

One of the earliest reports of EMG in stroke was by Hirschberg and Nathanson [20] in 1952, using skin electrodes over gluteus medius, adductor longus, semitendinosus, vastus lateralis, medial gastrocnemius, and

tibialis anterior. They reported the EMG levels to be decreased on the paretic side in all 11 subjects, with the patterns of activation of the hip muscles showing only small deviations from normal. The quadriceps and hamstrings showed the highest levels of activity which began at the usual time but were prolonged to the end of stance phase.

Peat and colleagues [21] provided the following summary of EMG activity. The average EMGs across subjects showed that the level of all muscle groups increased simultaneously as initial contact was made, and peaked together in mid stance phase. The average profiles of tibialis anterior, quadriceps and calf muscles showed a relatively sharp peak of activity in mid stance, but the period of high level activity of the medial hamstring extended from early to late stance. After toe off, the activity of all groups decreased as the limb prepared to leave the supporting surface. Pinzur and colleagues [22] noted consistent out-of-phase activity in rectus femoris. Hirschberg and Nathanson [20] also reported activation of gastrocnemius with initial contact but described the amplitude as low and uniform, not showing the increase that characterizes push off, while Pinzur and colleagues [22] reported consistent out-of-phase activity in both tibialis anterior and gastrocnemius-soleus.

There is unanimous agreement by researchers reporting on EMG that there is a great deal of inter-individual variation [11,21] and the descriptions given above should be regarded as itemizations of variations seen in the group of subjects rather than features generally seen in all subjects. This claim has been substantiated by Knutsson and Richards [11] who attempted to reveal inter-individual differences in disturbed motor control by classifying the subjects. This approach revealed inter-individual differences in the disturbed motor control that was not revealed when the EMG of compiled groups was examined for common features. In a group of 26 subjects with hemiparesis, all in the chronic recovery stage except one, Knutsson and Richards [11] described three types of disturbed motor control during gait. Subjects with a Type I disorder demonstrated hyperactive stretch reflexes leading to premature activation of the calf muscles in early and mid stance as the muscles were stretched following initial contact and the lower leg began pivoting over the fixed foot for weight acceptance and transfer. Early contraction of the calf muscles precluded the accomplishment of an effective push off by the plantarflexors. The activation patterns of subjects with a Type II disorder were characterized by a lack of activation during both shortening and lengthening contractions. Characterized by low levels of muscle activity in two or more major muscle groups, sometimes activity was present only during loading. In most cases, the distal groups were more disturbed than the proximal, but otherwise the disturbances were irregular. The ability to generate sufficient muscle force to
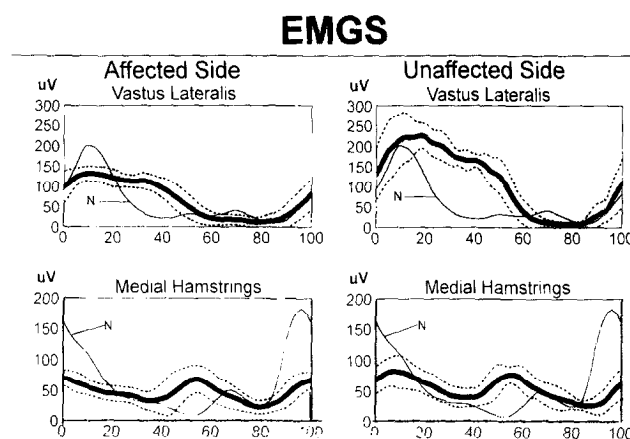
EMGS

Fig. 3. Mean and standard deviation of EMGs showing co-activation of hamstrings and knee extensors sustained into stance phase, classified as Type III-S by Shiavi et al. [23] shown with mean of normal profile (N) from Winter [25].

stabilize and to generate power is diminished in this group. Gait ability varied among these subjects, ranging from those who could walk only a few metres to others who could walk several kilometres, reflecting the variety and extent of deficiencies. There was no attempt to categorize further, though there appeared to be several sub-groups. Subjects with Type III disorder had neither responses to muscle stretch nor lack of muscle activation. Instead, they showed excessive and stereotyped coactivations of several muscle groups. Normal sequencing was disrupted: in some cases several muscle groups became co-activated during late swing and sustained into stance, but, in others, co-activation occurred only during stance phase. Shiavi and colleagues [23] classified those with activation predominantly during stance as Type III-S and during transitions as Type III-T. An example is shown in Fig. 3. Type IV pattern, showing combined components of the other three, was also reported.

Shiavi and colleagues [23] applied the Knutsson and Richards classification of both affected and unaffected extremities to the evaluation of change in locomotor control with recovery, evaluating 12 subjects early (1–10 weeks) and late (6–24 months) post-stroke. In the early phase, all disturbed types were observed on the affected side but two had normal profiles. Interestingly, however, when reevaluated later, the classification changed for all subjects with disturbed patterns. Abnormal patterns were also seen on the unaffected side, but it was not possible to determine whether they were due to neurological insult or to biomechanical compensation.

Few authors have reported on EMG of the unaffected limb. Wortis and colleagues [24] in 1952, reporting on the results of 19 subjects, expressed surprise that in many instances the electromyograms looked more atypical on the unaffected side than the affected, at-

tributing this appearance to the fact that the amplitudes were higher in the extensor muscles while many of the abnormalities present in the affected paretic extremity were also present. They noted particularly that the activity of the quadriceps muscle was prolonged into the second half of the stance phase, and that, frequently, there were bursts of activity of the semitendinosus that was uncommon in the paretic leg. In addition, although the patterns of the gastrocnemius muscles were higher than those of the affected limb, they remained abnormal, showing even contraction throughout the stance phase.

5. Kinetic characteristics

Because kinetic variables are the cause of the kinematic and spatio-temporal outcomes of the walking subject, kinetic information is particularly satisfying and useful in understanding and interpreting the characteristics of stroke gait. Unfortunately, there have been very few studies offering full link segment kinetic analyses.

5.1. Moments

The research and clinical community has failed to agree on the conventions for expressing joint moments. We will use the convention proposed to the International Society of Biomechanics and included in Winter [25], which expresses extensor and plantarflexor internal joint moments as positive. These internal moments are usually attributable to muscle activity. For example, during early stance phase the knee usually briefly flexes and then extends with an accompanying net knee extensor moment generated by the knee extensor muscles. This we refer to as a knee extensor moment.

The earliest large study of joint moments identified was reported in 1951 by Wortis and colleagues [24]. Using stroboscopic methods with force plate data, they reported the internal knee moment to be flexor throughout stance phase in 19 hemiplegic subjects. The results of Lehmann and colleagues [14] did not show a continuous internal flexor moment. However, comparisons of the average knee moments during the mid stance interval showed the mean internal extensor moment (i.e. the mean during the time the net moment was extensor) to be significantly smaller in the seven subjects with stroke than in able-bodied subjects walking at similar speeds (5.3 N.m compared to 8.6 N.m). Further, the mean internal knee flexor moment appeared to be much larger (17.3 N.m compared to 9.4 N.m), though values did not reach a level of statistical significance. The knee flexor moment may, in fact, have been attributable to hamstring activity, made necessary by the need for a large extensor moment at the hip to balance the forward lean noted by the authors. Although not pertinent in these cases, an exaggerated knee flexor moment in stance may be associated with a position of full exten-
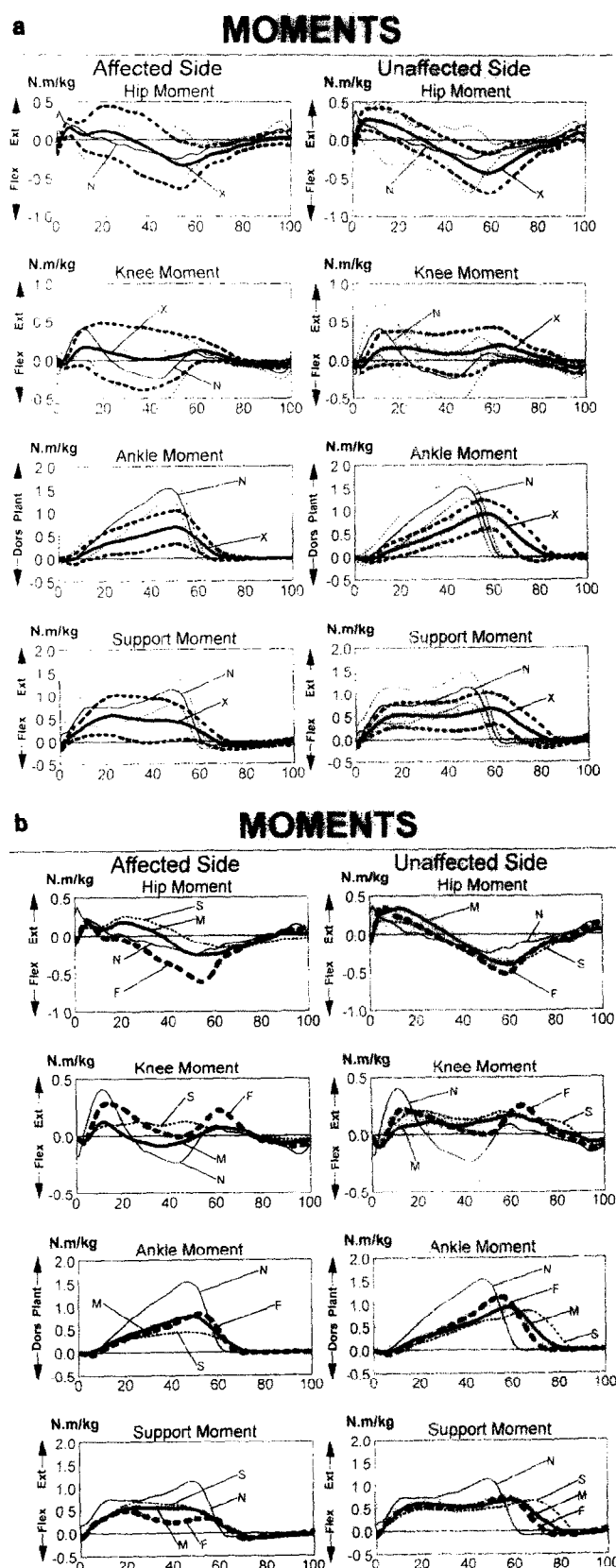

Fig. 4. (a) Mean and standard deviation of joint moments of 30 subjects with hemiparesis (X) shown with mean and standard deviation of profiles of normal subjects (N) walking at slow speed from Winter [25]. (b) The mean joint moment profiles for slowest (S) subjects with hemiparesis (mean 0.25 m/s, S.D. 0.05 m/s), medium speed (M) subjects (mean 0.41, S.D. 0.08 m/s) and fastest (F) subjects (mean 0.63 m/s, S.D. 0.08 m/s) shown with mean profiles of normal subjects walking at slow speed (N), from Winter [25].

sion, or hyperextension, and can be attributed to the resistance offered by the knee ligaments.

Fig. 4a,b shows data from the same subjects as those in Figs. 2 and 3. Note that the slowest group demonstrated, on average, exaggerated extensor moments at both hip and knee through most of stance on the affected side, whereas the fastest group showed exaggerated flexor profiles at the hip during stance and a knee moment that was normal in shape but biased towards extension throughout stance. Of note is the fact that the peak hip flexor moment of the affected side has been shown to be highly correlated with speed [15]. As will be seen when powers are discussed, below, the large pull-off powers at the hip are partly attributed to the presence of large flexor moments in late stance and early swing. Noteworthy also is the fact that the fastest group is willing to tolerate a very low support moment during single support on the affected side.

5.2. Ground reaction forces

A few authors have reported on ground reaction forces. Wortis and colleagues [24] described the vertical force curve in 19 subjects. In contrast to the double-peaked curve that is typical of the able-bodied, the vertical force curve was more variable and had an initial low peak. Carlsoo and colleagues [26] reported three patterns: one with a first peak during heel contact phase and the second during push off; a second pattern showed a continuous plateau, but no discernible peaks; and a third with a single peak in mid stance. More recently, researchers have used the vertical force curve as an indicator of progress in treatment using therapeutic electrical stimulation [27,28], and have suggested that trends towards the re-institution of higher forces and a double-peaked vertical force pattern be regarded as positive.

Rogers and associates have reported on the laterally-directed ground reaction forces in the frontal plane, in order to characterize the initiation of single limb stance [29]. During double support the able-bodied showed a simultaneous increase in medially-directed force on the lifting limb and decrease in medially-directed force on the stance limb in a proportion of about 4:1. In subjects with stroke, however, when the subjects transferred weight to the affected side, 86% of the resultant laterally-directed impulse was contributed from beneath the unaffected side. This resulted in a 6:1 lifting-to-stance contribution from beneath the unaffected side. In contrast, when subjects transferred towards the unaffected side, 70% of the contribution was from under the stance limb, resulting in a 1:2.3 lifting-to-stance proportion. In some instances, there was a lack of synchrony and reversals in the usual directions of the lateral forces, suggesting disturbances in inter-limb co-ordination.

5.3. Mechanical energy

The mechanical energy of walking has been calculated using a two-dimensional, seven-segment mechanical energy analysis. The instantaneous energy of one segment is calculated as:

$$E_i = m_i g h_i = 1/2\ m_i v_i^2 + 1/2\ I_i w_i^2$$

where m_i is the mass of the segment, assuming it to be a certain proportion of body mass; g is the gravitational constant; h_i is the height of its centre of mass above the reference point of the floor surface; v_i is the linear velocity of the centre of mass; I_i is the rotational moment of inertia about the centre of mass; and w_i is the angular velocity of the segment. When an increase in potential energy is simultaneous with a decrease in kinetic energy, or vice versa, an energy-conserving exchange is assumed to have occurred, and is known as a within-segment exchange. There are two mechanisms of changing the mechanical energy level of a segment. The first mechanism is passive, calculated as the product of the joint reaction force and the linear velocity of the joint. The second mechanism acts via the tendo-muscular interface as a result of muscle action. Calculated as the product of the moment at the joint and the angular velocity of the segment, energy is generated to the segment if the polarities are the same (concentric contraction) and absorbed if they are different (eccentric contraction). In addition, when the segments are moving in the same direction, passive transfer occurs to the segment whose moment and angular velocity are in the same direction, and the rate of transfer is the lesser of the two power components at the joint. The total energy of the body at any instant in time is the algebraic sum of the energy of each of its segments.

The total energy cost of a single stride is the absolute sum of the differences between energy levels at each instant in time over the full cycle; that is, the total cost is the sum of the rises and falls. In the able-bodied, the total body cost has been reported to be 1.1 J/kg·m, with energy conservation to be an average of about 70%. There are three reports of energy studies of stroke subjects [30–32], a total of only 15 subjects. These studies reported energy conservation from 22 to 62%. In the largest study, the costs per unit distance ranged from 0.8 J/kg·m to 3.9 J/kg·m, though most were over the normal 1.1 J/kg·m.

Three disturbances causing these reductions in conservation were identified [31]. The first disturbance was characterized by the absence of the mirroring pattern of potential and kinetic energy of the large head, arms and trunk segment, indicating that little exchange between kinetic and potential energy was occurring. In the second pattern, there was some mirroring of the energy patterns of the head, arms and trunk segment, but the magnitude of the kinetic energy was so low that little exchange could occur. The third pattern was termed the hip-hiking pattern (Fig. 5) because it was characterized by a single large rise and fall of the potential energy pat-
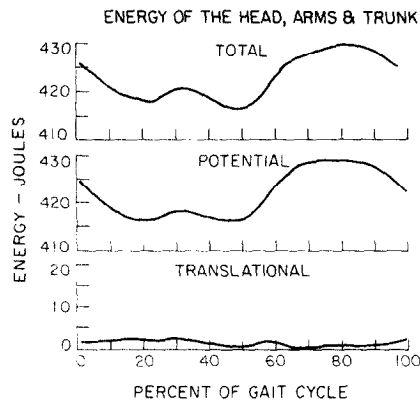
ENERGY OF THE HEAD, ARMS & TRUNK

Fig. 5. Pattern of mechanical energy of head, arms and trunk segments during one gait cycle showing hip-hiking patterns. Note large potential energy changes and very low kinetic energy throughout, offering virtually no possibility for exchange [31].

tern that corresponded to the time of the swinging of the affected leg, which was accomplished with excessive rise of the trunk. This was caused by the fact that at initial contact the potential energy of the body is at its lowest point while the kinetic energy, a reflection of the velocity, is at its highest. During the period until mid stance, the body loses speed while gaining height, exchanging kinetic energy for potential energy.

Another approach to using the centre of gravity information to assess gait was reported by Iida and Yamamuro [33]. Double integration of the total floor reaction force yielded the acceleration of the centre of mass when divided by body force — a method which the authors acknowledge is limited by the fact that it assumes the trajectory of the centre of mass of the whole body represents the energy changes of the arms and lower limbs which move in opposite directions relative to the centre of mass. The authors compared the potential and kinetic energies during gait with the total energy (derived from the algebraic sum of potential and kinetic curve). In able-bodied subjects, the potential energy costs were greatest, the kinetic energy costs about 15–20% less, and total costs at least 30% less than the potential energy costs. In contrast, in four persons with stroke, the kinetic energy costs were reduced by well over 50%, but total costs were rarely more than 20% less than potential costs. Moreover, the mean work per unit distance ranged from about 0.58 J/kg·m to 1.26 J/kg·m in contrast to 0.54–0.62 J/kg·m, the range expressed for the able-bodied. These values are predictably lower due to the error in their technique.

5.4. Joint power

The mechanical powers have also been reported for both the affected and unaffected side of the body for three groups of 10 hemiparetic subjects grouped accord-ing to their natural walking speeds: slow (mean 0.25 m/s, S.D. 0.05 m/s), medium (mean 0.41 m/s, S.D. 0.08 m/s), and fast (mean 0.63 m/s, S.D. 0.08 m/s) [5]. The mean and standard deviations of profiles of the whole group are shown with normal values from Winter [25] in Fig. 6a; individual group means appear in Fig. 6b. A brief background to these analyses follows. The body can move only when work is done on it. In walking, the energy level of the body returns to approximately the same level at the same point in the gait cycle for each stride, and successive bursts of positive work and negative work occur in known patterns [35]. Positive work is performed by concentric (shortening) contractions while work done against gravity or other external forces is called negative work and it is performed by eccentric (lengthening) contractions. Both forms of work require metabolic energy.

The work that is performed by a muscle group that crosses a particular joint during one stride is calculated by integration of the power curve over time. The power P (watts) of a muscle group at joint j at a given instant in time is:

$$P_j = M_j \cdot \omega_j$$

where M_j is the net moment of force at joint j (N-m), and ω_j is the joint angular velocity (radians/s) [36].

Mechanical work and power in normal walking has been investigated [35–38]. In normal slow walking the single largest burst of positive work is performed by the ankle plantarflexors during push off and early swing (A2). The knee has three phases attributable to knee extensor activity: one of eccentric work at weight acceptance, designated as K1; a very small concentric period during mid stance (K2); and a large eccentric phase at push off and early swing (K3). A fourth phase (K4) occurs at the end of swing and is caused by the knee flexors acting eccentrically. The hip shows a small amount of concentric work by the extensors in early stance (H1), and low-level eccentric flexor work during much of the rest of stance (H2). A substantial period of concentric flexor work occurs in late stance and early swing (H3) and may be termed pull off.

As shown in Fig. 6a,b, the positive A2 burst by the ankle plantarflexors during push off was a major contributor to the positive work accomplished on both sides with one exception: it is barely evident on the affected side at the slowest speed. The muscles of the knee, like their normal counterparts, acted primarily eccentrically, with K3 at the fast speed on the unaffected side shown to be even larger than normal. A small burst of positive work by the knee extensors (K2) occurred in early stance on the affected side for the fastest speed group. At the hip the pull off phase (H3) was evident in all plots though the level was very low on the affected side of the slowest group. In contrast, the mean of the H1 phase,

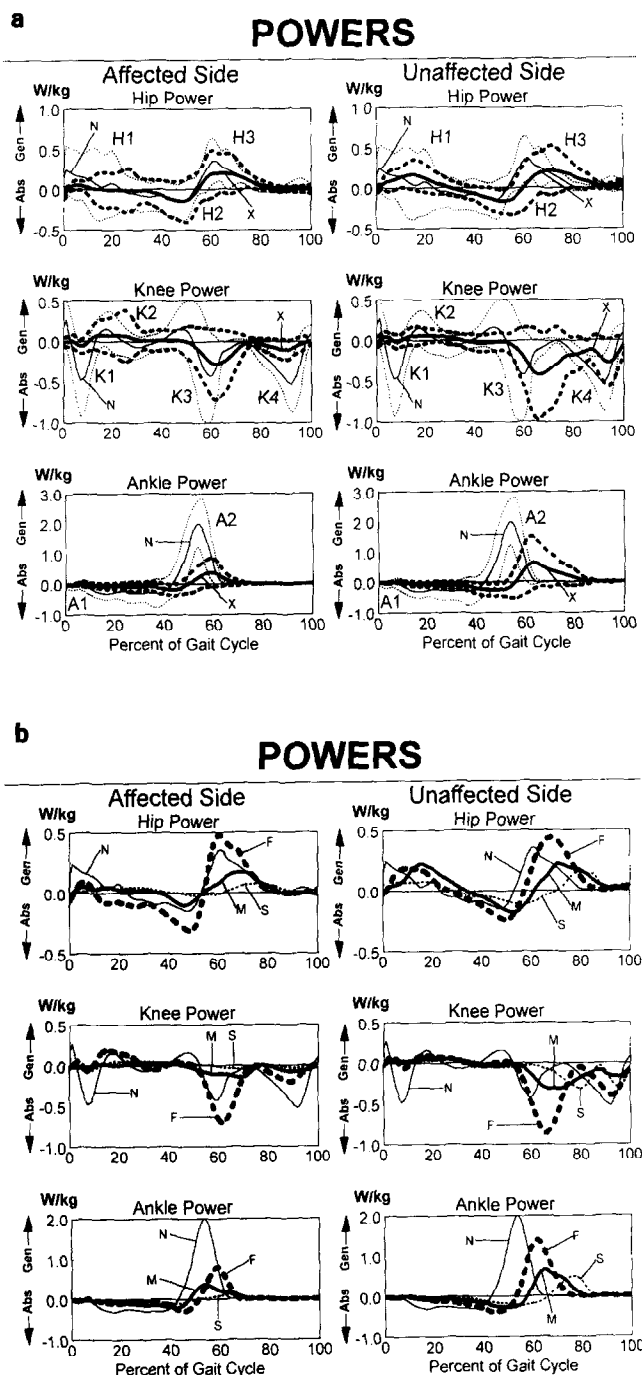**a**

POWERS

b

POWERS

Fig. 6. (a) Mean and standard deviation of joint powers of 30 subjects with hemiparesis [5] (X) shown with mean and standard deviation of profiles of normal subjects (N) walking at slow speed from Winter [25]. (b) The mean joint power profiles for slowest (S) subjects with hemiparesis (mean 0.25 m/s, S.D. 0.05 m/s), medium speed (M) subjects (mean 0.41, S.D. 0.08 m/s) and fastest (F) subjects (mean 0.63 m/s, S.D. 0.08 m/s) [5] shown with mean profiles of normal subjects walking at slow speed (N) from Winter [25].

caused by hip extension in early stance, was positive only on the unaffected side.

The speed of walking has been shown to be related to work and power [5]. The peak ankle and hip power was

less for both affected and unaffected sides with slower speeds, but the trend for knee power was less clear in this regard. Work values for the affected side tended to decrease with declining speed, the exception being positive knee and hip work which were about the same at slow and medium speeds. On the unaffected side, the effects of speed were less consistent, though the positive work of the ankle decreased with declining speed. Overall, the unaffected side performed a greater proportion of the work at all speeds, roughly in a 60:40 ratio. This resulted from large discrepancies between the work of the affected and unaffected ankle and moderate differences at the hip. There were no differences between contributions of the knee muscles.

The relationships have been reported between speed of walking and single gait variables as well as between speed of walking and groups of variables which, when taken together, offer advantages [15]. Variables that correlated significantly with self-selected speed included the maximum hip extension angle and the maximum hip flexor moment on the affected side, and the maximum ankle and hip powers on both sides. For the affected side, the faster walker would show a large hip flexor moment at the end of stance phase, a large range between maximum dorsiflexion and plantarflexion ankle moments, and a small range between maximum knee extensor and knee flexor moments. Based on data from the unaffected side, a fast walker should have a short period of weight bearing, substantial ankle plantarflexor power at push off and a strong pull off by the hip flexors. This approach explores factors that are important in attaining high levels of gait performance. Although the specific results require further empirical studies before serious application in gait training, some insight into the specific factors that may be important in attaining high levels of gait performance is offered.

6. Asymmetry

The achievement of symmetry in gait has been assumed to be an important objective in the rehabilitation of persons with hemiparesis [39–41] and, indeed, has often been used to measure success of treatment. Wall, for example, has said, 'An ideal objective in the functional rehabilitation of hemiplegia following stroke is the reduction of the asymmetrical nature of movement patterns' [39]. However, it is difficult to offer theoretical defense for this position: one would not expect a bilateral machine with motors of unequal power on each of its sides to produce an optimal solution by using equal outputs from those motors. Empirical support for this position is given by Winter and colleagues [42], who have identified many examples of compensations for deficient motor patterns in which values of gait variables are clearly different on each side of the body.

In an effort to determine whether symmetry in gait

was related to higher walking speeds in rehabilitated subjects, the symmetry properties of 34 gait variables in 31 subjects with hemiparesis were examined [43]. A variable was defined as symmetric if subjects with the highest speeds had equal values on both sides of the body. If the highest speeds were achieved when the values of the affected side significantly exceeded the unaffected, the variable was called asymmetric. Correlations of the differences and absolute differences between the variables from the two sides with speed were used to detect symmetric and asymmetric variables, as well as variables exhibiting other forms of symmetry. Only seven variables were detected that were not indifferent to symmetry, and only one of these showed symmetry itself. Four asymmetric variables, and three variables with other symmetry properties were identified. In summary, there was only weak evidence that symmetry played any role at all in promoting speed in the study group; in fact, asymmetric variables seemed more important. At present the only clear argument for making temporal, spatial or kinematic symmetry during walking a goal of treatment in chronic stroke is an aesthetic one, and there is no defensible argument for targeting kinetic symmetry as a goal of treatment for this group. This directive is probably not appropriate for persons in the early stages of treatment when one hopes to attain a high level of performance of the affected side as recovery occurs, without the introduction of compensations. Also, encouragement of high levels of performance by the affected side is important at all stages of treatment. In view of the needs of rehabilitated subjects, however, it is time the unquestioned goal of gait symmetry for all persons with hemiparesis be laid to rest.

7. Impairment and adaptations

If a gait variable is substantially different from a value for the able-bodied, it is usually referred to as a gait deviation. Although this term has traditionally included only those variations that can be seen by the trained observer, namely spatio-temporal and kinematic deviations, kinetic deviations should also be included. The term deviation should not be interpreted negatively, as it is, in fact, advantageous that the neuromuscular system has many different ways of fulfilling the kinematic and kinetic requirements for forward progression, balance control and maintenance of support during walking. A gait deviation may be the direct effect of an impairment, in which case it appears below with the designation of *impairment*. On the other hand, the gait deviation may occur to mitigate the effects of an impairment, in which case the deviation appears below under the designation of *adaptation*. Certain adaptations provide compensations for impairments and are of the same type as the deficit. Adaptations that are also compensations are specifically noted in the text below. Gait devia-

tions that commonly occur in spatio-temporal, kinematic, EMG and kinetic categories are identified below, with causes attributed to the possible impairments and adaptations. Note that any given deviation may be caused by one or more of the impairments or adaptations.

7.1. Spatio-temporal deviations

7.1.1. Slow speed of walking; long stance phase durations and proportions, both sides; long double support period

Impairment: diminished strength: low levels of power generation by many muscle groups.

Adaptation: to impaired balance: reduced push-off power to reduce unbalancing effect of piston-like drive of push-off at the ankle on the unaffected side.

7.1.2. Early foot contact by unaffected side

Impairment: diminished strength: inability to generate sufficient hip flexor moment on affected side to reverse the thigh extension and balance the trunk while unaffected limb approaches initial contact; inability to generate sufficient push-off power by affected side to produce longer duration of swing phase of unaffected side.

Adaptation: to impaired balance: fear of lateral instability: need for two-foot contact to control medio-lateral stability.

7.1.3. Comments on particular spatio-temporal deviations

Decreased strength or impaired balance results in slow forward progression. The failure to achieve adequate speeds, in turn, results in loss of opportunity for energy-conserving exchanges between potential and kinetic energy of the upper body [31]. It also results in higher energy costs per unit of distance covered.

Although extended periods of double support may be functional for balance, they are almost undoubtedly detrimental to energy conservation. The period of double support is characterized by simultaneous energy generation by the pushing limb and absorption by the weight-accepting limb, which is an inefficient mechanism if prolonged.

7.2. Kinematic deviations

7.2.1. Limited ankle dorsiflexion at initial contact and during stance

Impairments: diminished strength: inadequate activation of ankle dorsiflexors; coactivation of ankle plantarflexors. Changes in mechanical properties (increased stiffness [4]) of ankle plantarflexors, making ankle dorsiflexion activation inadequate to clear the floor in swing.

Adaptation: to impaired balance: provision of stability of a flat foot landing at initial contact.

7.2.2. Knee hyperextension in stance phase, affected side

Impairments: spasticity of ankle plantarflexors. Changes in mechanical properties (increased stiffness [4]) of ankle plantarflexors.

Adaptation: to diminished strength: seeking of position of stability with knee in a position of extension, due to diminished moment-generating capabilities by combination of hip extensors, knee extensors, and ankle plantarflexors.

7.2.3. Diminished knee flexion in swing phase, affected side

Impairments: diminished strength: diminished power generation by affected ankle plantarflexors and/or hip flexors. Spasticity of knee extensors.

Adaptation: to impaired balance: desire to keep foot close to the ground during entire swing phase in preparation for next initial contact.

7.2.4. Hip hiking or circumduction in swing phase, affected side

Adaptations: same as for deviation of diminished knee flexion, swing, affected side. Diminished strength: inadequate strength of ankle dorsiflexors, with subsequent difficulty with floor clearance; coactivation of ankle plantarflexors. Changes in mechanical properties (increased stiffness [4]) of ankle plantarflexors, making ankle dorsiflexion activation inadequate to clear the floor.

7.2.5. Excessive knee flexion in stance, affected side

Impairments: diminished strength: diminished support moment generation at appropriate joint angles by combination of knee extensors, ankle plantarflexors, hip extensors; knee may flex excessively to point where muscle length effect makes it more effective.

7.2.6. Comments on particular kinematic deviations

Many authors have reported on the gait adaptation of knee hyperextension that is apparent during mid stance and frequently continues into late stance [28,44,45]. The actual knee position reported varies with the anatomy of the subject's normal knee excursion, and indeed the technical methods used for data collection and analysis, and the definition of the neutral position. Thus the position of maximum extension of the knee may range from about zero to several degrees of hyperextension. When observed, the knee appears to be abnormally extended in stance, probably because this position of full knee extension is not reached in the gait of the able-bodied [46]. The pattern of knee hyperextension in late stance precludes the normal knee flexion-extension pattern during early stance, though frequently the subject makes initial contact with the knee in flexion and proceeds rapidly to full knee extension or hyperextension.

Although knee hyperextension is a gait adaptation, it has a number of undesirable biomechanical effects especially when continued into late stance. It usually precludes an effective push off [15]. In this case, there is failure to flex the knee, and the subject must either carry the limb through swing in an extended position, or resort to circumduction [14] or hip hiking. Either of these manoeuvres virtually prevents generation of energy by both the ankle plantarflexors and the hip flexors. Because generation by the unaffected side is usually in the order of 40% of the total cost of walking [5], this is a serious loss.

7.3. EMG and kinetic deviations

7.3.1. Coactivation of knee muscles during stance; continued activation of hamstrings in stance phase of the affected side

Impairment: inappropriately timed/graded muscle contraction: an impairment of motor control.

Adaptations: to diminished strength of affected side: attempt to generate extensor power in stance (H1) to compensate for a generally low power generation of affected side. To diminished ankle plantarflexor strength, affected side: necessity of providing adequate extensor support moment in compensation for diminished ankle plantarflexor flexor moment in late stance. To postural forward lean: necessity to generate sufficient hip extensor moment to balance forward-leaning body.

7.3.2. Large bursts of hip flexor activity at pull off, power burst H3, either side of body

Adaptation: to diminished strength ankle plantarflexors: attempt to provide additional power for work of walking; compensation for low ankle plantarflexor power.

7.3.3. Large bursts of ankle plantarflexor activity, power burst A2, unaffected side of body

Adaptation: to diminished strength, affected side: attempt to provide additional power for work of walking; compensation for low power generation, affected side.

7.3.4. Excessive energy costs per unit distance walked

Impairments: diminished strength: insufficient to cause velocity adequate for good exchange of kinetic-potential energy. Inappropriately timed/graded muscle contraction: providing irregular bursts of energy not timed for optimal energy exchange.

7.3.5. Comments on particular EMG and kinetic deviations

The need to provide support for the limbs and trunk [47] of the affected side through stance despite very reduced moments of the ankle plantarflexors may account for the extended activity of hip extensors and knee

extensors usually reported in EMG studies [20]. In a kinetic example showing compensation to secure support during stance [42], an unusually large flexor moment at the hip was compensated by an unusually large extensor moment at the knee, resulting in a good support moment profile.

8. Directions for further research

The serious study of gait in persons with stroke has hardly begun. Myriad questions remain unaddressed, most of which require knowledge of kinetics. One might question why there are so few reports on the kinetics of stroke gait, given the obvious potential for increasing understanding. Gait analysis historically has been regarded with ambivalence to the degree that it cannot offer unequivocal solutions to clinical problems. Gait analysis has received the most enthusiastic support when it can guide the selection of dramatic intervention such as surgery [48]. To date, too few studies of gait analysis in persons with stroke have been conducted to identify any but the most obvious of the pathological patterns. The following directions appear to offer fertile ground for advancing this knowledge:

- *Use gait analysis to determine optimal treatment protocols.* Targeting specific biomechanical treatment objectives and subsequently assessing the effects would increase knowledge of the behaviour and responsiveness of the system.
- *Relate outcome measures of treatment, such as gait velocity, to specific kinetic changes.* Failure to do so impairs our ability to target specific kinetic variables in treatment and to use these variables to understand the nature of change in gait.
- *Use large group studies to identify clusters of biomechanical features associated with good performance.* Unfortunately, little is known about the different strategies that people with specific characteristics use to solve their movement problems. These may be developed into principles of treatment for certain subject groups.
- *Develop measures of mechanical work and efficiency for use in meaningful ways.* Energy is a paramount concern, and our tools are seriously deficient.

Acknowledgements

References

[1] Jorgensen H S, Nakayama H, Raaschou H O, Olsen T S. Recovery of walking function in stroke patients: The Copenhagen Study. *Arch Phys Med Rehabil* 1995; 76: 27–32.

[2] Wade D T, Wood V A, Heller A, Maggs J, Hewer R L. Walking after stroke: measurement and recovery over the first 3 months. *Scand J Rehabil Med* 1987; 19: 25–30.

[3] Burdett R G, Borello-France D, Blatchly C, Poptter C. Gait comparison of subjects with hemiplegia walking unbraced, with ankle-foot orthosis, and with Air-Stirrup brace. *Phys Ther* 1988; 68: 1197–1203.

[4] Dietz V, Berger W. Interlimb coordination of posture in patients with spastic hemiparesis. *Brain* 1984; 107: 965–978.

[5] Olney S J, Griffin M P, Monga T N, McBride I D. Work and power in gait of stroke patients. *Arch Phys Med Rehabil* 1991; 72: 309–314.

[6] Tang A, Rymer W Z. Abnormal force-EMG relations in paretic limbs of hemiparetic human subjects. *J Neurol Neurosurg Psychiatry* 1981; 44: 690–698.

[7] Bourbonnais D, Vanden Noven S. Weakness in patients with hemiparesis. *Am J Occup Ther* 1989; 43: 313–319.

[8] McComas A J, Sica R E P, Upton A R M, Aguilera N. Functional changes in motoneurones of hemiparetic patients. *J Neurol Neurosurg Psychiatry* 1973; 36: 183–193.

[9] Bohannon R W, Larkin P A, Smith M B, Horton M G. Relationship between static muscle strength deficits and spasticity in stroke patients with hemiparesis. *Phys Ther* 1987; 67: 1068–1071.

[10] Conrad B, Benecke R, Meick H M. Gait disturbances in paraspastic patients. In: Delwaide P J and Young R R, eds. *Clinical Neurophysiology in Spasticity*. Amsterdam: Elsevier, 1985: 155–174.

[11] Knutsson E, Richards C. Different types of disturbed motor control in gait of hemiparetic patients. *Brain* 1979; 102: 405–430.

[12] von Schroeder H P, Coutts R D, Lyden P D, Billings E Jr. Gait parameters following stroke: a practical assessment. *J Rehabil Res Dev* 1995; 32: 25–31.

[13] Nakamura R, Handa T, Watanabe S, Morohashi T. Walking cycle after stroke. *Tohoku J Exp Med* 1988; 154: 241–244.

[14] Lehmann J F, Condon S M, Price R, deLateur B J. Gait abnormalities in hemiplegia: their correction by ankle-foot orthoses. *Arch Phys Med Rehabil* 1987; 68: 763–771.

[15] Olney S J, Griffin M P, McBride I D. Temporal, kinematic, and kinetic variables related to gait speed in subjects with hemiplegia: a regression approach. *Phys Ther* 1994; 74: 872–885.

[16] Cozean C D, Pease W S, Hubbell S L. Biofeedback and functional electric stimulation in stroke rehabilitation. *Arch Phys Med Rehabil* 1988; 69: 401–405.

[17] Intiso D, Santilli V, Grasso M G, Rossi R, Caruso I. Rehabilitation of walking with electromyographic biofeedback in foot-drop after stroke. *Stroke* 1994; 25: 1189–1192.

[18] Knutsson E. Gait control in hemiparesis. *Scand J Rehabil Med* 1981; 13: 101–108.

[19] Wagenaar R C, Beek W J. Hemiplegic gait: a kinematic analysis using walking speed as a basis. *J Biomech* 1992; 25: 1007–1015.

[20] Hirschberg G G, Nathanson M. Electromyographic recording of muscular activity in normal and spastic gaits. *Arch Phys Med Rehabil* 1952; 33: 217–225.

[21] Peat M, Dubo HIC, Winter DA, Quanbury AO, Steinke T, Grahame, MC. Electromyographic temporal analysis of gait:

hemiplegic locomotion. *Arch Phys Med Rehabil* 1976; 57:421–425.

[22] Pinzur M S, Sherman R, Dimonte-Levine P, Trimble J. Gait changes in adult onset hemiplegia. *Am J Phys Med* 1987; 66: 228–237.

[23] Shiavi R, Bugle H J, Limbird T. Electromyographic gait assessment, part 2: Preliminary assessment of hemiparetic synergy patterns. *J Rehabil Res Dev* 1987; 24: 24–30.

[24] Wortis S B, Marks M, Hirschberg G G, Nathanson M. Gait analysis in hemiplegia. *Trans Am Neurol Assoc* 1951; 76: 181–183.

[25] Winter D A. *The Biomechanics and Motor Control of Human Gait: Normal, Elderly and Pathological.* 2nd edn. Waterloo: University of Waterloo Press, 1991.

[26] Carlsoo S, Dahllof A-G, Holm J. Kinetic analysis of gait in patients with hemiparesis and in patients with intermittent claudication. *Scand J Rehabil Med* 1974; 6: 166.

[27] Bogataj U, Gros N, Malezic M, Kelih B, Kljajic M, Acimovic R. Restoration of gait during two to three weeks of therapy with multichannel electrical stimulation. *Phys Ther* 1989; 69: 319–327.

[28] Malezic M, Kljajic M, Acimovic-Janezic R, Gros N, Krajnik J, Stanic U. Therapeutic effects of multisite electric stimulation of gait in motor-disabled patients. *Arch Phys Med Rehabil* 1987; 68: 553–560.

[29] Rogers M, Hedman L D, Pai Y C. Kinetic analysis of dynamic transitions in stance support accompanying voluntary leg flexion movements in hemiparetic adults. *Arch Phys Med Rehabil* 1993; 74: 19–25.

[30] Lowery L L. Mechanical energy analysis of hemiplegic gait: monitoring and diagnosis. *Proc Can Soc Biomech* 1980: 56–57 [Abstr].

[31] Olney S J, Monga T N, Costigan P A. Mechanical energy of walking of stroke patients. *Arch Phys Med Rehabil* 1986; 67: 92–98.

[32] Winter D A. Energy assessments in pathological gait. *Physiother Can* 1978; 30: 183–191.

[33] Iida H, Yamamuro T. Kinetic analysis of the center of gravity of the human body in normal and pathological gaits. *J Biomech* 1987; 20: 987–995.

[34] Winter D A. A new definition of mechanical work done in human movement. *J Appl Physiol* 1979; 46: 79–83.

[35] Winter D A. *The Biomechanics and Motor Control of Human Gait.* Waterloo: University of Waterloo Press, 1987.

[36] Winter D A. *Biomechanics and Motor Control of Human Movement.* 2nd edn. New York: Wiley, 1990.

[37] Winter D A. Biomechanical motor patterns in normal walking. *J Motor Behav* 1983; 15: 302–330.

[38] Winter D A, Eng P. Energy generation and absorption at the ankle and knee during fast, natural, and slow cadences. *Clin Orthop Relat Res* 1983; 175: 147–154.

[39] Wall J A, Turnbull G I. Gait asymmetries in residual hemiplegia. *Arch Phys Med Rehabil* 1986; 67: 550–553.

[40] Hesse S A, Jahnke M T, Schreiner C, Mauritz K-H. Gait symmetry and functional walking performance in hemiparetic patients prior to and after a 4-week rehabilitation program. *Gait Posture* 1993; 1: 166–171.

[41] Dewar M E, Judge G. Temporal asymmetry as a gait quality indicator. *Med Biol Eng Comput* 1980; 18: 689–693.

[42] Winter D A, Olney S J, Conrad J, White S C, Ounpuu S, Gage J R. Adaptability of motor patterns in pathological gait. In: Winters J M and Woo S L Y, eds. *Multiple Muscle Systems: Biomechanics and Movement Organization.* New York: Springer, 1990: 680–693.

[43] Griffin M P, Olney S J, McBride I D. The role of symmetry in gait performance of stroke subjects with hemiplegia. *Gait Posture* 1995; 3: 132–142.

[44] Morris M E, Matyas T A, Bach T M, Goldie P A. Electrogoniometric feedback: its effect on genu recurvatum in stroke. *Arch Phys Med Rehabil* 1992; 73: 1147–1154.

[45] Stanic U, Acimovic-Janezic R, Gros N, Trnkoczy A, Badj T, Kljajic M. Multichannel electrical stimulation for correction of hemiplegic gait. *Scand J Rehabil Med* 1978; 10: 75–92.

[46] Winter D A, Patla A E, Frank J S, Walt S E. Biomechanical walking pattern changes in the fit and healthy elderly. *Phys Ther* 1990; 70: 340–347.

[47] Winter D A. Overall principle of lower limb support during stance phase of gait. *J Biomech* 1980; 13: 923–927.

[48] Gage J R. Gait analysis for decision-making in cerebral palsy. *Bull Hosp Jt Dis Orthop Inst* 1983; XLIII(2): 147–163.