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Observation and analysis of hemiplegic gait: stance phase

People with hemiplegia resulting from cerebrovascular accident commonly demonstrate one or more deviations from the kinematics of normal gait. This paper presents a list of common kinematic deviations for which physiotherapists might look when making clinical observations of hemiplegic gait. A number of likely causes of those kinematic deviations are described, based on a review of the literature, biomechanical considerations and clinical observations. Particularly common and significant stance phase deviations are a decreased peak hip extension in late stance, increased or decreased peak lateral pelvic displacement, increased or decreased knee extension in early or mid stance and decreased plantarflexion at toe-off. The causes of these kinematic deviations lie in the inability to appropriately activate muscles and in the adaptive muscle shortening which commonly occurs following stroke.

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In keeping with contemporary views of motor control, the disordered gait commonly seen following hemiplegic stroke can be seen as an emergent motor behaviour (Shepherd and Carr 1991). In other words, hemiplegic gait evolves over time in a way that is determined by the effects of the neural lesion, secondary motor problems such as adaptive muscle shortening, and the interaction of these problems with the complex dynamics of the motor control system. The complexities of hemiplegic gait mean that the clinical processes of observation, analysis and intervention present a significant challenge to physiotherapists.

The intention of this paper is to provide a clinically orientated review of the mechanics of hemiplegic gait. The first aim is to identify those kinematic deviations from normal gait which commonly occur following stroke and which are of clinical significance. The important kinematic deviations which will be described here are based on the essential components of walking presented by Carr and Shepherd (1987) and the major determinants of walking described by Saunders et al (1953). Clinically significant deviations are those which would be expected to impair walking performance, for example, by decreasing walking speed.

A second, more important aim is to speculate about the likely causes of the kinematic deviations of hemiplegic gait. Physiotherapy intervention requires that the movement problems be identified in terms that are amenable to intervention, such as the inability to activate particular muscle groups, the over-activity of muscle groups and adaptive muscle shortening. The identification of probable causes of particular kinematic deviations is difficult, however, because the clinical processes of ascertaining movement problems are imperfect and the relevant biomechanical literature is far from complete. The clinical decision-making process is made complex because the movement problems may be removed from their kinematic manifestations. For example, failure to extend the hip in stance phase may be caused by adaptive shortening of the plantarflexor muscles; in this case the most significant consequence of adaptive changes to the ankle musculature is a kinematic deviation at the hip. Moreover, kinematic deviations may be displaced in time from their causes. To use the same example, failure to attain normal peak hip extension late in stance phase may also be caused by an inability to produce a large hip extensor muscle moment early in stance phase.

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Another complication in the analysis of hemiplegic gait arises from the need to differentiate between those departures from normal gait kinematics which arise as a direct consequence of the motor problem and those which arise as learned (or adaptive) compensations for the motor problem. Failure to extend the hip late in stance phase may arise as a direct consequence of an inability to produce a sufficiently large moment with the hip extensors, or it may arise as a compensation which keeps the body's centre of mass over the base of support when adaptive shortening of the plantarflexor muscles prevents dorsiflexion. Differentiating between motor problems and their compensations is essential if effective intervention is to occur. Intervention aimed at increasing the ability of the hip extensors to generate tension is unlikely to increase hip extension late in stance phase if the problem is that short plantarflexor muscles are preventing the forward inclination of the leg. Likewise, intervention aimed at increasing the length and compliance of the plantarflexor muscles may not increase the amount of stance phase hip extension if the major problem is an inability to generate sufficient moments with the hip extensors. A particular focus of this paper will be the identification of both the motor problems which cause common kinematic deviations and their compensations.

Throughout the paper, the kinematic and kinetic data of Winter (1987) have been used as a normal model with which to compare the kinematics and kinetics of hemiplegic gait. Specifically, Winter's 1987 data for slow walking have been used, because they are the most appropriate for comparison with the mechanics of hemiplegic gait (Lehmann et al 1987). It is recognised that Winter's data (in which subjects walked at a cadence which was 20 steps.min^{-1} less than that of their chosen walking speed) may not provide an entirely satisfactory normal model, because many people walk at considerably slower velocities after stroke. In fact, in 21 reviewed papers which measured walking speed after stroke, the mean walking speed reported was only 0.4 m.s^{-1}. The use of Winter's 1987 slow walking data probably presents little problem when considering the kinematics of hemiplegic gait, as the kinematics are almost independent of walking speed over a modest range of speeds (Winter 1987), but it means that kinetic comparisons should be made with caution.

This paper, which is the first of two parts, considers the mechanics of the stance phase of hemiplegic gait. The mechanics of the swing phase of hemiplegic gait will be considered in the second part (Moore et al 1993). Each part is divided into sections which are concerned with a particular kinematic deviation. In each section, there is a brief overview of normal mechanics relevant to the kinematic deviation and the potential causes of the kinematic deviation. [Readers seeking clarification of the mechanical terminology used could consult the recent paper by Herbert et al (1993)]. The kinematic deviations have been presented in terms of segmental kinematics such as decreased hip extension in late stance phase, rather than in terms of broad kinematic measures such as decreased step length, because segmental kinematics better provide the information necessary for analysis. Abnormalities of ankle and foot motion (except the sagittal plane kinematics of the ankle) and other transverse plane kinematics are arguably important but they have not been dealt with here because of the paucity of relevant biomechanical information available.

For simplicity, each of the kinematic deviations has been presented separately. However, physiotherapists usually see people who present with a combination of kinematic deviations. When this occurs, the task for the physiotherapist becomes one of reconciling a large amount of information about potential causes of the movement problems. It is not the intention of this paper to map out the processes by which physiotherapists make these complex decisions, although it is considered that these processes require further examination in the physiotherapy literature. Instead, hypotheses about common kinematic deviations and their causes have been presented in the belief that they can provide physiotherapists with information on which to base these decisions.

Stance phase

The kinematics of the stance phase of normal gait have been extensively documented in the biomechanics literature (Eberhart et al 1968, Murray et al 1984, Saunders et al 1953, Winter 1987). The most important kinematic features can be briefly summarised as follows. For most of stance phase, the hip extends. Hip extension, together with ankle dorsiflexion, transports the vertical trunk segment from behind to in front of the stance foot. Rapid ankle plantarflexion at the end of stance phase further propels the body forward. Early in stance phase, the trunk is displaced laterally, accompanied by adduction of the stance hip and eversion of the stance foot, so that the centre of body mass is moved to a point nearly over the stance foot for the duration of single support phase. The knee remains relatively extended throughout the single support phase, but a small amount of flexion occurs early in stance phase. During the final third of stance phase, the knee flexes in preparation for swing phase.

During the single support phase of normal stance, the motion of the body resembles that of an inverted pendulum, with the body rotating over the stance foot (Cavagna et al 1976, Mochon and McMahon 1980). The analogy of an inverted pendulum is particularly suitable because, like an inverted pendulum, the forward motion of the body during this phase of the gait cycle occurs largely under the influence of weight moments; muscle moments contribute relatively little to the motion of the body during single support. At the beginning and end of stance phase, however, the pendular motion of the body is

Table 1.
Commonly observed stance phase kinematic deviations and some of their possible causes.

Kinematic deviation	Potential causes
Decreased peak hip extension in late stance phase	▲ Inability to produce sufficient active tension with the hip extensor muscles early in stance ▲ Adaptive shortening of hip flexor muscles ▲ Production of excessive active tension with the hip flexor muscles in stance ▲ Production of excessive active tension with the ankle plantarflexor muscles in stance ▲ Adaptive shortening of ankle plantarflexor muscles ▲ Inability to produce sufficient active tension with the hip flexor muscles late in stance ▲ Inability to produce sufficient active tension with the knee extensor muscles throughout stance ▲ Inability to produce sufficient active tension with ankle plantarflexor muscles in stance
Decreased peak lateral pelvic displacement in stance phase	▲ Inability to produce sufficient active tension with the hip adductor muscles in early stance ▲ Inability to produce sufficient active tension with the hip abductor muscles in early to mid stance
Increased peak lateral pelvic displacement in stance phase	▲ Adaptive shortening of the hip adductor muscles ▲ Production of excessive active tension with the hip adductor muscles in stance ▲ Inability to produce sufficient active tension with the hip abductor muscles in early to mid stance
Decreased knee flexion (or knee hyperextension) in stance phase	▲ Inability to produce sufficient active tension with the knee flexor muscles in mid stance ▲ Inability to produce sufficient active tension with the knee extensor muscles in stance ▲ Production of excessive active tension with the ankle plantarflexor muscles in early or mid stance ▲ Adaptive shortening of ankle plantarflexor muscles
Increased knee flexion in stance phase	▲ Inability to produce sufficient active tension with the knee extensor muscles in a shortened range during stance ▲ Adaptive shortening of the knee flexor muscles or decreases in the compliance of other tissues on the flexor aspect of the knee ▲ Production of excessive active tension with the knee flexor muscles in stance
Decreased ankle plantarflexion at toe-off	▲ Inability to produce sufficient active tension with the ankle plantarflexor muscles in late stance ▲ Unnecessary due to segmental alignment

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initiated and terminated by large bursts of muscle activity (Mochon and McMahon 1980). That is, muscle activity in the double support phases sets the initial and final conditions for stance and swing. The initial conditions for stance phase appear to be set by a large burst of concentric hip extensor muscle activity, and the final conditions are controlled by eccentric hip flexor muscle activity and concentric plantarflexor muscle activity (Winter 1987).

Table 1 summarises important deviations from the normal kinematics of stance phase which are commonly observed following hemiplegic stroke. Each of these kinematic deviations will be considered in subsequent sections of the paper.

Decreased hip extension

During stance phase, when walking with a slow cadence, the hip normally extends from about 16 degrees of flexion (SD 7 degrees) at heel strike to 11 degrees of extension (SD 8 degrees). The peak hip extension occurs near the end of stance phase at approximately 54 per cent of the gait cycle. During the final 6 per cent of stance phase (between 54 per cent and 60 per cent of the gait cycle) the hip flexes to about 8 degrees (SD 7 degrees) of extension (Winter 1987, Figure 1). Hip extension during stance phase is important because it moves the vertical trunk segment forward over the stance foot, contributing to a normal contralateral step length.

Decreased hip extension is a commonly reported kinematic deviation following hemiplegic stroke (Bogardh and Richards 1981, Knutsson and Richards 1979, Lehmann et al 1987, Olney et al 1988 and 1989, Pinzur et al 1986 and 1987, Richards and Knutsson 1974). One probable cause is a reduced net hip extensor moment at the beginning of stance phase. Normally, an extensor muscle moment acts at the hip in the first third of stance phase (Winter 1987, Figure 2). Bursts of EMG activity have been recorded in the gluteus maximus, gluteus medius, semitendinosis and

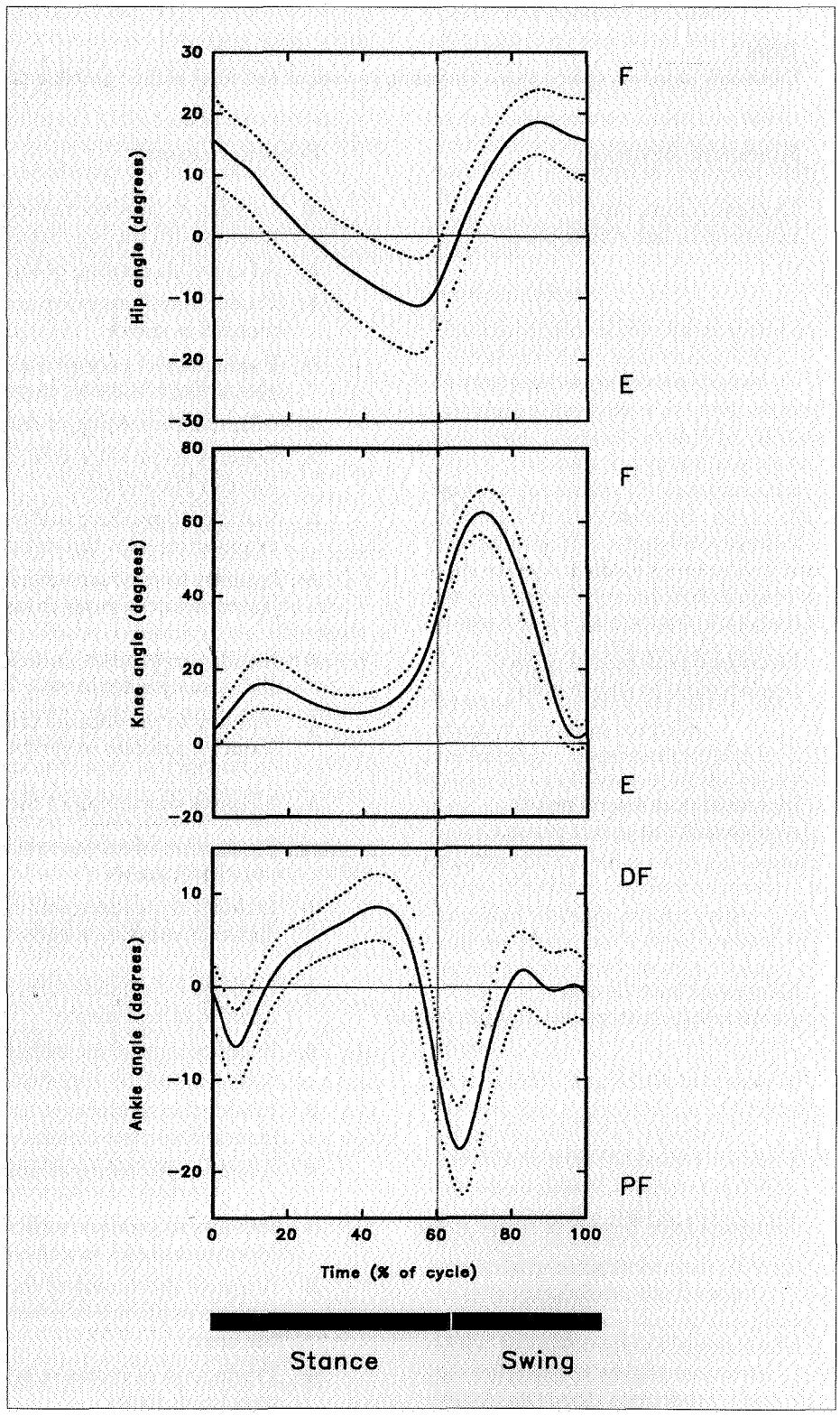

Figure 1.
Normal sagittal plane hip, knee and ankle angular displacements versus time during level walking at a slow cadence (redrawn from the data of Winter 1987 with permission from the author). Graphs are of means and one standard deviation about the mean.

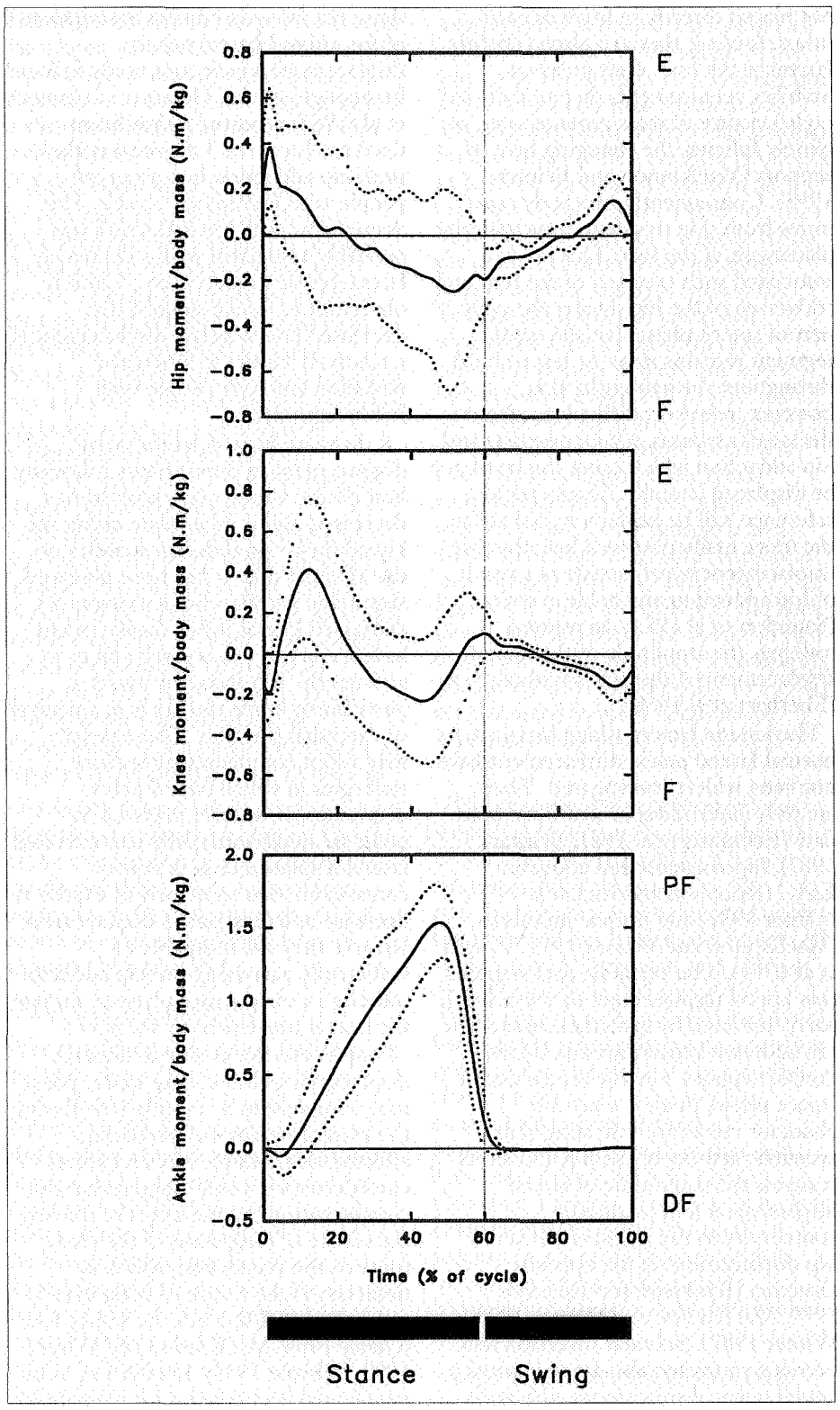

Figure 2.
Normal hip, knee and ankle muscle moments during level walking at a slow cadence (redrawn from the data of Winter 1987 with permission from the author). Graphs are of means and one standard deviation about the mean.

biceps femoris during this period, implicating all of these muscles in the initiation of hip extension (Winter 1987). A failure to set the initial conditions for the inverted pendular movement of the stance leg, caused by an inability to generate a burst of hip extensor muscle activity, may limit the forward acceleration of the hip and result in reduced hip extension later in stance phase. Adaptive shortening of the hip flexor muscles or excessive activity of the hip flexor muscles can also decrease the net hip extensor moment, limiting the amount of hip extension which occurs in stance phase.

It is likely that some of the most common causes of the decreased hip extension in stance phase are problems with the length and appropriate activation of the plantarflexor muscles. Between approximately 6 and 44 per cent of a normal gait cycle, the ankle dorsiflexes from about 7 degrees (SD 4 degrees) of plantarflexion to about 9 degrees (SD 4 degrees) of dorsiflexion (Winter 1987, Figure 1). Ankle dorsiflexion permits forward inclination of the leg, which is necessary if hip extension and forward transport of the vertical trunk segment are to occur. An increased net plantarflexor moment, which could result from adaptive shortening of the plantarflexor muscles or excessive plantarflexor muscle activity, can limit the ankle dorsiflexion, and thus the amount of hip extension that occurs in late stance phase (Lehmann et al 1987).

An inability to activate the hip flexor muscles is another possible cause of decreased hip extension during stance phase. Winter (1987) has reported that normal subjects generate a net hip flexor moment in the second half of the stance phase. During this period, the hip is extending, indicating that eccentric hip flexor muscle activity is acting to slow down the hip movement (Winter 1987, Winter et al 1991, Figures 1 and 2). It is possible that, following hemiplegic stroke, a person who has reduced or absent eccentric hip flexor muscle activity may compensate for their inability to control hip motion by restricting the amount of hip extension that occurs.

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It is also possible that a decrease in hip extension may be caused by an inability of the plantarflexor muscles to generate sufficiently large active tensions. Normally, the plantarflexor muscles contract eccentrically to produce large moments throughout most of the stance phase (Knutsson and Richards 1979, Winter 1987, Figure 2). Presumably this occurs in order to control the tendency of the body to rotate forward over the foot as the centre of body mass passes anterior to the ankle (Sutherland et al 1980). Hip extension may be restricted in mid to late stance phase as a compensation for a decreased ability to contract the plantarflexor muscles eccentrically. That is, the person who is unable to produce plantarflexion moments large enough to control the rotation of the body over the foot may employ movement strategies in which the body mass is not allowed to pass too far anterior to the ankle (Sutherland et al 1980). If the centre of body mass does not pass in front of the hips, and if the trunk remains vertical or inclines forward, then a consequence must be a decrease in peak hip extension.

Finally, reduced hip extension and increased ankle dorsiflexion may be associated with increased knee flexion during stance phase. This more indirect cause of decreased hip extension will be addressed in the section on excessive knee flexion in stance phase.

If the hip is insufficiently extended at the end of stance phase, contralateral step length will be reduced. Lehmann et al (1987) reported that a mean 14 degree decrease in hip extension of a group of people with hemiplegic stroke was associated with an 8cm reduction of contralateral step length from normal values. People with hemiplegic stroke may compensate for a lack of hip extension by excessively rotating their trunk forward on the swing side in late stance phase, which slightly increases the contralateral step length.

Decreased or increased lateral pelvic displacement

During normal walking, the feet are not placed directly in front of each other. Instead, they are placed slightly lateral to the line of progression. Stability requirements dictate that the body's centre of mass remains over, or closely follows, the changing base of support (MacKinnon and Winter 1993). Consequently, the body must move from side to side in time with the placement of the feet. This is associated with eversion of the foot and adduction of the hip during the early part of stance phase, but the trunk segment remains more or less vertical throughout the gait cycle. It is, however, relatively difficult to observe the small amounts of foot eversion and hip adduction which cause the trunk to be displaced laterally. Consequently, reference will be made here instead to the more easily observed lateral pelvic displacement which occurs as a result of hip adduction and ankle eversion (Saunders et al 1953). In normal walking, the amplitude of the lateral displacement of the pelvis is about 5cm (Eberhart et al 1968).

The kinetic factors which bring about normal lateral pelvic displacement have not been widely investigated. There are only isolated reports of force plate data (Lehmann et al 1987, Winter 1987), hip abductor and adductor EMG (Knutsson and Richards 1979, Winter 1987) and muscle moments (MacKinnon and Winter 1993, Winter et al 1991). The available data suggests that lateral displacement of the pelvis is partly initiated by ipsilateral concentric hip adductor muscle activity during double support. For the remainder of stance phase, there is a net hip abductor moment, indicating that eccentric activity of the hip abductors controls the magnitude of lateral displacement and eventually contributes to the initiation of lateral hip displacement in the opposite direction (Knutsson and Richards 1979, MacKinnon and Winter 1993, Winter 1987). Subtalar inversion and eversion moments also act to control lateral pelvic displacement, although they are highly variable (MacKinnon and Winter 1993).

As the majority of studies on hemiplegic gait have investigated only gait deviations in the sagittal plane, there are few quantitative descriptions of the altered lateral pelvic displacement which commonly follows hemiplegic stroke. However, Lehmann et al (1987) reported a significant decrease (mean of 3 degrees) in the peak hip adduction in a group of people with hemiplegic stroke. This decrease in peak hip adduction was probably associated with a decreased lateral pelvic displacement. Clinical observations would suggest that decreased lateral pelvic displacement is a relatively common kinematic deviation amongst people with hemiplegic stroke.

A decrease in peak lateral pelvic displacement in stance phase following hemiplegic stroke may result from a decreased ability to activate either the hip adductor or abductor muscles on the affected side. It has been observed that many people who demonstrate a decreased lateral pelvic displacement have a markedly decreased ability to activate the hip abductor muscles, particularly when the hip is in a neutral or extended position. These people may adopt compensatory walking strategies in which lateral pelvic displacement is never initiated, in order to avoid potentially uncontrolled lateral displacements. It is also conceivable that some people exhibit a decrease in lateral pelvic displacement because they are incapable of sufficiently activating the hip adductor muscles in early stance phase to initiate the lateral movement of the pelvis.

People who are unable to laterally displace the pelvis during stance phase may compensate by rapidly side-flexing the trunk towards the ipsilateral, affected side. Simple biomechanical considerations would suggest that this compensation has the effect of moving the centre of body mass of the trunk towards the stance side, which is necessary if the centre of body mass is to be displaced towards the stance foot (Cerny 1984, MacKinnon and Winter 1993, Whittle 1991). In addition, some people may compensate for an inability to generate sufficient moments with the hip abductors by increasing step width, or base of support, to ensure that the centre of body mass does not pass lateral to the foot. Decreased

lateral pelvic displacement may also be associated with decreased ipsilateral hip extension during stance phase, a decrease in contralateral step length or a shortened duration of contralateral swing phase.

While there have been no biomechanical studies which have described increases in lateral pelvic displacement following hemiplegic stroke, clinical observations indicate that this is also a relatively common kinematic deviation (Carr and Shepherd 1987). An increase in the net hip adductor moment, caused by either excessive activation or adaptive shortening of the hip adductor muscles, may result in an increase in lateral pelvic displacement. Conversely, if a person is unable to sufficiently activate the hip abductor muscles to constrain lateral pelvic displacement in stance phase, they may adopt a walking strategy in which the pelvis is allowed to be displaced excessively laterally, to the point at which further displacement is constrained by the passive resistance of tissues on the lateral aspect of the hip.

Excessive lateral pelvic displacement may be accompanied by lateral flexion of the trunk towards the contralateral, unaffected side. This compensation occurs in order to displace the body's centre of mass towards the base of support (Whittle 1991).

Knee hyperextension

When walking with a slow cadence, the knee flexes from about 4 degrees (SD 5 degrees) to 16 degrees (SD 7 degrees) in the first quarter of stance phase, after which it extends to about 8 degrees (SD 5 degrees) of flexion by about two thirds of the way through stance phase. Presumably this yield at the knee is important for shock absorption and to minimise the vertical displacement of the body's centre of mass (Eberhart et al 1968, Saunders et al 1953). The knee then flexes to approximately 35 degrees (SD 6 degrees) by toe-off in preparation for swing phase (Winter 1987, Figure 1).

In early stance phase, the weight of the thigh and trunk segments tends to flex the knee because the centre of mass of these segments falls posterior to the knee. Also, at this stage of the gait cycle, the body is decelerating and this is associated with a backwardly orientated ground reaction force which further tends to flex the knee (Boccardi et al 1981). In order to prevent an excessive acceleration of the knee into flexion, a net extensor moment acts at the knee. Eccentric knee extensor muscle activity controls (slows) the flexion component of yield, and concentric knee extensor muscle activity then acts to move the knee back towards extension. In mid stance, the weight of the trunk segment acts to extend the knee; in order to prevent knee hyperextension a net flexor moment acts at the knee, with eccentric knee flexor muscle activity controlling knee extension (Knutsson and Richards 1979, Winter 1987, Figure 2). Soon thereafter, as the knee moves forwards over the foot, external forces act to flex the knee. The kinetics producing knee flexion prior to toe-off are discussed in more detail in the paper on swing phase (Moore et al 1993).

The preceding description of knee kinetics is based on the mean data of 19 subjects (Winter 1987). There is a large variability, however, in the muscle moment values reported, with the knee and hip having coefficients of variation of 171 per cent and 207 per cent, respectively (Winter 1987). There appears to be a flexible trade-off between the extensor muscle moments generated at the hip and knee during stance phase, so that the relative contribution of the individual moments may vary both within and between subjects (Winter 1987). However, the sum of the hip, knee and ankle moments, called the total support moment by Winter (1980), remains extensor and relatively constant producing relatively invariant kinematics.

Knee hyperextension is one of the most commonly reported kinematic deviations in the gait of people with hemiplegic stroke (Knutsson and Richards 1979, Lehmann et al 1987, Morris et al 1991, Olney et al 1988, Pinzur et al 1986 and 1987, Richards and Knutsson 1974, Takebe and Basmajian 1976, Van Griethuysen et al 1982). As with the other common kinematic deviations, knee hyperextension in stance phase probably has a number of causes.

Knee hyperextension commonly arises as a compensation for a decreased ability to generate a knee extensor muscle moment (Cerny 1984) caused by a reduced or absent ability to activate the knee extensor muscles. In order to achieve single support on the affected leg in the absence of the ability to generate forces with the knee extensor muscles, the knee is fully extended, sometimes to beyond the neutral position, and the trunk segment is inclined forward by flexion of the hip. Knee hyperextension combined with hip flexion has the effect of moving the centre of mass of the thigh and trunk anterior to the knee, producing a large weight moment which tends to extend the knee. The presence of passive structures posterior to the knee which can limit knee extension, including muscles, joint capsule and ligaments, allow this compensatory strategy to prevent collapse of the knee even when the knee extensor muscles cannot be activated.

The knee flexors (predominantly the hamstrings and the gastrocnemius muscles) produce a moment which acts to prevent rapid knee hyperextension throughout a large part of the middle of stance phase. This suggests that it is also possible that a decrease in the knee flexor muscle moment, attributable to decreased or absent gastrocnemius or hamstring muscle activity, may enable the knee to rapidly hyperextend in mid stance. Unfortunately the precise action of the two-joint gastrocnemius and hamstring muscles in walking is not well understood. While they produce a flexor muscle moment at the knee, it is possible that under certain conditions they could actually act to extend the knee in walking (Zajac and Gordon 1989). Further investigation of the role of these muscles in causing knee hyperextension in stance phase is warranted.

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Throughout most of stance phase, there is a net plantarflexor moment controlling the forward rotation of the leg on the fixed foot (Winter 1987). If this plantarflexor muscle moment is excessive, it will tend to rotate the leg backwards on the foot whilst the thigh continues to move forwards, resulting in excessive knee extension. Increases in the plantarflexor muscle moment commonly occur as a result of excessive muscle activity or adaptive shortening of the calf muscles (Halar et al 1978, Knutsson 1981, Knutsson and Richards 1979, Thilmann et al 1991).

One last cause of knee hyperextension during stance is an increased knee extensor muscle moment. Some people with hemiplegic stroke may have difficulty regulating the tension in the knee extensor muscles, particularly in the stance phase of walking. Excessive activation of the knee extensor muscles can cause the knee to be hyperextended throughout stance phase.

Increased knee flexion during stance phase

Excessive knee flexion throughout stance phase is well documented in people with hemiplegic stroke (Bogardh and Richards 1981, Carlsoo et al 1974, Knutsson and Richards 1979, Olney et al 1986, 1988 and 1989, Takebe and Basmajian 1976, Trueblood et al 1989). The increased knee flexion cannot simply be explained by a decreased ability to generate knee extensor muscle moments – in fact the knee extensor muscles probably have to produce a larger extensor moment when the knee is flexed than when it is extended. However, excessive knee flexion may be caused by an inability to generate sufficient tension in the knee extensor muscles when these muscles are in a shortened position. People with hemiplegic stroke may be unable to generate significant knee extensor muscle moments when the knee is close to full extension, particularly when they are in standing. Perhaps this is a reflection of the length-tension properties of muscles which dictate that muscles are least able to generate tension at short lengths. Alternatively, it could reflect a difficulty in activating the knee musculature under conditions in which it is necessary to change rapidly between activating the knee extensors and knee flexors.

An increase in the net knee flexor moment, which could result from excessive production of tension by the knee flexor muscles (hamstrings and gastrocnemius) or adaptive shortening of the soft tissues on the flexor aspect of the knee, can be another cause of increased knee flexion during stance phase. However, in the absence of information about the size of the moment arms of the knee flexor muscles, it is hard to be certain about the exact effect of increased knee flexor muscle tension. The mechanics of these two-joint muscles are such that it is conceivable that they could act to extend the knee during stance phase (Zajac and Gordon 1989). Therefore, while adaptive shortening and excessive activation of the knee flexor muscles would appear to be likely causes of excessive knee flexion in stance phase, the true importance of these putative causes awaits confirmation.

During mid stance phase, the plantarflexors contract eccentrically to constrain the forward rotation of the leg (Winter 1987). If the plantarflexors are not capable of producing the required active tension, forward rotation of the leg may continue until further rotation is prevented by passive tension in structures on the plantar aspect of the ankle. However, if the body's centre of mass is to remain over the base of support, the thigh cannot also rotate forward. As a consequence, an inability to contract the plantarflexor muscles eccentrically during mid stance can result in excessive knee flexion.

Unless compensated for, increases in the amount of knee flexion during stance phase may produce decreases in peak ipsilateral hip extension and contralateral step length because decreased hip extension and excessive knee flexion decrease the extent to which the hips are transported forward over the stance foot.

Decreased plantarflexion at toe-off

The ankle rapidly plantarflexes from about 9 degrees of dorsiflexion (SD 4 degrees) to 18 degrees of plantarflexion (SD 5 degrees) in the last quarter of stance phase (Winter 1987, Figure 1). Rapid plantarflexion may contribute to forward propulsion of the centre of mass (Hof et al 1983, Winter 1987) and probably serves to increase contralateral step length. Plantarflexion at the end of stance phase probably also has a profound influence on the dynamics and energetics of swing phase (Winter 1987).

Several studies have reported decreased ankle plantarflexion at toe-off in people with hemiplegic stroke (Bogardh and Richards 1981, Knutsson and Richards 1979, Olney et al 1988 and 1989, Trueblood et al 1989). This is most likely to be attributable to an inability to contract the plantarflexors concentrically with sufficient tension to overcome the inertia of the rest of the body. Perhaps, given the difficulty of generating large muscle forces during concentric contractions at high velocities, it is not surprising that the ability to plantarflex the ankle at toe-off is so often lost following stroke. It is likely that, following stroke, many people have difficulty activating the plantarflexor muscles sufficiently (Knutsson 1981, Knutsson and Richards 1979). But even when the muscles are appropriately activated, it is likely that they will have undergone secondary adaptations which make them less capable of generating large forces during fast concentric contractions. The decreases in length of the plantarflexor muscles which have been reported following hemiplegic stroke (Halar et al 1978), and which are often observed in clinical practice, are likely to reduce significantly the plantarflexor muscles' ability to generate force at high velocities.

Plantarflexion may also be restricted if segmental alignment at the end of

stance phase is such that plantarflexion would result in a vertical, or even posterior, displacement of the rest of the body, rather than forward propulsion. If the centre of body mass is not sufficiently forward of the ankle at the time at which the plantarflexors contract most strongly, the plantarflexor muscle moment may act to rotate the leg backwards rather than lift the heel off the ground. In this situation, contraction of the plantarflexor muscles would actually impede the goal of getting the body forward over the stance foot. For this reason, people who are unable to advance their centre of body mass forward of the ankle in late stance phase may adopt a movement strategy in which they do not contract the plantarflexor muscles concentrically to plantarflex the ankles just before toe-off.

Summary

This paper has identified several of the commonly-observed kinematic deviations in hemiplegic gait and has sought to generate hypotheses about the potential causes of these deviations. Testing of these hypotheses provides a basis for structuring specific intervention. A subsequent paper (Moore et al 1993) will address these issues for swing phase of walking following hemiplegic stroke.

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