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Functional Recovery of Gait and Joint Kinematics After Right Hemispheric Stroke

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ABSTRACT. Huitema RB, Hof AL, Mulder T, Brouwer WH, Dekker R, Postema K. Functional recovery of gait and joint kinematics after right hemispheric stroke. Arch Phys Med Rehabil 2004;85:1982-8.

Objective: To gain insight into the relation between changes in gait patterns over time and functional recovery of walking ability in stroke patients.

Design: Cohort study.

Setting: Inpatient rehabilitation center of a university hospital in the Netherlands.

Participants: Thirteen stroke patients admitted, or awaiting admission, for inpatient rehabilitation 3 weeks poststroke, and 16 healthy control subjects.

Interventions: Not applicable.

Main Outcome Measures: At 3, 6, 12, 24, and 48 weeks poststroke, functional recovery of walking ability was assessed with the Rivermead Mobility Index (RMI) and the Functional Ambulation Categories (FAC). When possible, kinematics of the knee, hip, and pelvis were assessed through gait analysis in an 8×4m gait laboratory. Minimal scores of 8 on the RMI and 4 on the FAC were necessary before patients were classified as functionally recovered.

Results: Patients whose joint kinematics during ambulation had recovered to within the range of the control group showed functional recovery of walking ability. However, some patients whose kinematics had developed toward an abnormal pattern also showed functional recovery.

Conclusions: Recovery of joint kinematics toward a normal pattern is not required for functional recovery of walking ability. Early recognition of compensatory walking patterns that facilitate functional recovery may have implications for rehabilitation programs.

Key Words: Cerebrovascular accident; Hemiplegia; Gait; Rehabilitation; Walking.

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R EGAINING WALKING ability is of great importance to stroke patients, and is a major goal of all rehabilitation programs.[1,2] Although the reported figures vary, approximately 50% to 80% of patients who survive a stroke will eventually regain some degree of walking ability.[3] Several studies have shown that most of the motor recovery occurs within the first 3 months poststroke, and that the initially steep recovery curve levels at about 6 months to a year poststroke.[3-7]

Recovery of walking ability is often quantified with clinical measures, such as the Rivermead Mobility Index[8] (RMI) or the Functional Ambulation Categories[9] (FAC), but gait velocity is also often used as a measure of recovery.[10] Another way to record recovery of gait after stoke is through gait analysis of specific characteristics of hemiplegic gait patterns. Several studies have suggested classification of different types of hemiplegic gait patterns in stroke patients.[11-14] However, much is still unknown about the relation between the changes in gait patterns over time and functional recovery of walking ability in stroke patients. Our purpose in this study, therefore, was to gain more insight into this relation. We addressed the changes that take place in joint kinematics in the first year poststroke and how they relate to the recovery of functional walking ability.

Though gait patterns of stroke patients may vary greatly, some specific movement patterns can be observed in subgroups of patients, and several studies have attempted to classify these hemiplegic gait patterns. In their review, Olney and Richards[12] concluded that, concerning patterns in joint kinematics, hemiplegic gait can be classified by a combination of the following: (1) a reduced hip joint angle amplitude in the sagittal plane, caused by a decreased hip flexion at heel strike and a decreased hip extension at toe-off; (2) a reduced knee joint angle amplitude caused by increased knee flexion at heel strike and decreased knee flexion at toe-off and during swing; and (3) increased plantarflexion of the ankle at heel strike and during swing and decreased plantarflexion at toe-off. Abnormalities in these joint kinematics often lead to secondary compensations in other body segments. For example, reduced knee flexion during swing can be accompanied by circumduction, vaulting, or upward pelvic tilt.[15,16] These secondary compensations may be energy inefficient in a normal healthy gait pattern. In a stroke patient, however, structural changes have taken place in the central nervous system so that the changed motor patterns may reflect adaptations that are optimal for the system's altered state. The relation between the recovery of joint kinematics and functional recovery of walking ability therefore might not be as straightforward as one might think, and the observed kinematic changes toward a compensatory pattern that differ from the normal pattern might even facilitate functional recovery of walking ability.

Table 1: Control Subjects

Variable	Mean ± SD	Min	Max
Speed (m/s)	1.24±0.095	1.08	1.42
Dknee (deg)	38.0±2.13	33.2	44.6
Ahip (deg)	46.5±3.86	40.3	53.7
Dpelvis (deg)	0.1±1.46	−2.7	3.0

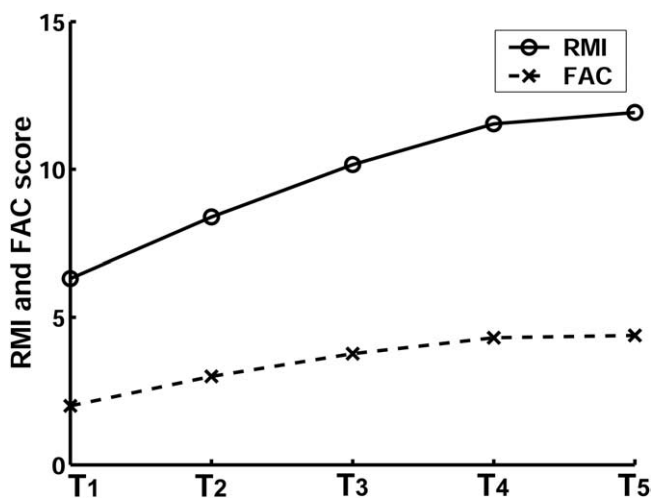Abbreviations: Max, maximum; Min, minimum.

Fig 1. Mean group score on the RMI and FAC for each measurement.

In this study, we recorded recovery of knee and hip joint kinematics in stroke patients and compared that recovery with functional recovery of walking ability as measured with the FAC and the RMI. Furthermore, pelvic rotation in the sagittal plane was recorded because it was expected that pelvic rotation might be used to compensate for a reduced hip joint angle amplitude in the sagittal plane.

METHODS

Participants

This study was part of a larger study in which the effects on gait of hemineglect in right hemisphere stroke patients was researched. The patients in this study formed a control group in the larger study and was composed, therefore, only of right hemisphere stroke patients. All stroke patients in the neurologic wards of 2 local hospitals were screened, and if they met the inclusion criteria they were asked to participate in the

study. Patients had to (1) be from 20 to 80 years of age; (2) have suffered a first time, single right hemisphere cerebrovascular accident; (3) have no severe cognitive disorders that would have interfered with the study's purpose; (4) have no other premorbid disorders that would have affected the study's results; and (5) have been admitted for inpatient rehabilitation 3 weeks poststroke.

Over a period of 20 months, 15 patients were included. Two patients dropped out; 1 suffered a second stroke 1 month after the first stroke and the other never attained any walking ability at 48 weeks poststroke. The remaining 13 patients included 7 men and 6 women. Average age $\pm$ standard deviation (SD) was 59.4 ± 12.7 years (range, 35–79y). For comparison purposes, 16 healthy control subjects (8 men, 8 women) volunteered. Average age of the control group was 61.3 ± 11.1 years (range, 33–77y). None of the control subjects had a history of motor disability, vestibular disorder, or neurologic damage that would have interfered with the study's aims. The study was approved by the hospital's ethics committee and informed consent was obtained from each participant.

Procedure and Materials

At 3, 6, 12, 24, and 48 weeks poststroke (T_1 to T_5), patients' motor function and, if possible, gait characteristics were assessed. Motor function was assessed with the RMI and the FAC. Gait analysis, however, did not begin until a patient could walk several meters independently. Assistive devices were allowed. Gait analysis was done while patients walked at a self-selected, comfortable speed across the floor of an 8×4m gait laboratory. Patients were asked to walk from 1 side to the other side of the laboratory and back, resulting in 2 walking cycles. In the control group, all gait characteristics were assessed once.

Knee angle was measured with Penny & Giles goniometers.[a] Piezoelectric gyroscopes were used to record angular velocity in the sagittal plane of the thigh and pelvis. Temporal parameters, used for normalizing kinematic data (heel strike, toe-off, midstance, midswing), were recorded with an ultrasonic motion analysis system.[17] Data were sampled at 200Hz and further processed on a personal computer using Matlab, version 5.3.[b]
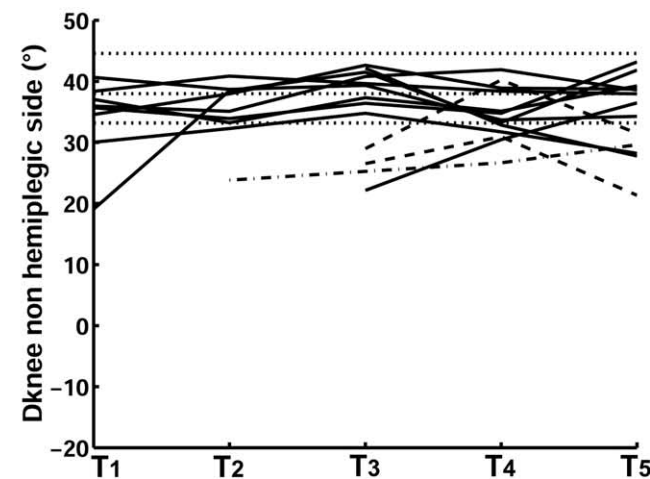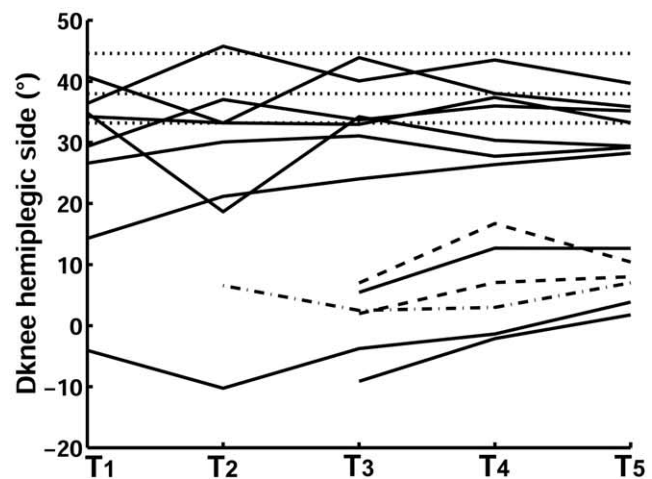
Fig 2. Dknee, the difference between knee flexion during midswing and midstance, presented for each patient and each measurement. Legend: —, patients whose gait had functionally recovered at T_5 according to the FAC (score $\geq$4) and RMI (score, $\geq$8); - - -, patients whose gait had not functionally recovered at T_5 according to the FAC (score, <4) and RMI (score, $\geq$8); - · - · -, FAC score <4 and RMI score <8; · · ·, mean, minimum, and maximum Dknee value of the control group.

Fig 3. Mean knee angle profiles for each patient at T_1, or first possible measurement, and T_5. Both hemiplegic side (HS) and nonhemiplegic side (NHS) are presented. (A, B) Profiles of patients whose Dknee had recovered at T_5; (C, D) profiles of patients whose Dknee had not recovered at T_5. Abbreviations: Ext, extension; Flex, flexion. Legend: see figure 2. Knee angle profiles at T_5 of patients for whom Dknee had recovered closely resemble profiles of healthy controls.

Data Analysis

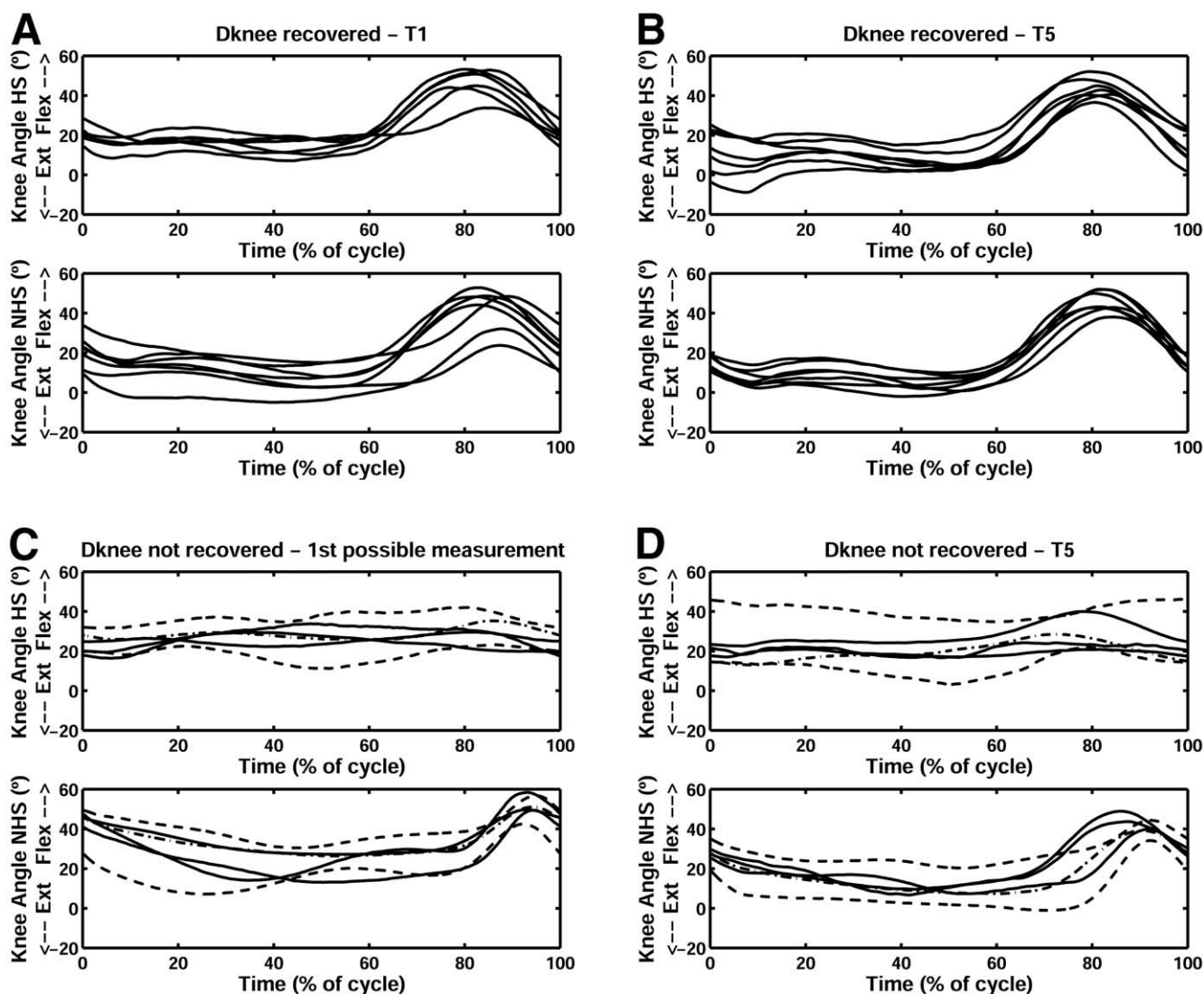Kinematics were calculated for each stride of both walking cycles and then averaged. The first and last strides of each cycle were omitted from analysis. Maximum knee flexion during swing can be used to quantify abnormalities in knee flexion patterns.[15,18] However, typical of a stiff-knee pattern, which often occurs in a hemiplegic gait, is the decrease in flexion *amplitude*, although a constant flexion during the full stride cycle may still be present. Therefore, we introduced the difference between knee flexion at midswing and midstance (Dknee) to quantify abnormalities in knee flexion patterns. It was expected that Dknee would be especially sensitive for revealing a stiff-knee pattern. Thigh and pelvis angle were obtained by integrating the angular velocity signals from the piezoelectric gyroscopes. An integration procedure always introduces an unknown constant in the output signal. As a result, the absolute values of the thigh and pelvis angle are arbitrary, but the shape and amplitude of the calculated signals are valid.

Reduced hip flexion and extension can be compensated by sagittal rotation of the pelvis. To quantify this compensatory pelvis rotation, we calculated the difference between pelvis angle in the sagittal plane at heel strike of the hemiplegic side and heel strike of the nonhemiplegic side (Dpelvis). In a symmetric gait pattern, the value of Dpelvis should be about zero, because the pelvis angle in the sagittal plane at heel strike left equals the angle at heel strike right.

The FAC and RMI were used to classify whether a patient's gait had functionally recovered. Minimum scores of 4 on the FAC and 8 on the RMI were required for a patient to be classified as functionally recovered.

Statistical Analysis

To determine whether the patient group's scores improved in time on the FAC and RMI, a repeated-measures analysis of variance with 1 within-subjects factor (measurement) with 5 levels (T_1 to T_5) was performed. If justified, post hoc median

tests on Dknee, Dpelvis, and hip rotation amplitude in the sagittal plane (Ahip) were performed between patients (sub-)groups and the control subjects. Reduced knee flexion would result in a decreased Dknee and reduced hip flexion and extension in a decreased Ahip. Therefore, Dknee and Ahip were tested single-sided, because in a hemiplegic gait pattern these variables will be lower than normal.

RESULTS

Table 1 shows mean, SD, and minimum and maximum values of the comfortable walking speed (Speed) for the control group. Furthermore, the difference between knee flexion during midswing and midstance (Dknee) and hip rotation amplitude in sagittal plane (Ahip) are shown, as is the difference between pelvis angle in the sagittal plane at heel strike left and heel strike right (Dpelvis).

The mean comfortable walking speed of the patients at T_5 was .81±.43m/s. Five patients used a walking cane and 2 required an ankle-foot orthosis during the experiment. Seven patients did not need an assistive device while walking.

RMI and FAC Scores

Figure 1 shows the mean group scores on both the RMI and the FAC for each measurement. Both tests showed a significant main linear effect for measurement ($F_{RMI_{1,12}}$=34.4, P<.001; $F_{FAC_{1,12}}$=29.3, P<.001).

Dknee

Dknee is presented for each patient and for each measurement in figure 2. Visual assessment of the hemiplegic side clearly reveals 2 subgroups. In a subgroup of 7 patients in the upper part of the figure, Dknee recovered to values at T_5 close to the control group. In a subgroup of 6 patients in the lower part of the figure, Dknee was low and did not recover. A median test at T_5 showed a significant difference between the subgroups (single sided, P=.010). Furthermore, median tests showed that the subgroup in which Dknee recovered did not significantly differ from the control group (single sided, P=.097), whereas the subgroup that did not recover did significantly differ from the control group (single sided, P=.018).

Figure 3 shows the mean knee angle profiles for each patient. Based on both the joint angle profiles and the value of Dknee in figure 2, the gait of patients in which Dknee had not recovered at T_5 can all be classified as a stiff-knee gait at T_5.

Dpelvis

Dpelvis is presented for each patient and for each measurement in figure 4. Visual assessment of Dpelvis did not reveal different subgroups at T_5 as clearly as it did with Dknee in figure 2. Therefore, patients with a Dpelvis within the range of that of the control group were classified as subjects with a recovered Dpelvis and patients with a Dpelvis outside the range of the control group were classified as subjects with a Dpelvis that had not recovered.

Figure 5 shows the mean hip and pelvis angle profile in the sagittal plane for each patient. Median tests showed a reduced hip angle amplitude (Ahip) on the hemiplegic side in patients whose Dpelvis had not recovered when compared with patients whose Dpelvis had recovered (single sided, P=.015), and compared with healthy controls (single sided, P=.035). Patients whose Dpelvis had recovered did not significantly differ from controls (single sided, P=.318). The pelvis angle in figure 5 clearly shows that the large negative values of Dpelvis at T_5 in figure 4 reflect a pendulum pelvis movement: the lower part of the pelvis is rotated forward at heel strike of the hemiplegic
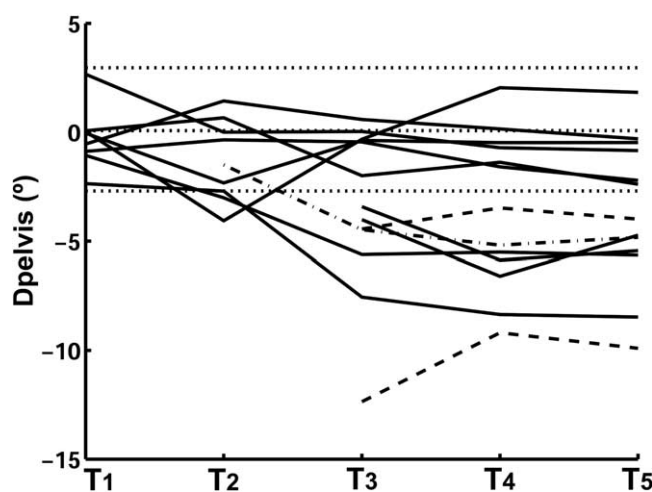

Fig 4. Dpelvis, the difference between the sagittal pelvis angle at heel strike of the hemiplegic side and heel strike of the nonhemiplegic side, presented for each patient and each measurement. Legend: see figure 2; · · · , mean, minimum, and maximum Dpelvis value of the control group.

side and it is rotated backward at heel strike of the nonhemiplegic side.

DISCUSSION

Maximum knee flexion during swing is often used to quantify abnormalities in knee flexion patterns[15,18] so that a spastic paretic stiff-legged gait is defined as reduced knee flexion during swing.[19,20] However, typical of a stiff-*knee* pattern is the decrease in flexion *amplitude*, although constant flexion during the full stride cycle may still be present. The lower part of figure 3 shows that, for some patients, maximum knee flexion during swing of the hemiplegic side reached values close to values in a healthy gait pattern, while at the same time the amplitude of the signal was far from normal. Therefore, maximum knee flexion during swing is not suitable for revealing a stiff-knee pattern. We used the difference between knee flexion at midswing and midstance (Dknee) to quantify stiff-knee gait. It appears that this parameter is quite sensitive for detecting a stiff-knee gait. Because no electromyographic recordings were made, the stiff-knee gait patterns cannot be classified with full certainty as spastic paretic stiff-legged gait, but it is likely that Dknee is more sensitive in detecting a spastic paretic stiff-legged gait than the maximum knee flexion during swing.

From figures 2 and 3 it can be concluded that when no stiff-knee gait is present after stroke, patients' gait will also functionally recover. Absence of a stiff-knee gait is not, however, a requirement for functional recovery because 3 patients with a stiff-knee gait also showed functional recovery. The question arises whether a stiff knee may be functional or, to go further, would these 3 patients have recovered walking in a functional sense when no stiff knee had developed? Several mechanisms have been proposed as a cause of stiff-knee gait. Spasticity of mainly the quadriceps was long considered to be the sole cause of stiff-knee gait. However, recent work[18,21,22] has indicated hip flexor weakness or poor ankle mechanisms are possible causes also. When a stiff-knee gait in a stroke patient is mainly caused by spasms, it could be the consequence of a lack of inhibition from higher cortical areas on spinal structures. However, we argue here that a stiff knee may emerge also as a result of a compensatory strategy caused by
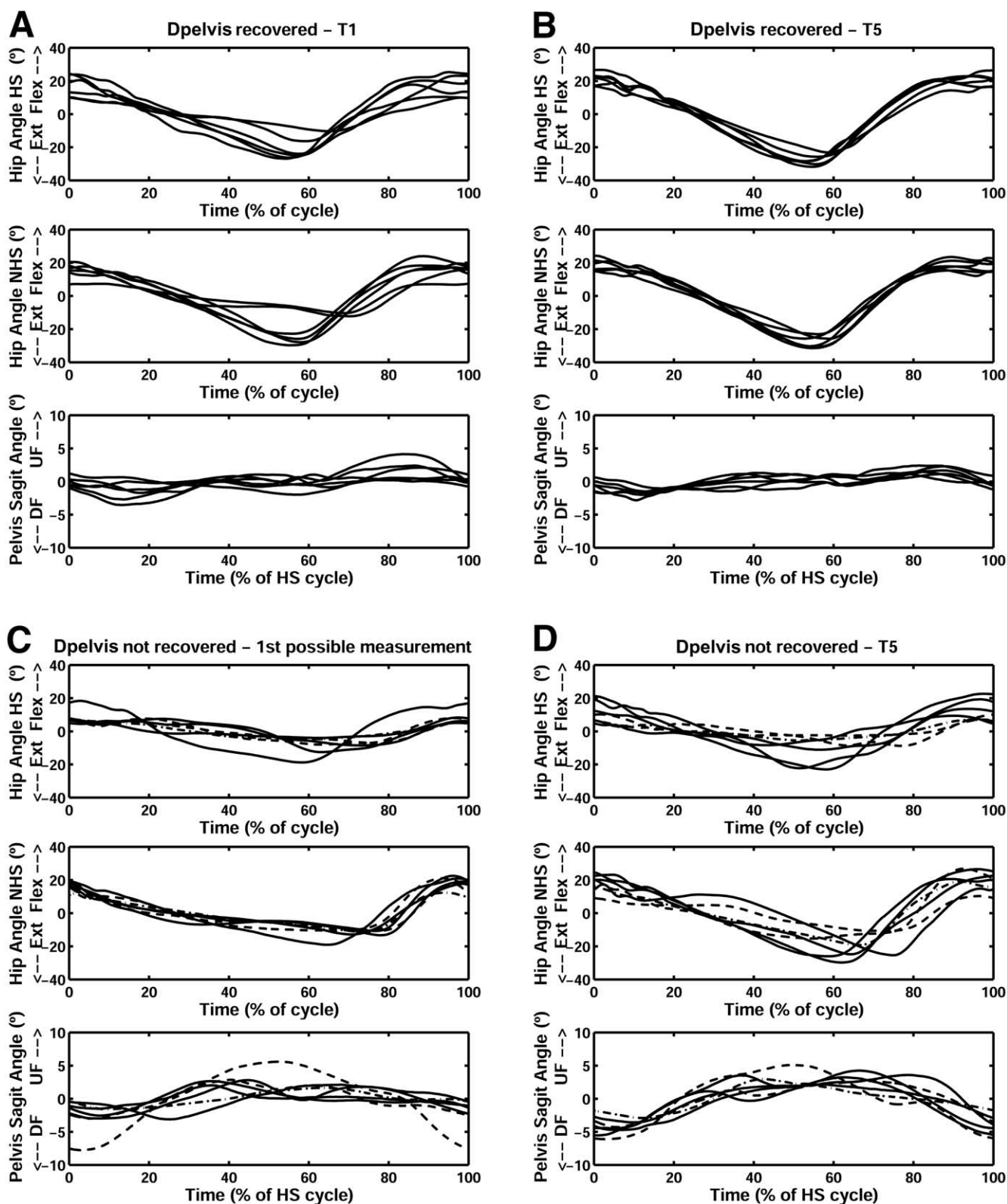
Fig 5. Mean hip and pelvis angle profile in the sagittal plane for each patient at T_1, or first possible measurement, and T_5. Both hemiplegic side and nonhemiplegic sides of hip angles are presented. Positive values for pelvis angle denote rotation of the upper part of the pelvis forward (UF); negative values denote rotation of the lower part forward (DF). Pelvis rotation is normalized according to a cycle of the hemiplegic side (heel-strike hemiplegic side to heel-strike hemiplegic side). (A, B) Profiles of patients whose Dpelvis had recovered at T_5; (C, D) profiles of patients whose Dpelvis had not recovered at T_5. Legend: see figure 2. Hip and pelvis angle profiles at T_5 of patients whose Dpelvis had recovered closely resembled profiles of controls.

cortical lesions having destroyed the smooth exploitation of motor programs required for normal gait. To optimize the output, the brain constructs a second best option in that a novel motor strategy (program) develops that enables the patient to support his weight during single support on the hemiplegic side and regain functional walking ability. This principle of output optimization on the basis of a novel strategy is an emergent characteristic of the neural system that has clear survival value.[23,24] Nevertheless, why did 3 stiff-knee gait patients show functional recovery and 3 others did not? Part of the answer may be found in radiologic or clinical differences between subjects: for example, the more severe the stroke, the less chance of functional recovery. These variables were not included in the experimental design as independent variables, however, and were therefore insufficiently recorded to be part of a post hoc analysis. Location and type of stroke did not, however, appear to differ between these patients and neither did the use of an assistive device during walking.

Figure 2 shows that development of a stiff-knee gait can be predicted by Dknee at the first possible gait analysis: if Dknee is below 10° it will not reach a normal value and a stiff-knee gait will develop. However, when a stiff knee can be functional, early recognition may be valuable for rehabilitation because in some cases it might be favorable to train the patient in using his stiff-legged gait rather than trying to change the gait to a normal, symmetrical gait.

We used the difference between pelvis angle in the sagittal plane at heel strike of the hemiplegic side and heel strike of the nonhemiplegic side (Dpelvis) to quantify possible compensatory pelvis rotation resulting from a reduced hip flexion and extension. For healthy subjects with a normal gait pattern, Dpelvis should be about 0°, which was indeed the case. Furthermore, all patients whose Dpelvis recovered within the range of the controls showed hip rotation amplitudes (Ahip) within the normal range. Patients whose Dpelvis did not recover, however, showed a significantly smaller hip rotation amplitude on the hemiplegic side. The lower right part of figure 5 shows that the divergent values of Dpelvis indicated a pendulum movement of the pelvis that compensates for the reduced hip rotation on the hemiplegic side: the lower part of the pelvis is rotated forward during flexion of the hip and backward during extension.

All patients whose Dpelvis recovered, and who thus had no pendulum movement of the pelvis and a sagittal hip rotation amplitude within the normal range, had functional recovery of gait. However, 4 patients whose Dpelvis did not recover also showed functional recovery. Therefore, the recovery of Dpelvis, indicating a normal pelvis rotation and a hip rotation amplitude within the normal range, is not required for functional recovery of gait. Because the pendulum movement of the pelvis in these patients appears to be a compensation for a reduced hip rotation, it is of course not unexpected that functional recovery is possible. For these patients, it may even be a requirement for functional recovery. Again, the question arises whether, in some cases, it would not be favorable to train a patient to use a pendulum movement of the pelvis rather than trying to change their gait to a normal, uncompensated gait. In this experiment, the compensatory pelvis rotation could not be predicted in an early phase, as it could with the stiff-knee gait. However, supportive training once this type of gait develops might still have promise. As with stiff-legged gait training, more research is needed to explore cases that might benefit from supportive training.

CONCLUSIONS

Recovery of joint kinematics in hemiplegic stroke patients toward a normal pattern is not required for functional recovery of walking ability. A stiff-knee gait and a pendulum movement of the pelvis do not always impede functional recovery of walking ability. In some cases, the abnormal gait patterns may even be a compensatory or adaptive strategy that facilitates functional recovery. Early recognition of these compensatory walking patterns may have implications for rehabilitation programs. The question arises whether physical therapy for these patients should aim at changing their gait toward the normal pattern. We argue that training focused on the use of the compensatory pattern may be indicated here.

References

1. Bohannon RW, Horton MG, Wikholm JB. Importance of four variables of walking to patients with stroke. Int J Rehabil Res 1991;14:246-50.
2. Mumma CM. Perceived losses following stroke. Rehabil Nurs 1986;11(3):19-24.
3. Skilbeck CE, Wade DT, Hewer RL, Wood VA. Recovery after stroke. J Neurol Neurosurg Psychiatry 1983;46:5-8.
4. Friedman PJ. Gait recovery after hemiplegic stroke. Int Disabil Stud 1990;12:119-22.
5. Fugl-Meyer AR. Post-stroke hemiplegia assessment of physical properties. Scand J Rehabil Med Suppl 1980;7:85-93.
6. Cassvan A, Ross PL, Dyer PR, Zane L. Lateralization in stroke syndromes as a factor in ambulation. Arch Phys Med Rehabil 1976;57:583-7.
7. Collen FM, Wade DT. Residual mobility problems after stroke. Int Disabil Stud 1991;13:12-5.
8. Collen FM, Wade DT, Robb GF, Bradshaw CM. The Rivermead Mobility Index: a further development of the Rivermead Motor Assessment. Int Disabil Stud 1991;13:50-4.
9. Holden MK, Gill KM, Magliozzi MR, Nathan J, Piehl BL. Clinical gait assessment in the neurologically impaired. Reliability and meaningfulness. Phys Ther 1984;6:35-40.
10. Collen FM, Wade DT, Bradshaw CM. Mobility after stroke: reliability of measures of impairment and disability. Int Disabil Stud 1990;12:6-9.
11. Knutsson E, Richards C. Different types of disturbed motor control in gait of hemiparetic patients. Brain 1979;102:405-30.
12. Olney SJ, Richards C. Hemiparetic gait following stroke. Part I: Characteristics. Gait Posture 1996;4:136-48.
13. Mulroy S, Gronley J, Weiss W, Newsam C, Perry J. Use of cluster analysis for gait pattern classification of patients in the early and late recovery phases following stroke. Gait Posture 2003;18:114-25.
14. De Quervain IA, Simon SR, Leurgans S, Pease WS, McAllister D. Gait pattern in the early recovery period after stroke. J Bone Joint Surg Am 1996;78:1506-14.
15. Kerrigan DC, Bang MS, Burke DT. An algorithm to assess stiff-legged gait in traumatic brain injury. J Head Trauma Rehabil 1999;14(2):136-45.
16. Kerrigan DC, Frates EP, Rogan S, Riley PO. Hip hiking and circumduction: quantitative definitions. Am J Phys Med Rehabil 2000;79:247-52.
17. Huitema RB, Hof AL, Postema K. Ultrasonic motion analysis system—measurement of temporal and spatial gait parameters. J Biomech 2002;35:837-42.
18. Waters RL, Garland DE, Perry J, Habig T, Slabaugh P. Stiff-legged gait in hemiplegia: surgical correction. J Bone Joint Surg Am 1979; 61:927-33.

19. Kerrigan DC, Karvosky ME, Riley PO. Spastic paretic stiff-legged gait: joint kinetics. Am J Phys Med Rehabil 2001;80:244-9.
20. Riley PO, Kerrigan DC. Kinetics of stiff-legged gait: induced acceleration analysis. IEEE Trans Rehabil Eng 1999;7:420-6.
21. Kerrigan DC, Gronley J, Perry J. Stiff-legged gait in spastic paresis. A study of quadriceps and hamstrings muscle activity. Am J Phys Med Rehabil 1991;70:294-300.
22. Kerrigan DC, Burke DT, Nieto TJ, Riley PO. Can toe-walking contribute to stiff-legged gait? Am J Phys Med Rehabil 2001;80:33-7.
23. Mulder T, den Otter R, van Engelen B. The regulation of fine movements in patients with Charcot Marie Tooth, type Ia: some ideas about continuous adaptation. Motor Control 2001;5:200-14.
24. Donker SF, Mulder T, Nienhuis B, Duysens J. Adaptations in arm movements for added mass to wrist or ankle during walking. Exp Brain Res 2002;146:26-31.

Suppliers

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b. The MathWorks Inc, 3 Apple Hill Dr, Natick, MA 01760-2098.