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Abstract

Background. Hemiparetic gait following stroke requires substantial energy consumption, which would promote deconditioning and disability. Optimal modalities for decreasing this energy cost remain challenging. Excessive energy consumption, however, seems to be mainly due to extra positive muscle work to substantially lift the body's center of mass (CM) against gravity during the paretic limb swing. **Objective.** The authors tested a new rehabilitation strategy in a pilot study to specifically reduce the energy cost in hemiparetic gait. **Methods.** Six chronic hemiparetic patients underwent a 6-week gait training program on a treadmill with real-time feedback of their CM and were asked to reduce its increased vertical displacement. The authors assessed the walking energy cost, vertical CM displacement, kinematics, and electromyogram activity without feedback before and after treatment. **Results.** After treatment, the vertical CM displacement decreased by 10% ($P = .005$), particularly when the CM vaulted over the nonparetic limb in stance, and the energy cost decreased markedly by 30% ($P = .009$). The paretic knee flexion in swing increased concomitantly by 45% and muscle co-contraction decreased significantly in both thigh muscles by 15%. **Conclusions.** The rehabilitation approach followed in this study seems remarkably effective in decreasing the walking energy cost. By treating the compensatory strategy (ie, the increased CM displacement), we also appear to treat primary deviations such as poststroke knee impairments, which is novel and complementary to current concepts in rehabilitation. This new approach is promising and merits further investigation.

Keywords

stroke, rehabilitation, gait, center of mass, energy consumption

Introduction

Hemiparetic gait following stroke is a leading cause of disability in adults and requires up to twice the metabolic energy of healthy gait.^{1,2} Elevated energy demands are of particular concern, especially in elderly individuals, because they promote activity intolerance with lower walking speed and a sedentary lifestyle that leads to physical deconditioning. This, in turn, compromises the patients' capacity to meet the energy-demanding gait, thus increasing the risk of cardiovascular disease and restraining social participation.^{1,3}

Normal walking seems easy because it costs less than 50% of the maximal aerobic capacity and does not require anaerobic activity. A hemiparetic gait, however, draws on 75% of the maximal oxygen capacity, leaving little in reserve.^{1,3} To this end, a physical conditioning program can increase aerobic capacity, but decreasing the walking energy cost is quite challenging because it represents the ambulation task as such and is directly related to gait impairments.¹ Despite

considerable advances in treatments that have aimed at improving ambulation function in hemiparetic patients, the energy cost did not seem to decrease more than 10% to 15%.^{4–8} Accordingly, effective and cost-efficient interventions that more specifically reduce energy costs are of the utmost need.⁹

The walking energy costs depend on muscle work requirements and the efficiency of muscle work production.^{10,11} The increased energy cost in hemiparetic gait seems to be primarily due to substantial muscle work provided by the nonparetic lower limb to excessively lift the body's center of mass (CM) against gravity.² This excessive vertical CM

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Table 1. Patients Characteristics at Time of Baseline Study Before Treatment

| Patient Number | Gender | Paresis Side | Time Since Injury at Entry (months) | Age (years:months) | Height (m) | SIAS | Treadmill Speed (km h ⁻¹) |
|----------------|--------|--------------|-------------------------------------|----------------------|------------------------|---------------------------|---------------------------------------|
| 1 | Female | Right | 120 | 42:5 | 1.6 | 47 | 1.5 |
| 2 | Female | Right | 111 | 65:7 | 1.6 | 55 | 2 |
| 3 | Male | Left | 285 | 58:1 | 1.75 | 56 | 3 |
| 4 | Female | Left | 116 | 47:5 | 1.59 | 57 | 3.3 |
| 5 | Male | Right | 276 | 32 | 1.88 | 67 | 3.5 |
| 6 | Female | Right | 48 | 35:4 | 1.69 | 64 | 3.7 |
| Mean | | | 159 ± 98 ^a | 47 ± 13 ^a | 1.7 ± 0.1 ^a | 56.5 (47-67) ^b | 2.8 ± 0.9 ^a |

Abbreviation: SIAS, Stroke Impairment Assessment Set.

^aValues are mean ± standard deviation.

^bValues are median (range).

bobbing, up to 3 times more than normal, is likely a compensatory strategy to clear the stiff paretic limb in swing.^{2,12,13} Similar excessive bobbing simulated by healthy humans when provided with their CM biofeedback also increases energy demands.^{14,15} In normal walking, some typical features of lower limb kinematics result in a normal intermediate vertical CM displacement.^{16,17} Although healthy subjects can modulate their CM displacement when provided with the CM biofeedback, they consume the least energy by naturally adopting an intermediate strategy between excessive CM bobbing akin to patients with stroke and extreme flat walking with little bobbing akin to a waiter carrying a bowl of soup.^{14,18}

Recent developments promoted the use of biofeedback in neurorehabilitation to provide patients with sensorimotor impairments a means to better assess and possibly learn self-control of their abnormal physiological responses.¹⁹ Thus, we tested a new rehabilitation strategy to reduce the walking energy costs in hemiparetic patients by helping them to actively reduce excessive vertical CM displacement through biofeedback.

Methods

Thirteen chronic patients with stroke were initially screened. Inclusion criteria for patients were chronic hemiparesis at least 6 months after injury, an increased vertical CM displacement, and the ability to walk on a treadmill without any aid at minimum 1 km h⁻¹. Excluded were patients with additional neurological or orthopedic impairments affecting ambulation, cardiovascular problems that precluded a training program, and severe cognitive deficits that would impede following instructions. Six patients were eventually recruited for this pilot/feasibility study (2 men and 4 women, aged 47 ± 13 years, 159 ± 98 months poststroke [mean ± SD]; see Table 1). Their neurological impairments were assessed using the Stroke Impairment Assessment Set.²⁰ All patients had completed conventional rehabilitation and were

considered “plateaued” in their recovery. This study was approved by the local ethics committee, and all patients provided written informed consent.

Gait Training

Each patient had 18 training sessions (3 times per week for 6 weeks), during which the patients were provided with real-time visual feedback to help them modulate their CM displacement as they walked on a motorized treadmill. A marker over the mid-sacrum representing their CM was videotaped from a posterior view and projected onto a screen projector in front of them according to a recent study in which we validated this biofeedback procedure (Figure 1).¹⁴ The only instruction given was to decrease the marker's vertical displacement. All patients wore a safety harness, but body weight was not supported.

The training sessions comprised 30-minute walking at comfortable speed (3 trials of 10 minutes each). Five-minute rest periods were given every 10 minutes or when the patient needed to stop. The walking period increased approximately 5 minutes every 2 weeks as tolerated to achieve, by the end, about 40 to 45 minutes of walking with rest when necessary. We continuously verified that the patients were really decreasing their vertical CM displacement by computing this displacement from ground reaction forces.

Gait Analysis

Gait analyses were performed in similar conditions before and after gait training. Both pretraining and posttraining gait analyses were performed at the same walking speed (ie, the comfortable treadmill speed for the patient at baseline; see Table 1) to rule out any speed effect on the outcome measures. Before gait analysis, the subjects walked on the treadmill at least 10 minutes in a practice session to become accustomed to treadmill and testing procedures. After a rest

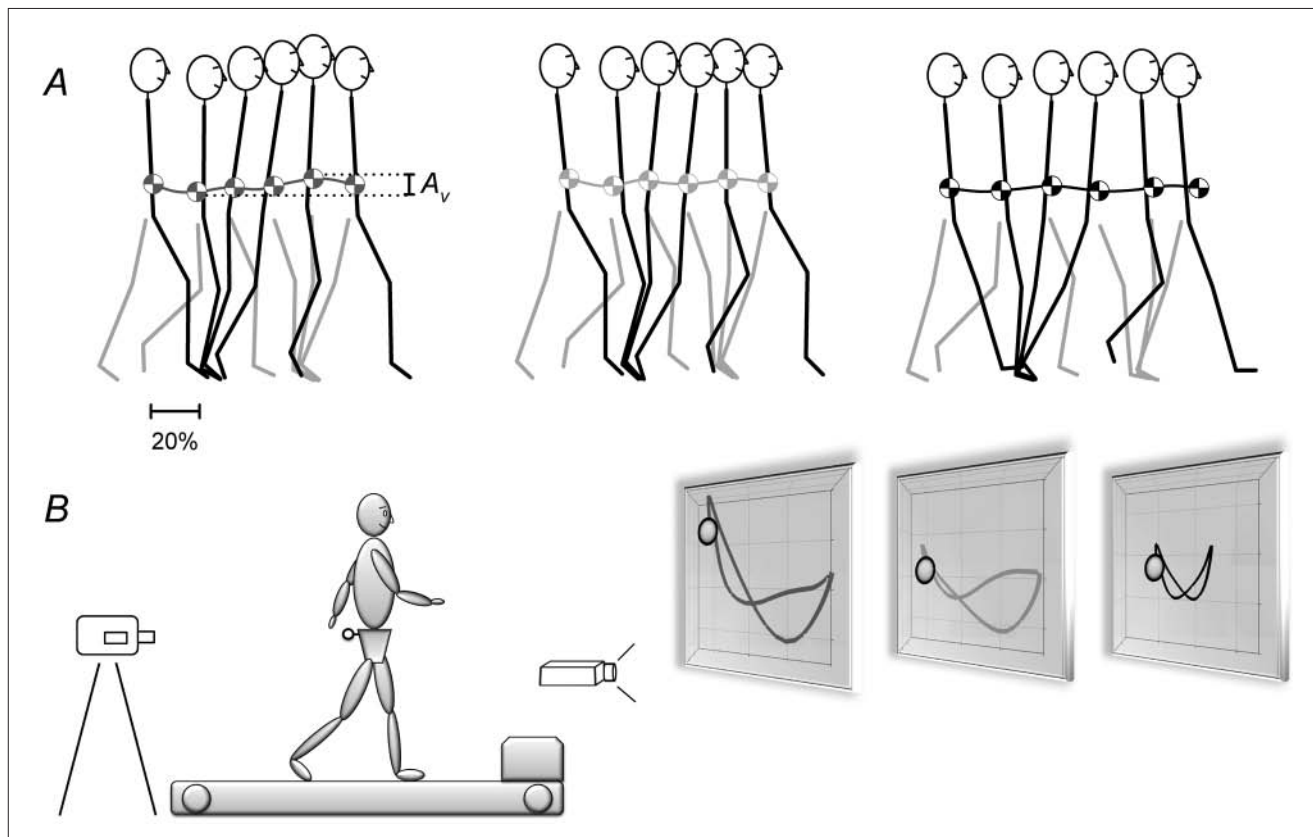


Figure 1. Gait training with biofeedback of the body's center of mass (CM). (A) The segments' positions (paretic limb in thick lines) are shown every 20% of a walking stride (ie, between successive paretic foot contacts) for a male patient with right hemiparesis. The patient walked naturally (left figure), with little vertical CM displacement (middle figure) versus a healthy walking subject (right figure). The hour glass symbol depicts the CM. The left figure shows the excessive CM displacement (dark gray curve), particularly when the paretic limb is in swing. Dotted lines indicate the vertical CM amplitude A_v . The middle figure shows the patient reducing his vertical CM displacement through the biofeedback (light gray curve). Note the smooth and symmetric head bobbing and the increase in paretic knee flexion in swing. (B) A sacral marker representing the CM is videotaped and projected onto a screen while being magnified 10 times. From left to right are depicted the marker projection for the male patient over a walking stride. On the screen, the marker shows simultaneous lateral displacement toward each supporting leg while bobbing up over this leg, hence the 8 or U-shaped figure. In the left screen, the marker rises excessively particularly over the left nonparetic limb in stance, thus distorting the figure 8. In the middle screen, the patient was asked to reduce the vertical marker displacement. Note the more symmetric vertical displacement between each step, thus getting closer to that in normal walking on the far-right screen.

period, gait analysis was performed as described in detail by Massaad et al.¹⁴ We simultaneously measured metabolic energy consumption, electromyogram (EMG) activity, 3D kinematics, and ground reaction forces while the patient walked on a force-measuring treadmill²¹ without the CM biofeedback.

Three-dimensional kinematics analysis was performed with 6 infrared cameras at 100 Hz (BTS, Milan, Italy) that measured the coordinates of 20 reflective markers positioned on specific anatomical landmarks to compute angular displacements. The vertical CM displacement was calculated from ground reaction forces recorded by the force-measuring treadmill at 100 Hz and digitized synchronously with the Elite system. Indeed, the vertical acceleration of the CM was

computed from the vertical components of the ground reaction forces and the mass of the subject. A double mathematical integration of the vertical CM acceleration was then performed to determine the vertical CM displacement.²² We measured the amplitude of the vertical CM displacement (A_v , in m) as the peak-to-peak amplitude on the vertical CM displacement curve over a walking stride (ie, beginning and ending with paretic foot contact; see Figure 1). We also measured the peak-to-peak amplitude during the paretic step ($A_{v \text{ Paretic Limb}}$; from paretic foot contact to nonparetic foot contact) and the peak-to-peak amplitude during the normal step ($A_{v \text{ Nonparetic Limb}}$; from nonparetic foot contact to paretic foot contact). The metabolic cost of walking was determined from oxygen consumption and carbon

dioxide production with an ergospirometer (Quark b²; Cosmed, Rome, Italy). The net metabolic cost of walking (C_{net} in $\text{J kg}^{-1} \text{m}^{-1}$) was calculated by dividing the energy expended above the resting value (ie, walking energy consumption for at least 3 minutes of steady metabolic state minus that while standing) by the walking speed. Simultaneously, we recorded the EMG activity of the vastus lateralis, biceps femoris, tibialis anterior, and medial gastrocnemius muscles in both lower limbs (BTS, Milan, Italy). The EMG signal was digitized at 1000 Hz, filtered (bandwidth 25-300 Hz), and full-wave rectified. The onset and cessation of muscle activity were both visually²³ and mathematically determined by computing the EMG threshold voltage as described in detail by Van Boxtel et al.²⁴ By combining the visual and mathematical methods, we obtained the EMG packets (onset to cessation) for each muscle. The strides were normalized to 100% in time before averaging the EMG activity of each muscle. Finally, the co-contraction index between the main antagonistic thigh muscles (biceps femoris and vastus lateralis) and between the shank muscles (tibialis anterior and medial gastrocnemius) was temporally quantified as the percentage of the walking stride during which these antagonistic muscles were simultaneously activated.²⁵

Statistics

For each gait analysis, data obtained during 10 strides for each variable were averaged, and the mean values were used for statistical analysis. For the global variable measured without taking into account the paretic/nonparetic side (C_{net}), the effect of training was tested with a paired *t* test after normality assumption was verified. For the variables measured in the paretic and nonparetic limbs, the effect of training was tested with a 2-way repeated measures analysis of variance with 2 factors (treatment and affected side) and Tukey's post hoc tests where appropriate (SigmaStat version 3.5, SPSS). The assumption of homoscedasticity was always verified. The significance level α was set at .05 for all the comparisons.

Results

Training sessions were well tolerated with no adverse experiences. During training sessions with biofeedback, the patients were able to decrease the amplitude of the vertical CM displacement A_v from 0.045 ± 0.01 to 0.033 ± 0.01 m, which is 30% less (Figure 1). After gait training, the gait analysis performed without biofeedback revealed that A_v significantly decreased by 10% from 0.045 ± 0.01 to 0.04 ± 0.01 m ($P = .005$; Figure 2). Post hoc tests revealed that this decrease was primarily related to a significant decrease in the $A_{v \text{ Nonparetic Limb}}$ from 0.039 ± 0.01 to 0.035 ± 0.01 m

($P = .016$; Table 2). The $A_{v \text{ Paretic Limb}}$ (which was already closer to normal amplitude [0.021 ± 0.004 m] reported for healthy subjects¹⁴) also tended to decrease but not significantly from 0.030 ± 0.01 to 0.027 ± 0.01 m ($P = .074$). We therefore analyzed the changes in the main lower limb kinematics that may influence the CM^{16,17} when the CM reached its maximum and minimum with the nonparetic limb in stance. The main significant change was a 47% increase in paretic knee flexion in swing when the CM reached its maximum with the nonparetic limb in stance ($21 \pm 15^\circ$ to $31 \pm 14^\circ$, $P = .008$; see Figure 3 and Table 2).

The decrease in A_v was associated with a significant 30% decrease in the net energy cost, C_{net} , from 3.86 ± 1.7 to $2.66 \pm 1.0 \text{ J kg}^{-1} \text{m}^{-1}$ ($P = .009$; Figure 2). In parallel, muscle co-contraction in thigh muscles decreased significantly by 15% in the nonparetic limb and 10% in the paretic limb (Table 2).

Discussion

The concept of this intervention emerged from previous findings that the increased energy cost in hemiparetic gait would mainly be due to substantial work requirements provided particularly by the nonparetic limb muscles to excessively lift the CM against gravity.² In other words, the overuse of the nonparetic limb to walk would underpin the excessive energy consumption in patients with stroke. Research involving normal walking, however, revealed that healthy human subjects can modulate their CM displacement when provided with their CM biofeedback and that they naturally adopt an intermediate strategy between excessive CM bobbing and extreme flat walking that minimizes energy consumption.¹⁴ Hence, in this pilot study, we tested if hemiparetic patients would be able to decrease their energy consumption by practicing a gait pattern with less vertical CM displacement as in normal walking. Indeed, after about a 10-hour training program with CM biofeedback, the vertical CM displacement decreased by 10%, which was associated with a marked 30% decrease in walking energy cost. Concomitantly, paretic knee flexion in swing improved and the EMG co-contraction activity decreased particularly in the thigh muscles.

Gait rehabilitation after stroke has witnessed dramatic changes over the past years. Based on pioneering animal studies²⁶ and increasing evidence that task-repetitive training can induce adaptive neuroplasticity,^{27,28} treadmill training has received widespread attention as a promising new rehabilitation technique combined with or without body weight support.^{5,29,30} Indeed, treadmill training has been shown to be effective in improving ambulatory function. It has also been shown to increase maximal aerobic capacity and decrease energy consumption by 10% to 15% after 6 months of training.^{4,5} It is still argued, however, that as many

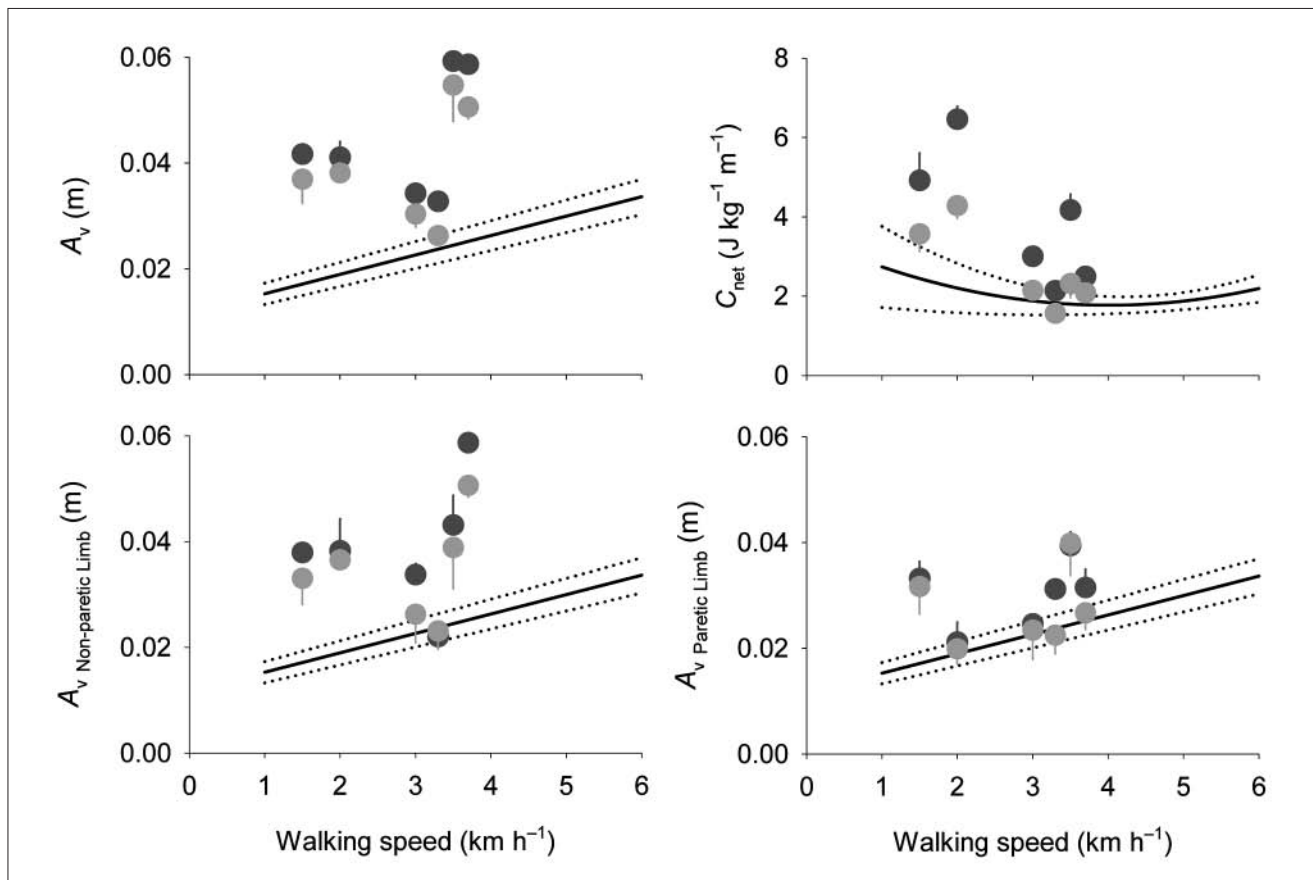


Figure 2. Mechanics and energetics of hemiparetic gait after training. The figure depicts the amplitude of vertical center of mass (CM) displacement over a walking stride (A_v), the net energy cost (C_{net}), the amplitude of vertical CM displacement when the nonparetic limb is in stance phase ($A_{v \text{ Nonparetic Limb}}$), and that when the paretic limb is in stance ($A_{v \text{ Paretic Limb}}$) versus walking speed for the patients before (dark gray symbols) and after gait training (light gray symbols). Symbols represent means \pm standard deviations of 10 walking strides for each patient. The results are compared with normal walking data (solid line with dotted area [mean \pm standard deviation]).

hemiparetic patients are unable to volitionally activate their muscles properly, they may continue repetitively reinforcing some abnormal movements during treadmill walking particularly in swing phase although some stance phase training opportunities were reported.³¹ In contrast, some gait training methods have emphasized the restoration of normal CM displacement because it was believed to strongly affect the sensory experience necessary for achieving optimal training.^{30,32} These techniques controlled the CM movement passively either by changing the harness stiffness in treadmill training³² or in the Gait Trainer,³⁰ which is, however, quite different from our approach in which the patients actively reduced their own CM displacement particularly the vertical one.

Energetics and Mechanics

Indeed, an efficacious intervention that induces a motor skill acquisition requires active practice of close-to-normal

movements, task specificity, intensive practice (repetition), and focused attention.^{12,27,33} Our new rehabilitation approach likely achieves these prerequisites, as it was executed on treadmill (task specific and repetition) with active control and focused attention to reach normal CM displacement representing the whole body movement. This active control of the vertical CM displacement being the main possible cause of the increased energy consumption would have probably helped decrease energy consumption further, as the latter is directly related to walking performance.¹ Indeed, a remarkable 30% decrease in the energy cost was present after only 6 weeks of gait training. This decrease is rapid and unusual when compared with the around 10% to 15% decrease commonly reported after 6 months of treadmill training.^{4,5} Interestingly, a follow-up of our patients 6 months after the training ended revealed that the decrease in the energy cost is still sustained by 15% with respect to pretraining gait analysis (Figure 4). This would indicate a long-term effect and likely a sustained motor learning.²⁸ Recent advances in gait

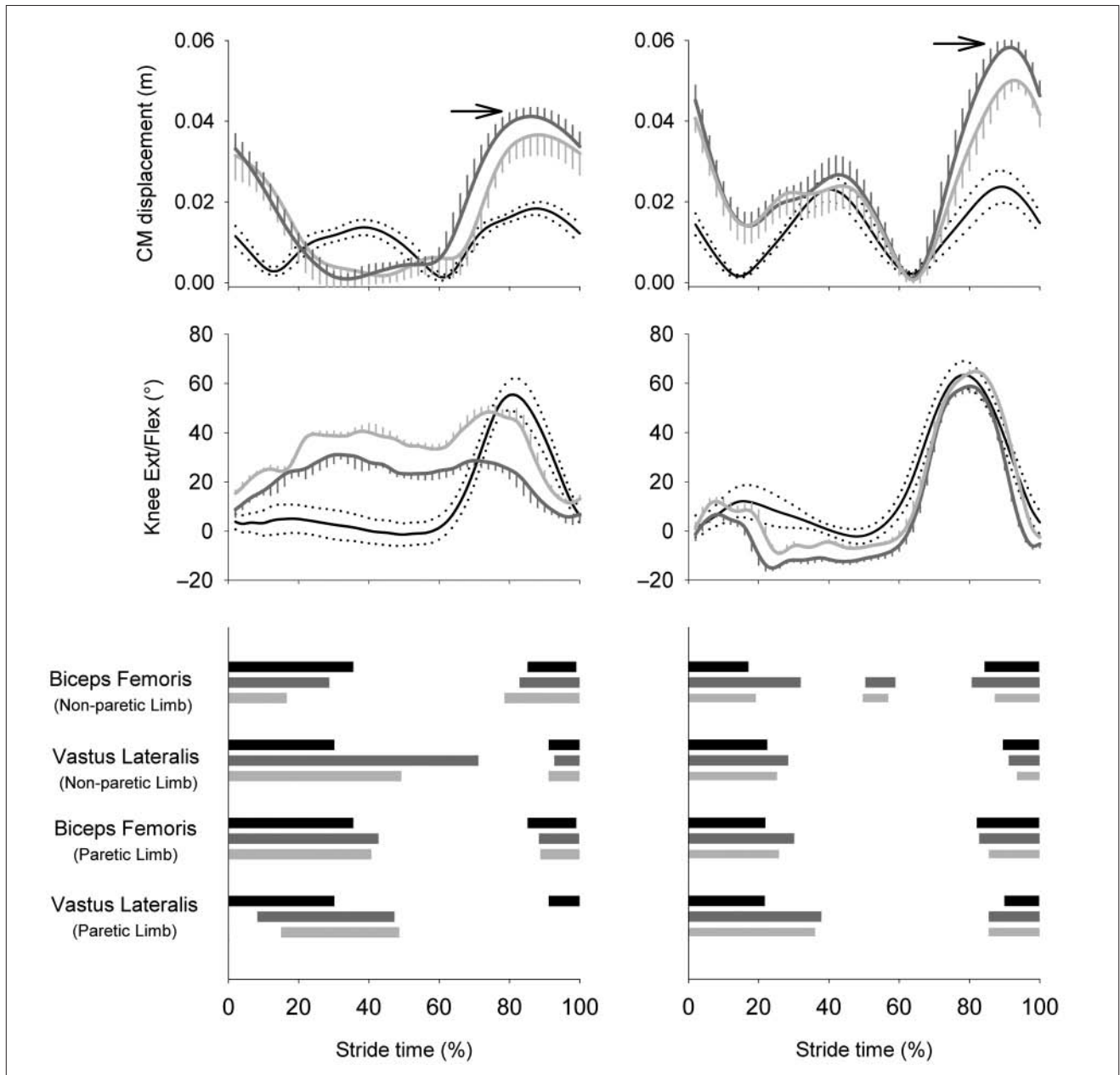


Figure 3. Body's center of mass (CM), kinematics, and electromyogram (EMG) activity after training. Typical traces of the vertical CM displacement (amplitude in m), sagittal plane knee kinematics (positive and negative values indicate respectively flexion [flex] and extension [ext]), and EMG activity of antagonistic thigh muscles in both limbs pretraining (dark gray symbols) versus posttraining (light gray symbols) as a function of the percentage of a walking stride (beginning and ending with paretic foot contact). The traces are compared with normal walking data (black symbols). The left column shows a female with right hemiparesis walking at 1.5 km h⁻¹. After training, the CM displacement decreased by 10%, particularly when the CM reached its maximum while the paretic limb is in swing (arrow). At this moment, knee flexion increased by 60% from 17° to 27°, which likely facilitated limb clearance at this moment. EMG co-contraction timing also decreased. The right column shows another female patient with right hemiparesis walking at 3.7 km h⁻¹. The CM was almost double the normal value, particularly during paretic limb swing, and decreased by 1 cm (15%) after treatment. Knee flexion increased by 10% to reach normal values, and muscle co-contraction decreased.

rehabilitation using high doses of botulinum toxin injections and sophisticated orthoses have proven to be effective in enhancing impairments and locomotion ability in patients

with stroke and have also been able to decrease the energy cost by 10% to 15%.⁶⁻⁸ This would encourage combining these latter interventions with our approach that aims more

Table 2. Vertical CM Displacement, Lower Limb Kinematics, and EMG Activity Before and After Treatment^a

| | Kinematics at Maximum CM Position With Nonparetic Limb in Stance | | | | EMG Activity ^b | | |
|--------------------------|---|-----------------------|----------------------|------------------------|---------------------------|--|--|
| | CM, A _v (m) | Ankle Flex/Ext (°) | Knee Flex/Ext (°) | Pelvis Rotation (°) | Pelvis Tilt (°) | Muscle Co-Contraction in Shank (%) | Muscle Co-Contraction in Thigh (%) |
| Paretic limb | | | | | | | |
| Pre | 0.030 ± 0.01 | -3 ± 7 | 21 ± 15 | -8 ± 8 | -0.1 ± 5 | 21 ± 16 | 44 ± 9 |
| Post | 0.027 ± 0.01 | 1 ± 8 | 31 ± 14 | -8 ± 6 | 3 ± 3 | 18 ± 17 | 39 ± 13 |
| P ^c | .074 | .097 | .008 | .726 | .214 | .516 | .026 |
| Nonparetic limb | | | | | | | |
| Pre | 0.039 ± 0.01 | 11 ± 3 | 7 ± 9 | 7 ± 7 | 0.3 ± 6 | 27 ± 21 | 40 ± 7 |
| Post | 0.035 ± 0.01 | 11 ± 2 | 9 ± 11 | 9 ± 5 | -2 ± 3 | 21 ± 9 | 34 ± 10 |
| P ^c | .016 | .972 | .394 | .478 | .295 | .249 | .012 |
| ANOVA^d | | | | | | | |
| P | .005 | .27 | .01 | .441 | .503 | .24 | .018 |

Abbreviations: CM, center of mass; EMG, electromyogram.

^aValues are mean ± standard deviation.

^bEMG activity is expressed as a percentage of stride time.

^cP values for Tukey's post hoc multiple comparisons between pretreatment and posttreatment within paretic and nonparetic limbs. Significant P values are in boldface.

^dANOVA P values are shown in the last row.

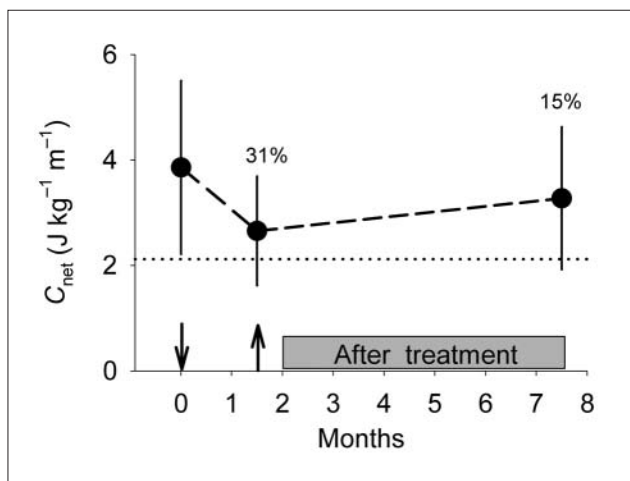


Figure 4. Follow-up of the energy cost. Net energy cost for the 6 patients (mean ± standard deviation) at baseline (downward arrow), at the end of training (upward arrow), and 6 months later at follow-up. Horizontal dotted line indicates normal walking data. The figure above each symbol shows the percentage of change compared with the baseline value.

specifically at decreasing the gait energy cost to probably decrease this energy cost further.

Previous research pointed at the excessive positive mechanical work provided by muscles as the main possible cause of the elevated energy demands in hemiparetic gait where the CM bobs up and down excessively. Indeed, both energy cost and muscle mechanical work show a

similar pattern of increase and reach twice the normal values particularly at the lowest walking speeds.² Excessive CM bobbing simulated by healthy humans similarly increases energy cost due to extra muscle work.¹⁴ The total positive mechanical work performed by muscles was classically estimated as the sum of the external work necessary to move the CM relative to the surroundings and the internal work to move body segments relative to the CM.^{10,11} To explain the decrease in energy cost in our results, we tried to estimate this total mechanical muscle work in our patients. This muscle work appeared to decrease by 10% after training (from 0.76 ± 0.2 to 0.68 ± 0.1 J kg⁻¹ m⁻¹), which may explain in part the decrease we see in the energy cost and is similar to the improvement in muscle work we recently observed in hemiparetic patients with stiff knee impairments after botulinum toxin injections.⁸ However, other variables may also account for the reduction in energy cost in our patients, especially considering that recent methodological considerations to estimate the muscle mechanical work showed that relevant muscle work is also needed to redirect the CM from one circular arc to the next during the transition between steps.³⁴⁻³⁶ This work performed with one leg pushing against the other during the double-support phase was ignored by the classic measurements of the positive external and internal muscle work done during walking,^{10,11} yet it could have relevant energetic consequences in normal gait.³⁴⁻³⁸ Other authors, however, argued that a part of this step-to-step transition work could be realized passively so that it would only account for maximum 10% of the total mechanical muscle work.³⁹ Nonetheless, this step-to-step transition work is not yet well

investigated in pathological gaits where it could likely be particularly relevant as the excessive CM bobbing would require an increased amount of work of redirection.⁴⁰ Taking this additional work into account would require measuring the forces under each foot separately on different force plates during the double-support phase. This is not possible when the patients are assessed on a force-measuring treadmill as in our experiment because both feet are on the same belt during double support. Therefore, further investigation of this mechanical muscle work in hemiparetic patients could shed further light on the reasons of the decrease in energy cost seen in our patients after training.

Knee Flexion and EMG Activity

The reduction in vertical CM displacement was primarily related to a decrease in the vertical amplitude during the stance phase of the nonparetic limb, which showed the greatest deviation from normal at baseline. This decrease was associated with a 47% increase in the paretic knee flexion in swing, which likely by facilitating the ground clearance reduced the compensatory increase in CM displacement necessary to avoid dragging the floor with the paretic foot.^{12,13} Knee flexion during swing resisted improvement in treadmill walking alone even when the latter was associated with body weight support, handrail hold, increased treadmill speed, or different stiffness in the harness support.³² This would indicate that the increase in knee flexion we found was likely not simply related to treadmill training as such. An increase in knee flexion was, however, reported after weight-supported treadmill training was combined with functional neuromuscular stimulation of main lower limb muscles.¹² Knee flexion also increased in treatments that specifically addressed the stiff knee impairments by botulinum toxin injections.^{7,8} This would also encourage researchers to test the association of the former interventions with our approach for a possible further improvement in stiff knee impairments.

The increase in muscle co-contraction has been suggested to compensate for impaired postural control, but excessive co-activation may also be associated with energy waste, especially when it concerns large muscle groups such as those at the knee and hip joints.⁴¹ The decrease in muscle co-contraction in our patients could be due to the practice of a more normal gait pattern, which would have optimized the sensory inputs that are important in facilitating spinal and supraspinal locomotor networks.^{33,42} A part of this decrease could be explained by the treadmill training that appears to produce significant improvements in strength and spastic reflex in the paretic limb.^{4,9} The marked decrease in co-contraction of the nonparetic limb is, therefore, probably explained in part by additional factors. The increased co-contraction in thigh muscles reflects rather a compensatory

activity to optimize the gait pattern in the presence of muscular weakness and reduced coordinative control.⁴³ This may indicate that this compensatory activity was probably less necessary after treatment, which, interestingly, parallels the decrease in the vertical CM displacement, particularly when the nonparetic limb was in stance.

Biofeedback Approach

Our approach is defined as dynamic task-oriented biofeedback, which suggests that the feedback is delivered during functional movement.¹⁹ An effective biofeedback therapy requires motivating cues to keep the subject attentive as well as of multiple variables that characterize the task performance without overwhelming a patient's cognitive ability. An information fusion approach is, therefore, necessary to avoid information overload.¹⁹ Our patients showed a marked motivation during training, and our study revealed that the point representing the fusion of all body mass would be the most suitable to give this unique and simple information, which may also inspire the use of biofeedback for other tasks. This simple information becomes more relevant when considering the extraordinary complexity of human locomotion. Contrary to diverse pathological gaits, however, similar patterns of CM movement are usually found.^{44,45} Therefore, the concept that fundamentally locomotion is the translation of the CM through space along a pathway requiring the least energy^{14,16} may extend the application of our method to other pathological gaits. In addition, the cost-benefits of our simple approach using a standard camera and projector may ultimately help facilitate its adoption into many rehabilitation centers.

Finally, although the only instruction to the patients was to decrease their CM bobbing, both global and segmental variables changed. Actually, by acting on the compensatory strategy (ie, the CM bobbing), we treated the primary gait impairments such as the stiff knee. To our knowledge, this is novel and complementary to common paradigms in rehabilitation that treat the primary deviations. Furthermore, as our perception of the patients' limp, the most evident symptom, is actually an expression of their abnormal and irregular CM bobbing,⁴⁵ we can say that, by handling the limp, we may manage its cause. The limp as such also appears to account for a relevant part of the patients' excessive energy consumption.

Limitations of This Pilot Study and Future Direction

We deliberately enrolled patients in our pilot/feasibility study that walk independently to eliminate any assistance effect. Our approach still needs to be verified in more-impaired patients with stroke who need gait assistance or body weight support. In addition, training with CM biofeedback was inherently associated with treadmill training

in our study, thus making it difficult to see which part of the improvement was due to the biofeedback as such. However, by comparing the main changes we found with that reported for treadmill training in the literature, we found that the present changes in our outcome variables seem much larger to be only explained by treadmill training alone. Nonetheless, as in every new approach, our pilot approach still needs further investigation in randomized controlled trials with a larger group of patients and combined with other interventions to be definitely validated in stroke rehabilitation.

In conclusion, training hemiparetic patients to walk with active practice of little vertical CM displacement decreased their energy consumption markedly. The CM seems to be a pathophysiological determinant of increased energy cost in patients with stroke and a trigger point on which to act. Our pilot study merits further investigation, but it also illustrates a typical example of how concepts from basic sciences, such as gait analysis, can elaborate on new rehabilitation approaches in clinical trials.

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Declaration of Conflicting Interests

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