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Energy cost, mechanical work, and efficiency of hemiparetic walking

C. Detrembleur a,*, F. Dierick a, G. Stoquart b, F. Chantraine b, T. Lejeune b

a Rehabilitation and Physical Medicine Unit, Université catholique de Louvain, Tour Pasteur 5375, Avenue Muontier 53, B-1200 Brussels, Belgium
b Department of Physical Medicine and Rehabilitation, Cliniques universitaires Saint-Luc, Université catholique de Louvain, Brussels, Belgium

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Abstract

The energy cost of walking (C) in nine chronic hemiparetic patients was calculated by measuring the total mechanical work (Wtot) done by the muscles and the efficiency of this work production (η). The energy cost was twice normal in slow walkers and 1.3 times greater in fast walkers. The increase in C was proportional to the increase in Wtot and η was normal at around 20%, despite an increase in muscle tone and muscle co-contractions. This type of approach gives a greater understanding into how segmental impairments increase Wtot and C and contribute to a patient’s disability.

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1. Introduction

In hemiparetic walking, many authors [1–4] have reported an increase in the metabolic energy cost related to the neurological impairments [1,2]. In classical exercise physiology, the metabolic energy expenditure is determined by the workload and the efficiency of work production [5]. The increase in the energy cost (C) of hemiparetic walking must then be related either to an increase in the total mechanical work done by the muscles (Wtot) or to a decrease in the efficiency of positive work production by the muscles (η). Wtot could increase as a result of kinematic impairments or an increase in step frequency [6]; or η could decrease as a result of excessive muscle co-contraction or an increase in muscle tone [7]. However, until now, neither the Wtot nor the η have been measured in pathological walking.

Several different methods have been described to measure the total mechanical work during walking. The technique described by Cavagna et al. [8] was selected because it has numerous methodological advantages [9]. Previous studies have demonstrated the strengths of this method, including its ability to explain why there is an optimal walking speed at which the energy cost is minimal [10], why there is an optimal step frequency for each walking speed [11], the effect of gravity [12], the maturation of the pendular mechanism of walking during growth [13], the difficulty of walking on sand [14] and how African women carry loads with ease [15]. However, despite its strengths, Cavagna et al.’s method has not been applied to any extent to pathological gait. Thys et al. [16] studied the mechanics and energetics of crutch gait, but in normal subjects. Only the energy changes of the body center of mass (COMB) have been studied in hemiparetic walking [17,18] and in lower limb amputees [19,20].

Classical gait analysis only focuses on segmental abnormalities such as abnormal patterns of muscle activation, joint motion or joint moment. The assessment of these segmental abnormalities is very useful in clinical practice to help in therapeutic decisions but provides little information on the walking patient’s disability such as energy cost. It is possible to study the repercussions of these segmental abnormalities on locomotor function using mechanical work and energy cost measurements. These mechanical and metabolic measurements could be more relevant to the walking patient’s disability and also be very useful in clinical research. By studying the energy changes of the COMB it is also possible to gain further insight into the mechan-
isms of pathological walking and whether or not the normal pendulum mechanism is preserved in hemiparetic walking.

The aim of the present study was to measure the energy cost of walking among post-stroke chronic hemiparetic patients, and to understand the increase in the energy cost by measuring the total positive mechanical work done and the efficiency of positive work production. Moreover, we investigated the usefulness and feasibility of these simultaneous mechanical and energetic analyses during pathological walking.

2. Materials and methods

2.1. Study population

Nine chronic hemiparetic post-stroke patients (5 men and 4 women) were recruited from our outpatient rehabilitation unit between April and June 2001; their main anthropometric and historic data are presented in Table 1. The patients were able to walk independently irrespective of the walking surface, and scored 5/5 for the Functional Ambulation Classification scale (FAC) [21]. They were also able to walk on a treadmill for sufficient time to complete a metabolic analysis and had no other major medical disorders. In the acute phase, all patients had been treated in a stroke unit and, thereafter, following an intensive rehabilitation program. They were still having physiotherapy twice a week to maintain their functional capacities. All gave informed consent before participating in the study.

2.2. Clinical examination

The same physician performed the clinical examination for all patients. The passive joint range of motion (ROM) of the hip, the knee, and the ankle was measured by goniometry. The muscle strength of the hip extensors and flexors, the knee extensors and flexors, and the ankle plantar and dorsal flexors was graded according to the Medical Research Council (MRC) criteria [22]. Muscle tone was assessed by the modified Ashworth scale. Tendon reflexes were assessed by the deep tendon reflex scale [23]. Impairment was evaluated by the Stroke Impairment Assessment Scale (SIAS) [24] and the functional capacity by the Functional Independence Measurement (FIM) [25].

2.3. Gait analysis

Gait was assessed by three-dimensional (3D) analysis, including synchronous kinematic, dynamic, and electromyographic (EMG) recordings. Patients were instructed to walk alone without any device at their comfortable speed in the measurement field (10 m long).

Foot-switch soles (Elite V5, BTS, Italy) were attached under the patients’ feet and data recorded at a sampling rate of 1000 Hz. These data were necessary to compute the global temporal parameters—walking speed, step length, and step frequency that were used in the analysis of all gait variables. Segmental kinematics were measured with the Elite system V5 (BTS, Italy) at 50 Hz. Four CCD infrared cameras measured the co-ordinates, in the 3 spatial planes, of 10 reflective markers positioned on specific anatomical landmarks to compute the angular displacements of pelvis, hip, knee, and ankle based on Euler angles and Newtonian mechanics [26].

The muscle electrical activity of rectus femoris (RF), biceps femoris (BF), tibialis anterior (TA), and lateral gastrocnemius (LG), was recorded by a telemetry EMG system (Telemg, BTS, Italy) with surface electrodes (Medi-Trace, Graphic Controls Corporation, NY, USA). The signal was digitized at 1000 Hz, full-wave rectified, and filtered (bandwidth 25–300 Hz). The onset and cessation of muscle activity were both visually and mathematically determined by computing the EMG threshold voltage as described by Van Boxtel et al. [27]. The EMG activity of each muscle was normalized to 100% in time of gait cycle before averaging. The co-contractions between TA and LG muscles (TA–LG), and between RF and BF muscles (RF–BF) were temporally quantified as the percentage of the gait cycle during which the antagonistic muscles were co-activated.

The total positive mechanical work ($W_{\text{int}}$) done by the muscles during walking was divided into the external work ($W_{\text{ext}}$) performed to move the COMb, relative to the surroundings, and the internal work ($W_{\text{int}}$) performed to move the body segments relative to the COMb [9].

$W_{\text{int}}$ was computed from kinematic data following the method described by Willems et al. [9]. The body was divided into seven rigid segments: head–arm–trunk (HAT), thighs, shanks and feet. The internal mechanical energy of the body segments corresponded to the sum of the rotational and translational energies of these segments due to their movements relative to the COMb. For each lower limb, the internal mechanical energy–time curves of the thigh, shank and foot were summed (Fig. 1). The $W_{\text{int}}$ of each lower limb and the HAT segment were then calculated separately as the sum of the increments of the respective internal mechanical energy curves during one stride. Finally, $W_{\text{int}}$ during walking corresponded to the sum of the $W_{\text{int}}$ done to move the lower limbs and the HAT segment.

$W_{\text{ext}}$ was computed from the measurement of the 3D ground reaction forces (GRFs) (Pharos Systems Inc., MA) at a sampling rate of 50 Hz, following the method described by Cavagna [28] and adapted to pathological walking [19]. The 3D accelerations of the COMb were computed from the 3D GRFs and the mass of the
patient. The mathematical integration of the 3D accelerations gave the 3D speeds of the COM, allowing computation of the COM kinetic energy due to forward ($E_{kf}$), vertical ($E_{kv}$) and lateral ($E_{kl}$) speeds. A second mathematical integration of the vertical speed gave the vertical displacement ($S_v$) of the COM, used to compute the gravitational potential energy ($E_p$) of the COM. The positive work done by the muscles during walking to move the COM in the forward ($W_f$), vertical ($W_v$), and lateral ($W_l$) directions was obtained by adding the increments of the respective energy curves $E_{kf}$, $E_v = E_{kv} + E_p$, and $E_{kl}$. By adding $E_{kf}$, $E_v$, and $E_{kl}$, the total mechanical energy ($E_{tot}$) of COM was obtained. Finally, the $W_{ext}$ during walking was obtained by adding the increments of the $E_{tot}$ curve during one stride. $W_{tot}$ during walking was calculated as the sum of $W_{ext}$ and $W_{int}$.

Thereafter, the metabolic cost of walking was determined by the patient's oxygen consumption ($\dot{V}O_2$) as they walked on a motor driven treadmill (Mercury LT med, HP Cosmos, Germany). Breath by breath $\dot{V}O_2$, carbon dioxide production ($\dot{V}CO_2$) and heart rate (HR) were measured throughout the treadmill test (Quark b2, Cosmed, Italy). Values were automatically converted by a software (Cosmed Quark b2 win, version 5.1a) to standard temperature, pressure and dry $\dot{V}O_2$. The respiratory quotient (RQ) was computed as the ratio between $\dot{V}CO_2$ and $\dot{V}O_2$. Each energy measurement started with a rest period while the subject was standing on the treadmill. Thereafter, the patients walked at a speed equivalent to the mean speed spontaneously chosen during the mechanic trials. Each period was continued until a steady state was reached and maintained for a period of 165 s (315 s ± 107). RQ always remained less than 1. The Joules of energy expended per liter of oxygen consumed were computed depending on RQ [29]. The energy expended at rest was subtracted from the energy expended when walking to obtain the net rate of energy expenditure expressed in J kg⁻¹ min⁻¹. The net rate of energy expenditure was then divided by the speed of walking to obtain the net energy cost of walking ($C$).

The efficiency of positive work production by the muscles was calculated as the ratio between the total positive mechanical work done, $W_{tot}$, and the energy expended, $C$ [10].

3. Results

The patients had the same functional independence, with a median FIM score of 123 (ranging from 118 to 125) (Table 2) as well as the same FAC score for ambulation. However, the walking speed adopted spontaneously varied considerably between the patients, and ranged from 1.7 to 3.6 km h⁻¹. Hence, the patients were
Fig. 1. Typical internal and external mechanical energy, kinematics and electromyographic activity in two patients (#3—left column; #8—right column) are presented as a function of normalized gait stride (%), and 0% is the onset of the stride on the affected side. From top to bottom the curves refer to the mean and SD (n = 5) internal mechanical energy changes obtained in unaffected and affected limbs, and in HAT (head–arm–trunk segment), the external mechanical energy changes of the COM, due to motion in vertical ($E_v$), forward ($E_{kf}$) and lateral ($E_{kl}$) directions and total mechanical energy changes ($E_{tot} = E_v + E_{kf} + E_{kl}$). The following curves refer to mean hip, knee and ankle displacements in the sagittal plane and EMG timing of Tibialis Anterior (TA), Triceps Surae (TS), Rectus Femoris (RF) and Hamstrings (HM) muscles. The normal EMG timing is indicated by white rectangles.
3.1. Typical traces

Fig. 1 presents the typical gait analysis results obtained from two patients walking at slow and fast speeds, respectively. The walking mechanics of patient #8, walking at 3.6 km h\(^{-1}\), were almost normal (right part of Fig. 1). \(W_{\text{int}}\) was mainly done during the swing phase to bring the lower limb from behind to in front of COM\(_b\). The patient used as much \(W_{\text{int}}\) to move the hemiparetic lower limb as the unaffected lower limb. The internal mechanical energy changes of the HAT segment were negligible. \(W_{\text{int}}\) was 0.35 ± 0.02 J kg\(^{-1}\) m\(^{-1}\), 1.3 times greater than the normal value [9]. The \(E_{v}\) curve showed that the COM\(_b\) was lifted up and down during the stride. It reached its highest position during mid stance and its lowest position during the double stance phase. COM\(_b\) reached a higher position on the hemiparetic lower limb than on the unaffected lower limb. The mechanical energy changes on the \(E_{k}\) curve were negligible. The \(E_{v}\) and \(E_{k}\) curves were out of phase, allowing transformations between kinetic and potential energies of the COM\(_b\) like in a pendulum. This explains why the fluctuations of \(E_{tot}\) were smaller than the sum of the fluctuations of \(E_{v}\) and \(E_{k}\), and, consequently, why \(W_{\text{ext}}\) was smaller than the sum of \(W_{v}\) and \(W_{k}\). \(W_{\text{ext}}\) was 0.34 ± 0.03 J kg\(^{-1}\) m\(^{-1}\), 1.3 times greater than the normal value [9]. The amount of mechanical energy recovered (\(R\)) due to passive transformations between the gravitational potential energy and the kinetic energy was calculated according to Cavagna et al. [8].

\[
R = 100 \frac{W_{t} + W_{v} + W_{i} - W_{\text{ext}}}{W_{f} + W_{v} + W_{i}}.
\]

\(R\) was equal to 59\%, corresponding to normal value [9].

The hip, knee, and ankle kinematics were almost normal [30]. EMG showed phasic muscle activity but with significant periods of co-contraction between RF and BF muscles, and between TA and LG muscles.

The walking mechanics of patient #3, walking at 1.9 km h\(^{-1}\), were abnormal (left part of Fig. 1). \(W_{\text{int}}\) was asymmetric and mainly done to move the unaffected lower limb during the swing phase. \(W_{\text{int}}\) was 0.27 ± 0.03 J kg\(^{-1}\) m\(^{-1}\), 1.9 times greater than the normal value [9]. The \(E_{v}\) curve showed that the COM\(_b\) was increased during the single stance phase on the hemiparetic lower limb, and reached its highest position during the following double stance phase. At this walking speed, the mechanical energy changes on \(E_{k}\) and \(E_{k}\) curves are normally negligible. Only a few transformations between kinetic and potential energies were allowed. \(R\) was equal to 28\% of the corresponding normal value at this slow speed. \(W_{\text{ext}}\) was 0.6 ± 0.09 J kg\(^{-1}\) m\(^{-1}\), 1.7 times greater than the normal value [9].

The hip kinematics were almost normal [30]. The knee was maintained in extension during stance phase and there was no ankle plantar flexion at the end of the stance phase. EMG showed prolonged muscles activities with significant periods of co-contraction between RF and BF muscles, and between TA and LG muscles.

### Table 2

<table>
<thead>
<tr>
<th>Clinical examination, gait parameters, joint kinematics, and muscles co-contractions</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n = 5)</td>
</tr>
<tr>
<td>(\text{FIM total score} \pm \text{SD})</td>
</tr>
<tr>
<td>(\text{FIM motor score} \pm \text{SD})</td>
</tr>
<tr>
<td>(\text{Ashworth scale score} \pm \text{SD})</td>
</tr>
<tr>
<td>(\text{Deep tendon reflex scale score} \pm \text{SD})</td>
</tr>
<tr>
<td>(\text{Ankle dorsiflexion knee extended} \pm \text{SD})</td>
</tr>
<tr>
<td>(\text{Ankle dorsiflexion knee flexed} \pm \text{SD})</td>
</tr>
<tr>
<td>(\text{SIAS Score} \pm \text{SD})</td>
</tr>
<tr>
<td>(\text{Gait parameters} \pm \text{SD})</td>
</tr>
<tr>
<td>(\text{Walking speed (km h}^{-1})</td>
</tr>
<tr>
<td>(\text{Cadence (step min}^{-1})</td>
</tr>
<tr>
<td>(\text{Step length (m)} \pm \text{SD})</td>
</tr>
<tr>
<td>(\text{Stance (% stride)} \pm \text{SD})</td>
</tr>
<tr>
<td>(\text{Joint kinematics} \pm \text{SD})</td>
</tr>
<tr>
<td>(\text{Ankle dorsiflexion (stance)} \pm \text{SD})</td>
</tr>
<tr>
<td>(\text{Ankle plantarflexion (toe-off)} \pm \text{SD})</td>
</tr>
<tr>
<td>(\text{Knee flexion (initial contact)} \pm \text{SD})</td>
</tr>
<tr>
<td>(\text{Knee extension (stance)} \pm \text{SD})</td>
</tr>
<tr>
<td>(\text{Knee flexion (swing)} \pm \text{SD})</td>
</tr>
<tr>
<td>(\text{Hil flexion} \pm \text{SD})</td>
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<tr>
<td>(\text{Hil extension} \pm \text{SD})</td>
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<tr>
<td>(\text{Muscles co-contractions (%) stride)} \pm \text{SD})</td>
</tr>
<tr>
<td>(\text{RF–BF})</td>
</tr>
</tbody>
</table>

Mean (±SD) except a, median (quartile).
knee kinematics were heterogeneous because some of the patients walked in knee flexion and other in knee extension irrespective of the walking speed. However, all patients had a decreased knee flexion during the swing phase. The ankle had a normal dorsiflexion during the stance phase, but a decreased plantar flexion at the end of this same phase.

The pattern of muscle activation was perturbed, especially among slow walking patients. In normal walking [31] the muscle activation pattern was phasic, the index of co-contraction of the ankle flexor extensor muscles was around 10%, and that of the knee flexor extensor muscles around 40%. Among our hemiparetic patients, muscle activity periods were prolonged and the co-contraction index was 1.5–6 times greater than normal [31]. The weight-specific positive mechanical work done per unit distance and energy cost were plotted in Fig. 2 as a function of walking speed. The upper left panel shows an important increase in \( W_v \) reaching twice the normal value of that of slow patients. This was related to an increase in the vertical displacement of the COM \( b \) from 2.2 cm in normal walking to 7.0 ± 2 cm in our hemiparetic patients. On the contrary, \( W_t \) was overall normal and \( W_t \) remained small despite an increase in the slow patients. \( R \) was nearly normal, ranging from 25% in the slow patients to 55% in the fast patients (lower left panel). Consequently, the increase in \( W_{\text{ext}} \) decreased with the walking speed, from twice the normal value in the slow patients, to near normal value in the fast patients (upper middle panel). The \( W_{\text{int}} \) was twice normal in the slow patients and slightly increased in the fast patients (upper right panel). On the whole, \( W_{\text{tot}} \) was twice normal in the slow patients and only slightly increased in the fast patients. The same pattern was found for \( C \): a two-fold increase was seen in the slow patients and a slight increase in the fast patients. Consequently, \( \eta \) was almost normal at around 20% [10,32].

4. Discussion

The present study showed that the increased energy cost of hemiparetic walking was related to an increase in the positive mechanical work done by the muscles and not to a decrease in the efficiency of this work production.

4.1. Energy cost

Various methods are available to estimate the energy expenditure during walking. The physiological cost index [33] or Energy Expenditure Index [34], based on HR measurement, are commonly used in gait analysis to estimate the energy expenditure. However, they do not quantify the energy expenditure and their reliability is limited [35]. The classical indirect calorimetric method, based on measurement of oxygen consumption, remains the most reliable and practical method to compute the energy expenditure. Our results confirm that hemiparetic patients expend more energy per unit distance traveled (energy cost) during walking at a given speed than healthy subjects. Bard and Ralston [36] and Bard [1] were the first to describe an increase in the gross energy cost of walking among hemiparetic patients. Among patients with severe or moderate spasticity, the gross energy cost was up to 1.4 times greater than the normal values. But surprisingly, among patients with minimal spasticity, the gross energy cost was less than normal, raising concerns about the methods used. Corcoran et al. [37] showed that, at a given walking speed, the gross energy cost was 1.5–1.7 times greater in hemiparetic patients than in healthy subjects. More recently, and with a more reliable method, Bernardi et al. [38] and Zamparo et al. [3] confirmed the increased gross energy cost of hemiparetic walking. In agreement with the present study, these authors also reported that the energy cost increase was inversely related to the walking speed spontaneously adopted by the patients. Among very slow walking patients, the gross energy cost reached twice the normal value, whereas it was normal or only slightly increased in patients who had a spontaneous speed similar to the healthy subjects (4.8 km h\(^{-1}\)). Therefore, the rate of energy expenditure per unit of time (power) was similar between patients, whatever the spontaneously adopted walking speed (11.7 ± 2 ml kg\(^{-1}\) min\(^{-1}\)). Moreover, the patients’ rate of energy expenditure was equivalent to the rate of energy expenditure of healthy subjects walking at their spontaneously adopted speed (4.8 km h\(^{-1}\))[37]. It seems that our patients decreased their walking speed to maintain their rate of energy expenditure at a level they could sustain for a long time. The same strategy, i.e. decreasing speed of motion to maintain a reasonable power in spite of increasing energy cost, is adopted by hemiparetic patients when climbing stairs [38].

4.2. Total mechanical work

Three methods are available to measure the total mechanical work done by the muscles during walking: (1) analysis of the muscle power around each joint [39] (2) analysis of the energy changes of a finite number of body segments from their movements relative to the ground [30] and (3) analysis of the center of mass of the whole body (COM\(_b\)) relative to the ground and of the body segments relative to the COM\(_b\) [10,40]. While any one of these methods can be used to estimate the total energy level of the body, the third method has several advantages as detailed by Willems et al. [9]. Their method is easy to implement, requiring few assumptions that cause only small errors (that air resistance and
skidding are negligible), and yields particularly simple to interpret, noise-free tracings.

4.3. External mechanical work

The speed dependent increase in $W_{\text{ext}}$, reaching twice the normal values among slow patients, is in agreement with the data reported in the literature [17,18,41]. This increase was principally explained by an increased COM$_b$ vertical displacement and increased $W_v$. Kerri-gan et al. [42] also reported an increase in the COM$_b$ vertical motion, measured by the displacement of the sacrum. It is, however, impossible to compare data because the COM$_b$ vertical displacement was speed dependent and they did not report the walking speed of their hemiparetic patients. Tesio et al. [43] and Zamparo et al. [3] postulated that the pendulum mechanism of walking [8] was maintained in their hemiparetic patients because the ‘U’ shaped relation between the walking speed and the energy cost was preserved. Our results confirm that the amount of mechanical energy recovered through the walking pendulum mechanism, quantified by $R$, was similar to that among healthy subjects at a given speed. However, analysis of the COM$_b$ energy curves showed that the transformations between kinetic and potential energies were limited, especially at slow speeds. Kinetic energy available at slow speed was very limited whereas the potential energy changes were important. Moreover the $E_v$ curve presented a single bump pattern and lost its phase opposition with the $E_{\text{kf}}$ curve. The mirroring effect between $E_v$ and $E_{\text{kf}}$ curves was lost. Consequently, the $E_{\text{tot}}$ curve also showed a single bump pattern. The $W_{\text{ext}}$ was mainly done at the beginning of the stride probably by the unaffected limb. The same asymmetry in $W_{\text{ext}}$ has already been reported in hemiplegic and unilateral hip osteoarthritis patients [17] and in below or above knee amputees [19].

4.4. Internal mechanical work

$W_{\text{int}}$ was twice greater than normal in slow patients and only slightly increased in fast patients. In our study, the $W_{\text{int}}$ done to move the unaffected lower limb ($0.205 \pm 0.03$ J kg$^{-1}$ m$^{-1}$) was significantly greater ($t = 3.2; P = 0.0005$) than the $W_{\text{int}}$ done to move the affected lower limb ($0.166 \pm 0.01$ J kg$^{-1}$ m$^{-1}$) in all patients. This implies that the unaffected lower limb made larger or faster movements than the affected limb. This could be secondary either to increased movement...
amplitude or to increased speed of motion of the lower limb segments because of a shortened swing phase. Until now, $W_{\text{tot}}$ has not been studied in pathological walking, allowing no comparisons with previous studies.

4.5. Efficiency of positive work production

Efficiency of positive work production by muscles among our patients was around 20% and similar to healthy subjects at a given speed. To the authors’ knowledge, there is no previous study where $W_{\text{tot}}$ and $C$ have been simultaneously measured during pathological walking allowing the calculation of $\eta$. The only references for $\eta$ among spastic subjects come from experiments done on a bicycle ergometer by two teams. Lundberg [44–46] measured a lower $\eta$ among young diplegic adults with cerebral palsy than among healthy subjects, but workload and pedaling rates were different. At low workloads ($<25$ W), Lundberg [47] showed a roughly similar $\eta$ among diplegic patients and controls. At a similar workload (0.5 W kg$^{-1}$), Dresen et al. [48] also found a normal $\eta$ among hemiplegic and diplegic children but a decreased $\eta$ among tetraplegic children.

The $\eta$ was surprisingly normal in our study. Indeed, a decreased $\eta$ was expected in hemiparetic spastic walking secondary to (1) significant excessive co-contraction between agonist–antagonist lower limb muscles, moderate to severe spasticity, assessed by the Ashworth scale score; (2) a modification of the histochemical properties of spastic muscle, i.e. the white fibers show atrophy related to the increase in tone [49] and (3) an increased passive stiffness of spastic muscle due to structural changes [50]. Two hypotheses could explain this normal $\eta$. Firstly the unaffected lower limb performed most of the work and secondly, the total work done by the muscles has a greater influence on the energy cost [14] than the tension developed by the muscles [51].

4.6. Clinical relevance

A better understanding of walking pathophysiology would be useful in clinical practice and in particular how segmental impairments increase $W_{\text{tot}}$ and $C$ and lead to a patient’s disability. Segmental impairments could then be treated appropriately to reduce $W_{\text{tot}}$. For example, injection of Botulinum Toxin A (BT-A) into spastic calf muscles of children with cerebral palsy reduced the foot equines deformity and led to a significant decrease in the vertical displacement of COM$\delta$ and therefore of $W_{\text{c}}$. An improvement in walking mechanics, quantified by $R$, was still present 6 months after BT-A injection. [52]

This method would also allow a better understanding of the effects of certain treatments, such as the decrease in $C$ after treadmill training in hemiparetic patients [4]. The unaffected limb that performs the majority of the mechanical work, could then be trained as much as the affected limb with objective assessments of training and response.

The quantitative and objective method of mechanical and energy analysis in pathological walking described here could also represent a powerful tool in clinical research, providing more valuable data than other, more qualitative and subjective, approaches. This study demonstrates the feasibility and clinical relevance of this technique.

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