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SURFACE EMG PROFILES DURING DIFFERENT WALKING CADENCES IN HUMANS [1]

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Movement is the result of the interaction between muscle tension, dictated by the central nervous system, and the mechanical demands of the task. Changes in these mechanical demands necessitate appropriate responses of the nervous system. An understanding of how the nervous system responds to changes in the mechanical demands of a task is essential to understanding movement control. The mechanical demands of locomotion vary considerably at different walking speeds. For this reason, walking at different speeds is an ideal paradigm for the investigation of the interaction between these demands and the behaviour of the nervous system.

The electrical activity of muscle contraction, the electromyogram (EMG), reflects both the output of the neurological system and the input to the mechanical system. EMG amplitudes are directly linked to both the neural 'drive' and the muscle tension (Bigland and Lippold 1954). Hence EMG amplitudes and their time course can provide information about both the neural and mechanical systems.

Engberg and Lundberg (1969) reported EMG patterns in intact cats walking at various speeds. The authors were only interested in the phasic similarities in EMG patterns and thus removed all EMG amplitude differences between different speeds of walking. Most studies on humans have reported qualitative EMG changes (Cavanagh and Gregor 1975), or 'on-off' phasic changes only (Grieve and Cavanagh 1971; Van der Straaten et al. 1975). The problems inherent in the use of 'on-off' EMG patterns are well described by Winter (1984). Milner et al. (1971) and Brandell (1977) both studied amplitude changes, but reported the results of a global measure: EMG averaged over the whole stride. Such a global measure could mask important differences in the time course of the EMG over the stride period. Thus there is a need to determine, more precisely, the EMG changes with walking speed both in the amplitude and the time domain. The purpose of this study was to determine the ensemble average EMG patterns associated with different walking cadences in 5 lower extremity muscles.

Methods

Eleven healthy subjects, 6 males and 5 females, participated in the study. The subjects ranged in age from 18 to 33 years; in height from 1.67 to 1.86 m; in weight from 64 to 80 kg. Five muscles of the right lower extremity were examined: the rectus femoris, vastus lateralis, lateral hamstring, tibialis anterior and soleus. Bipolar Ag/AgCl surface electrodes 0.5 cm in diameter were attached over the bellies of the 5 muscles, 1 cm apart rim to rim, after reducing the skin impedance with rubbing alcohol.

The myoelectric signals were telemetered via an FM multichannel biotelemetry system, full-wave rectified and low-pass filtered with a second order filter (cutoff frequency 3 Hz), producing an analog linear envelope. The linear envelope signals underwent analog to digital (A/D) conversion on-line at a sampling rate of 250 Hz with a Hewlett-Packard 9845T computer. The digitized data were stored on flexible discs for further analysis.

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The signal from footswitches attached to the heel, ball and toe of the right shoe was telemetered and recorded simultaneously with the EMG. Each switch generated a different voltage level when in contact with the floor, thus allowing different phases of a stride to be identified. Two photoelectric cells at shoulder level, spaced approximately 5 m apart along the middle portion of the 10 m walkway, initiated and terminated the A/D conversion. Only strides taking place between these photoelectric cells were converted. The photocell signal was simultaneously recorded on a separate channel, for a record of traverse times.

The subjects walked at 3 cadences: 115, 95 and 75 steps/min, in time to a metronome. The 115 steps/min cadence was chosen because it is close to the natural cadence for most people. The 95 and 75 steps/min cadences were chosen to represent slow cadences, in the range commonly reported for patients with neuromuscular pathology. Walking cadence was manipulated instead of walking speed, in order to account for differences in subject height. A given walking speed will require different levels of exertion for subjects of different stature. A given cadence and a fixed decrement in walking cadence should require a similar degree of exertion for all subjects, regardless of height. The order of presentation of walking cadences was randomized. Subjects practiced at each of the cadences for 15 min. A minimum of 16 sec of data were collected in each trial.

The EMG and footswitch data from each subject were examined, and a minimum of 6 strides selected from each speed for averaging. The selection criteria for averaging were based on the clarity of the footswitch signal for one complete stride, and the absence of movement artifacts or telemetry dropout across all channels. Heel contact to heel contact was considered 100% stride time for each stride selected for averaging. The EMG data at 0% and each consecutive 5% stride time were averaged across the selected strides, generating a 20-point ensemble EMG pattern of one stride for that cadence. Details concerning the selection and averaging techniques have been reported elsewhere (Winter 1984). Five strides have been shown to adequately represent a subject's walking pattern on one day (Arsenault 1982), thus the subject ensembles generated as described were considered representative of a subject's walking pattern at that cadence on a given day.

To compare the EMG amplitude changes, the mean EMG level in swing and in stance were calculated for each subject, for each muscle. A one-way repeated measures analysis of variance (ANOVA) design was used to determine whether the mean EMG amplitudes were significantly different at the 0.05 level for the 3 cadences, for each muscle. Post hoc tests were performed for two pairs of contrasts using the Bonferroni t test: the 115 and 95 steps/min, and the 115 and 75 steps/min conditions. The level of significance was set at 0.02 for the post hoc tests. These two contrasts were chosen to demonstrate the changes associated with slowing down from one's natural cadence.

The phasic similarity of the ensemble EMGs at two cadences was quantified by the Pearson's product-moment correlation. The 20 points specifying the ensemble EMG of a subject at one cadence were paired with the corresponding 20 points at another cadence from the same subject. Thus if the shape of the ensembles were similar, a high correlation would result. Correlations between the 115 and 95 steps/min cadences, and between the 115 and 75 steps/min cadences, were obtained for each muscle of each subject.

Results

A summary of the temporal results is shown in Table I. The values represent the average and standard deviation of the 11 subjects. The be-

TABLE I

Temporal results. Means and standard deviations (S.D.) of walking cadence (steps/min), walking speed (m/sec), and stance time (% stride time), for the 3 experimental conditions.

Cadence	mean	115	94.5	74.2
	S.D.	2.1	1.6	0.8
Speed	mean	1.38	1.06	0.75
	S.D.	0.12	0.10	0.06
Stance	mean	60.1	60.7	62.1
time	S.D.	2.5	1.5	1.7

TABLE II

Mean EMG amplitude in stance and in swing. An * indicates the mean (in μV) to be significantly different ($P < 0.02$) from the mean at the 115 steps/min condition. The muscles examined were: rectus femoris (RF), vastus lateralis (VL), biceps femoris (BF), tibialis anterior (TA) and soleus (SO).

Muscle	Mean EMG					
	Stance (steps/min)			Swing (steps/min)		
	115	95	75	115	95	75
RF	44	29 *	13 *	36	12 *	7 *
VL	89	64 *	31 *	24	19	10 *
BF	79	47	30 *	64	33 *	22 *
TA	140	113 *	101 *	198	159 *	140 *
SO	180	146 *	115 *	49	36 *	33

tween-subject variability of cadence, walking speed and stance time was very low. Each decrement in cadence of 20 steps/min resulted in a corresponding decrease in walking speed of approximately 0.31 m/sec.

The overall EMG pattern changes associated with walking cadence are shown in Fig. 1. These patterns represent averages of the 11 subjects. Ensemble peaks decreased with decreasing cadence in all muscles. This was most pronounced in the rectus femoris, in which the second peak disappeared at the slower cadences. To quantify the changes in the ensemble EMGs, two aspects were analyzed, as previously mentioned: (1) the average amplitude in stance and in swing; (2) the shape.

The ANOVA on mean EMG in stance and in swing revealed significant F tests for all muscles. The vast majority of post hoc contrasts revealed significant differences (Table II).

The correlations reflecting the similarity in shape are shown in Table III. Most muscles showed high correlations, indicating similarity in ensemble

TABLE III

Pearson's product-moment correlations for EMG ensembles. The frequency distribution for two sets of correlations: 115 and 95 and 115 and 75 steps/min, for all subjects. Same muscle groups as in Table II.

r	RF	VL	BF	TA	SO
1.0 –0.91	10	17	9	16	20
0.90–0.81	5	2	4	3	2
0.80–0.71	1	0	5	3	0
0.70–0.61	0	0	1	0	0
0.60–0.51	1	0	0	0	0
0.50 to –0.1	5	3	3	0	0
Total no. of comparisons	22	22	22	22	22

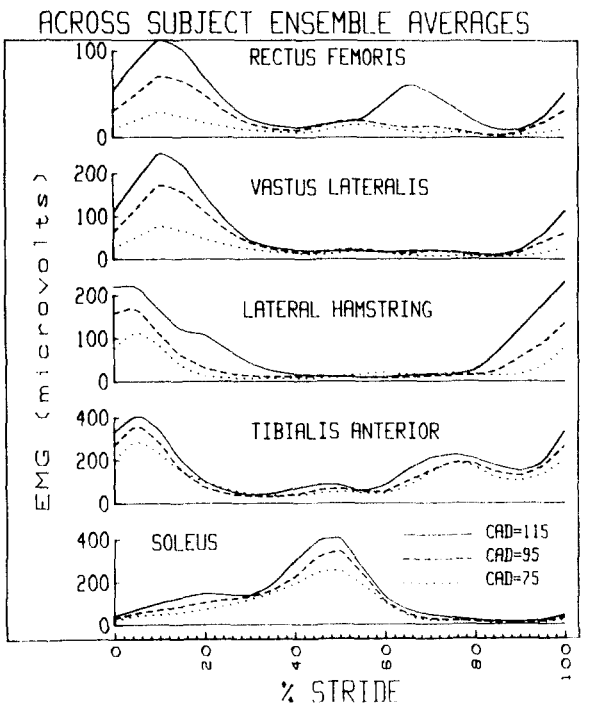

Fig. 1. Linear envelope EMG changes with walking cadence. These patterns represent the average of 11 subjects for 3 different cadences: 115, 95 and 75 steps/min. The stride time was normalized for all speeds, such that 0% and 100% correspond to heel contact.

shape with a change in walking cadence. This was particularly true for the soleus and tibialis anterior. In some subjects, low correlations were noted in the rectus femoris, vastus lateralis and lateral hamstring muscles.

Discussion

The results demonstrated that with changes in the cadence of walking, the EMG patterns changed primarily in amplitude; the shape of the ensembles remained almost exactly the same (Table III). The degree of amplitude change was muscle specific. Muscles about the ankle showed smaller amplitude changes than those about the hip and knee. These results will be discussed from 2 perspectives: (1) the response of the neurological system to changes in the mechanical demands of different walking cadences; and (2) the possible neurological mechanisms that are effecting the appropriate cadence changes.

Mechanical considerations

The EMG changes associated with walking cadence can be predicted based on the changes in the mechanical demands of the movement. The mechanical functions which must be accomplished during walking can be categorized into the following: (1) propulsion for forward progression; (2) control of foot position; (3) energy absorption in early stance; (4) forward acceleration of the limb in early swing; (5) deceleration of the limb in late swing.

The first two functions are accomplished by the ankle muscles, and the latter three by the hip and knee muscles. The mechanical demands of each function do not change uniformly with walking cadence, hence one would expect that the muscle adaptive responses would be different.

To facilitate the discussion, results from a similar study on the kinetics of walking at different cadences are included (Winter 1983). The net joint moments at the hip, knee and ankle are shown in Fig. 2, for 3 walking cadences. Although the individual subjects from the two studies were not identical, they were all drawn from the same subject pool of university students, and similar experimental protocols were followed. There is thus no

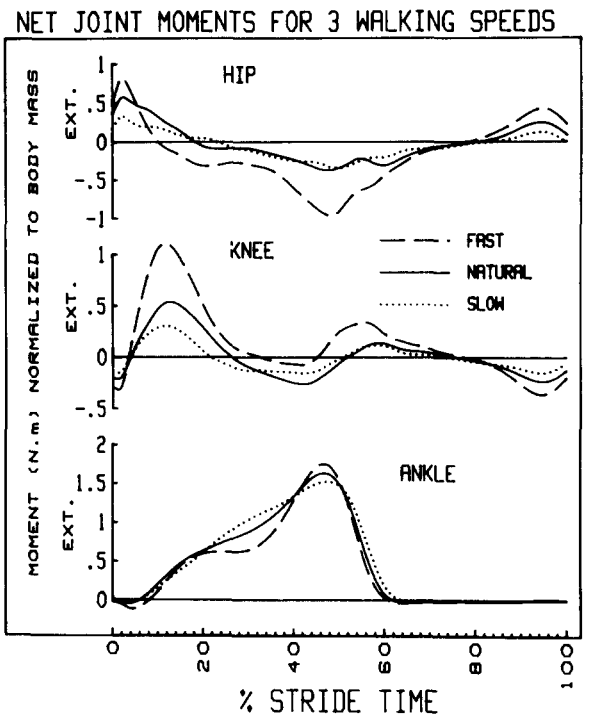

Fig. 2. Net joint moment patterns at the hip, knee and ankle, for 3 walking speeds. The moment patterns represent averages of 16, 14 and 14 subjects for the natural, fast and slow speeds respectively. The moments were normalized to the body mass of the subject prior to averaging. The stride time was normalized as in Fig. 1.

reason to suspect major differences between the two groups.

Muscle forces called upon to generate movement must perform two major tasks: (a) overcome gravity, whose effect is proportional only to the weight of the body segments to be supported during the movement, and (b) accelerate or decelerate the body segments. A change in walking cadence involves changing the second task only. By knowing the relative importance of tasks (a) and (b), one can predict the necessary changes the muscles must make to change walking cadence.

The first two mechanical functions of walking, forward propulsion (1) and control of foot position (2), contain a large gravity component. During push-off, the ankle plantar flexors must overcome body weight, a large 'overhead' cost, before they can accelerate the body upward and forward. Likewise, the control of foot position is primarily an anti-gravity task. Because of this, most of the

activity of the ankle muscles serves to overcome gravity; only a small percentage of the activity accelerates or decelerates the limbs. A change in walking cadence thus produced only small differences in ankle muscle activity (Fig. 1). This is in agreement with the small ankle moment changes associated with walking cadence, as shown in Fig. 2.

In contrast, the other 3 mechanical functions, energy absorption (3), forward acceleration of the swinging limb (4), and deceleration of the swinging limb (5), contain a large dynamic component. Although gravity is certainly a part of these functions, they are dominated by the rapid accelerations and decelerations necessary at the appropriate points in the walking cycle. Unlike the ankle, most of the activity of the knee and hip muscles serves to accelerate or decelerate the limbs. Since a change in walking cadence results in proportional changes in angular accelerations and decelerations (Winter 1983), these muscles show pronounced cadence-related changes in both EMG and moment patterns (Figs. 1 and 2).

The disappearance of the second rectus femoris EMG peak in most subjects is of particular interest with respect to the mechanics of movement. This activity is associated with forward acceleration of the limb in early swing. At slower walking cadences, the acceleration necessary to swing the limb decreases to the point that the initial part of swing can be accomplished virtually without muscle activity, as in a pendulum.

Neurological considerations

The existence of a central pattern generator for locomotion has been confirmed in many vertebrates (Grillner 1975). Although its structure is unknown, it is generally agreed that appropriate tonic excitation of the generator will result in a cyclical output typified in locomotion. The neurological mechanisms which control walking speed, however, are still unclear. Two possibilities were suggested by Shik and Orlovski (1976): the control of cadence alone, and the control of muscle forces alone. If cadence alone were controlled, the mechanism which controls the speed of walking only speeds up the rate at which the cyclical activity occurs, the central pattern generator oscillation rate. The pattern of muscle forces employed through the stride would remain unchanged at different speeds, but they would occur at different rates. Under this possibility, the EMG amplitudes would remain unchanged at the different cadences, and the timing of the EMG activity stride time dependent. The present findings, as well as those of Winter (1983), rule out this possibility. If cadence alone were controlled, the EMG amplitude would remain the same, thus resulting in similar or shorter stride lengths. An increase in walking speed would be strictly the result of a higher cadence. This clearly is not the case.

The second possibility suggested by Shik and Orlovski (1976), and the one they supported, was the control of muscle forces alone. That is, the mechanism which controls walking speed directly controls the magnitude of muscle forces. Hence, to increase walking speed, the muscle forces would be increased, resulting in greater accelerations and decelerations, which in turn would result in longer step lengths and shorter step times. A third possibility which can be added to Shik and Orlovski's original suggestions is the control of both cadence and muscle force in combination, suggested by Winter (1983). Other possibilities may exist, but have not been proposed as yet.

The second and third possibilities are difficult to isolate. Shik and Orlovski (1976) concluded, from tonic midbrain stimulation of mesencephalic cats (Shik et al. 1966), that muscle forces were directly controlled. The EMG amplitude changes observed in this study and the proportional joint moment changes reported by others (Cavanagh and Gregor 1975; Winter 1983) would also support the second option, but not to the exclusion of the third. Indeed, whether cadence is controlled in combination with muscle forces, or whether it is merely an effect of muscle force changes remains unresolved.

The EMG changes with cadence were different for each of the muscle groups studied. The method by which the nervous system recruits a muscle to the required degree to generate a certain speed of walking is unknown. The EMG patterns from the current study do not support a simple 'gain' phenomenon, whereby the entire EMG pattern would change by a multiplicative constant with cadence.

Indeed, not only did the gain appear different between muscles, but it varied with stride time within one muscle, the rectus femoris.

One potential cause for these differences is the relationship between motor unit recruitment threshold and the rate of rise of tension. An increase in the rate of tension rise is associated with a lower threshold of recruitment force for motor units (Budingen and Freund 1976). Since the rate of rise of tension in muscles, as reflected by the joint moments (Fig. 2), changes with the walking speed, the EMG observed at the surface will reflect these differences. The large percentage EMG changes in the knee extensors in contrast to the small percentage changes in the soleus could be partly explained by this phenomenon. As seen in Fig. 2, the rate of rise of tension changes more dramatically with walking speed in the knee extensors than in the ankle plantar flexors. In addition, the differences in EMG changes of the rectus femoris muscle in stance and in swing may also be associated with the higher velocities of movement in swing than in stance.

A tight coupling between EMG bursts and events in the stride was evident. It is clear from Fig. 1 that the peaks and valleys in EMG amplitude are related to the normalized stride time. For example, regardless of walking speed, the plantar flexor EMG burst responsible for push-off always occurred near 50% stride time. This was independent of what 50% stride time represented in absolute time. The effect of this invariant EMG timing is a tight coupling.

It must be emphasized that there was considerable between-subject variability in cadence-related EMG changes. This phenomenon has been reported previously (Shiavi et al. 1981). Whereas the EMG pattern changes averaged across subjects revealed a seemingly systematic trend, the individual subject responses varied greatly. These individual differences could be related to the trade-off in function between synergistic muscles, differences in fibre type, or kinetic differences in the way subjects walked. It appears that the apparently simple task of reducing one's walking cadence can be achieved by an enormous number of combinations of muscle activity patterns. Although trends can be identified in this small sample, the prediction of walking cadence-related changes in EMG patterns for individuals will require more extensive investigation.

Summary

The ensemble electromyogram (EMG) patterns associated with different walking cadences were examined in 11 normal subjects. Five muscle groups were studied: the rectus femoris, vastus lateralis, lateral hamstring, tibialis anterior and soleus muscles of the right lower extremity. The myoelectric signals were telemetered, full-wave rectified and smoothed. Subjects walked at cadences of 115, 95 and 75 steps/min. Footswitches indicated the different phases of the stride. Six or more strides per subject were averaged for each cadence. Cadence-related changes in (1) mean EMG amplitude during stance, and during swing, and (2) the shape of the EMG patterns, were analyzed. One-way repeated-measures analyses of variance on the mean EMG amplitude in stance and in swing revealed significant changes with cadence ($P < 0.05$) in all muscles examined. The magnitude of these changes could be related to the mechanical function of the muscles involved. The shape of the EMG patterns generally remained similar at the different cadences. The timing of EMG activity was closely related to the normalized stride time and remained invariant at different cadences.

Résumé

Aspect de l'EMG de surface chez l'homme à différentes cadences de marche

Les patterns d'EMG globaux associés à différentes cadences de marche ont été examinés chez 11 sujets normaux. Cinq groupes de muscles ont été étudiés: rectus femori, vastus lateralis, tendon du biceps crural latéral, tibialis anterior, et soleus à l'extrémité du membre inférieur droit. Les signaux myoélectriques étaient transmis par télémétrie, rectifiés et lissés. Les sujets marchaient à des cadences de 115, 95 et 75 pas/min. Des microinterrupteurs indiquaient les différentes phases du cycle de marche. On a moyenné au moins 6 cycles

de marche par sujet et pour chaque cadence. On a analysé les modifications, liées à la cadence, (1) de l'amplitude EMG moyenne au cours de la phase d'appui et la phase de balancement, et (2) de l'aspect des patterns EMG. Une analyse de variance à mesures repétées a démontrée un effet significatif de la cadence de marche sur la valeur moyenne de l'EMG pendant l'appui et le balancement ($P < 0,05$), et ce, pour tous les muscles examinés. L'importance de ces modifications a pu être associée à la fonction mécanique des muscles impliqués. La forme des patterns EMG restait en général la même quelle que soit la cadence. Le décours temporel de l'activité EMG était étroitement lié à la durée normalisée du cycle de marche et restait identique quelle que soit la cadence.

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