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Detection of non-standard EMG profiles in walking[☆]

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Abstract

The amplitude of an EMG and the temporal pattern can be used when considering if an EMG profile is normal or not. In the method described in this paper a gain factor of the complete EMG profile was determined and then the profile normalised with this gain factor. This normalised individual profile was then compared with a standard profile, predicted on the basis of walking speed. Deviating profiles were identified when they fell outside the upper and lower 95% limits range for the average profiles of 14 leg muscles. The amount of deviation from the normal profile can be quantified with the normalised mean square difference D^2 . Gain factors varied over a factor of 4 within a group of 10 normal subjects. For a normal population D^2 was below 1. Most muscles had consistent profiles but some patterns could be discerned which showed marked variability among muscles and subjects.

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Keywords: Electromyogram; Human walking; Speed dependence; Intersubject variation

1. Introduction

Electromyography (EMG) has proved to be a valuable tool in the evaluation of gait disorders. To assess deviations from the normal pattern, the ‘normal’ pattern should first be defined. In a previous study [1], we have presented average EMG profiles of young normal subjects and shown how to take the influence of walking speed into account. The present paper tries to answer the question when the EMG profile from a subject or patient is to be classified as non-standard and to give a quantitative measure of this deviation of the standard.

Two aspects should be considered: firstly the *timing pattern*, which is directly related to the neural control of the muscle and secondly the *amplitude* of the EMG signal, which is known to be influenced by a great number of factors, e.g. electrode positioning, muscle fibre orientation and thickness of the subcutaneous fat layer [2,3]. When possible, it is good practice to calibrate the rectified EMG against muscle force, but this is impractical in patient

recordings of many EMGs and it poses difficult problems in case several synergistic muscles are active simultaneously [4].

2. Methods

2.1. Subjects, EMG recording

The experimental data were the same as used in the study [1] on averaged profiles. The starting material for the proposed procedure was the average EMG profiles of our control group of 10 healthy subjects. In Fig. 1a a grand average was computed (thick line) for 10 individual profiles. Next, a gain factor equal to the ratio between individual and mean profile was determined for each individual curve. By dividing by this gain factor, each individual profile had about the same amplitude as the mean profile (Fig. 1b). From these normalised curves a high and a low limit was assessed (thick lines). A patient profile was processed in a similar way and was normalised firstly. If the normalised profile fitted between the high and low limits, the timing was normal. If not, some measure of the deviation from normal can be evaluated. Secondly the gain factor can be evaluated. A very low gain factor, for example, suggests that the muscle is paretic.

[☆] Data related to this paper can be downloaded from the CGA Normative Gait Database, <http://www.univie.ac.at/cga/data/index.html>.

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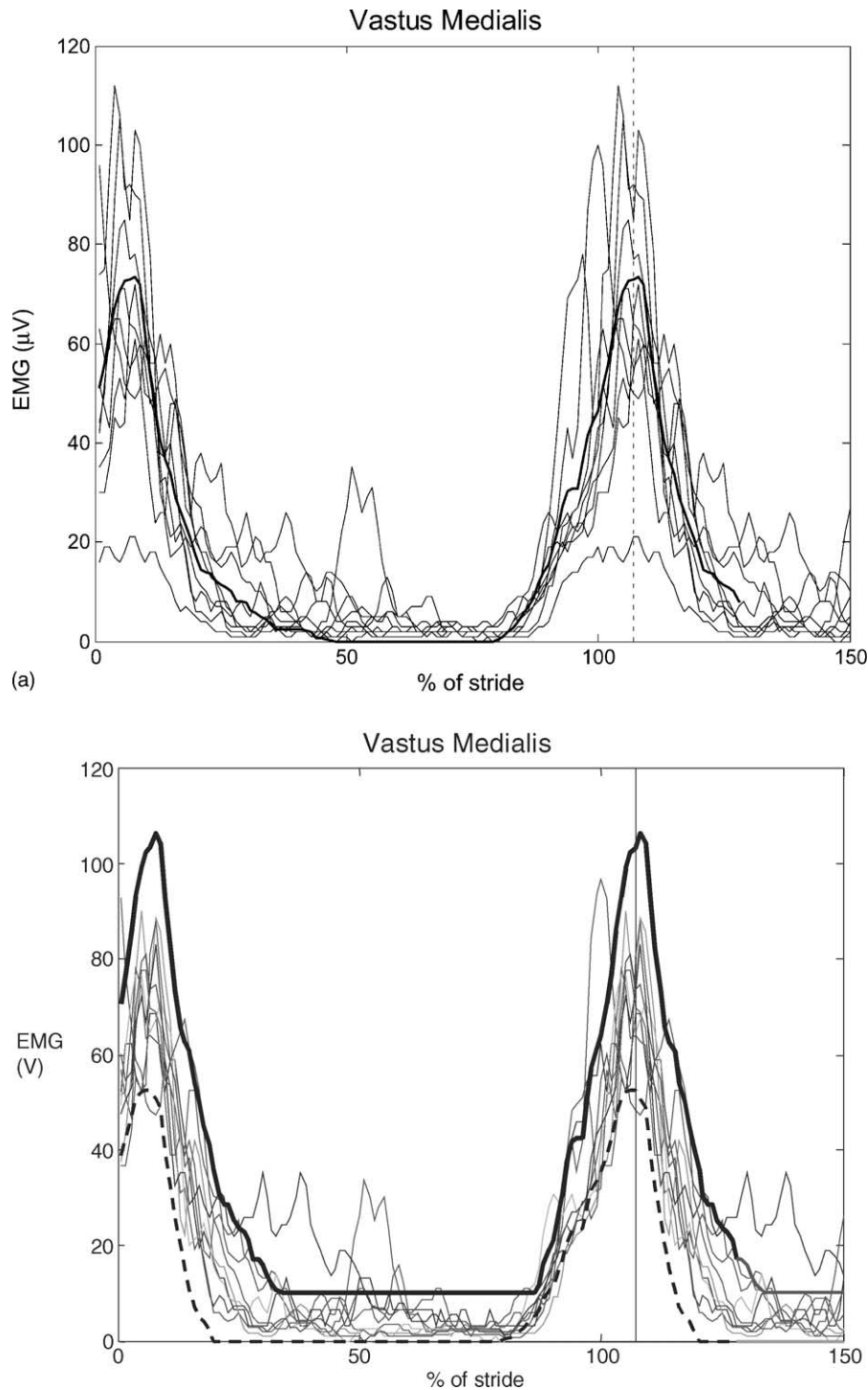


Fig. 1. (a) Average EMG profiles of vastus medialis muscle in 10 normal subjects (thin lines) with the average profile (thick line). (b) Average EMG profiles of vastus medialis muscle after normalising with the gain factor. Also shown low and high limits, as obtained from the basic patterns $F_0(k)$ and $F_1(k)$. Time is given as a percentage of stride, starting at heel contact, but in a scale running 0–100–50%, in order to represent better the activity around heel strike.

Surface EMGs of 14 leg muscles (Table 1) were recorded from two homogeneous groups ($n = 9$ and 11, respectively) of young healthy male subjects (mean age 22 years (S.D. 1.5), stature 1.85 m (S.D. 0.05), leg length 0.98 m (S.D. 0.04), body mass 73 kg S.D. 8). The division in two groups

was made for practical reasons, but care had been taken to match the average personal data of both groups. Electrode placements were in accordance with the recommendations of Perotto [5] and of SENIAM [6]. Subjects walked barefoot on a 10 m indoor walkway at speeds of 0.75, 1.00, 1.25, 1.50,

Table 1

List of muscles, their abbreviations as used in the text, the contributing basic patterns $F_0(k)$ and $F_1(k)$, see Fig. 1, expressed as the weighting coefficients for the low, average and high limits, **L**, **D** and **H**, respectively

Muscle	$F_0/F_1/F_2$	k	L	D	H
Soleus (SO)	F_0	1	0.7	1	1.47
	F_1	1	0.75	1	1
	F_1	5	0	0.39	0.63
	F_1	8	0	1	1.88
Gastrocnemius medialis (GM)	F_0	1	1	1.50	2.00
	F_1	1	0.51	0.83	1.27
	F_1	2	0	0	0.37
Gastrocnemius lateralis (GL)	F_0	1	0	0.25	0.65
	F_1	1	0.32	0.64	1.10
	F_1	2	0	0	0.33
Peroneus longus (PL)	F_0	1	0	0.61	1.19
	F_0	6	0	0	0.32
	F_1	1	0.65	0.91	1.15
	F_1	5	0	1	1.23
	F_1	8	0	1.10	3.28
Tibialis anterior (TA)	F_0	3	0	0.50	0
	F_0	4	0	0.94	2.66
	F_0	6	0.56	1	1.46
	F_1	5	1.12	1.72	2.39
	F_1	6	0.54	1	1.50
Vastus medialis (VM)	F_0	2	0	1	2.09
	F_1	2	0.76	1	1.38
Vastus lateralis (VL)	F_0	2	0.57	1.19	2.74
	F_1	2	0.7	0.84	1.22
Rectus femoris (RF)	F_0	2	0.44	1.31	2.30
	F_1	2	0.30	0.78	1.48
	F_2	9	0.30	1	1.78
Biceps femoris (BF)	F_0	3	0.17	0.55	1.16
	F_1	3	0.26	0.44	0.64
Semitendinosus (ST)	F_0	2	0	0	2.40
	F_0	3	0.39	1	1.63
	F_0	5	0	0	0.77
	F_1	3	0.48	1	1.36
Semimembranosus (SM)	F_0	3	0.20	0.30	0.80
	F_1	3	0.30	0.58	0.90
	F_1	5	0	0	1.20
	F_1	9	0.20	1	1.18
Gluteus maximus (GX)	F_0	4	0	0	0.25
	F_0	5	0	0	0.73
	F_1	4	0.50	1	1.37
Gluteus medius (GD)	F_0	4	0.64	1	1.44
	F_0	6	0	0	0.42
	F_1	2	0	0.60	0.60
	F_1	4	0.75	1	2.40
	F_1	5	0	0.50	1.20
Adductor magnus (AM)	F_0	3	0.20	0.22	0.60
	F_0	5	0.54	1	1.88
	F_1	7	0	1	1.70

and 1.75 m s^{-1} . Average walking speed was assessed from the interval between passing two light beams at both ends of the walkway, 7 m apart. To accommodate differences in leg length, speed v will be expressed in non-dimensional form as

$\hat{v} = v/\sqrt{gl_0}$, in which l_0 is the leg length and g the acceleration of gravity. The EMGs were high-pass filtered at 20 Hz, rectified and smoothed with a 25 Hz third order Butterworth low-pass filter. Smoothed rectified EMGs were, after A/D conversion with a sample frequency of 100 Hz, linearly interpolated to 100 points per stride, triggered by heel contact of the leg of interest. The recorded steps were screened to exclude those with obvious artefacts or incorrect foot contacts. In this way for every individual i , normalised speed \hat{v} , and muscle m , average individual profiles $e(p, m, \hat{v}, i)$ were determined from at least 10 steps over $p = 1 - 100\%$ of the gait cycle.

2.2. Gain factor

The individual gain factors $g(m, i)$ were determined by linear regression (without intercept) with the average profile

$$g(m, i) = \frac{\sum_p (e(p, m, \hat{v}, i)E(p, m, \hat{v}))}{\sum_p E^2(p, m, \hat{v})} \quad (1)$$

in which $E(p, m)$ is the grand average profile, obtained from the basic patterns F_0 and F_1 , as described previously [1]. This was done at a single speed, 1.25 m/s. It was assumed that the gain factor was constant at all speeds in the same session.

2.3. Limits

The upper limit $h(p, m, \hat{v})$ and lower limit $l(p, m, \hat{v})$ were obtained from the set of individual profiles. First the profiles were normalised by dividing with the appropriate gain factor: $e^*(p, m, \hat{v}, i) = e(p, m, \hat{v}, i)/g(m, i)$, cf. Fig. 1b. From this set of normalised profiles the upper and lower limit of the range were estimated for every p . A simple choice would have been to take the maximum and minimum of the set of $e^*(p, m, \hat{v}, i)$ for every p . To minimise the influence of possible outliers, an estimate based on fractiles was used assuming that the profiles had a uniform distribution. To this end the n samples $e^*(p, m, \hat{v}, i)$ were sorted for every p in ascending order and the limits were estimated from the two-but-lowest and two-but-highest samples $e^*(p, m, \hat{v}, 3)$ and $e^*(p, m, \hat{v}, n - 2)$:

$$\begin{aligned} l(p, m, \hat{v}) &= e^*(p, m, \hat{v}, 3) - \text{mar}(p, m, \hat{v}), \\ h(p, m, \hat{v}) &= e^*(p, m, \hat{v}, n - 2) + \text{mar}(p, m, \hat{v}), \\ \text{mar}(p, m, \hat{v}) &= \frac{2.5}{n - 5} (e^*(p, m, \hat{v}, n - 2) - e^*(p, m, \hat{v}, 3)) \end{aligned} \quad (2)$$

In addition, $l(p, m, \hat{v})$ is limited to be above zero.

In a manner identical with the procedure for average data [1] the $h(p, m, \hat{v})$ and $l(p, m, \hat{v})$ functions were fitted by a linear plus quadratic dependency with speed and assembled

from three sets of basic patterns F_0, F_1, F_2 :

$$\begin{aligned} l^*(p, m, \hat{v}) &= \mathbf{F}_0 \mathbf{L}'_0 + (\hat{v} - 0.16) \mathbf{F}_1 \mathbf{L}'_1 + (\hat{v} - 0.16)^2 \mathbf{F}_2 \mathbf{L}'_2, \\ h^*(p, m, \hat{v}) &= \mathbf{F}_0 \mathbf{H}'_0 + (\hat{v} - 0.16) \mathbf{F}_1 \mathbf{H}'_1 + (\hat{v} - 0.16)^2 \mathbf{F}_2 \mathbf{H}'_2 \end{aligned} \quad (3)$$

Next to this, h^* was limited to be above $10 \mu\text{V}$. In (3) \mathbf{F}_0 is a 6×100 matrix containing the six basic $F_0(k, p)$ patterns, similarly \mathbf{F}_1 is a 9×100 matrix containing the nine basic $F_1(k, p)$ patterns, and \mathbf{F}_2 is a 1×100 matrix containing the single basic $F_2(p)$ pattern. All these basic patterns were the same as in the previous study, Fig. 2 of \mathbf{L}_0 and \mathbf{H}_0 are 14×6 matrixes containing weighting coefficients, similarly for $\mathbf{L}_1, \mathbf{H}_1$, and $\mathbf{L}_2, \mathbf{H}_2$. These coefficients were determined from the experimental $l(p, m, \hat{v})$ and $h(p, m, \hat{v})$ functions in a similar way as described for the average profiles [1], by first determining the speed independent and speed dependent parts, and then fitting these parts to the $F_0(k, p)$ and $F_1(k, p)$ functions, respectively, by a linear regression (1).

2.4. Deviation

To obtain a measure for the ‘non-standardness’ of an individual averaged EMG profile, first the p 's were obtained for which the patient's $e^*(p, m)$ is outside the range between $l(p, m)$ and $h(p, m)$ for the relevant \hat{v} . Then the difference between the individual normalised EMG and the standard profile were squared over these outlying points p_{out} , summed and divided by the summed square of the standard profile:

$$D_m^2 = \frac{\sum_{p_{\text{out}}} (e_{mi}^* - E_m)^2}{\sum_1^{100} E_m^2} \quad (4)$$

3. Results

The gain factors for all nine or 11 subjects and 14 muscles varied within a range of 0.2–5. In Fig. 3 they are given in a logarithmic normal probability plot, which suggests that their distribution could be fitted by a log-normal

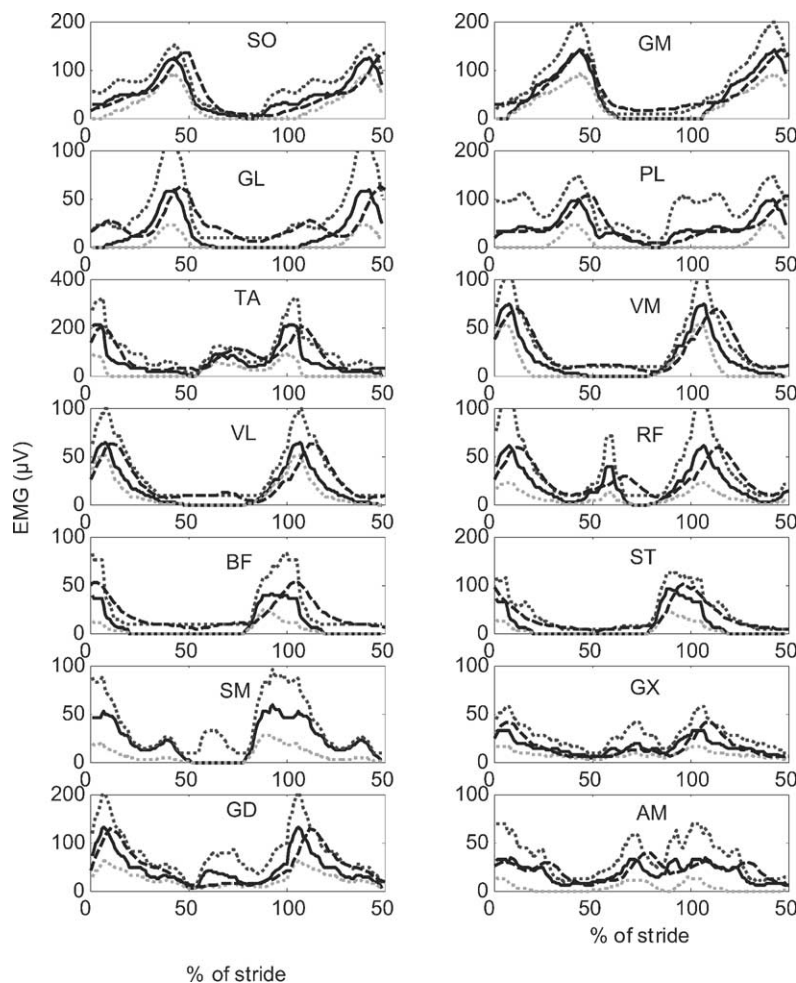


Fig. 2. Mean EMG profiles (solid lines) and lower and upper limits (dotted lines) for all 14 muscles and a walking speed of 1.25 m s^{-1} (normalised speed 0.40). For abbreviations of muscle names, see Table 1. As a comparison data from Winter have been included (dashed line). The amplitudes of Winter's profiles have been normalised, cf. Eq. (1). The gain values were: SO 2.6, GM 2.2, GL 3.6, PL 1.5, TA 1.8, VM 2.4, VL 3.2, RF 1.0, BF 3.1, ST 2.2, SM not available, GX 1.1, GD 0.7, AM 2.0. Winter's data show a delay of 4–7% due to low-pass filtering at 3 Hz.

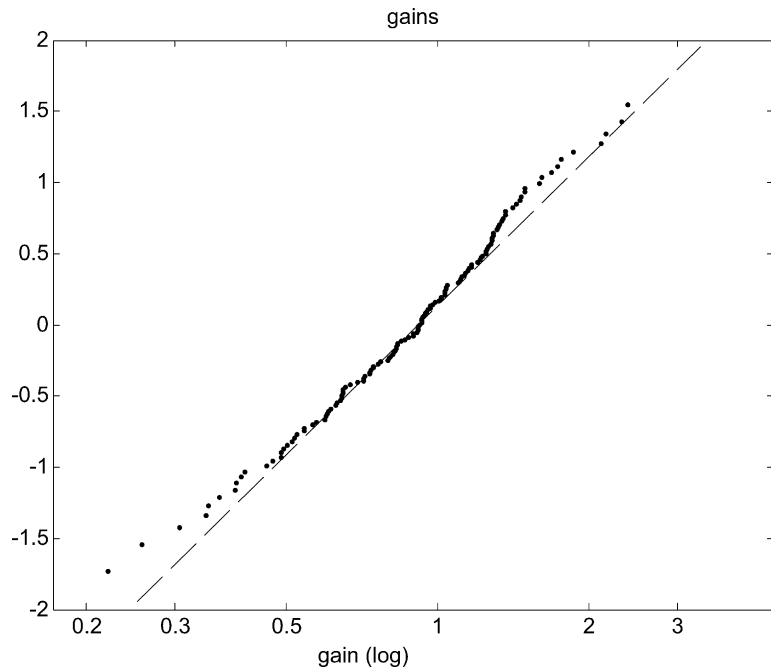


Fig. 3. log-normal probability plot of all gain values of 9 (11) subjects \times 14 muscles. Horizontal scale: logarithm of gain factor g , vertical scale: normal probability, 0: mean, +2: mean + 2 standard deviation in a normal distribution, etc. The straight line suggests that gains have a log-normal probability distribution. Percentiles 2.5% (-2 S.D.) and 97.5% ($+2$ S.D.) can be read from the fitted straight line, and are 0.25 and 4, respectively.

distribution with a mean of 1 (as could be expected from the procedure) and a S.D. of $\log(g) = 0.30$. By extrapolation, the 2.5–97.5% confidence interval ranged from $g = 0.25$ to $g = 4$. The lowest g -values referred to GL, the highest to GX (the outlier $g = 4.7$) and RF.

The weighting coefficients for the **L**, and **H** matrices have been given in Table 1. For comparison the coefficients of

the **D** matrix, relating to the average, which were reported in the previous paper, have been included as well. As an example Fig. 2 gives the average, lower and upper limits as calculated by (3) for a walking speed of 1.25 m s^{-1} . As a comparison Winter’s [7] data have been included. It should be noted that in the latter data a low-pass filter of only 3 Hz had been used, resulting in a considerable delay.

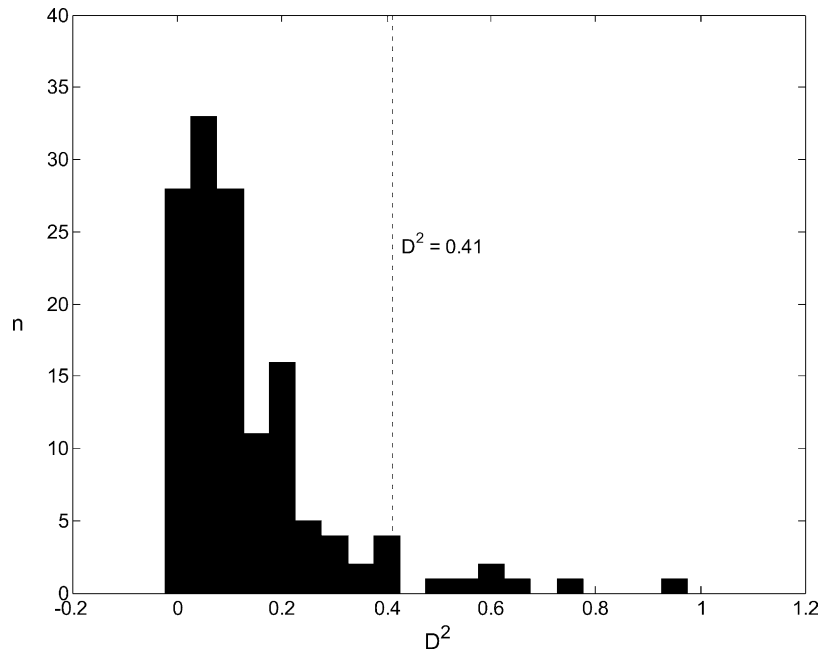


Fig. 4. Histogram of the normalised mean square difference D^2 in 9 (11) normal subjects \times 14 muscles at a speed of 1.25 m s^{-1} . Gain was also determined at 1.25 m s^{-1} . The 95% percentile was 0.42 in this case, cf. Table 2.

Table 2

The 95% percentile values for normalised mean square difference D^2 for individual profiles in our normal subject group

Speed (m s^{-1})	Gain same speed	Gain 1.25 m s^{-1}	Gain 1.75 m s^{-1}
0.75	1.10	1.35	1.38
1.00	0.51	0.73	0.79
1.25	0.42	0.42	0.70
1.50	0.43	0.93	0.45
1.75	0.48	2.18	0.48

Gain factors were fitted, from left to right, for the same speed as the measured profiles, for the middle speed 1.25 m s^{-1} , and for the highest speed, 1.75 m s^{-1} .

To give an idea of the properties of the D^2 measure of deviation, it was determined for the individual profiles of our subject group. Fig. 4 shows the distribution of D^2 for the speed of 1.25 m s^{-1} . In this case the 95% percentile was determined as 0.42. In principle the gain factors do not depend on walking speed as speed dependent effects have already been discounted in (3). When measurements are done at several speeds, it is thus sufficient to fit the gain factors only once for each muscle, preferably at the highest speed. When a recording at a single speed is evaluated it is, of course, only possible to fit at this one speed. For the deviation this makes a difference; when gain and deviation are determined for the same profile the fit will be better and the deviation lower. This can be seen in Table 2, where 95% percentiles of D^2 have been given three times: for this case the gain was determined at the same speed as evaluated, for this case the gain was determined at 1.25 and 1.75 m s^{-1} .

4. Discussion

When an average profile from a patient is subjected to the method described here, two questions can be answered. The first question is: how much does the form of this profile agree with the standard profile, predicted for the relevant walking speed and the patient's leg length from a set of assumedly normal walkers? A deviation can be expressed in the normalised mean square difference D^2 . When D^2 is zero this indicates perfect agreement; when it is above 1.0 the profile is certainly non-standard, when it is between 0.5 and 1.0 it is doubtful (cf. Table 2). Our group of subjects consisted of young healthy subjects, with age and stature within a narrow range. When the profile of a subject or patient is different from our 'standard profile', this should not, therefore, be interpreted as 'abnormal' or 'pathological' without further interpretation, but only as 'not in agreement with the present group of young healthy male subjects'. If EMG profiles for patients of other age or sex are to be investigated, this requires, in principle, that data from a corresponding healthy control group should be used as a reference. When, on the other hand, a patient's profile of a particular muscle is in agreement with the presented 'young healthy male' stan-

dard profile, this is certainly an indication that the profile is normal.

In Fig. 2 the profiles found in this study have been presented for a moderate walking speed of 1.25 m s^{-1} . Data from Winter have been included as a comparison, after normalisation for amplitude (see Section 2). Winter's data were filtered with a low-pass filter of 3 Hz, whereas ours were filtered at 25 Hz, and therefore show a delay between 4 and 7%. If this effect is considered, all profiles are very similar. The amplitudes are not: Winter's 'gain factors' range between 0.7 and 3.6 (average 2.1); see Fig. 2.

The second question regards the amplitude of the EMG, expressed in the gain factor g . It is assumed that this factor depends on details of the electrode placement (see Section 1) but that the effects of speed are completely accounted for by the **D**, **L** and **H** factors. With a proper electrode placement, it is not to be expected that g will change when walking at different speeds. When g is around 1, the amplitude is comparable to the average of our normal group, but can vary over a factor of 4 (i.e. it can be between 0.25 and 4.0) within perfectly normal subjects. An anecdotal example is the high gain values once found in a young, very lean child. On the other hand, a gain markedly below 0.25 might well indicate a paresis. If EMG gains are followed within the same subject, the random error is considerably less [8–10].

An example how the present method may be applied has been given by Boerboom et al. [11]. Anterior cruciate ligament deficiency causes in some patients a functional instability of the knee ('non-copers'), but other patients are able, after considerable rehabilitation, to function normally ('copers'). In contrast to non-copers, many copers showed an atypical activity of semitendinosus or biceps femoris during stance. Values of D^2 for these two muscles have been given in Table 3. This atypical hamstring activity may be a compensatory mechanism by which copers have learnt to function on a normal level. Ironically, in this case the 'healthy' subjects have an 'abnormal' profile.

The D^2 -deviation, as defined in (4), is more or less the logical form of a mean squared error measure. It is only slightly different from the variation ratio (VR) [12,13]. In the VR the denominator term contains $(E_m - \bar{E}_m)^2$ instead of E_m^2 . Subtracting the means in this way does not seem useful for EMG profiles, for which the mean over the

Table 3
Patients without anterior cruciate ligament

	Semitendinosus		Biceps femoris	
	Median	Range	Median	Range
Copers	0.54*	0.30–0.66	0.48	0.02–0.96
Non-copers	0.14	0.08–0.29	0.27	0.07–0.57

D^2 values for two hamstring muscles in five patients able to function normally ('copers') and five with persistent knee instability ('non-copers'). From Boerboom et al. the non-copers have a normal EMG pattern, while many of the copers have adopted a typical non-standard pattern, with hamstring activity around midstance.

complete profile has no special meaning. Including only the data points outside the lower–upper interval does influence the D^2 -deviation only mildly, but the situation in which $D^2 = 0$ has now a special significance: the profile lies entirely between the lower and upper limits.

The entries in Table 1 give some clue as to whether the individual EMG profiles were constant among subjects, when the **L**, **D** and **H** values are relatively close together, or when the profiles were variable, which is the case when **L** = 0 and **H** has a high value.

For the *plantarflexor group*, GM and GL show quite constant profiles, although it should be noted that in a few subjects the $F_0(1)$ pattern had zero weight in GL. In these cases GL activity was thus confined to a single burst at push-off, $F_1(1)$. SO had, above a stereotypical pattern similar to GM, rather variable activity before and around heel strike, $F_1(8)$. In the individual subjects this activity could vary between 0 and 100 μV , about 2/3 of the peak activity at push-off. PL shows this activity even more so. Here EMG at heel contact could be very high, up to twice the push-off peak. Next to this, PL can show marked activity during swing. TA had a constant profile in swing and early stance, $F_0(6)$, $F_1(5)$, and $F_1(6)$. Activity in stance, $F_0(4)$, was variable from 0 to some 100 μV (1/3 of peak). It thus seems that there was sometimes co-contraction activity of TA and PL or SO during stance. This was verified by comparing profiles of individual subjects. The co-contraction might have a role in the control of foot positioning. It may have been prominent here because our subjects walked barefoot.

Profiles of the *vasti* were constant among subjects. In RF the F_2 peak around toe was very variable in height. For this muscle, Nene et al. [14] have reported a remarkable finding. With intramuscular electrodes Nene et al. [14] showed that the $F_0(2)$ and $F_1(2)$ patterns are in fact crosstalk, probably originating from vastus intermedius. Only the F_2 peak corresponds to real RF activity.

Profiles in the *hamstrings* are similar, with the exception that in several subjects SM showed a definite activity in stance. Four out of 10 subjects showed a peak around 40%, also four, but not the same subjects, had a peak around 65% and two had no stance phase activity after the regular hamstring profile.

In all muscles where they were manifest, PL, SM, GX, GD and AM, the adductor and abductor swing phase activities, $F_0(5)$ and $F_1(5)$, respectively, were very variable. In AM versus GD the activities were to some extent reciprocal. Our interpretation is that these patterns are related to the foot placement after swing, which seems a major control mechanism to ensure stability during walking

[15,16]. To study this interesting mechanism further, step-to-step variability should be studied in longer series of steps.

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