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THE CONTROL OF TIMING AND AMPLITUDE OF EMG ACTIVITY IN LANDING MOVEMENTS IN HUMANS

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SUMMARY

The control of self-initiated falls from different heights was studied. The objective of the study was to investigate in a quantitative manner the modulation of EMG timing (i.e. onset from take-off and duration from onset to touch-down) and amplitude (before and after foot contact) as a function of fall height. The muscles studied were m. soleus and m. tibialis anterior. Kinematic (ankle joint angle) and kinetic (ground reaction force) variables were also measured. Six subjects took part in the experiments that consisted of ten landings from each of five heights (0.2, 0.4, 0.6, 0.8 and 1 m) onto a force platform. We found a consistent pattern of co-contraction before and after touch-down across the fall heights studied. In both muscles, the onset of pre-landing EMG activity occurred at a longer latency following take-off when landing from greater heights. The absolute EMG duration was affected to a lesser extent by increasing fall height. These findings suggest that the onset of muscle activity of the muscles studied prior to foot contact is timed relative to the expected time of foot contact. Pre- and post-landing EMG amplitude tended to increase with height. Despite a doubling in the magnitude of ground reaction force, the amplitude of ankle joint rotation caused by the impact remained constant across heights. These findings suggest that the observed pattern of co-contraction is responsible for increasing ankle joint stiffness as fall height is increased. The attainment of an appropriate level of EMG amplitude seems to be controlled by (a) timing muscle activation at a latency timed from the expected instant of foot contact and (b) varying the rate at which EMG builds up.

INTRODUCTION

Leaping down to land on a lower surface is an essential skill for animals whether they be in the role of predator or prey. The effective performance of this movement depends on early motor learning and on the ability of the motor control system to modify the details of the action to the exact circumstances of the task at hand. The goal of this movement can be postulated in terms of physics: the minimization of the impact force of the landing collision.

To avoid injury the ground reaction force experienced throughout the impact has to be spread in time (Dufek & Bates, 1990). This may seem a simple task since healthy individuals can land smoothly without too much conscious thought. However, in reality this is a major control problem for the human brain or for a robotic system. To achieve a successful landing the motor system must predict the characteristics of the ground reaction force and control the amplitude and velocity of joint rotations in the lower body by activating some energy absorption mechanism. This objective is achieved by appropriately controlling the compliance of the limb.

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A naive view would be that the momentum of the body could be braked by short latency stretch reflexes. However, due to reflex delays, these responses would occur too late to fully achieve the required result and, in addition, the gain of the reflex might be too low to provide the required force (Rack, 1981; Stein, 1982). We should therefore expect an increase in muscle stiffness prior to impact, produced by the activation the relevant musculature. Such pre-landing activation has indeed been found using electromyographic techniques (EMG), both in animals (Laursen, Dyhre-Poulsen, Djorup & Jahnsen, 1978; Dyhre-Poulsen, Laursen, Jahnsen & Djorup, 1980; McKinley & Smith, 1983; McKinley, Smith & Gregory, 1983; Dyhre-Poulsen & Laursen, 1984) and humans (Melvill Jones & Watt, 1971a,b; Greenwood & Hopkins, 1976; Dietz & Noth, 1978; Dietz, Noth & Schmidtbleicher, 1981; Sidaway, McNitt-Gray & Davis, 1989; Dyhre-Poulsen, Simonsen & Voigt, 1991; Thompson & McKinley, 1995).

Controlling the timing of this preparatory muscle activity is crucial, if a given level of limb compliance has to be attained at the time of foot contact. The time between take-off and the onset of the EMG activity and the duration of EMG activity prior to landing are both likely to be key variables which ensure that a sufficient level of muscular force is developed by the time impact occurs. Most of the studies on landing movements performed by humans, however, have not provided a quantitative analysis of EMG timing (Melvill Jones & Watt, 1971b; Greenwood & Hopkins, 1976; Dyhre-Poulsen & Laursen, 1991). To examine quantitatively how EMG timing was influenced by a change in fall height, we addressed this question by using a method which provides an objective measure of onset latency.

It has been suggested from work on catching (Lacquaniti & Maioli, 1989) that EMG amplitude before and after impact increases with the expected momentum of the collision. It is interesting to ask if this is a general CNS strategy for dealing with increasing levels of expected impact force. One study (Dyhre-Poulsen & Laursen, 1984) noted that prior to landing EMG amplitude seemed to depend on the height from which monkeys fell. As with the EMG timing, however, this relationship has not been formally or quantitatively investigated in landing movements. If EMG amplitude does scale with fall height, three testable mechanisms for achieving this come to mind: (1) the onset of EMG activity occurs at the same time prior to landing but increases more rapidly for the higher heights; (2) EMG amplitude grows at a constant rate but its onset is earlier as fall height increases; or (3) both. In the present experiments we tested these hypotheses. We varied flight time and impact momentum by having the subjects fall from a range of heights and we investigated how ankle joint rotation, pre-landing EMG amplitude and EMG timing were modulated with variation in fall height. The EMG measurements were taken from the m. soleus and its antagonist, m. tibialis anterior. The m. soleus is part of the triceps surae muscle–Achilles tendon complex which is an important muscle group involved in spreading the impact shock of landing over time (Devita & Skelly, 1992). We also examined EMG responses after touch-down as this activity may contribute to the later stages of braking and stabilization of body posture.

METHODS

Protocol

Six male subjects were asked to land on a force platform from take-off stages set at five different heights: 1, 0.8, 0.6, 0.4 and 0.2 m. All the subjects were physically active students from the department (means ± s.d.: age, 19.6 ± 0.8 years; weight, 73.2 ± 5.5 kg; height, 1.76 ± 0.07 m). The subjects were not experienced in jumping manoeuvres of the kind studied.
The bare-footed subjects were instructed to start and terminate the landing movement in a standing position, to take off and touch down with both feet, to lean forward with the body at take-off, rather than jumping off the stage, and finally to brake the fall smoothly. All the falls were self-initiated. Before the main experiment took place, the subjects were habituated by a period of landing practice consisting of 300 landings (twenty jumps from each of five fall heights performed during three sessions). This was done to reduce the variability of the main features of the movement.

Experimental sessions with the habituated subjects consisted of five groups of ten landings with rests of 2 min between each group. Between each group of ten landings, the height of the stages was reduced in descending order, starting from the highest height (i.e. 1 m). Prior to the participation in the experiments, written consent was obtained from the subjects. All experimental procedures were approved by the University Ethical Committee.

Apparatus and technical procedures

Measurements of the ankle joint (i.e. talocrural joint) angle of the right foot were taken using an electrogoniometer (Penny and Giles Biometrics, ‘M’ series twin axis; Penny and Giles Ltd, Blackwood, Wales). One endblock of the electrogoniometer was attached to the posterior surface of the calcaneus, whilst the other endblock was attached to the posterior surface of the middle third of the lower leg. The accuracy of the electrogoniometer was ±1 deg for each reading. Ninety degrees was defined as the right angle between the horizontal foot surface and a vertical line passing through the most lateral point of both the lateral condyle of the tibia and the lateral malleolus. In the electrogoniometer traces (Fig. 2) plantar flexion is represented by values larger than 90 deg, and dorsiflexion by values smaller than 90 deg.

The electrical activity (EMG) of the m. soleus and m. tibialis anterior of the right leg was recorded using bipolar silver electrodes (inter-electrode distance, 17 mm; diameter, 7 mm). The electrodes were part of an encapsulated amplifier mounted directly on the skin (gain, × 1000; input impedance, 10 MΩ; common mode rejection ratio at 50 Hz, 100 dB; band pass, 10−1000 Hz). They were developed from an original design by Johnson, Lynn, Miller & Reed (1977). Jumps from different heights by an individual subject were performed in a single experimental session so that the electrodes were left in place throughout the EMG recording. Movement artifacts were checked by tapping the electrodes, by creating passive movements of the limb and by shaking the wires. No relevant artifact was found using these procedures. Cross-talk between the EMG signals from different muscles was also checked by examining alternate contractions of the muscles on an oscilloscope before starting the experiment. We tested for the presence of cross-talk between (a) m. soleus and tibialis anterior and (b) m. soleus and m. gastrocnemius. For (a), we recorded the EMG from both muscles while the m. tibialis anterior was contracting maximally (isometric dorsiflexion). For (b) we recorded the EMG from m. soleus and m. gastrocnemius and we asked the subject to flex his knee against resistance while keeping the ankle joint at a constant angle (90 deg) as monitored by the electrogoniometer. No cross-talk was present.

A multicomponent force platform (Kistler 9281 A11, Kistler Winterthur, Switzerland; resonant frequency in situ 1-6 kHz) was used for measuring the vertical component of the ground reaction force after touch-down. The charge yielded by the piezoelectric load cells in the platform was converted into voltage output by a charge amplifier (Kistler 5006 ). A pressure pad switch (Radio Spares 317-140, 26 × 16 cm, Radio Spares, UK) was also placed near the edge of the stage in order to determine the instant of take-off.

Signal processing

EMG, goniometer, force platform and switch signals were amplified and then passed to separate channels of an analog-to-digital converter (1401 Cambridge Electronic Design, Cambridge, UK). The signals were sampled at 2 kHz and stored on a computer. The onset of the force platform signal (i.e. the touch-down) was used to trigger the recordings of all the other signals. The trigger was set so as to record 500 ms before and 200 ms after foot contact. Further processing of the signals took place off line. Part of the procedure involved averaging the records from ten separate jumps from each subject. The records were averaged electronically using the programme ‘Sigavg 5.20’ (Cambridge Electronic Design). The point at which the force record first indicated the landing impact was used as the reference point to line up the records of jumps prior to electronic averaging. Signals electronically averaged in this way are shown in Fig. 2.

For the ankle joint, we measured (a) the joint angle at touch-down; (b) the amplitude of joint rotation; (c) the minimum angle attained at the end of joint rotation and (d) the time from foot contact to minimum
joint angle. Visual inspection of the electrogoniometer traces revealed that ankle joint rotation during the period of 50 ms after foot contact could be approximated by a linear relationship. An estimate of the velocity of the ankle joint rotation was obtained by fitting a regression line through the ankle joint signal for each trial. For the vertical component of the ground reaction force ($F_z$), we measured the peak $F_z$ amplitude and the time from foot contact to peak $F_z$.

The EMG signals were subjected to additional processing. This involved taking the records from each single jump and defining the time at which the EMG signal started to build up prior to landing (the onset time point). Identification of this time point enabled us to define two time periods: the time period between take-off and this point (EMG onset latency) and the time period from this point until landing (EMG duration). The sum of EMG onset latency and EMG duration is a function of fall height. Theoretically, direct measurement of both variables would not be necessary. However, this relationship holds for bodies falling from a constant height. Since we could not be sure that subjects always faithfully followed the instruction not to jump, there could have been slight differences between the theoretical and experimental fall times. This means that – even for falls from the same height – the EMG onset latencies could have varied and the EMG durations could have been constant. Therefore, we found it more accurate to measure directly both variables. The method used to define the onset time point is now described.

An algorithm was designed to distinguish the later continuous build-up of EMG occurring shortly before foot contact from the early bursts of EMG occurring after take-off. We found that the timing of muscle activity preceding touch-down could not be analysed using methods previously employed by other authors (Sidaway et al. 1989; McKinley & Pedotti, 1992; Thomson & McKinley, 1995). McKinley & Pedotti (1992) and Thomson & McKinley (1995) defined the EMG activity onset as the point in time when the amplitude of the EMG signal exceeded and remained above the baseline confidence limits (95 %) for a given period of time (i.e. 10 ms). Cessation of activity was defined as the point in time when the EMG amplitude remained below baseline activity for a period of 10 ms. It should be noted that intertrial variability in the mean value of the EMG baseline would yield a different estimate of onset latencies even when the build-up of a continuous EMG activity occurs at similar latencies. Although intertrial variability in the EMG baseline might not have been a problem for the analysis of jumps from a constant height (i.e. 45 cm, as studied by McKinley & Pedotti, 1992), we found that this method was not suitable to analyse EMG traces for falls from different heights.

In a study on falls from different heights, Sidaway et al. (1989) estimated the EMG onset as the point in time when the rectus femoris muscle EMG first departed from a baseline bandwidth of ±25 mV. One problem with this method is that it does not take into account how long the EMG activity should last before being considered ‘preparatory EMG activity’. Therefore, the start of an EMG burst of amplitude larger than ±25 mV and lasting 10 ms would be considered onset latency, as would an EMG burst of similar amplitude lasting 100 ms.

We wished to use a method that could distinguish between ‘early’ (i.e. occurring soon after take-off), brief EMG bursts and ‘late’ (i.e. occurring shortly before foot contact), continuous EMG activity. Muscle activity occurring shortly before touch-down represents a motor control strategy which prepares the muscles to absorb an impact, whose time of occurrence and magnitude are anticipated by the CNS. In contrast, short-duration EMG bursts, often found shortly (~30–50 ms) after the subjects took off, are more likely to be associated with the event of take-off.

The raw EMG signal recorded during the period between take-off and touch-down (i.e. fall time) was full wave rectified (upper trace in Fig. 1), and a continuous (‘running’) integration of all the data points (with a time interval equal to the sampling rate of 2 kHz) was performed. The integrated EMG (IEMG) and the fall time were then normalized such that both the final IEMG value and the fall time were given the value of 1. Figure 1 (lower trace) shows the normalized IEMG plotted against the normalized fall time. It should be noted that the slope of the normalized IEMG line is dependent on the rate of increase of the EMG signal.

The normalized IEMG trace is then compared to a reference line with slope equal to 1. This reference line represents the relationship between normalized IEMG and normalized time when there is a constant level of EMG amplitude during the fall. If the baseline of the rectified EMG is flat throughout the fall, the rate of increase of the normalized IEMG with time is constant and the slope of the IEMG line is equal to 1, i.e. the same slope of the reference line. If, on the other hand, the baseline is characterized by brief EMG bursts, the increases in the slope of the normalized IEMG line would also last for a short time, returning afterwards to the same rate of increase as before the bursts occurred. The slope of the
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Fig. 1. Method for calculating EMG onset latency. The upper trace is the full-wave rectified EMG signal from m. soleus (one landing, subject S4) recorded during the period between take-off and touch-down (i.e. fall time). A continuous integration of all the EMG data points was performed with a time interval equal to the sampling rate (2 kHz). The integrated EMG (IEMG) and the fall time were then normalized such that both the final IEMG value and the fall time were given the value of 1. In the lower trace the normalized IEMG is plotted against the normalized fall time. The normalized IEMG trace is then compared to a reference line with slope equal to 1. This reference line represents the relationship between normalized IEMG and normalized time when there is a constant level of EMG amplitude during the fall. The EMG onset latency was defined at the point in normalized fall time when the distance between the normalized IEMG slope and the reference line was the greatest (d, lower trace). This occurs when there is a continuous increase in the EMG amplitude, i.e. when the slope of the normalized IEMG line becomes > 1.

normalized IEMG line would only increase in a continuous fashion, however, if a continuous increase in the EMG amplitude occurs. As a consequence, the slope of the normalized IEMG line becomes >1.

The EMG onset latency was defined at the point in time when the distance between the normalized IEMG slope and the reference line was the greatest (d, lower trace; indicated by an arrow in the upper trace; Fig. 1). This is the point when the slope of the normalized IEMG line starts to increase continuously (∆(normalized IEMG)/∆(normalized time) > 1), therefore indicating the onset of a continuous build-up of muscle activity. For the estimation of EMG onset latency, the only requirement is that the EMG increase has to be continuous, hence EMG bursts of short duration would be ignored. This condition was arbitrarily chosen in order to perform a repeatable quantitative analysis of the EMG onset latency.

The computer algorithm worked well in ~95% of cases. For a small percentage of the EMG traces (~5% of the total), the computer program calculated the onset at a very short latency from take-off.
Fig. 2. Landing from a 0·2 m height. Averaged recordings of ten landings from a height of 0·2 m are shown (subject S4). From top to bottom, the traces shown are: pressure switch, ankle joint angle, rectified EMG of m. soleus and m. tibialis anterior and vertical component \((F_z)\) of ground reaction force. The dashed line indicates take-off (as signalled by the pressure switch) and the continuous line indicates touch-down (as signalled by the onset of \(F_z\)). Three time periods can be distinguished: A, the time during which the subject is standing on the stage (to the left of dashed line); B, the time during which the subject falls (between dashed and continuous lines); C, the time during which the subject lands on the force platform and cushions the impact (to the right of continuous line). An increasing positive value in the electrogoniometer signal indicates plantar flexion, and vice versa for dorsiflexion. The additional calibration bar of ground reaction force is in body weights (bw).

\(<50\text{ ms}\). From examination of the original traces, it was consistently found that this was due to the presence of large and short-lasting \((\sim 30-50\text{ ms})\) EMG bursts similar to those described by Greenwood & Hopkins (1976), after which a more continuous EMG activity build-up could be seen. In such traces, the program gave two distinct \(d\) values (see above), of which the second one was chosen as it fulfilled the above-mentioned criteria.

The average amplitude of EMG activity was calculated over a period of 80 ms before touch-down (pre-landing EMG) and 100 ms after touch-down (post-landing EMG).

**Statistical analysis**

The data, averaged across ten trials for each height, were analysed using univariate one-way analysis of variance (ANOVA) with repeated measures. Where statistically significant effects were found \((P < 0·05)\) the Tukey’s *post-hoc* \(t\) test was used to determine the significance in the differences between the values found when falling from a height of 0·2 m and the remaining fall heights.
RESULTS

Original records and qualitative observations

The main types of data obtained from each subject during a jump are shown in Fig. 2. The records are the averages of ten landings from one subject (S4) falling from a height of 0.2 m. Take-off (dashed line, Fig. 2), as signalled by a pressure switch (top trace), was associated with plantar flexion of the ankle joint. The slowness of this action showed that subjects did not thrust the body upwards at take-off. This plantar flexion continued until approximately 200 ms before touch-down (continuous line, Fig. 2). From this time, the position of the foot was kept constant until touch-down occurred. On landing the impact caused a rapid foot dorsiflexion, followed by a gradual plantar flexion as a standing position was assumed.

The impact of landing is shown in the force platform record (bottom trace, Fig. 2; ‘bw’ on the y-scale represents body weight) which displays the gradual rise of $F_z$ before reaching its peak at $\sim 90$ ms after foot contact. After the largest peak, $F_z$ slowly decreased until the value of body weight was attained. Although only one peak is present in the average trace shown in Fig. 2, most of the individual force records (approximately 90%) were characterized by another peak that occurred at a latency of 10–15 ms from foot contact. This ‘early’ peak had an amplitude that was approximately 60% smaller than the largest peak described above. Only the peak with the maximum amplitude (i.e. peak $F_z$) was analysed quantitatively (see below).

Prior to the impact of landing there was, as predicted, preparatory EMG activity in the leg muscles (Fig. 2). Shortly before take-off, m. soleus and m. tibialis anterior (upper and lower EMG traces, respectively) are coactivated, although the former muscle is much less active than the latter. After take-off, a decrease in m. tibialis anterior EMG is seen, followed by a short burst of activity in m. soleus. A gradual increase in activity is seen at about 100 ms prior to touch-down, when coactivation of both muscles occurs. Following touch-down, both muscles are active throughout the foot dorsiflexion, after which EMG activity slowly decreases.

In addition to the typical pattern of muscle activation described above, other features were also observed. In most of the traces (approximately 70% of the records), the EMG of both muscles was found to decrease shortly (30–50 ms) before foot contact. The post-landing EMG also showed a decrease in activity after foot contact followed by a distinct burst of activity about 40 ms after contact. This feature becomes more evident for greater heights (see Fig. 3).

Figure 3 shows the rectified and averaged EMG records from m. soleus and m. tibialis anterior (left and right panel, respectively) during landing from a range of heights. All the records are from the same subject (S3). EMG activity from both muscles is low during the fall and starts to increase at about 100 ms before foot contact (indicated by the arrows). When landing from a height of 0.2 m (top trace), the duration of preparatory EMG activity from m. soleus is shorter than when landing from the remaining heights. This feature was common to all subjects. In contrast, EMG duration from m. tibialis anterior is rather invariant across fall heights. This feature was seen only in this subject, the EMG duration in the remaining subjects being characterized by a larger variability.

Visual inspection of the traces showed that the major part of the EMG activity in the m. soleus after touch-down is found during the period between the onset and termination of ankle joint rotation. This feature was seen across all the fall heights studied, irrespective of the fact that the duration of joint rotation decreased with increasing fall heights (see below). In other words, the major part of EMG activity after touch-down from m. soleus (left panel, Fig. 3) tended to be of shorter duration for higher heights than for lower ones. The m. tibialis anterior, on the other hand, tended to remain active also after joint rotation ended (right panel, Fig. 3).
Two main points should be noted in Fig. 3. First, the pattern of EMG activity is rather invariant despite a change in fall height. Second and in contrast, the amplitude of muscle activation before and after touch-down is strongly modulated with the height of the fall as is the timing of the activation from take-off. These variables are analysed quantitatively below.

**Quantitative relationships of variables to fall height**

We studied the effect of increasing fall height on the control of (a) foot rotation before and after touch-down, (b) the amplitude and latency of peak ground reaction force and (c) EMG timing and amplitude. No effect of practice (i.e. 10 trials) was found in the variables studied ($P > 0.7$).

**Ground reaction force**

The amplitude of the vertical component of the ground reaction force ($F_z$) increased with fall height as would be expected. The mean peak $F_z$ values following a fall for fall heights of 0.2, 0.4, 0.6, 0.8 and 1 m were equivalent to $\sim 3.9$, $4.7$, $5.6$, $6.9$ and $7.9$ average body weights, respectively. Interestingly, peak $F_z$ occurred at shorter latencies from touch-down when falling from greater heights in all the subjects. The mean values of time to peak $F_z$ (means $\pm$ S.E.M.) were $70 \pm 6$, $62 \pm 5$, $53 \pm 3$, $46 \pm 2$ and $40 \pm 2$ ms. This suggests that the stiffness of the lower limbs increased with increasing fall height (see below).
Fall height had a significant effect on both peak $F_z$ and time to peak $F_z$ ($F(4,20) = 22.77$ and $30.77, P < 0.01$, respectively). For both variables, post-hoc $t$ tests showed that the values found when falling from 0-6, 0-8 and 1 m were significantly different from the value found when falling from a height of 0-2 m ($P < 0.05$).

**Ankle joint rotation**

The amplitude of foot plantar flexion on approach to the ground tended to increase when falling from greater heights, i.e. +11% for a change in height from 0-2 to 1 m height (●, Fig. 4). This tendency was not statistically significant.

Foot dorsiflexion was faster when falling from higher heights than lower heights. The mean velocity of foot dorsiflexion after touch-down ranged from ~400 deg s$^{-1}$ when falling from 0-2 m to ~750 deg s$^{-1}$ when falling from 1 m. Nevertheless, the amplitude of ankle joint rotation after foot contact remained relatively constant irrespective of fall height (dashed lines, Fig. 4), the only change in joint excursion (+8 deg) occurring between 0-2 and 0-4 m heights. This suggests that the ankle joint was stiffer when landing from greater heights despite a doubling in the magnitude of $F_z$. The minimum ankle joint angle reached after the impact did not vary systematically with fall height (●, Fig. 4). Fall height had a significant effect on the amplitude of ankle joint rotation ($F(4,20) = 5.11, P < 0.05$), the only significant difference ($P < 0.05$) being between the value at 0-2 m and the remaining heights.

The time to attain minimum ankle joint angle, calculated as the time between touch-down and minimum angle at the end of joint rotation, tended to decrease with increasing fall height. The mean data from all the subjects (means ± s.e.m.) were: 133 ± 12, 116 ± 13, 106 ± 13, 83 ± 5 and 95 ± 10 ms, when falling from 0-2, 0-4, 0-6, 0-8 and 1 m heights, respectively. The effect of fall height was significant ($F(4,20) = 5.66, P < 0.01$).
Table 1. **Relationship between EMG amplitude and fall height**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Pre-landing m. soleus $r^2$</th>
<th>Pre-landing m. tib. ant. $r^2$</th>
<th>Post-landing m. soleus $r^2$</th>
<th>Post-landing m. tib. ant. $r^2$</th>
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<td>S1</td>
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<td>0.702</td>
<td>0.920</td>
<td>0.152</td>
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<tr>
<td>S2</td>
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<td>0.004</td>
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<td>0.920</td>
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<tr>
<td>S4</td>
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<td>0.947</td>
</tr>
<tr>
<td>S5</td>
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<td>0.837</td>
<td>0.709</td>
<td>0.930</td>
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<tr>
<td>S6</td>
<td>0.732</td>
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<td>0.833</td>
<td>0.847</td>
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</tbody>
</table>

The coefficients of determination ($r^2$) of the relationship between EMG amplitude (averaged across 10 trials for each height) and fall height are shown for each subject.

**EMG activity**

**EMG amplitude.** The muscle activity – averaged 80 ms before and 100 ms after touch-down – tended to increase linearly with increasing fall height (Fig. 5). However, this strategy was not observed in all subjects and both muscles (Table 1).

Table 1 shows the coefficients of determination ($r^2$) of the regression lines fitted through the data averaged across trials ($n = 10$). A linear increase with fall height in pre- and post-landing EMG amplitude from both muscles was found in three subjects (S3, S5 and S6). Although post-landing EMG amplitude from m. soleus also increased linearly in the remaining three subjects, their EMG modulation to height was more variable. For example, for subject S1 only the pre-landing EMG from m. soleus and post-landing EMG from m. tibialis anterior increased linearly. For subject S2, both the pre- and post-landing EMG amplitude of m. soleus exhibited a linear increase, while the EMG from m. tibialis anterior did not. Finally, for subject S4 a linear increase was found in the post-landing activity only from both muscles.

Figure 5 shows the data from all subjects expressed as a percentage of the values at 0.2 m. The increase in the pre-landing EMG amplitude with fall height (Fig. 5, upper panel) was significant in m. soleus ($F(4,20) = 4.69, P < 0.05$) but not in m. tibialis anterior. The post-landing EMG amplitude (Fig. 5, lower panel) was significantly affected by increasing fall height (m. soleus: $F(4,20) = 12.16, P < 0.01$; m. tibialis anterior: $F(4,20) = 6.66, P < 0.05$). Although the values in Fig. 5 are expressed in normalized form, the statistical analysis was performed on the raw data.

**Timing of pre-landing EMG activity.** The onset of preparatory muscle activity following take-off tended to occur later when falling from greater heights in both muscles. The absolute EMG duration was affected to a lesser extent by increasing fall height.

When subjects fell from a height of 0.2 m, the EMG from the two muscles was characterized by bursts of variable duration, before a continuous muscle activity could be detected (Figs 1 and 2). In two subjects (S2 and S5), an almost continuous period of muscle activity was often seen occurring from take-off to touch-down when falling from 0.2 m. In the rest of the subjects and for the remaining heights, the EMG was more distinctly quiet following take-off until the onset of muscle activity at about 200 ms prior to landing. The time from take-off to onset was termed onset latency, whilst the EMG activity from this point to impact was termed EMG duration (see Methods).
EMG ACTIVITY IN LANDING MOVEMENTS

Fig. 5. EMG activity amplitude before and after touch-down. The EMG amplitude was averaged over a period of 80 ms before touch-down and 100 ms after touch-down (upper and lower panel, respectively). The mean ± S.E.M. values from all the subjects are shown for m. soleus (■) and m. tibialis anterior (□). The values, plotted against fall height, are expressed as a percentage of the value found when falling from a height of 0-2 m (i.e. 100, indicated by the dashed line). Asterisks indicate significant differences (* P < 0.05) between the values at 0-2 m and the remaining heights.

For the m. soleus the mean onset latency (■, upper panel, Fig. 6) increased from 180 to 299 ms (equivalent to an increase of +66 %) when the fall height was increased from 0-2 to 1 m. The largest increase in onset latency, however, occurred when falling from 0-2 to 0-6 m, after which it levelled off. This feature was common to all subjects. The duration of pre-landing muscle activity (□) was less affected by an increase in the fall height, increasing from 86 to 115 ms (+34 %) with a change in fall height from 0-2 to 1 m. Fall height had a statistically significant effect on EMG onset latency ($F(4,20) = 16-01, \ P < 0-01$), but not on EMG duration.

Interestingly, the shape of the relationships between fall height and (a) EMG onset latency and (b) duration in m. tibialis anterior closely resembles the same relationships in m. soleus. The onset latency (■, lower panel, Fig. 6) increased from 200 to 306 ms (+53 %) for a change of fall height from 0-2 to 1 m. Although the EMG duration (□) increased from 66 to 108 ms (+70 %) for the same change in fall height, its absolute rate of change was lower than that for EMG onset latency, especially within the range 0-2–0-6 m fall height. Only the EMG onset latency was significantly affected by fall height ($F(4,20) = 17-25; \ P < 0-01$).
DISCUSSION

Our study is in qualitative agreement with previous studies (Melvill Jones & Watt, 1971a,b; Greenwood & Hopkins, 1976; Dyhre-Poulsen et al. 1991; McKinley & Pedotti, 1992; Thompson & McKinley, 1995) concerning the observation that the control of landing movements consists of more than reflex responses caused by the impact. However, our study provides new evidence on the quantitative aspects of the EMG amplitude and timing as a function of fall height.

It is clear that during the downward flight the leg muscles receive neural input to control the mechanical characteristics of the muscle in a manner appropriate to smoothly absorb the impact of landing. The neural input to the muscles responsible for controlling ankle joint rotation appears to affect both agonist and antagonist muscles in a very similar fashion, i.e. a similar scaling of EMG onset latency and amplitude to fall height. Our data suggest that this pattern of muscle activation is responsible for increasing ankle joint stiffness in response to increased joint rotation velocities.
Control of ankle joint stiffness

The kinematic and kinetic consequences of a larger EMG amplitude when falling from greater heights are difficult to determine, since the relationship between EMG amplitude and muscle tension is complex. However, the observed co-contraction of m. soleus and m. tibialis anterior – both before and after foot contact – is likely to be responsible for controlling joint stiffness. In this respect, it is interesting to note that despite a doubling in the magnitude of the force of impact as fall height is increased from 0.2 to 1 m, the amplitude of joint rotation caused by the impact increased little (Fig. 4). An invariant amplitude of ankle joint rotation when landing from different heights (0.32, 0.72 and 1.28 m) has also been reported by McNitt-Gray (1991, 1993), although the question of how this strategy is implemented at the neural level (i.e. EMG) was not addressed. An increased amplitude in the input perturbation (i.e. initial joint rotation velocity when falling from greater heights) and a constant output response (i.e. amplitude of joint rotation) clearly indicates that the stiffness of the ankle joint is increasing, so as to keep the joint excursion within a desired range of motion.

A high stiffness of the ankle joint is essential if foot dorsiflexion has to be stopped before it approaches its physiological range of movement (~60–70 deg from maximal foot plantar flexion to maximal foot dorsiflexion; Kapandji, 1970). When falling from greater heights, excessive rotation at the ankle joint may also be prevented by a larger extension of the forefoot and flexion of other joints with a larger range of motion, such as the knee and hip joints (M. Santello & M. J. N. McDonagh, in preparation). In this respect, it should be noted that rotation at the knee joint may affect rotation at the ankle joint by changing the length of the bi-articular m. gastrocnemius.

EMG timing

We found that the latency at which EMG activity was activated following take-off increased with fall height. The rate of increase of pre-landing EMG duration with height was lower than that found for EMG onset latency (Fig. 6), although this was clearer for lower heights.

Our results are in qualitative agreement with the observations reported by other authors who found that pre-landing EMG duration was little affected by fall height (Greenwood & Hopkins, 1976; Laursen et al. 1978; McKinley et al. 1983; Thompson & McKinley, 1995). Similar findings were also reported by Dietz & Noth (1978) in a study of humans falling on the arms. However, in a later study Dietz et al. (1981) reported that the duration of pre-landing activity in m. triceps brachii tended to increase with increasing fall time. Since no quantitative analysis of the EMG timing was performed in these studies (with the exception of Thompson & McKinley, 1995; but see Methods), we are unable to quantitatively compare their results with ours.

In contrast, Sidaway et al. (1989) reported that pre-landing EMG duration increased as a function of fall height (i.e. 0.72, 1.04 and 1.59 m), the EMG duration ranging from 60 to 149 ms. Due to the different procedures used to analyse EMG timing (see Methods), a quantitative comparison with their data is not possible. However, their Table 1 shows that the major increase in EMG duration (on average +66 ms) occurred only when fall height was changed from 1.04 to 1.59 m. Therefore, their results are also in agreement with our data within the range of fall heights that we studied.

The fact that the (a) EMG onset tended to increase with increasing fall height and (b) pre-landing EMG duration was affected to a lesser extent indicates that muscle activity before impact is timed relative to the expected instant of foot contact, rather than take-off. If the onset of EMG activity were timed to take-off, EMG onset latency would be constant.
regardless of fall height. Timing pre-landing EMG activity to foot contact makes intuitive sense, since touch-down is the event of interest that must be taken into account by the CNS in controlling the absorption of the impact. It is still a matter of debate, however, how the mechanisms responsible for timing motor actions operate in controlling interceptive movements, such as landing and catching.

Lee (1976) proposed that the information about the time of contact with a moving object can be extracted by visual estimation of its combined distance and velocity. More precisely, the ratio between the displacement and the velocity of the image on the retina (\( \tau \)), is equal to the time remaining before the object reaches the point of observation (Lee, 1980; Lee & Thomson, 1982). The \( \tau \)-hypothesis proposes that the onset of motor actions is geared to a value of \( \tau \), called ‘\( \tau \)-margin’. In other words, when the ratio between velocity and distance of an approaching object reaches a prescribed value, the initiation of motor actions is triggered.

In landing movements, where acceleration occurs, a given \( \tau \)-margin would be reached earlier for falls from higher heights, i.e. the EMG onset after take-off would occur earlier. Sidaway et al. (1989) tested whether the \( \tau \)-hypothesis could account for the control of EMG timing in landing from different heights. These authors reported that onset of EMG activity was triggered at an approximately fixed \( \tau \)-margin (i.e. ranging from 521 to 592 ms) regardless of fall height, thus causing the pre-landing EMG duration to increase with increasing height. However, this result could have been affected by some limitations in the method used to estimate EMG onset (see Methods) and the estimated value for the \( \tau \)-margin (Wann, 1996).

Although our experiments were not designed to directly test the \( \tau \)-hypothesis, our data indicate that activity was not timed at a constant \( \tau \)-margin. It should also be noted that tasks such as landing are generally characterized by a short fall time, where a strategy relying on \( \tau \) would give unreliable estimates of time to contact. As noted by other authors (Tresilian, 1995; Wann, 1996), the control of timing of motor actions in this type of task is more likely to rely on other variables, such as predicting fall time from measurement of the distance over which the fall occurs.

**EMG amplitude**

**Pre-landing EMG activity**

We found a highly consistent pattern of muscle co-contraction prior to foot contact across the range of fall heights studied. This feature is likely to be responsible for the increase in ankle joint stiffness discussed above. Pre-landing EMG amplitude tended to increase with increasing fall height in both muscles (Fig. 5, upper panel). Although we found in general a linear scaling of EMG amplitude with height, this ‘strategy’ was not systematically observed in all the subjects (Table 1).

A scaling of pre-landing EMG amplitude has previously been noted in experiments on cats (Prochazka, Schofield, Westerman & Ziccone, 1977), monkeys (Dyhre-Poulsen & Laursen, 1984) and man (Melvill Jones & Watt, 1971a,b; Dietz & Noth, 1978; Dietz et al. 1981), but the relationship between EMG amplitude and fall height was not systematically investigated. However, experiments on self-initiated falls in cats (McKinley et al. 1983) and unexpected falls in humans (Greenwood & Hopkins, 1976) indicate that no modulation of pre-landing EMG amplitude occurred when the height of the fall was changed.

The results of McKinley et al. (1983) could be explained by the fact that the initial phases of landing were characterized by a static limb position, caused by hyperextension of the forelimb, as reported by the authors. Scaling of pre-landing EMG amplitude, on the other hand, might be related to scaling limb compliance so as to control the joint rotation caused by
touch-down. If the limb position is static at touch-down these joint rotations do not occur, and muscle tension prior to foot contact might not need to be modulated with fall height. Data from human subjects (Greenwood & Hopkins, 1976) also seem to indicate that amplitude of pre-landing EMG remained constant when subjects were released at unpredictable times from 0-3, 0-4 and 0-6 m heights. It is conceivable that a key factor underlying modulation of EMG amplitude is that subjects must have control over the timing of the initiation of the movement.

The results obtained on the pre-landing EMG timing (see above) and amplitude indicate that EMG amplitude scaling was achieved by varying the rate at which EMG activity built up before the expected time of contact. As suggested above, visual estimation of fall height alone might provide the necessary input to predict fall time and scaling of EMG amplitude. A greater height would be associated with a longer fall time which, in turn, would be associated with experiencing larger ground reaction forces. As a result, limb compliance would be modulated accordingly. Sensorimotor memories of the dynamic interactions between the body and the environment have been shown to provide a remarkably robust mode of control in other types of movements such as grasping (Johansson & Westling, 1987; Johansson, 1991) and catching (Lacquaniti & Maioli, 1989; Lacquaniti, 1992).

An alternative mechanism for controlling EMG amplitude and muscle tension might rely on a continuous estimation of distance, time, or a function of these as the subject falls. If such an on-line mechanism is indeed used in the task we studied, onset of preparatory EMG activity appears to be geared to a threshold time period from foot contact, rather than a threshold distance from the floor or the instantaneous velocity of the subject. The length of this time period is likely to be controlled so as to ensure the attainment of different levels of muscle tension at foot contact. It should be noted that, when falling from different heights, triggering the onset of EMG at a given distance from the floor would not be desirable. In these circumstances, the instantaneous velocities at similar distances from the floor will be different, and the remaining time to contact will also vary. Therefore, contact might occur too early or late with respect to the onset of EMG activity.

Post-landing EMG activity

As found for the pre-landing activity, the amplitude of the post-landing EMG also tended to increase linearly with fall height (Table 1; Fig. 5). Pre-landing muscle activity tended to merge with the activity associated with the deceleration of the ankle joint rotation after foot contact.

Our protocol did not allow us to determine the contribution of reflex mechanisms to this activity. However, tendon taps on our subjects indicate that short-latency spinal reflex activity of m. soleus cannot be evoked any sooner than \( \sim 35 \) ms after touch-down. In most of the traces the EMG pattern after touch-down was characterized by a continuous activity, where no distinct EMG burst or peak could be easily defined. Some of the records (Fig. 4) do show evidence of increased activity at a latency of \( \sim 40 \) ms but we cannot unequivocally label such activity as being ‘reflex‘. In fact, Dhyre Poulsen et al. (1991) have shown that the H reflex is inhibited at this time. In any case, a reflex response would contribute to muscle tension at a latency when ground reaction force has already reached a significant amplitude. Despite these considerations further work needs to be carried out to establish the exact contribution of the stretch reflex to post-landing EMG activity.

Although most of the above studies on landing movements emphasized a dichotomy between reflex and ‘preprogrammed’ muscle activity after foot contact, more recent studies (Lacquaniti & Soechting, 1986; Gielen, Ramaekers & van Zuylen, 1988; Smeets & Erkelens, 1991) suggest that the stretch reflex does not control a single variable at a single joint (i.e. muscle length or
tension), but rather constitutes a more global mode of control of the whole limb (Lacquaniti, 1992).

**Common features in the control of landing and catching movements**

Interestingly, EMG timing and amplitude are controlled in a very similar fashion in catching movements (Lacquaniti & Maioli, 1989), where co-contraction of forearm muscles occurs before and after contact with the ball is made. This pattern of muscle activation was found to be responsible for modulating the time history of hand compliance (Lacquaniti, Borghese & Carrozzo, 1992). Furthermore, (a) the amplitude of EMG activity – before and after contact with the ball – of agonist and antagonist forearm muscles scales linearly with ball momentum and (b) the EMG onset latency is modulated with fall height to a greater extent than EMG duration. Lacquaniti and co-workers (Lacquaniti & Maioli, 1989) further proposed that preparatory and reflex EMG activity are both involved in the control of hand compliance. These observations strongly suggest that the control of interceptive actions is based on a similar control strategy irrespective of the muscles involved and their functional role.

**Conclusions**

The attainment of a desired level of muscle tension at foot contact seems to be achieved by (a) timing the onset of muscle activation to foot contact and (b) modulating the rate of EMG build-up. This strategy is likely to rely on sensorimotor memories about ground reaction forces associated with the height of the fall.

A successful landing, however, does not depend solely on the adjustment of a desired limb compliance at touch-down. The time history of limb compliance after foot contact must also be controlled, if joint rotation is to be controlled from touch-down throughout the deceleration of the whole body. The fact that EMG amplitude before and after touch-down was scaled to fall height suggests that the two time periods are not separable from a motor control perspective, as has been implied in previous studies on landing.

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The control of timing and amplitude of EMG activity in landing movements in humans
M Santello and MJ McDonagh

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