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J. Physiol. 1976;254;507-518

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MUSCLE RESPONSES DURING SUDDEN FALLS IN MAN

BY RICHARD GREENWOOD AND ANTHONY HOPKINS

From the Department of Neurology, St Bartholomew's Hospital, London EC1A 7BE

(Received 10 June 1975)

SUMMARY

1. E.m.g. activity in soleus during an unexpected fall is found to be more complex than that described by Melvill Jones & Watt (1971b). After a silent period of about 80 msec an initial peak of activity lasts until about 200 msec after release. In falls from sufficient heights a second peak of activity occurs before landing.

2. The initial peak of activity is found in muscles throughout the body and is absent during falls in which the subject releases himself. It is suggested that this initial peak is a startle response to release and on landing during the initial peak any deceleration due to tension in the leg muscles is in part coincidental.

3. The second peak of activity is found in muscles of the lower limbs. Its timing is related to the timing of landing. It is suggested that this is the activity concerned in the voluntary control of landing.

4. No initial peak of activity could be recorded in two patients with absent labyrinthine function.

INTRODUCTION

The interaction of central commands and segmental afferent input during walking in the cat was described by Engberg & Lundberg (1969). They found that electromyographic (e.m.g.) activity in extensors of the leg begins 5–10 msec before the foot touches the ground. This activity is therefore not initiated by stretch of the extensors at the start of the stance phase. Rather, it is a centrally programmed event, anticipating stance, organized at spinal and probably supraspinal levels. A similar conclusion was drawn by Melvill Jones & Watt (1971a), who reported observations on the e.m.g. activity of gastrocnemius in man during a single downward step. They found that activity begins before contact with the ground. In addition they found no evidence that long loop stretch reflexes were concerned in landing. In order to test the hypothesis that the response before landing was pre-programmed centrally they dropped
subjects suspended from an electromagnet at unexpected moments (Melvill Jones & Watt, 1971b). They found a consistent response in gastrocnemius beginning about 74 msec after release. They suggested that this response originated from the otolith apparatus due to the change in acceleration at the onset of fall.

Since the latency of gastrocnemius e.m.g. was 74 msec and the electromechanical coupling time 28 msec, they suggested that falls of less than 102 msec (5·1 cm) do not allow sufficient time for build up of tension in the muscles concerned with decelerating the body, so that the body is jolted on landing from short falls. Falls of over 5·1 cm allow some build up of muscle tension, and they suggested that after about 195 msec voluntary control of muscle activity results in a ‘truly comfortable, well controlled landing’.

However, Melvill Jones & Watt only studied gastrocnemius activity during free fall from heights of up to 20 cm, which last about 200 msec. We have studied the activity in a number of muscles in subjects dropped from greater heights. Our findings suggest that the contribution of the response described by Melvill Jones & Watt during unexpected fall to deceleration on landing is coincidental. This response is probably related to the startle response. Voluntary control of landing is manifest by a second peak of activity following this initial, or startle, response. We have found evidence that the vestibular apparatus is involved in the initiation of this response. A preliminary report of some of these findings has been published elsewhere (Greenwood & Hopkins, 1974).

**METHODS**

Normal volunteers were suspended in a parachute harness by a rope passing through freely running pulleys to a metal plate held by an electromagnet (Fig. 1). The distance of the subjects’ toes above the landing platform could be varied by an electric winch. Subjects were instructed to concentrate their attention on the landing platform while hanging before the fall. Switching off the current to the electromagnet resulted in an unexpected fall after a delay of about 500 msec due to decay of the magnetic field. The exact moment of release was indicated by a microswitch operated by separation of the plate from the magnet; this triggered a crystal controlled oscillator (Devices Digitimer 3290). The moment of landing was also indicated by a microswitch located in the landing platform. Sometimes the angle of the ankle or knee during the fall was measured by means of a potentiometer. In other experiments the subject hung by his hands from a bar attached to the electromagnet. He could either let go of this voluntarily, the moment of release being indicated by an accelerometer, or alternatively the bar itself was released unexpectedly using the electromagnet. E.m.g. activity was recorded using 8 mm silver-silver chloride surface electrodes placed 20 mm apart over the muscle under study. To minimize movement artifacts, signals were amplified by small pre-amplifiers strapped to the limb. These were then further amplified and filtered and also rectified and integrated. The integrator (Devices Signal Processor/Audio Unit 4010)
was re-set every 20 msec by a Devices Gated Pulse Generator 2521 so that the amount of activity in each epoch could be measured. All signals were displayed on a Tektronix D13 oscilloscope, photographed and enlarged for measurement.

The time taken for a fall from a given height was found to be slightly greater than the calculated time for free fall using Newtonian physics. Although this disparity could have been due to movements of the limbs during fall, we found that the duration of the fall from a given height was the same for both a subject and for lead weights of the same mass. We therefore concluded that the acceleration during falls was slightly decreased by friction and inertia in the system to about 0.9 g.

The height of each fall in any experiment was varied randomly. The first four falls in each experiment were rejected. In some experiments auditory cues of release, consisting of the noise of the plate separating from the electromagnet, were excluded by adding a randomly variable electronic sound delivered through head-phones, and visual cues of the moment of release were excluded by a blindfold.

Four subjects with defective labyrinthine function were examined with their understanding and consent. One (P.M.), whose name was given to us by Dr P. A. Merton, was also studied by Matthews & Whiteside (1960) during seated falls.

Case reports:

(1) P.M., a man of 41 years, developed a pseudomonas meningitis due to chronic otitis media in 1948. During a 13-day period he received 17 g intramuscular and 1.3 g intrathecal streptomycin. A left radical mastoidectomy was performed. He was unsteady immediately following the illness but there were no other neurological signs. At that time no nystagmus could be induced by irrigation of the external auditory meati with water at 0°C. A recent repeat irrigation with water at 0°C
produced a minimal response from the right ear, and no response from the left. Impulsive rotation to the right and left at 40 and 60°/sec resulted in nystagmus to the right and left respectively lasting only 2–4 sec. Normally such rotatory accelerations produce nystagmus lasting 30 sec. Hearing was essentially normal.

(2) W.B., a man of 53 years, complained in 1970 of unsteadiness in the dark. On three occasions he found himself to be disorientated while swimming under water. Examination in 1970 showed ‘minimal residual left labyrinthine function’. It was thought that this was due to an unknown amount of streptomycin received during treatment of a septicaemia in 1954. Recent caloric testing, using water at 0° C, showed no response from either ear. Impulsive rotation to the right at 60°/sec produced nystagmus to the right for 3–4 sec only on rotation to the right. No nystagmus was produced on impulsive rotation to the left. Hearing was essentially normal.

(3) J.W., a man of 18 years, developed a pneumococcal meningitis in 1967. He was unconscious for 10 days. At this time he received 14 g intramuscular and 0·4 g intrathecal streptomycin. Since then he has had occasional grand mal and atonic seizures. Recent testing revealed absent caloric responses bilaterally to water at 0° C. No nystagmus was produced by impulsive rotation to the right and left at 60°/sec. This subject had a total perceptive deafness.

(4) D.E., a man of 34 years, developed episodes of vertigo and oscillopsia in 1968. Over the next five years there was a gradual decline in the duration of the nystagmus produced by caloric testing with water at 0° C. For the last year he has had no further attacks. Recently, there was no response at all to water at 0° C, nor was there any response to impulsive rotation to the right and left at 60°/sec. Hearing was essentially normal. We do not know the cause for this man’s illness.

RESULTS

Subjects hanging in the parachute harness were relaxed and no activity was recorded from soleus before release. Fig. 2 shows the record of a typical experiment for an unexpected free fall from 95 cm. After release there is, initially, no activity in soleus; then a burst occurs with about the latency described by Melvill Jones & Watt (1971b) in their study of falls lasting up to 200 msec. However, the longer falls that we have studied show that this initial burst of activity terminates by about 200 msec. There is then a period of relative silence followed by a second peak of activity before landing. Our evidence suggests that these two peaks are different phenomena and descriptions of each are given separately.

The initial peak

Unexpected free fall

The mean latency of onset of the initial peak in soleus for falls of 20 cm was 81·6 msec (s.e. of mean = 1·0 msec, n = 32). The difference between this value and this reported by Melvill Jones & Watt (74·2 msec, s.e. of mean = 1·4 msec) may be accounted for by the difference in recording sites—soleus as opposed to gastrocnemius. Falls from greater heights, up to 120 cm, tended to have a rather more variable latency but the mean
Fig. 2. Upper trace: soleus e.m.g. during unexpected free fall from 95 cm; lower trace: integrated activity from the same muscle. Integrator re-set every 20 msec.

Fig. 3. E.m.g. activity in the periocular muscles, biceps, forearm extensors and soleus in a normal subject during an unexpected free fall from 20 cm.
Table 1. Latency of onset of e.m.g. activity during free fall from 20 cm in various muscles of normal subjects and of two subjects with absent labyrinthine function (means and s.e. of mean)

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Periocular muscles</th>
<th>Forearm extensors</th>
<th>Quadriceps femoris</th>
<th>Hamstrings</th>
<th>Tibialis anterior</th>
<th>Soleus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latency (msec)</td>
<td>37-8 ± 1-6</td>
<td>68-3 ± 2-8</td>
<td>70-4 ± 1-9</td>
<td>72-1 ± 1-2</td>
<td>71-5 ± 1-2</td>
<td>80-7 ± 1-5</td>
</tr>
<tr>
<td>No. subjects</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>4</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>No. falls</td>
<td>20</td>
<td>33</td>
<td>48</td>
<td>37</td>
<td>20</td>
<td>21</td>
</tr>
</tbody>
</table>

Abnormal subjects

| Latency (msec) | 39-1 ± 0-38       | 66-4 ± 1-8        | 78-75 ± 1-7       |
| No. subjects  | 2                 | 2                 | 2                 |
| No. falls     | 14                | 14                | 14                |

was not significantly different. The latency of the maximum and end, and amplitude of the initial peak were also not related to the height of fall. In a few subjects, the amplitude of the initial peak was greater for the first one or two than for subsequent falls but after this no habituation occurred, even after as many as thirty falls. As noted in Methods, the first four falls during each experiment were rejected.

The distribution of e.m.g. activity in muscles other than soleus was also studied. All muscles that we have examined, whether flexor or extensor, show a period of silence after release followed by a burst of activity as was found in soleus. This is illustrated in Fig. 3 which shows the response from four muscles during an unexpected fall from 20 cm. Although the amount of activity in biceps was not great, in other muscles, notably the periocular muscles, the burst of activity was as large as in soleus, using a similar interelectrode distance. Table 1 shows the latency of onset of e.m.g. in a number of muscles throughout the body during unexpected falls from 20 cm in five subjects.

Voluntary release compared to an unexpected fall

If the subject released himself voluntarily, by letting go of a bar, an accelerometer indicating the moment of release, it was found that the initial peak was almost absent in three subjects during falls from 20, 55 and 80 cm in all muscles examined including the periocular muscles (Fig. 4).
Fig. 4. E.m.g. activity in soleus during voluntary O—O and unexpected •—• falls from 80 cm. Each curve is the average of three falls in one subject. Points showing the amplitude of successive 20 msec epochs of integrated e.m.g. activity have been joined.

The second peak

Unexpected free fall

In falls from more than about 20 cm there was a period of relative silence between the end of the initial peak and a second burst of activity which we have called the second peak. The latency of onset of the second peak was often poorly defined but when a clear distinction was seen the second peak began about 200–300 msec after release. The onset was never seen earlier than 200 msec after release. The timing of the maximum of the second peak, defined as the start of the largest epoch in the second peak, was not related to the timing of release, occurring later in falls from greater heights. However, inspection of the data showed that it occurred at a consistent time before landing in falls between 50–120 cm (Fig. 5). The maximum was followed by a short period of diminished e.m.g. activity before landing.

The distribution of e.m.g. activity in muscles other than soleus was examined during free falls lasting more than 200 msec. The second peak was found to occur consistently in flexors and extensors of the knee and ankle. However, in the periocular muscles, biceps and forearm extensors the second peak was not consistently seen. This is illustrated in Fig. 3 which shows a second peak of e.m.g. activity in soleus but not in the periocular or arm muscles before landing.
Fig. 5. Soleus activity during unexpected free falls in one subject from: 20, ○; 55, ■; and 95, Δ, cm. Each curve is the average of three falls for each height. Points showing the amplitude of successive 20 msec epochs of integrated e.m.g. activity have been joined.

<table>
<thead>
<tr>
<th>Patient and age</th>
<th>Calorics at 0 °C</th>
<th>Impulsive rotation at 40 or 60°/sec to the right</th>
<th>Soleus e.m.g. activity during free fall</th>
</tr>
</thead>
<tbody>
<tr>
<td>P.M. (41)</td>
<td>Minimal response, right ear</td>
<td></td>
<td>200 μV Land</td>
</tr>
<tr>
<td>W.B. (53)</td>
<td>Absent</td>
<td></td>
<td>200 μV Land</td>
</tr>
<tr>
<td>J.W. (18)</td>
<td>Absent</td>
<td></td>
<td>200 μV Land</td>
</tr>
<tr>
<td>D.E. (34)</td>
<td>Absent</td>
<td></td>
<td>200 μV Land</td>
</tr>
</tbody>
</table>

Fig. 6. E.m.g. activity in soleus during a 15 or 20 cm free fall in four subjects with defective labyrinthine function. Rotational and caloric tests showed that subjects P.M. and W.B. still had minimal labyrinthine function.
The effect of restricting afferent inputs

Further experiments were performed using normal subjects and those with neurological disease to define the sensory information necessary for the initial peak of muscle activity.

Normal subjects

We examined the effects of blindfolding and of excluding auditory cues during free falls from 20 cm. It was found that the timing of the onset and peak and the amplitude of the initial peak were unchanged. The latency of onset of muscle activity in the periocular muscles and flexors and extensors of the upper and lower limbs was also unchanged.

Subjects with absent labyrinthine function

Experiments were performed on each patient, whose clinical details are described in Methods, with and without blindfolding and the exclusion of auditory cues. The response in each subject to a short free fall is shown in Fig. 6.

The latency and characteristics of the initial and second peaks of muscle activity in subjects P.M. and W.B., who had minimal responses on caloric or rotational testing, or both, were the same as in normal subjects, despite exclusion of visual and auditory cues. Subjects D.E. and J.W. had no response to caloric and rotational tests. The initial peak of muscle activity was never seen in J.W., who was totally deaf. It was present in D.E. but disappeared when all auditory cues were excluded. In these two subjects, during 20–25 cm free falls, muscle activity in soleus and the periocular
muscles showed a variable latency. Activity in soleus gradually increased until landing occurred (Fig. 7). This abnormal response was the same whether J.W. or D.E. were blindfold or not. (They had previously inspected the height of fall.) During free falls from 50 cm muscle activity in soleus was not divided into two peaks, as in normal subjects, but instead soleus activity increased to a single peak up to the moment of landing, much as the second peak of activity in normal subjects.

**DISCUSSION**

The results of these experiments show that the pattern of e.m.g. activity during an unexpected fall in man only becomes clear during falls lasting more than about 200 msec. There is initially a widespread burst of e.m.g. activity throughout the body which is present in all muscles that we have studied. This initial peak begins between about 60 and 90 msec after release in muscles of the upper and lower limbs and lasts up to about 200 msec after release. In falls of sufficient duration a period of relative silence is then seen, followed by a second peak of activity only in muscles of the lower limbs. This second peak is timed to occur before landing. The short falls, lasting for only 200 msec or less, employed by Melvill Jones & Watt (1971b) revealed only the initial peak.

Since the initial peak occurs during free falls from 120 cm (520 msec), when clearly the activity of the initial peak is superfluous to landing, release rather than landing is implicated in its genesis. Any deceleration on landing during the initial peak due to tension in the leg muscles may therefore be, at least in part, coincidental. In support of this we have found activity in muscles which are not obviously concerned with landing – the periocular muscles.

The presence of this activity in periocular muscles suggests to us that the initial peak of activity is the electrical counterpart of a startle reaction in response to release. The startle reaction was described and recorded photographically by Strauss (1929) and Landis & Hunt (1939). The latter authors emphasized that the startle reaction is an integrated motor response, the pattern of which is relatively independent of the type of sudden afferent input. They found that the startle reaction was variably inhibited if the subject triggered the stimulus himself. In our experiments, the initial peak was absent if the subject released himself. They also found that the most consistent component of the response was the blink reflex. The consistent activity in the periocular muscles during fall occurs with a latency (about 38 msec) similar to that of the second component of the blink reflex recorded with surface electrodes after a loud sound (Suhren, Bruyn & Tuyman, 1966; Shahani, 1968). There have not been any detailed studies...
in normal subjects of the latency of onset of e.m.g. activity in other muscles. However, Jones & Kennedy (1951) obtained polygraph recordings of the reaction to a pistol shot, and inspection of their Fig. 2 suggests that activity in quadriceps femoris and gastrocnemius begins with about the same latency after a shot as we have found after release.

In some subjects we found that the latency of the e.m.g. of the initial peak was more variable during falls from higher heights. During falls of more than 20 cm the initial peak becomes superfluous to landing. It is known that supraspinal influences change the latency of polysynaptic reflexes. Thus the latency of a habituated blink reflex is shortened by apprehension (Rushworth, 1962). We suggest that in these subjects at lower heights voluntary set contributed to facilitation of the neurones concerned in the genesis of the initial peak. Thus the latency of onset of e.m.g. activity in falls from less than about 20 cm was minimal while the latency during higher falls was more variable.

What are the pathways through which the nerve impulses responsible for the initial peak travel? Melvill Jones & Watt (1971b) suggested that the response originated from the otolith apparatus, transient stimulation of which occurs when the body passes suddenly into a zero gravitational state. However, this stimulus must act upon peripheral limb and visceral receptors as well, as suggested by Matthews & Whiteside (1960). These receptors might be effective in triggering the initial startle response at the onset of the fall. Our patients, J.W. and D.E., both had absent vestibular function as judged by caloric and rotational tests, which, of course, do not test otolith function. The initial startle response was absent whether visual input was excluded or not in the case of J.W., who was deaf; in D.E., whose cochlear function was normal, the initial response was absent if auditory input was excluded. This must indicate that the stimulus for this response is derived very largely, if not entirely, from the vestibular apparatus. Our data does not allow us to decide whether the semicircular canals or the otolith organs were responsible for this response since the head of the subject was not fixed during fall. Our results confirm previous observations that labyrinthectomized man is unable to react to a sudden change in effective gravitational force, such as occurs with a sudden rapid tilt (Martin, 1965) even when visual pathways are available.

Melvill Jones & Watt (1971a) suggested that during a downward step deceleration was brought about by an accurately timed burst of pre-programmed muscle activity rather than a stretch reflex resulting from the mechanical event of landing. The inference from their two papers is that the control of landing during a downward step (1971a) is effected by muscle activity similar to that found to control landing from an unexpected free fall from heights of up to 20 cm (1971b). Downward stepping is a
voluntary manoeuvre, imposed by the subject upon himself, as in our experiments in which the subject releases himself, when we found the initial peak or startle reaction to be almost completely absent. We would emphasize therefore that the initial activity during an unexpected fall, which we believe to be a startle reaction, is qualitatively different from that found during a downward step.

The second peak of muscle activity occurring during an unexpected fall was found only in muscles of the lower limb. The timing of the maximum of the second peak was related to the timing of landing. This suggests that the second peak if responsible for the smooth, voluntary control of landing and that on landing during the initial peak from a short fall deceleration due to tension in the leg muscles is probably to a large extent coincidental.

This work was supported by the Joint Research Board of St Bartholomew’s Hospital and Medical College. We thank Mr Christopher Sheldon and Mr John Hare for technical assistance.

REFERENCES


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