The Effect of Long Duration Spaceflight on Postural Control During Self-Generated Perturbations

Charles S. Layne[1]

Ajitkumar P. Mulavara[2]

P. Vernon McDonald[2]

Casey J. Pruett[3]

Innessa B. Kozlovskaya[4]

Jacob J. Bloomberg[5]

[1] 104 Garrison, Department of Health and Human Performance, University of Houston, Houston
Texas, USA, 77024,
Telephone 713.743.9868, fax 713.743.9860,
e-mail - clayne2@bayou.uh.edu

[2] Wyle Life Science Laboratories, Houston, TX, USA

[3] TECMATH, Troy, MI, USA

[4] Institute of Biomedical Problems, Moscow, Russia
[5] Life Sciences Research Laboratories, NASA/Johnson Space Center, Houston, TX, USA

ABSTRACT

This report is the first systematic evaluation of the effects of prolonged weightlessness on the bipedal postural control processes during self-generated perturbations produced by voluntary upper limb movements. Spaceflight impacts humans in a variety of ways, one of which is compromised postflight postural control. We examined the neuromuscular activation characteristics and center of pressure motion (COP) associated with arm movement of eight subjects who experienced long duration spaceflight (3-6 months) aboard the Mir space station. Surface electromyography (EMG), arm acceleration, and COP motion were collected while astronauts performed rapid unilateral shoulder flexions prior to and after spaceflight. Subjects displayed compromised postural control after flight as evidenced by modified peak-to-peak COP anterior-posterior and medio-lateral motion and COP pathlength relative to preflight values. These changes were associated with disrupted neuromuscular activation characteristics, particularly after the completion of arm acceleration (i.e. when subjects were attempting to maintain their upright posture). These findings suggest that although the subjects were able to assemble coordination modes that enabled them to generate rapid arm movements, the subtle control necessary to maintain bipedal equilibrium evident in their preflight performance is compromised after long duration spaceflight.

Key words: spaceflight, postural control, neuromuscular activation

INTRODUCTION

Astronauts returning from spaceflight exhibit a variety of postural control problems. These include deficits while balancing on rails of varying widths (13) increased sway of the body's center of gravity (COG) (33,34) modifications in body segment motion (1) and increased response latencies to external perturbations (18). Preliminary reports indicate that returning astronauts have difficulty assembling the coordination strategies necessary to efficiently perform rapid voluntary arm raises during bipedal stance (18,20). These deficits are accompanied by decreases in lower limb strength (12), in part stemming from muscular atrophy (24) and hyperactive proprioceptive and neuromuscular reflexes (16,17,35). Moreover, returning astronauts experience alterations in vestibular system functioning (36), head movement control (4) and abnormal proprioceptive functioning (38) which also can contribute to postural control deficits.

Previous research examining postural control has primarily centered on various manipulations of sensory input or responses to external perturbations. Few investigations have assessed returning astronauts' ability to perform voluntary limb movements with the constraint that bipedal equilibrium must be maintained (7,8,30). The present study addresses the question of whether humans who have experienced extended periods of microgravity (3-6 months) are able to perform a vigorous arm movement while maintaining bipedal upright stance.

The arm movement utilized, a rapid unilateral arm raise, has been used extensively as a method to investigate the ability of normal and patient populations to control self-generated postural perturbations. Belen'kii and his colleagues (2) were the first investigators to report that trunk and lower limb muscles are activated prior to the initiation of arm motion. This "anticipatory" postural activity is specific to the particular arm raise task (e.g. unilateral versus bilateral, weighted versus non-weighted) and counters the potentially destabilizing reactive forces arising from upper limb motion (6). A variety of patient populations with postural control problems also have difficulties while performing voluntary arm movements. These difficulties are manifested as inappropriate anticipatory neuromuscular activation strategies, increased sway of the COP, and decreased arm movement velocity relative to normal subjects (14,37). Therefore, the rapid arm raise is an ideal task with which to evaluate the ability of returning astronauts to perform a voluntary limb movement while maintaining an upright bipedal posture. We hypothesized that during the arm raise task after spaceflight subjects would display diminished postural stability quantified using COP measures (see Methods). Measures of COP motion as indices of postural stability have often been used to assess differences in postural control between normals and patients (9, 23,25) and healthy young adults and the elderly (11, 26,28).

In addition to determining if returning astronauts could maintain preflight levels of postural control and arm acceleration, we were interested in how neuromuscular activation characteristics associated with the arm raise were affected by spaceflight. Therefore, we assessed potential modifications in muscle activation strategies in response to long duration spaceflight. We were particularly interested in two questions regarding neuromuscular activation: whether, after spaceflight, subjects could produce neuromuscular patterns that were similar to preflight patterns 1) during the movement initiation phase and 2) after the self-generated perturbation (i.e. after arm acceleration was completed). Previous investigators have detailed changes in proprioceptive functioning and loss of muscle strength, both of which may impact the ability to produce task-appropriate neuromuscular control (24,38). Thus, we hypothesized that the neuromuscular patterns associated with the maintenance of postural stability in response to the reactive forces produced during the arm movement would be more disrupted after flight than those associated with the inittiation of the arm movement.

METHODS

Subjects

Eight subjects (two US astronauts, six Russian cosmonauts, mean age 43 ± 8 years) who experienced three to six months of microgravity aboard the Mir space station participated in this study. All were volunteers and had completed the NASA Institutional Review Board for Human Research Informed consent form.

Protocol

The task comprised 15 right shoulder flexions performed from a bipedal standing position. Subjects assumed a comfortable stance on a force plate (Kistler Instruments Inc., Amherst, New York), arms resting at their sides with the right elbow extended. The self-initiated movements consisted of first closing their eyes and then raising the arm by flexing the shoulder as rapidly as possible until the arm was parallel to the force plate. Throughout the task subjects were required to maintain their upright bipedal stance (i.e. no stepping or falling). Testing was performed approximately 10 days before spaceflight and, with one exception, one day after landing. One crewmember was tested on landing day. All subjects were well-practiced prior to the final preflight data collection. To ensure that subjects adopted the same foot placement before and after spaceflight, during preflight testing the borders of the feet were marked relative to the axes of the force plate. These markings were then used to properly position the subjects during postflight testing.

Data Collection and Processing

Tangential arm acceleration was measured using a uniaxial accelerometer (Kistler Instruments Inc.) mounted on a wrist splint. Additionally, ground reaction forces from the force plate (Kistler Instruments Inc.) and surface EMG from the right anterior deltoid (RAD), left (LBF) and right biceps femoris (RBF), left paraspinals (LPA), right lateral gastrocnemius (RGA) and right tibialis anterior (RTA) were obtained during data collection. These muscles were monitored because they are activated in most subjects in preparation for and/or during the arm raise task (2,7,14,22). After cleaning the skin, preamplifier electrodes (Therapeutics Unlimited Inc. Iowa City, Iowa) were attached to the skin over the muscles using adhesive collars. To prevent motion artifacts, the electrodes were further secured with neoprene wraps or hypoallergenic tape. All data were digitally sampled at 500 Hz. Due to temporal and programmatic constraints we were unable to obtain kinematic data.

Arm angular acceleration:

For each trial, the gravity component of the tangential arm acceleration was removed and the remaining linear acceleration component was divided by the radius of the arm to provide arm angular acceleration (29). Arm movement initiation was determined using the resulting angular acceleration waveform. For each trial, the time of arm movement initiation was used to synchronize the force plate and EMG data records that were collected on separate computers. The arm acceleration signal was recorded on both computers which enabled data synchronization. Arm movement initiation time was also used to obtain a data window for each trial that consisted of 1 second before and 1.25 seconds after arm movement initiation. The 1 second interval prior to arm movement initation was chosen to obtain a quiet EMG and COP baseline prior to the initiation of "anticipatory" neuromuscular activity and associated COP motion. The 1.25 second interval after arm movement initation encompassed the arm movement itself and the time required for the subjects' COP maximal excursion to be reached and begin to return to its starting point. This data was of appropriate length to investigate the features of postural stability and the underlying neuromuscular activation during the arm raise task. Using the appropriate zero crossing of the accelerometer waveform, two movement phases were identified: 1) the *initiation* phase - from the beginning of the data record through the end of arm acceleration and 2) the *recovery* phase - from the beginning of arm deceleration until the end of the data record, (Figure 1). The division of the trial into the initiation and recovery phases enabled us to assess the similarity of the initial phasic activation features used to prepare for and initiate arm movement separately from the activity primarily used to arrest the arm motion and maintain/regain bipedal postural control. Peak acceleration values were obtained from the acceleration records of each trial.

Insert Figure One about here

Center of pressure:

For each trial, the COP signals were obtained from commercially available software (Bioware 2.0, Kistler Instruments Inc.) and then low pass filtered with a 10 Hz cut-off (Butterworth, 4th order, zero phase response). Our operational measures of postural stability were anterior-posterior and medio-lateral peak-to-peak motion and COP pathlength. Peak-to-peak COP motion within each trial in the anterior-posterior (A-P) and medio-lateral (M-L) plane and the COP pathlength within the two identified phases were calculated.

Since the unilateral arm raise creates a torque about the body's longitudinal axis, the COP pathlength measure includes motion in both the M-L and the A-P planes. As our subjects were healthy and well-practiced in the task, we considered our preflight measures of COP motion (A-P and M-L peak-to-peak motion, COP pathlength) as representative of stable postural control. Therefore, we considered subjects experiencing significantly different COP motion during the postflight arm raise task relative to their preflight measures, as demonstrating deficits in bipedal postural control.

Muscle Activation:

For each muscle of each subject, the EMG signals were first band-passed filtered (20-300 Hz), full-wave rectified, smoothed (10 ms time constant) and averaged. Arm movement initiations for each of the 15 trials obtained from the accelerometer waveform were used as the synchronization point for signal averaging. Mean muscle activation latencies (relative to arm movement initiation) were determined using an interactive graphics program (EGAA, RC Electronics Inc.) and visual inspection (Figure 1). To be considered active, a muscle's voltage had to exceed the baseline voltage by two SDs and remain active for at least 30 ms (5). Since the nature of the task dictated that the subjects adopt quiet stance prior to arm movement initiation, in all cases muscle activation levels were very low. This made muscle activation onset identification straightforward. To assess the degree of similarity between pre- and postflight muscle activation features, cross correlation coefficients were calculated for the two phases of the individual subject mean waveforms.

RESULTS

One purpose of this report is to provide quantitative information that illustrates how spaceflight differentially impacts individuals in terms of postural control during self-initiated perturbations. Consistent with our previous work and others (4,16,19,31), we have observed that in holistic tasks requiring sensory-motor integration, spaceflight is associated with a wide range of adaptive postflight behavioral responses. Therefore, we believe it is important that individual subject data be presented whenever appropriate. Thus, throughout this report each individual's pre- and postflight responses are presented. However, to provide a statistical indication of the magnitude of the potential pre- versus postflight differences of the measures, paired *t* tests were applied to the data of each individual subject.

Arm angular acceleration:

Figure 2 displays the pre- and postflight peak arm acceleration data for each subject. Four subjects significantly decreased (A,C, D, F), two subjects increased (G,H) and two subjects (B,E) displayed no change in their peak acceleration after spaceflight.

Insert Figure Two about here

Center of pressure:

Figure 3 displays pre- and postflight peak-to-peak A-P COP motion. Peak-to-peak A-P COP motion increased significantly in six subjects (B, C, D, F, G, H), decreased in one subject (A) and was unchanged in the remaining subject (E) after spaceflight. Figure 4 displays pre- and postflight peak-to-peak M-L COP motion. Four subjects significantly increased their peak-to-peak M-L motion (B,C,E,G) after spaceflight while Subject A displayed decreased motion. Two subjects's M-L peak-to-peak motion was unaffected by spaceflight (D,H).

Insert Figures Three and Four about here

Figure 5 displays exemplar single trial COP data from one subject during pre- and postflight arm movements. It can be observed that postflight COP motion is increased in both the initiation and recovery phase of the task after spaceflight. Figure 6 shows that the COP pathlength in the *initiation* phase of the movement significantly increased in six subjects (B, C, D, F, G, H) and was unchanged in two subjects (A,E) after spaceflight. Figure 7 shows that COP pathlength during the *recovery* phase was significantly increased in six subjects (B,C,E,F,G,H), one subject had no change (D), while subject A displayed a decrease after spaceflight. Although COP motion was significantly increased after spaceflight, none of the subjects fell during the testing.

Insert Figures Five, Six and Seven about here

Muscle activation:

Pre- and postflight muscle activation latencies are tabulated in Table 1. Although there were large individual differences, there was no consistent trend to suggest that spaceflight modifies the time of initial activation of muscles during the task. Spaceflight had minimal effect on the sequence of muscle activation. In general, and consistent with previous reports (2,14,22), the postural muscles RBF and LPA were activated in an anticipatory fashion well in advance of arm movement onset, during both pre- and postflight testing.

Insert Table 1 about here

Table 2 lists the cross correlation coefficients for each muscle of each subject representing the maximum degree of similarity between the neuromuscular activation patterns within the initiation and recovery phases between the pre- and postflight waveforms. Consistent with the recommendations of Dickey and Winter (10) we used a coefficient value of 0.71 ($r^2 = 0.50$) as the criterion to indicate that the pre- and postflight activation patterns were significantly different. On the basis of this criterion, nine of the 48 (19.0%) waveforms during the initiation phase were modified by spaceflight. Seven of the nine modified initiation waveforms were obtained from the two shank muscles (RGA and RTA). Thirty-three of the 48 (69%) of the activation patterns during the recovery phase were altered after flight. If the RAD comparisions are not considered, 80% (32 of 40) of the postflight lower limb and trunk neuromuscular activation patterns during the recovery phase were significantly different. Despite accounting for the phase lag between the pre and postflight waveforms, the results of the cross correlation analyses indicate that the phasic features of the waveforms were modified by spaceflight. Further analyses of the phase lag data indicated that 95.8% (92/96) of all waveform comparisons displayed either a lag or lead between the pre- and postflight waveforms. However there was no consistent direction or magnitude associated with the lags either across subjects or within subjects.

Insert Table 2 about here

DISCUSSION

The present findings are the first describing the degree to which *long duration* spaceflight affects returning astronauts' ability to initiate and control *self-generated* postural perturbations in the form of voluntary arm movements. The results generally indicate that although subjects can initiate the necessary neuromuscular activation sequences to perform rapid arm movements, upright postural control during the task is compromised after long duration flight. The results of this study are consistent with those of other investigators who have reported that astronauts returning from spaceflight display a variety of postural control problems (1,3,19,31,34). Previous spaceflight related research has primarily focused on postural control in the context of bipedal stance in response to externally-generated perturbations and manipulations of the sensory input (for exceptions see 7,8,30).

The arm raise task contains at least two explicit behavioral goals: 1) move the arm as rapidly as possible until parallel to the floor; and 2) maintain an upright bipedal posture with the feet remaining in contact with the support surface. These two goals are not mutually compatible and therefore suggest a possible trade-off such that the potential postural perturbation resulting from the arm movement can be reduced or increased by reducing or increasing arm acceleration. This potential trade-off in postural stability for arm acceleration makes the subjects' perception of, and confidence in, their ability to control bipedal stance an important consideration. Astronauts who perceive themselves as having postural control decrements after spaceflight can reduce their arm acceleration relative to preflight levels to insure they remain upright. Conversely, returning astronauts with full confidence in their ability may choose to increase arm acceleration at the risk of challenging upright stance. Most interesting, perhaps, is the possibility that astronauts may misperceive the degree of diminished postural control after space flight due to modified proprioceptive processing (17). Thus, they may still threaten their bipedal stability despite decreased arm acceleration.

Measures of peak-to-peak A-P COP motion and COP pathlength reflect deficits in postflight postural control relative to preflight. With the exception of subject A, our subjects generally displayed increases in COP motion. These increases in COP motion were observed despite the fact that the majority of our subjects decreased their peak arm acceleration. The increases in COP motion may be related to subjects' perceptions of their postflight postural control capabilities. We suggest that these subjects correctly perceived they were experiencing compromised postural control but were unable to perceive the *degree* to which their postural control was compromised after spaceflight. This possibillity is reflected in the increased COP motion despite decreases in arm acceleration. Additionally, modifications in proprioceptive processing associated with spaceflight (17), may have resulted in the misperception of arm acceleration during the movement and therefore generated greater potential postural disturbances than they realized.

Two subjects significantly increased their peak arm angular acceleration resulting in large increases (87% for Subject G, 41% for Subject H) in postflight peak-to-peak A-P COP motion. On the assumption that these subjects had full confidence in their ability and wished to retain their preflight performance levels, these increases suggest the inability of these subjects to correctly perceive their postflight postural control capabilities.

It is noteworthy that after spaceflight only Subject A displayed decreases in peak arm acceleration, peak-to-peak COP motion and pathlength in both movement phases compared to his preflight values. In other words, this subject accompanied his decreased postflight arm acceleration by a generalized depression of the associated COP motion. This pattern of decreased motion may suggest that this subject was able to accurately perceive that his postural control was compromised after spaceflight. Therefore, he utilized a strategy that enabled him to complete the task while maintaining postural stability. This is in contrast to the remaining subjects who, for most measures, showed a significant increase in COP motion.

The suggestion that subjects may experience adaptive postflight proprioceptive problems is reasonable. Both Watt et al., (38) and Kozlovskaya et al. (17) have reported that returning astronauts display disordered proprioception that results in inaccurate perceptions of the interaction between themselves and the environment. Additionally, anecdotal evidence indicates that many astronauts experience sensations of 'heaviness' and/or illusions of 'sinking' into the floor while standing (Layne, personal observation). Such sensations would be expected to influence our subjects' perceptions of their postural control capabilities. This disruption in perceptual abilities and associated neuromuscular control may be related to a combination of several physiological changes associated with space flight. Changes in postflight ankle proprioceptive functioning could result in greater ankle sway prior to adequate detection and/or interpretation by the proprioceptive system. Altered functioning of the vestibular system could also result in deficits in sway detection after spaceflight (3,33,34). It is also possible that spaceflight affects muscle spindle sensitivity in such a way that the interaction between central motor commands and peripheral feedback is altered. Thus, although the command for movement is initiated properly after spaceflight, as evidenced by the high correlations between the pre- and postflight waveforms in the initiation phase, the ability to sustain or generate additional bursts of muscle activity is impaired. Loss of muscle strength, particularly in the ankle and trunk musculature, may also play a role in our subject's inability to prevent excessive COP motion. The anti-gravity musculature, including the trunk muscles, tends to show a preferential loss of strength after spaceflight (12,24) which may also have influenced the ability to generate the subtle neuromuscular features necessary for optimal control.

The loss of optimal neuromuscular control after spaceflight would negatively impact the kinematic strategies used to produce the arm movement and associated postural control. Although cross correlation analysis revealed that the phasic features of the neural activation patterns needed for the preparation and initiation of the arm movement remained similar during testing one day after spaceflight, we observed increases in COP motion during the initiation phase. These seemingly paradoxical findings can be explained as followed. Despite the fact that the *shape* of pre- and postflight EMG waveforms were quite similar during the initiation phase of the movement, the timing of the activation features were generally altered by spaceflight. Nintey-seven percent of the lower limb and trunk muscle comparisons indicated that the postflight waveforms either lagged or led the preflight waveforms at the point of maximum correlation. Thus, the postflight temporal relationships associated with muscle force generation relative to arm movement initiation were different than those observed preflight. Moreover, the magnitudes of the muscle force associated with the altered postflight neuromuscular activation features were unlikely to be the same as preflight, particularly since loss of muscle strength typically accompanies extended stays in weightlessness. Additionally, we only obtained EMG from a limited number of muscles. Other musculature undoubtedly contributed to the control of the bipedal arm raise task. The force generating capabilities of these muscles can also be expected to be impacted by exposure to long duration spaceflight. Thus, precise force magnitudes and temporal relationships between the muscles we obtained EMG from and unmonitored muscles may have been significantly altered as a result of spaceflight. Disruptions of these relationships could lead to the diminishment of postural control reflected in the observed increases in COP motion during the initiation phase.

The cross correlation analyses of the lower limb and trunk EMG waveforms during the *recovery* phase revealed that 87.5 percent of the comparisions indicted either a lag or lead in the postflight waveform at the point of maximum correlation relative to preflight. Additionally, 80 percent of the lower limb and trunk muscle cross correlation coefficients during the recovery phase were significantly different indicating that the phasic features of postflight neuromuscular activation generally did not conform with those observed preflight. These findings further suggest a consequential loss of neuro-motor control after spaceflight that is reflected in the increases in postflight COP motion observed during the recovery phase.

The finding that the vast majority of initial muscle activation *patterns* during the initiation phase of the movement were not different pre- versus postflight is somewhat inconsistent with the report of Massion and his colleagues (30). These authors reported that during backward trunk bending, the early activation of the soleus observed preflight was replaced by early tibialis anterior activation during their first postflight session. They attribute this change in neuromuscular patterning as a vestige of the neuromuscular activation sequence used during inflight trunk bending. The normal sequence of activation was restored by the second postflight data collection session (8 days after landing). However, in general, our subjects did display the same *initial* phasic muscle activation characteristics pre- and postflight. These patterns were quite similar to the patterns our subjects used during rapid inflight arm movements performed when restrained to the support surface of the Mir space station (Layne, unpublished data). Thus, the neuromuscular synergies observed preflight were also appropriate to accomplish the inflight arm movement, so it is not particularly surprising that we found the 'shapes' of the pre- and postflight activation waveforms during the initial phase of movement to be similar. However, the fact that 97 percent of the postflight EMG waveforms obtained during the initiation phase either led or lagged the preflight waveforms is consistent with Massion et al.'s findings of disrupted postflight neuromuscular activation.

Primarily due to programmatic constraints, data were collected for seven of the subjects one day after landing. Undoubtedly the diminished postural control and modified neuromuscular activation characteristics exhibited by our subjects would have been exacerbated had we had the opportunity to test them on landing day. One of the subjects, who was scheduled for testing on landing day, was unable to perform the task despite a strong desire to do so. This finding is consistent with previous reports indicating that bipedal postural control recovers toward preflight performance rapidly after spaceflight, especially in the first hours after landing, but recovery is not complete for several days after landing. In particular, Paloski and his colleagues (34) calculated that subjects recover 50% of the postflight equilibrium deficiets experienced at the time of landing within 2.7 hours after short duration spaceflight.

To summarize, the present results indicate that astronauts returning from long duration spaceflight are able to initiate rapid voluntary arm movements without difficulty. However, these movements are accompanied by decreases in bipedal postural control as assessed by measured of COP motion. This is consistent with previous reports that postflight postural control is compromised in response to external perturbations and/or during tests of static postural control in altered sensory environments (3,16,34). Additionally, there were often significant modifications in neuromuscular activation that may have contributed to the compromised postural control exhibited by our subjects. These modifications in neuromuscular activation may have resulted from central and peripheral physiological changes associated with spaceflight. Our subjects' behavior may also suggest that returning crewmembers ability to perceive the full functional capabilities of their postural control systems may also be compromised, particularly after long duration spaceflight. Our findings contribute to a growing body of evidence defining the precise nature of task specific sensory-motor integration deficits experienced by crewmember's returning from spaceflight (4,7,17,19,30,31,34). Although we chose to investigate a task that included a well-documented "anticipatory" postural component associated with vigorous arm motion, all movements necessarily involve a postural component. Therefore, the finding that the postural control associated with voluntary limb motion is compromised after flight is important. Understanding the underlying adaptive processes is an important step towards mitigating the postflight postural control problems experienced by returning astronauts.

Acknowledgements

We thank Brian Peters, Shannon Smith, Matthew Mueller and Elisa Allen, whose support made this project possible. We also thank the participating crewmembers, whose cooperation was essential to the project.

REFERENCES

1 **Anderson, D.J., M.F. Reschke, J.E. Homick, and S.A.S. Werness.** Dynamic posture analysis of Spacelab-1 crewmembers. *Exp. Brain Res.* 64: 380-391, 1986.

2 **Belen'kii, V.E., V.S. Gurfinkel, and R.I. Pal'tsev.** On the elements of voluntary movement control. *Biofizika* 12: 135-141, 1967.

3 **Black, F.O., W.H. Paloski, D.D. Doxey-Gasway, and M.F. Reschke.** Vestibular plasticity following orbital space flight: Recovery from postflight postural instability. *Acta. Otolaryngol.* 520: 450-454, 1995.

4 **Bloomberg, J.J., M.F. Reschke, W.P. Huebner, B.T. Peters, and S.L. Smith.** Locomotor head-trunk coordination strategies following space flight. *J. Vestib. Res.* 7: 161-177, 1997.

5 **Bogey, R.A., L.A. Barnes, J. Perry.** Computer algorithms to characterize individual subject EMG profiles during gait. *Arch. Phys. Med. Rehabil.* 73:835-841, 1992.

6 **Bouisset, S., and M. Zattara.** Biomechanical study of the programming of anticipatory postural adjustments associated with voluntary movement. *J. Biomech.* 20(8): 735-742, 1987.

7 **Clement, G., V.S. Gurfinkel, F. Lestienne, M.I. Lipshits, and K.E. Popov.** Adaptation of postural control to weightlessness. *Exp. Brain Res.* 57: 1-72, 1984.

8 **Clement, G., V.S. Gurfinkel, F. Lestienne, M.I. Lipshits, and K.E. Popov.** Changes of posture during transient perturbations in microgravity. *Aviat. Space Environ. Med.* 56: 666-671, 1985.

9 **Daley, M. L., and R. L. Swank.** Quantitative posturopgraphy: Use in multiple sclerosis. *IEEE Trans. Biomed. Engin.* 28: 668-671, 1981.

10 **Dickey, J.P., and D.A. Winter.** Adaptations in gait resulting from unilateral ischaemic block of the leg. *Clin. Biomech.* 7(4): 215-225, 1992.

11 **Era, P., and E. Heikkinem.** Postural sway during standing and unexpected disturbance of balance in random samples of men of different ages. *J. Gerentol.* 40: 287-295, 1985.

12 **Hayes, J.L., J.J. McBrine, L. Roper, M.D. Stricklin, S.F. Siconolfi, and M.C. Greenisen.** Effects of space shuttle flights on skeletal muscle performance. *FASEB J.* 6(5): A1770, 1992.

13 **Homick, J.L., and M.F. Reschke.** Postural equilibrium following exposure to weightless space flight. *Acta Otolaryngol.* 83: 445-464, 1977.

14 **Horak, F.B., P. Esselman, M.E. Anderson, and M.K. Lynch.** The effects of movement velocity, mass displaced, and task certainty on associated postural adjustments made by normal and hemiplegic individuals. J. *Neurol., Neurosurg. Psychiatry* 47: 1020-1028, 1984.

15 **Koozekanani, S.H., C.W. Stockwell, R.B. McGhee, and F. Firoozmand.** On the role of dynamic modeling in quantitative posturography. *IEEE Trans. Biomed. Engin.* 27, 605-609, 1980.

16 **Kornilova, L.N., A.M. Goncharenko, G. Bodo, K. Elkin, V. Grigorova, V., and A. Manev.** Pathogenesis of sensory disorders in microgravity. *Physiologist* 34(1): S36-39, 1991.

17 **Kozlovskaya, I.B., Y.U.V. Kreidich, V.S. Oganov, and O.P. Koserenko.** Pathophysiology of motor functions in prolonged manned space flights. *Acta Astronautica* 8(9): 1059-1072, 1981.

18 **Kozlovskaya, I.B., V.A. Barmin, V.I. Stepantsov, and N.M. Kharitonov.** Results of studies of motor functions in long-term space flights. *Physisologist* 33(1): S1-3, 1990.

19 **Layne, C.S., P.V. McDonald, and J.J. Bloomberg.** Neuromuscular activation patterns during treadmill walking after space flight. *Exp. Brain Res.* 113(1): 104-116, 1997.

20 **Layne, C.S., A.P. Mulavara, P.V. McDonald, C.J. Pruett, I.B. Kozlovskaya, and J.J. Bloomberg.** The impact of long duration space flight on upright postural stability during unilateral arm raises. *Soc. Neurosci. Abs.* 23: 1562(610:12), 1997.

21 **Layne, C.S., P.V. McDonald, C.J. Pruett, G. Jones, and J.J. Bloomberg.** Preparatory postural control after space flight. *Soc. Neurosci. Abs.* 21: 684(275.11), 1995.

22 **Lee, W.A., T.S. Buchanan, and M.W. Rogers.** Effects of arm acceleration and behavioral conditions on the organization of postural adjustments during arm flexion. *Exp. Brain Res.* 66: 257-270, 1987.

23 **Lee, R.G., I. Tonolli, F. Viallet, R. Aurenty, and J. Massion.** Preparatory postural adjustments in parkinsonian patients with postural instability. *Can. J. Neurol. Sci.* 22: 126-135, 1995.

24 **LeBlanc, A., R. Rowe, V. Schneider, H. Evans, and T. Hedrick.** Regional muscle loss after short duration space flight. *Aviat. Space Environ. Med.* 66(12): 1151-1154, 1995.

25 **Lehmann, J.F., S. Boswell, R. Price, A. Burleigh, B.J. deLateur, K.M. Jaffe, and D. Hertling.** Quantitative evaluation of sway as an indicator of functional balance in post-traumatic brain injury. *Arch. Phys. Med. Rehabil.* 71: 955-962, 1990.

26 **Lord, S. R., R.D. Clark, and I.W. Webster.** Postural stability and associated physiological factors in a population of aged persons. *J. Gerentol.* 46: M69-76, 1991.

27 **Maki, B.E.** Selection of perturbation parameters for identification of the posture control system. *IEEE Trans. Biomed.* 34: 797-810, 1987.

28 **Maki, B.E., P.J. Holliday, and G. R. Fernie.** Aging and postural control: A comparison of spontaneous- and induced-sway balance tests. *J. Am. Geriatr. Soc.* 38: 1-9, 1990.

29 **Maki, B.E.** Biomechanical approach to quantifying anticipatory postural adjustments in the elderly. *Med. Biomed. Engin. Comput.* 31: 355-362, 1993.

30 **Massion, J., V. Gurfinkel, M. Lipshits, A.Obadia, and K. Popov.** Axial synergies under microgravity conditions. *J. Vestib. Res.* 3: 275-287, 1993.

31 **McDonald, P.V., C. Basdogan, J.J. Bloomberg, and C.S. Layne, C.S.** Lower limb kinematics during treadmill walking after space flight: implications for gaze stabilization. *Exp. Brain Res.* 112: 325-334, 1996.

32 **Newman, D.J., D. K. Jackson, and J.J. Bloomberg.** Altered astronaut lower-limb and mass center kinematics in downward jumping following space flight. *Exp. Brain Res.* 117: 30-42, 1997.

33 **Paloski, W.H., F.O.Black, M.F. Reschke, D.S. Calkins, and C. Shupert.** Vestibular ataxia following shuttle flights: Effects of transient microgravity on otolith-mediated sensorimotor control of posture. *Am. J. Otolaryngol.* 13(3): 254-262, 1992.

34 **Paloski, W.H., M.F. Reschke, F.O. Black, D.D. Doxey, and D.L. Harm.** Recovery of postural equilibrium control following space flight. In: *Sensing and controlling motion: vestibular and sensorimotor function*, edited by B. Cohen, D.L. Tomko, and F. Guedry. New York, NY: New York Academy of Sciences, 1992, p. 747-754.

35 **Reschke, M.F., D.J. Anderson, and J.L. Homick.** Vestibulo-spinal response modification as determined with the H-reflex during Spacelab-1 flight. *Exp. Brain Res.* 64: 367-379, 1986.

36 **Reschke M.F., L.N. Kornilova, D.L. Harm, J.J. Bloomberg, and W.H. Paloski.** Neurosensory and sensory-motor function. In: *Space biology and medicine, vol. III: Humans in spaceflight, Book 1: Effects of Microgravity*, edited by C.S. Leach Huntoon, V.V. Antipov, and A.I. Grigoriev. Washington, DC and Moscow: American Institute of Aeronautics and Astronautics, 1996, p. 135-193.

37 **Riach, C.L., K.C. Hayes, and S.D. Lucy.** Changes in centre of pressure of ground reaction forces prior to rapid arm movement in normal subjects and patients with cerebellar ataxia. *Clin. Biomech.* 7: 208-214, 1992.

38 **Watt, D.G.D., K.E. Money, R.L. Bondar, R.B. Thirsk, and P. Scully-Power.** Canadian medical experiments on shuttle flight 41-G. *J. Can. Aeronaut. Space* 31(3): 215-225, 1985.

FIGURE LEGENDS

Figure 1. Preflight (A) and postflight (B) exemplar mean LPA activation waveforms (+1 SD, upper trace) and accelerometer records (lower trace). The *initiation* phase consists of data 1 second prior to the initiation of arm motion through arm acceleration. The *recovery* phase consists of data from the completion of acceleration until the end of the data record.

Figure 2. Individual subject means (+1SD) pre- and postflight peak arm angular acceleration. As with the remaining figures, the dark bar represents preflight performance and the lighter shaded bar represents postflight performance. The asterisks indicate statistical significance at $p < 0.05$.

Figure 3: Individual subject means (+1SD) pre- and postflight A-P peak-to-peak COP motion.

Figure 4: Individual subject mean (+1SD) pre-and postflight M-L peak-to-peak COP motion.

Figure 5: One example trial showing COP trace from pre- (A) and postflight (B) displaying increased postflight motion during both the initiation and recovery phases of the arm movement.

Figure 6: Individual subject means pre- and postflight COP pathlength during the *initiation* phase.

Figure 7: Individual subject means pre- and postflight COP pathlength during the *recovery* phase.

Table 1 Pre- and postflight mean EMG activation onsets. # indicates that no burst was present in the averaged record. The values are reported in milliseconds and are relative to arm movement initiation.

	RAD		LBF		RBF		LPA		RGA		RTA	
Subject	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
A	-46	-44	-68	-6	-118	-114	-58	-96	-16	-32	-96	-60
B	-26	-32	-8	-12	-156	-162	-92	-90	-6	2	-8	-2
C	-34	-26	6	26	-110	-102	-56	-46	-70	18	-82	6
D	-54	-38	30	-6	-106	-72	-94	-56	-24	-44	-62	-54
E	-24	-22	-6	-10	-126	-142	-64	-74	-30	-42	#	#
F	-48	-52	#	#	#	#	-78	-70	#	#	#	#
G	-54	-86	-12	-18	-124	-122	-78	-90	-34	-28	#	#
H	-32	-48	-14	-10	-142	-134	-32	-48	-16	-28	-34	-40
Mean	-39.8	-43.5	-18.9	-5.1	-126	-121.1	-69	-71.6	-28	-22	-56.4	-30
SD	12.2	20.1	24.2	14.3	17.7	29.2	20.7	19.8	20.8	23.2	35.7	30.2

Table 2 Cross correlations between the pre- and postflight EMG records during each phase of the movement. Init = initiation phase, Rec = recovery phase.

	RAD		LBF		RBF		LPA		RGA		RTA	
Subject	Init	Rec	Init	Rec	Init	Rec	Init	Rec	Init	Rec	Init	Rec
A	0.97	0.75	0.96	0.58	0.95	0.46	0.92	0.42	0.87	0.43	0.96	0.57
B	0.95	0.92	0.99	0.24	0.98	0.59	0.93	0.72	0.60	0.51	0.90	0.78
C	0.98	0.96	0.96	0.69	0.88	0.48	0.96	0.46	0.80	0.59	0.94	0.88
D	0.99	0.93	0.96	0.58	0.98	0.49	0.89	0.66	0.94	0.75	0.96	0.82
E	0.99	0.89	0.92	0.38	0.96	0.88	0.98	0.64	0.50	0.33	0.26	0.31
F	0.97	0.58	0.22	0.29	0.53	0.18	0.88	0.69	0.18	0.34	0.43	0.36
G	0.98	0.82	0.93	0.58	0.91	0.73	0.94	0.71	0.52	0.33	0.47	0.29
H	0.98	0.95	0.97	0.63	0.97	0.59	0.98	0.61	0.90	0.65	0.95	0.71
Mean	0.98	0.85	0.86	0.50	0.89	0.55	0.94	0.61	0.66	0.49	0.73	0.59
SD	0.01	0.13	0.26	0.17	0.15	0.21	0.04	0.12	0.26	0.16	0.29	0.24

Figure 1

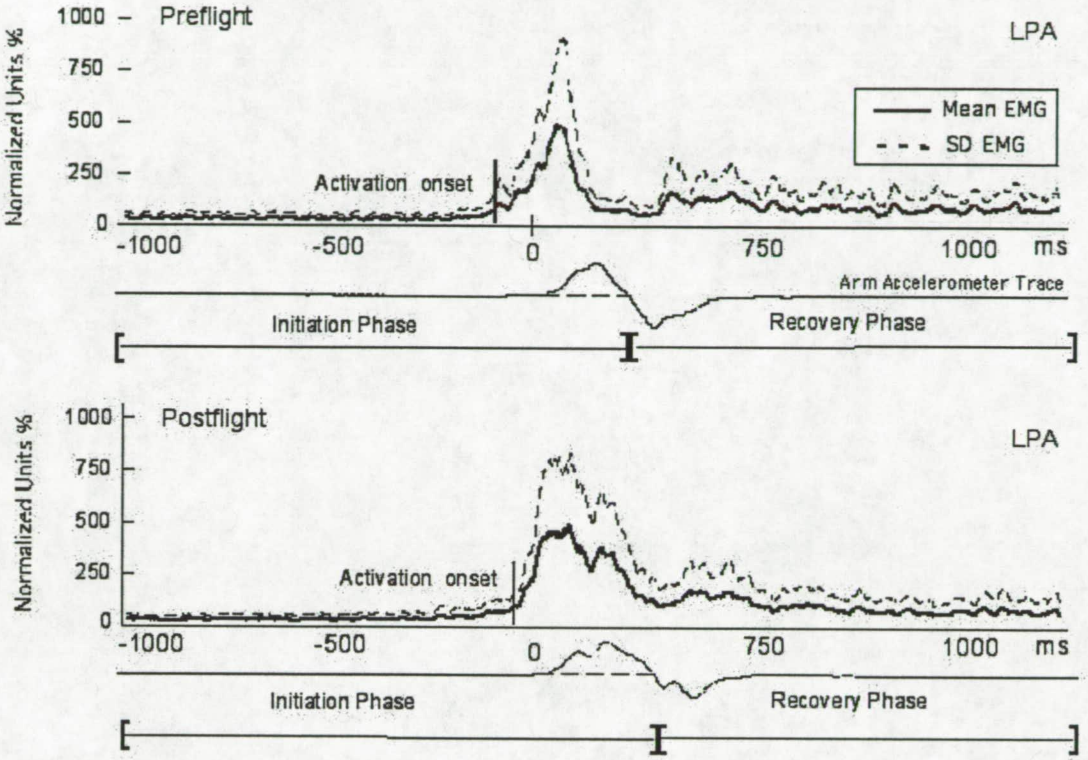
1000
750
500
250
0
Normalized Units %
Preflight
LPA
Mean EMG
SD EMG
Activation onset
-1000
-500
0
750
1000
ms
Arm Accelerometer Trace
Initiation Phase
Recovery Phase
1000
750
500
250
0
Normalized Units %
Postflight
LPA
Activation onset
-1000
-500
0
750
1000
ms
Initiation Phase
Recovery Phase

Figure 2

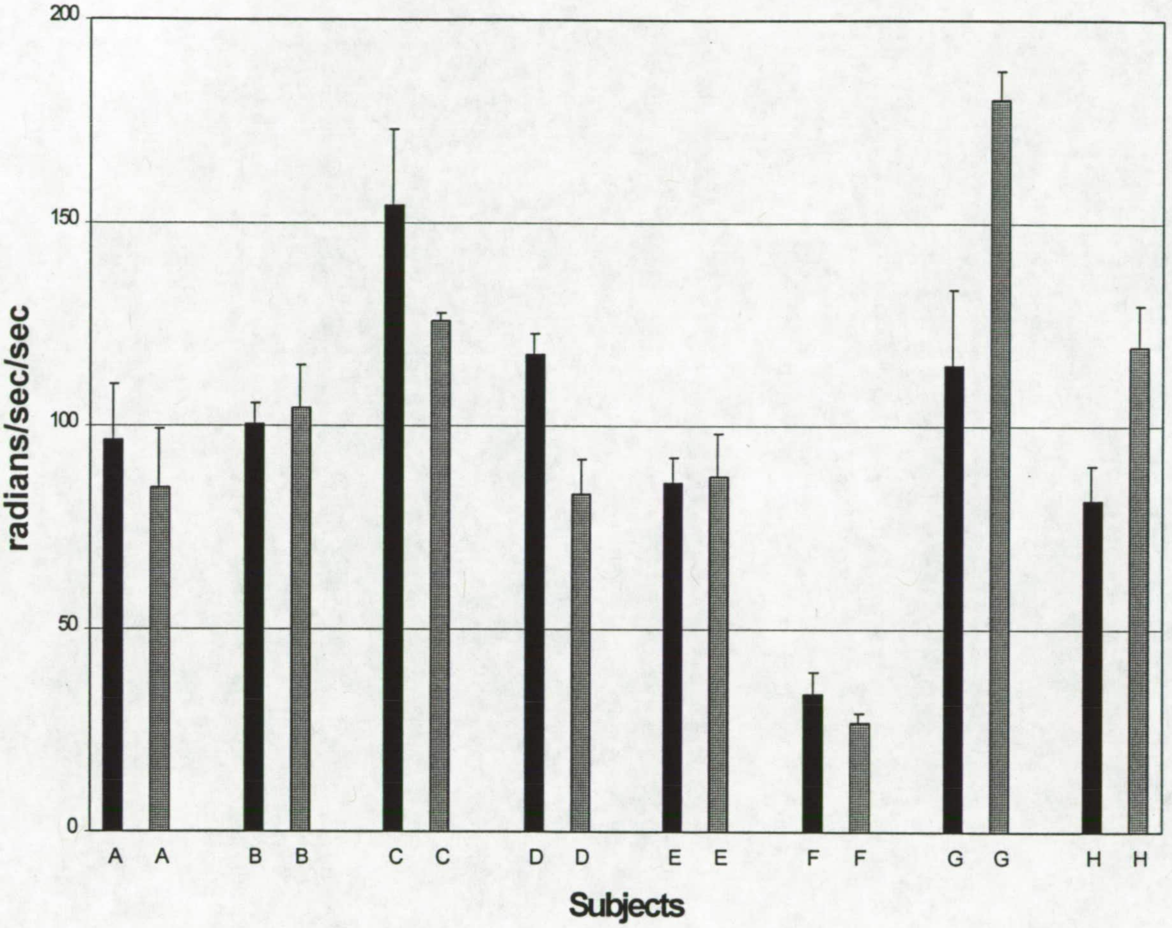

Figure 3

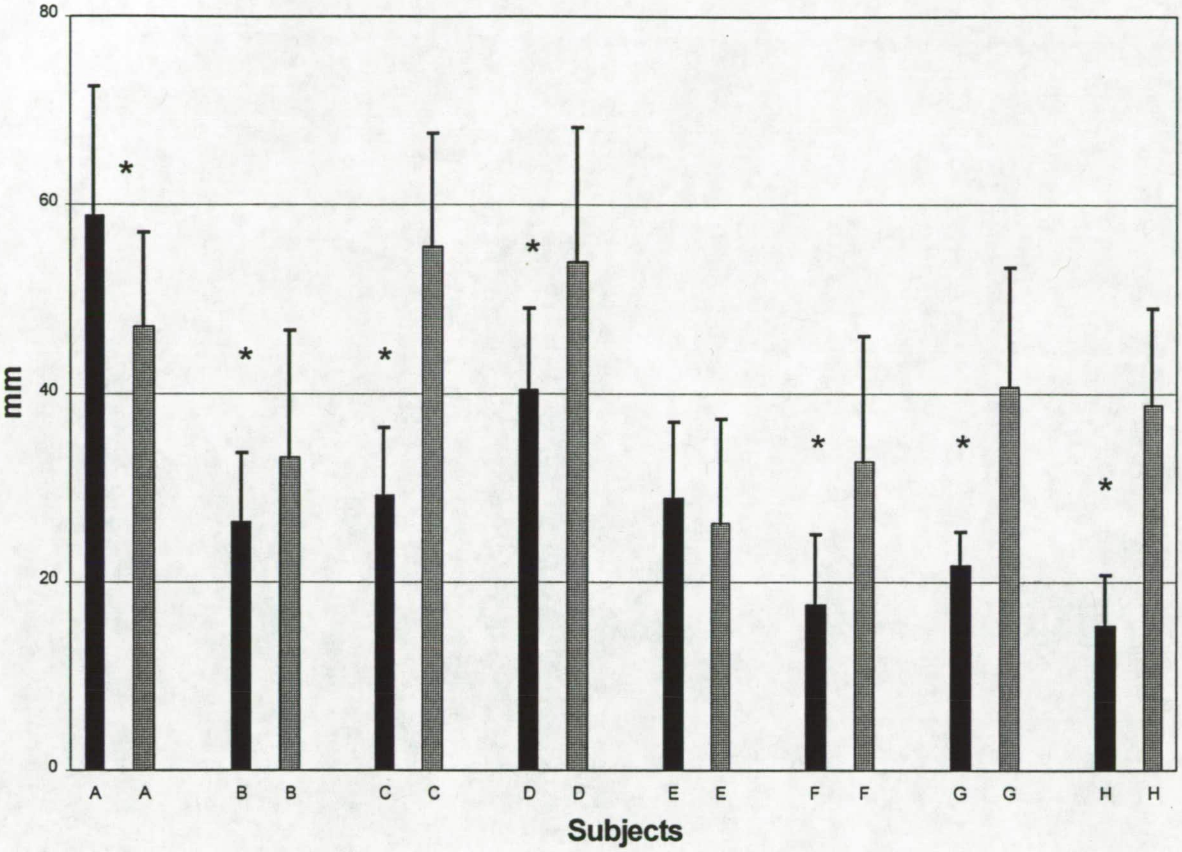

Figure 4

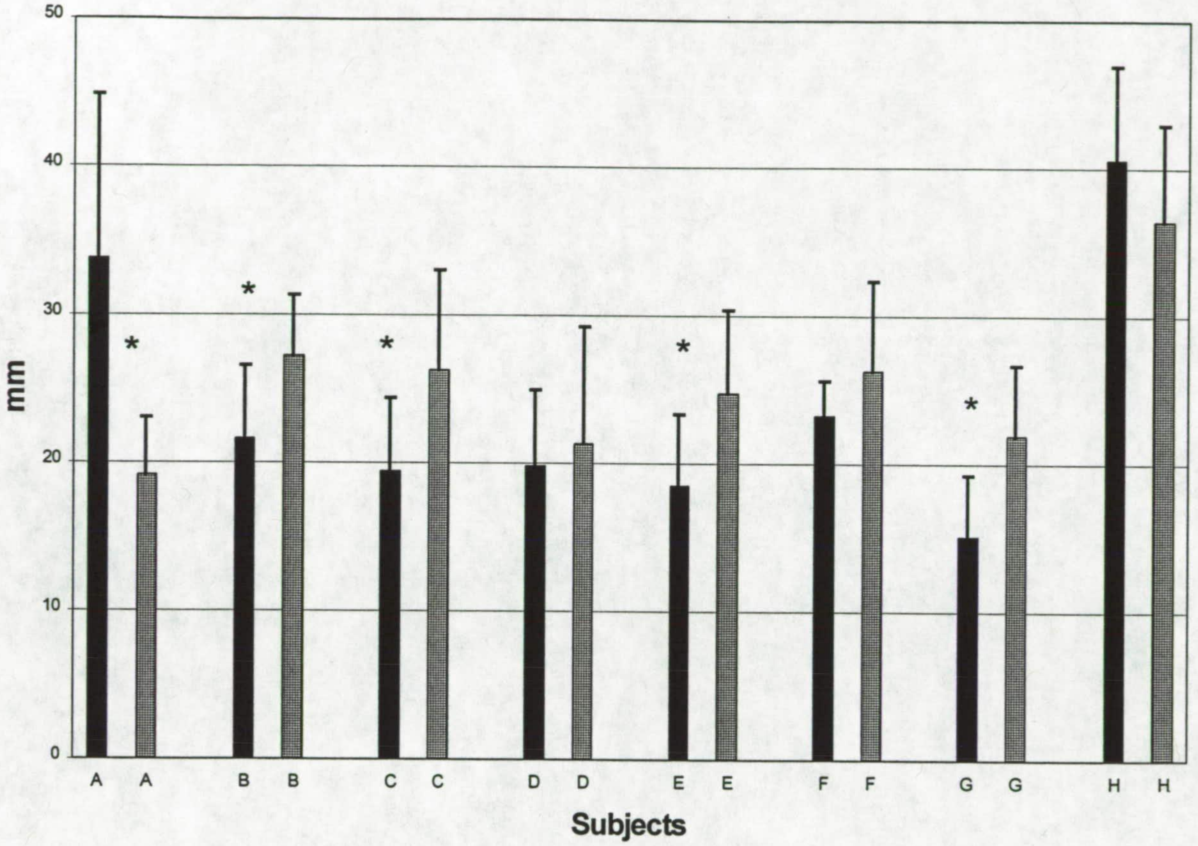

Figure 5

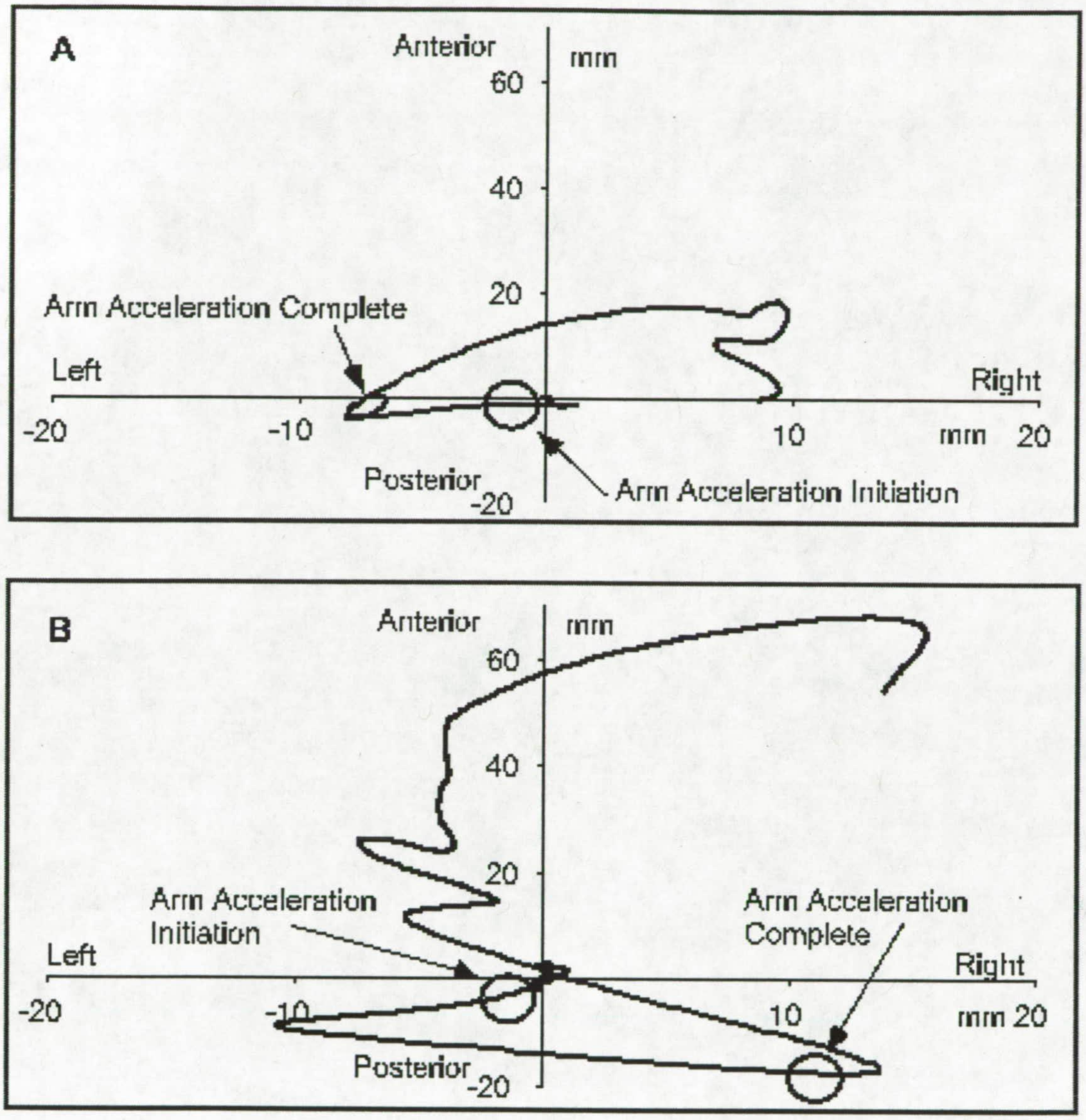
A
Anterior
mm
60
40
20
Arm Acceleration Complete
Left
Right
-20
-10
10
mm 20
Posterior
-20
Arm Acceleration Initiation
B
Anterior
mm
60
40
20
Arm Acceleration
Initiation
Arm Acceleration
Complete
Left
Right
-20
-10
10
mm 20
Posterior
-20

Figure 6

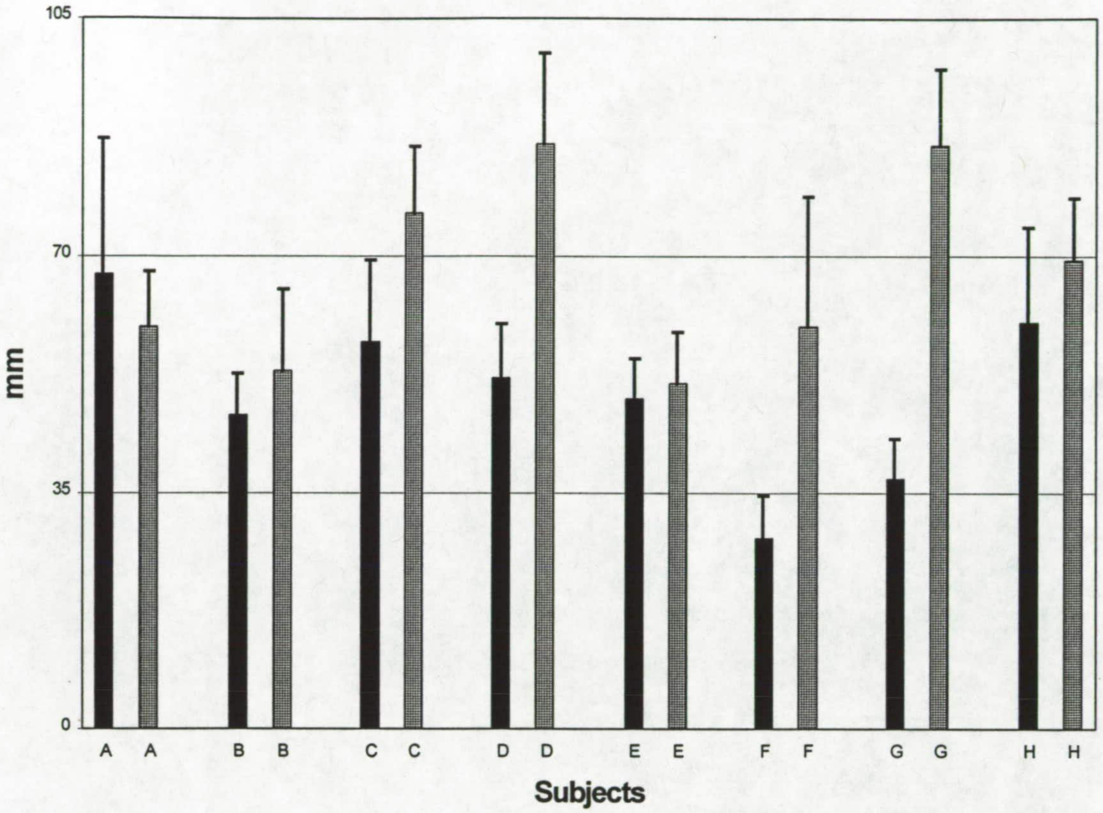

Figure 7