

Electromyographic evaluation of anti-gravity muscle function after 6 hr exposure to dry flotation in young Indian army recruits

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Thirteen healthy army recruits between the ages of 18-22 yrs were chosen for the study. Surface EMG from gastrocnemius and tibialis anterior muscles were recorded during maximum voluntary isometric and isotonic contractions with the subject supine. Ankle jerk evoked EMG for different forces of percussion was also recorded from the calf muscles. The subjects were exposed to 6 hrs of dry flotation in the dry immersion chamber at IAM IAF. Post exposure EMG parameters were recorded in the same manner as the pre-exposure recordings. EMG data was then analysed using integration and power spectrum analysis. The comparative results are discussed.

Keywords: Electromyography, hypogravity, neuromuscular deconditioning, space medicine

Deterioration of anti-gravity muscle function on exposure to microgravity of space has been well documented [1,2,3]. Similar findings have been reported in ground based simulation experiments, both in human and animal subjects [4,5]. A long term exposure to microgravity results in structural and biochemical changes in muscles themselves as well as readjustment of their neural control [5,6]. Relatively short term exposures have not shown significant structural and biochemical changes in anti-gravity muscles but their functional properties have shown alterations in as short an exposure to hypogravity as 40 s in parabolic flight [7]. It has been suggested that these functional alterations may be as a result of readjustment of neural control of anti-gravity muscles [8]. Exact site and time course of these readjustments is still debated. It is possible that changes in neural control of anti-gravity muscles are a forerunner of local changes in these muscles on long exposure to microgravity and may lead to them as a trophic phenomenon. If such be the case, new strategies in evolving counter measures against neuromuscular

deconditioning may be developed, the primary target of which will be neural control readjustment. This study was undertaken to get a better insight into functional properties of anti-gravity muscles and their neural control in human volunteers on 6 hr exposure to hypogravity simulation by dry flotation method, using surface electromyography as a tool.

Method

Thirteen healthy male army recruits between the age of 18-22 yrs volunteered for this study. All subjects were within stipulated height and weight limits for their age as per the army standards and maintained a moderate physical fitness schedule. All subjects were instructed to have adequate sleep on

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the night previous to the day of the experiment and to avoid any unaccustomed physical activity for 24 hrs prior to it. They were, however, advised to continue their routine activities. The subjects reported to hypogravity simulation laboratory at IAM at 0830 hrs on the day of the test. A brief clinical history was obtained and a clinical examination with special emphasis to the examination of neuromuscular system was performed to rule out any pre-existing disease or abnormality.

The subjects were instrumented for surface electromyography of calf and tibialis anterior muscles. Bipolar Ag-AgCl electrodes were applied over the maximum bulge of medial head of gastrocnemius muscle with reference electrode on Achilles tendon, 2 cm above the ankle joint, and 2-4 cm below tibial tuberosity on the belly of tibialis anterior muscle with reference electrode over the tendon of the muscle 2 cm above the ankle joint anteriorly. The area chosen was cleaned thoroughly using acetone and electrodes fixed using EC-2 electrolyte cream (GRASS, USA). The electrodes were connected to two wide-band AC preamplifiers (Model 7P5 GRASS Polygraph, USA). The output of preamplifiers was fed to driver amplifiers (7DA GRASS, USA). The EMG was recorded on chart paper using GRASS 7D polygraph. The output from amplifiers was fed to integrators (7P10 GRASS, USA), FM Tape recorder (TEAC R-61) and dual trace oscilloscope (HM-412).

Calibrations: The preamplifier sensitivity was set to 500 μ v at input producing 1 volt output at the driver amplifier output. The EMG signal was monitored on the oscilloscope at a sensitivity of 1 volt/cm and a sweep of 10 cm/sec. Chart recorder registered a 1 cm deflection for 1 volt driver output. The FM tape-recorder was calibrated to saturation at 500 μ v signal at pre-amplifier input i.e. 1 volt signal at driver output and tape recorder input.

Pre-immersion recording: Following instrumentation, the subject was made to lie down on the hy-

draulic platform of the dry flotation tank at IAM, IAI, the details of which have been described by earlier workers [9]. Passive supine EMG was recorded for 30 s period. After this, ankle jerks were elicited using a percussion hammer fitted with a force transducer, which was connected to a DC preamplifier (7P1, GRASS, USA) and recorded on chart recorder through driver amplifier (7DA, GRASS). It was calibrated to 0.25 kg/mm. Gradually increasing force of percussion with corresponding EMG from calf and tibialis anterior muscles were recorded. No 'reinforcement' was used to alter the sensitivity of the reflex response.

The subjects were then asked to plantarflex the ankle with maximum possible force. This isotonic activity was recorded from the calf and anterior tibialis muscles. After a 2 min rest, the subjects were again asked to plantarflex at the ankle joint but at this time, resistance was applied against the sole to prevent any movement and to ensure maximum isometric contraction. EMG for calf and tibialis anterior muscles was recorded again. In both instances, recordings were made for 15 s.

Dry immersion (DI): Following pre-immersion recordings, the platform was lowered hydraulically and the subject floated freely on the surface of the water, pre-heated to 32-33°C to avoid any thermal discomfort, but physically separated from the water by a pliant, non anchored plastic sheet. He was maintained in this position for 6 hr and his food and toilet needs were catered for in this posture. At the end of 6 hr, the platform was raised again and post immersion recordings made.

Post immersion recordings: Passive supine EMG, T-reflex, max isometric and max isotonic EMG were recorded similar to preimmersion recordings.

Analysis of EMG: EMG recorded on FM tape recorder was digitised using PCL-201 AD converter at a sampling rate of 2048 samples/sec. Digitized

data was analysed using discrete Fourier Transform power spectral analysis, based on University of Texas algorithm [10]. A 512 point data burst was collected and surge effects smoothed using Blackman Window function [11] which is defined as:

$$W(n) = 0.42 - 0.5 \cos(2\pi n/N) + 0.08 \cos(4\pi n/N)$$

Where, N=No of samples per data burst,

n=0,1,2,3...etc position of each data point in the sequence and

W(n)=weighting index for nth data point.

Smoothed data burst was subjected to power spectral analysis using discrete Fourier Transform given by [10]:

$$H(k) = \sum_{n=0}^{n=N} h(n) e^{-j2\pi kn} \text{ for } 0 \leq k \leq N-1$$

where, H is the fourier transform of amplitude function h for the nth data point,

k is the frequency index component, N is the sample size and $j = \sqrt{-1}$

From the transform, total EMG power of an epoch and its centroid frequency were calculated. Centroid frequency is defined as the frequency below and above which the power distribution is equal. It was calculated using the following equation [11]:

$$C = \frac{\sum_{k=3}^{256} H(k) \cdot k}{\sum_{k=3}^{256} H(k) / 0.511}$$

where C=Centroid frequency and H(k)=Fourier Transform for kth frequency.

Latency of T-reflex was calculated as the interval between the first deflection of the percussion hammer tracing and appearance of the EMG response of the calf muscles. The amplitude of reflex evoked EMG (sensitivity 5mV/cm) and force of percussion (sensitivity 2.5 kg/cm) were calculated from

respective tracings and EMG amplitude per unit percussion force was derived.

Statistical analysis: All the measured and derived parameters including EMG total power, centroid frequency, T-reflex latency and T-reflex EMG amplitude/unit force were tested for significant difference between pre and post immersion recordings using Student's paired 't' test. Total power and centroid frequency were analysed for passive supine, maximum isotonic and maximum isometric contractions before and after immersion.

Results

Passive supine EMG total power from calf muscle did not show a significant difference between pre exposure and post-exposure values (pre-exp 97.12±0.25 mV/sec, post-exp 95.13±4.99 mV/sec; p>0.05). Similarly, max isotonic (pre-exp 240.13±9.46 mV/sec, post-exp 243.60±0.57 mV/sec; p>0.05) and max isometric (pre-exp 378.77±31.3 mV/sec, post-exp 389.65±1.09 mV/sec; p>0.05) also did not show significant differences between pre exposure and post exposure values. Centroid frequency values also did not show significant shift between pre-exposure and post exposure values for passive supine (pre-exp 122±5 Hz, post-exp 118±8 Hz, p>0.05), max isotonic (pre-exp 116±14 Hz, post-exp 118±8 Hz, p>0.05), max isometric (pre-exp 109±15 Hz, post-exp 102±20 Hz, p>0.05) EMG for calf muscles. Similarly, EMG for tibialis anterior muscle did not show significant differences for total power and centroid frequencies between pre and post exposure values in passive supine state and max isotonic and max isometric contractions.

T-reflex latency showed a significant increase after exposure to 6 hr dry immersion (pre-exp 108.84±51.57 ms, post-exp 172.27±24.20 ms; p<0.001). T-reflex EMG/unit percussion force showed a significant decrease after dry immersion (pre-exp 4.17±1.2 mV/kg, post-exp 2.36±0.99 mV/kg, p<0.01).

Discussion

Various changes in contractile, structural and biochemical properties of anti-gravity muscles, in both human and animal subjects, after long term exposure to hypogravity have been reported. These include decrease in max isometric tetanic force, increase in twitch/tetanus tension ratio [4,12,13], reduction in type I fibres and a relative increase in type II fibres [13], loss of myosin protein and enzymatic shift with increase in glycolytic potential with a comparative reduction in oxidative respiratory capacity [14]. EMG studies have revealed an increase in burst activity over soleus after hypogravity exposure [13]. Such spectral shifts towards higher frequencies are found in clinical myopathies [15,16] while in neuropathic patterns, the dominant frequency shifts towards the lower range [15,16]. Thus, muscle changes on long term hypogravity exposure are more akin to myopathic disorders rather than neuropathic disorders.

In the present study, however, there was no shift in centroid frequency of calf muscles, indicating that no local or motor end plate changes occurred in the calf muscles in 6 hrs of dry flotation. Similarly, there were no changes in total EMG power during passive lying. Since this EMG represents motor unit activity at rest to maintain muscle tone [17], maintained by control from reticulo-spinal tracts over T-spinal moto-neurons, clearly this control is not modified in 6 hrs of hypogravity exposure. Maximum isotonic and isometric contractions represent cortico-spinal drive of α -motoneurons, causing maximum motor unit recruitment [17]. Since greater recruitment of motor units results in greater power in measured EMG [18,19], no significant alteration in total EMG power during max isotonic and isometric contractions of calf muscles after dry immersion exposure indicates that voluntary motor unit recruitment is unaffected by 6 hrs DI.

T-reflex latency showed a significant lengthening after DI. Also, amplitude of evoked EMG/unit

force of percussion also showed a reduction. This indicates a suppression of the monosynaptic reflex loop. T-reflex suppression has also been reported by Kozlovskaya et al after 7 day exposure to dry immersion [8]. Our results indicate that this suppression begins as early as after 6 hrs of exposure to DI. The possibility of reduction in the sensitivity of muscle spindle or the sensory neuronal suppression is negated by the finding of decreased threshold of T-reflex after 4-5 days of hypogravity exposure by some workers [20]. Since a suppression of otolith-spinal reflex has been reported [21] on hypogravity exposure, it is likely that suppression of T reflex as seen in this study may be due to a partial withdrawal of vestibulo-spinal facilitation of α -motoneurons.

The results of this study indicate that while 6 hrs of DI simulation of hypogravity does not produce any structural and functional changes at the level of the anti-gravity muscles themselves and their cortical and reticular control, a suppression of spinal α -motoneurons projecting to these muscles does occur, possibly due to withdrawal of vestibulo-spinal facilitatory drive.

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