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VOLUME 66 NUMBER 1

JANUARY 1995

AVIATION, SPACE & ENVIRONMENTAL MEDICINE

Is There Central Fatigue During Simulated Air Combat Maneuvering?

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BAIN B, JACOBS I, BUICK F. *Is there central fatigue during simulated air combat maneuvering?* Aviat. Space Environ. Med. 1995; 66:1–5.

This study tested the hypothesis that repeated exposure to high levels of +Gz acceleration, in conjunction with repeated execution of an Anti-G Straining Maneuver (AGSM), causes central fatigue, presumably by impairing central nervous system (CNS) function. We speculated that central fatigue would impair the ability to recruit sufficient musculature at the intensity required to perform an adequate anti-G straining maneuver. Central fatigue was evaluated by measuring maximal force generation and surface electromyographic activity of leg extensor muscles before, during, and immediately upon termination of an SACM, and comparing these values to those obtained when the muscles were electrically stimulated during maximal voluntary contractions (MVCs). We assumed that any observed increase in force generation during the MVCs, caused by the stimulation, would indicate central fatigue. G-tolerance time was 230 ± 172 s. Hypoxia was induced by the SACM as the arterial oxygen saturation decreased significantly from 97% to 90%. In spite of this hypoxia, there was no significant change in MVC force when the pre- and post-SACM values were compared. Electrical stimulation during the MVC's did not cause an increase in force generation. The average forces generated during the +7 Gz phase of the SACM were only about 35% of MVC force. This force value did not change significantly during the SACM. The results indicate that the inability to continue to perform the AGSM during an SACM is not likely due to central fatigue or to fatigue of the large skeletal muscle groups we have examined. Taken together with other recent studies, all indicating that the force levels generated in large muscle groups during an SACM are submaximal, these results question the rationale for advocating strength training for the purpose of improving tolerance of +Gz acceleration forces.

PILOTS FLYING high-performance aircraft are often subjected to high levels of headward (+Gz) acceleration for varying lengths of time. These high +Gz accelerations decrease the hydrostatic pressure of the column of blood above the heart, thereby decreasing brain perfusion pressure which can lead to loss of peripheral vision, blackout (defined here as total loss of vision), and loss of consciousness (termed G-induced loss of consciousness or G-LOC) (8). The Anti-G Straining Maneuver (AGSM) is an effective means of increasing head-level arterial blood pressure via a combination of increases in intrathoracic pressure and peripheral vascular resistance. The AGSM consists of a Valsalva maneuver held for three seconds, released for one second and then repeated, combined with isometric tensing of skeletal muscles especially in the legs, arms, and abdomen. The AGSM is continued for the duration of the acceleration or until the pilot becomes fatigued.

The inability to perform an effective AGSM during +Gz exposure has been linked to local muscular fatigue of skeletal muscles recruited during the AGSM (9,12), but this explanation has been questioned recently (2,3). We reported previously that the intensity of muscular contraction during the AGSM is submaximal, ranging from 20–50% of maximum voluntary isometric contraction (MVC) force, depending on the muscle examined (3). We also interpreted the changes in muscle lactate and glycogen concentrations after an AGSM during exposure to high +Gz as not being sufficiently large to be causally related to muscular fatigue during this type of exertion (2). Taken together, these results suggest that it is unlikely that a metabolic cause of peripheral muscular fatigue in the muscles examined limits the ability to perform the AGSM. We speculated that there may be a central nervous system (CNS) component to the fatigue perceived during AGSM, possibly caused by prolonged hypoxic insult to the CNS during exposure to high +Gz levels, because decreases in oxygen saturation and the partial pressure of oxygen in arterial blood occur during exposure to high levels of +Gz acceleration (8,14).

A number of studies have suggested that central fatigue contributes to force failure during isometric muscular contractions (1,6,11). Central fatigue was defined by Asmussen as being impairment of force generation,

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This manuscript was received for review in September 1993. It was revised and accepted for publication in May 1994.

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the cause of which occurs proximal to the motor neurons (1). Isometric muscle contraction increases mean arterial blood pressure as a function of the contraction intensity, duration and the mass of the recruited musculature (15,16,19). If central fatigue reduces the ability to generate or maintain the intensity of muscle contraction required to increase arterial pressure during an AGSM, then the effectiveness of the straining maneuver will be impaired.

This investigation tested the hypothesis that repeated exposure to high levels of $+G_z$ acceleration, in conjunction with repeated execution of an AGSM, causes central fatigue. In this study we defined central fatigue, a priori, as being manifested when electrical stimulation of a muscle during a maximum voluntary contraction (MVC) causes an increase in the force produced by the muscle in question. While electrical stimulation added to MVC does not increase force production in unfatigued leg muscle, it does do so after intense fatiguing exercise (4,5,11).

METHODS

Subjects

From a trained pool of centrifuge subjects, eight subjects, five males and three females, volunteered to participate in the experiment. The experimental protocol was approved by the institutional Human Ethics Committee. Written informed consent was obtained from the subjects. The subjects were experienced and regular participants in centrifuge acceleration experiments. Their participation was subject to medical consent/certification to participate by a physician or medical officer authorized to approve participation in centrifuge experiments.

Simulated Air Combat Maneuver (SACM)

The SACM centrifuge profile was an alternating $+4\ G_z/+7\ G_z$ cycle, with each G level maintained for 10 s. The cycle was repeated until the subject reached his or her endpoint. Endpoint was individually defined by each subject as that time during the SACM when they felt that they could no longer maintain the required AGSM intensity to sustain consciousness. Subjects terminated the run by releasing a hand-held brake switch. A few days before the experiment the subjects underwent refamiliarization training runs in the centrifuge to practice the Simulated Air Combat Maneuver (SACM). Good reproducibility of their SACM tolerance times was indicated by the test-retest reliability coefficient of 0.86.

Experimental Protocol

Fig. 1 shows that muscle force generation was measured during two different types of movements: leg extension and leg presses. With the exception of the leg press MVC's done at $+4\ G_z$ towards the end of the SACM (described below), all of the other MVC's were performed with the centrifuge spinning at $+1.4\ G_z$. This was done because our centrifuge is programmed to slow down to $+1.4\ G_z$ after the termination of a run and remain there for approximately 30 s before coming to a complete stop. This period would have constituted recovery time for the subject; therefore, both the pre- and post-run MVC's and electrical stimulations were done at $+1.4\ G_z$.

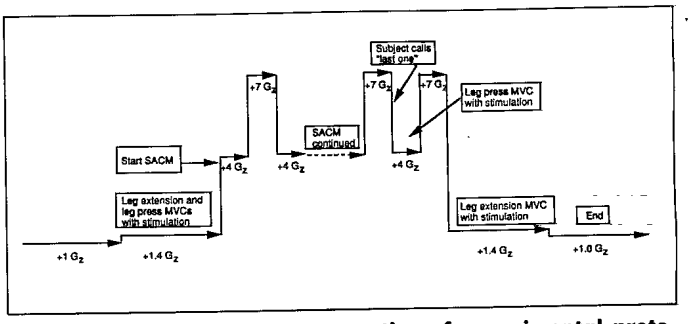

Fig. 1. Diagrammatic representation of experimental protocol. Subjects performed pre-SACM leg press and leg extension maximum voluntary muscle contractions (MVC's) sitting in the centrifuge gondola spinning at $+1.4\ G_z$. They then did the SACM until they indicated impending exhaustion by calling "last one," after which they did the post-SACM leg press MVC's at $+4\ G_z$. The final leg extension MVC's were done at $+1.4\ G_z$.

Leg press MVC's were performed before the SACM and at $+4\ G_z$ between the second-to-last and last $+7\ G_z$ plateau (Fig. 1). Recovery of muscle force occurs rapidly, thus our desire to evaluate force generation as quickly as possible when impending exhaustion was perceived by the subject. Since it takes several seconds for the centrifuge to brake, we decided to evaluate force generation at $+4\ G_z$, but the 10-s time interval restricted our evaluation to only one movement; i.e., leg press. Subjects had to estimate when this point occurred. This procedure was practiced on at least two occasions in order to ensure coordination between subject and experimenter and to establish the reproducibility of G tolerance time. The leg extensor MVC's were performed before and immediately after the SACM runs.

To ensure that full recruitment of available motor units was voluntarily achieved in the unfatigued state, both the leg press and leg extension MVC's were performed twice prior to the centrifuge runs, once with and once without electrical stimulation.

Muscle Force Measurements

Leg extension isometric MVC's were performed in the seated position in the centrifuge gondola by extending the leg at the knee joint. The subjects' ankles were attached via a strap to a force transducer mounted at an angle which allowed the pull on the ankle strap to be directed in a straight line (180°) from the transducer. Leg press isometric MVC's were also done seated in the gondola by pushing against calibrated force plates in the same manner as during an AGSM. The action is similar to that performed with a leg press weight machine; i.e., extension of the thigh at the hip, extension of the leg at the knee, and plantar flexion of the foot at the ankle. The normal footplates in the centrifuge gondola were replaced by an instrumented force plate for each foot, so that both left and right leg forces were measured independently. In addition to the pre- and post-SACM force measurements, the leg press forces were recorded continuously during the AGSM to quantify the relative intensity of muscle contraction during the SACM.

Electrical Stimulation

The subjects arrived dressed in shorts, t-shirts, and flight boots. After appropriate skin preparation, silver-silver chloride EMG electrodes (Medi Trace, Graphic Controls Canada, Gananoque, Ontario, Canada) were placed on the left leg on the surface of the bellies of the vastus lateralis and vastus medialis muscles. The inter-electrode distance was 10 mm. A common ground electrode was placed on the bony part of the lateral aspect of the left knee. After the EMG electrodes were in place, a pair of flexible 3 cm × 4 cm muscle stimulation electrodes were placed on the left leg with the proximal electrode attached to the rectus femoris approximately 5 cm distal to the inguinal ligament and 2 cm lateral to the femoral sheath and the distal electrode attached to the vastus medialis approximately 5 cm proximal and 2 cm medial to the patella. These were held in place using elastic fabric straps with hook and loop closures. Upon stimulation, the electrical current then traveled somewhat transversely across the leg.

After the electrode placement, the subjects donned a standard CSU-15/P anti-G suit which inflated with a pressure of 1.5 psi/G from +2 G_z. The subject then entered the centrifuge gondola and was strapped in for the centrifuge run. All muscle contractions were then performed in this position. The electrical stimulus was delivered to the muscle from a muscle stimulator (Digitimer Model DS7, Digitimer, Welwyn Garden City, England) via skin electrodes described above. The stimulus was regulated by a trigger generator (Digitimer Model DG2, ibid) sufficient to produce a muscle tetanus which generated force in the range of 50–70% MVC in the normal, unfatigued state. The stimulus consisted of a 1 ms pulse at a voltage sufficient to produce a current of 100 mA at a frequency of 100 Hz. The stimulus lasted 150 ms, therefore producing a stimulus train of 15 shocks. The subject was asked to contract the quadriceps muscle maximally using either the leg extension or leg press maneuvers. As they were contracting the muscle, and after 2–3 s to ensure a maximal voluntary effort, the experimenter triggered the generator and the shock was superimposed on the maximal contraction. Any increase in the recorded force caused by the stimulation, above the force generated voluntarily, is suggestive of central fatigue (4,5,13,17,20).

Data Acquisition and Analysis

Signals from the force transducers were recorded on a strip chart recorder (Gould Instruments, Cleveland, OH, Model ES-1000) along with oxygen saturation measured by ear oximetry (Hewlett-Packard, Waltham, MA, Model 47201A), the G level and G-suit inflation pressure. EMG signals were recorded on the strip chart and also on FM magnetic tape using a Hewlett-Packard, Model HP 3968A FM tape recorder. Prior to recording, the signal was bandpass filtered at 10–1000 Hz. The signals were down-loaded to a Macintosh IIci computer and digitized at a sampling rate of 2000 Hz. These signals were subsequently processed to yield root mean square (RMS) amplitude and mean power frequency (MPF) as described elsewhere (3). RMS EMG amplitude and force production were expressed relative to maximal values measured during the pre-SACM MVC's. Since each subject had a different SACM-tolerance time, the EMG and muscle force values were examined at 0, 25, 50, 75, and 100% of the duration of the SACM for each subject.

Statistical analysis was performed using Statview II software (Abacus Concepts, Berkeley, CA). One-way analysis of variance with repeated measures was used to assess differences in force output, RMS EMG and MPF over time. Paired *t*-tests were used to compare the pre- and post-SACM oxygen saturation values. Regression analysis was used to determine if relationships existed between measured variables and SACM time. Significance was accepted at the 0.05 level. All values are expressed as mean ± SD.

RESULTS

Muscle force generation during the leg press and leg extension MVC's did not increase with superimposed electrical stimulation, neither before nor after the SACM. The MVC force levels were similar before and after the SACM for leg extension (14.8 ± 4.1 kg vs. 12.5 ± 3.7 kg before and after the SACM, respectively) and for leg press (157.8 ± 42.5 kg and 149 ± 28.3 kg before the SACM for the left and right legs, respectively, vs. 141.3 ± 39 kg and 132.3 ± 37.9 kg after the SACM). The leg press forces were also measured continuously during the SACM and they did not change over time (Table I); the average relative force output at +7 G_z was 35% MVC for the left leg and 36% MVC for the right leg.

The relative RMS EMG did not change significantly over time for either vastus lateralis or vastus medialis (Table I). Average RMS EMG at +7 G_z (left leg only) was 41.1 ± 17.5% maximal for vastus lateralis and 39.2 ± 24.6% maximal for vastus medialis. Mean power frequency (MPF) decreased significantly over time for both muscles ($p < 0.05$). The decreases were small however, amounting to 8% and 9% for vastus lateralis and vastus medialis, respectively.

The average SACM tolerance time was 230 ± 172 s. While there was large inter-subject variation in the tolerance times, no centrifuge profile was terminated for any reason other than the subject's own subjective perception of exhaustion. Tolerance time was not significantly correlated with any of the force variables examined such as maximal leg press force, maximal leg extension force, or average leg press force (relative or absolute).

Arterial oxygen saturation (S_aO_2) decreased significantly from 97 ± 1 to 90 ± 4% during SACM exposure. There was no relationship between SACM tolerance time and S_aO_2.

DISCUSSION

We speculated that the SACM might cause a hypoxic insult to the CNS, impairing the ability to optimally activate the skeletal muscles in the limbs during the AGSM. The results of the present study do not support this hypothesis. Before the SACM, force did not change with added electrical stimulation indicating that the subjects could voluntarily recruit available motor units during the leg MVC's. A similar level of MVC force after SACM, again with no measurable effects of electrical

TABLE I. FORCE GENERATED AGAINST THE FOOT PLATES AND EMG ACTIVITY OF THE ANTERIOR THIGH MUSCULATURE DURING THE +7 Gz SEGMENTS OF THE SACM. THE FORCE AND THIGH EMG VALUES ARE MEAN ± SD EXPRESSED RELATIVE TO VALUES FOR MAXIMUM CONTRACTIONS OF THE SAME MUSCLES. ONLY MEAN POWER FREQUENCY (MPF) DECREASED SIGNIFICANTLY ($p < 0.05$) OVER TIME.

	Elapsed SACM Time Expressed Relative to Total Time				
	0%	25%	50%	75%	100%
Footplate Force (% max)					
Left	37 ± 24	36 ± 25	34 ± 23	36 ± 20	38 ± 22
Right	37 ± 26	38 ± 23	35 ± 23	38 ± 21	39 ± 22
Thigh EMG (% max)					
v lateralis	43 ± 19	44 ± 20	38 ± 19	42 ± 20	41 ± 20
v. medialis	46 ± 27	39 ± 26	36 ± 25	38 ± 25	39 ± 22
EMG MPF (Hz)					
v. lateralis	78 ± 14	77 ± 15	75 ± 15	75 ± 13	72 ± 14
v. medialis	75 ± 8	74 ± 7	74 ± 7	70 ± 4	70 ± 5

stimulation, indicates that the ability to achieve maximal force and recruit all motor units was sustained during the SACM. Thus no fatigue of the leg musculature was evident during the SACM.

In a previous study (3), we showed that the intensity of muscle contraction of several large muscle groups during an AGSM at +7 Gz was submaximal. In the present study, we measured both EMG and force production for the legs during the SACM. The present results are consistent with our previous data. The average RMS EMG for vastus lateralis at +7 Gz was 41% of maximal values in the present study and 43% of maximal values in the previous investigation. In the present study, we measured leg press force production directly during the SACM, and the contraction levels observed were 35% and 36% MVC for left and right legs, respectively. These were the values at +7 Gz; during the +4 Gz portion of the SACM the values were only around 10% MVC or less. Such a low relative intensity of muscle contraction during half of the SACM would allow for substantial recovery to take place. These results substantiate our contention that peripheral muscle fatigue, at least in the leg musculature, is not likely to be the major cause of a subject's inability to continue an SACM. The lack of a significant increase in RMS EMG over time, a classical electrical index of local muscular fatigue, further supports this contention. We did observe a statistically significant decrease in mean power frequency over time; however, this decrease was very slight. There was also no relationship between the decrease in MPF and SACM time. It is not clear why we observed a decrease in MPF over time in this study but not previously (3).

Our investigations into the role of muscular fatigue as a limiting factor to SACM tolerance time have focused on large skeletal muscle groups, the groups for which strength training exercises have been recommended for high performance aircraft pilots (10). Based on our results (2,3, present), however, one could infer that since minimal fatigue of these large muscles occurs during SACM, then perhaps the time devoted to strength training could be more effectively used in other activities.

We cannot, however, preclude potential fatigue of muscle groups not evaluated in our studies, such as the respiratory musculature. Tolerance time during an SACM is related to the ability to maintain head-level arterial pressure within certain limits, especially at +7 Gz, over a relatively long period of time. This, in turn, depends on the subject's ability to generate sufficient intra-thoracic pressure and to increase peripheral vascular resistance. Assuming that the greatest contribution to raising arterial pressure is related to the increase in intrathoracic pressure generated during the AGSM, then factors which affect this ability will affect SACM tolerance time. These factors include the ability to increase lung volume and then to generate pressure within the thoracic cavity via contraction of the expiratory muscles. If lung volume is not optimal (i.e., not close to 80–100% vital capacity), then the pressures generated will be sub-maximal (7,18). Further research in this area should include examination of respiratory muscle EMG, intrathoracic pressure, and lung volumes in order to examine the relationships between these variables and the inability to continue to perform the AGSM.

In summary, we detected no evidence of central fatigue in subjects performing the AGSM during a +4–7 Gz SACM, even though significant decreases in arterial oxygen saturation were demonstrated. We confirmed previous results (3), showing that the intensity of force generation during the AGSM by major muscle groups, in this case the quadriceps, is submaximal. This investigation indicates that the ability to continue to perform the AGSM during an SACM centrifuge profile is likely not due to central fatigue.

ACKNOWLEDGMENTS

The authors thank LCdr. Bill Bateman, MD, for medical assistance and supervision, Mr. Michel Paul, Capt. Andrew Morris, Mr. Jim Maloan, Mr. Tom Gee, Mr. Gene Steffler, and Cpl. Kim Morey for technical assistance, and the members of the DCIEM A-team for another experiment well-done.

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