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Comparison of the influence of two stressors on steadiness during index finger abduction

Adam R. Marmon, Ph.D and **Roger M. Enoka, Ph.D**

Department of Integrative Physiology, University of Colorado, Boulder CO, 80309 USA

Abstract

Although several stressors have been used to examine the influence of arousal on motor performance, including noxious electrical stimulation, cold pressor test, and mental math calculations, no study has compared the influence of different physical stressors on motor output. The purpose of the study was to compare the influence of two stressors (cold pressor test and electrical stimulation) on the steadiness of the abduction force exerted by the index finger. Sixteen subjects (22.8 ± 3.5 yrs, 8 women) performed steadiness trials before (anticipatory phase), during (stressor phase), and after (recovery phase) each stressor. The steadiness task involved isometric contractions with the first dorsal interosseus muscle, which is the muscle that produces most of the abduction force exerted by the index finger. Subjects were required to match the abduction force on a monitor to a target force set to 5% of the maximal voluntary contraction (MVC) force for 60 s. In contrast to previous studies that examined the influence of stressors on pinch grip steadiness, the two stressors did not decrease steadiness. Furthermore, the absence of a change in steadiness contrasted with the increases in cognitive (State-Trait Anxiety Index, Visual Analog Scale) and physiological (heart rate) arousal during the stressor phase and the subsequent decline during recovery. The null effect of the stressors on index finger steadiness may be due to the relative simplicity of the task compared with those examined previously.

Introduction

When an individual attempts to produce a constant force during a steady contraction, the force is not constant but varies about an average value. The fluctuations in force, which are often used as an index of steadiness, change with contraction intensity [1,2,3,4], contraction type [2,3], and the muscle group used to perform the task [1,5,6]. The magnitude of the fluctuations in force can be influenced by both neural and muscular factors. The muscular factors that can modulate the force fluctuations appear to include muscle size [7,8] and the contractile properties of the motor units activated during the task [9]. A principal neural factor is modulation of the descending command, such as due to the visual requirements of the task or the presence of a stressor that increases physiological arousal [10. 11. 12]. Sosnoff and Newell [13], for example, reported that the greater fluctuations in force observed in old adults compared with young adults was associated with a reduction in visual acuity. However, variation in the intermittency of visual feedback altered force variability in both age groups. Furthermore, Welsh et al. [14] found that, the fluctuations in force were similar for young and old adults in

Adam R. Marmon, McKinly Laboratory, University of Delaware, Newark, DE 19716, Phone: 1-215-882-1507, Fax: 1-302-831-4234, marmon@udel.edu.

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the absence of visual feedback, suggesting that visuomotor corrections contribute to differences in force fluctuations. The fluctuations in force were reduced in both groups when visual feedback was removed during the steady contractions, but to a larger extent in the old group of subjects.

Exposure to stressors has had mixed effects on the magnitude of force fluctuations during steady contractions. The physiologic response to a stressor includes activation of the hypothalamic-pituitary-adrenal axis and the sympathetic nervous system [15], which stimulates the release of glucocorticoids that can influence neuronal communication [16], increase motor neuron discharge variability [17], and suppress the response of muscle spindles [18]. Increases in physiological arousal, therefore, can modulate muscle activation and alter motor output. However, performance of the cold pressor test, which increases physiological arousal, did not alter the steadiness with which healthy young subjects were able to match a target torque of 10% of maximum for 20 s with the ankle dorsiflexor muscles [19]. The constant torque contractions were performed before, during, and 1 and 5 minutes after submerging the left hand in ice water. In contrast, noxious electrical stimulation increased the force fluctuations during pinch grip tasks [10,11,12]. The pinch grip protocols involved healthy young and old adults pinching a force transducer between the thumb and index finger for either 10 s to an absolute target of 4 N [8,9] or for 60 s to a relative target force of 2% of maximum [10].

Although there is evidence suggesting that both the cold pressor test [20] and noxious electrical stimulation [21] increase sympathetic activity, the influence on steadiness appears to be modulated by other factors. One possibility may be related to the muscle groups examined in the different studies. To minimize the confounding influence of the muscle group studied, the present study compared changes in the magnitude of the force fluctuations (steadiness) in a relatively simple model in which the force is primarily produced by a single muscle [22]. The purpose of the current study was to compare the influence of two stressors (cold pressor test and electrical stimulation) on the steadiness of the abduction force exerted by the index finger in the presence and absence of visual feedback.

Methods

Sixteen healthy young adults (18–32 yr, 8 women) volunteered to participate in the study. All subjects were free from neurological diseases and disorders, and were not taking any medications known to influence neuromuscular function. Additionally, all subjects were right handed (Edinburgh Handedness Inventory [23]), had moderate levels of trait anxiety (≥ 40 on the trait portion of the State-Trait Anxiety Index [24]), and had not previously been subjected to an ice bath treatment. The Institutional Review Board for the University of Colorado at Boulder approved the experimental protocol, which was in compliance with the Declaration of Helsinki. Each subject signed an informed consent prior to participating in the study.

Experimental setup

Subjects were seated in a modified dental chair facing a computer monitor positioned at eye level. The elbow was flexed to approximately 110° , the shoulder was abducted slightly, and the wrist and forearm were restrained by two Velcro® straps. The left hand was fitted to a customized device so that the hand was in a neutral position (midway between supination and pronation). The device limited movement of the index finger to the abduction-adduction plane about the metacarpophalangeal joint (Figure 1). The index finger was secured to a bar with Velcro® to prevent flexion of the finger about the metacarpophalangeal joint. Index finger abduction forces were recorded with two transducers (Sensotec, model 13, Columbus, Ohio): a low-sensitivity transducer (0.025 V/N; linear range ~0–222 N) was used to record maximal forces and a high-sensitivity transducer (0.436 V/N; linear range ~0–9.81 N) was used for the low-force steadiness tasks. The abduction force produced by the subjects was sampled at 1

kHz with an analog-to-digital converter (Model 1401+, Cambridge Electronic Design, Cambridge, UK) and stored on a personal computer for off-line analyses.

Maximal Voluntary Contraction (MVC)

The maximal abduction force exerted by the index finger was determined by asking subjects to increase the force from rest to maximum gradually in ~3 s and to then maintain the maximum for an additional 2 s. Repeated contractions were performed until two attempts were within 5% of each other and the greater peak force was used as the subject's MVC force.

Steadiness task

The steadiness task consisted of a 60-s contraction during which subjects were asked to maintain 5% of the previously measured MVC force as steadily as possible. Feedback was provided to subjects on a computer monitor with a custom-written program (LabVIEW 7.1, National Instruments, Austin, TX). The feedback comprised a white target line on a black background and the force exerted by the subject was shown in red. The subject's force line moved left to right across a computer monitor placed 1.2 m in front of the subject and was displayed intermittently; 5 s with feedback and 5 s without. Subjects were instructed to match the index finger force to the target force and to maintain the force even when the feedback was removed. The feedback gain on the computer monitor was 1.36% MVC/cm.

Electromyography (EMG)

Surface EMG was recorded from the first dorsal interosseus muscle using silver-silver chloride electrodes (4-mm diameter). Antagonist muscle activity (second palmar interosseus) was recorded as an intramuscular EMG signal with a fine-wire electrode that comprised two insulated wires (50- μ m diameter, California Fine Wire Company, Grover Beach, CA) and was inserted into the muscle with a 27-gauge hypodermic needle. EMG data were sampled at 2 kHz using an analog-to-digital converter (Model 1401+, Cambridge Electronic Design, Cambridge, UK), amplified by 1,000 (S-series Coulbourn, Allentown, PA), bandpass filtered from 13 Hz – 1 kHz (S-series, Coulbourn, Allentown PA), and stored on a personal computer for off-line analysis. EMG data are expressed as the root mean square and normalized to the EMG obtained during an MVC.

Stressors

Two stressors were administered during the experiment: noxious electrical stimulation and the cold pressor test. The electrical stimulation stressor comprised a series of stimuli (1 ms) delivered to the dorsal surface of the right hand through two stimulating electrodes (Ag/AgCl; ConMED, New York, NY). The intensity of the electrical stimuli was determined by multiplying a threshold by *a priori* specified factors (Table 1). The threshold was the minimal stimulus needed to produce a visible twitch of the hand muscles. The applied current was varied using a constant current unit (Model CCU1; Grass Electronics). The voltage was held at 150 V. A single bout of the electrical stimulation stressor lasted 2 minutes, during which time the lights in the room were alternately switched on and off every 20 s. When the lights were on, there was no threat of stimulation, whereas subjects received 2–5 electrical stimuli when the lights were off. The cold pressor test involved subjects submerging the right hand into a bucket of ice water ($0.93 \pm 0.42^{\circ}\text{C}$) up to the styloid process of the ulna. Subjects were instructed to keep the hand in the bucket for 1–2 minutes or for as long as it could be tolerated.

Assessment of arousal

The Visual Analog Scale (VAS) and the state portion of the State-Trait Anxiety Index (SAI; [25]) were used to assess the influence of the stressor protocols on cognitive arousal. The VAS comprised a 10-cm horizontal line with 'Not at all Anxious' anchored on the left end, and

'Extremely Anxious' anchored on the right end. Subjects were handed a pen and asked to place a vertical tic mark indicating the level of anxiety at that moment. The SAI consisted of 20 statements, such as 'I feel content' and 'I feel secure'. Subjects were asked to respond to each statement, using a 4-point Likert scale, indicating how they felt at the moment. Additionally, heart rate was measured using a Polar® heart rate monitor (Vantage XL, Polar Electro Inc., Lake Success, NY) to assess changes in physiological arousal. The VAS and heart rate were obtained multiple times during each exposure to a stressor, and SAI was administered once during each phase of the protocol (Figure 2).

Procedures

All subjects participated in a single experimental session lasting ~2.5 hours. Each subject completed the experimental protocol twice, once for each stressor, with the order of the two stressors counterbalanced across subjects. The protocol for each stressor comprised three phases: Anticipatory, Stressor, and Recovery (Figure 2). The Anticipatory phase comprised three trials of the steadiness task, each separated by 5 min. Heart rate was measured and the VAS was administered between each steadiness trial, and the SAI was obtained between the second and third trials. Subjects were exposed to three bouts of the stressor during the Stressor phase and a steadiness trial was performed immediately after each bout. Heart rate and VAS were also recorded after each bout and the SAI was again obtained between the second and third trials. Two steadiness trials were performed during the Recovery phase: the first occurred 5 min after the last trial of the Stressor phase and the second was performed 15 min later. The SAI was administered approximately 15 min into the Recovery phase. Heart rate and VAS were recorded at the beginning, middle, and end of the Recovery phase.

Statistical analyses—The influence of the stressors was assessed with repeated-measures ANOVAs (SPSS version 16.0; Chicago, IL). Force steadiness and EMG data were evaluated with three-factor repeated-measures ANOVAs (2 interventions $\times$ 3 phases $\times$ 2 feedback conditions). Quantification of steadiness and EMG were determined for selected segments from the 60-s plateau of each steadiness trial (Figure 3). The plateau was subdivided into six 10-s sweeps; each sweep consisted of 5 s with feedback and 5 s without. The first and last sweeps were discarded and the middle four sweeps were used for analysis. The last 2.3 s of the feedback segment and the first 2.3 s of the no feedback segment were analyzed, excluding a 600 ms transition period between conditions. The force signal during the no feedback condition was detrended prior to the analysis. Force steadiness was quantified as the coefficient of variation for force and EMG was normalized to the level of activity recorded during an MVC.

Bonferroni corrections were applied for multiple comparisons and Huynh-Feldt corrections were applied when sphericity was not assumed. Independent t-tests were used to evaluate differences between phases within an intervention and to compare the same phase across interventions. The cognitive (VAS and SAI) and physiological (heart rate) assessments of arousal were evaluated with two-factor repeated-measures ANOVAs (2 interventions $\times$ 3 phases). The α -level was set to 0.05 for statistical tests and all data are reported as mean $\pm$ standard deviation in the text and tables, and as the mean $\pm$ standard error in the figures.

Results

The results comprise estimates of the change in physiological arousal during two stressor protocols and the accompanying changes in the magnitude of force fluctuations during steady contractions.

Cognitive and Physiological Assessments of Arousal

All assessments of arousal (VAS, SAI and heart rate) were significantly greater during the Stressor phase compared with the Anticipatory and Recovery phases ($p < 0.001$; Figure 4). There were no significant differences in these measures between the Anticipatory and Recovery phases for either stressor.

Steadiness of index finger abduction force

The target force during the steady contractions was 5% of the MVC force, which was 29.8 ± 9.12 N. The mean force produced by each subject did not differ between stressor protocols (cold pressor test: $4.83 \pm 0.38\%$ MVC force, electric stimulation: $4.90 \pm 0.34\%$ MVC force; $p = 0.192$), did not change across the phases of each protocol (4.78 to 4.90% MVC force; $p = 0.268$), and there were no phase-by-stressor interactions. However, the mean force was significantly lower in the absence of visual feedback ($4.81 \pm 0.39\%$ MVC force) than when feedback was provided ($4.93 \pm 0.32\%$ MVC force; $p < 0.001$). The fluctuations in force, as expressed by the coefficient of variation, were also not significantly different for the two stressors (cold pressor test: $2.97 \pm 3.28\%$ MVC force, electric stimulation: $2.52 \pm 1.05\%$ MVC force; $p = 0.390$) and did not change across the phases of each protocol ($p = 0.212$). However, there was a significant main effect for feedback conditions ($F(1,15) = 5.966$, $p = 0.027$) as the relative fluctuations were larger when feedback was provided ($2.91 \pm 2.82\%$ MVC force) compared with when feedback was removed ($2.58 \pm 2.00\%$ MVC force).

Muscle activity

Surface EMG from the first dorsal interosseus muscle was obtained in 13 of the 16 subjects. The amplitude of the surface EMG from the first dorsal interosseus was not significantly different between stressors (cold pressor test: $14.4 \pm 6.9\%$ MVC force, electric stimulation: $17.3 \pm 9.3\%$ MVC force; $p = 0.200$) or across the phases of the protocols ($p = 0.242$). There was significantly less muscle activity when feedback was removed ($15.1 \pm 8.6\%$ MVC force) compared with when feedback was provided ($16.6 \pm 8.0\%$ MVC force) ($F(1,15) = 4.819$, $p = 0.049$). Due to technical difficulties, intramuscular EMG recordings from second palmar interosseus (antagonist to first dorsal interosseus) were only obtained for 10 of the 16 subjects. There were no significant differences in the magnitude of muscle activity in the second palmar interosseus between the two interventions ($p = 0.605$). However, there was a significant main effect for phase ($F(2,18) = 4.031$, $p = 0.036$) as activation of the second palmar interosseus progressively increased from anticipatory to stressor and stressor to recovery phases for both interventions. When the Bonferroni adjustment for multiple comparisons was applied, however, the differences were no longer significant.

Discussion

The purpose of the current study was to compare the influence of two stressors (cold pressor test and electrical stimulation) on the steadiness of the abduction force exerted by the index finger in the presence and absence of visual feedback. In contrast to previous findings, there was no association between increases in physiological arousal and declines in force steadiness [10,11,12].

Why might a stressor modulate steadiness?

In response to stressor-induced increases in physiological arousal, activation of the sympathetic nervous system may influence the ability to control motor output by interfering with proprioceptive information and up-regulating the release of neuromodulators. For example, evidence of depressed muscle spindle sensitivity in response to artificial stimulation of sympathetic nerves has been observed in muscles from several animal models [18,26,27]. In

one study on the masseter muscle in rats, sympathetic activation resulted in a rapid inhibition (~ 0.5 s) of muscle spindle activity [27]. Although the site of modulation remains unclear, findings from these studies suggest that the effects occur at the level of the intrafusal fibers by modulation of the fusimotor drive [18] or as a result of increased calcium reuptake that can inhibit afferent discharge, as has been shown to occur with sympathetic nerve activation [26] and with administration of a calcium antagonist [23,28].

In addition to depressed spindle sensitivity, sympathetic activation increases the release of various neuromodulators that can alter the membrane potential of motor neurons. In the presence of such neuromodulators as serotonin and norepinephrine, for example, spinal motor neurons can develop persistent inward currents that depolarize the membrane potential towards voltage threshold. The activation of persistent inward currents increases the input-output gain of motor neurons and can alter the recruitment and rate coding of motor units [17]. These mechanisms, which are modulated by physiological arousal, would presumably interfere with normal motor output. In the current study, however, no significant changes in steadiness were observed in response to either noxious electrical stimulation or the cold pressor test, even though a previous study in our laboratory demonstrated that the electrical stimulation protocol does impair the steadiness of a pinch grip. The current findings suggest that although the stressors likely activated the sympathetic nervous system, the neuromodulators either did not evoke a functional response in the motor output from the spinal cord or the motor system was able to compensate for these changes.

Specificity of stressors—Although Selye hypothesized that the physiologic response to stressors is non-specific [29], different stressors (insulin, cold exposure, hemorrhage, formaldehyde, and immobilization) have been shown to produce markedly different neuroendocrine responses [30]. For example, different levels of blood epinephrine and norepinephrine have been measured in response to a series of stimuli that included the cold pressor test and administration of caffeine [31]. Also, measures of regional cerebral blood flow have indicated a more pronounced response to pain than cold in the pons [31]. Moreover, differences in the stress response have been associated with the duration [31] and intensity [32] of the stressor stimuli. For example, more intense stressors have been associated with the release of larger concentrations of catecholamines [32]. Nonetheless, the current study found similar changes in cognitive (VAS and SAI) and physiological (heart rate) assessments of arousal in response to the cold pressor test and noxious electrical stimulation, which is consistent with the hypothesis of a non-specific response to stressors [29].

Although there are differences in the response to various stressors, there is a common response that includes increases in the activation of the hypothalamic-pituitary-adrenal axis, activation of the sympathetic nervous system, and in heart rate, blood pressure, and catecholamine release. Although the neuroendocrine responses to the cold pressor test and noxious electrical stimulation were not measured in the current study, it has previously been shown that plasma concentrations of epinephrine, norepinephrine, and adrenocorticotrophic hormone are increased significantly after both noxious electrical stimulation [10] and the cold pressor test [30].

The context of a stimulus and previous exposure to a stimulus also influences the response. For example, a unique, non-aversive stimulus such as exercise can activate the hypothalamic-pituitary-adrenal axis similarly to a stimulus that threatens the animal (33). When an individual has been habituated to an aversive stressor, however, the stress response could be larger for the non-aversive stressor. In the current study, two different physiological stimuli were utilized to increase physiological arousal and habituation of the stimuli was controlled for by ensuring that the subjects had not been previously treated with an ice bath. Because no measurements of catecholamine concentrations or brain stem activation patterns were recorded in the present study, it is not possible to comment on differences in the specificity of the arousal response.

Future studies on animal models are needed to identify the specific pathways and neural circuits that are activated by each stressor, including a quantitative analysis of the timing, concentrations, and types of hormones associated with the response to each stressor.

Influence of visual feedback—The fluctuations in force were significantly smaller without visual feedback compared with when visual feedback was provided, and there was no phase-by-feedback interaction. Findings in the current study are not novel, but agree with previous work (13, 14); when visual feedback is removed, subjects are left with proprioception to determine the intensity of force required for the task and for making any adjustments. A lack of change in steadiness for either feedback condition across phases suggests that increased physiological arousal did influence a person's ability to use proprioceptive feedback when generating constant force using hand muscles.

Contrasting findings—In contrast to the current results, previous results have shown that increased arousal has a detrimental influence on steadiness in an upper extremity model [10, 11,12], but not in a lower extremity model (ankle dorsiflexion [19]). The primary methodological differences between these studies and the current study are the task performed, the contraction intensity maintained, and the stressor to which the participants were exposed. The current study and the study by Christou et al. [10] were also different in that Christou and colleagues examined middle-aged and old adults in addition to the young adults, which may have contributed to the different results obtained in young adults in the current study. Christou et al. [10] also collected blood samples, which may have increased the arousal response. In the current study, however, fine-wire electrodes were inserted into the second palmar interosseus, which likely increased the level of arousal. Subjects in the previous studies on the influence of a stressor on the steadiness of hand muscles performed a pinch task, which involves more muscles than the index finger abduction task used in the current study. Whereas a single muscle is largely responsible for the abduction torque exerted by the index finger [34], the pinch task involves many muscles acting across multiple joints [35]. Findings with another simple model involving a single joint, ankle dorsiflexion, also showed that increased physiological arousal did not influence the fluctuations during a constant force task [14]. These results suggest that increases in physiological arousal may influence the ability to coordinate the actions of multiple muscles, especially when the muscles produce opposing actions, as occurs during pinching and grasping.

Previous studies used either a physical stressor (noxious electrical stimulation [7,8,9] or the cold pressor test [14]), or both physical and mental stressors (mental math and noxious electrical stimulation [9]). The current study was performed to compare the influence of two different physical stressors (noxious electrical stimulation and cold pressor test) on steadiness. Although neither stressor altered steadiness in the current study, the noxious electrical stimulation protocol was based on a previously used protocol that did impair steadiness [7]. As the stressors in these studies were applied to the limb not performing the steadiness task, thereby invoking a systemic response, it seems unlikely that the contrasting findings are related to the type of stressor. The intensity of the constant force contractions also differed between the current study and the previous studies in which stressor-related impairments in steadiness were observed. As the target force (5% MVC) used in the current study is within the range of forces previously examined [7,8,9], contraction intensity is also not likely a factor contributing to the differences in findings between studies. Therefore, the different results obtained in the current and previous studies appear attributable to differences in the experimental task.

Conclusions—The main finding of the current study was that although two physical stressors both increased physiological arousal, neither altered steadiness during an index finger abduction task. The absence of a change in steadiness with exposure to noxious electrical stimulation contrasted with the result obtained in a previous study in which the same stressor

reduced the steadiness of a pinch grip. These results suggest that a stressor-induced modulation of sympathetic nervous system activity does not have a simple, uniform effect on the motor output from the spinal cord.

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Figure 1.

Experimental set-up. Index finger abduction force was recorded with a force transducer located above the proximal interphalangeal joint (a). Surface EMG was recorded from the first dorsal interosseus (b) and intramuscular EMG was recorded from the second palmar interosseus muscle (c; antagonist). Illustrated by Michael Pascoe.

Figure 2.

Experimental protocol. Each subject completed the protocol twice, once for each stressor. Each protocol consisted of three phases; Anticipatory, Stressor, and Recovery. The State portion of the State-Trait Anxiety Index (SAI) was measured once in each phase, whereas the Visual Analog Scale (VAS), heart rate, and steadiness were measured multiple times during each phase.

Figure 3.

Selected force segments for steadiness analysis. Each steadiness trial consisted of six 10-s sweeps, where feedback was presented intermittently (5 s with, 5 s without). The coefficient of variation for force during each trial was determined from the last 2.3 s of the feedback condition and the first 2.3 s of the no-feedback condition, excluding a 0.6 s transition period, of the middle 4 sweeps.

Figure 4.

Assessments of cognitive and physiological arousal. Heart rate (HR; A), State portion of the State-Trait Anxiety Index (SAI; B), and the Visual Analog Scale (VAS; C) were compared between the stressors (electrical stimulation [ES] and cold pressor test [CPT]) and across the phases. * significant difference between the Stressor phase and both the Anticipatory and Recovery phases ($p < 0.001$).

Table 1

Electric stimulation characteristics. Stimulus intensity and quantity of stimuli were adjusted within and across the three bouts of stress during the electrical stimulation protocol. The stimulus intensity was controlled by adjusting the applied amperage using a constant current unit. The intensity factors listed in the table were multiplied by the minimal threshold intensity needed to evoke a visible twitch in the hand.

		Bout 1			Bout 2			Bout 3		
		1	2	3	1	2	3	1	2	3
Intensity Factor (X Threshold)	4.0		4.5	5.0	4.5	5.0	5.5	5.0	5.5	6.0
No. of shocks	2		2	2	4	2	5	4	4	5