Effects of Psychological Stress on State Anxiety, Electromyographic Activity, and Arpeggio Performance in Pianists

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The present study examined the effects of psychological stress, as manipulated by performance evaluation, on the cognitive, physiological, and behavioral components of music performance anxiety (MPA) and performance quality. Twelve skilled pianists (five women, seven men) aged 21.9 ± 3.3 yrs performed arpeggios on a digital piano at the metronome-paced fastest possible tempo under the evaluation and no-evaluation conditions. Measurements were made of self-reported state anxiety, heart rate (HR), sweat rate (SR), and electromyographic (EMG) activity from eight arm and shoulder muscles, and MIDI signals were obtained. The increases in self-reported anxiety score, HR, and SR in the evaluation condition confirmed the effectiveness of stress manipulation. The EMG activity of all the muscles investigated significantly increased from the no-evaluation to evaluation condition, suggesting that psychological stress can add to the risk of playing-related musculoskeletal disorders. Furthermore, the elevated muscle activity in the forearm was accompanied by increased key velocities. We also obtained the first evidence of increased arm stiffness associated with MPA by estimating the co-contraction levels of antagonist muscles in the forearm and upper arm. Consistent with the three systems model of anxiety, the three MPA components were moderately intercorrelated. Participants with high trait anxiety showed stronger correlations between the self-reported anxiety score and other objective measures, which indicated their heightened perceptual sensitivity to physiological and behavioral changes caused by psychological stress. These results provide some practical implications for understanding and coping with MPA. *Med Probl Perform Art* 2008;23:120–132.

Music performance anxiety (MPA) is a serious and frequent problem for many musicians, sometimes leading to impaired performance[1] and even to dropout. Similar to anxiety in general,[2] MPA has been indicated to comprise a loosely correlated constellation of cognitive, physiological, and behavioral components or systems.[3] The cognitive component of MPA includes disturbing mental processes such as the arousal of state anxiety, loss of confidence, and lack of concentration. The physiological component involves somatic symptoms, such as increased heart rate, sweating, and shortness of breath. The behavioral component may manifest itself as tremor, arm and neck stiffness, and shoulder lifting.[4] Since these three components can combine and interact with each other to affect performance quality, research on MPA should assess all three components.

However, most previous studies have focused on only the cognitive or subjective component of MPA on the basis of questionnaire surveys, presumably because of the practical difficulties in monitoring the physiological and behavioral components during live performances in recitals or competitions. Still, it is possible to cause musicians a moderate level of psychological stress in an experimental performing setting while obtaining comprehensive measurements of the three components. Researchers have consistently found that performances entirely aimed at evaluation, such as examinations or competitions, are sources of stress for many musicians.[4] Thus, in the present study, we attempted to manipulate musicians' stress levels by evaluating their performance quality in a laboratory setting and to examine how changes would occur in the three components.

BACKGROUND

So far, several attempts have been made to objectively assess the physiological component of MPA. The most frequently used index among measures of physiological arousal is heart rate (HR), because the development of portable HR monitors has allowed researchers to continuously and telemetrically measure HR without disturbing performing musicians. Previous studies have reported substantial HR increases ranging from 6.4 to 39.2 beats/min (bpm) during performances in front of an audience compared with private performances[3–7] or during live concerts compared with rehearsals.[8,9] These HR changes clearly reflect the activation of sympathetic division of the autonomic nervous system.

Among other physiological measures, sweating or skin conductance has recently been recommended for use as a useful indicator of mental stress or anxiety.[10] Despite the fact that many pianists complain of sweaty palms during public performances, which sometimes cause their fingers to slip on the keyboard, the only study that assessed skin conductance in performing musicians failed to find any significant changes in the measure when piano students were exposed to

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an audience. In the present study, therefore, in addition to reexamining the robustness of HR increases, we attempted to observe the changes in mental sweating response during stressful performance by using an apparatus for measuring sweat rate (SR) directly.

The behavioral manifestations of MPA include arm and neck stiffness, shoulder lifting,[4] and difficulty in maintaining posture and moving naturally. The occurrence of these symptoms appears to be largely explained by changes in muscle activity. In the previous studies on MPA, little attention has been paid to muscle activity, which may influence performance quality more directly than subjective state anxiety, HR, or SR. While MPA research has used electromyographic (EMG) activity of the frontalis muscle as an indicator of anxiety,[11] no study to date has examined in detail the relationship between MPA and muscle activity. Meanwhile, research has consistently demonstrated increased EMG activity in the upper extremity muscles in response to psychological stress by using a variety of tasks including a ball-stroking task,[12] a computer game,[13] computer mouse tasks,[14,15] or demanding mental tasks such as the Stroop color word test and mental arithmetic tests.[16] Visser et al.[14] concluded that the hyperactivity in upper extremity muscles generated by psychological pressure could be one of the risk factors of work-related upper-extremity musculoskeletal disorders.

In the domain of performing arts medicine, on the other hand, musicians with playing-related pain in the neck and shoulder region have been found to show higher levels of EMG activity compared to those without pain.[17] If increased EMG activity can also be observed in musicians under stress, therefore the prevalence of playing-related musculoskeletal disorders may be explained partly by the fact that many musicians are obliged to experience a high level of psychological stress in their daily lives. The changes in muscle activity can affect not only the risk of musculoskeletal disorders but also the task performance itself. Predictably, the elevated muscle activity associated with psychological stress is usually accompanied by increased force outputs, as reflected in the increased movement speed of a stroked ball[12] or increased grip- and click-forces on a computer mouse.[14,15] Based on these findings, the present study aimed to examine whether the same effects (i.e., increased muscle activity and force outputs) can also be observed in performing musicians.

Psychological stress has been suggested to increase not only EMG amplitudes but also co-contraction levels of antagonist muscles in the upper-extremity muscle group.[13] Although muscle co-contraction is metabolically expensive, findings have suggested that it contributes to an increase in joint stiffness, which is a mechanically effective way to stabilize the position of the limb to achieve accurate movements.[18] Osu et al.[19] proposed that co-contraction may be a strategy used by the central nervous system (CNS) early in learning a novel motor task to temporarily improve movement accuracy in the absence of a fully formed internal model of dynamics, and that co-contraction may be reduced as the learning proceeds and the contribution of a feedforward component increases. In the meantime, elevated anxiety has been suggested to provide a new condition for which a new perceptual-motor solution has to be found,[20] possibly because it leads to a deterioration in overall signal-to-noise levels in the motor control system.[13] Thus, there seem to be parallels between the motor performance early in learning and that under psychological stress. To cope with the higher levels of neuromotor noise concomitant with elevated anxiety, the CNS may use limb stiffness through co-contraction to stabilize the movements.[13]

Furthermore, as musicians are repeatedly exposed to anxiety-provoking performance situations, the effects of anxiety on performance quality seem to decrease gradually through adaptation. Some professional musicians even report the “facilitating” effects of anxiety, implying that these experienced musicians have already learned to perform well under the influence of anxiety. Based on these findings, we could assume that muscle co-contraction increases when nonprofessional musicians, who are not exposed to large audiences as frequently as professionals, are required to perform under psychological stress. So far, no study has objectively assessed the changes in stiffness or muscle co-contraction associated with MPA, although musicians report arm stiffness during performance as one of the behavioral manifestations of MPA.[4] Therefore, the present study attempted to collect the first evidence of co-contraction increases in response to psychological stress by employing skilled amateur musicians.

According to the three systems model of anxiety,[2,3] the cognitive (i.e., self-reported state anxiety), physiological (i.e., HR and SR), and behavioral (i.e., EMG amplitudes and co-contraction) components of MPA should be moderately intercorrelated. However, research has often failed to find any significant relationship among the three response systems, probably because the individual differences that may modulate the concordance among the systems were not considered.[21] So far, two studies[3,21] have suggested that individuals with high trait anxiety tend to reveal higher levels of concordance among anxiety components. According to Calvo and Miguel-Tobal,[21] one possible explanation for the finding is that the physiological and behavioral responses are stronger and perceptually salient in individuals with high trait anxiety, causing their subjective experience to be more related to the objective measures of anxiety. An alternative explanation is that individuals with low trait anxiety possess a cognitive bias to avoid processing threat stimuli (perceptual suppression) and/or to inhibit reporting of subjective feelings of distress (verbal response suppression), which may be a defensive function to avoid awareness of negative affects. Based on these previous studies, we investigated whether the degree of anxiety concordance significantly differs between musicians with low trait anxiety and those with high trait anxiety, and which of these two interpretations can better account for our data.

Clearly, the central issue concerning MPA would be the actual deterioration of performance quality, which results from the changes in movements and muscle activities. Therefore, examining the relationship between EMG activity and performance quality may offer some explanations as to why MPA often leads to impaired performance quality. In order

FIGURE 1. Test piece used in the experiment.

to correlate EMG activity with performance quality, it is necessary to objectively quantify performance quality. Recently, the development of digital recording technology based on the musical instrument digital interface (MIDI) system has allowed researchers to analyze keyboard motor skills in detail, making it possible to describe technical aspects of the skills in statistical terms.[22] The MIDI technology has actually been shown to be effective in quantifying movement impairment caused by overuse syndrome[23] or focal dystonia.[22,24] Thus, a descriptive analysis of MIDI data collected in real time can provide information about subtle differences in performance quality between experimental conditions, which may be otherwise undetectable to performers or listeners.

The purpose of the present study was threefold. First, we examined whether a moderate level of psychological stress induced in a laboratory setting produces significant changes in the three components of MPA. Second, we investigated the relationships among the three components, especially focusing on the difference in the degree of concordance between musicians with low trait anxiety and those with high trait anxiety. Third, we examined the changes in performance quality in relation to EMG activity by employing a MIDI-based performance quantification technique.

METHODS

Participants

Twelve skilled amateur pianists (5 women, 7 men; participants P1 to 12) were recruited from a university piano club in Tokyo, Japan. The participants were between 18 and 31 yrs of age (mean 21.9, SD 3.3), with a mean of 15.8 yrs (SD 5.9) of playing experience and a mean of 11.7 yrs (SD 3.7) of private instruction, and included five prize winners of celebrated domestic and/or international piano competitions held in Japan. All of them were capable of sight reading, and none was familiar with the musical piece used in this experiment. The subjects gave written informed consent to participate. This study was approved by the Ethics Committee of the Graduate School of Arts and Sciences, the University of Tokyo.

Experimental Task

On the basis of a professional pianist's advice, the A-flat major and G-sharp minor arpeggios were combined to form a test piece in six-eight time (Figure 1). Participants were asked to play the piece mezzoforte (medium loudness) and in staccato style (notes were played in a detached and distinctly separate manner) on a digital piano (P-60s; Yamaha, Tokyo, Japan), whose action is very similar to that of a regular acoustic piano keyboard. Fingerings were according to the standard accepted arpeggio fingerings; i.e., the pianists played 2, 1, 2, 4, 1, 2, 4, 2, 1, 4, 2, 1 . . . by their right hand and 3, 1, 4, 2, 1, 4, 2, 4, 1, 2, 4, 1 . . . by their left hand, where 1 is the thumb, 2 the index finger, 3 the middle finger, 4 the ring finger, and 5 the little finger. The piece was repeatedly performed at the fastest possible tempo which was set for each pianist prior to the experiment. The tempo was paced by the metronome of the digital piano, which was set to eighth-notes. After the metronome was turned on, participants waited for the first four bars with their hands on the keyboard, carefully listening to the metronome ticks (*preparatory* phase). Then they performed eight bars of arpeggios (*performance* phase). We instructed participants to reduce the number of pitch errors, to synchronize the note onsets with the metronome ticks, and to maintain constant loudness and tone durations as much as possible.

Apparatus and Data Collection

Screening questionnaire: Prior to the experiment, participants completed the trait subscale of the State-Trait Anxiety Inventory (STAI).[25] The Cronbach's α coefficient for the subscale was 0.75, indicating a high level of internal consistency.

Self-reported state anxiety measure: To measure the levels of subjective state anxiety, we used the "anxiety thermometer," which is a 100-mm continuous scale ranging from 0 (not anxious at all, the left end) to 100 (extremely anxious, the right end). The validity and reliability of the scale have been confirmed by Houtman and Bakker.[26] Just prior to performing the arpeggios in each trial, participants were asked to place a short vertical line on the scale to indicate their anxiety level at the moment.

Signal recording: To assess the mean HR as an indicator of physiological arousal, electrocardiograms (ECG) were recorded with bipolar Ag/AgCl surface electrodes placed on the anterior chest.

We also assessed mental sweating, which usually appears on palms and soles. In order not to disturb participants, we assessed the local SR on the sole of the foot with a SR meter (TS100; Techno Science, Tokyo, Japan) utilizing the ventilated capsule technique. A detecting probe (capsule) of 1 cm^2 was tightly attached to the left plantar arch. A stream of air was passed continuously through the lumen of the capsule via inlet and outlet housings at a fixed flow rate, and the amounts of moisture in the inlet and outlet housings were measured by two hygrosensors, respectively. The amount of evaporated water in the capsule was obtained from the difference between the amounts of moisture in the inlet and outlet housings.

EMG activity was recorded with bipolar Ag/AgCl surface electrodes placed on the extensor digitorum (ED) and flexor carpi ulnaris (FCU) muscles of the right forearm, the long heads of triceps brachii (TB) and biceps brachii (BB) of the bilateral upper arms, and the upper trapezius (TR) of the bilateral shoulders. Before the electrodes were attached, the surface of the skin was treated with alcohol and then rubbed with fine sandpaper to reduce the inter-electrode resistance.

Participants wore a jacket on which the transmitter of a telemeter (SYNA ACT; NEC, Tokyo, Japan), which wirelessly sent the ECG and EMG data from the right TR to the receiver, was attached. EMG signals were amplified 500 times. All signals were sampled at 1000 Hz with an analog-digital converter (NR-2000; Keyence, Tokyo, Japan) and stored in a personal computer. Figure 2 shows a typical example of signal records for a single participant.

Performance recording: The keyboard of the digital piano was equipped with a MIDI, the output of which was fed to a personal computer via a MIDI translator (MIDI sport uno; M-audio Japan, Tokyo, Japan). For recording and generating MIDI files, we used a commercially available music-editing software (Singer Song Writer Lite 4.0 for Windows; Internet, Tokyo, Japan). The software was used to measure pitch values, key velocities (an indirect measure of loudness ranging from 0 to 127, in MIDI units), tone durations (time between note onset and end of note, in ms), and time off the metronome beat (the difference between the actual time and the expected time of the key press, in ms).

We attached a switch on the lowest A key of the piano, which was connected to a 3-V battery and the NR-2000. When the A key was depressed, an electric signal and a tone signal were recorded simultaneously by the wave-analyzing software and the music editing software, respectively. During the preparatory phase, one of the experimenters depressed the A key in order to synchronize the MIDI data with the ECG, SR, and EMG signals for the later analyses.

Design and Procedure

All participants were instructed not to drink caffeinated or alcoholic beverages within 24 hrs before the experiment. Upon arrival at the laboratory, where two experimenters were present, the detecting probe of the SR meter and the ECG and EMG electrodes were attached. We then obtained the baseline measurements of anxiety. Participants were instructed to sit quietly for a 5-min adaptation period during which they completed the trait subscale of the STAI. After the adaptation period, the baseline data of HR and SR were collected while participants were seated in a relaxed manner in an adjustable piano chair for 8 min. We also asked participants to indicate their level of anxiety at the moment on an AT, which was regarded as their baseline level of subjective anxiety.

After measuring the resting levels of anxiety, participants practiced the test piece until they became able to play it fluently (usually for about 5 min). Then, the performing tempo was set for each participant. First, we asked a participant to play the piece in time to the metronome set at a tempo at which he or she could play it easily. Second, the participant repeatedly played the piece while raising the tempo by five quarter-notes/min at a time. When performance quality was greatly impaired, the tempo was again reduced gradually. Finally, the tempo at which the participant could fluently play the piece with less than three pitch errors was selected as his/her performing tempo. The mean performing tempo among participants was 133.2 quarter-notes/min (SD 46.9).

Subsequently, participants received a detailed explanation of the experimental procedure. We first told participants that they could gain cash rewards according to the total score they achieve over the four blocks of the experiment, which consisted of five trials each. We also told them that the score would be calculated based on the number of pitch errors they committed during the performance phase of each trial. Three types of errors were regarded as "pitch errors": a) A *substitution* involved an intruder (an unintended note) replacing a target (an intended note), b) an *addition* involved an intruder being added without replacing a target, and c) a *deletion* involved a target being deleted.[27]

For the first four trials of each block, psychological stress was increased in the *evaluation* condition. In this condition, every time a participant committed a pitch error during the performance phase, 20 points were subtracted from the original 100 points. For example, if a participant made three pitch errors, the score for the trial resulted in 40 points. If the participant made more than five pitch errors, the score was 0 point. In the *no-evaluation* condition, on the other hand, participants always won 100 points regardless of the number of pitch errors. Two of the first four trials of each block had the evaluation condition and the other two had the no-evaluation condition. To exclude order effects, these two types of conditions were presented to participants in a randomized order. Participants received feedback on the obtained score immediately after performing the test piece in each trial.

To keep participants highly motivated over the course of the experiment that lasted for 3 hrs on average, a special trial called the *final trial* was inserted into the end of every block. Two of the four final trials had the reward condition, and the other two had the penalty condition. The score for a block was determined based on the total score of the first four trials and the number of pitch errors made in the final trial, and the total score for a participant was calculated by summing

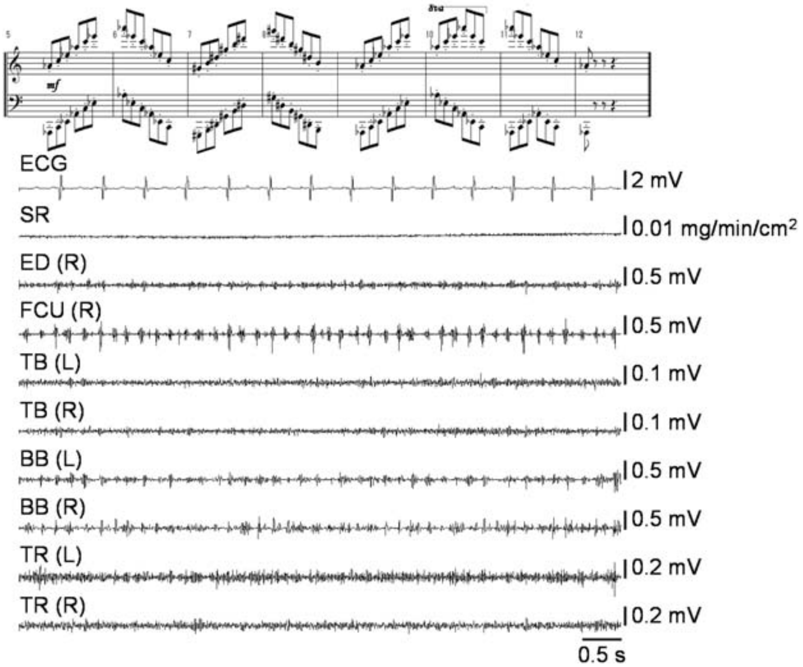

FIGURE 2. Typical example of signal records. SR, sweat rate; ED, extensor digitorum; FCU, flexor carpi ulnaris; TB, triceps brachii; BB, biceps brachii; TR, trapezius; L, left; R, right.

up the four block scores. Because of the absence of sufficient number of trials, the data obtained from the final trials were excluded from the main analyses.

Data Analysis

Anxiety measures: For each trial, the distance from the left end to the vertical line of the anxiety thermometer (AT, in mm) was measured. Then the *AT change* (in mm) was computed for each trial by subtracting the baseline anxiety level of the participant from the obtained distance.

We calculated the mean HR (in bpm) during the performance phase for each trial from the R-R intervals of the collected ECG data. Then the *HR change* (in bpm) was computed for each trial by subtracting the resting HR level of the participant from the mean HR during the performance phase. Similarly, we computed the *SR change* (in mg/min/cm^2) for each trial.

EMG activity. EMG signals were full-wave rectified and then low-pass filtered at 50 Hz. To facilitate comparisons between electrodes and across subjects, EMG values (in mV) for each muscle were normalized relative to the maximum EMG activity for that muscle observed over the trials of the no-evaluation condition. Since the EMG data included some noisy signals, the maximum level of EMG activity for each trial was determined as the 95 percentile point in the EMG-amplitude distribution of the performance phase. The mean value of the normalized EMG amplitudes (in %max EMG) during the performance phase was computed for each muscle, and for each trial.

Additionally, we computed a measure of co-contraction.[18] For each of the agonist-antagonist pairs—i.e., for the ED and FCU and for the TB and BB—at each sampling point in time, the minimum value of normalized EMG was computed, discarding the portion of EMG in one muscle that was not matched by the EMG in the opposing muscle. The resulting time-varying signal is considered to represent EMG activity in antagonist muscles that increases joint stiffness. We computed the mean value of the signals (*co-contraction index*) observed during the performance phase for each muscle pair and for each trial.

Performance quality. To evaluate performance quality, we first counted the number of substitutions, additions, and deletions and the total number of pitch errors for each trial. After excluding the notes with pitch errors, we computed the means and standard deviations of key velocities and tone durations for the remaining notes of the trial. Additionally, we calculated two measures of timing error: the *constant error* (the difference between the actual time and the expected time of the key press) and the *absolute error* (the absolute value of the difference between the actual time and the expected time of the key press) for each note. We then computed the means of constant errors and absolute errors for each trial.

Statistical Analysis

Nonparametric statistical models were selected because of the small sample size and the distribution patterns of the dependent variables. Differences in the AT change, HR change, SR change, mean normalized EMG amplitudes, co-contraction index, and performance measures between the evaluation and no-evaluation conditions were assessed by Wilcoxon matched-pair signed-rank tests. Prior to the application of the tests, the obtained values of all the variables

TABLE 1. Medians and Quartile Deviations of Anxiety Measures for the Evaluation and No-evaluation Conditions

	Median (Quartile Deviation)			Change		
Measurement*	Baseline	No-evaluation	Evaluation	No-evaluation	Evaluation	p†
AT score (mm)	26.0 (13.5)	30.7 (6.6)	53.1 (6.0)	3.2 (15.5)	22.3 (9.4)	<0.01
HR (bpm)	75.3 (5.7)	82.5 (10.4)	88.5 (7.3)	9.3 (3.9)	14.3 (3.7)	<0.01
SR (mg/min/cm^2)	0.0296 (0.0281)	0.0568 (0.0338)	0.0580 (0.0479)	0.0214 (0.0265)	0.0248 (0.0257)	<0.10

*AT, anxiety thermometer; HR, heart rate; SR, sweat rate.

†The p values indicate the significance of the differences in the AT change, HR change, and SR change between the evaluation and no-evaluation conditions.

were averaged across trials for each condition and for each participant.

To determine the significance of associations among the three anxiety measures or between anxiety measures and EMG activity, we computed Spearman's rank correlation coefficients on a trial-by-trial basis. The data for all 20 trials, including final trials, were incorporated into the analyses. Prior to the correlation analyses, the z-score value for a given variable was computed for each trial by using the mean and SD of the variable over all the trials of the participant. The normalization to z-scores had the effect of eliminating differences in the means and SDs of variables between participants.

To examine the individual differences in the degree of anxiety concordance, we first assigned participants to either the *low-anxiety* or *high-anxiety* group on the basis of a median split of the trait subscore of the STAI. We then performed two types of analyses[21]: a) correlations between variables within the low- or high-anxiety group, and b) Fisher's Z-transformation tests of significant differences between the correlation coefficients for the low- and high-anxiety groups.

To exploratively evaluate the significance of associations between EMG activity and key velocities, we first computed Spearman's rank correlation coefficients between the z-scores of mean EMG amplitudes and those of mean key velocity on a trial-by-trial basis. Then, we performed a stepwise multiple regression analysis with the mean EMG amplitudes of the right arm muscles as the independent variables and the mean key velocity of the right hand as the dependent variable, because the EMG data for both the forearm and upper arm were available in terms of the right side. Considering the individual differences in the relationship between EMG activity and key velocities, we also conducted an individual-based correlation analysis between these variables.

The p-values <0.05 or <0.10 were regarded as statistically significant or marginally significant, respectively.

RESULTS

State Anxiety at the Subjective and Physiological Level

Table 1 shows the medians and quartile deviations of the AT score, HR, and SR for the evaluation and no-evaluation conditions. The Wilcoxon matched-pair signed-rank test performed on the AT change revealed that participants reported a significantly higher level of anxiety in the evaluation condition than in the no-evaluation condition: Wilcoxon $Z = -3.06$, $p < 0.01$. Similarly, the HR change was significantly greater in the evaluation than in the no-evaluation condition: Wilcoxon $Z = -2.67$, $p < 0.01$. In terms of the SR change, a marginally significant difference was found between the two conditions: Wilcoxon $Z = -1.65$, $p < 0.10$. The effectiveness of stress manipulation was confirmed by these subjective and physiological changes associated with anxiety.

EMG Activity

The psychological stress had a remarkable effect on the pianists' EMG activity during the performance phase. The mean normalized EMG amplitudes of all of the muscles investigated significantly increased in the evaluation condition: Wilcoxon $Z = -3.06$, $p < 0.01$ for the right ED; Wilcoxon $Z = -2.04$, $p < 0.05$ for the right FCU; Wilcoxon $Z = -2.59$, $p < 0.05$ for the left TB; Wilcoxon $Z = -2.75$, $p < 0.01$ for the right TB; Wilcoxon $Z = -2.82$, $p < 0.01$ for the left BB; Wilcoxon $Z = -2.59$, $p < 0.05$ for the right BB; Wilcoxon $Z = -2.67$, $p < 0.01$ for the left TR; Wilcoxon $Z = -2.98$, $p < 0.01$ for the right TR, respectively (Figure 3).

The co-contraction activity of antagonist muscles in the forearm and upper arm also significantly changed from the no-evaluation to evaluation condition. The co-contraction index for all of the muscle pairs revealed significantly higher values in the evaluation condition: Wilcoxon $Z = -2.67$, $p < 0.01$ for the right forearm (i.e., ED and FCU); Wilcoxon $Z = -2.75$, $p < 0.01$ for the left upper arm (i.e., TB and BB); Wilcoxon $Z = -2.59$, $p < 0.05$ for the right upper arm, respectively (Figure 4).

Relationships among Anxiety Measures and EMG Activity

Correlations among anxiety measures: In order to investigate how the individual anxiety symptoms were related to each other, we computed Spearman rank correlations among anxiety measures (see the rightmost column of Table 2). The AT change positively and significantly correlated both with the HR change ($p < 0.001$) and SR change ($p < 0.05$). Moreover, a significant positive correlation was found between the HR change and SR change ($p < 0.05$).

Correlations between anxiety measures and EMG activity: As the consistent increases in the AT change and HR change were found in the evaluation condition, we examined the correlations of these anxiety measures with the mean EMG amplitudes and co-contraction index values (see Table 3). The HR change was positively and consistently related to the mean EMG amplitudes of all the muscles investigated ($p < 0.05$ for each). The AT change positively and significantly correlated with the mean EMG amplitudes of five arm muscles ($p < 0.05$ for each). The co-contraction index was associated with the two anxiety measures more strongly. Both the AT change and HR change had significant positive correlations with the index for all of the three antagonist muscle pairs ($p < 0.01$ for each).

Comparison of the degree of anxiety concordance between the low- and high-anxiety groups: First, we attempted to confirm the validity of the median-split method of grouping participants. Based on a Mann-Whitney *U* test, we confirmed that the participants in the high-anxiety group exhibited higher trait anxiety score (median = 55, *QD* = 2.3) than those in the low-anxiety group (median = 44, *QD* = 3.3), $Z = -2.89$, $p < 0.01$. Because the state-trait theory of anxiety predicts that the individuals with high trait anxiety tend to show increased state anxiety when exposed to stressful situations,[25] we computed the *AT increase* for each participant by subtracting the mean AT change in the no-evaluation condition from that in the evaluation condition. A Mann-Whitney *U* test suggested that the AT increase was larger for the high-anxiety group than for the low-anxiety group, $Z = -1.85$, $p < 0.10$. Furthermore, a significant positive correlation was found between the STAI trait anxiety score and the AT increase: $r_s = 0.71$, $df = 10$, $p < 0.05$ (Figure 5). These results clearly justified the median-split method. Although the AT increase tended to be larger for the high-anxiety group, no significant difference was found in the *HR increase* or in the *SR increase* between the two groups.

Second, we compared the correlations among anxiety measures between the low- and high anxiety groups (Table 2). For the low-anxiety group, significant positive correlations were found between the AT change and HR change ($p < 0.05$) and between the HR change and SR change ($p < 0.05$). For the high-anxiety group, the AT change was positively and significantly related both to the HR change ($p < 0.001$) and SR change ($p < 0.01$). The correlation between the AT change and HR change was significantly stronger for the high-anxiety group than for low-anxiety group ($p < 0.05$). Additionally, a marginally significant difference was found in the correlation between the AT change and SR change between the two groups ($p < 0.10$).

Third, we compared the correlations between anxiety measures (i.e., AT change and HR change) and EMG activity between the low- and high-anxiety groups (Table 3). The AT change positively and significantly correlated with the mean EMG amplitudes of six muscles ($p < 0.05$ for each) for the high-anxiety group, whereas no significant correlation was found between the variables for the low-anxiety group. Additional tests revealed that four out of eight comparisons involved correlations that were significantly different between the two groups ($p < 0.05$ for each). The HR change was positively and significantly related to the EMG amplitudes of five muscles ($p < 0.01$ for each) for the high-anxiety group, whereas only two significant correlations ($p < 0.01$) were found between the variables for the low-anxiety group. We found significant differences in the correlations between the variables only in two out of eight muscles investigated between the groups ($p < 0.05$).

In terms of the co-contraction activity of antagonist muscles, both the AT change and HR change positively and significantly correlated with the index for all the muscle pairs for the high-anxiety group ($p < 0.01$ for each). For the low-anxiety group, however, the AT change was related to none of the co-contraction index values, and the HR change was significantly related only to the co-contraction index for the right upper arm ($p < 0.05$). Additional tests demonstrated that the correlations between the AT change and co-contraction index values were significantly stronger for the high-anxiety group than for low-anxiety group in two out of three

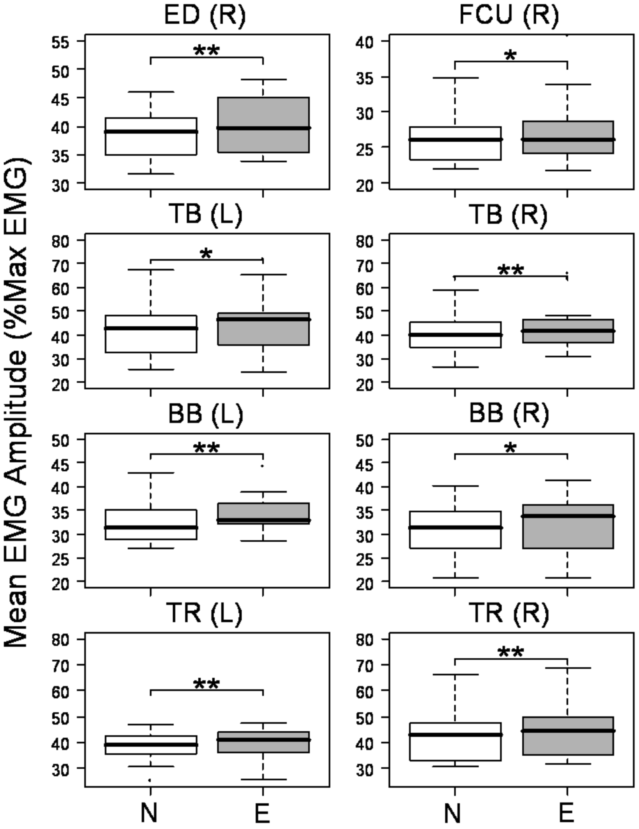

FIGURE 3. Box and whisker plots of the mean EMG amplitudes. The box represents the middle half of the data, the horizontal line in the box is the median, and the whisker is at 1.5 × the range of the middle half of the data from the ends of the box: ED, extensor digitorum; FCU, flexor carpi ulnaris; TB, triceps brachii; BB, biceps brachii; TR, trapezius; L, left; R, right; N, no-evaluation condition; E, evaluation condition. * $p < 0.05$, ** $p < 0.01$.

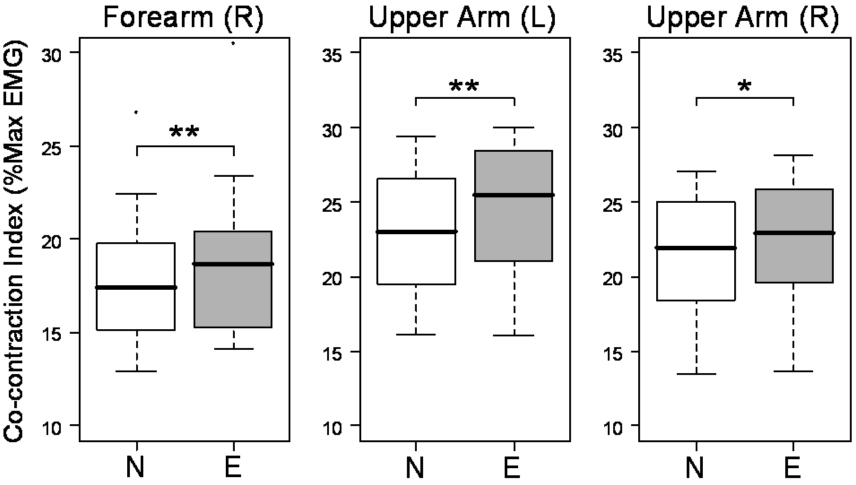

FIGURE 4. Box and whisker plots of the co-contraction index. L, left; R, right; N, no-evaluation condition; E, evaluation condition. * $p < 0.05$, ** $p < 0.01$.

muscle pairs ($p < 0.05$ for both). The HR change correlated with the co-contraction index for the left upper arm more strongly for the high-anxiety group than for low-anxiety group ($p < 0.05$).

Performance Quality

Comparisons of performance measures between two conditions: Substantial individual differences were found in the effects of psychological stress on the total number of pitch errors. Of the 12 participants, 7 committed fewer pitch errors and the remaining 5 more pitch errors in the evaluation condition compared to the no-evaluation condition. Accordingly, no statistically significant difference was found at the group level (Figure 6). In terms of the changes in the number of each type of pitch errors, we found an interesting tendency; although more participants showed increased number of substitutions (seven vs. five) and additions (nine vs. three), more participants showed decreased number of deletions (eight vs. four) in the evaluation condition (Figure 6).

We also examined the means and SDs of key velocities and tone durations and the means of constant errors and absolute errors (Figure 7). We found a marginally significant increase in the mean key velocity in the evaluation condition: Wilcoxon $Z = -1.88$, $p = 0.06$. No significant difference was found in the other measures of performance quality.

Relationship between EMG activity and key velocities: As a statistically meaningful difference in performance quality between the two experimental conditions was found only in the mean key velocity, we further investigated what change in muscle activity resulted in an increase in key velocities.

First, we computed the correlations between the mean normalized EMG amplitudes of the left arm and shoulder muscles and the mean key velocity of the left hand, and between the mean EMG amplitudes of the right arm and shoulder muscles and the mean key velocity of the right hand respectively, on a trial-by-trial basis. As a result, the mean EMG amplitudes of both the left TB and BB positively and significantly correlated with the mean key velocity of the left hand: $r_s = 0.32$, $df = 236$, $p < 0.001$, and $r_s = 0.24$, $df = 236$, $p < 0.001$, respectively. As for the right arm, the mean EMG amplitudes of ED, FCU, and TB positively and significantly correlated with the mean key velocity of the right hand: $r_s = 0.41$, $df = 236$, $p < 0.001$; $r_s = 0.40$, $df = 236$, $p < 0.001$; and $r_s = 0.27$, $df = 221$, $p < 0.001$, respectively. Additionally, a marginally significant correlation was found between the right BB activity and mean key velocity: $r_s = 0.12$, $df = 236$, $p < 0.10$. On the other hand, the EMG activity of neither left TR nor right TR was related to the mean key velocity. These group-based correlation analyses indicated that increased muscle activity in the forearm and upper arm was associated with increased key velocities.

TABLE 2. Correlations among Anxiety Measures

	Low-Anxiety Group (n = 6)		High-Anxiety Group (n = 6)			All (n = 12)	
	r_s	(df)	r_s	(df)	z	r_s	(df)
AT–HR	0.23*	(118)	0.46***	(116)	2.03*	0.34***	(236)
AT–SR	0.03	(118)	0.26**	(116)	1.81†	0.14*	(236)
HR–SR	0.19*	(118)	0.14	(116)	0.38	0.17*	(236)

AT, anxiety thermometer score change; HR, heart rate change; SR, sweat rate change.
The z values indicate the significance of the differences in the correlation coefficients between the low- and high-anxiety groups.
† $p < 0.10$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

TABLE 3. Correlations between Anxiety Measures and EMG Activity

	AT change							HR change						
	Low (n = 6)		High (n = 6)			All (n = 12)		Low (n = 6)		High (n = 6)			All (n = 12)	
	r_s	(df)	r_s	(df)	z	r_s	(df)	r_s	(df)	r_s	(df)	z	r_s	(df)
ED (R)	–0.06	(118)	0.22*	(116)	2.18*	0.08	(236)	0.09	(118)	0.17†	(116)	0.68	0.14*	(236)
FCU (R)	–0.02	(118)	0.40***	(116)	3.38**	0.20**	(236)	0.09	(118)	0.29**	(116)	1.58	0.20**	(236)
TB (L)	0.09	(118)	0.40***	(116)	2.51*	0.25***	(236)	0.05	(118)	0.38***	(116)	2.60**	0.24***	(236)
TB (R)	0.07	(114)	0.39***	(105)	2.53*	0.23**	(221)	0.13	(114)	0.37***	(105)	1.91†	0.25***	(221)
BB (L)	0.15†	(118)	0.25**	(116)	0.79	0.20**	(236)	0.12	(118)	0.37***	(116)	2.05*	0.24***	(236)
BB (R)	0.17†	(118)	0.16†	(116)	0.08	0.15*	(236)	0.15	(118)	0.27**	(116)	0.96	0.21**	(236)
TR (L)	0.08	(118)	0.09	(116)	0.09	0.09	(236)	0.27**	(118)	0.13	(116)	1.16	0.20**	(236)
TR (R)	–0.00	(118)	0.19*	(116)	1.52	0.09	(236)	0.25**	(118)	0.16†	(116)	0.67	0.20**	(236)
CI-F (R)	–0.06	(118)	0.42***	(116)	3.83***	0.18**	(236)	0.12	(118)	0.32***	(116)	1.62	0.23***	(236)
CI-U (L)	0.11	(118)	0.37***	(116)	2.11*	0.24***	(236)	0.12	(118)	0.41***	(116)	2.41*	0.28***	(236)
CI-U (R)	0.15†	(118)	0.28**	(116)	0.99	0.21**	(236)	0.20*	(118)	0.33***	(116)	1.12	0.28***	(236)

Low, low-anxiety group; High, high-anxiety group. AT, anxiety thermometer score; HR, heart rate; ED, mean EMG amplitude of extensor digitorum; FCU, mean EMG amplitude of flexor carpi ulnaris; BB, mean EMG amplitude of biceps brachii; TB, mean EMG amplitude of triceps brachii; TR, mean EMG amplitude of trapezius; CI-F, co-contraction index for the forearm; CI-U, co-contraction index for the upper arm; L, left; R, right.

The z values indicate the significance of the differences in the correlation coefficients between the low- and high-anxiety groups.

† $p < 0.10$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

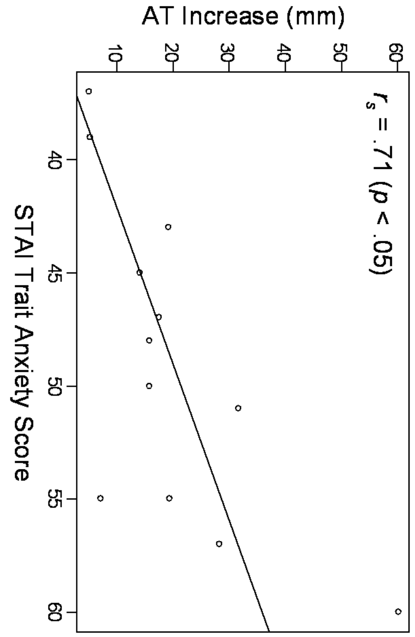

FIGURE 5. Direct relationship between the STAI trait anxiety score and AT increase. The AT (anxiety thermometer) increases are plotted as a function of the trait anxiety score of the STAI (State-Trait Anxiety Inventory).

Second, to specify the activity of which arm muscles affected key velocities, we performed a multiple regression analysis with the mean EMG amplitudes of the right arm muscles as the independent variables and the mean key velocity of the right hand as the dependent variable. Because of the exploratory nature of the analysis, we utilized the stepwise method. The mean EMG amplitudes accounted for a statistically significant portion of the variance of the mean key velocity: F (2, 220) = 22.22, p <0.001. The mean EMG amplitudes of the right ED and FCU emerged as the significant predictors of the mean key velocity: standardized β = 0.27, p <0.001, and standardized β = 0.21, p <0.01, respectively. Figure 8 shows the scatter diagrams in which the standardized mean key velocities of the right hand are plotted as functions of the standardized mean EMG amplitudes of the right ED and FCU.

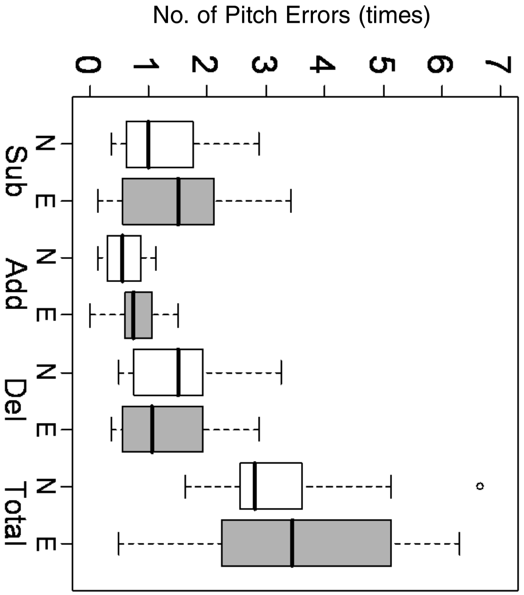

FIGURE 6. Box and whisker plots of the number of pitch errors. Sub, substitution; Add, addition; Del, deletion; Total, total number of pitch errors; N, no-evaluation condition; E, evaluation condition.

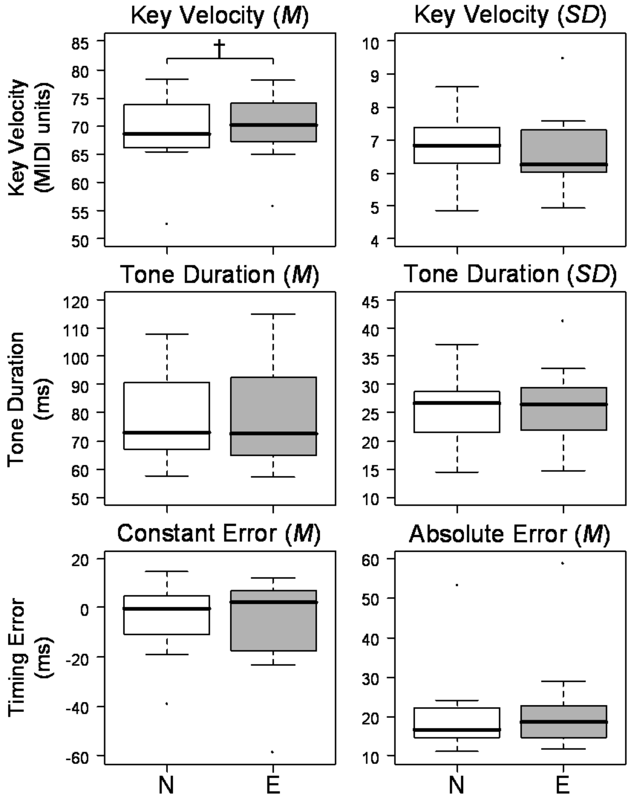

FIGURE 7. Box and whisker plots of the means and SDs of key velocities and tone durations, and means of timing errors: N, no-evaluation condition; E, evaluation condition.† $p < 0.10$.

Since the multiple regression analysis suggested that the activity of forearm muscles influenced key velocities at the group level, we further computed the correlations between the mean EMG amplitudes of the right ED and FCU and the mean key velocity of the right hand for each participant (Table 4). Ten out of the 12 participants revealed meaningful associations between the EMG activity of these two muscles and the mean key velocity. Of the 10 participants, 6 (i.e., participants P1, P2, P7, P8, P10, and P12) showed positive correlations between the mean EMG amplitudes of both muscles and the mean key velocity (p <0.10 for each). As for the remaining 4 participants, the mean key velocity significantly correlated only with the ED activity in P3 and P6 ($p < 0.05$ for both), and only with the FCU activity in P4 and P5 (p <0.01 for both), respectively.

DISCUSSION

The aim of the present study was to examine the changes in cognitive, physiological, and behavioral components of MPA and performance quality in response to increased psychological stress and their interrelationships for better understanding the nature of MPA.

The comparisons of anxiety measures between the two experimental conditions confirmed that the present method of manipulating participants' stress levels was highly effective. The AT score showed that participants felt more anxious in the evaluation than in the no-evaluation condition, supporting the idea that performance evaluations are sources of stress for many musicians.[4]

In addition to subjective experience, both the mean HR and SR were elevated in the evaluation condition. The HR change during performance increased by a median of 5 bpm from the no-evaluation to evaluation condition (Table 1). In comparison with previous research, the amount of increase was comparable to that found in members of the BBC Symphony Orchestra (+6.4 bpm) from rehearsals to live performances. The present data are highly valuable in that a considerable increase in physiological arousal was induced without employing a large audience, lending further support for the validity of conducting MPA research in a laboratory setting. Although Craske and Craig[3] failed to find any change in skin conductance in pianists performing under stress, we found a marginally significant increase in the mean SR in the evaluation condition. Thus, SR may be a more sensitive measure of the mental sweating response than skin conductance, which has long been used in anxiety research. However, the increase of SR was not as substantial as that of HR. Therefore, relatively greater individual differences may exist in the mental sweating than in cardiac acceleration associated with MPA. Obviously, future research should use larger samples to determine if the SR increases consistently occur during stressful music performances.

Psychological stress also produced significant effects on muscle activity levels. Congruent with previous studies using different tasks,[12–16] the mean EMG amplitudes of all the upper extremity muscles investigated significantly increased in the evaluation condition (Figure 3). Obviously, the present experiment is the first to have systematically verified that the activity of musicians' arm and shoulder muscles can be elevated during stressful performances. Philipson et al.[17] reported that professional violinists with playing-related pain exhibited higher levels of EMG activity in the right BB and the bilateral TR during performance compared with those without pain, which indicated that the elevation of muscle tension during performance can be a risk factor of playing-related musculoskeletal disorders. Although the increases in EMG amplitudes observed in the present experiment were relatively small, they can modify the risk of developing the disorders if continued over long periods of time. It is also worth remarking that the bilateral TR activity substantially increased from the no-evaluation to evaluation condition, because the upper TR has been indicated to be especially sensitive to psychological stress.[14–16] The increased TR activity may have led to shoulder lifting, which is one of the common symptoms of MPA.[4]

As hypothesized, the co-contraction activity of antagonist muscles in the right forearm and in the bilateral upper arms was elevated in the evaluation condition. Despite the fact that arm stiffness can be seen as one of the behavioral symptoms of MPA,[4] no objective data on its change in response to psychological stress had been available. Therefore, the present study obtained the first evidence of increased arm stiffness as estimated by muscle co-contraction in the arm, in

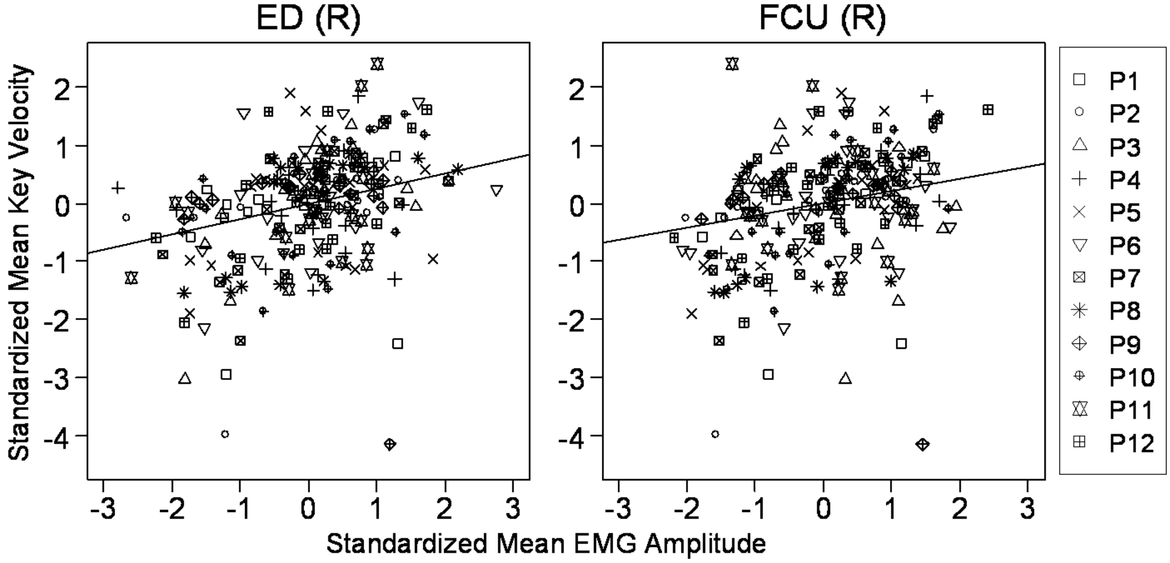

FIGURE 8. Direct relationship between the mean EMG amplitudes of the right forearm muscles and the mean key velocity. The standardized mean key velocities of the right hand are plotted as functions of the standardized mean EMG amplitudes of the right ED (extensor digitorum) and FCU (flexor carpi ulnaris).

musicians performing in a stressful situation. Even though significant emotional changes, as reflected in the AT score, mean HR, and SR, occurred in the evaluation condition, we found no significant difference in performance accuracy measures (i.e., number of pitch errors, standard deviations of key velocities and tone durations, and the means of timing errors) between the two conditions. The results are accountable if we consider that the CNS may have used changes in co-contraction as a compensatory way to maintain movement accuracy, which might have otherwise been impaired, under the stressful or unstable condition.

Since the ED contributes to the extension and the FCU to the flexion of wrist joint, the co-contraction of these two muscles may have led to wrist stiffness and hence to the stabilization of hand positions relative to the keyboard, which may in turn have helped fingers to strike the keys accurately. The biarticular muscles of upper arm, i.e., the long head of TB and the BB, have been suggested to be activated by the CNS to control the elbow joint rather than the shoulder joint,[28] indicating that the co-contraction of these two muscles mainly contributed to elbow stiffness. Therefore, by increasing elbow stiffness, participants may have attempted to stabilize the position of the forearm relative to the keyboard to achieve accurate movements. To test these assumptions, future studies should combine the EMG measures of co-contraction with direct measurements of limb stiffness, and with motion analyses of performance.

Since the significant activation in the three MPA components was caused by psychological stress, we further investigated the interrelationships among the components. Consistent with the three systems model of anxiety,[2] statistically significant yet moderate levels of correlations were found between the subjective and physiological measures of anxiety (Table 2), between the anxiety measures and mean EMG amplitudes, and between the anxiety measures and co-contraction index (Table 3). Some previous studies[6,7] have failed to find any significant relationship between the subjective and physiological aspects of MPA, presumably because these studies employed the inter-individual correlations. Since physiological measures also may be affected by nonemotional factors[3] that can hardly be controlled in experiments, the significant associations might have been masked by the individual differences in other factors. On the other hand, the repeated measurements of variables and the calculation of intra-individual correlations in the present study enabled us to effectively demonstrate the significant relationships among the three components.

The findings are considered reasonable, because from an evolutional perspective, an adaptive response that helps an

TABLE 4. Correlations between Mean EMG Amplitudes of Right Forearm Muscles and Mean Key Velocity of Right Hand for Each Participant

Participant	ED (R)	FCU (R)	*df*
P1	0.56*	0.65**	18
P2	0.39†	0.67**	18
P3	0.71***	−0.27	18
P4	0.04	0.61**	17
P5	0.13	0.62**	18
P6	0.46*	0.33	18
P7	0.67**	0.74***	18
P8	0.68**	0.70**	17
P9	0.12	0.15	18
P10	0.56*	0.68**	18
P11	0.23	0.05	18
P12	0.49*	0.51*	18

† $p < 0.10$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

organism to avoid dangerous situations would be to mobilize most organismic resources in coordination to maximize effectiveness.[21] A remarkable discovery here would be that the relationship between HR and EMG activity was consistent across different muscles (Table 3), in accordance with previous findings.[16] On the basis of the present results, it may safely be inferred that the sympathetic activation associated with MPA may have led to the hyperactivity of alpha motoneurons in the spinal cord. We also found that the co-contraction index values were related to anxiety measures more consistently than the mean EMG amplitudes. Therefore, we argue that the co-contraction index can be used as an indicator of anxiety more effectively than the simple activation levels of muscles.

We observed a higher level of concordance among the three MPA components in pianists with high trait anxiety than in those with low trait anxiety, supporting the previous findings.[3,21] The difference between the low- and high-anxiety groups was particularly marked in the correlations between self-reported state anxiety and other objective measures. With respect to the interrelationships among anxiety measures, both the correlation between the AT change and HR change and the correlation between the AT change and SR change were stronger for the high-anxiety group than for the low-anxiety group (Table 2). Furthermore, the AT change was associated with EMG activity (i.e., mean EMG amplitudes and co-contraction index) more strongly for the high-anxiety group, with significant differences in the correlation coefficients between the two groups in 6 out of 11 comparisons (Table 3). Considering that no significant difference was found in the HR increase or SR increase between the two groups, Calvo and Muguel-Tobal's[21] first hypothesis, which insisted that the concordance is higher in individuals with high trait anxiety because their physiological responses are stronger, might not explain our results. Rather, their second hypothesis appears to reasonably account for the difference between the two groups; i.e., the concordance was higher in pianists in the high-anxiety group because those in the low-anxiety group possessed a cognitive bias to avoid processing threat stimuli or to inhibit reporting subjective feelings of distress.[21]

Compared with the correlations between self-reported anxiety and other objective measures, the differences in the correlations among physiological and behavioral measures seemed smaller between the low- and high-anxiety groups. The correlation between the HR change and SR change was not significantly different between the two groups. Concerning the relationship between the HR change and EMG activity, the high-anxiety group exhibited significantly stronger correlations in only 3 out of 11 comparisons. Therefore, what distinguishes individuals with high trait anxiety from those with low trait anxiety seems to be the perceptual sensitivity to the physiological and behavioral changes associated with psychological stress, rather than the physiological responsiveness to threat stimuli or the concordance among physiological and behavioral responses.

The comparisons of performance measures between the evaluation and no-evaluation conditions showed that only the mean key velocity differed between the two experimental conditions. Although key velocity has a nonlinear relationship with fingertip force applied to the key,[29] it nevertheless provides a rough estimate of keystroke force. Therefore, consistent with previous research using other tasks,[12,14,15] the pianists' force outputs appear to have increased in response to psychological stress. The increased levels of key velocity might have led to more substitutions and additions and less deletions in the evaluation condition. The present finding is practically important, because generating the required levels of loudness by striking the keys with appropriate force levels is absolutely one of the most fundamental skills for artistic expressions. The loss of control over loudness under psychological stress can greatly impair performance quality. The elevated muscle activity might partly explain this change in force outputs.

We found significant positive correlations between the mean EMG amplitudes of arm muscles and the mean key velocity of the ipsilateral hand. A multiple regression analysis indicated that the elevated muscle activity especially in the forearm contributed to increased key velocities. Furuya et al.[30] suggested that pianists are likely to inhibit the proximal joint movements as the performing tempo increases. Furthermore, when performing at relatively low loudness (*pp*–*mf*), pianists tend to control loudness through changes in the wrist joint angles by mainly using the distal muscles.[30] These findings may explain why the activity of forearm muscles positively predicted key velocity levels, since in the current study pianists performed the arpeggios at the fastest possible tempo and at medium loudness. The individual-based correlation analyses, however, revealed considerable individual differences in the relationship between the EMG activity in forearm muscles (i.e., ED and FCU) and the mean key velocity. The results might partially be attributed to the individual differences in the key-striking movements.

The present findings provide some important implications for understanding and coping with MPA. Although previous studies have demonstrated the utility of MIDI keyboard technology for the analysis of movement disorders,[22–24] none has applied this technology to MPA research. The present study, however, showed that MIDI technology can also be an effective and reliable tool for quantification of changes in performance quality associated with MPA. This study also is the first to examine in detail the effects of psychological stress on muscle activity in musicians. The results showed that the EMG activity of arm and shoulder muscles and the co-contraction activity of antagonist muscles in the forearm and upper arm were heightened in conjunction with elevated anxiety. Although these changes appeared in part to be an adaptive response to maintain movement accuracy, they can add to the risk of playing-related musculoskeletal disorders and impair performance quality by making it difficult for performers to generate smaller loudness. To avoid these detrimental effects, musicians should train themselves to gain full control of their muscle activity during practice or through muscle relaxation training.[11] Finally, we observed the higher level of concordance among the three MPA components in pianists with high trait anxiety than in those with low trait anxiety, indicating that anxious pianists are more sensitive to the physiological or behavioral changes in response to psychological stress than their nonanxious counterparts. It is recommended, therefore, that when put under great stress, musicians with high trait anxiety try to divert their attention away from the physiological or behavioral responses and to concentrate on music itself to prevent subjective state anxiety from growing.

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