Bursts and variability of beta oscillations mediate the effect of anxiety on motor exploration and motor learning.

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Abstract

Anxiety results in sub-optimal motor performance and learning through mechanisms still unknown. Here we addressed whether state anxiety impairs motor learning through changes in behavioral and neural variability. Participants completed a reward-based motor sequence learning paradigm, with separate phases for exploration (baseline) and learning. Anxiety was manipulated either during baseline or learning. We show that anxiety at baseline reduces motor variability, undermining subsequent reward-based learning. By contrast, unconstrained baseline exploration led to successful motor learning, even under the effect of anxiety. The behavioral changes were driven by changes in the variability of sensorimotor beta oscillations (13-30Hz, SBO). Moreover, bursts of SBO, a marker of physiological beta, lasted longer under the effect of anxiety, resembling recent findings of pathophysiological beta in movement disorders. Our findings suggest that changes in variability and burst duration in SBO represent a neural mechanism through which anxiety constrains movement variability, with detrimental consequences for motor learning.

Introduction

Anxiety involves anticipatory changes in physiological and psychological – cognitive, emotional, behavioral – responses to a potential and uncertain future threat[1-2]. Previous work on the neurobiology of anxiety established that trait anxiety interferes with prefrontal control of attention in perceptual tasks, whereas state anxiety modulates the amygdala during detection of threat-related stimuli[2-3]. In the area of motor control, research has shown that stress and anxiety have detrimental effects on performance[4-5]. These results have been partially interpreted as the interference of anxiety with information-processing resources[6]. However, the effects of anxiety on motor learning are often inconsistent and a mechanistic understanding is still lacking. Delineating mechanisms through which anxiety influences motor learning is important to ameliorate its impact in different settings, including in motor rehabilitation programmes.

Motor variability could be the primary component of motor learning that is affected by anxiety; it is defined as the variation of performance across repetitions[7], and is driven by various factors including sensory and neuromuscular noise[8]. As a form of action exploration, movement variability is increasingly recognized to benefit motor learning[9-11]. These findings are consistent with the vast amount of research on reward-based reinforcement learning demonstrating increased learning following initial exploration[12]. More recently movement variability was shown to benefit motor learning when it takes the form of 'intentional' exploration of the task space, not as motor noise[13]. Yet contextual factors can reduce variability. For instance, recent work on ritualistic behavior reveals that state anxiety leads to movement redundancy, repetition, and rigidity to regain a feeling of control[14]. This finding resembles the reduction in behavioral variability and exploration that manifests across animal species during the fight or flight response in stressful environments[15]. Based on these results we set out to test the hypothesis that state anxiety modulates motor learning through a reduction in motor variability and action exploration.

Additionally, we posited that changes in motor exploration are driven by neural variability in premotor and motor areas. Support for our hypothesis comes from recent data in animal studies demonstrating that variability in the primate premotor cortex tracks behavioral variability during motor planning[16]. Further evidence in rodents and primates supports that changes in variability in single-neuron activity in motor cortex drive motor exploration during initial learning, and reduce it following intensive training[17-18]. Also, the basal ganglia are crucial for modulating variability during learning and production, as shown in songbirds and, indirectly, in patients with Parkinson's disease[11, 19-20].

In the present study, we analyzed sensorimotor beta oscillations (SBO, 13-30Hz) as a candidate mechanism

driving motor exploration and variability. Beta oscillations have been linked to different aspects of performance and motor learning[21-23], as well as reward-based learning[24]. Although amplitude or power changes was traditionally the primary focus of research on oscillations, there is a renewed interest towards assessing dynamic properties of oscillations, such as the presence of brief bursts[25], which are considered to be a central feature of physiological beta in motor-premotor cortex and the basal ganglia[26-28]. The assessment of variability and burst duration of SBO thus allows us to capture dynamic changes in neural variability induced by anxiety and their link to behavioral effects.

To test our hypotheses, we recorded electroencephalography (EEG) in three groups of participants while they completed a reward-based motor sequence learning paradigm, with separate phases for motor exploration (baseline) and reward-based learning. Crucially, different sequences were used in each phase of the task to exclude carry-over effects of learning from the baseline period. We manipulated anxiety by informing participants about an upcoming public speaking task that would require them to describe an unknown art object to a panel of experts[14]. Using a between-subject design, the anxiety manipulation targeted either the baseline or the reward-based learning phase. Analysis of the EEG signals aimed to assess anxiety-related changes in the variability and burst duration in SBO in relation to changes in behavioral variability.

Our primary finding was that anxiety impairs reward-based learning by constraining motor variability and action exploration during the baseline phase. Importantly, these effects were mediated by increased within-trial variability and burst duration in SBO. A second experiment served to demonstrate that anxiety during reward-based learning has an opposing effect on motor variability and learning rates depending on the presence or absence of a preceding baseline exploration phase.

Results

Sixty participants completed our reward-based motor sequence learning task, consisting of three blocks of 100 trials each over two phases (**Figure 1**): a baseline motor exploration (block 1) and a reward-based learning phase (blocks 2 and 3: termed training hereafter). Prior to the experimental task, we recorded in each participant 3 min of EEG at rest with eyes open. Next, on a digital piano, participants played two different sequences of seven and eight notes during the exploration and training phases respectively (**Figure 1A**). They were explicitly taught the tone sequences prior to the start of the experiment, yet precise instructions about the timing or loudness (keystroke velocity) were not provided.

During the exploration phase, participants were informed they could freely change the rhythm and/or the loudness of the performance of sequence1 every trial, and that no reward or feedback would be provided. During training, however, participants received performance-based feedback in the form of a 0-100 score at the end of each trial, and were informed that the overall average score would be translated into monetary reward. They were directly instructed to explore the temporal or loudness dimension (or both) and to use feedback scores to discover the unknown performance objective (which, unbeknownst to them, was a specific rhythmic pattern). The task-related dimension was therefore timing, whereas keystroke velocity (Kvel) was the non-task related dimension. Timing in our task referred to the pattern of inter-onset-intervals between consecutive keystrokes (IOI, ms). The score increased when the difference between the coefficient of variation of the performed and target rhythm patterns (IOIs) decreased (see *Materials and Methods*).

Participants were pseudo-randomly allocated to either a control group or to one of two experimental groups (**Figure 1B**): anxiety during exploration (anx1); anxiety during the first block of training (anx2). The lack of anxiety manipulation during block3 thus allowed us to assess the dissociable effects of anxiety

during baseline exploration or training on the learning rates during the last training block. We measured changes in heart-rate variability (HRV), heart-rate (HR) and state anxiety scores four times throughout the experimental session: resting state (3 min, prior to performance blocks); block1; block2; block3. The HRV significantly dropped during the targeted blocks relative to the initial resting phase in each group (**Figure 1 – figure supplement 1**), confirming that the experimental manipulation succeeded in inducing physiological responses consistent with an anxious state[29]. Statistical analysis of behavioral and neural measures focused on the separate comparison between each experimental group and the control group (contrasts: anx1 – controls, anx2 – controls). See *Materials and Methods*.

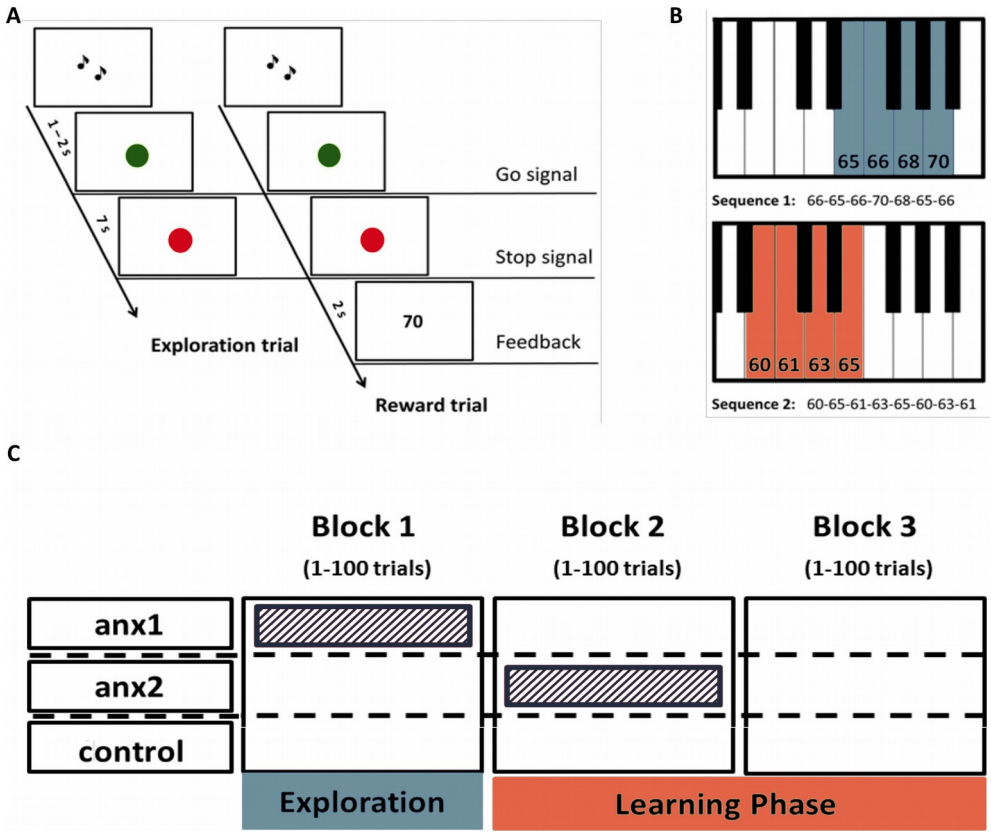

Figure 1. A Novel Paradigm for Testing Exploration and Reward-Based Learning during Sequence Performance. (A) Schematic of the task. Participants played sequence1 during 100 exploration trials, followed by 200 trials of reward-based learning performing sequence2. After each reward-based learning trial, participants received a performance-related score between 0-100. (B) Pitch content of the sequences used in the exploration (sequence1) and reward-based learning blocks (sequence2), respectively. (C) Schematic of the anxiety manipulation. The shaded area denotes the phase in which anxiety was induced in each group, using the threat of an upcoming public speaking task, which took place immediately after that block was completed.

General Effects of Baseline Task-related Variability and Exploration on Reward-based Learning

All groups of participants demonstrated significant improvement in the achieved scores during reward-

based learning, confirming they effectively used feedback to approach the hidden target performance (**Figure 2**: $p < 0.05$, after control of the false discovery rate at level $q = 0.05$ due to multiple comparisons[30], termed FDR-corrected thereafter; anx1: non-parametric effect size[31], $PS_{dep} = 0.80$; anx2: $PS_{dep} = 0.88$; controls: $PS_{dep} = 0.90$). Detailed analysis of the trial-by-trial changes in scores and performance will be reported elsewhere.

Assessment of motor variability was performed separately in the task-related temporal dimension and the non-task-related keystroke velocity dimension. Temporal variability – and similarly for keystroke velocity – was estimated using two different measures (**Figure 2B)**: the within-trial and across-trials coefficient of variation of IOI (cvIOI). The within-trial cvIOI provided a total of 100 values across each experimental block. By contrast, the across-trials cvIOI provided one single value per experimental block. Because the score obtained during reward-based learning was explicitly related to the within-trial cvIOI, we predicted that higher values of this parameter at baseline would be associated with higher reward during the subsequent training phases. Of note, higher within-trial cvIOI values denote a larger departure from an isochronous performance of the sequence. However, we also hypothesized that a higher degree of exploration across trials at baseline (that is, playing different temporal patterns in each trial), and therefore higher across-trials cvIOI, would improve subsequent reward-based learning.

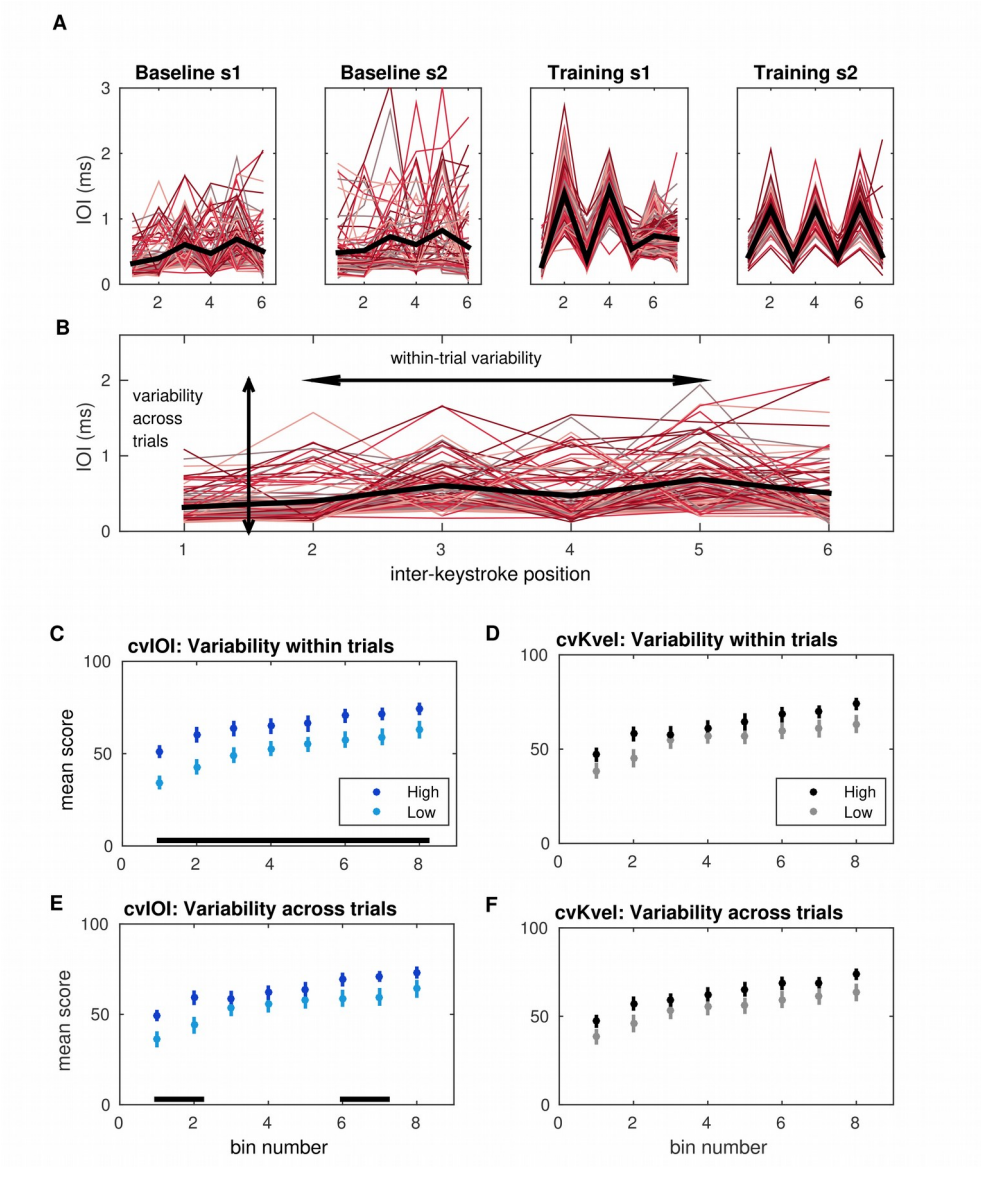

Figure 2.Temporal variability within and across trials at baseline contributes to subsequent reward-based learning. (A) Illustration of timing performance during baseline exploration (left panels) and training (right panels) blocks in two representative participants, s1 and s2. X-axis represents the position of the inter-keystroke interval (sequence1: 7 notes, corresponding to 6 inter-keystroke temporal intervals; sequence2: 8 notes, 7 inter-keystroke intervals). Y -axis shows the inter-onset interval (IOI) in ms. Black lines represent the mean IOI pattern. (B) Task-related variability was measured using two parameters: the within-trial and across-trials coefficient of variation of IOI, cvIOI. (C) Scores achieved by participants during training following a median split of all 60 participants into high and low within-trial cvIOI at baseline. Trials were split into bins of 25 trials and scores were averaged within each bin. Black bars at the bottom indicate the bins of significant between-group differences ($p < 0.05$, FDR-corrected). (D) Same as C but for keystroke velocity, using cvKvel to do a median split of participants. E-F. Same as C-D but using the across-trials cvIOI and cvKvel, respectively. Bars around the mean display ±SEM.

To first evaluate the effect of baseline within-trial temporal variability on subsequent reward-based learning, regardless of the group, we did a median split of all 60 participants based on the within-trial cvIOI, averaged across trials. This analysis revealed that larger within-trial cvIOI at baseline was associated with higher scores during training ($p < 0.05$, FDR-corrected; PS_{dep}= 0.91; **Figure 2C**). Corresponding with this result, there was a significant non-parametric rank correlation between the values of within-trial cvIOI at baseline, and also later during training – as expected, and the average scores obtained (Spearman $\rho = 0.474$, $p = 0.001$, at baseline; $\rho = 0.646$, $p = 0.00001$, during training). A control analysis performed with groups of low and high values of within-trial cvKvel demonstrated a non-significant difference in subsequent scores ($p > 0.05$; **Figure 2D**).

We also stratified participants based on the degree of across-trials cvIOI at baseline exploration. Participants whose performance exhibited a higher across-trials cvIOI at baseline achieved higher scores during training ($p < 0.05$, FDR-corrected; PS_{dep} = 0.81; **Figure 2E**). Changes in across-trials cvKvel did not influence subsequent reward-based learning ($p > 0.05$; **Figure 2F**).

Notably, the amount of within-trial variability expressed by participants in timing and keystroke velocity was not correlated ($\rho = 0.019$, $p = 0.898$). Neither was the across-trials cvIOI and cvKvel ($\rho = 0.021$, $p = 0.788$). This supports that the temporal and velocity dimensions in our task were uncorrelated and, in principle, participants could vary them separately. Participants, however, generally used a lower amount of variability in Kvel relative to timing at baseline, likely due to the higher difficulty required to precisely control loudness during piano performance.

Influence of Anxiety on Baseline Variability and Subsequent Reward-based Learning

Next, we assessed pair-wise differences between each experimental group (anx1, anx2), separately, and the control group. Participants affected by state anxiety at baseline (anx1) achieved significantly lower scores in the subsequent reward-based learning phase relative to control participants (**Figure 3A:** $p < 0.05$, FDR-corrected, between-group non-parametric effect size[31], PS_{sup} = 0.78). By contrast, in the anx2 group scores did not significantly differ from the scores in the control group ($p > 0.05$). Converging with the previous analysis, the total average score (related to the amount of money received) achieved by anx1 participants was significantly smaller than the amount received by control participants (52 [SEM 3] for anx1, 63 [3] for controls, $p = 0.02$, PS_{sup} = 0.85). Anx2 and controls did not achieve significantly different average scores than control participants (61 [3] for anx2; $p > 0.05$). A planned comparison between both experimental groups demonstrated significantly higher total average scores in anx2 ($p = 0.045$, PS_{sup} = 0.67).

At baseline, anx1 used a lower degree of within-trial and across-trials cvIOI than the control group (**Figure 3BC**. Within-trial cvIOI: $p < 0.05$, FDR-corrected; PS_{sup} = 0.67; Across-trials cvIOI: $p = 0.032$; PS_{sup} = 0.67). There was no between-groups (anx1-controls) difference in within-trial or across-trials variability in Kvel ($p > 0.05$, **Figure 3 – figure supplement 1**). Performance at baseline in anx2 did not significantly differ from performance in the control group, both for cvIOI or cvKvel, and for within and across-trials variability ($p > 0.05$).

Performance in the Training Phase: Exploration and Exploitation

We evaluated whether the significant increase in scores found in each group from beginning to end of the training blocks was paralleled by a significant drop in the across-trials cvIOI, reflecting exploitation of the rewarded options (**Figure 3**). A 2x2 factorial analysis of the across-trials cvIOI with factors Group (anx1, control) and Phase of training (block2, block3) demonstrated a significant main effect Phase and interaction effect ($p < 0.05$, FDR-corrected). Further exploration of the interaction effect established that in control

participants – not in anx1 – the across-trials cvIOI dropped from training block2 to block3 ($p < 0.05$, FDR-corrected, $PS_{sup} = 0.66$). A similar 2x2 analysis comparing anx2 and control groups revealed a significant main effect Phase ($p < 0.05$, FDR-corrected), due to smaller across-trials cvIOI values in block3 in both groups. Collectively, these findings support that during reward-based learning exclusively participants in the anx2 and control groups went through a gradual transition from an explorative regime (characterized by higher across-trials cvIOI) to an exploitative regime, in parallel to their achieving higher scores.

Additional similar 2x2 factorial analyses of the average score and within-trial cvIOI with the abovementioned Phase and Group factors demonstrated significant main effects for Phase in all cases ($p < 0.05$, FDR-corrected: all groups had larger scores and within-trial cvIOI in the second training block), a main effect Group for anx1 and controls ($p < 0.05$, FDR-corrected) and no significant interaction effects. This finding suggested that the transition in scores and within-trial task-related variability from the first to the second training blocks was similar in all groups, despite anx1 having significantly overall lower within-trial cvIOI and lower scores than control participants.

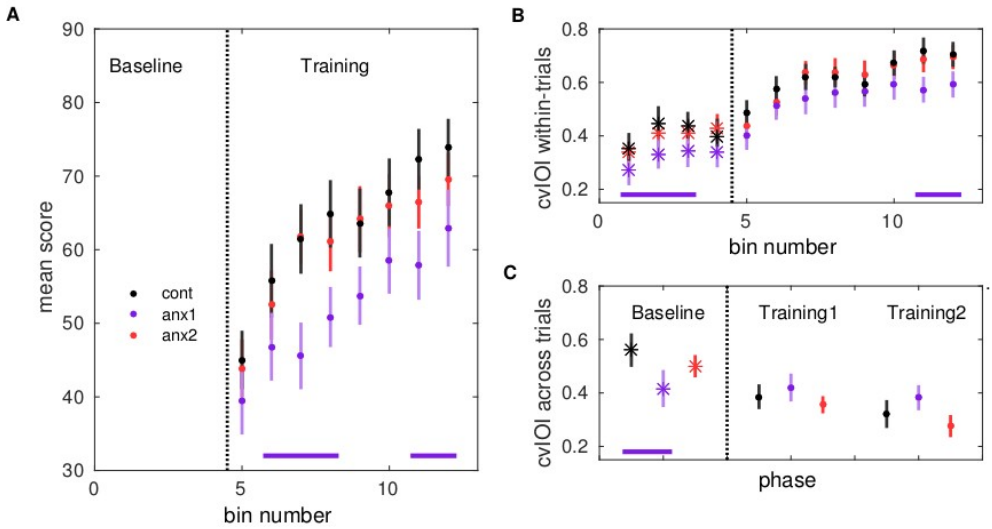

Figure 3. Effects of anxiety on behavioral variability and reward-based learning. The score was computed as a 0-100 normalized measure of proximity between the pattern of inter-onset intervals performed in each trial and the target rhythm ([0.2, 1, 0.2, 1, 0.2, 1, 0.2] s). (A) Scores achieved by participants in the anx1, anx2, and control groups across bins 5:12 (bins of 25 trials: trial range 101-300), corresponding with blocks 2 and 3 and the training phase. Participants in anx1 achieved significantly lower scores than control participants in bins 6:8 and 11:12 (trials 125-200 and 250-300, $p < 0.05$, FDR-corrected, denoted by the bottom purple line). (B) Changes in within-trial cvIOI from the exploration phase (bins 1-4) to the training phase (bins 5-12). Participants in anx1 used smaller within-trial cvIOI than controls during exploration (bins 1-3) and at the end of the training blocks (bins 11-12, $p < 0.05$, FDR-corrected). Anx2 participants did not differ from control participants. (C) Same as (B) but for the across-trials cvIOI, revealing a signifcant drop in task-related exploration at baseline in anx1 relative to control participants ($p < 0.05$, FDR-corrected). Bars around the mean show ±SEM.

Without Baseline Exploration, State Anxiety during Reward-based Learning Reduces Learning Rates.

Because participants in anx2 performed at a level not significantly different from that found in control participants, we asked whether the initial unconstrained motor exploration at baseline might have counteracted the effect of anxiety during training. To that aim, we performed a control behavioral experiment with new control and anx2 groups (N =13 each). Participants in each group performed the two

training blocks 2 and 3 (**Figure1**), but without completing a preceding baseline exploration block. In anx2, state anxiety was induced exclusively during the first training block, as in the original experiment. We found that HRV and within-trial temporal variability were significantly reduced in anx2 relative to controls during the manipulation phase ($p < 0.05$, FDR-corrected, **Figure 3 – figure supplement 2).** Moreover, anx2 participants achieved significantly lower scores than control participants during the first training block ($p < 0.05$, FDR-corrected), yet not during the second training block ($p > 0.05$). Importantly, however, overall anx2 participants achieved a lower average score (and monetary reward) than control participants ($p = 0.0256$; $PS_{sup} = 0.64$). The degree of across-trial temporal variability did not differ between both groups, yet in the control group – not in anx2 - there was a significant transition from an explorative to an exploitative regime (drop in across-trials cvIOI, $p = 0.0001$, $PS_{dep} = 1$), as expected.

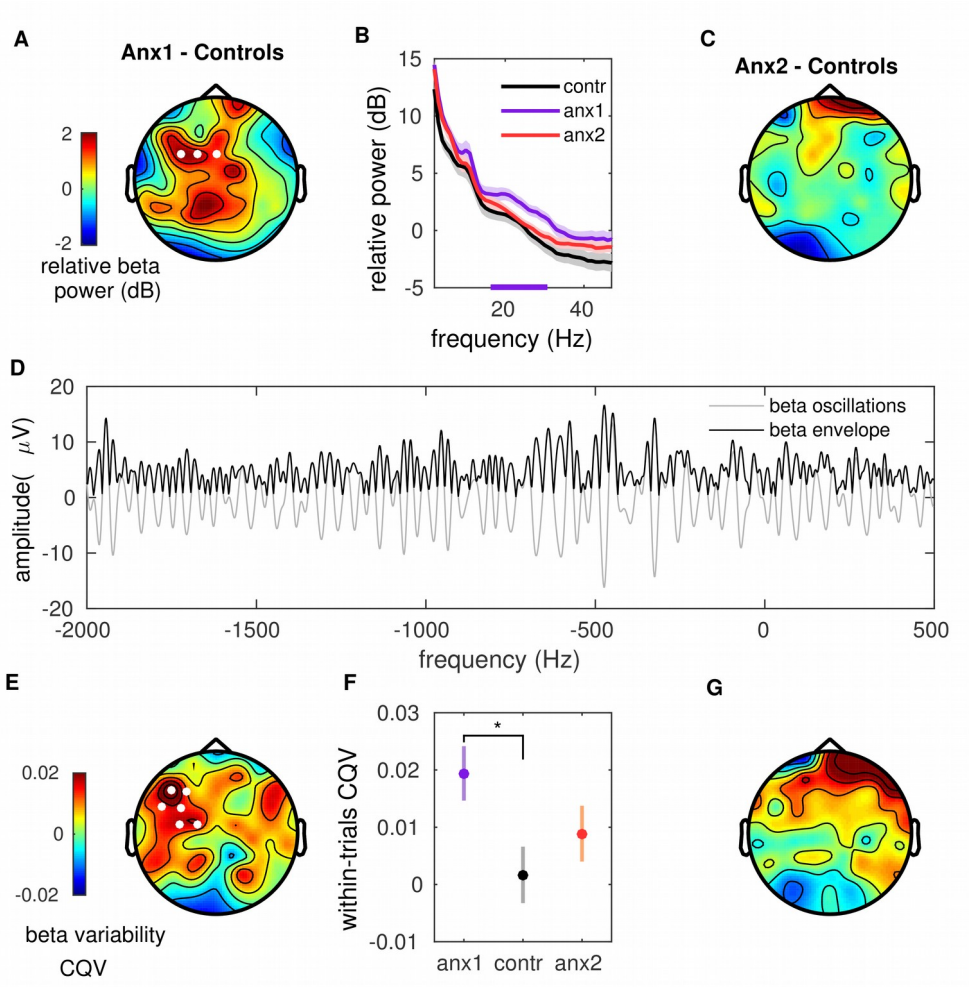

Figure 4. Sensorimotor beta activity during baseline exploration is modulated by anxiety. (A) Topographical representation of the between-group difference (anx1-controls) in normalized beta-band power spectral density (PSD) in dB. A larger beta-band PSD increase was found in anx1 relative to control participants in a small cluster of contralateral sensorimotor electrodes (white dots indicate significant electrodes, two-tailed cluster-based permutation test, $p < 0.025$, FWE-corrected). (B) Averaged PSD within 4-45Hz for each experimental and control group corresponding to the cluster shown in (A). Beta-band power differences were additionally assessed within the broader range 4-45Hz, revealing an effect exclusively within 17-30Hz ($p < 0.05$, FDR-corrected), denoted by the purple line at

the bottom). No significant effects outside the beta range were found. Anx2 and control participants did not differ in power modulations. Shaded areas denote mean ±SEM. (C) Same as (A) but for differences in beta-band PSD between anx2 and control participants. No significant clusters were found. (D) Illustration of the amplitude of beta oscillations (gray line) and amplitude envelope (black line) for one representative subject and channel. (E) Scalp topography for between-group differences in the coefficient of quartile variation (CQV) of the beta-band amplitude envelope, as a measure of beta-band amplitude variability. We obtained one significant cluster of left sensorimotor electrodes (white dots, $p < 0.025$, FWE-corrected), due to larger beta-band variability in anx1 than in control participants. (F) Beta-band CQV index averaged within the electrodes pertaining to the significant positive cluster shown in (E). Data shown as mean and ± SEM. Significant differences between anx1 and control groups are indicated by the asterisk. (G) Same as (E) but for beta-band CQV differences between anx2 and control participants. No significant differences were found.

Variability in Beta Oscillations at Baseline is Enhanced by State Anxiety

We assessed whether the changes in motor variability found during baseline exploration are associated with changes in sensorimotor beta-band oscillatory activity. Specifically, we tested whether within-trial variability in the amplitude envelope of beta oscillations is influenced by state anxiety at baseline in anx1 relative to control participants – using the coefficient of quartile variation (CQV[32]) as a measure of relative dispersion. In addition, between-group differences in the averaged normalized power spectral density (PSD) of beta oscillations were evaluated. Normalization of the raw PSD into decibels (dB) was carried out using as reference the average PSD from the initial rest recordings (3 min). Results on the effects of anxiety on the modulation of beta oscillations by feedback-locked reward processing will be reported elsewhere.

We found a significantly higher beta-band power in a reduced set of three channels in the contralateral sensorimotor region in anx1 relative to control participants at baseline ($p < 0.025$, two-sided cluster-based permutation test[33]; $PS_{sup} = 0.73$. **Figure 4A-B**). By contrast, in anx2 participants, the beta power was not significantly different than in controls (**Figure 4C**, $p > 0.05$). No significant between-group changes in PSD were found in lower (<13Hz) or higher (>30Hz) frequency ranges ($p > 0.05$). Crucially, in anx1, the CQV of beta oscillations was significantly higher than the values in the control group across an extended set of channels in the left sensorimotor region ($p < 0.025$, $PS_{sup}= 0.80$ **Figure 4D-E**). No difference in the CQV of beta oscillations was found between anx2 and control participants (**Figure 4F)**. Thus, the anxiety manipulation during baseline exploration led to a pronounced enhancement of within-trial beta variability in contralateral sensorimotor electrodes. This indicates a more irregular range of dynamic changes of beta amplitude. To a lesser degree, the anxiety manipulation at this phase also increased contralateral sensorimotor beta power, although in a more locally confined set of electrodes.

A similar analysis in the training period revealed no significant between-group beta power differences (**Figure 4 – figure supplement 1**). There was, however, significantly larger within-trial beta-band variability in contralateral sensorimotor electrodes in anx1 relative to control participants ($p < 0.025$). Accordingly, despite the targeted effect of the anxiety manipulation in the anx1 group, which led to changes in HRV exclusively in the baseline phase, the larger variability of beta oscillations found during baseline extended to the training period as well. Anx2 participants also exhibited larger beta-band CQV values relative to control participants, albeit in a region of frontal electrodes ($p < 0.025$).

State Anxiety during Exploration Prolongs Beta Bursts

To explore further the result of anxiety-related increases in within-trial variability in beta oscillations, we assessed the distribution and duration of beta bursts. To identify bursts of beta oscillations and assess the distribution of their duration, we applied an above-threshold detection method, which was adapted from previously described procedures[25,27](see **Figure5A** and *Materials and Methods*). Bursts extending for at least one cycle were selected. Using a double-logarithmic representation of the probability distribution of burst

durations, we obtained a power law and extracted the slope, τ, also termed "life-time" exponent[25]. Modelling work has revealed that a power law in the burst-duration distribution (slope τ = 1.5), reflecting that the oscillation bursts or neuronal avalanches have no characteristic scale, indicates that the underlying neural dynamics operate in a state close to criticality, and thus benefitial for information processing[25,34].

During baseline exploration, beta bursts lasted significantly longer in anx1 as compared to control participants (**Figure 5B**, $p < 0.025$, $PS_{sup} = 0.75$). This effect was most pronounced in a cluster of electrodes in the contralateral sensorimotor area, resembling the topography of the CQV effects (**Figure 4**). The mean burst duration in these electrodes was 147 (2) ms in control participants and 168 (10) ms in the anx1 group, with a difference of 20 ms corresponding with at least 2 cycles of 13Hz oscillations (5 cycles of 30Hz oscillations). A further between-group comparison focusing on the distribution of burst duration demonstrated that shorter bursts were significantly more frequent in control relative to anx1 participants (130-194ms, $p < 0.05$, FDR-corrected; PS_{sup}= 0.70; **Figure5CD**). By contrast, long bursts of 630-1130ms were more frequent in anx1 than control participants ($p < 0.05$, FDR-corrected, PS_{sup}= 0.92). The life-time exponents were smaller in anx1 than in the control group at left sensorimotor electrodes (1.43 [0.30]; 1.70 [0.15]; $p < 0.05$, FDR-corrected; PS_{sup}= 0.81). No differences in mean burst duration, life-time distribution, or exponents were found between anx2 and control participants. Regarding the distribution of beta bursts throughout the trial, the probability in all groups increased significantly at the completion of the trial-wise performance, as reported previously[28,30] ($p < 0.05$ in all groups, FDR-corrected; **Figure 6**). Interestingly, between-group comparisons demonstrated that, during sequence performance, the probability of oscillation bursts dropped in anx1 relative to control participants ($p < 0.05$, FDR-corrected), due to the smaller rate of brief bursts in this experimental group (**Figure 6 – figure supplement 1)**.

During training, the mean duration of bursts in anx2 or anx1 was not significantly different from values obtained in the control group (**Figure 5E**, $p > 0.05$). However, long bursts were more frequent in anx2 than in control participants (**Figure 5FG**, duration 630-930 and 1380-1680ms; $p < 0.05$, FDR-corrected, PS_{sup}= 0.71), supporting that each experimental group exhibited longer beta bursts relative to control participants during the blocks affected by the anxiety manipulation. Within-group comparisons further confirmed this outcome, demonstrating that the average burst duration was longer during baseline exploration than during training in anx1 across left sensorimotor electrodes ($p < 0.05$, FDR-corrected, PS_{sup} = 0.73) – despite anx1 also exhibiting significantly more frequent long bursts during training than controls (630-770ms, $p < 0.05$, FDR-corrected, PS_{sup} = 0.68). Also, the burst duration was significantly longer during the first block of the training phase than at baseline in anx2 ($p < 0.05$, FDR-corrected, PS_{sup} = 0.71). In control participants, the duration of beta bursts did not change across the experimental blocks ($p > 0.05$). Throughout the trial, the probability of beta bursts did not differ between groups; yet there was a significant within-group increase in burst probability from beginning to end of the trial in all channels and all groups ($p < 0.05$, FDR-corrected, **Figure 6**). Following the feedback presentation, the burst probability dropped significantly relative to the end of the trial in each group ($p < 0.05$, FDR-corrected) and similarly in all groups. The life-time exponent, τ, did not differ between groups ($p > 0.05$, around 1.6 on average in all groups).

Lastly, smaller slope values τ – corresponding with long-tailed distributions of burst duration due to the more frequent long bursts, as in anx1 – were associated with higher beta-band CQV across participants, and both during exploration and during training (Spearman $\rho = 0.496$, $p = 6 \times 10^{-4}$ for exploration, $\rho = 0.413$, $p = 0.0011$ for training; N = 60; **Figure 6 – figure supplement 2**).

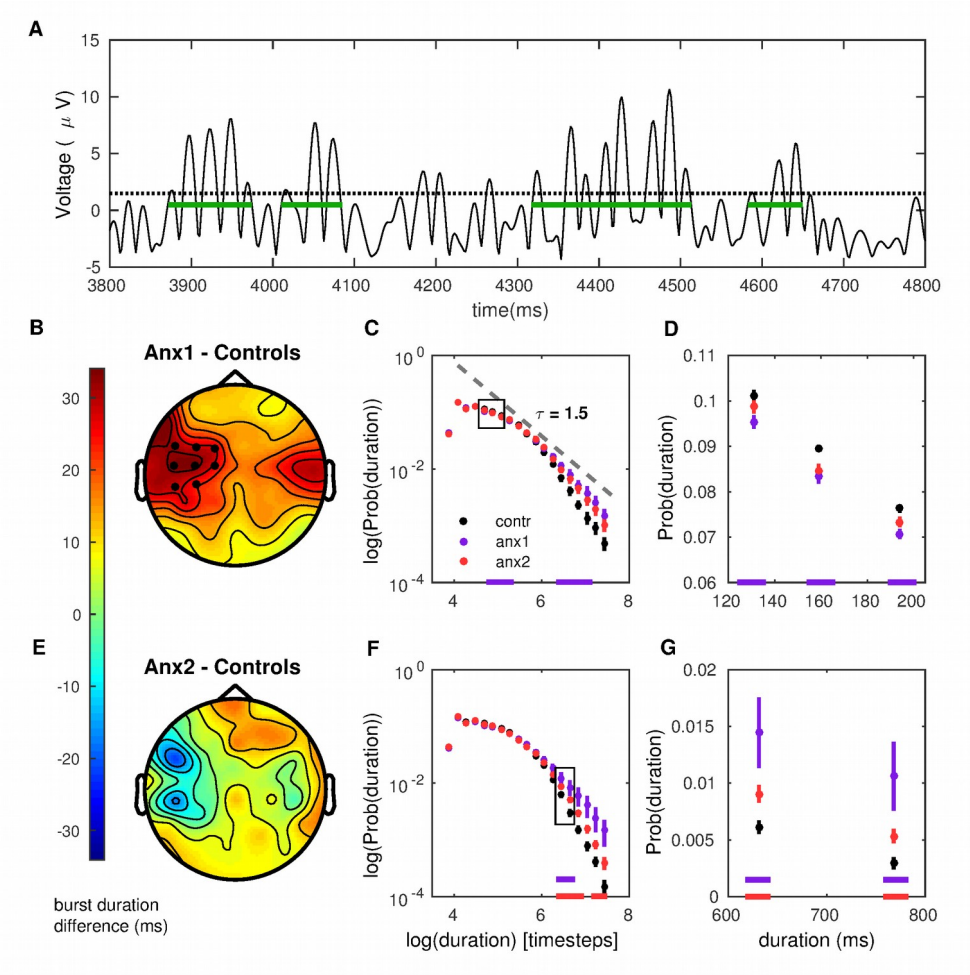

Figure 5. Anxiety during baseline exploration modulates the duration of sensorimotor beta-band oscillation bursts. (A) Illustration of the threshold-crossing procedure to detect beta oscillation bursts[25,27]. A threshold of 75% of the beta-band amplitude envelope was selected and beta bursts extending for at least one cycle were accepted. Windows of above-threshold oscillation bursts detected in the beta-band amplitude envelope (black line) are denoted by the green lines. (B) Scalp topography for between-group changes in the mean burst duration during baseline exploration. A significant positive cluster was found in an extended cluster of left sensorimotor electrodes, due to a longer average burst duration in anx1 than in control participants (20-30ms longer; Black dots indicate significant electrodes, two-tailed cluster-based permutation test, $p < 0.025$, FWE-corrected). (C) Probability distribution of beta-band oscillation-burst life-times within range 50-2000ms for each group during baseline exploration. The double-logarithmic representation reveals a power law within the fitted range (timesteps in logarithmic x-axis 4.09-7.62, corresponding to time windows 59.64 – 2053ms; first timestep excluded from the fit[25]). For each power law we extracted the slope, τ, also termed life-time exponent. The dashed line illustrates a power law with $\tau = 1.5$. Significant differences between anx1 and control participants in oscillation-burst durations are denoted by the purple line at the bottom ($p < 0.05$, FDR-corrected). The rectangle highlights the area enlarged and displayed in the right panel (D). Data shown as mean and ± SEM. (E) Same as (B) but for differences in mean burst duration between anx2 and control groups during training. No significant differences were found. (F) Same as (C) but during training. Significant between-group differences were found for long-lived oscillation bursts within 630-930 and 1380-1680ms (anx2-controls, red bar at the bottom; $p < 0.05$, FDR-corrected) and 630-770ms (anx1-controls, purple bar at the bottom). (G) Enlarged display of the region of between-group significant differences highlighted by the rectangle in (F).

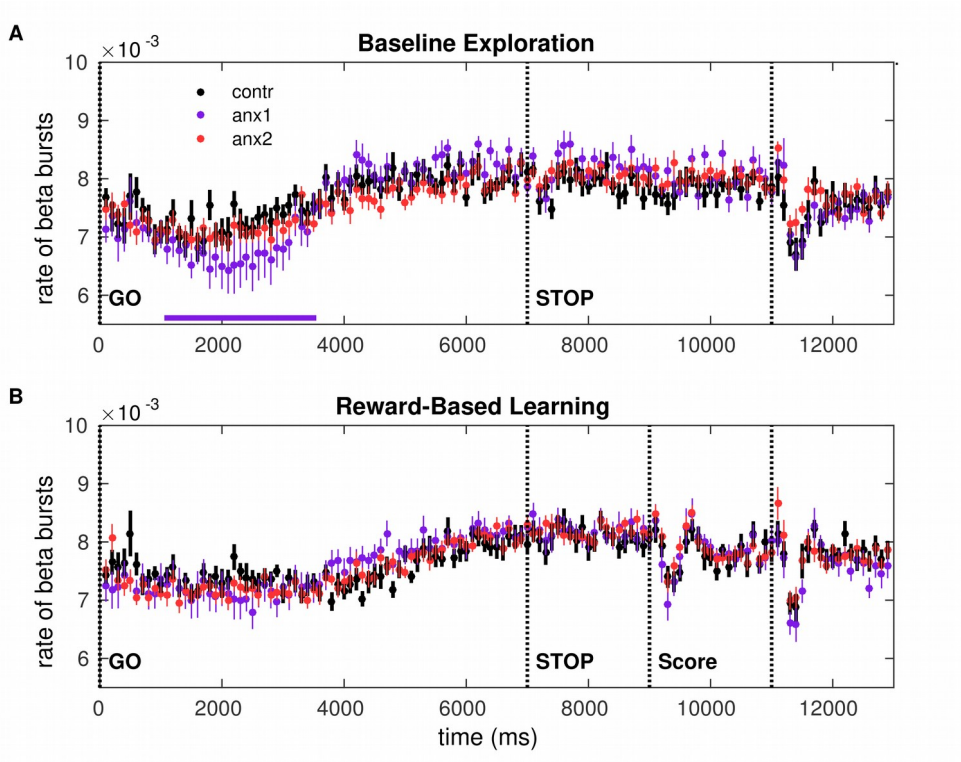

Figure 6. Time course of the rate of beta-band oscillation bursts throughout trial performance. (A) Rate of beta bursts during sequence performance in the baseline exploration phase. Participants completed sequence1 on average between 600 (SEM 100) and 3600 (100) ms (non-significant differences between groups, p> 0.05). The STOP signal (red ellipse on the monitor) was displayed 7000 ms after the GO signal. At 11000 ms the trial ended (the red ellipse vanished). In all groups there was a significant increase in the rate of oscillation-bursts duration following completion of the sequence performance (0-3500ms versus 3500 – 7000s trial segments, p < 0.05, FDR-corrected). In addition, between-group comparisons demonstrated a significant drop in the burst rate in anx1 participants relative to control participants during sequence performance (1100-3500 ms, denoned by the purple bar at the bottom; p < 0.05, FDR-corrected). Data display the mean and ± SEM. (B) Same as (A) but for the training period, when participants played sequence2. At 9000 ms, 2000 ms after the STOP signal, the feedback score was displayed for 2000 ms. There was a within-group significant increase in burst rate following completion of the sequence performance (0-3500ms versus 3500 – 7000s trial segments, p < 0.05, FDR-corrected) and a subsequent significant drop following feedback presentation (p < 0.05, FDR-corrected). No significant between-group effects were found.

Discussion

Our findings expand previous computational modelling and experimental work that linked anxiety levels (trait) and poorer performance, albeit in aversive environments[2-3,35]. The results demonstrate that state anxiety impaired motor variability and exploration at baseline, decreasing performance in a subsequent reward-based learning phase. Participants with larger task-related variability and exploration at baseline scored higher during the following training phase, extending recent findings on the faciliatory effect of exploration on motor learning[10,36]. Crucially, combining evidence from both experiments, we were able to show that reward-based learning is not affected by concurrent state anxiety if participants were given the opportunity of unlimited exploration during a preceding baseline phase. On the neural level, state anxiety during baseline exploration increased variability of beta oscillations in the contralateral sensorimotor

cortex; and to a lesser degree, also enhanced average beta power. Finally, bursts of sensorimotor beta oscillations, a marker of physiological beta, lasted longer under the effect of anxiety, resembling recent findings of abnormal burst duration in movement disorders.

These results thus provide the first evidence for changes in variability and burst duration of sensorimotor beta oscillations mediating the effects of anxiety on motor exploration, with negative consequences for reward-based motor learning.

Anxiety constrains motor variability and exploration

Previous studies manipulating psychological stress and anxiety to assess motor learning showed both a deleterious and faciliatory effect[37-38]. Differences in experimental tasks, which often assess motor learning during or after high-stress situations but not during anxiety induction in anticipation of a stressor, could account for the previous mixed results. Here, we adhere to the neurobiological definition of anxiety as a psychological and physiological response to an upcoming diffuse and unpredictable threat[1,2]. Accordingly, anxiety was induced using the threat of an upcoming public speaking task reliably shown to lead to anticipatory changes in heart-rate and perceived anxiety[14,29.]. The analysis of HRV confirmed that the experimental manipulation succeeded in modulating activity in the autonomic nervous system in association with the anxiety induction during the targeted blocks. Behaviorally, state anxiety at baseline reduced task-related variability within the trial but also exploration across trials. This converges with recent evidence demonstrating that anxiety leads to ritualistic behavior (repetition, redundancy, rigidity of movements) to regain a sense of control[14]. Crucially, however, anx1 participants continued to exhibit a limited use of temporal variability and exploration during subsequent non-anxiety-related training – despite this phase requiring an unrelated piano sequence performance and the HRV returning to normal levels. Moreover, they achieved lower scores and an overall smaller monetary reward. By contrast, participants in the control and anx2 groups who freely explored the temporal dimension during baseline achieved higher scores during training. Our results thus extend previous work[10,36] on the beneficial effect of motor variability on motor learning to the context of anxiety. In particular, the data support that mechanistically the anxiety-induced reduction in behavioral exploration impairs performance in successive tasks that depend on exploration for learning.

Significantly, the control experiment demonstrated that removal of baseline motor exploration leads to anxiety diminishing reward-based learning, establishing the relevance of unconstrained exploration for successful motor learning. Our results thus have implications for research on anxiety disorders and performance anxiety, by supporting that intervention programs exploring movements during a non-anxious phase could preserve subsequent motor learning when anxiety re-emerges.

We accounted for two sources of temporal variability. Within-trial variability was directly linked to the computation of feedback scores during training. Across-trials variability was higher in participants exploring different performance options in successive trials. Operationally, however, higher levels of across-trials variability could reflect both an intentional pursuit of an explorative regime; or, an unintentional higher level of motor noise. Similarly, motor variability in previous studies reflected contributions from motor noise and intentional exploration, and it is possible that both sources of variability could be beneficial for reward-based learning[10-11]. A recent study, however, established that motor learning (and decision-making) is improved by the use of intended exploration, not motor noise[13]. Although our paradigm cannot dissociate between intended and unintended exploration, the successful transition from an explorative to an exploitative regime in anx2 and control participants from baseline to training blocks, and further during the training blocks, shows they were capable of context-dependent modulation of task-related variability. This outcome aligns well with animal studies where evidence shows a reduction in motor exploration when

stakes are high (high-reward situations, social context)[36,39-40]. Furthermore, the transition was paralleled by an increase in within-trial task-related variability to achieve higher scores, demonstrating that separately controlling within-trial and across-trials variability was possible and necessary for success. The results are consistent with computational approaches to motor control emphasizing that during task performance, some variables are controlled by the central nervous system, whereas others are left unconstrained[9, 41].

Variability and burst duration of beta oscillations mediate the effects of anxiety on behavior.

An important finding was that anxiety at baseline increased variability in the amplitude envelope of beta oscillations during performance. This increase was observed in a region of contralateral sensorimotor channels, supporting that in humans changes in sensorimotor beta variability by anxiety track the changes in motor variability and exploration. Although EEG does not allow for a detailed anatomical localization of the effect, the finding is consistent with the involvement of premotor and motor cortex in driving motor variability and learning, as previously reported in animal studies[16-18], as well as with the changes in motor cortical excitability found in anxious individuals in clinical settings[42]. Moreover, the data suggest that an excessive degree of variation in the amplitude of sensorimotor beta oscillations might be detrimental for performance.

The observed anxiety-related changes in beta variability at baseline and during training were correlated with the life-time exponents of the distribution of oscillation bursts across contralateral sensorimotor channels. These correlation results indicate that a tendency towards more frequent long bursts was associated with more variable amplitude of beta oscillations during trial performance. A similar association has been recently observed in work comparing beta oscillation properties in real and shuffled data[28]. Our data demonstrate for the first time a context-dependent anxiety-related modulation of the burst distribution of cortical sensorimotor beta oscillations. Although bursts of 50-100ms were the most frequent in all experimental groups, the most pronounced presence of long bursts was found in anx1 during exploration, and partially also during training. The outcomes thus tentatively link the more frequent presence of long-lived oscillation bursts in sensorimotor regions to reduced motor exploration and learning.

Brief bursts of alpha and beta oscillations extending from one to several cycles have been linked to the normal physiological state during rest and motor performance, respectively[25-26]. In the case of alpha oscillations at rest, it has been suggested that bursts represent neuronal avalanches propagating in neural networks operating near a critical state[25]. The life-time exponents reported for sensorimotor alpha oscillations lies within 1.5-1.99[25], in line with the values of the beta-band oscillation-burst distribution we obtained, 1.4-1.9. This range of exponents is consistent with neural dynamics operating in a state close to criticality[25], which would be beneficial for information processing as it supports a balance between flexibility and stability[34]. A link between beta-band oscillation bursts and information processing has also been proposed in recent studies, which showed that the timing and distribution of beta bursts influence motor processing on a trial-by-trial basis[26,28]. These brief bursts of beta oscillations emerge most prominently in the pre- and post-movement period[26,28], which converges with the time course of burst probability in our study. Alternative hypotheses posit that beta bursts contribute to inhibitory processes[43], in line with the suggested anti-kinetic role of beta oscillations[44]. This interpretation would apply to the power effects in our study, as anxiety at baseline increased the average beta power, which could have limited the expression of motor variability in anx1 participants.

Interestingly, during baseline the exponents in contralateral sensorimotor electrodes dropped in anx1 relative to control participants, corresponding with the long-tailed distribution of burst duration in this experimental group. This finding at the cortical level converges with recent data from the basal ganglia in patients with Parkinson's disease, showing that beta bursts last longer in association with more severe

motor symptoms[27]. The link is also interesting considering the evidence for a role of basal ganglia variability driving movement variability[19-20]. Previous songbird studies demonstrated that contextual cues such as the presence of a partner alter the expression of neuronal variability during singing via modulation of dopamine release[39]. In Parkinson's disease, a condition characterized by a loss of dopaminergic cells in the substantia nigra, reward-based modulation of movement variability is limited[11]. Our data thus imply that corresponding changes in the duration of beta-band oscillation bursts in basal ganglia structures could be driving the cortical effects, thereby shaping the use of movement variability. Future work, combining recordings in the human basal ganglia and cortex, should test this prediction.

In conclusion, this study provides the first evidence that contextual modulation of beta bursts and variability by anxiety biases motor behavior, leading to changes in motor variability and exploration, with consequences for motor learning.

Materials and Methods

Participants and sample size estimation

In the main experiment, 60 right-handed healthy volunteers (37 females) aged 18 to 44 (mean 27 years, standard error of the mean, SEM, 1) participated in this study. In the second, control experiment, 26 right-handed healthy participants (16 females, mean age: 25.8, SEM 1, range 19-40) took part in the study. Sample size estimation can be found in S.I. Materials and Methods. Participants gave written informed consent prior to the start of the experiment, which had been approved by the local Ethics Committee at Goldsmiths University. Participants received a base rate of either course credits or money (£15) (equally distributed across groups) and were able to earn an additional sum up to £20 during the task depending on their performance.

We used pilot data from a behavioral study using the same experimental paradigm (data not shown) to estimate the minimum sample sizes for a statistical power of 0.95, with an α of 0.05, using the MATLAB (The MathWorks, Inc., MA, USA) function sampsizepwr. In the pilot study we had one control and one experimental group of 20 participants each. In the experimental group we manipulated the reward structure during the first training block (in this block feedback scores did not count towards the final average monetary reward). For each behavioral measure (within-trial cvIOI and mean score), we extracted the standard deviation (sd) of the joint distribution from both groups and the mean value of each separate distribution (e.g. m1: control, m2: experimental), which provided the following minimum sample sizes:

Between-group comparison of within-trial cvIOI and mean score parameters (using 2-tailed t-test): MinSamplSizeA = sampsizepwr('t',[m1 sd],m2, 0.95) = 18-20 participants.

Accordingly, we recruited 20 participants for each group in the main experiment. Next, using the behavioral data from the anx1 and control groups in this main experiment (as we found large non-parametric effect sizes in the anx1-control comparison, PS_{sup} in range 0.7-0.8), we estimated the minimum sample size for the second, control experiment:

Between-group (anx1-controls) comparison of within-trial cvIOI and mean score parameters (using 2-tailed t-test): MinSamplSizeA = sampsizepwr('t',[m1 sd],m2, 0.95) = 13 participants.

Therefore for the second control experiment we recruited 13 participants in each group.

Apparatus and Materials

Participants were seated at a digital piano (Yamaha Digital Piano P-255, London, United Kingdom) and in

front of a PC monitor in a light-dimmed room. They sat comfortably in an arm-chair with their forearms resting on the armrests of the chair. The screen displayed the instructions, feedback and visual cues for start and end of a trial. Participants were asked to place four fingers of their right hand (excluding the thumb) comfortably on 4 pre-defined keys on the keyboard. Performance information was transmitted and saved as Musical Instrument Digital Interface (MIDI) data, which provided time onsets of keystrokes relative to the previous one (inter-onset-interval – IOI in ms), MIDI velocities (related to the loudness, in arbitrary units, a.u.), and MIDI note numbers that corresponded to the pitch. The experiment was run using Visual Basic and additional parallel port and MIDI libraries.

The sequence patterns for the baseline exploration and training blocks were designed so that the key presses would span a range of four neighbouring keys on the piano (**Figure 1A**).

Experimental design

In all blocks, participants initiated the trial by pressing a pre-defined key with their left index finger. After a jittered interval of 1-2 s, a green ellipse appeared in the centre of the screen representing the "go" signal for task execution. Participants had 7 s to perform the sequence which was ample time to complete it before the green circle turned red indicating the end of the execution time. If participants failed to perform the sequence in the correct order or initiated the sequence before the "go" signal, the screen turned yellow (**Figure 1B**). In blocks 2 and 3 during training, performance-based feedback in form of a score between 0 and 100 was displayed on the screen 2 s after the red ellipse, that is, 9 s from the beginning of the trial. The performance-based feedback (scores) provided participants with information regarding the target performance. The pattern of inter-onset-intervals (IOIs) was used to assess the timing or rhythm of the performance, whereas the MIDI keystroke velocity was used to quantify the dynamics (changes in loudness).

The target rhythm consisted of a pattern of alternating short and long IOIs: $t \equiv [0.2, 1, 0.2, 1, 0.2, 1, 0.2]$ s (7 IOIs for an 8 notes-long sequence). The score was computed using a measure of proximity between the pattern of IOIs performed in each trial (p) and the rewarded rhythm. Specifically, we computed the norm of the differences between adjacent IOI values (MATLAB function *diff*) for the performed pattern $normDp = \|diff(p)\|$ and, separately, for the target pattern $normDt = \|diff(t)\|$. Next, the score was calculated using this expression:

$$score = 100\, e^{-|normDp - normDt|}$$

In practice, different rhythm patterns could achieve the same reward, as any pattern of IOIs leading to the same $normDp$ value obtained identical scores. The scores correlated with the difference between the cvIOI of the performed and target patterns (ρ = 0.53, p < 0.0001), and accordingly, same values of cvIOI led to identical scores. Participants were unaware of the existence of various solutions and their performance demonstrated that they approached a single-solution. The rationale for accepting different timing patterns as maximally rewarded solutions was our aim to enable a steady learning rate in all participants, by diminishing the difficulty that would be associated with requiring one single solution.

Anxiety Manipulation

Anxiety was induced during block1 performance in group anx1, and during block2 performance in the anx2 group by informing participants about the need to give a 2-minute speech to a panel of experts about an unknown art object at the end of that block[14]. We specified that they would first see the object at the end of the block (it was a copy of Wassily Kandinsky' Reciprocal Accords [1942]) and would have 2 min to prepare for the presentation. Participants were told that the panel of experts would take notes during their speech and would be standing in front of the testing room (due to the EEG setup participants had to remain

seated in front of the piano). Following the 2-min preparation period, participants were informed that due to the momentary absence of panel members they instead had to present in front of the lab members. Participants in the control group had the task to describe the artistic object to themselves, not in front of a panel of experts. They were informed about this secondary task at the beginning of the exploration phase.

Assessment of State Anxiety

To assess state anxiety we acquired two types of data: (1) the short version of the Spielberger State-Trait Anxiety Inventory (STAI, state scale X1, 20 items)[45] and (2) a continuous electrocardiogram (ECG, see EEG and ECG recording session). The STAI X1 subscale was presented four times throughout the experiment. A baseline assessment before the start of the experiment before the resting state recording was then followed by an assessment immediately before each experimental block to determine changes in anxiety levels. In addition, we a continuous ECG recording was obtained during the resting state and experimental blocks to assess changes in autonomic nervous system responses. The indexes of heart rate variability (HRV, coefficient of variation of the inter-beat-interval) and mean heart rate (HR) were evaluated, as their reduction has been linked to changes in anxiety state due to a stressor[29].

EEG, ECG and MIDI recording

EEG and ECG signals were recorded using a 64-channel (extended international 10–20 system) EEG system (ActiveTwo, BioSemi Inc.) placed in an electromagnetically shielded room. During the recording, the data were high-pass filtered at 0.16 Hz. The vertical and horizontal eye-movements (EOG) were monitored by electrodes above and below the right eye and from the outer canthi of both eyes, respectively. Additional external electrodes were placed on both left and right earlobes as reference. The ECG was recorded using two external channels with a bipolar ECG lead II configuration. The sampling frequency was 512 Hz. Onsets of visual stimuli, key presses and metronome beats were automatically documented with markers in the EEG file. The performance was additionally recorded as MIDI files using the software Visual Basic and a standard MIDI sequencer program on a Windows Computer.

EEG and ECG pre-processing

We used MATLAB and the FieldTrip toolbox[46] for visualization, filtering and independent component analysis (ICA; runica). The EEG data were highpass-filtered at 0.5 Hz (Hamming windowed sinc finite impulse response [FIR] filter, 3380 points) and notch-filtered at 50 Hz (847 points). Artifact components in the EEG data related to related to eye blinks, eye movements and the cardiac-field artifact were identified using ICA. Following IC inspection, we used the EEGLAB toolbox[47] to interpolate missing or noisy channels using spherical interpolation. Finally, we transformed the data into common average reference.

Analysis of the ECG data focused on detection of the QRS-complex to extract the R-peak latencies of each heartbeat and use them to evaluate the HRV and HR measures in each experimental block.

Analysis of power spectral density and variability of oscillations

We first assessed the standard power spectral density (PSD, in mV^2/Hz) of the continuous raw data in each performance block and separately for each group. The PSD was computed with the standard fast Fourier Transform (Welch method, Hanning window of 1s with 50% overlap). The raw PSD estimation was normalised into decibels (dB) with the average PSD from the initial rest recordings (3 min). Specifically, the log normalized PSD during the performance blocks was calculated as the natural logarithm of the quotient between the performance-block PSD and the resting state power.

In addition, variability of cortical beta-band (13-30Hz) activity in each performance block was assessed using

the coefficient of quartile variation (CQV[32,48]). The CQV is a descriptive statistic based on the first (lower) and third (higher) quartile of the data:

$$\text{CQV} = \frac{Q_3 - Q_1}{Q_3 + Q_1}$$

The difference Q_3-Q_1, termed interquartile range, is a measure of the dispersion of the data when ranked. To measure the CQV of beta oscillations, the amplitude envelope of the instantaneous analytic signal was computed after applying the Hilbert transform to the bandpass-filtered raw data (12–35 Hz; Hamming windowed two-way least-squares FIR filter applied with the eegfilt.m routine from the EEGLAB toolbox. See **Figure 4D**) spanning the full continuous recording in each performance block. Next, from the total beta-band amplitude envelope we selected data epochs of 10s corresponding to each performance trial (trial of 7 s, post-performance period of 3 s). This step provided 100 epochs during baseline exploration and 200 epochs during training. The beta-band CQV index was computed for each of these single-trials, and was then averaged across trials within each block.

Extraction of beta-band oscillation bursts

The time series of beta-band amplitude envelope obtained in the CQV analyses were used to detect oscillation bursts. We followed a procedure adapted from previous work on oscillation bursts[25,27]. In brief, we used as threshold the 75% percentile of the amplitude envelope of beta oscillations (after rectification)[27]. Amplitude values above this threshold were considered to be part of an oscillation burst if they extended for at least one cycle (50ms: as a compromise between the duration of one 13 Hz-cycle [76 ms] and 30 Hz-cycle [33 ms]). Threshold-crossings that were separated by less than 25 ms were considered to be part of the same oscillation burst. As an additional threshold the median amplitude was used in a control analysis, which revealed similar results (significantly more frequent short bursts in control relative to anx1 participants but less frequent long bursts, $p < 0.05$, FDR-corrected), as expected from previous work[25]. Importantly, because threshold crossings are affected by the signal-to-noise ratio in the recording, which could vary between the baseline and training blocks, we selected a common threshold across all experimental blocks separately for each participant[27].

Distributions of the rate of oscillation bursts per duration were estimated using equidistant binning on a logarithmic axis with 20 bins between 50-2000 ms. In all participants the double-logarithmic representation of the distributions of burst duration followed a decaying power-law with slope values, τ, in the range 1.4-1.9, in agreement with previously reported values for sensorimotor alpha bursts[25].

Statistical Analysis

Between-group comparison focused on each experimental group, separately, and the control group (contrasts: anx1 – control, anx2 – control). Differences between experimental groups anx1-anx2 were evaluated exclusively concerning the overall achieved monetary reward. When appropriate, we tested main effects and interactions for factors Group (anx, control) and Phase (baseline, training) using a 2x2 synchronized permutations test[49]. This analysis was complemented with non-parametric permutation tests to assess differences between conditions or between groups in the statistical analysis of behavioral or neural measures. To evaluate differences between sets of multivariate EEG signals corresponding to two conditions or groups, we used two-sided cluster-based permutation tests[33] and an alpha level of 0.025. Control of the family-wise error rate was implemented in these tests to account for the problem of multiple comparisons[33]. When multiple testing was performed with permutation tests and synchronized permutations, the FDR was controlled at level $q = 0.05$[30].

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References.

1. Grupe DW, Nitschke JB (2013) Uncertainty and anticipation in anxiety: an integrated neurobiological and psychological perspective. Nat Rev Neurosci 14:488–501.
2. Bishop SJ (2007) Neurocognitive mechanisms of anxiety: an integrative account. Trends Cogn Sci 11(7):307–316.
3. Bishop SJ (2009) Trait anxiety and impoverished prefrontal control of attention. Nat Neurosci 12(1):92–98.
4. Baumeister RF (1984) Choking under pressure: self-consciousness and paradoxical effects of incentives on skillful performance. J Pers Soc Psychol 46(3):610-620.
5. Beilock SL, Carr TH (2001) On the fragility of skilled performance: what governs choking under pressure? J Exp Psychol Gen 130: 701–725, 2001.
6. Eysenck MW, Calvo MG (1992) Anxiety and Performance: The Processing Efficiency Theory. Cogn Emot 6: 409-434.
7. van Beers R, Haggard P, Wolpert D (2004) The role of execution noise in movement variability. J Neurophysiol 91: 1050-1063.
8. He K et al (2016) The Statistical Determinants of the Speed of Motor Learning. PLoS Comput Biol 12(9):e1005023.
9. Todorov E, Jordan MI (2002) Optimal feedback control as a theory of motor coordination. Nat Neurosci 5: 1226–1235.
10. Wu HG et al (2014) Temporal structure of motor vriability is dynamically regulated and predicts motor learning ability. Nat Neurosci 17(2):312–321.
11. Pekny SE, Izawa J and Shadmehr R (2015) Reward-dependent modulation of movement variability. Journal of Neuroscience, 35(9): 4015-4024.
12. Sutton RS, Barto AG (1998) Reinforcement Learning: An Introduction: MIT press.
13. Chen X, Mohr K, Galea JM (2017) Predicting explorative motor learning using decision-making and motor noise. PLoS Comput Biol 13(4):e1005503.
14. Lang M, Krátký J, Shaver JH, Jerotijević D, Xygalatas D (2015) Effects of Anxiety on Spontaneous Ritualized Behavior. Curr Biol 25(14):1892–1897.
15. Morgan KN and Tromborg CT (2007). Sources of stress in captivity. Applied animal behavior science, 102(3-4): 262-302.
16. Churchland MM, Afshar A, Shenoy KV (2006) A Central Source of Movement Variability. Neuron 52(6):1085–1096.
17. Mandelblat-Cerf Y, Paz R, Vaadia E (2009) Trial-to-trial variability of single cells in motor cortices is dynamically modified during visuomotor adaptation. J Neurosci 29:15053–15062.
18. Santos FJ, Oliveira RF, Jin X, Costa RM (2015) Corticostriatal dynamics encode the refinement of specific behavioral variability during skill learning. eLife 4. doi:10.7554/eLife.09423.
19. Kao MH, Doupe AJ, Brainard MS (2005) Contributions of an avian basal ganglia–forebrain circuit to real-time modulation of song. Nature 433(7026):638–643.
20. Ölveczky BP, Andalman AS, Fee MS (2005) Vocal Experimentation in the Juvenile Songbird Requires a Basal Ganglia Circuit. PLoS Biol 3(5):e153.

21. Herrojo Ruiz M et al (2014) Beta-band amplitude oscillations in the human internal globus pallidus support the encoding of sequence boundaries during initial sensorimotor sequence learning. Neuroimage, 85: 779-793.
22. Bartolo R, Merchant H (2015) β oscillations are linked to the initiation of sensory-cued movement sequences and the internal guidance of regular tapping in the monkey. J Neurosc 35(11):4635-40.
23. Tan H, Jenkinson N, Brown P (2014) Dynamic neural correlates of motor error monitoring and adaptation during trial-to-trial learning. J Neurosci 34(16):5678–5688.
24. HajiHosseini A, Rodríguez-Fornells A, Marco-Pallarés J (2012) The role of beta-gamma oscillations in unexpected rewards processing. Neuroimage 60(3):1678–1685.
25. Poil SS, van Ooyen A and Linkenkaer-Hansen K (2008). Avalanche dynamics of human brain oscillations: relation to critical branching processes and temporal correlations. Human brain mapping, 29(7): 770-777.
26. Feingold J, Gibson DJ, DePasquale B, Graybiel AM (2015) Bursts of beta oscillation differentiate postperformance activity in the striatum and motor cortex of monkeys performing movement tasks. Proc Natl Acad Sci 112(44):13687–13692.
27. Tinkhauser G, et al. (2017) Beta burst dynamics in Parkinson's disease OFF and ON dopaminergic medication. Brain 140(11):2968–2981.
28. Little S , Bonaiuto J, Barnes G and Bestmann S (2018) Motor cortical beta transients delay movement initiation and track errors. *bioRxiv*: 384370. Preprint, posted August 9, 2018.
29. Feldman PJ, Cohen S, Hamrick N, Lepore SJ (2004) Psychological stress, appraisal, emotion and cardiovascular response in a public speaking task. Psychology & Health 19(3):353-68.
30. Benjamini Y, Krieger AM and Yekutieli D (2006). Adaptive linear step-up procedures that control the false discovery rate. *Biometrika*, *93*(3), pp.491-507.
31. Grissom RJ, Kim JJ (2012): Effect Sizes for Research: Univariate and Multivariate Applications, 2nd ed. New York: Taylor & Francis.
32. Iscan Z, Nazarova M, Fedele T, Blagovechtchenski E, Nikulin VV (2016) Pre-stimulus alpha oscillations and inter-subject variability of motor evoked potentials in single-and paired-pulse TMS paradigms. Frontiers in human neuroscience 10:504.
33. Maris E, Oostenveld R (2007): Nonparametric statistical testing of EEG-and MEG-data. J Neurosci Methods 164:177–190.
34. Chialvo DR (2010) Emergent complex neural dynamics. Nature physics 6(10):744.
35. Browning M, Behrens TE, Jocham G, O'reilly JX, Bishop SJ (2015) Anxious individuals have difficulty learning the causal statistics of aversive environments. Nat Neurosc 18(4):590.
36. Dhawale AK, Smith MA, Ölveczky BP (2017) The role of variability in motor learning. Annu Annu Rev Neurosci. 40:479-98.
37. Hordacre B, Immink MA, Ridding MC, Hillier S (2016) Perceptual-motor learning benefits from increased stress and anxiety. Hum Mov Sci 49:36–46.
38. Vine SJ et al (2013) Evaluating stress as a challenge is associated with superior attentional control and motor skill performance. J Exp Psychol Appl 19(3): 185-194.
39. Woolley SC, Rajan R, Joshua M, Doupe AJ (2014) Emergence of context-dependent variability across a basal ganglia network. Neuron 82(1):208-23.
40. Kao MH, Wright BD, Doupe AJ (2008) Neurons in a forebrain nucleus required for vocal plasticity rapidly switch between precise firing and variable bursting depending on social context. J. Neurosci 28:13232–13247.
41. Scholz JP, Schöner G (1999) The uncontrolled manifold concept: identifying control variables for a

functional task. Exp Brain Res 126:289–306.
42. Wassermann EM, Greenberg BD, Nguyen MB, Murphy DL (2001) Motor cortex excitability correlates with an anxiety-related personality trait. Biol Psychiatr 50(5):377-82.
43. Shin H, Law R, Tsutsui S, Moore CI, and Jones SR (2017) The rate of transient beta frequency events predicts behavior across tasks and species. Elife 6: e29086.
44. Engel AK, Fries P (2010) Beta-band oscillations—signalling the status quo?Curr Opin Neurobio 20(2):156-65.
45. Spielberger CD (1970) STAI manual for the state-trait anxiety inventory. Self-Evaluation Questionnaire 1-24.
46. Oostenveld R, Fries P, Maris E, Schoffelen JM. FieldTrip: open source software for advanced analysis of MEG, EEG, and invasive electrophysiological data (2011) Comput Intell Neurosci 2011:156869.
47. Delorme A, Makeig S. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis (2004) J Neurosci Methods 34(1):9-21.
48. Bonett DG (2006) Confidence interval for a coefficient of quartile variation. Comput Stat. Data Anal 50: 2953–2957.
49. Basso D, Chiarandini M and Salmaso L (2007) Synchronized permutation tests in replicated I× J designs. Journal of Statistical Planning and Inference 137(8): 2564-2578.

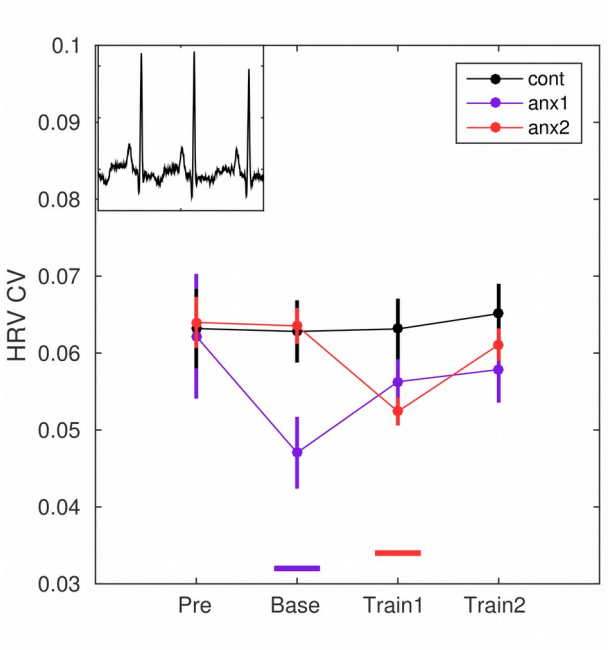

Figure 1 – figure supplement 1. Heart-rate varibility (HRV) modulation by the anxiety manipulation. Average HRV measured as the coefficient of variation of the inter-beat-interval is displayed across the experimental blocks: initial resting state recording (Pre), baseline exploration (Base), first block of training (Train1), last block of training (Train2). Relative to Pre, there was a significant drop in HRV in anx1 participants during baseline exploration ($p < 0.05$, FDR-corrected, $PS_{dep} = 0.81$). In anx2 participants the drop in HRV was found during the first training phase, which was affected by the anxiety manipulation ($p < 0.05$, FDR-corrected, $PS_{dep} = 0.78$). In addition, relative to the control group, anx1 demonstrated a significantly lower HRV at baseline ($p < 0.05$, FDR-corrected, $PS_{dep} = 0.75$). The second experimental group, anx2, exhibited a significant drop in HRV relative to controls during the first training block ($p < 0.05$, FDR-corrected, $PS_{dep} = 0.71$). These results demonstrate a group-specific modulation of anxiety relative to controls during the targeted blocks. No changes in mean heart-rate were found ($P > 0.05$). Neither was the STAI state anxiety subscale able to dissociate between the different phases in each group or between-groups ($p > 0.05$; mean values within 29-37 in all groups and experimental blocks). This is likely due to the habituation of the participants to the questionnaire.

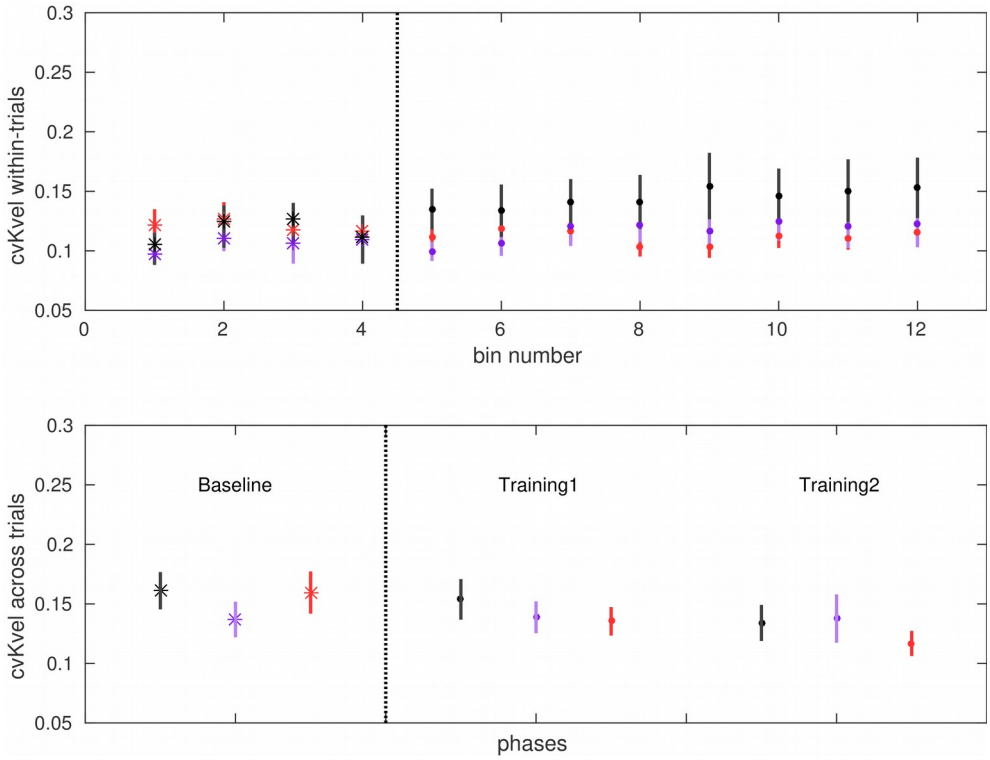

Figure 3 – figure supplement 1. Non-Task related variability in keystroke velocity. Top: Within-trial variability in Kvel across the experimental blocks (trials were split into bins of 25 trials and values were averaged within each bin). No significant between-group differences were found ($p > 0.05$). Bars around the mean display ±SEM. Data for control participants are shown in black, wherease data in purple / red indicate values in the anx1 and anx2 groups, respectively. Bottom: Same as in the upper panel but for the across-trials cvKvel. No significant between-group differences were found either ($p > 0.05$).

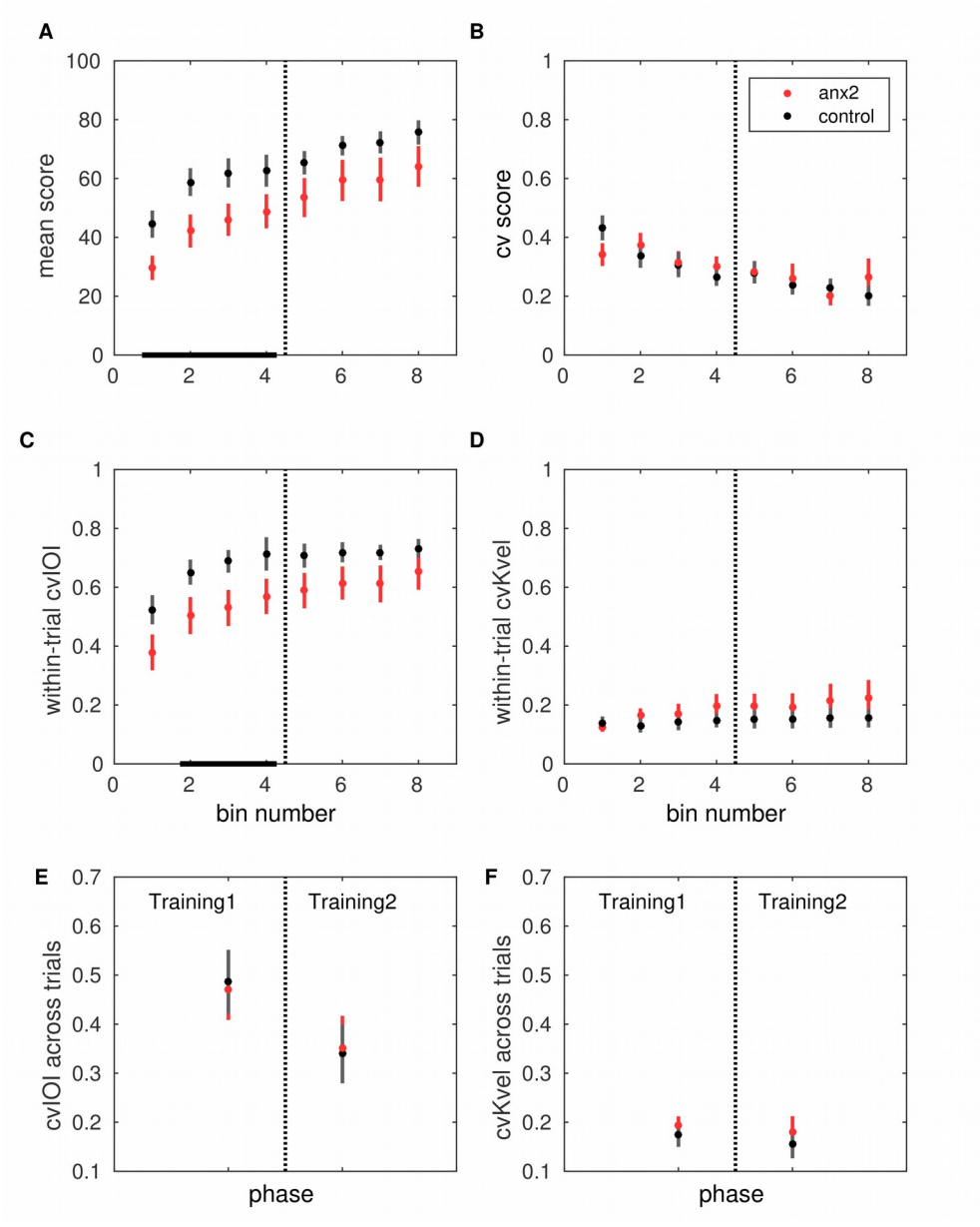

Figure 3 – figure supplement 2. Control experiment: Effect of anxiety on variability and learning after removal of the baseline exploration phase. Results of the control experiment in which new anx2 and control participants completed the reward-based learning phases without a prior exploration phase. Panels A/C/E are displayed as Figure 3A-C. Significant between-group differences are denoted by the black bar at the bottom ($p < 0.05$, FDR-corrected). (B) Coefficient of variability of the average score, showing similar dispersion in both groups. (D) Same as Figure 3 – figure supplement 1. (E) There was a significant drop in across-trials cvIOI for controls, not for anx2 participants ($p < 0.05$, FDR-corrected). (F) Same as Figure 3 – figure supplement 1.

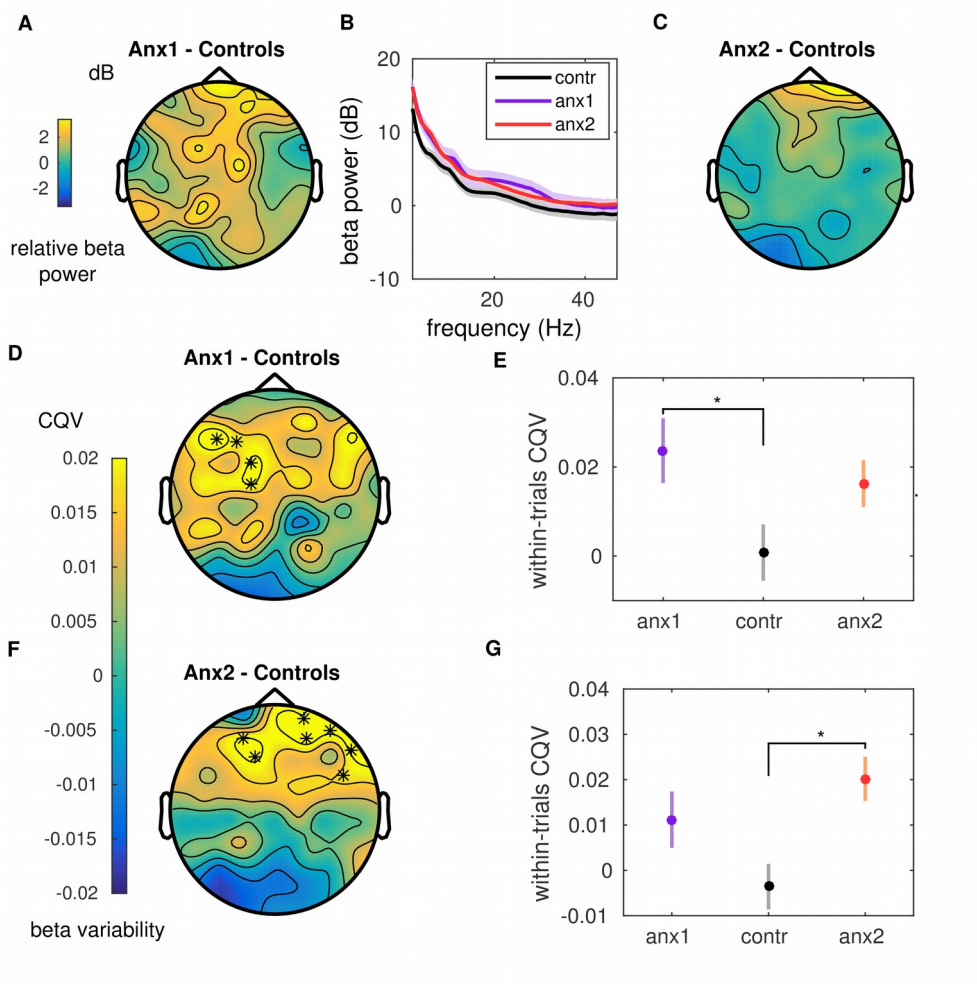

Figure 4 – figure supplement 1. Beta power and variability during reward-based training. Displayed as Figure 4: Power changes are shown in panels A-C. No between-group differences were found ($p > 0.05$). Changes in variability of beta amplitude envelope (Beta CQV) are shown in panels D-G. Anx1 participants had larger beta CQV values than control participants across sensorimotor electrodes ($p < 0.025$, two-tailed cluster-based permutation test, denoted by the asterisks). Anx2 participants also had larger beta CQV than control participants, albeit in a region of frontal electrodes ($p < 0.025$, two-tailed cluster-based permutation test, denoted by the asterisks).

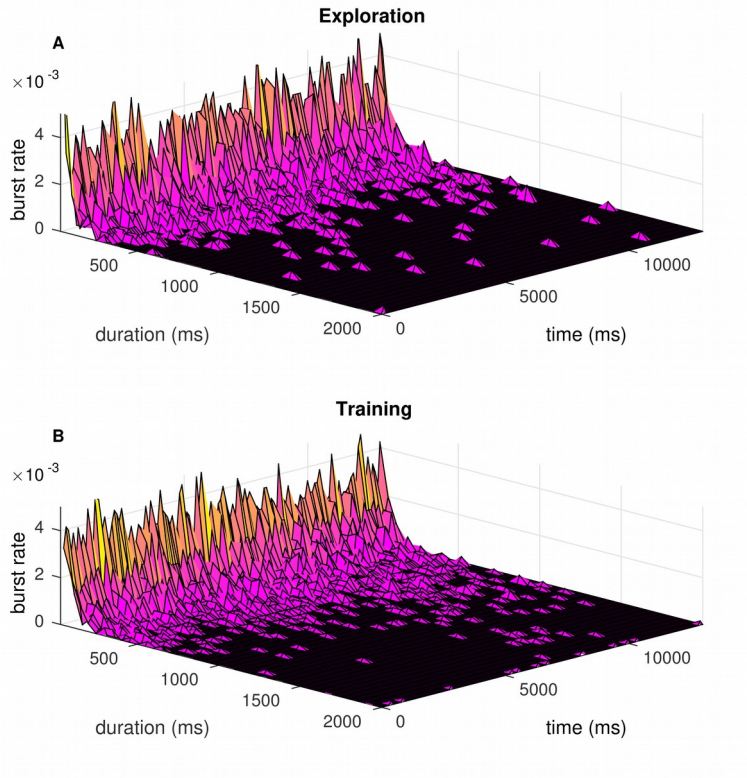

Figure 6 – figure supplement 1. Rate of beta bursts as a function of burst duration (range 50 – 2000ms) and time during trial-wise performance (trial lengh 0 – 12000ms). (A) Rate of oscillation bursts in one representative subject during exploration. (B) Same as (A) but during training.

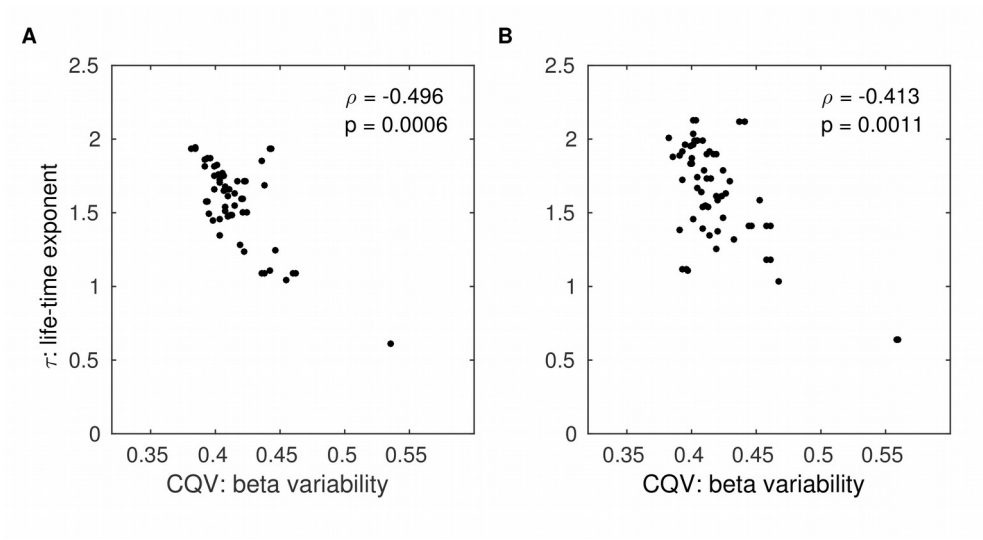

Figure 6 – figure supplement 2. Association between the life-time exponent of the beta-bursts distribution and the measure of variability of the beta amplitude envelope. Non-parametric rank correlation (Spearman ρ) across all 60 participants between the life-time exponent, τ, of the oscillation-bursts distribution and the beta CQV index during exploration (A) and training (B).