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RUNNING HEAD: PHYSIOLOGICAL AROUSAL AND DURATION PERCEPTION

The relationship between pain induced autonomic arousal and perceived duration

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## Abstract

Emotional distortions of the perceived duration of events are often explained in terms of increases and decreases in arousal. Whilst this explanation is theoretically plausible, there is a lack of evidence for a direct relationship between physiological arousal and perceived duration. The aim of the current study was to investigate whether physiological arousal, defined by autonomic nervous system (ANS) activity is directly related to perceived duration. In two experiments we measured skin conductance level (SCL) and high frequency heart rate variability (HF HRV) during verbal estimation tasks. In Experiment 1, participants estimated the duration of electro-cutaneous stimuli previously rated as inducing no pain, low pain and high pain. High intensity stimuli were perceived as lasting for longer than low intensity stimuli, and these changes in duration estimation were associated with changes in ANS activity. In Experiment 2, participants estimated the duration of a neutral visual stimulus whilst experiencing different intensities of background thermal pain (no pain, low pain, and high pain), to determine whether task-irrelevant arousal also affects time perception. Duration estimations for the neutral stimulus did not increase with pain intensity despite significant increases in SCL. Furthermore, there was no association between ANS activity and time estimation in Experiment 2. These findings suggest that the relationship between physiological arousal and time perception is more complex than previously described. Whilst physiological arousal can influence the perceived duration of events, it appears to have a greater capacity to do so when the to-be-timed stimulus is itself the source of arousal.

Keywords: Perceived duration, Physiological arousal, Pain, SNS, PSNS

## Introduction

Common adages such as “time flies when you are having fun” suggest that subjective perceptions of duration are influenced by the activities we perform and the emotions we experience (Wearden, O’Donoghue, Ogden, & Montgomery, 2014). A consistently reported finding is that fear inducing stimuli are perceived as lasting for longer than neutral stimuli (Droit-Volet, Brunot, & Niedenthal, 2004). For example, angry faces (Droit-Volet et al., 2004), negative valence high arousal images (Angrilli, Cherubini, Pavese, & Manfredini, 1997), sounds (Noulhiane, Mella, Samson, Ragot, & Pouthas, 2007) and somatosensory stimulation (Fayolle, Gil, & Droit-Volet, 2015) are all perceived as longer than neutral stimuli.

These effects are usually explained through an increase in arousal (see Gil & Droit-Volet, 2012 for discussion). In Scalar Expectancy Theory (SET; Gibbon, Church & Meck, 1984) arousal increases the rate at which the pacemaker emits output, resulting in greater accumulation and longer perceived duration. In the Striatal Beat Frequency model (SBF), time is processed by oscillation frequencies of cortex neurons that are detected by the striatum. Arousal is thought to modulate dopamine levels in the cortico-striatal circuits, modulating the oscillation frequencies (Matell & Meck, 2004). In interoceptive models of timing, distortions to time result from concurrent activation of the anterior insular cortex during timing and homeostatic regulation (Craig, 2002). These suggestions are supported by observations that cardiovascular activity (Lambourne, 2012), pain (Ogden, Moore, Redfern & McGlone, 2014; Fayolle et al., 2015), increased body temperature (Wearden & Penton-Voak, 1995) and the administration of dopamine agonists (Cheng, Ali, & Meck, 2007) all lengthen perceived duration. Furthermore, when judging the duration of emotional stimuli, perceived

duration can be determined by how arousing the stimuli is and not just its valence (Gil & Droit-Volet, 2012; Noulhiane et al., 2007).

Although arousal is consistently implicated in temporal distortions, there is not a clear and shared definition of arousal in the time perception literature (e.g., Wearden, Philpott & Win, 1999). For example, the term “arousal” is used interchangeably to describe arousal resulting from emotion induction (see Gil and Droit-Volet, 2012) but also from hypothesised changes in cortical activity as a result of repetitive stimulation (see Droit-Volet, 2010; Jones, Allely & Wearden, 2011 for examples). Furthermore, the relationship between arousal and perceived duration is often assumed rather than tested. For example, the association between heightened arousal and longer perceived durations has been evidenced by participants stating that they feel aroused, or, experimenters choosing stimuli which they believe to be arousing (e.g., Tipples, 2008) rather than measuring arousal directly. In studies in which measures of the physiological response to the to-be-timed stimuli are taken, the measures are rarely related back to the time estimates themselves. For example, although Angrilli et al., (1997), Droit-Volet (2010) Fayolle et al., (2015), Mella Conty & Pouthas (2011) demonstrate that their arousing stimuli do produce physiological arousal, they do not then demonstrate that the change in physiological arousal itself is related to the change in duration perception.

One way to define arousal is through changes in the activity of sympathetic and parasympathetic branches of the autonomic nervous system (ANS). The sympathetic nervous system (SNS) is dominant during stress and fight/flight responding. Increases in SNS activity increase heart rate (HR), decrease heart rate variability (HRV) and increase peripheral vasoconstriction (Sztajzel, 2004; Mendes, 2009). Increased SNS activity also modulates the electrical activity of the skin resulting in increased sweating (Critchley, 2002). Skin

Conductance Level (SCL) can be measured as an index of SNS activity. The parasympathetic nervous system (PSNS) is dominant during relaxation and rest. Increases in PSNS activity decrease HR, increase HRV and increase peripheral vasodilatation (Sztajzel, 2004; Mendes, 2009). PSNS activity is measured by calculating High Frequency Heart Rate Variability (HF HRV), which is HRV in the range of 0.15-0.4 Hz. This range is associated with the respiratory sinus arrhythmia (the increase and decrease of heart rate during inhalation and exhalation, respectively), which is considered to be solely determined by the PSNS (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). Increases in HF HRV correspond to increased PSNS activity.

Few studies have directly tested the relationship between physiological arousal defined as ANS activity and time perception (Cellini, Mioni, Levorato, Grondin & Stablum, 2015; van Hedger, Necka, Barakzai & Norman, 2017; Fung, Crone, Bode & Murawski, 2017; Meissner & Wittmann, 2011; Pollatos, Yeldesbay, Pikovsky & Rosenblum, 2014). Of those existing studies, the majority have focused on how resting state cardiac activity is related to temporal perception, rather than how changes in ANS reactivity relate to temporal distortions. For example, Cellini et al., (2015) observed that higher vagal tone was associated with lower error rates on a temporal production task and Pollatos et al., (2014) observed that greater vagal control was associated with less error on a reproduction task. Both Cellini et al., (2015) and Pollatos et al., (2014) acknowledge that this may just reflect the influence of vagal tone on attention and working memory (see Thayer, Hansen, Saus-Rose & Johnsen, 2009). Other studies have used measures which, although indicating changes in ANS activity, are non-specific to the SNS and PSNS branches (Fung et al., 2017; Hawkes, Joy & Evans, 1962; Osato, Ogawa & Takaoka, 1995). Fung et al., (2017), for example, tested the relationship between baseline levels of low and high frequency HRV and duration reproduction and found

an association between low frequency HRV and less accurate duration reproduction.

Although this indicates a relationship between the ANS and perceived duration, low frequency HRV is difficult to interpret as it is influenced by both SNS and PSNS activity (see Reyes del Paso, Langewitz, Mulder, Roon & Duschek, 2013 for discussion).

van Hedger et al., (2017) used more direct measures of SNS (Pre-ejection Period) and PSNS activity (HF HRV) to test the effect of a social stressor on temporal reproductions of neutral, negative and positive images. By manipulating emotional state, they were therefore able to explore how changes in ANS reactivity relate to emotional distortion of time. There was a significant correlation between changes in reproduction durations for the negative images (before and after the stressor) and changes in SNS activity, although this correlation was found only for short stimulus durations (400ms) and not long ones (4000ms). No relationships were found between SNS activity and reproductions of positive or neutral stimuli. There were also no relationships between PSNS and any reproduction. The absence of an overall lengthening effect of the stressor on duration perception suggests that the relationship between ANS activity and duration perception may be more complex than initially indicated. The fact that a relationship between SNS activity and perceived duration was only observed for the negative stimuli perhaps suggests that SNS activity only affects perceived duration when there is a large change in SNS activity, not a small one. Negative stimuli produce larger physiological responses than positive stimuli (Cacioppo & Gardner, 1999). Thus, physiological change may only have been sufficient to affect timing when the effect of the stressor and the negative IAPS images combined. It may also suggest that ANS activity only affects duration processing when the to-be-timed stimulus is itself arousing. However, it should also be noted that the use of a reproduction method in van Hedger et al., (2017) meant that participants experienced the to-be-timed-stimulus and made their

reproduction in the same state (i.e. prior to the stressor and after the stressor). The absence of a state change between stimulus presentation and stimulus reproduction is likely to have limited any temporal distortion observed as any effect of the stressor (i.e. change in internal clock speed) would be present during the timing of the stimulus and the timing of the reproduction. Furthermore, because separate physiological recordings were not taken for the different emotional categories (positive, negative and neutral) it is not possible to identify how the physiological response to the emotional stimuli itself related to their perceived duration.

A complex relationship between arousal and temporal distortions is evident in other studies. Angrilli et al. (1997) found that positive and negative valence had opposing effects on duration perception at high and low levels of arousal. Similarly, Gil & Droit-Volet (2012) indicated that the semantic content of an image can influence the extent to which it distorts time, even when the arousal ratings are comparable. This suggests that a basic model of temporal distortions in which increases in arousal result in increases in perceived duration may be too simplistic (Cheng, Tipples, Narayanan & Meck, 2016; Lake, 2016; Lake, Labar & Meck, 2016). Reducing the differences between the stimuli used in high and low arousal conditions is necessary to clarify the relationship between ANS activity and perceived duration.

One effective way of manipulating arousal is through the induction of noxious somatosensory stimulation (i.e. experimental pain). Two common methods of inducing noxious somatosensory stimulation are through electro-cutaneous stimulation and thermal stimulation (see Kyle & McNeil, 2014 for review). Both are consistently reported as painful and increasing stimulus intensities are positively correlated with both ANS activity and verbal



reports of pain experience (Möltner, Hölzl & Strian, 1990; Vassend & Knardahl, 2005). SNS and PSNS activity can therefore be modified by administering differing intensities of electro-cutaneous and thermal stimulation to participants. Electro-cutaneous stimulation (Fayolle et al., 2015) and thermal stimulation (Ogden et al., 2014) are also effective methods of distorting perceived duration. To-be-timed events including electro-cutaneous or thermal stimuli are consistently perceived as longer than neutral events of the same duration (Fayolle et al., 2015; Ogden et al., 2014). The magnitude of subjective lengthening of duration is typically greater than that observed for visual and auditory stimuli, suggesting that electro-cutaneous and thermal stimulation are particularly effective methods of distorting perceived duration (Ogden et al., 2014). Whilst their effectiveness is suggested to be because of their ability to modify arousal, the direct relationship between the arousal that they evoke and their perceived duration has never been tested.

The present study therefore aimed to test the relationship between physiological arousal, defined as ANS activity, and perceived duration using noxious somatosensory stimulation. Two experiments are reported. In Experiment 1, participants were required to estimate the duration of electro-cutaneous stimuli previously rated as inducing no pain, low level pain and high level pain. In Experiment 2, participants were required to estimate the duration of a neutral visual stimulus whilst concurrently experiencing task irrelevant thermal stimulation previously rated as inducing no pain, low pain and high pain. Throughout both experiments, measures of SCL and HF HRV were recorded as indicators of SNS and PSNS arousal, respectively.

For both experiments it was expected that the high pain stimulus would elicit greater SNS activity compared to the low pain and a no pain stimulus. It was also expected that duration

estimates would lengthen with increasing stimulus intensity. In all conditions, greater SNS reactivity was expected to be associated with longer perceived durations.

## Experiment 1

Experiment 1 tested the hypothesis that changes in ANS activity are correlated with distortions of perceived duration. Participants were asked to estimate the duration of electro-cutaneous stimuli they had previously rated as not painful, low level pain and high pain, whilst SCL and HF HRV were recorded. Different stimulus intensities (no pain, low pain and high pain) were used to establish whether different levels of arousal have different relationships to perceived duration. It was expected that electro-cutaneous stimuli rated as low level pain and high pain would be perceived as lasting longer than stimuli rated as not painful, replicating previous findings (Fayolle et al., 2015; Ogden et al., 2014). It was further expected that increased electro-cutaneous stimulus intensity would be associated with increased SCL, reflecting greater SNS activity for the high pain than for the low pain and no pain conditions. Critically, it was also expected that SNS activity would be positively related to perceived duration. As previous evidence suggests that only large changes in arousal affect perceived duration (e.g., Gil & Droit-Volet, 2012; van Hedger et al., 2017), we expected this relationship to be stronger in the high compared to the low pain condition. In contrast, we did not expect to find any correlation between PSNS arousal (indexed by HF HRV) and time estimation, as in van Hedger et al. (2017).

## Method

### *Participants*

Forty participants (30 females and 10 males; mean age = 26.20, SD = 3.91) were recruited. Sample sizes for Experiments 1 and 2 were determined by examining that used in previous research examining the effect of pain on time perception (Rey et al., 2017; Ogden et al., 2014). Participants were required not to be pregnant, not have a history of epilepsy and not to have chronic pain, heart disease, skin problems or any impairment of body sensation. Additionally, they were asked not to take any analgesic during the 8 hours prior to the experiment. Participants were reimbursed £10 in vouchers for taking part. The study was approved by the Liverpool John Moores University ethics committee and informed consent was obtained from all participants.

#### *Apparatus and materials*

*Pain stimulation:* Electro-cutaneous stimulation was used as the painful stimulus because of its high degree of temporal acuity. A Digitimer DS7A Current Stimulator (Digitimer Ltd) was used to present the stimulation, which provides up to 100mA current intensity at up to 400V voltage. Two electrodes were placed on the left volar forearm of participants 10cm from the wrist. The stimulation consisted of a train of 2ms pulses at 300V which were repeated for the desired duration.

*Physiological apparatus:* Biopac MP30B-CE was used to record EDA and ECG signals from which SCL and HF HRV were extracted, respectively (extrapolation is described below). Two sets of electrodes were used to record EDA and ECG separately. For EDA a set of two electrodes were applied on the index and middle finger of the right hand. For the ECG a set of three electrodes were applied on the torso to reproduce the Einthoven's triangle (one electrode on each shoulder and one on the left hip). The Biopac MP30B-CE was connected to a computer which recorded the physiological activity through the Biopac Student Lab Pro 3.7

software. The software was programmed to filter the EDA and the ECG signals in real time with band-pass of 0 – 35 Hz and .5 – 35 Hz, respectively.

### *Procedure*

Participants completed a health screening questionnaire to confirm suitability to participate. Participants then performed three tasks; 1) an initial intensity rating task to establish three stimulus intensity levels (no pain, low pain and high pain); 2) a verbal estimation task and 3) a further post-timing intensity rating task to establish whether the three intensity levels were still perceived as different after the verbal estimation task. These tasks were administered using E-Prime software (<http://www.pstnet.com>).

### *Initial intensity rating task*

This task was used to establish the three (no pain, low pain and high pain) stimulus intensity levels for each participant for subsequent use in the verbal estimation task. Participants were informed that their task was to use an 11-point Numeric Rating Scale (NRS, Jensen & McFarland, 1993) to indicate how painful a series of electro-cutaneous stimuli were (0 = no pain at all, 10 = worst pain imaginable). Initially, participants experienced a stimulus of 0.20mA for 750ms on their forearm. If at this initial level of intensity participants did not experience any pain, but the stimulus was clearly perceptible as stimulation, this was reported as 0 in the NRS and the electro-cutaneous stimulus was increased at a rate of 0.20mA per trial until participants indicated a score of 6 on the NRS, or until the stimulus intensity reached 4.00mA. If at the initial level of intensity participants experienced pain, the electro-cutaneous stimulus was decreased at a rate of 0.05mA per trial until participants indicated a score of 0 on the NRS. Then these participants were asked to rate again the initial stimulus of 0.20mA before increasing the stimulus at a rate of 0.20mA per trial until they indicated a score of 6 on

the NRS or until the stimulus intensity reached 4.00mA. The three stimuli selected for the verbal estimation task included the last one rated as 0 on the NRS (no pain) and the first ones rated as 3 (low pain; following Serlin, Mendoza, Nakamura, Edwards & Cleeland, 1995) and 6 (high pain; following Khoshnejad, Martinu, Grondin & Rainville, 2016).

### *Verbal estimation task*

Participants were asked to judge the duration of a series of electro-cutaneous stimuli delivered to their arm, which were set to the intensities established during the intensity rating task. The experiment consisted of three blocks, one for each pain intensity condition (no pain, low pain and high pain), presented in a counterbalanced order across participants. Before each block participants were asked to watch an 8 minute relaxing video-clip in order to measure their SCL and HF HRV in a baseline condition. This clip consisted of scenes of ocean life accompanied by relaxing music. Following this baseline recording period participants completed a block of verbal estimation.

At the start of each block, participants were instructed that a series of stimuli would be presented to their forearm, lasting between 50ms and 1700ms, and that their task was to verbally estimate the duration of each stimulus in milliseconds. After the participant pressed spacebar to initiate the start of the block, there was an inter-stimulus interval (ISI) randomly chosen from a 1500-2500ms range, which preceded the stimulus presentation. After stimulus presentation, there was an ISI of 1000ms then the response prompt was displayed cueing the participant to estimate the stimulus duration. A microphone and Audacity software were used to record participant's estimations. Participants then pressed the spacebar for the next trial. Each block contained 48 stimuli; five standard durations (242ms, 455ms, 767ms, 1058ms and 1296ms) each of which was repeated six times and eighteen additional stimuli, the duration

of which was selected at random from a uniform distribution ranging from 100ms to 1500ms. The purpose of these additional trials was to disguise the repeated use of the same 5 experimental stimulus durations across the experimental blocks. The data from these 18 additional stimuli were not analysed (as in Ogden, Moore, Redfern & McGlone, 2015). In each of the three blocks there were therefore 48 trials, giving a total of 144 trials for the entire task. The order of presentation of the trials was randomised by e-prime for each participant. SCL and HF HRV were recorded through each block.

#### *Post-timing intensity rating task*

To establish whether the three intensity levels were still perceived as different after the verbal estimation task, participants rated the three intensity levels (no pain, low pain and high pain) using an 11-point NRS (0 = no pain at all, 10 = worst pain imaginable). Each intensity level was presented 5 times (a total of 15 stimuli) in random order. Each stimulus lasted 750ms.

#### *Physiological data extrapolation*

EDA and ECG were measured during the three experimental blocks and the three baselines, resulting in a total of 6 recordings per participant. Using Biopac software, recorded signals were visually explored for artefacts which were manually removed. Six SCLs per participant were extrapolated averaging across each EDA signal (three representing the three baseline recording and a further three representing the no pain, low pain and high pain recordings). Three baseline-corrected SCLs were then calculated by subtracting baseline SCLs from their respective experimental SCLs. The baseline-corrected SCLs were later used for all the analyses described in the below results section.

ECG signals were imported into Kubios HRV software (University of Kuopio, Kuopio, Finland) for the frequency domain measure of the High Frequency band (0.15-0.4 Hz). From the original ECG signal, the software retrieved the inter-beat (or RR) intervals and applied the smoothness priors method to remove the low frequency baseline trend component. Frequency domain estimates of HF HRV (in normalized units) were then derived using the power spectrum density with the fast Fourier transformation based on Welch's periodogram method (Welch, 1967). Six estimates of HF HRV per participant were therefore obtained and three baseline-corrected estimates of HF HRV were then calculated by subtracting baseline values from their respective experimental values. The baseline-corrected estimates were later used for all the analyses described in the below results section.

### *Results*

Out of forty participants tested, data from six participants were excluded from the analysis because their estimates did not display temporal sensitivity or they did not comply with the task instructions. These participants were excluded for having estimates which did not show sensitivity to the stimulus duration in the neutral condition i.e. short estimates for long stimuli and long estimates for short stimuli, or, flat gradients due to the repetitive use of a single duration estimate e.g. 1500ms. Therefore, we report the results based on data from the remaining 34 participants. Datasets of Experiments 1 and 2 have been made publicly available at [https://osf.io/z2xj9/?view\\_only=6b50ce31b85247a5ba512bcf75b25398](https://osf.io/z2xj9/?view_only=6b50ce31b85247a5ba512bcf75b25398).

Table 1 about here

Table 1 shows means and standard deviations of intensity current (mA) for the three pain intensities individuated during the initial intensity rating task and used during the verbal estimation task. Repeated measures ANOVA indicated that stimulus mA's were significantly different to each other,  $F(1.15, 37.95) = 216.61, p < .001, \eta_p^2 = .87$ ; confirmed by post-hoc tests using Bonferroni correction ( $ps < .001$ ). Data from the post-timing intensity-rating task confirmed that the three intensity levels were still perceived as different to each other at the end of the task  $F(2, 66) = 200.61, p < .001, \eta_p^2 = .86$ .

### *Physiological response*

Table 1 shows SCL and HF HRV for each stimulus intensity. Examination of Table 1 suggests that participants had higher SCL in the high pain than low pain and no pain conditions. A repeated measures ANOVA showed a significant effect of electro-cutaneous intensity (no pain, low pain and high pain) on SCL  $F(2, 66) = 4.93, p = .01, \eta_p^2 = .13$ . Post-hoc tests (Bonferroni corrected) showed that SCL was significantly higher in the high pain condition than in the low pain ( $p = .049, 95\% \text{ CI } 0.01, 1.71$ ) and no pain condition ( $p = .024, 95\% \text{ CI } 0.12, 2.07$ ). SCL was not significantly different between the low pain and no pain condition ( $p = .99, 95\% \text{ CI } -0.70, 1.18$ ). Although examination of Table 1 suggests that participants HF HRV increased from the no pain to the high pain condition (which would indicate increased PSNS activity), a repeated measures ANOVA showed no significant effect of electro-cutaneous intensity (no pain, low pain and high pain) on HF HRV  $F(2, 66) = 1.12, p = .33, \eta_p^2 = .03$ .

### *Verbal Estimation*

Figure 1 shows mean verbal estimations for each electro-cutaneous stimulus intensity condition. Examination of Figure 1 suggests that longer duration estimates were given in the



high pain than in the low pain and no pain conditions. A repeated measures ANOVA with electro-cutaneous intensity (no pain, low pain and high pain) and stimulus duration (242ms, 455ms, 767ms, 1058ms, 1296ms) as within subject factors, showed significant main effects of stimulus duration  $F(1.78, 58.83) = 360.35, p < .001, \eta_p^2 = .92$  and of pain intensity  $F(2, 66) = 5.93, p = .004, \eta_p^2 = .15$  on time estimates. Post-hoc tests (Bonferroni corrected) showed that estimates were significantly longer in the high pain condition than in the no pain condition ( $p = .01, 95\% \text{ CI } 15.83, 145.57$ ). The difference between the high pain and low pain condition was approaching significance ( $p = .054, 95\% \text{ CI } -0.72, 112.24$ ). Estimates were not significantly different in the low pain and no pain condition ( $p = .91, 95\% \text{ CI } -35.03, 84.90$ ). There was no significant interaction between stimulus duration and pain intensity  $F(5.14, 169.46) = 1.58, p = .17, \eta_p^2 = .05$ .

Figure 1 about here

#### *ANS activity and perceived duration*

To investigate whether there was a relationship between ANS activity and time perception the mean duration estimate for each intensity condition was calculated (Table 1). The change in mean duration estimate, SCL and HF HRV across the three intensity conditions was then calculated producing three change scores. For SCL change: 1) no pain to low pain (low pain SCL – no pain SCL), 2) no pain to high pain (high pain SCL – no pain SCL) 3) low pain to high pain (high pain SCL – low pain SCL). The same calculations were conducted on the HF HRV and the mean duration estimates. One-tailed correlations were then conducted to test whether 1) there was a positive correlation between changes in SCL change in duration estimate, and 2), to test whether there was a negative correlation between changes in HF HRV and changes in time estimation (i.e., whether decreases in HF HRV were associated with increases in duration estimation). Table 2 shows this analysis. Examination of Table 2

suggests that there were significant positive correlations between SCL and time estimate change for all conditions. That is, increases in SCL with each intensity condition were associated with increases in duration estimation. Significant negative correlations were observed between HF-HRV and time estimate change for the no-low pain condition and the low-high pain condition.

Table 2 about here

Multiple regression tested whether changes in SCL and HF HRV predicted changes in duration estimates. ANS activity explained 18.58% of the variance in the increase in duration estimates from the no pain to low pain condition ( $R^2 = .24$ ,  $F(2, 31) = 4.76$ ,  $p = .016$ ); SCL was a significant predictor ( $\beta = .37$ ,  $p = .04$ , 95% CI 1.14, 45.61) but HF HRV was not ( $\beta = -.21$ ,  $p = .24$ , 95% CI -5.26, 1.35). ANS activity explained 16.35% of the variance in duration estimate change from the no pain to the high pain condition ( $R^2 = .21$ ,  $F(2, 31) = 4.22$ ,  $p = .024$ ), SCL was a significant predictor ( $\beta = .46$ ,  $p = .007$ , 95% CI 9.05, 52.12) but HF HRV was not ( $\beta = -.05$ ,  $p = .78$ , 95% CI -3.46, 2.61). ANS activity explained 22.02% of the variance in duration estimate change the low pain to the high pain condition ( $R^2 = .27$ ,  $F(2, 31) = 5.66$ ,  $p = .008$ ), HF HRV was a significant predictor ( $\beta = -.37$ ,  $p = .024$ , 95% CI -2.37, 0.02) and SCL trended towards significance ( $\beta = .32$ ,  $p = .051$ , 95% CI -0.08, 41.77).

Mediator analysis was conducted to assess whether ANS activity had a direct or indirect effect on perceived duration. Mediator analysis tests whether the independent variable X has a direct effect on the dependent variable Y, or, whether the effect is indirect because it is mediated by one or more other variable M, the mediator(s). Mediator analysis calculates the indirect effect of X on Y via M (indexed by the coefficient  $ab$ ), the direct effect of X on Y

( $c'$ ) and the total effect of X on Y ( $c$ ), which is the sum of the direct and indirect effect. See Figure 2.

Figure 2 about here

Mediation analysis was conducted using the Mediation and Moderation for Repeated Measures (MEMORE) macros for SPSS developed by Montoya and Hayes (2017) using a path-analytic form following the methodology of Judd, Kenny, and McClelland (2001). MEMORE has been specifically developed to assess mediation in within subject repeated measure design where X is defined as a change of condition, as in the present case where X is defined as a change pain intensity. Consequently, MEMORE macros can only calculate the mediation effect in a two condition design. We therefore conducted three mediator analyses investigating whether SCL ( $M_1$ ) and HF HRV ( $M_2$ ) mediated the effect of pain intensity (X) on time estimation (Y) when X changed (i) from the no pain to the low pain condition and (ii) from the low pain to the high condition and (iii) from the no pain to the high pain condition. Each mediator analysis first calculated the total effect of X on Y ( $c$ ). Mediator analysis then calculated the effect of X on mediators ( $a_1$  for  $M_1$  and  $a_2$  for  $M_2$ ) and the effect of mediators on Y ( $b_1$  for  $M_1$  and  $b_2$  for  $M_2$ ). The indirect effect of X on Y via  $M_1$  ( $a_1b_1$ ) and via  $M_2$  ( $a_2b_2$ ) and the total indirect effect of X on Y considering both  $M_1$  and  $M_2$  ( $ab$ ) were tested using a bootstrap estimation approach with 5000 samples. The direct effect of X on Y ( $c'$ ) has been also calculated. All coefficients have been reported in Table 3.

Table 3 about here

Complete mediation was obtained for the no pain - high pain condition, supporting the hypothesis of a direct relationship between SNS reactivity and duration distortion. Although complete mediation was not obtained for the no-low pain conditions and the low-high pain

conditions, the effect of SCL on time estimates was either significant, or trending towards significant ( $p = .056$ ).

### *Experiment 1 Discussion*

Experiment 1 tested whether perceived duration was related to changes in ANS activity. As expected, SCL was significantly higher in the high pain, compared to the low pain, and no pain conditions, indicating increased SNS arousal. SCL did not differ between the no and low pain conditions. The effects of stimulus intensity on SCL were mirrored in the changes in time perception across the conditions; perceived durations were significantly longer in the high pain than low and no pain conditions. However, there was no significant difference in perceived duration between the no and low pain conditions.

The correlational analysis suggested that increases in SCL were associated with increases in duration estimation. This was confirmed by the regression and the mediation analysis, which showed that changes in SCL significant predicted changes in time estimation. Together, these findings suggest a direct relationship between SNS activity and perceived duration, with increased SNS activity being associated with longer perceptions of duration.

HF HRV did not differ significantly between the conditions, indicating no significant differences in PSNS activity. Despite this, the correlational analysis suggested that increases in HF HRV from the no pain to the low pain condition and from the low pain to the high pain condition (indicating increased PSNS activity with greater pain intensity) were associated with decreases in duration estimation. Furthermore, the regression analysis found change in HF HRV to be a significant predictor of change in duration estimation from the low pain to the high pain condition. This contrasts with the mediator analysis (which did not indicate any

mediation role of HF HRV) and with van Hedger et al.'s (2017) finding that changes in HF HRV after a stressful situation were not related to changes in the perceived duration of positive or negative images.

Together, the results of Experiment 1 suggest that ANS activity influences perceived duration. This confirms the predictions of models of timing such as SET and which suggest that our internal representation of duration is influenced by our level of arousal.

## Experiment 2

Experiment 2 sought to further understand the circumstances in which ANS activity can influence perceived duration. Specifically, the experiment aimed to test whether task-irrelevant changes in ANS activity (i.e. from a source other than the to-be-timed-stimulus) also influence the perceived duration.

Previous studies investigating the effect of task-irrelevant arousal on the perceived duration of neutral stimuli have produced inconsistent effects. For example, fear induced by a short film has been found to lengthen the perceived duration of neutral stimuli in a subsequent temporal bisection task (Droit-Volet, Fayolle & Gil, 2011). However, experiencing negatively valenced tactile stimulation (unpleasant rough touch to the arm) does not affect the perceived duration of concurrently presented neutral visual stimulus (Ogden et al., 2015). Furthermore, ANS activation induced by task-irrelevant stress is associated with changes in the perceived duration of negative stimuli but not neutral and positively valenced stimuli (van Hedger et al., 2017). This latter finding is inconsistent with the SET and SFB models of timing, which both predict that increased arousal from the stressor should affect the central

timing mechanism and therefore influence the perceived duration of all subsequent stimuli, not just negative stimuli. The relationship between arousal and perceived duration therefore appears less clear when the to-be-timed stimulus is not itself the source of arousal and arousal is therefore not task-relevant.

Experiment 2 therefore aimed to test whether task-irrelevant arousal can alter the perceived duration of neutral visual stimuli. To do this participants completed a verbal estimation task whilst in states of no-pain, low pain and high pain. SNS and PSNS activity were indexed through changes in SCL and HF HRV. Thermal stimulation was used to induce experimental pain because electro-cutaneous stimulation could not be safely delivered for the length of time required for this task (Reilly, 2012). As in Experiment 1, it was expected that SNS activity, indexed by SCL, would increase with increasing pain intensity. It was also expected that increases in SCL would be associated with increases in perceived durations. Given the finding of a negative relationship between HF HRV and duration estimation in Experiment 1, increases in perceived duration in the low and high pain conditions were also expected to be associated with decreases in HF HRV.

## Method

### *Participants*

Thirty-one participants (26 females and 5 males; mean age = 22.23, SD = 4.74) were recruited. Participants were required to meet the same criteria of Experiment 1 and they were reimbursed £10 in vouchers for taking part. The study was approved by the Liverpool John Moores University ethics committee and informed consent was obtained from all participants.

### *Apparatus and materials*

*Pain stimulation:* A Medoc PATHWAY-Advanced Thermal Stimulator was used to induce sustained pain stimulation. This equipment is designed for use in clinical and research settings, and induces pain through a metal plate, placed on the skin. The temperature is delivered and controlled through specialist hardware and software, designed for experimental purposes. Here, we induced thermal pain through a 30mm x 30mm Peltier thermode attached to the participants' left volar forearm. This equipment is able to increase the temperature at a ramp rate up to 8°C/second and to decrease it at a ramp rate of 4°C/second.

*Physiological apparatus:* As in Experiment 1, Biopac MP30B-CE was used to record EDA and ECG signals from which SCL and HF HRV were extracted, respectively. Technical characteristics were identical to the equipment used in Experiment 1.

### *Procedure*

Participants were initially asked to complete a health screening questionnaire to confirm their suitability to participate. Participants then performed two tasks 1) an intensity rating task to establish three stimulus intensity levels of the thermode (no pain, low pain and high pain) and 2) a verbal estimation task where participants judged the duration of a neutral visual stimulus under the three stimulus intensity levels. These tasks were administered using E-Prime software (<http://www.pstnet.com>).

### *Intensity rating task*

A search protocol was used to establish three subjective intensity levels of stimulation that were then used during the verbal estimation task. The thermode was first applied to the participant's left volar forearm. Participants were then informed that their task was to use the

11-point Verbal Numeric Rating Scale (NRS, Jensen & McFarland, 1993; 0 = no pain at all, 10 = worst pain imaginable) to identify three levels of stimulation: 0 (No pain), 3 (Low pain) and 6 (High pain), matched to the self-report intensities used in Experiment 1. Starting from a baseline temperature of 32°C participants were instructed to increase the temperature by pressing a mouse button. Each time the participant pressed the button a small increase of approximately .1°C occurred. Participants' aim was to increase the temperature until it was considered warm but not painful (as 0 on the NRS). Once this percept was achieved participants were asked to keep the temperature at that intensity for 15 seconds before being asked to confirm whether the sensation was still at the same intensity. If participants reported that the sensation was not the same they were asked to adjust the temperature and this check was performed again until a reliable percept was reached. Participants then repeated this procedure with the target intensity level replaced with a 3 on the NRS. After 15 seconds, participants were asked to confirm whether the pain was still at the same intensity and, if not, to adjust it. Finally, participants were instructed to increase the temperature further until the pain reached the intensity level of 6 on the NRS. Again, after 15 seconds, participants were asked to confirm whether the pain was still at the same intensity and, if not, to adjust it. A temperature of 48°C was never exceeded given the sustained period of stimulation to ensure participants' safety.

#### *Verbal estimation task*

Participants completed six verbal estimation tasks; two whilst experiencing no pain, two whilst experiencing low pain and two whilst experiencing high pain. The order of blocks was randomised for each participant. Each verbal estimation task contained 24 trials. Within each task there were three presentations of each of the standard durations; 242ms, 455ms, 767ms, 1058ms and 1296ms and nine additional durations which were selected at random from a



uniform distribution ranging from 100ms to 1500ms. The order of presentation of the trials was randomised by e-prime for each participant. Data from all trials was recorded but only data from the standard presentation durations was analysed (as in Ogden et al., 2015). Across the whole task participants therefore received 48 no pain, 48 low pain and 48 high pain trials. Trials were divided in this way at the request of the ethics panel to avoid lengthy initial exposures to high levels of pain.

Each verbal estimation task began with a four-minute baseline recording period which was followed by the verbal estimation task itself. During the baseline recording participants watched a 4-minute clip of the video used in Experiment 1 whilst baseline measures of SCL and HF HRV were recorded and no heat stimulation was applied.

Following completion of the baseline recording, heat stimulation was applied to the participants' volar forearm. For each of the three thermal intensities established (0, 3 and 6 on an NRS) a protocol was developed for concurrent testing. The temperature increased at a rate of 8<sup>0</sup>C/second to 1<sup>0</sup>C above each participant's set threshold. This then oscillated between 1<sup>0</sup>C above and 1<sup>0</sup>C below the participant's threshold at 8<sup>0</sup>C/second for 10 oscillations before returning to the baseline temperature (32<sup>0</sup>C) at a rate of 8<sup>0</sup>C/second. This procedure was repeated on a continuous cycle until participants completed each verbal estimation task. This protocol was used to reduce habituation to the thermal stimulus.

During the verbal estimation task participants were instructed to estimate, in milliseconds, the presentation duration of a white square (300x300pixels) which appeared on a black computer screen. Participants were told that square would be presented for between 50ms and 1700ms and were asked to verbalise their responses so that they could be recorded by a microphone.

After the participant pressed spacebar to initiate the start of the block, there was an ISI randomly chosen from a 1500-2500ms range, which preceded the stimulus presentation. After stimulus presentation, there was an ISI of 1000ms then the response prompt was displayed queuing the participant to estimate the stimulus duration. Participants then pressed the spacebar for the next trial. Measures of SCL and HF HRV were recorded throughout.

#### *Physiological data extrapolation*

EDA and ECG were measured during the six experimental blocks and the six baselines, for a total of twelve times per participant. For each participant, twelve SCLs and twelve estimates of HF HRV were therefore retrieved using the same extrapolation procedure as in Experiment 1. Baseline-corrected SCLs were then calculated subtracting baseline SCLs from the respective experimental SCLs, giving a total of six baseline-corrected SCLs per participant, two per each intensity level (no pain, low pain and high pain). Baseline-corrected SCLs of the same intensity level were then averaged to obtain a single SCL value per pain intensity that was used for further analysis. An identical procedure was used to obtain three estimates of baseline-corrected HF HRV, one per each intensity level.

#### *Results*

Out of thirty-one participants tested, one chose temperatures too low to be considered painful (37.5 °C as low pain and 39.5 °C as high pain). Therefore, we report the results based on data from the remaining 30 participants (Yarnitsky, Sprecher, Zaslansley & Hemli, 1995).

Table 4 about here

Table 4 shows means and standard deviations of temperatures (°C) for the three stimulus intensities individuated during the initial intensity rating task and used during the verbal estimation task. A repeated measures ANOVA indicated that the temperatures in each condition were significantly different  $F(1.42, 41.15) = 594.69, p < .001, \eta_p^2 = .95$ .

### *Physiological response*

Table 4 shows SCL and HF HRV of participants for each stimulus intensity. Examination of Table 4 suggests higher SCL in the high pain than in the low pain and no pain conditions. Meanwhile, HF HRV does not appear to decrease or increase consistently with pain intensity. A repeated measures ANOVA showed a significant effect of stimulus intensity (no pain, low pain and high pain) on SCL  $F(1.53, 44.48) = 12.41, p < .001, \eta_p^2 = .30$ . Post-hoc tests (Bonferroni corrected) showed that SCL was significantly higher in the high pain condition than in the low pain ( $p = .013, 95\% \text{ CI } 0.12, 1.23$ ) and no pain condition ( $p = .001, 95\% \text{ CI } 0.38, 1.60$ ). SCL was not significantly different in the low pain and no pain conditions ( $p = .09, 95\% \text{ CI } -0.04, 0.67$ ). A repeated measures ANOVA showed no significant effect of stimulus intensity on HF HRV  $F(2, 58) = 0.11, p = .89, \eta_p^2 = .004$ .

### *Perceived duration*

Figure 3 shows mean verbal estimates in each pain intensity condition. Examination of Figure 3 suggests that similar duration estimates were given for the no pain, low pain and high pain conditions. A repeated measures ANOVA with stimulus intensity (no pain, low pain and high pain) and stimulus duration (242ms, 455ms, 767ms, 1058ms, 1296ms) as factors showed a significant main effect of stimulus duration on duration estimates  $F(1.77, 51.26) = 246.14, p < .001, \eta_p^2 = .90$ . There was no significant effect of pain intensity  $F(2, 72) = 0.10, p = .91, \eta_p^2$

= .003 and no significant interaction between stimulus duration and pain intensity  $F(4.49, 130.09) = 0.83, p = .52, \eta_p^2 = .03$ .

Figure 3 about here

#### *ANS activity and perceived duration*

As in Experiment 1, to test the relationship between ANS activity and time perception, the change in mean duration estimate, SCL and HF HRV across the three conditions was calculated producing three change scores. One-tailed correlations were then conducted to investigate whether there was a positive correlation between changes in SCL and changes in time estimation and to test whether there was negative correlation between changes in HF HRV and changes in time estimation (see Table 5). Examination of Table 5 shows that there were no significant correlations between ANS reactivity and changes in time estimate.

Table 5 about here

Multiple regressions tested whether changes in SCL and HF HRV between each intensity condition predicted changes in perceived duration. The ANS activity was not able to explain any of the variance in changes from the no pain to the low pain condition ( $R^2 = .01, F(2, 27) = 0.11, p = .89$ ); from the no pain to the high pain condition ( $R^2 = .12, F(2, 27) = 1.80, p = .18$ ) and from the low pain to the high pain condition ( $R^2 = .01, F(2, 27) = 0.15, p = .86$ ).

To confirm that the absence of an effect of pain on perceived duration was not due to habituation to pain across the task, data from the first block and the second block of each condition was compared and analyzed separately. Paired samples t-tests show that time estimates and PSNS activity did not differ from block 1 to block 2 (all  $ps > .05$ ). In contrast,

paired samples t-tests show that SNS activity was significantly higher during block 1 than block 2. SCL was significantly higher in the no pain (0.82  $\mu$ mho), low pain (1.23  $\mu$ mho) and high pain (1.99  $\mu$ mho) tasks of block 1 than block 2 (no pain: 0.23  $\mu$ mho,  $p = .025$ ; low pain: 0.38  $\mu$ mho,  $p = .001$ ; high pain: 0.83  $\mu$ mho,  $p < .001$ ). Despite this, repeated measures ANOVAs indicated that stimulus intensity (no pain, low pain and high pain) had a significant effect on SCL in both blocks 1 and 2,  $F(1.59, 47.58) = 6.46$ ,  $p = .006$ ,  $\eta_p^2 = .18$  and  $F(1.49, 44.77) = 6.71$ ,  $p = .006$ ,  $\eta_p^2 = .18$ , respectively. Furthermore, paired samples t-tests show that changes in SCL between tasks (from the no pain to the low pain, from the no pain to the high pain or from the low pain to the high pain) were not significantly different in blocks 1 and 2 (all  $ps > .05$ ). Moreover, even in block 1, where SCL was significantly greater than in block 2 there was no significant correlation between changes in SCL and time estimations (all  $ps > .05$ ).

As in Experiment 1, path analytic mediator analysis was conducted to test whether physiological arousal (indexed by SCL and HF HRV) mediated the effect of pain intensity on time estimation. The findings of the mediator analysis are shown in Table 6. Pain intensity changes affected SCL in all three conditions, with increases in pain intensity increases being associated with increases in SCL. However, neither pain intensity nor physiological arousal affected time estimations. These results suggest that when the to-be-timed stimulus is neutrally valenced, and changes in ANS activity are task-irrelevant, the ANS reactivity does not influence perceived duration.

Table 6 about here

### *Experiment 2 discussion*

Experiment 2 tested whether changes in ANS activity from a task-irrelevant source can influence the perceived duration of a neutral stimulus. As expected, SCLs were significantly higher in the high pain compared to the low pain and no pain conditions, indicating increased SNS activity. SCL did not differ between the no and low pain conditions. In contrast to expectations and to Experiment 1, these effects were not mirrored in the changes in duration perception with similar estimates being given in the three conditions (no pain, low pain, high pain). The correlation and regression analyses did not show any association between ANS activity and time estimation. The absence of an effect of task-irrelevant arousal on perceived duration is compatible with Ogden et al. (2015) who observed that unpleasant tactile stimulation did not affect the perceived duration of a neutral stimulus. The absence of a relationship between ANS activity and perceived duration replicates van Hedger et al., (2017). Together these findings suggest that when the to-be-timed stimulus is neutrally valenced and changes in ANS activity are task irrelevant, the ANS change does not influence perceived duration.

## General Discussion

This study tested the hypothesis that the perceived duration of an event is influenced by physiological arousal, defined as ANS activity. This was tested in two experiments. In Experiment 1, the to-be-timed stimulus itself was arousing and thus arousal was task relevant. In Experiment 2, the to-be-timed stimulus was neutral and arousal originated from a task-irrelevant secondary source.

In both experiments, increased stimulus intensity was associated with greater SCL, indicating greater SNS activity. Stimulus intensity did not affect HF HRV suggesting no influence on PSNS activity. Despite similar relationships between stimulus intensity and ANS activity in

both experiments, stimulus intensity had different effects on perceived duration in the two tasks. In Experiment 1, when the to-be-timed stimulus was itself arousing, high intensity stimuli were perceived as lasting for longer than neutral stimuli, confirming previous findings (Ogden et al., 2014; Fayolle et al., 2015). However, in Experiment 2, when the to-be-timed stimulus was neutral and arousal originated from a task irrelevant source, there was no effect of arousal on duration estimation. These contrasting findings support previous suggestions that the relationship between arousal and perceived duration is more complex than previously predicted (Burle & Casini, 2001; Mella et al., 2011).

The lengthening effect of electro-cutaneous stimulation on perceived duration observed in Experiment 1 is compatible with previous suggestions that “arousal” increases the perceived duration of events (Gil & Droit-Volet, 2012). By examining both SNS and PSNS, Experiment 1 clarifies when and how the different branches of the ANS affect timing. SNS activation is positively related to perceived duration for lower and higher levels of stimulus intensity. This confirms van Hedger et al’s., (2017) observation that the perceived duration of sub-second presentations of negatively valenced stimuli was positively related to SNS activation. Whilst a number of previous studies have confirmed the arousing properties of their stimuli by measuring SNS response (Angrilli et al., 1997; Fayolle et al., 1995) few have established PSNS stimulus responses. Those which have directly tested the relationship between PSNS activity and perceived duration have concluded that the two were not related (van Hedger et al., 2017). In the current study however, PSNS activation was found to be related to perceived duration, but only when the SNS activity was moving from a moderate to a high level (i.e. from the low pain to the high pain condition). In these circumstances, increases in HF HRV (indicating increased PSNS activity) were associated with shorter duration estimates. This suggests that perceived duration can be influenced by PSNS activation, but

only when SNS activation is already high. Differing findings in relation to HF HRV in this study and others perhaps reflects differences in the levels of ANS reactivity produced by the stimulus, with van Hedger et al.'s (2017) study not using sufficiently arousing stimuli to observe an effect of PSNS on perceived duration. This highlights the importance of using sufficiently arousing stimuli in these types of studies. However, it is acknowledged that in the current study, PSNS reactivity was not affected by the pain manipulation itself. It is therefore possible that manipulations specifically designed to increase PSNS activity may produce changes which are more clearly related to temporal distortions. Future research should explore this.

Whilst the results of Experiment 1 suggest a clear and relatively simple relationship between ANS activity and perceived duration, the findings of Experiment 2 suggest that this relationship is unique to certain circumstances. Experiment 2 required participants to judge the duration of a neutral stimulus whilst experiencing arousing stimulation (heat pain) from a task-irrelevant secondary source. Although SCL was significantly higher in the high pain condition than in the other conditions, there was no effect of pain on duration estimation and changes in ANS activity were not related to duration estimation. This null effect is unlikely to be due to the use of thermal stimulation per se (as opposed to electro-cutaneous) because thermal stimulation has previously been demonstrated to be an effective method of distorting duration perception when the stimulus is task relevant (Ogden et al., 2014). Instead, these results suggest that there is no relationship between time perception and physiological arousal when the arousal is not task relevant.

Models of timing such as SET and SBF do not specify that different sources of arousal will have different effects on perceived duration. Instead, they imply that a change in arousal



resulting from “any” source will affect perceived duration. It is therefore unclear why, in Experiment 2, task irrelevant increases in arousal did not affect perceived duration. One possible explanation is that the ANS activation evoked in Experiment 2 was not sufficient to affect perceived duration. A comparison of SCL and HF HRV recorded in Experiments 1 and 2<sup>1</sup> suggests that mean SCL was higher and HF HRV lower in Experiment 1 than Experiment 2 for all conditions. Thus, SNS activation was greater and PSNS was lower in Experiment 1 than in Experiment 2. However, comparison of the change scores for SNS and PSNS activation (i.e. the change in SCL from one condition to another) do not differ between Experiments 1 and 2 suggesting that the condition-condition changes in arousal were similar in both experiments. However, it remains possible that despite similar changes in SCL and HF HRV across the experiments, the lower baseline levels in Experiment 2 reduced the effect of SNS and PSNS activity on perceived duration.

An alternative explanation is that the SNS activation evoked by thermal stimulus did increase perceived duration, however, this effect was “wiped-out” by the distracting effect of pain. Pain captures attention, reducing the attentional resources available for concurrent tasks (Moore, Keogh & Eccleston, 2012). Reduced attention to time can result in shorter perceptions of duration (Zakay & Block, 1996), as is observed in dual task studies (Brown, 1997). In Experiment 2, dividing attention between pain and the timing task may therefore have negated any effect of arousal induced increases in pacemaker/oscillation rate. Mella et al., (2011) provide a similar argument to account for the absence of temporal distortions when estimating the duration of negatively valenced sounds. Attention to time and pain may also have contributed to the lengthening effects observed in Experiment 1. Emotional stimuli, particularly negatively valenced stimuli, affect attention through both endogenous top-down

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<sup>1</sup> See Footnotes

mechanisms (i.e., high cognitive function driven processing) and exogenous bottom-up mechanisms (i.e., automatic, stimulus driven processing; Vuilleumier, 2005). This results in faster identification of negatively valenced stimuli and improved performance on tasks requiring their processing (see Vuilleumier, 2005 for a review). In Experiment 1, both endogenous and exogenous orientation was aligned to the same stimulus (i.e. the electrocutaneous stimulation), perhaps leading to an attentional advantage during temporal processing which may have contributed to longer perceptions of duration.

Although reduced attention to time can perhaps account for the null effects observed in Experiment 2, this explanation should be taken with caution. One effect of the attentional capture of pain is that it increases errors on ongoing tasks (Moore, Keogh & Eccleston, 2013). Similarly, one effect of divided attention during timing tasks is increased error or reduced accuracy (e.g. Brown, 1997; Brown & Boltz, 2002). If pain reduced the attentional resources devoted to the timing task we would expect performance to be different in the no pain condition (in which attention could be fully dedicated to the processing of duration), and the high pain condition (in which attention to time was reduced). However, in the current study no differences were observed suggesting that an attentional explanation for the differing findings from Experiments 1 and 2 may be too simplistic.

Alternatively, it is possible that previous suggestions that increased arousal lengthens perceived duration have been too general, and that the actual relationship between arousal and perceived duration is limited to situations in which arousal results from a to-be-timed stimulus itself. Although it remains unclear precisely how time is processed in the brain, one possibility is that there are a series of sensory specific timers associated with each sense (van Wassenhove, Buonomano, Shimojo & Shams, 2008), the output of which contributes to the central timing system. This suggestion is supported by Coull et al. (2015) who observed that

activation in the right inferior occipital cortex increased parametrically with overestimations of duration. Coull et al. (2015) suggest that this reflects sensory specific low-level passive coding of duration. Active temporal processing instead occurs in the supplementary motor area. If time is initially processed in sensory specific timing units then greater neural responses to emotional stimuli in those sensory specific regions in the brain may contribute to the temporal distortions observed. For example, emotional faces and sounds produce greater neural responses than neutral ones in the fusiform face area (Vuilleumier, Armony, Driver & Dolan, 2001) and auditory cortex (Mitchell, Elliott, Barry, Cruttenden & Woodruff, 2003) respectively. This increased neural responding may contribute to the subjective lengthening of perceived duration for these types of stimuli. In Experiment 1, increased neural responding in the motor cortices in response to the electro-cutaneous stimulation (McKay, Ridding & Miles, 2003) may have increased temporal processing in that area, leading to a lengthening of duration. In Experiment 2 however, because the to-be-timed visual stimulus was neutrally valenced, there would not have been increased neural activity in any sensory specific timing unit in the visual cortex and less opportunity for duration distortion. This theory therefore suggests that arousal originating from a different sense to the to-be-timed stimulus may have less capacity to modulate time processing in other modalities. This suggestion is supported by the observation that emotional distortions to time are often observed in studies where duration and emotion are presented in the same modality (Droit-Volet et al., 2011) but absent or inconsistent in studies in which they are presented in different modalities (Ogden et al., 2015; van Hedger et al., 2017).

Limited evidence for temporal distortions in cross-modal tasks may be, in part, because there is limited contrast between the arousing and in the neutral stimuli. Matthews, Stewart & Wearden (2011) demonstrated that the contrast between the intensity of the stimulus and the intensity of the background is determinant of the stimulus' perceived duration, rather than a

stimulus' absolute intensity. In Experiment 1, the contrast between the stimulus and the background changed from trial to trial. In Experiment 2, however the arousing stimulus was presented as a constant background the task. Therefore the absence of trial-to-trial contrast change may have contributed to the absence of a temporal distortion.

Limited effects of arousal on time perception may also result from the *function* of arousal induced distortions to time. Emotional distortions to duration are thought to have an evolutionary origin (Mella et al., 2011) wherein the subjective lengthening of the duration of arousing events provides some perceptual and cognitive advantage for survival (Craig, 2009). However, if lengthening of subjective duration is to be adaptive, it must also be limited to circumstances of specific threat. It would not be adaptive for perceived duration to speed up and slow down due to task-irrelevant or time irrelevant changes in ANS activation. Craig (2009) explains this in terms of salience; stimuli which are highly salient on a moment to moment basis increase right anterior insular cortex activity, resulting in a lengthening of perceived duration. In Experiment 1, moment-moment salience was high because the electrocutaneous stimulation, which has a high threat value, was being anticipated and processed. The salience of the stimulation may have increased throughout the task due to sensitization resulting in heightened neural responding, heightened pain experience and greater stimulus salience (Davis & Sheard, 1974). This, coupled with the endogenous goal of the task being to process the pain stimulus itself may have resulted in a subjective lengthening of duration. In Experiment 2 however moment-moment salience of the thermal pain was perhaps lower. This is because, although pain stimulation was present throughout the task resulting in nociceptive activation, cognitive evaluation of the stimulus likely determined that the pain is unavoidable and not task relevant (i.e., the task goal was to judge the visual stimulus not the thermal pain). In these circumstances antinociceptive mechanisms may have been endogenously activated leading to habituation and reduced pain experience (see Bingel, Schoell, Herken, Büchel &

May, 2007 for discussion). The combined effect of antinociceptive mechanisms reducing pain salience and pain processing being task irrelevant may therefore have reduced the likelihood of a lengthening of subjective duration.

This explanation is also biologically plausible. Electro-cutaneous stimulation produces increased activation in the right anterior insular cortex (Freund, Stuber, Wunderlich & Schmitz, 2007) which Craig (2009) associates with increases in perceived duration.

Furthermore, right AIC activation in response to pain is positively correlated to the perceived intensity of the stimulus (Carlsson et al., 2006; Freund et al., 2007; Frot & Mauguiere, 2003). Therefore, in Experiment 1 greater stimulus estimates may result from increased AIC activation, as suggested by Craig (2009). Conversely, antinociception and habituation to pain are associated with reductions in AIC activation (relative to when pain is first experienced) (Bingel et al., 2007). Therefore, in Experiment 2 reducing AIC activity may have prevented temporal distortions from manifesting through the combined processing of pain and time in the AIC. Further investigation of the role of the AIC in the timing of short durations is therefore required.

### *Limitations*

In Experiment 1, it is possible that the sample size may have led to an overestimation of the effect sizes observed (see Button et al., 2013 for discussion). Similarly, in Experiment 2 it is possible that the null effects observed may have been due to insufficient statistical power (Button et al., 2013). Future research in this area should therefore employ larger sample sizes.

### *Conclusions*

The experiments presented in this study confirm that there is a relationship between ANS activation and perceived duration. However, this relationship appears to be more complex

than previously suggested. ANS activity is only predictive of perceived duration when the source of ANS activation is task-relevant. When ANS activity results from a secondary source, its capacity to influence perceived duration appears limited. Furthermore, when ANS activation and perceived duration are related, ANS activation only accounts for a small proportion of the variance in perceived duration, suggesting that other factors are contributing to temporal distortions observed. Although a number of theories are offered relating to attention, stimulus modality and stimulus relevance, further behavioural and neuroimaging work is required to understand the precise circumstances in which ANS activation can alter the perceived duration of events.

#### Additional information

Datasets have been made publicly available at

[https://osf.io/z2xj9/?view\\_only=6b50ce31b85247a5ba512bcf75b25398](https://osf.io/z2xj9/?view_only=6b50ce31b85247a5ba512bcf75b25398).

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## FOONOTES

<sup>1</sup>Independent samples t-tests show that SCL was significantly higher in Experiment 1 than in Experiment 2 for the no pain ( $p = .024$ , 95% CI 0.11, 1.47), low pain ( $p = .043$ , 95% CI 0.02, 1.43) and high pain ( $p = .022$ , 95% CI 0.13, 1.65) conditions. HF HRV was significantly higher in Experiment 2 than in Experiment 1 for the no pain ( $p < .001$ , 95% CI -20.23, -6.22), low pain ( $p = .005$ , 95% CI -21.44, -3.98) and high pain ( $p = .031$ , 95% CI -15.83, -0.79) conditions. However, between Experiment 1 and Experiment 2 the changes in SCL (or HF HRV) from one condition to another were not significantly different (all  $ps > .05$ ). Changes in SCL from the no pain to the low pain condition ( $p = .85$ , 95% CI -0.88, 0.72), from the no pain to the high pain condition ( $p = .82$ , 95% CI -0.81, 1.02) and from the low pain to the high pain condition ( $p = .66$ , 95% CI -0.63, 0.99) were not significantly different in Experiment 1 and 2. Changes in HF HRV from the no pain to the low pain condition ( $p = .90$ , 95% CI -7.74, 8.77), from the no pain to the high pain condition ( $p = .24$ , 95% CI -3.36, 13.19) and from the low pain to the high pain condition ( $p = .32$ , 95% CI -4.31, 13.11) were not significantly different in Experiment 1 and 2.

TABLES

<i>Pain intensity</i>	<i>Mean electrical current (SD)</i>	<i>Mean post-timing rating (SD)</i>	<i>Mean SCL (SD)</i>	<i>Mean HF HRV (SD)</i>	<i>Mean time estimation (SD)</i>
<i>No pain</i>	0.65 (0.40)	0.28 (0.60)	1.28 (1.65)	-14.26 (14.66)	602.43 (195.33)
<i>Low pain</i>	1.70 (0.68)	2.37 (1.38)	1.51 (1.72)	-13.25 (17.76)	627.36 (178.56)
<i>High pain</i>	3.02 (1.03)	4.83 (1.57)	2.37 (1.76)	-10.39 (14.86)	683.12 (186.68)

Table 1. Means (and standard deviations) of electrical current (mA), post-timing pain ratings, baseline-corrected Skin Conductance Levels (SCL,  $\mu\text{mho}$ ), baseline-corrected High Frequency Heart Rate Variability (HF HRV, normalized units) and time estimations (ms) in the three intensity level conditions (no pain, low pain and high pain) in Experiment 1.



	<i>Changes from no pain to low pain</i>			<i>Changes from no pain to high pain</i>			<i>Changes from low pain to high pain</i>		
	1	2	3	1	2	3	1	2	3
1. Time estimation	—			—			—		
2. Skin Conductance Level	<b>.45**</b> <b>[0.21, 0.69]</b>	—		<b>.46**</b> <b>[0.25, 0.66]</b>	—		<b>.37*</b> <b>[0.17, 0.63]</b>	—	
3. High Frequency Heart Rate Variability	<b>-.35*</b> <b>[-0.64, -0.07]</b>	<b>-.39*</b> <b>[-0.64, -0.09]</b>	—	-.04 [-0.34, 0.27]	.01 [-0.31, 0.30]	—	<b>-.41**</b> <b>[-0.65, -0.20]</b>	-.14 [-0.53, 0.34]	—

Table 2. Correlation coefficients [95% confidence intervals] between changes of (1) time estimation (ms), (2) Skin Conductance Level (SCL,  $\mu\text{mho}$ ) and (3) High Frequency Heart Rate Variability (HF HRV, normalized units) from the no pain to the low pain, from the no pain to the high pain and from the low pain to the high pain condition in Experiment 1.

\*  $p < .05$ ; \*\*  $p < .01$

<i>Dependent variable, Y</i>	<i>Independent variable, X</i>	<i>Mediating variable, M</i>	<i>Effect of X on M (a)</i>	<i>Effect of M on Y (b)</i>	<i>Indirect effect (ab)</i>	<i>Total indirect effect (ab)</i>	<i>Direct effect (c')</i>	<i>Total effect (c)</i>	<i>Degree of mediation</i>
Time estimation	<i>Pain intensity (0) no pain (1) low pain</i>	SCL	0.24 [-0.52, 0.99]	<b>27.15*</b> [ <b>4.79, 49.51</b> ]	6.46 [-16.45, 30.70]	4.35 [-27.71, 32.50]	20.58 [-23.10, 64.27]	24.94 [-23.43, 73.31]	None
		HF HRV	1.01 [-4.08, 6.11]	-2.08 [-5.39, 1.23]	-2.10 [-16.92, 10.61]				
	<i>Pain intensity (0) low pain (1) high pain</i>	SCL	<b>0.86*</b> [ <b>0.17, 1.55</b> ]	21.91 <sup>†</sup> [-0.64, 44.46]	<b>18.78*</b> [ <b>1.90, 53.18</b> ]	10.51 [-19.52, 50.91]	45.25 [-1.40, 91.90]	<b>55.76*</b> [ <b>10.20, 101.32</b> ]	None
		HF HRV	2.86 [-2.82, 8.54]	<b>-2.89*</b> [ <b>-5.56, -0.22</b> ]	-8.27 [-31.28, 7.47]				
	<i>Pain intensity (0) no pain (1) high pain</i>	SCL	<b>1.10**</b> [ <b>0.31, 1.88</b> ]	<b>32.09**</b> [ <b>9.47, 54.71</b> ]	<b>35.15*</b> [ <b>6.91, 70.20</b> ]	<b>33.38*</b> [ <b>3.07, 71.17</b> ]	47.32 [-8.99, 103.63]	<b>80.70**</b> [ <b>28.37, 133.03</b> ]	Complete
		HF HRV	3.87 [-1.74, 9.48]	-0.46 [-3.57, 2.65]	-1.77 [-17.89, 15.69]				

Table 3. Mediation coefficients [95% confidence intervals] of Experiment 1.

SCL = Skin Conductance Level ( $\mu\text{mho}$ ). HF HRV = High Frequency Heart Rate Variability (normalized units).

<sup>†</sup> = .056; \*  $p < .05$ ; \*\*  $p < .001$

<i>Pain intensity</i>	<i>Mean temperature (SD)</i>	<i>Mean SCL (SD)</i>	<i>Mean HF HRV (SD)</i>	<i>Mean time estimation (SD)</i>
<i>No pain</i>	35.70 (1.29)	0.48 (1.04)	-1.04 (13.18)	608.89 (182.34)
<i>Low pain</i>	40.72 (1.12)	0.80 (0.98)	-0.54 (17.06)	605.72 (160.59)
<i>High pain</i>	43.60 (1.28)	1.48 (1.26)	-2.08 (15.17)	616.73 (174.39)

Table 4: Means (and standard deviations) of temperature (°C), baseline-corrected Skin

Conductance Levels (SCL,  $\mu\text{mho}$ ), baseline-corrected High Frequency Heart Rate Variability (HF HRV, normalized units) and time estimations (ms) in the three intensity level conditions (no pain, low pain and high pain) in Experiment 2.

	<i>Changes from no pain to low pain</i>			<i>Changes from no pain to high pain</i>			<i>Changes from low pain to high pain</i>		
	1	2	3	1	2	3	1	2	3
1. Time estimation	—			—			—		
2. Skin Conductance Level	-0.09 [-0.40, 0.25]	—		-.28 [-0.55, 0.02]	—		-.10 [-0.32, 0.15]	—	
3. High Frequency Heart Rate Variability	-.02 [-0.36, 0.26]	.24 [-0.15, 0.56]	—	-.16 [-0.56, 0.33]	-.16 [-0.46, 0.17]	—	-.02 [-0.30, 0.30]	-.10 [-0.42, 0.24]	—

Table 5. Correlation coefficients [95% Confidence intervals] between changes of (1) time estimation (ms), (2) Skin Conductance Level (SCL,  $\mu\text{mho}$ ) and (3) High Frequency Heart Rate Variability (HF HRV, normalized units) from the no pain to the low pain, from the no pain to the high pain and from the low pain to the high pain condition in Experiment 2.

\*  $p < .05$ ; \*\*  $p < .01$

<i>Dependent variable, Y</i>	<i>Independent variable, X</i>	<i>Mediating variable, M</i>	<i>Effect of X on M (a)</i>	<i>Effect of M on Y (b)</i>	<i>Indirect effect (ab)</i>	<i>Total indirect effect (ab)</i>	<i>Direct effect (c')</i>	<i>Total effect (c)</i>	<i>Degree of mediation</i>
Time estimation	<i>Pain intensity (0) no pain (1) low pain</i>	SCL	<b>0.32*</b> [0.03, 0.60]	-11.44 [-70.99, 48.10]	-3.61 [-18.31, 14.88]	-3.33 [-19.83, 17.76]	0.16 [-46.37, 46.68]	-3.17 [-45.54, 0.20]	None
		HF HRV	0.50 [-6.38, 0.37]	0.56 [-2.13, 3.24]	0.28 [-7.51, 9.39]				
	<i>Pain intensity (0) low pain (1) high pain</i>	SCL	<b>0.68**</b> [0.23, 1.12]	0.27 [-48.12, 48.66]	0.18 [-25.46, 19.37]	0.76 [-25.05, 21.03]	10.26 [-53.30, 73.81]	11.02 [-43.80, 65.93]	None
		HF HRV	-1.54 [-8.48, 5.40]	-0.38 [-3.40, 2.65]	0.58 [-8.30, 10.01]				
	<i>Pain intensity (0) no pain (1) high pain</i>	SCL	<b>0.99**</b> [0.50, 1.48]	-33.74 [-83.01, 15.54]	-33.51 [-88.24, 10.94]	-31.63 [-87.68, 13.40]	39.48 [-38.02, 116.97]	7.84 [-53.32, 69.01]	None
		HF HRV	-1.04 [-7.41, 5.32]	-1.80 [-5.61, 2.01]	1.88 [-18.14, 21.32]				

Table 6. Mediation coefficients [95% confidence intervals] of Experiment 2.

SCL = Skin Conductance Level ( $\mu$ mho). HF HRV = High Frequency Heart Rate Variability (normalized units).

\*  $p < .05$ ; \*\*  $p < .01$

## FIGURES

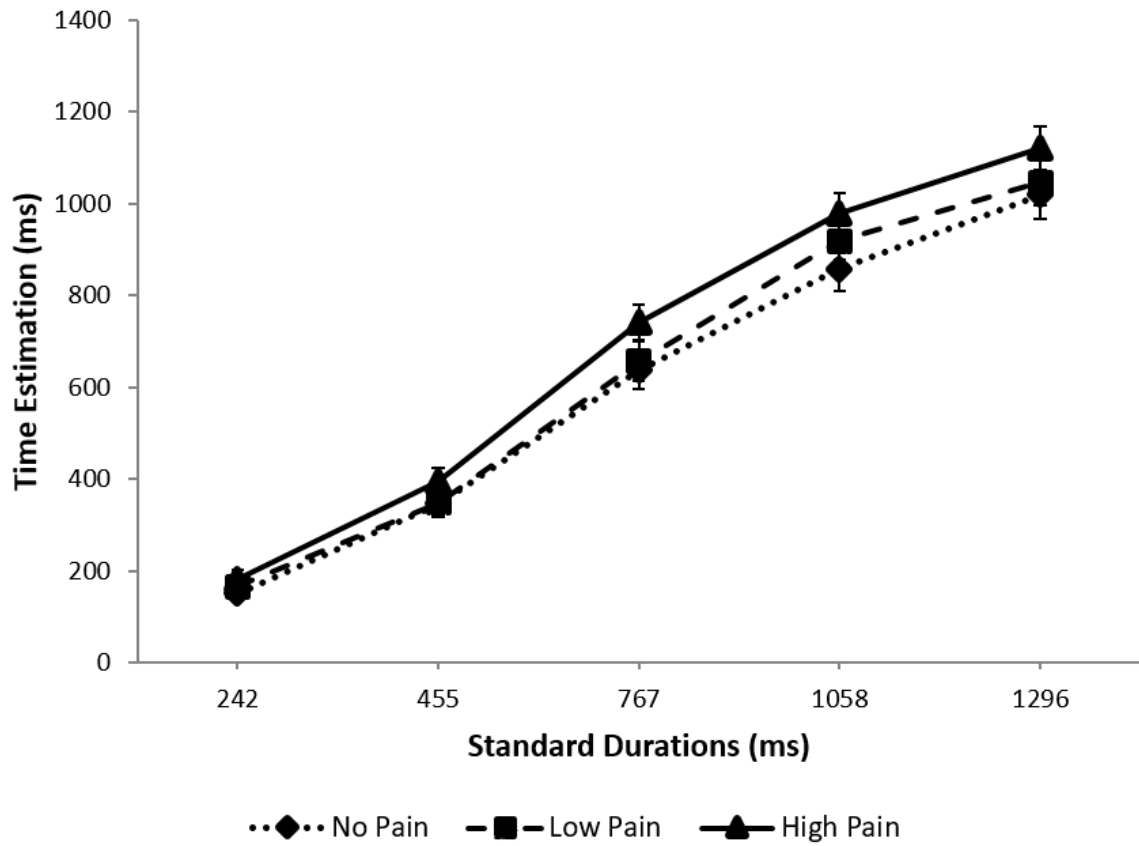


Figure 1: Means (and standard errors) of the verbal estimations (ms) plotted against the standard durations and divided by the intensity level conditions (no pain, low pain and high pain) in Experiment 1.

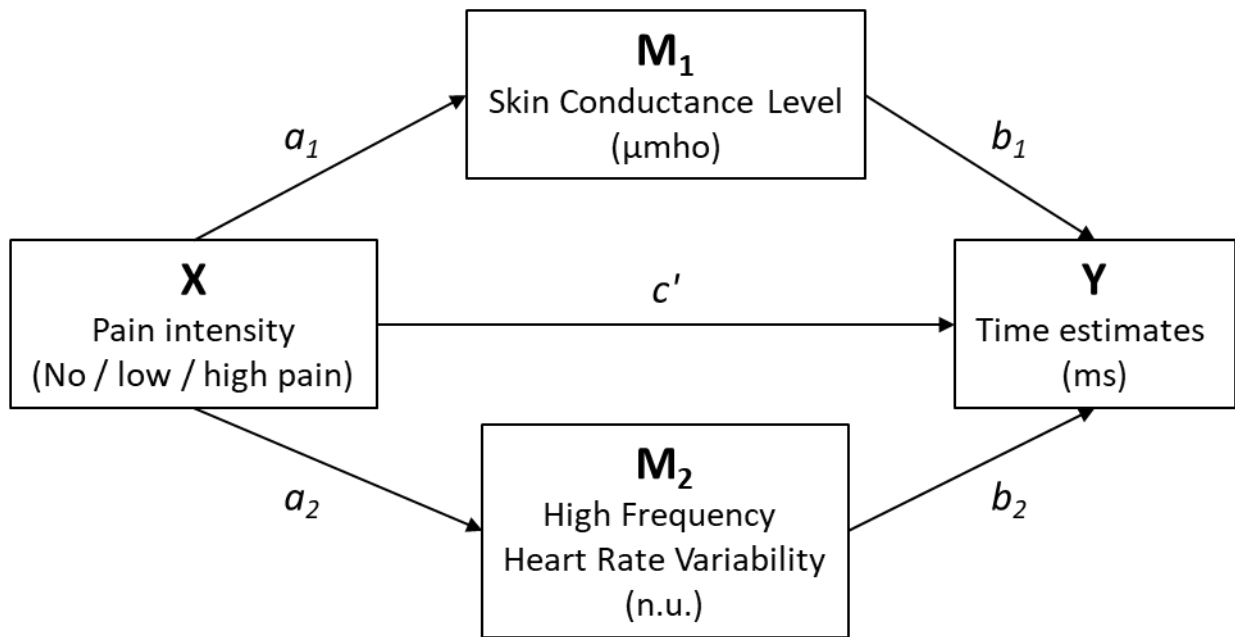


Figure 2. Model of the within-participant parallel mediation in path analytic form, showing the effect of pain intensity (X) on time estimation (Y) mediated by SCL (M<sub>1</sub>) and HF HRV (M<sub>2</sub>).

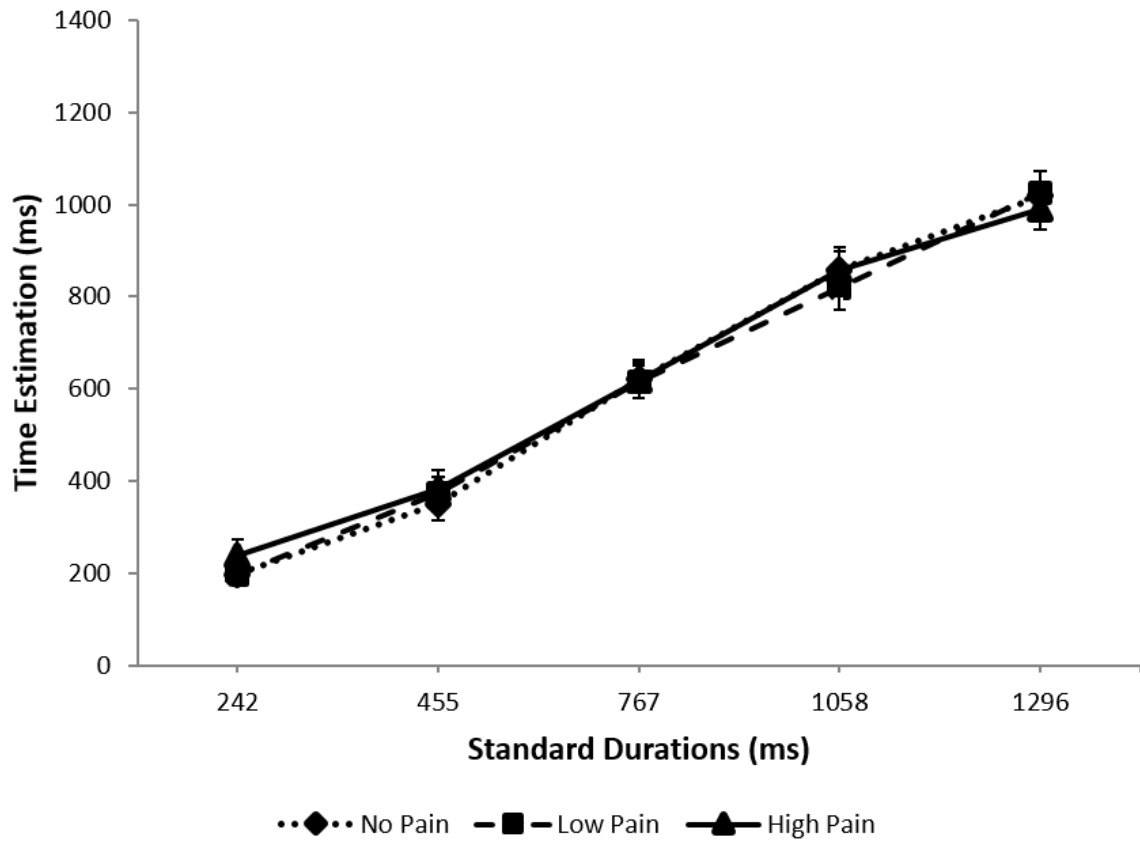


Figure 3: Means (and standard errors) of the verbal estimations (ms) plotted against the standard durations and divided by the intensity level conditions (no pain, low pain and high pain) in Experiment 2.