

# **THE EFFECT OF PAIN ON HUMAN TIME PERCEPTION**

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A thesis submitted in the partial fulfilment of the requirements of Liverpool John

Moores University for the degree of Doctor of Philosophy

February 2019

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## **Abstract**

This thesis explored the effect of pain on human temporal perception. This aim was achieved firstly by systematically testing the way in which pain experience affects duration estimates, and memory for duration and secondly by examining whether it was possible to reduce perceived duration of pain in clinical and no clinical population.

Chapter 5 examined the effect of different pain intensities on perceived duration when pain was the to-be-timed stimulus (i.e., task-relevant) and when pain was in the background (i.e., task-irrelevant). Participants were required to verbally estimate the duration of no pain, low pain and high pain electro-cutaneous stimulations and the duration of a neutral visual stimulus whilst being exposed to no pain, low pain and high pain thermal stimulation. Increases in the intensity of the electro-cutaneous stimulation were associated with longer verbal estimates, reflecting a multiplicative effect. However, low pain thermal stimulation did not affect the perceived duration of the visual stimulus and high pain thermal stimulation led to shorter verbal estimates. The lengthening effect of pain therefore appeared to be limited to circumstances when pain was task-relevant.

Chapter 6 examined whether changes in physiological arousal mediated the effect of task-relevant and task-irrelevant pain on time perception. Participants' physiological activity (skin conductance level and high frequency heart rate variability) was measured while they were asked to verbally estimate the duration of an electro-cutaneous stimulation at different intensities and a neutral stimulus whilst perceiving a thermal stimulation at different intensities. The lengthening effect of task-relevant pain on time perception, although did not replicate the multiplicative effect, was mediated by sympathetic arousal, supporting previous suggestions that temporal distortions due to pain are caused by changes in the arousal level. However, task-irrelevant pain did not affect verbal estimates of participants, despite it increased their physiological arousal, and there was no relationship between physiological arousal and verbal estimates. This suggests that changes in arousal do

not affect time perception when arousal arises from sources other than the to-be-timed stimulus.

Chapter 7 examined whether pain enhanced or disrupted the memorization of duration by using a temporal generalisation task. Participants were required to encode the duration of a tone whilst experiencing neutral or painful thermal stimulation and to recall the duration immediately after learning or after a delay. Delay affected neutral and pain related durations in a comparable way, suggesting that pain does not have any unique effect on the memorization of duration: pain does not enhance nor disrupt the memorization of duration information.

Chapter 8 tested whether a mindfulness intervention could reduce the lengthening effect of pain in healthy people and in chronic pain patients. Participants were asked to estimate the duration of visual, vibrotactile and electro-cutaneous stimuli before and after practicing mindfulness meditation for a week. Healthy participants gave similar verbal estimates before and after the intervention, suggesting that mindfulness was not able to modulate the perceived duration in any stimulus modality. In chronic pain patients mindfulness practice led to longer verbal estimates in any stimulus modality including pain, suggesting that mindfulness was not an appropriate intervention to reduce the lengthening effect of pain, however, caution should be taken when interpreting this latter finding due to the small sample.

Together the findings of this thesis show that task relevant pain distorts time, in part due to its capacity to increase sympathetic nervous system activity. Pain, however, appears to have no influence on memory for duration. Furthermore, interventions which reduce the intensity of pain do not appear to be effective in reducing the perceived duration of pain. Further research is therefore required to understand how the lengthening effect of pain can be mitigated in clinical and non-clinical settings.

## **Acknowledgements**

Firstly, I would like to thank Dr Ruth Ogden for introducing me to the field of time perception and for her support and insights during my PhD. I would also like to express my gratitude to the rest of my supervisory team: Dr Laura Mirams and Dr Helen Poole, for their advice, comments and encouragement. I also thank my colleagues in the PhD office, who are too many to list, for sharing joyful and stressful moments during the last three years. I am also very grateful to Miss Letizia Palumbo for her constant support, encouragement and advice in the last five years of my personal and academic life. Last but not least, I would like to thank my parents and sister for supporting me throughout writing this thesis, despite the 1400km between us, and for my life in general.

## Research Publications

The research reported in this thesis has led to the following publications:

### Based on Chapter 6

Piovesan, A., Mirams, L., Poole, H., Moore, D., & Ogden, R. (2018). The relationship between pain induced autonomic arousal and perceived duration. *Emotion*. <http://dx.doi.org/10.1037/emo0000512>

The research reported in this thesis has also led to the following conference presentations:

### Based on Chapter 5

Piovesan A., Mirams L., Pool H., Moore, D., Ogden, R. S. (2017) Judging the duration of painful stimuli and non-painful stimuli during a state of pain. EPS conference, London, January, 2017.

### Based on Chapter 6

Piovesan A., Mirams L., Pool H., Moore, D., Ogden, R. S. (2017) Does pain lengthen time estimation because it increases body arousal? A physiological study. EPS conference, Belfast, April, 2017.

Piovesan A., Mirams L., Pool H., Moore, D., Richter M., Ogden, R. S. (2017) How pain affects time estimation. A physiological study. 1st Conference of the Timing Research Forum, Strasbourg, October, 2017.

### Based on Chapter 7

Piovesan A., Mirams L., Pool H., Ogden, R. S. (2018) The effect of pain on memory for duration. 4<sup>th</sup> International Conference on Time Perspective, Nantes, August, 2018.



# **PART I**

## **THEORETICAL BACKGROUND**

# Chapter 1

## Time perception

### 1.1 – Internal clock models of time perception

#### 1.1.1 – *The history of internal clock models of timing*

The idea that humans have an internal clock to perceive duration was first suggested by François (1927) and then Hoagland (1933), following their observation of the effect of body temperature change on perceived duration. François (1927) asked participants to tap their finger on a regular basis while their body temperature was experimentally modulated, which led participants to tap their finger more frequently when their body temperature increased. This was similarly observed by Hoagland (1933), who repeatedly asked his wife to produce a time interval of 60 seconds while her body temperature fluctuated due to a natural fever. His wife produced the 60 seconds in a shorter amount of time when her body temperature was high in comparison to when it was low. The results of both studies suggested that the same time interval could be perceived differently depending on the perceiver's body temperature; with longer estimates and shorter productions when body temperature increases.

This led Hoagland (1933) to hypothesize that humans perceive duration through a biological clock, which follows the Arrhenius equation in which chemical reactions occur more quickly at higher temperatures than lower temperatures. Therefore, if humans possess a biological clock, its chemical reactions would be faster at higher temperatures leading to longer perceived durations, and slower at lower temperatures leading to shorter perceived duration. Hoagland (1933) suggested that this biological clock consisted of a pacemaker, which regularly produces pulses, and the perceived duration of an event is based on the quantity of pulses produced during the event. Hoagland (1933) suggested that the frequency of produced pulses is not constant but varies depending on body temperature, with higher body temperature corresponding to higher frequency or, in other words, to a greater number of pulses produced in the same time interval.

Treisman (1963) developed the first widely accepted model of timing (Figure 1.1). The model described a pacemaker, a counter, a store, a comparator and a verbal selective mechanism. The pacemaker constantly produces pulses with some variation of the inter-pulse interval, variation that leads to the scalar variance of the timing representation (see section 1.1.2, page 20). The pulses are recorded by the counter when an interval needs to be estimated and the count is then transferred into the store (which is part of the long-term memory) where, when needed, the comparator mechanism retrieves timing information assisted by the verbal selective mechanism. The model explains distortions to time (e.g., due to temperature) by proposing that the frequency with which pulses are outputted from the pacemaker is modulated by arousal; increases of arousal accelerate the production of pulses, leading to a greater count and longer perceived duration. In contrast, decreases of arousal slow down the production of pulses leading to a lower count and shorter perceived duration.

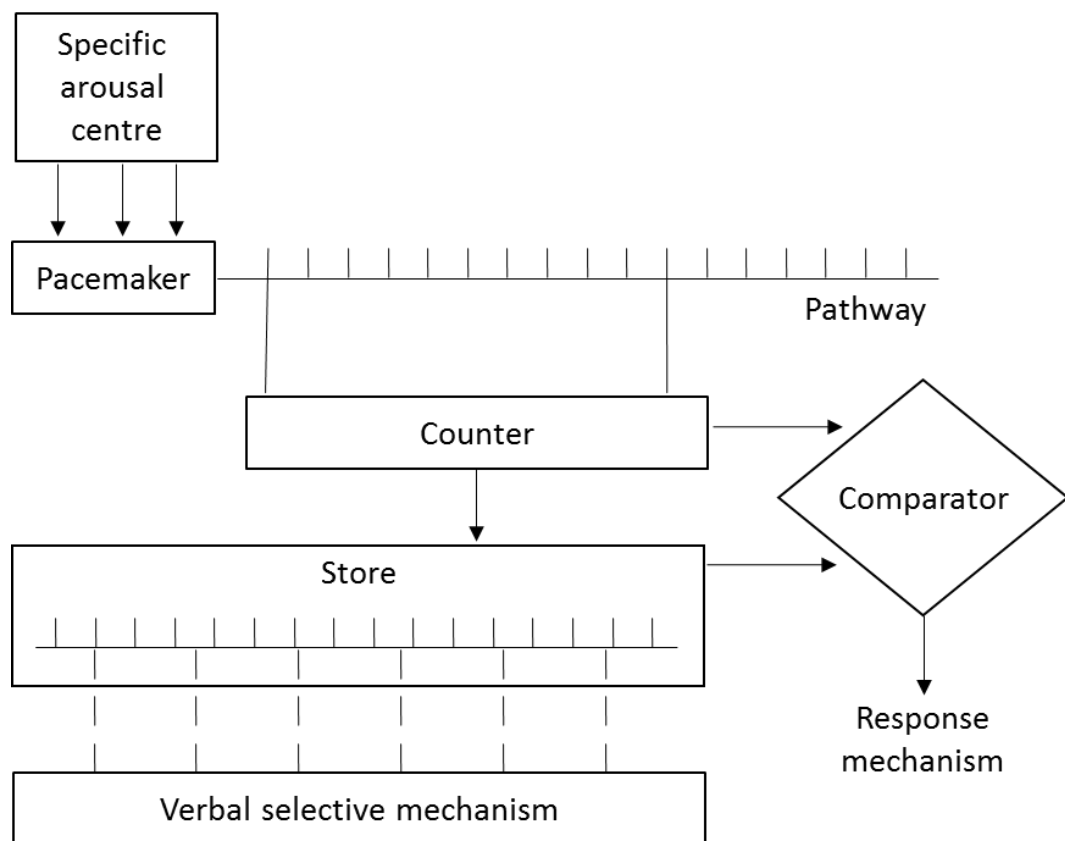


Figure 1.1. Treisman's (1963) internal clock model.

The main principles of Treisman's (1963) model (e.g., the pacemaker and the arousal effect) were adapted by later time perception models; particularly, the most popular models of timing, as the Scalar Expectancy Theory (Gibbon, Church, & Meck, 1984; see section 1.1.3, page 22) and the Striatal Beat Frequency (Matell & Meck, 2000, 2004; see section 1.2, page 35), have adopted the scalar variance properties of time.

### 1.1.2 – Principles of scalar timing

Human and animal timing conforms to two principles to produce what is commonly referred to as scalar timing: 1) mean accuracy and 2) scalar variability (or Weber's law) (Lejeune & Wearden, 1991, 2006; Wearden & Lejeune, 2008). Mean accuracy refers to the observation that timing behaviour linearly and accurately varies with the to-be-timed interval; longer intervals are associated with longer time representations (e.g., estimates) that are, on average, accurate. The second principle, scalar variability, or Weber's law, refers to the observation that the variability of time representations (i.e., the standard deviation) is a constant proportion of the mean (i.e., standard deviation/mean = C, where C is a constant). This constant proportion is often referred to as the *coefficient of variation* or a Weber-fraction like measure of time sensitivity.

Conformity to Weber's law can also be demonstrated through the property of superimposition in which time responses for different duration ranges superimpose when plotted on the same relative scale (Wearden, 1991). Superimposition can be tested in the temporal generalisation task, which involves indicating whether a series of comparison intervals are similar to a previously learnt standard interval using a 'Yes' or 'No' response option (for a complete description of the task see section 7.1.1.2, page 147). When the proportion of 'Yes' responses are plotted against comparison duration in a relative scale, that is, as proportions of the standard (Figure 1.2), the graph shows a reverse 'V' shape with the peak of 'Yes' responses at the standard duration. Superimposition can be observed by plotting data obtained from

two different duration ranges (e.g., 200-800ms and 320-1280ms) on the same relative scale, resulting in overlapping psychophysical functions (Figure 1.2).

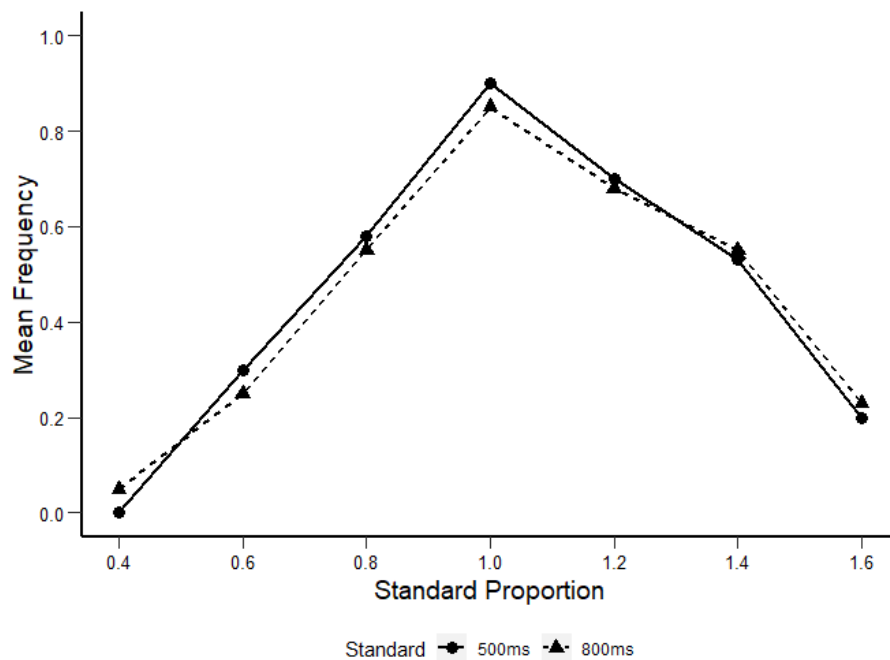


Figure 1.2. Simulated data of a temporal generalisation task using a standard of 500ms and range of comparison between 200ms to 800ms (solid line) and using a standard of 800ms and range of comparison between 320ms to 1280ms (dotted line).

Mean accuracy and scalar variability have been demonstrated in timing in both animals (Lejeune & Wearden, 1991, 2006; Maricq, Roberts, & Church, 1981) and humans (Wearden & Lejeune, 2008). However, conformity to Weber's law, although consistently observed in temporal generalisation and bisection tasks (Ortega & López, 2008; Wearden, 1992), is often violated in verbal estimation tasks (Wearden, 2015a). In verbal estimation, participants are required to provide a numerical estimate (generally in seconds or milliseconds) of the duration of a stimulus (e.g., the length of the sounding of a tone) immediately after its occurrence. Analysis of mean verbal estimates shows that variability is not a constant proportion of the mean and is typically greater at shorter durations than at longer durations. The coefficient of

variation is not therefore constant and violates the principle of scalar variability. It is presently unclear why Weber's law is violated in verbal estimation tasks, however, this may be a product of the quantization process that is unique to the verbal estimation tasks (for discussion on quantization see Wearden, 2015a and section 1.1.3.3, page 31).

### *1.1.3 – Scalar Expectancy Theory*

Gibbon et al. (1984) developed Scalar Expectancy Theory based on the scalar principles of timing. The model was originally conceived to explain animal timing and then modified by Wearden (1992) to accommodate human timing (see section 1.1.3.3, page 31). Gibbon et al. (1984) included three types of components in the model: (I) the pacemaker-accumulator clock, (II) memory components and (III) the decision component (Figure 1.3). The pacemaker-accumulator clock produces the 'raw material' enabling the perception of duration. It comprises of a pacemaker and an accumulator, which are connected via a switch. The pacemaker constantly produces pulses. When a to-be-timed event occurs, the switch between the pacemaker and the accumulator closes allowing pulses to enter into the accumulator. The amount of accumulated output forms the representation of duration. The contents of the accumulator then enter short-term memory (STM) where they remain if they are only required for a single trial. Representations that are valid for multiple trials are transferred to reference memory. To produce behavioural output the contents of reference memory and STM are compared.

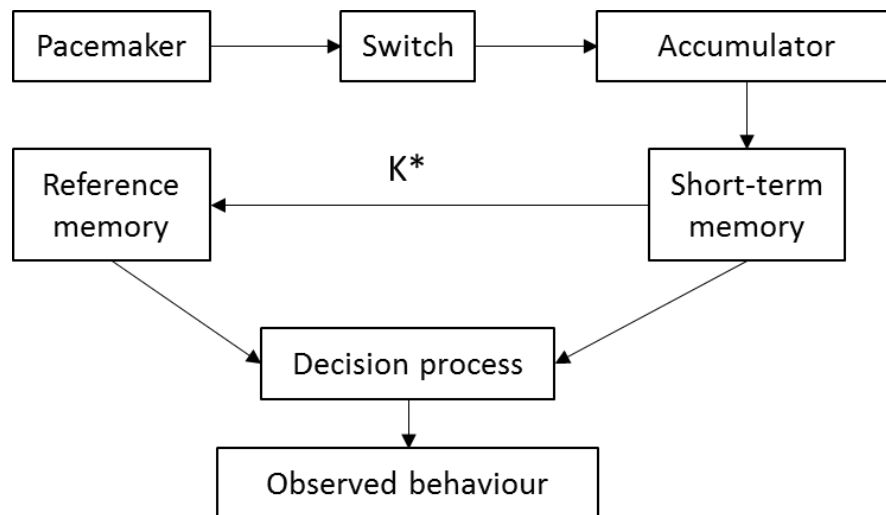


Figure 1.3. The Scalar Expectancy Theory model (Gibbon et al., 1984).

### 1.1.3.1 – Pacemaker-accumulator component

#### 1.1.3.1.1 – Pacemaker

Duration is initially encoded by the pacemaker, which generates the pulses that represent elapsed time. Pacemaker is conceived as a Poisson emitter: the interval between pulses is random but they are generated at an averagely constant rate (Gibbon, 1992; Wearden, 1999).

The rate at which the pacemaker emits output is affected by a number of factors, for example dopamine levels (Maricq et al., 1981) and physiological arousal (Mella, Conty, & Pouthas, 2011). The effect of the neurotransmitter dopamine (DA) on pacemaker rate was first demonstrated in drug manipulation studies of animal timing (e.g., Meck, 1983) and has been replicated many times using different drugs (Abner, Edwards, Douglas, & Brunner, 2001; Buhusi & Meck, 2002) and different tasks (Drew, Fairhurst, Malapani, Horvitz, & Balsam, 2003; Miller, McAuley, & Pang, 2006). DA agonists (e.g., methamphetamine) have been found to speed-up and DA antagonists (e.g., haloperidol) to slow-down pacemaker speed (Buhusi & Meck, 2002; Maricq et al., 1981; Meck, 1983). Moreover, time distortion magnitude generated by the DA-related drug administration is approximately linear to the dosage (Matell & Meck, 1997) and the affinity of the drug to the D2 receptors (Meck, 1986). However, it should be noted that, although DA modulation has been found to systematically

affect internal clock in mice (Abner, Edwards, Douglas, & Brunner, 2001; Buhusi & Meck, 2002; Meck, 1983, 1986), the relationship between DA and timing is less stringent in humans. For example, Wearden, Smith-Spark et al. (2008) found that healthy participants and patients with Parkinson's disease, which notably have DA deficiency (Damier, Hirsch, Agid, & Graybiel, 1999), have similar performance in timing tasks that do not involve motor responses (e.g., bisection task). This weakens the argument that DA affects internal clock speed in humans (see also Rakitin, Scarmeas, Li, Malapani & Stern, 2006).

Mathematical models of pacemaker operation suggest that changes in pacemaker speed have multiplicative effects on perceived duration, that is, the effects are greater at longer durations than at shorter durations (Maricq et al., 1981; see Figure 1.4). Multiplicative effects on perceived duration have been demonstrated in numerous behavioural studies (Droit-Volet, Tourret, & Wearden, 2004; Gil & Droit-Volet, 2012; Mella et al., 2011; Smith, McIver, Di Nella, & Crease, 2011). It is also often argued the presence of a multiplicative effect of a manipulation as evidence for a change of pacemaker speed, rather than attention or memory function change (Maricq et al., 1981; Wearden, 2015a). However, this is disputed by studies showing that it is possible to produce multiplicative differences in timed behaviour in circumstances in which pacemaker speed change was impossible (Matthews, 2011).



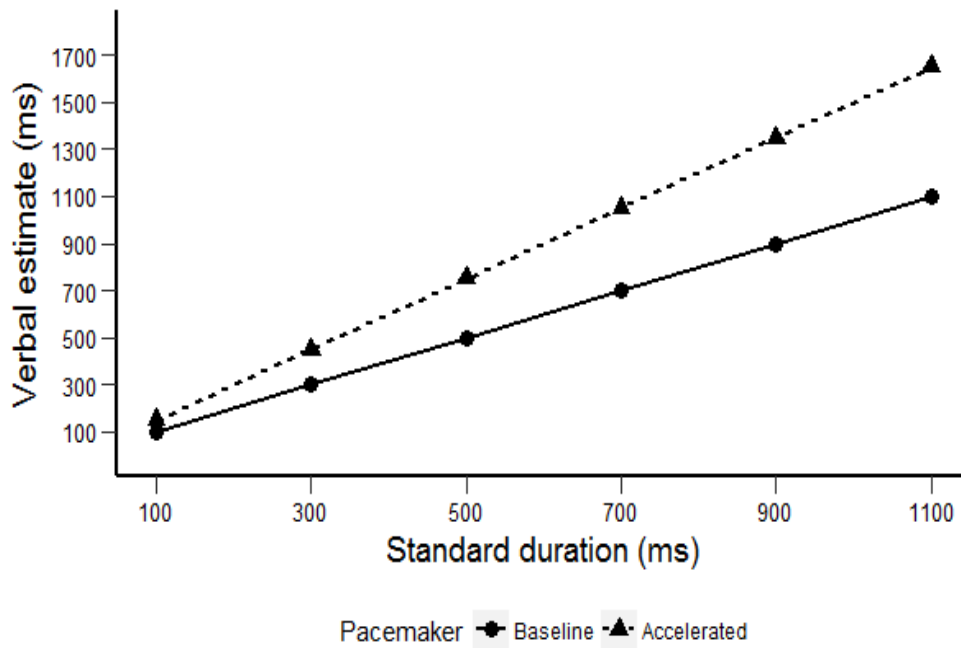


Figure 1.4. Simulated data of two verbal estimation tasks with a regular pacemaker (solid line) and an accelerated pacemaker (dotted line). Note that the distance between the two lines (i.e., the time distortion caused by the pacemaker acceleration) increases with stimulus duration.

Predominantly, multiplicative effects on perceived duration in line with changes in pacemaker speed have been shown when the arousal level of the perceiver was modulated (see section 2.3.1 and 3.3.1, page 47 and 67). However, multiplicative effects on perceived duration have been found also when arousal was not involved. For example, the duration of auditory stimuli is usually overestimated compared to the duration of visual stimuli and it has been argued that this difference in perceived duration is due to the pacemaker being faster during the encoding of auditory stimuli than during the encoding of visual stimuli (Wearden, Edwards, Fakhri & Percival, 1998). Similarly, the pacemaker is believed to be faster during the encoding of filled intervals, which are judged to last for longer than unfilled intervals when pacemaker is believed to be slower (Wearden, Norton, Martin & Montford-Bebb, 2007). Finally, it has been argued that also click-trains increase pacemaker speed lengthening the perceived duration of concurrent stimuli (Penton-Voak, Edwards, Percival & Wearden, 1996).

Previous studies have expressed the overestimation of the duration of click-train related stimuli as an increase of the arousal level of the perceiver. However, there is no evidence to support that click-trains increase arousal, suggesting that factors other than arousal could modulate the pacemaker speed.

#### *1.1.3.1.2 – Switch*

The switch was originally conceived as an on-off system, connecting pacemaker and accumulator at the onset of the stimulus and decoupling them at the offset of the to-be-timed event (Gibbon et al., 1984). When switch is closed, the pulses are allowed to move into the accumulator; in contrast, when switch is open transfer ceases.

The model predicts that the attention captured by the to-be-timed-stimulus affects the switch closure at stimulus onset, with greater attention dedicated to the stimulus associated with faster switch closure, resulting in more accumulation and longer perceived duration. Conversely, when attention is not orientated to the start of the to-be-timed event, switch closure latency increased resulting in less accumulation and a shorter perceived duration. Switch latency effects such as these result in additive effects on perceived duration, that is, time distortion due to change in switch closure are thought to be the same across stimuli durations (Wearden, 1999). In other words, faster switch closure causes the same time distortion independently of the interval duration (Figure 1.5).

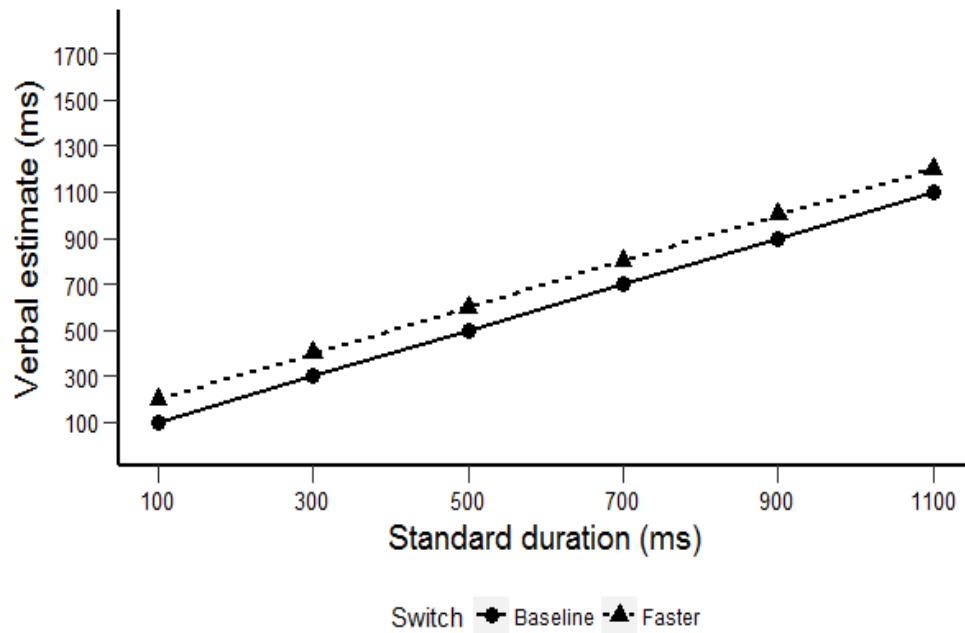


Figure 1.5. Simulated data of two verbal estimation tasks with a regular switch closure (solid line) and an accelerated switch closure (dotted line). Note that the distance between the two lines (i.e., the time distortion caused by the faster switch closure at stimulus onset) remains constant.

Zakay and Block (1995) modified the original switch in SET with an attentional gate, which can be open with different degrees depending by the amount of attention dedicated to time. Depending on this degree, different proportion of pulses are allowed to have access into the accumulator affecting the final perceived duration of the event. The more the attention dedicated, the wider the gate, the greater the number of pulses that reach the accumulator and the longer the perceived duration of the event. The attentional gate model therefore contrasted the on-off functioning of the switch, which implied an all or nothing effect of attention on time perception, introducing a gate that takes into account the variability of attentional resources dedicated to the passage of time.

This modification was supported by studies that found a relationship between time distortion and amount of attention dedicated to time; for example, during a timing task, participants reported shorter estimates when they had to concomitantly

complete a difficult object recognition task rather than a simpler version (Hicks, Miller, & Kinsbourne, 1976). The attentional gate provides an explanation for these results, suggesting that the increased difficulty of object recognition task implies a reduction of attentional resources dedicated to the timing task, leading the attentional gate to allow less pulses into the accumulator compared to the pulses accumulated with the simple recognition task. The hallmark of a change in gate openness is the multiplicative effect (Figure 1.4); the gate, in fact, allows a *proportion* of pulses to enter into the accumulator, proportion that depends on the duration of the to-be-timed event. Although the attentional gate is widely accepted it has been criticised by Lejeune (1998) who suggested that the functions attributed to the attentional gate can be ascribed to an attentional switch for parsimony.

#### 1.1.3.1.3 – Accumulator

The accumulator was conceived as a container that stores the pulses occurring during the to-be-timed event, that is, when the switch is closed and the pacemaker is connected to the accumulator (Gibbon et al., 1984). The short-term memory then retrieves the stored pulses from the accumulator for the following memory and decision components.

Because of its function of simple container, the accumulator was not conceived to be affected by external factors and it was not object of investigation. However, accumulator has been suggested to be shared between the processing of time and the processing of other dimensions that require cumulative properties (e.g., counting; Meck & Church, 1983). This hypothesis has been supported by studies showing common neural activity of the parietal cortex in time, number and spatial processes (Buetti & Walsh, 2009), and led Buetti and Walsh (2009) to outline A Theory Of Magnitude (ATOM). However, it is still under debate whether this integration between time and other magnitude dimensions is in the accumulator component or whether they are linked in other components such as attention or memory (for discussion see Cappelletti, Freeman, & Cipolotti, 2009).

### 1.1.3.2 – Memory components

Meck (1983) demonstrated the existence of separate clock and memory processes by comparing the effect of dopaminergic drugs (haloperidol and methamphetamine) with the effects of vasopressin, oxytocin, physostigmine and atropine in a temporal discrimination task. This procedure consisted of two phases: firstly rats were trained to press two levers that were associated to a 'short' (2 s) and a 'long' (8 s) standard duration (training phase), and then they categorized a series of comparison durations as 'short' or 'long' by pressing the respective lever (testing phase). Different rats were administered with one drug in the training or testing phase while saline was administered in the other phase. Methamphetamine induced right and left shift of the point of subjective equality (PSE, point at which 50% of responses are 'long'), when it was administered during the training and testing phase, respectively (Figure 1.6). This suggests that methamphetamine hastened the pacemaker, leading to longer perceived duration of the standard durations when administered in the training phase and leading to longer perceived duration of the comparison durations when administered in the testing phase. Haloperidol had opposite effect on PSE compared to methamphetamine, suggesting that it reduced pacemaker speed resulting in shorter perceived durations.

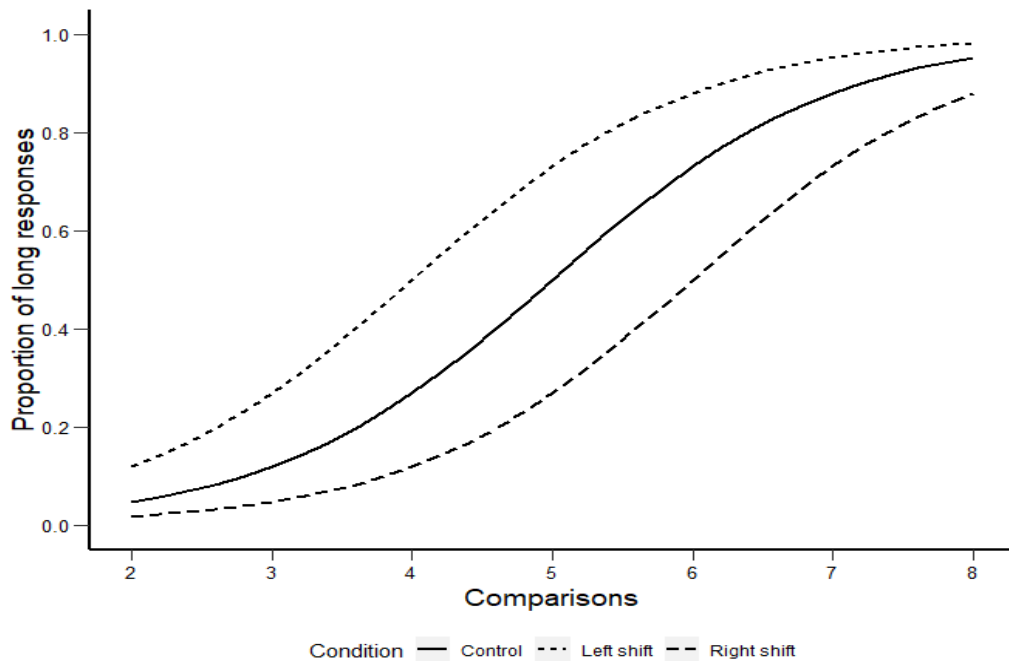


Figure 1.6. Simulated data of a temporal discrimination task in animal and of a temporal bisection task in humans under normal circumstances (solid line), with a left shift (short dotted line) and with a right shift (long dotted line).

In contrast, vasopressin, oxytocin or physostigmine shifted PSE to the left when administered in the training phase, but PSE remained unaffected when these drugs were administered during the testing phase. This suggested that these drugs affected the memorisation process rather than the temporal encoding. Meck (1983) expressed this suggesting that, during the transfer from the STM to the reference memory, the standard representations are multiplied by the transformer  $K^*$ , which has a mean of 1 and a Gaussian distribution variability when saline was administered. However, atropine administration increased the mean of  $K^*$  (i.e.,  $K^* > 1$ ) and the standard representations were longer than the actual duration of the standards. In contrast, vasopressin, oxytocin and physostigmine administration decreased the mean of  $K^*$  (i.e.,  $K^* < 1$ ) and the standard representations were shorter than their actual durations.

Reference memory is also hypothesised as a source of the scalar variance. When the stimulus representation is multiplied by the transformer  $K^*$ , the duration stored into the STM is transformed into a Gaussian distribution of values. In normal situation (i.e., when  $K^* = 1$ ), the mean of this distribution corresponds to the STM representation, meanwhile the variability corresponds to the coefficient of variation. Any value of this distribution can be recalled when the interval in the reference memory is needed for the task, causing the variability of the timing responses. Although there is evidence supporting reference memory's role in scalar variance, tasks which eliminate the use of reference memory, known as episodic tasks, still produce behaviour exhibiting scalar variance (e.g., Wearden & Bray, 2001). This suggests that the reference memory is not the only source of the scalar variance, which potentially is any number of other components of the model (e.g., pacemaker).

#### 1.1.3.3 – Decision component

Originally, Church and Gibbon (1982) proposed a model to explain decision rule employed by rats in a fixed interval task, the animal analogue of the temporal generalisation task (see section 1.1.2, page 20). Rats learnt a standard interval through reinforcement (i.e., food pellets) and then were rewarded if they recognized the same interval in a series of other interval presentations (comparisons) in a yes or no option. Church and Gibbon (1982) suggested that rats identified the comparison stimulus as the standard following the rule:

$$\frac{|s^* - t|}{s^*} < b^* \quad \text{Equation 1}$$

where  $t$  is the comparison duration stored in the STM,  $b^*$  is a threshold that varies from trial to trial with a coefficient of variation of  $x$  and  $s^*$  is the standard duration stored in the reference memory as a Gaussian distribution with coefficient of variation  $c$ , where scalar variance arises. In words, the rat would identify the comparison stimulus as the standard one when the absolute difference between the Gaussian representation of the standard and the comparison, divided by the

Gaussian representation of the standard, is lower than a threshold that varies from trial to trial.

The rule proposed by Church and Gibbon (1982), however, did not fit human behaviour on the human analogue of the task (temporal generalisation). The main difference between rats and human timing behaviour is due to the symmetry of the responses: rats confuse the comparison duration as the standard with equal probability whether the comparison is actually longer or shorter (symmetrical pattern). In contrast, humans produce right skewed gradients in which YES responses to durations longer than the standard are more common than YES responses to durations shorter than the standard (Figure 1.7). Wearden (1992) introduced a modified Church and Gibbon (MCG) model, suggesting that humans identify the comparison stimulus as the standard following the rule:

$$\frac{|s^* - t|}{t} < b^* \quad \text{Equation 2}$$

where the terms are as in Equation 1. Here, the comparison stimulus is identified as the standard when the absolute difference between the Gaussian representation of the standard and the comparison, divided by the comparison, is lower than a threshold that varies from trial to trial. In the MCG model, although the absolute difference between two comparisons and the standard may be the same, the response is more likely to be yes when comparison is greater than the standard rather than shorter, resulting the right skewed gradients observed from human subjects (Figure 1.7).



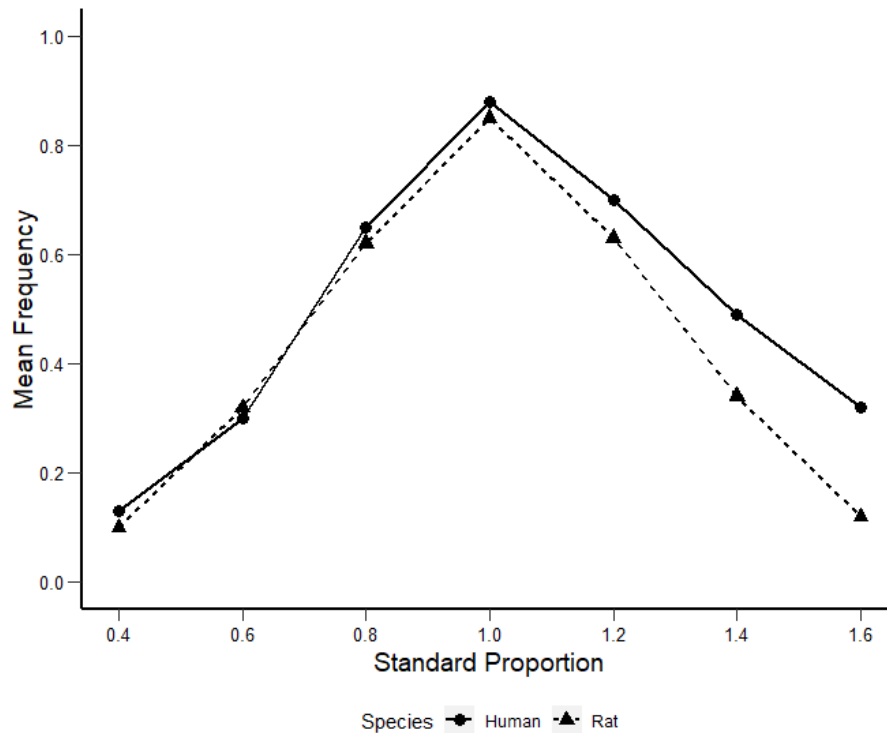


Figure 1.7. Simulated data of a temporal generalisation task completed by human participants (solid line) and rats (dotted line). Note the symmetry of the ‘YES’ responses in rats and the right skewed gradient in human participants.

The same structure model of Equations 1 and 2 has been also proposed to address the human behaviour during other temporal tasks, such as episodic temporal generalisation tasks (Wearden, 2004), temporal bisection tasks (Wearden & Ferrara, 1996) and temporal reproduction tasks (Ogden, Wearden, & Montgomery, 2014). In all the decision rules, a threshold is compared to a difference between two durations that is divided by one of the two durations or by their mean. The verbal estimation task has been found more difficult to model, however. Data from a verbal estimation task has been found to violate the scalar principles (see section 1.1.2, page 20) and presents the problem of ‘quantization’: when participants estimate the duration of an event in milliseconds, they almost always round their estimates adding ‘00’ or ‘50’ at the end. Participants therefore use only a fraction of the possible values. Wearden (2015a) proposed an ‘attractor model’ to address human behaviour during a verbal estimation task, which suggests that the used values are attractors that differ in

weight and distance to the presented stimulus and they compete for priority as output value. The priority is given to the value with greater weight and closer to the presented stimulus. Whilst this model provided an accurate fit for the data in Wearden (2015a), it is yet to be widely tested.

#### 1.1.3.4 – Strengths and limitations of the Scalar Expectancy Theory

SET has received recognition in the timing field, being able to accurately describe a great variety of timing behaviours (Church & Gibbon, 1982; Wearden, 1992, 2004; Wearden & Ferrara, 1996). Nevertheless, it has also received numerous criticisms. The principles of scalar timing have been criticised, in part, because these two principles can occur in contexts other than time perception (Wearden, 1991); scalar principles have been demonstrated when animals and humans discriminate stimuli by their numerosity (Emmerton & Renner, 2006), visual contrast (Gorea & Sagi, 2001) and brightness (Treisman, 1964). In part, the principles of scalar timing have been criticised because timing behaviour can violate the scalar principles (Lejeune & Wearden, 1991; Wearden & Lejeune, 2008). The second principle has been found to be violated when short durations (< 100ms) are used (Wearden & Lejeune, 2008), when participants were exposed to an extensive practice (Kristofferson, 1980), and when more versions of the same task with different difficulties are compared (Ferrara, Lejeune, & Wearden, 1997). Additionally, 'classical' timing procedures (i.e., verbal estimation task and (re)production tasks) frequently violate both scalar principles of timing. However, it should be noted that the absence of a scalar property in timing behaviour does not necessarily imply the absence of underlying scalar representation; there is the possibility that the motor/decision response is modulated, masking the scalar properties.

SET has been also accused of being so flexible in its components (particularly the decision process) that it can always explain the data independently of how they look, by simply choosing appropriate decision processes (Wearden, 1999). This makes the model impossible to falsify because any experimental design cannot definitively

prove that the model is erroneous (Staddon & Higa, 1999). In fact, previous studies used computer modelling and manipulated each SET component separately to identify their operating parameters (Jones & Wearden, 2004; Wearden & Grindrod, 2003). However, thanks to the flexibility of the parameters, a plausible mathematical model can be always found to fit the data, even if this requires adding bias responses (Droit-Volet, Clément, & Wearden, 2001). Therefore, “no data can disprove, or even modify, SET” as stated by Jones and Wearden (2003, p. 322).

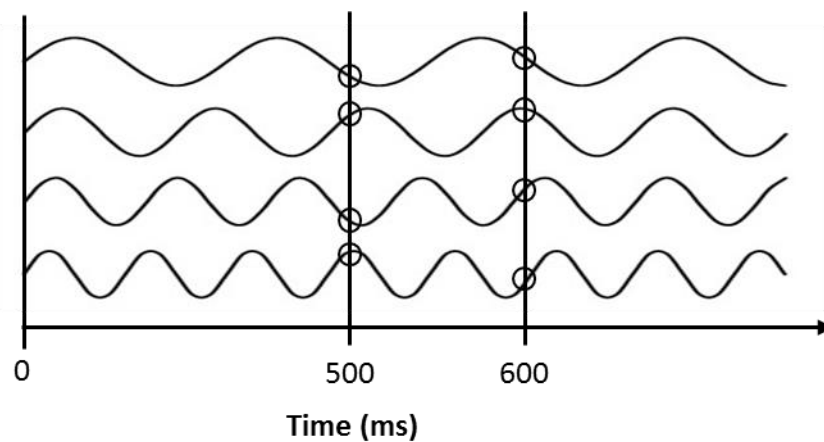
Finally, SET (together with the other internal clock models) has been criticised to be a cognitive model that lacks a neurobiological counterpart (Matell & Meck, 2004). In fact, SET has been said to lack neurobiological plausibility, because the components do not reflect the neural functions of the brain. New neurobiological models therefore have been developed to integrate timing behaviour and neural correlates. Among these, the Striatal Beat Frequency (Matell & Meck, 2000, 2004) has received great attention and popularity.

## **1.2 – The Striatal Beat Frequency Model**

Matell and Meck (2000, 2004) developed the Striatal Beat Frequency (SBF) model which aimed to explain the neurobiological basis of time perception. A number of different brain areas have been identified as potential sources of the neural timing mechanism: cerebellum (Breukelaar & Dalrymple-Alford, 1999), hippocampus (Meck, Church, & Olton, 1984), putamen (Coull, Vidal, Nazarian, & Macar, 2004), pre-frontal cortex (Harrington, Haaland, & Knight, 1998), pre-supplementary motor cortex (Coull, 2004), basal ganglia (Matell, Meck, & Nicolelis, 2003; Meck, 2006) and thalamus (Teki, Grube, Kumar, & Griffiths, 2011). Collectively, studies show that damage to these brain areas is associated with impaired timing behaviour and their neural activity is increased during timing tasks. Particularly consistent is the evidence indicating that correct functioning of the basal ganglia is necessary for timing (Matell & Meck, 2004; Matell et al., 2003; Meck, 2006). The basal ganglia is a section of the forebrain that includes the striatum, globus pallidus, subthalamic nucleus and

substantia nigra pars compacta (SNPC), which have extensive connections with the thalamus and cerebral cortex. Brain damage in this area prevents from temporal perception and production (Matell & Meck, 2004; Meck, 2006) and activity of the striatum mirrors the time behavioural patterns in a peak interval task (Matell et al., 2003).

Matell and Meck (2000, 2004) developed SBF model integrating the neurobiological evidence of timing with the Beat Frequency model (Miall, 1989), which proposed that intervals are encoded and represented by patterns of oscillators. Miall (1989) proposed that the encoding of the stimulus duration begins at stimulus onset by resetting a series of oscillators that soon lose synchrony due to their different frequency speed, leading to a new, unique oscillatory pattern at every moment. The encoding of the stimulus duration ends at stimulus offset by reading the oscillatory pattern, which is associated to a unique time interval (Figure 1.8).



*Figure 1.8. Visual representation of four oscillators with different speeds starting together at stimulus onset (0) and leading to different oscillatory patterns for a stimulus of 500ms and 600ms.*

SBF advanced Miall's model by making explicit links between the oscillator process and neural structures/networks previously associated with timing. The model integrates the neurobiological evidence that supports the role of dopamine, cortical oscillations and basal ganglia in time perception. SBF describes a neural network

responsible for timing, referred to as the cortico-striato-thalamic loop (Figure 1.9). Within it, the oscillators proposed by Miall (1989) have been replaced by the oscillating neural activity of the cortex, that is, the oscillations naturally generated by the cortical activity (e.g., alpha waves), which have been suggested to be associated to timing behaviour (Bartolo, Prado, & Merchant, 2014; Rohenkohl & Nobre, 2011). The rate at which the cortical activity oscillates is influenced by the DA levels, which is regulated by the thalamus; greater DA releases are associated to faster cortical oscillations, meanwhile minor DA releases are associated to slower oscillations.

As in Miall, the duration of an event is determined by resetting cortical oscillators at stimulus onset and by reading the pattern of the oscillators at stimulus offset, which is detected by the striatal integrators in the striatum and forms the representation of duration. Critically, the model predicts that the speed of the cortical oscillations is associated with time distortions: at the end of a time interval (e.g., 500ms), faster oscillations reach a pattern associated to longer durations (e.g., 600ms), meanwhile slower oscillations reach a pattern associated to shorter durations (e.g., 400ms). Because DA affects the speed of cortical oscillations, this model can account for the pharmacological studies showing that DA agonists (e.g., methamphetamine and cocaine) lengthen time perception meanwhile DA antagonists (e.g., haloperidol) shorten time perception in animals (Buhusi & Meck, 2002; Maricq et al., 1981).

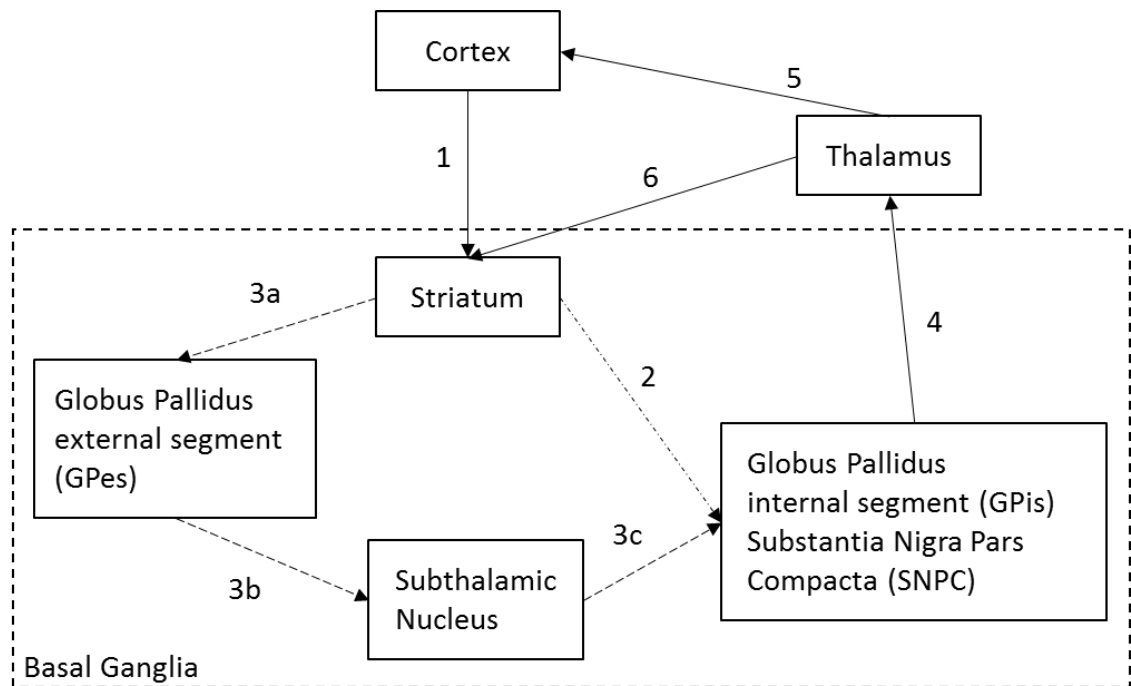


Figure 1.9. The Striatal Beat Frequency model (Matell & Meck, 2004) readapted for human timing showing the connections between cortex, basal ganglia and thalamus (continuous arrows), the excitatory direct pathway (short dotted arrow) and the inhibitory indirect pathway (long dotted arrows).

### 1.2.1 – The cortico-striato-thalamic loop

At stimulus onset, the cortical oscillators and striatal integrators are reset by a burst of DA release of the thalamus. During stimulus presentation, cortical oscillators lose synchrony, generating a unique oscillatory pattern every moment (as in Miall, 1989; Figure 1.8). The cortical oscillatory activity is detected by the medium spiny neurons of the striatum (1), each of which is sensitive to a specific pattern of oscillators (i.e., a specific time duration). The striatum is connected to the thalamus through direct and indirect pathways, which have opposite effects. Through the direct pathway (2), striatum activity activates the Globus Pallidus internal segment (GPis) and Substantia Nigra Pars Compacta (SNPC). Through the indirect pathway (3), striatum activity elicits the Globus Pallidus external segment (GPes, 3a), which elicits the Subthalamic Nucleus (3b), which *inhibits* GPis and SNPC (3c), contrasting the direct pathway's effect. The direct pathway is predominant during stimulus

presentation, leading in activation of GPs and SNPC that have an inhibitory effect on the thalamus (4), that is, the direct pathway prevents thalamus from releasing burst of DA into cortex (5) and striatum (6). In contrast, the indirect pathway is predominant during stimulus offset, leading in inhibition of GPs and SNPC that have an excitatory effect on the thalamus (4), that is, the indirect pathway promotes bursts of DA of the thalamus into cortex (5) and striatum (6). At stimulus offset, thalamus releases DA to the striatum (6) reinforcing the striatal integrators that are active in that moment and the associated oscillatory pattern, forming the mental representation of the stimulus duration. The mental representation is then stored in the hippocampus, where the long-term memory is located (Oprisan & Buhusi, 2014), if the stimulus duration has to be remembered; otherwise, the mental representation is immediately used for the ongoing task.

### *1.2.2 – Strengths and limitations of SBF*

Through data modelling, Matell and Meck (2004) and Oprisan and Buhusi (2014) showed that SBF model can provide a good fit for data from humans and animals. Furthermore, SBF explains scalar variance through variability of neural activity (e.g., varying dynamic threshold of the cortical activity between trials). Perhaps the most widely stated advantage is the neurobiological plausibility. However, this has some limitations; for example, the actual oscillatory activity of the cortex is more irregular than that allowed by the model (Matell & Meck, 2004) and lesions to the cortex do not lead to such a critical disruption of timing as the model would predict (Olton, 1989). Moreover, there is limited neural evidence supporting a reset of the cortical oscillators at the onset of the stimulus, without which the timing process would not be possible as described by SBF (Kononowicz & van Wassenhove, 2016). Furthermore, because the model lacks an accumulator component, SBF is not able to address the linear timing experience, which is the perception of time as an entity varying in magnitude. The accumulator in SET can easily address linear timing by indicating that the relationship between interval durations is determined by the amount of pulses generated; hence the interval where more pulses are generated is

longer than the interval with fewer pulses. In contrast, although SBF predicts that striatal neurons are related to specific oscillatory patterns and interval durations, it is not clear how the striatal neurons interact to establish the relationship of the duration they respond to.

The model also predicts the use of DA in multiple phases of the timing process: (I) DA is thought to reset cortical and striatal activity at stimulus offset, (II) regulate oscillatory speed during stimulus presentation and (III) 'save' the striatal activity at stimulus offset. However, the model does not indicate how DA achieves these multiple tasks. Finally, the model cannot take in account the ability of simultaneous temporal processing (Matell & Meck, 2004).

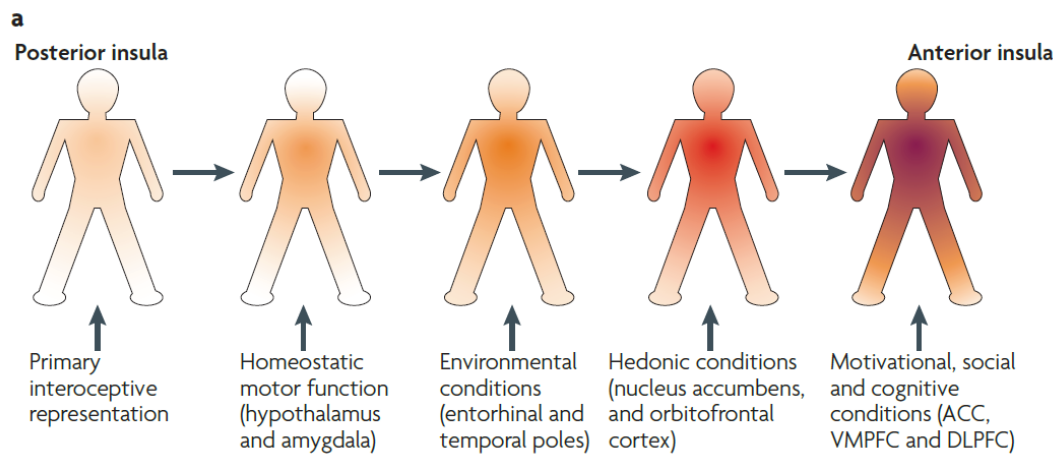
### **1.3 – Craig's model of awareness**

Craig (2002, 2009b) proposed a neurobiological model of time perception in which temporal processing is integrated into subjective awareness, where awareness is defined as the sum of the representation of self "*at each immediate moment (now) extended across a finite period of present time (the specious moment)*" (Craig, 2009a, p. 1933). Given that the model considered awareness as a process that occurs across time, Craig (2009a) elaborated the model to address subjective timing.

The single representations of self that compose awareness (also called global emotional moments) are defined as the integration of salient feelings in the immediate moment. Craig (2009a) used 'salient feelings' as a broad term to indicate the personal components (emotion, sensation, cognition, etc.) that are relevant to the individual homeostasis. The sum of the salient feelings in the immediate moment forms the global emotional moment. The model suggests that the anterior insular cortex (AIC) generates the global emotional moment after a series of stages in the insula with a posterior-to-mid-to-anterior progression, through which insula integrates interoception, situational context, hedonic impulses and motivational, social and cognitive feelings (Figure 1.10). Craig (2009b) suggested that the repetition



of the posterior-to-mid-to-anterior circuit leads to a meta-representation of the global emotional moment across a finite period of time, resulting in awareness.

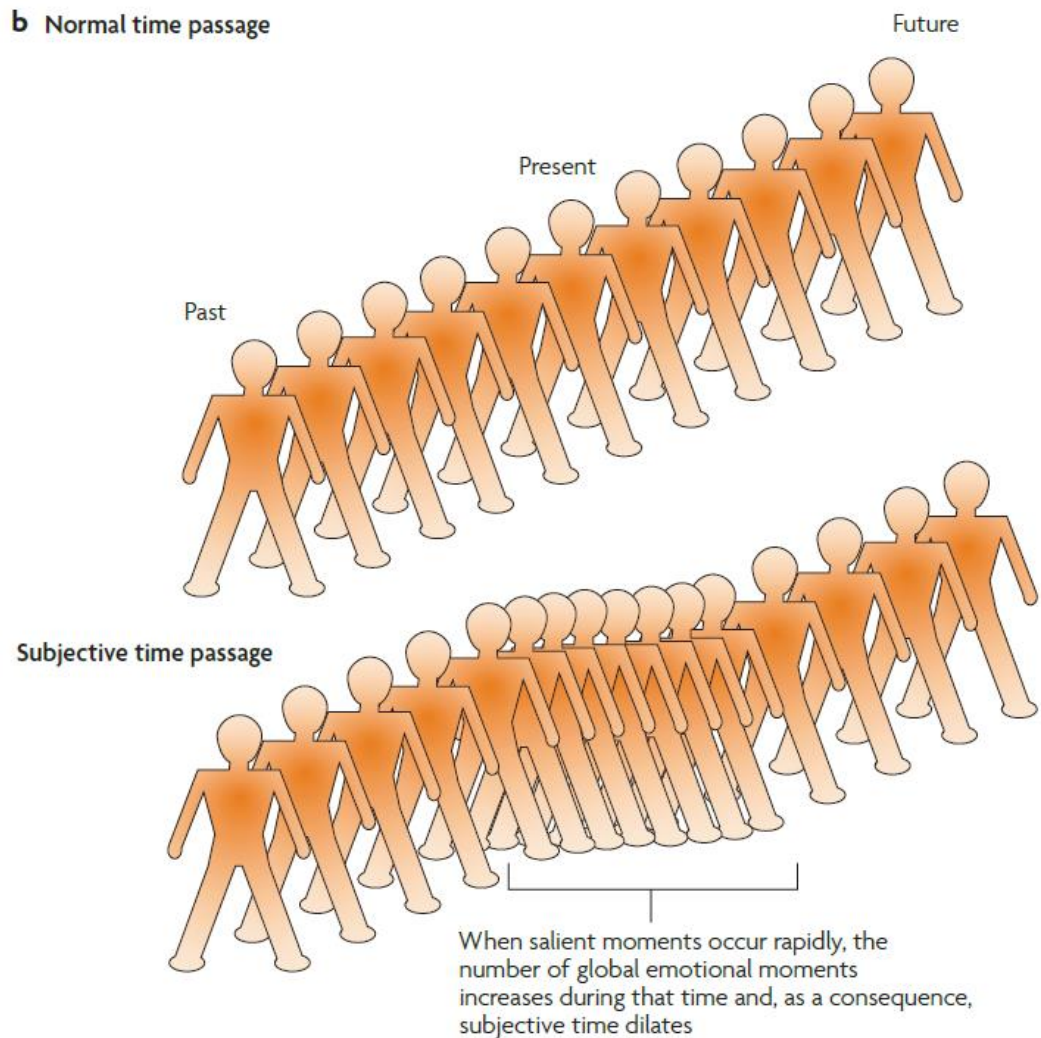


*Figure 1.10. The posterior-to-mid-to-anterior progression of the global emotional moment expected by the model of awareness (Craig, 2009b).*

### 1.3.1 – Adaptation of the model in time perception

With awareness being postulated as a process that occurs across time, Craig (2009a) suggested that perceived duration is linearly related to the number of generated global emotional moments, which varies depending on the AIC activity. AIC activity is also associated with homeostasis of the autonomic nervous system (ANS) (Craig, 2002; Wittmann, 2009). Right activation of the AIC is associated with increased activity of the sympathetic nervous system (SNS), which is the branch of the ANS that modulates body homeostasis in stressful situations. Whereas, left activation of the AIC is associated with increased activity of the parasympathetic nervous system (PSNS), which is the branch of the ANS that modulates body homeostasis in resting situations (see section 2.1, page 44). Craig (2009a) suggested that right AIC activation due to SNS activity leads to greater production of global emotional moments, which results in longer perceived duration (bottom part Figure 1.11). In contrast, left AIC activation due to PSNS activity increases leads to reduced production of global emotional moments, which results in shorter perceived

duration. This suggestion is supported by studies showing AIC activation during temporal perception and homeostasis processing simultaneously (Craig, 2009a).



*Figure 1.11. The generation of global emotional moments across time during left AIC activation (top part) and during right AIC activation (bottom part) (Craig, 2009b).*

### *1.3.2 – Strengths and limitations of the model of awareness*

The model of awareness (Craig, 2002, 2009b) has received recognition due to its integration of behavioural and neurological evidences. However, the original aim of the model was to address awareness rather than subjective timing (Craig, 2009a). Therefore, whilst the model is able to predict the directional effect that external

situations have on perceived durations, the model lacks mathematical modelling to compare expected and experimental results, contrarily to SET and SBF. Moreover, Craig (2009a) acknowledges that the model cannot explain the phenomenon 'time flies when having fun': the shortening effect that pleasant and engaging situations have on subjective timing. The model of awareness would predict that pleasant situation would increase the generation of global emotional moments increasing right AIC activation, resulting in lengthening effect as for dangerous situations.

## Chapter 2

### The effect of emotion on time perception

#### 2.1 – Emotions

Emotions are automatic patterns of behavioural and physiological responses that have developed to adapt body and mind to the current state of the external world, so as to provide the best survival and reproductive chances (James, 1884). Emotions therefore involve changes of physiological and cognitive states (Vuilleumier, 2005). Physiological changes occur through the autonomic nervous system (ANS), the division of the peripheral nervous system that automatically regulates the activity of the internal organs (e.g., the heart) adapting the body to the external context (Robertson, Low & Polinsky, 2011). The ANS consists of two branches, the sympathetic nervous system (SNS), which is dominant during stress and fight/flight responding, and the parasympathetic nervous system (PSNS), which is dominant during relaxation (Robertson et al., 2011). Emotional experience is associated with SNS dominance over PSNS, resulting in increases of heart rate, peripheral vasoconstriction (Mendes, 2009; Sztajzel, 2004) and electrical activity of the skin (Critchley, 2002). Particularly, the emotions related to survival (e.g., fear) induce greater SNS activity compared to other emotions (e.g., happiness) (LeDoux, 2012).

Emotions have also privileged routes in cognitive and neural processes. Early sensory processes (e.g., P1 and N1 event-related potentials) have greater amplitude when the stimuli are emotional than neutral (Grandjean et al., 2005; Vuilleumier, Armony, Driver, & Dolan, 2001). Emotional stimuli also capture more attention than non-emotional stimuli leading to enhanced detection of emotion stimuli (Ohman, Flykt, & Esteves, 2001) and impaired performance on tasks performed simultaneous to emotion processing (Richards & Blanchette, 2004). Memory and executive functions are also impacted by emotions, with emotional stimuli more likely to be remembered (Kensinger & Corkin, 2003; Lindström & Bohlin, 2011) and less likely to inhibit automatic responses (Verbruggen & De Houwer, 2007) than neutral stimuli. Critically however, although there is evidence that all emotional states can affect

cognitive processing, the greatest effects appear to occur when the emotion induced is survival-related (Lake, LaBar, & Meck, 2016; Vuilleumier, 2005).

## **2.2 – The effects of emotions on timing**

“Time flies when having fun” and “time drags when bored” are common statements that describe the ability of emotions to distort human time experience in daily life. However, these expressions frequently are passage of time judgments, that is, judgments of how fast or slow the time seems to pass compared to normal. Passage of time judgments are not related to duration judgment (i.e., judgments of how long an interval lasts), and to date it is still unclear how passage of time judgments are related to the underlying mechanisms of timing. For example, the feeling of “time flying” has been associated with longer perceived duration in some studies (Gil & Droit-Volet, 2011) and with shorter perceived duration in others (Droit-Volet, Bigand, Ramos, & Bueno, 2010). It is therefore difficult to interpret passage of time judgments to address the effects of emotions on time perception (see Wearden, 2015b for review).

In the last few decades, the effects of emotion on duration judgments have been intensively investigated. Studies consistently show that subjective time is distorted by emotional experience (e.g., Bar-Haim, Kerem, Lamy, & Zakay, 2010; Campbell & Bryant, 2007; Droit-Volet & Gil, 2009; Droit-Volet, Brunot, & Niedenthal, 2004; Stetson, Fiesta, & Eagleman, 2007; Yamada & Kawabe, 2011). The most consistently reported effect is that negatively valenced stimuli are perceived as lasting for longer than neutral stimuli presented for the same duration (Lake et al., 2016). The relative overestimation of threatening stimuli appears consistent across modalities. Static visual stimuli, negatively valenced IAPS images (Angrilli, Cherubini, Pavese, & Manfredini, 1997; Droit-Volet & Meck, 2007; Gil & Droit-Volet, 2012; Grommet et al., 2011), angry faces (Doi & Shinohara, 2009; Thayer & Schiff, 1975; Tipples, 2008), taboo words (Tipples, 2010), life-threatening situations (Campbell & Bryant, 2007; Castellà, Cuello, & Sanz, 2017) are all perceived as lasting for longer than their neutral counterparts.

Comparable effects are observed for negatively valenced, dynamic visual stimuli such as video-clips (Droit-Volet, Fayolle, & Gil, 2011). Similarly, the duration of emotional auditory stimuli are overestimated relative to neutral stimuli (Noulhiane, Mella, Samson, Ragot, & Pouthas, 2007). Furthermore, individual differences influence the extent to which emotion distorts time; the magnitude of the overestimation of negatively valenced stimuli correlates positively with the empathy quotient of the participant (Mondillon, Niedenthal, Gil, & Droit-Volet, 2007) and distortions are attenuated when embodiment is prevented (Efron, Niedenthal, Gil, & Droit-Volet, 2006).

The effect of threatening stimuli on perceived duration is also broadly consistent across experimental tasks; it has been observed on temporal bisection (Droit-Volet et al., 2011; Droit-Volet, Fayolle, Lamotte, & Gil, 2013), verbal estimation (Angrilli et al., 1997), temporal generalisation (Mella et al., 2011) and temporal reproduction tasks (Angrilli et al., 1997; Bar-Haim et al., 2010). However, findings are not consistent in the temporal generalisation and reproduction tasks. For example, Gil and Droit-Volet (2011) did not find emotional distortions of performance during temporal generalisation task and suggested that temporal generalisation and reproduction tasks demand greater memory resources compared to verbal estimation and bisection tasks, which might mask the emotional effects on the 'raw' temporal encoding (Baudouin, Vanneste, Isingrini, & Pouthas, 2006; Droit-Volet & Rattat, 2007).

Whilst fear inducing negatively valenced stimuli are consistently perceived as longer than neutral stimuli, positively valenced stimuli are generally perceived as lasting for less time than neutral stimuli (Smith et al., 2011). For example, participants perceived the duration of happy music (Droit-Volet et al., 2010) and positive images (Smith et al., 2011) as shorter than neutral stimuli. Similarly, Ogden et al. (2015) found that a square presented while experiencing pleasant touch was perceived as lasting shorter than a square presented while experiencing unpleasant or no touch. Stimulus valence therefore appears critical in determining the direction of emotional

distortions to time. However, it should be noted that the shortening effect of positively valenced stimuli is not universally observed: for example, positive sounds were found to be overestimated compared to neutral sounds (Noulhiane et al., 2007) and to lengthen the perceived duration of concomitant visual stimuli (Droit-Volet, Mermillod, Cocenas-Silva, & Gil, 2010). Moreover, the magnitude of the time distortion of positive stimuli is lower compared to the magnitude of the time distortion of negative stimuli (Noulhiane et al., 2007).

Other emotional states have been less widely studied making conclusions about their effects more difficult. For example, disgust has been found to induce (i) time overestimation when using high-arousal images (e.g., mutilated corpses; Gil & Droit-Volet, 2012), (ii) no time distortion when using low-arousal images (e.g., an ashtray; Gil & Droit-Volet, 2012), and (iii) time underestimation when using images of rotten food (Gil, Rousset, & Droit-Volet, 2009). It should be noted, however, that the choice of the high-arousal disgust stimuli might be inaccurate given that mutilated bodies can also be rated as threatening (Lang & Bradley, 2007). Similarly, attractive faces showed contrasting effects on perceived duration; for example, Arantes et al. (2013) found that attractive faces were overestimated compared to neutral faces, meanwhile Ogden (2013) found no difference in perceived durations between neutral and attractive faces.

### **2.3 – Theoretical explanation of emotion effects on time**

Within the frameworks of the models of time perception discussed in Chapter 1, two hypotheses have been proposed to explain the mechanisms that lead emotions to distort time: 1) the arousal hypothesis and 2) the attention hypothesis.

#### **2.3.1 – *The arousal hypothesis***

Since emotional distortions to time were first reported, it has been theorized that emotion induced changes in arousal is the causal mechanism by which emotion

distorts duration (Thayer & Schiff, 1975). Emotion evokes widespread changes in physiological arousal (Russell & Mehrabian, 1977), which are thought to act on the mechanisms used to judge duration. The role of arousal in emotional distortions to time is supported by observations that high arousal stimuli have greater effects on timing than low arousal stimuli; for example, Gil and Droit-Volet (2012) found that low arousing sad images induced a time distortion with reduced magnitude compared to high arousal sad images. Similarly, negative stimuli are thought to lead to greater time distortion than positive stimuli (as in Noulhiane et al., 2007) because negative stimuli are more arousing (Cacioppo & Gardner, 1999).

Arousal is determinant of perceived duration in the three main models of temporal perception (SET, SBF and Craig's model of awareness; see Chapter 1). In SET, arousal affects the speed at which the pacemaker emits output. At a basic level, increases in arousal are thought to increase the output rate, leading to a greater accumulation and a longer perceived duration. In contrast, decreases in arousal are thought to decrease output rate leading to less accumulation and a shorter perceived duration (Gibbon et al., 1984).

Because arousal acts on the pacemaker, its effect on perceived duration is thought to be multiplicative, that is, greater time distortions occur with longer stimulus durations (Maricq et al., 1981; see section 1.1.3.1.1, page 23). A series of studies investigated the arousal hypothesis by testing the multiplicative effect with emotional stimuli: if emotional stimuli induce time distortion because they are more arousing, then time distortion should be greater when presenting longer emotional stimuli. However, studies have shown contrasting findings with evidence of the multiplicative being found in some studies (e.g., Droit-Volet et al., 2004; Mella et al., 2011) but not in others (e.g., Grommet et al., 2011; Lui, Penney, & Schirmer, 2011). Additionally, to date no study has shown whether emotions affect the slope of time judgments during a verbal estimation task, which is an additional indication of a multiplicative effect (see section 1.1.3.1.1, page 23) and a direct evidence of pacemaker speed modulation (Matthews, 2011). In fact, the hastening effect of



emotions on pacemaker speed should result in steeper verbal estimates. Due to the inconsistent presence of multiplicative effect and the absence of studies showing the effect of emotions on slope, the argument that emotions affect the pacemaker speed is therefore unsupported by empirical data.

Inconsistency in the presence of multiplicative effect might arise because the multiplicative effect prediction assumes that emotions affect only arousal and therefore only pacemaker changes should be observed. This is unlikely given that emotional experience affects a range of cognitive and physiological processes (see section 2.1, page 44) and it is thus possible that multiplicative effects are masked by other affected components (e.g., memory). This suggestion is supported by studies that found that emotional events had both additive and multiplicative effects on time perception (Gil & Droit-Volet, 2012; Smith et al., 2011).

In SBF, arousal affects DA, which regulates the frequency of the cortical oscillators. At a basic level, increases in arousal are thought to increase DA release, leading to faster desynchronization of cortical oscillators and longer perceived duration (Matell & Meck, 2004). In contrast, decreases in arousal are thought to decrease DA release, leading to slower desynchronization of cortical oscillators and shorter perceived duration. During emotional events, the thalamus releases greater doses of DA into the cortex; for example, Bromberg-Martin et al. (2010a, 2010b) found a phasic DA release to the cortex in response to appetitive and aversive events. This hastens the cortical oscillators, resulting in longer perceived durations compared to neutral events (see Lake et al., 2016 for discussion).

In Craig's model of awareness, physiological arousal affects the activity of the anterior insular cortex (AIC), promoting the generation of global emotional moments (Craig, 2009a). At a basic level, increases in SNS dominance are thought to increase right side AIC activity, leading to a faster production of global emotional moments and resulting in longer perceived duration. In contrast, increases in PSNS dominance are thought to increase left side AIC activity, leading to a slower production of global

emotional moments and resulting in shorter perceived durations. During emotional events, there is an increase of SNS activity, particularly for threatening, negatively valenced stimuli. This leads to right side AIC activation, producing more global emotional moments during emotional stimuli presentation, which are therefore perceived to last longer than neutral stimuli.

The mediation role of arousal in the emotional distortions to time is supported by emerging evidence that there is indeed a direct relationship between physiological arousal and the perceived duration of sub-second stimuli (Cellini et al., 2015; Fung, Crone, Bode, & Murawski, 2017; van Hedger, Necka, Barakzai, & Norman, 2017). For example, Pollatos et al. (2014) observed that greater PSNS dominance was associated with less error on a reproduction task and Cellini et al. (2015) observed that higher PSNS activity was associated with lower error rates on a temporal production task. However, these findings showed how resting state of ANS activity is associated with temporal perception, rather than how changes in ANS activity are associated with temporal distortions. Both Pollatos et al. (2014) and Cellini et al. (2015) acknowledge that their findings might just indicate the influence of PSNS on attention and working memory required for performing the timing task (see Thayer, Hansen, Saus-Rose, & Johnsen, 2009).

In addition, there are studies using physiological measures that are not specific to the SNS and PSNS activity (Fung et al., 2017; Hawkes, Joy, & Evans, 1962; Osato, Ogawa, & Takaoka, 1995). For example, Fung et al. (2017) found that low frequency heart rate variability (LF HRV) was associated with less accurate perceived durations in a temporal reproduction task. However, LF HRV is modulated by both SNS and PSNS activity (see Reyes del Paso, Langewitz, Mulder, van Roon, & Duschek, 2013 for discussion), making it therefore difficult to disentangle the effects of ANS activity on temporal distortion.

Van Hedger et al. (2017) tested the effect of a social stressor on the temporal reproduction of neutral, negative and positive images and used Pre-ejection Period

(PEP) and high frequency heart rate variability (HF HRV) to measure changes in SNS and PSNS activity, respectively. They found a significant correlation between changes in PEP (before and after the social stressor) and changes in reproduction durations for the negative images at short durations (400ms). In contrast, changes in SNS activity did not correlate with changes in reproduction durations for the negative images at long durations (4000ms), and for the neutral and positive images. No relationship was also found between HF HRV and any reproduction. This study had methodological issues, however; in a reproduction task, a state change between the presentation of the to-be-timed stimulus and participants' reproduction is required to test temporal distortions. Here, the to-be-timed stimulus was presented and the reproductions were made in the same emotional state, i.e., before the social stressor and after the social stressor. The social stressor therefore equally affected the stimulus presentation and the stimulus reproduction, which may have masked the temporal distortions. Moreover, given that SNS and PSNS activities were not recorded separately for the different emotional categories (neutral, negative and positive), it is not possible to relate the physiological response to the emotional stimulus with its perceived duration.

The arousal hypothesis, in summary, states that the time distortion induced by the emotion is directly associated with its ability to regulate arousal: the greater the changes in arousal the greater the changes in perceived duration. Whilst this hypothesis enjoys some support, there are some limitations. Firstly, the hypothesis is unable to explain why positive stimuli are perceived as lasting for less time than neutral stimuli. Positive emotional stimuli have the capacity to increase arousal in a manner akin to that observed with negative stimuli (Lang & Bradley, 2010). If positive stimuli are increasing arousal, they should be perceived as lasting for longer than neutral stimuli, not shorter. Secondly, although van Hedger et al. (2017) demonstrated a direct relationship between changes in physiological arousal and temporal distortions, there were methodological issues that reduced the validity of the findings. Systematic measurement and evaluation of the relationship between

physiological arousal and perceived duration is therefore required before it can be concluded that arousal change directly influences perceived duration.

### *2.3.2 – The attention hypothesis*

Emotions affect attentional processing, capturing attention leading to enhanced processing of emotional stimuli often at the cost of ongoing tasks (Öhman, Lundqvist, & Esteves, 2001; Vuilleumier, 2005; see section 2.1, page 44). Because veridical timing is dependent on adequate attentional resources (see section 1.1.3.1.2, page 26), attention capture by emotion is thought to affect the perceived duration of emotional events.

Experimental evidence supports this; Ogden et al. (2015) suggested that visual stimuli are underestimated while receiving a pleasant stroke because the pleasant feeling detracts participants' attention away from the timing task. Similarly, it has been suggested that the perceived duration of rotten food (Gil et al., 2009), shameful expressions (Gil & Droit-volet, 2011) and unattractive faces (Ogden, 2013) are underestimated because attention is dedicated to avoiding rotten food and shameful expressions and locating atypical features in face-space, reducing attention to time and resulting in reduced perceived duration.

Enhanced attention to emotional stimuli has also been used to explain subjective lengthening of duration. For example, Grommet et al. (2011) presented participants with neutral (i.e., lamps) and threatening images (i.e., snakes) during a temporal bisection task and found that threatening images produced more long responses compared to neutral ones. Critically, Grommet et al. (2011) found that this effect was independent of the stimulus duration, suggesting an additive effect of emotion on verbal estimation, which is an index of a switch modulation rather than a pacemaker speed modulation within SET (see section 1.1.3.1.2, page 26).

Different theoretical models of time perception provide different levels of specificity about the precise way in which emotion alters attention to time. In SET, attention influences switch operation; because emotions capture more attention at stimulus onset than neutral stimuli, emotions reduce the latency with which the switch closes, allowing more pulses to access the accumulator compared to the usual switch latency when presenting neutral stimuli (see section 1.1.3.1.2, page 26). Furthermore, emotions increase sustained attention (see section 2.1, page 44), which is thought to affect the openness of the attentional gate, regulating the amount of pulses allowed into the accumulator (Zakay & Block, 1995).

SBF suggests that emotional affects attention to time in two ways; firstly, emotional stimuli capture more attention at their onset than neutral stimuli leading to faster resetting of cortical oscillators. This allows cortical oscillators to desynchronize during a greater time window, resulting in time overestimation for emotional stimuli compared to neutral ones. Secondly, SBF predicts that sustained attention to time has similar effects of the ones induced by arousal (Matell & Meck, 2004); that is, greater attention for emotional stimuli increases dopamine level in the brain leading to faster desynchronization of cortical oscillators and resulting in longer perceived duration. In contrast, a lack of sustained attention leads to a decrease in dopamine levels and fire ratings of the cortex, resulting in time underestimation.

Craig's model of awareness does not explicitly state how attention would affect time perception. However, the model predicts that the timing process is based on the *saliency* of the to-be-timed event (Craig, 2002) and salient events capture more attention than non-salient ones (Kim & Cave, 1995). Salient stimuli lead to greater right side AIC activity, greater production of global emotional moments and longer perceived duration. Emotional stimuli are more salient than neutral ones (Craig, 2002), therefore leading to greater AIC activity, which explains why they are perceived as lasting for longer. However, because the model predicts that time perception is affected by the stimulus saliency, which is always greater in emotional

stimuli than in neutral ones, Craig (2009a) acknowledged that the model is not able to address why sometimes emotions lead to time underestimation.

The attention hypothesis, in summary, states that the time distortion induced by the emotion is directly associated with the quantity of attention captured by the emotion at stimulus onset and with the quantity of sustained attention dedicated to time during stimulus presentation. It should be noted however, that whilst the attentional explanations used in these experiences are plausible they are simply inferred from the presence of underestimation or additive effect, but they are not explicitly tested. Moreover, additive effect was not consistently found across studies (e.g., Droit-Volet et al., 2004; Mella et al., 2011).

Although the field of time perception treats arousal and attention as fairly distinct components. There is substantive evidence demonstrating that arousal and attention are collaborative processes that enable priority for the detection and encoding of emotional events in the external world (see Vuilleumier, 2005 and Coull, Cheng, & Meck, 2011 for review). It therefore seems highly likely that arousal and attention interact during time perception. This is also supported by evidence showing that simply instructing participants to direct attention toward the emotional component rather than the time component of the event affects physiological responses and perceived duration (Mella et al., 2011). The current approach of prescribing either attentional *or* arousal explanations for findings is therefore problematic as any explanation is likely to be incomplete. An integrative account of how attention and arousal dually impact on time perception is therefore required.

## Chapter 3

### The effect of pain on time perception

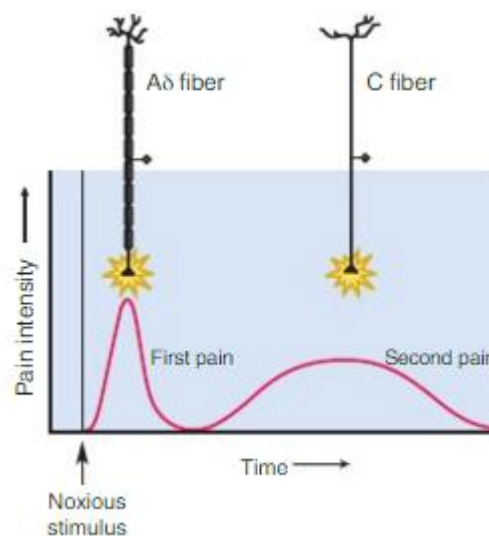
#### 3.1 – Pain

In the earliest models, pain was conceived only as a biological phenomenon to promote survival and avoid body damage. The psychological states of the sufferer were not thought to play any role on the pain experience (see Brown, 1989 for a description of the Cartesian Dualism). Nowadays, pain is considered a complex biopsychosocial construct, where physiological, social, affective and cognitive dimensions all interact to create the experience of pain (Engel, 1980). Pain is a subjective experience which can be measured only through self-reported techniques. This is because, although the activity of the skin receptors can be measured, this activity does not always reflect the pain intensity experienced by the sufferer (Smart, Blake, Staines, & Doody, 2012). Biopsychosocial models, therefore, considered pain as a survival mechanism that can be modulated by the sufferer's social and psychological state.

##### 3.1.1 – Physiology of pain

The skin contains a variety of receptors with different functions (Bear, Connors, & Paradiso, 2007); mechanoreceptors detect pressure and vibration, thermoreceptors detect variation of temperature and nociceptors detect noxious stimulations. Nociceptors are divided in sub-categories, with each sub-category detecting a single threat source; thermal nociceptors are responsible for the burning sensation when a hot object is in contact with the skin, meanwhile mechanical nociceptors are responsible for the pain sensation when a blunt or sharp object hits the body. Nociceptors are also divided in two categories based on the presence of myelination, which affects the axon's speed in transferring the information from the skin to the spinal cord. A $\delta$  nociceptors are myelinated and have a speed of 5-30m/sec, meanwhile C nociceptors are not myelinated and have a speed of 0.5-2m/sec. This difference in speed between C and A $\delta$  nociceptors are also responsible for the biphasic response to pain; A $\delta$  nociceptors are responsible for the first, sharp and

short-lasting sensation of pain; meanwhile C nociceptors are responsible for the second, dull and long-lasting sensation of pain (Figure 3.1). The first phase has the aim of interrupting the contact of the skin with the threatening source (e.g., removing the hand from the hot pan); meanwhile the second phase has the aim of making the injured body part sensitive so to promote protection and avoid further damage (e.g., the use of a burnt finger is reduced).



*Figure 3.1. First and second pain phases mediated by fast A $\delta$  and slow C nociceptors respectively (Bear et al., 2007).*

### 3.1.2 – Neural correlates of pain

Once a threat has been detected by the nociceptor, the information follows the spinothalamic medial and lateral pathways before being perceived (Brooks, Nurmikko, Bimson, Singh, & Roberts, 2002). The nociceptor's axon projects to the nerve in the dorsal column of the spinal cord, which follows the lateral side of the spinal cord projecting to the medial or lateral nuclei of the thalamus. The lateral nuclei of the thalamus project to the primary and secondary somatosensory cortex, meanwhile the medial nuclei project to the limbic regions of the brain (e.g., cingulate gyrus and insula). Historically, the lateral pain pathway would be responsible for the sensory-discriminative aspects of pain (e.g., the pain intensity; Treede, Kenshalo,



Gracely, & Jones, 1999); meanwhile the medial pathway would be responsible for the affective aspects (Vogt & Pandya, 1987). For example, Lamm et al. (2011) found that the anterior insula and cingulate gyrus were also activated while seeing someone in pain (that is, in empathy for pain), supporting the limbic systems relationship with the affective aspects of pain rather than with its sensation itself. Craig (2002) also suggested that pain has a privileged route, compared to other stimulations (e.g., thermal stimulation), to the posterior insular cortex, which is responsible for the sense of interoception. The sense of interoception, in fact, is enhanced while experiencing pain (Craig, 2002). However, the historical pathways were contested by Brooks et al. (2002), who observed that only a small area in the posterior insula has the sensory-discrimination role. Brooks et al. (2002) found that distracting participants from pain with a perceptive task (i.e., detecting the moving direction of a series of dots) changed the neural activity associated with pain processing (i.e., insula, inferior frontal gyrus, secondary sensory cortex and cingulate gyrus), with the only exception of that small area in posterior insula. Brooks et al. (2002) therefore suggested that this might be the only area with a sensory-discriminative role.

Pain also activates the hypothalamus, which regulates the activity of the autonomic nervous system (Bear et al., 2007). Pain alters the activity of the two branches of the ANS, the sympathetic and parasympathetic systems, leading to the SNS dominance over the PSNS because the SNS is activated during threatening situations and has the function of preparing the body for the fight/flight response in order to survive. Pain induced increases in SNS activity result in increased heart rate (Dowling, 1983; Hamunen et al., 2012), electro-dermal activity (Dowling, 1983; Dubé et al., 2009), breathing rate (Boiten, 1998) and peripheral vasoconstriction (Awad et al., 2001). These effects are thought to enhance muscle oxygenation, predisposing the body to react to the threat and reducing harm (Gordan, Gwathmey, & Xie, 2015).

### *3.1.3 – Experimental induction of pain*

Historically, in experimental settings, pain has been induced using two types of stimulus: electrical and thermal (Kyle & McNeil, 2014). The most widely used

technique to induce thermal pain is activating thermal nociceptors through the cold pressor test, which consists of placing the participant's hand in freezing water (0-5°C) (Richardson et al., 2013). The popularity of this test is due to the contained cost of the equipment, the ease of administration and its excellent validity and reliability (Edens & Gil, 1995). However, the method also has limitations: the temperature used and the number of hand immersions vary across studies, leading to difficulties when comparing the findings (Mitchell, MacDonald, & Brodie, 2004).

Other thermal pain stimulators include the use of a thermode (e.g., Moore, Keogh, & Eccleston, 2013) and a laser (e.g., Stancak, Raij, Pohja, Forss, & Hari, 2005) that activate thermal nociceptors. The former consists of a thermode placed on participant's skin, whose temperature can be controlled by the experimenter to reach from 0°C to 55°C. The latter consists of a laser beam on participant's skin whose intensity can be controlled to produce burning sensation. Although these techniques are less popular and have higher financial costs compared to the cold pressor technique, they bring other advantages. For example, the laser technique has the advantage that none of its components is in direct contact to the skin, avoiding the activation of mechanoreceptors (activated by the thermode and by the water in the cold pressor test), which may interfere with the nociceptors' activity. In contrast, the thermode can both increase and decrease its temperature based on the experimental design.

Electro-cutaneous stimulation, on the other hand, induces pain through alteration of the membrane potential of the stimulated cells and through alteration of electrical energy into thermal energy, resulting in (potential) tissue damage (Lee, Zhang, & Hannig, 2000). Electrical pain has been used for its high degree of temporal and intensity acuity: the electrical stimulation delivers the selected pain intensity from the stimulation onset until the stimulation offset, contrarily to the thermal stimulation that usually requires a ramp time before reaching the selected temperature and before returning to baseline. Moreover, the duration of the electrical stimulation can be in the sub-second range within an error of one

millisecond. However, electrical pain is not specific to any particular type of nociceptor (e.g., thermal nociceptor) because the electrical stimulation affect the membrane potential of all the cells invested by the shock. This leads the electrocutaneous stimulation to activate all receptors (i.e., nociceptors, mechanoreceptors, etc.), resulting in a complex sensation that is not only limited to pain, but involves also a feeling of vibration and heat (Lee et al., 2000). Moreover, the density of the receptors differs depending on the body area, leading to different sensations; for example, the perception of an electric shock on the arm may differ to the perception of a shock on the chest (Lee et al., 2000).

<i>Method</i>	<i>Nociceptors activated</i>	<i>Advantages</i>	<i>Disadvantages</i>
Cold pressor test	Thermal nociceptors	<ul style="list-style-type: none"> <li>▪ Contained costs.</li> <li>▪ Ease of administration.</li> <li>▪ Excellent validity and reliability.</li> <li>▪ Stimulation in minutes range.</li> </ul>	<ul style="list-style-type: none"> <li>▪ Impossible stimulation with hot temperature.</li> <li>▪ Impossible stimulation in sub-second range.</li> </ul>
Thermode	Thermal nociceptors	<ul style="list-style-type: none"> <li>▪ Dedicated software for experimental and clinical settings.</li> <li>▪ Stimulation in minutes range.</li> </ul>	<ul style="list-style-type: none"> <li>▪ High costs.</li> <li>▪ Impossible stimulation in sub-second range.</li> </ul>
Laser	Thermal nociceptors	<ul style="list-style-type: none"> <li>▪ Dedicated software for experimental settings.</li> <li>▪ Activates only thermal nociceptors and no other receptors.</li> </ul>	<ul style="list-style-type: none"> <li>▪ High costs</li> <li>▪ Impossible stimulation with cold temperature.</li> <li>▪ Impossible stimulation in minutes range.</li> </ul>
Electrical shock	All nociceptors (thermal, mechanical, etc.)	<ul style="list-style-type: none"> <li>▪ Contained costs.</li> <li>▪ High degree of temporal and intensity acuity.</li> </ul>	<ul style="list-style-type: none"> <li>▪ Impossible stimulation in minutes range.</li> <li>▪ Activates all receptors of the skin.</li> </ul>

*Table 3.1. The most common techniques to induce experimental pain with the activated nociceptors, the advantages and disadvantages.*

#### *3.1.4 – The effect of pain on cognition*

Pain is a highly salient stimulus that grabs attention from ongoing tasks (Eccleston & Crombez, 1999; Moore, Keogh, & Eccleston, 2012; Moore et al., 2013), resulting in rapid detection (Van Damme, Crombez, & Lorenz, 2007). These attention effects occur to maximize the likelihood of survival (Somov, 2000); the faster the attentional shift toward the noxious stimulus, the sooner the avoidance response, resulting in increased chances of survival. Pain also affects other cognitive functions, such as long-term memory (Kuhajda, Thorn, & Klinger, 1998), working memory (Buhle & Wager, 2010) and executive functions (e.g., inhibition and task switching; Moriarty, McGuire, & Finn, 2011). For example, people exposed to painfully cold water whilst viewing a series of words later recalled fewer words compared to people who were exposed to room temperature water (Kuhajda et al., 1998). Moreover, increasing the intensity of thermal pain disrupts working memory performance in an n-back task (Buhle & Wager, 2010). Overall, these findings suggest that painful stimuli receive priority in the distribution of the cognitive resources, which are limited, at the expenses of non-pain related tasks (see Moriarty et al., 2011 for review).

#### *3.1.5 – Regulation of pain*

Physiologically, perceived pain can be regulated through afferent (bottom-up) and descending (top-down) pathways; the former consists of activating the mechanoreceptors near the injury, for example rubbing the skin, to decrease the nociceptors fire rate and therefore the pain sensation (see Gate Control Theory by Melzack & Wall, 1965). The latter consists of activating the periaqueductal grey matter through cognitive and emotional regulation to induce body analgesia (Hampton, Hadjistavropoulos, Gagnon, Williams, & Clark, 2015).

Emotional suppression strategies modulate the perception of pain; for example, seeing arousing, positive pictures decreases pain ratings (Godinho, Magnin, Frot,

Perchet, & Garcia-Larrea, 2006), meanwhile seeing arousing, negative pictures increases pain ratings (Godinho et al., 2006) and inhibits pain threshold (Meagher, Arnau, & Rhudy, 2001). Descending regulation of pain can be also achieved by distracting sufferers with classic cognitive tasks, such as the Stroop task (Valet et al., 2004), perceptive tasks (Brooks et al., 2002) and working memory task (Buhle & Wager, 2010). More ecological techniques, such as playing video games (Stamp, Dobbins, Fairclough, & Poole, 2017) and reading a book (Hussein, 2015), can be also used to reduce pain experience. Finally, control over pain has also been found to affect pain ratings; pain ratings are higher when the sufferer lacks of control (Turk, Meichenbaum, Genest, & Berntzen, 1984), meanwhile increasing the control decreases the pain ratings (Kanfer & Seidner, 1973).

In clinical contexts, pain is effectively management using psychologically based therapies, such as Cognitive Behavioural Therapy (Eccleston & Crombez, 1999) and Acceptance and Commitment Therapy (Hayes, Levin, Plumb-Villardaga, Villatte, & Pistorello, 2013). Particularly, mindfulness-based interventions (e.g., Mindfulness-Based Stress Reduction) have recently increased their popularity for the pain management (Malinowski, 2017; Zeidan, Gordon, Merchant, & Goolkasian, 2010). For example, mindfulness-based interventions reduce perceived intensity of clinical pain (Veehof, Trompetter, Bohlmeijer, & Schreurs, 2016) and reduce stress and depression symptoms improving the quality of life and wellbeing of chronic pain patients (Kuyken et al., 2015).

### **3.2 – The relationship between pain experience and time perception.**

Einstein famously observed that “put your hand on a hot stove for a minute and it feels like an hour”. This distorting effect of pain experience on time experience has since been explored in numerous experimental (Ogden, Moore, Redfern, & McGlone, 2014; Thorn & Hansell, 1993) and clinical (Somov, 2000) studies. Here, experimental studies have been referred to those studies that induced experimental pain to healthy participants comparing their time perception between painful and non-

painful condition. In contrast, clinical studies have been referred to those studies where time perception of healthy participants was compared to the time perception of clinical patients who suffered pain (e.g., back pain).

### 3.2.1 – Experimental studies

In experimental settings, pain has been shown to lengthen perceived duration in a range of studies using a variety of pain induction techniques. Hare (1963) tested the effects of electric shocks on the verbal estimation of empty intervals delimited by a click sound at the beginning and at the end. The study adopted a prospective verbal estimation paradigm, where participants were asked to estimate the duration of a series of 5- and 20-second intervals, half of which were followed by a painful shock. Participants were told before the interval whether the shock would occur. Participants overestimated the duration of the intervals with the shock compared to intervals without a shock, suggesting that pain experience lengthened the perceived duration of the intervals. The lengthening effect of electric shock was recently replicated by Fayolle et al. (2015) using a temporal bisection task.

Ogden et al. (2014) observed comparable results when testing the effects of heat pain on the perceived duration of neutral visual stimuli. In this study, participants were first conditioned to associate one visual symbol with the occurrence of pain and another with no pain. Participants then completed a prospective verbal estimation task in which the pain-conditioned stimulus and the neutral conditioned stimuli were re-presented, however pain was only delivered on 30% of pain-stimulus trials. The stimuli could last between 50 and 1500 milliseconds. Results showed that the pain-related stimulus was overestimated compared to the neutral stimulus, suggesting a lengthening effect of pain on perceived duration. Overestimation also occurred in trials where the pain-related stimulus was presented but the pain was not delivered, suggesting that the *anticipation* of pain induces time distortions. The authors also showed that the pain effect on subjective timing has a greater magnitude than the

time distortion magnitude generated by visual emotional stimuli in previous studies (e.g., angry faces in Gil & Droit-volet, 2011).

Rey et al. (2017) also found time overestimation of neutral visual stimuli (e.g., a gray square) using the cold pressor test. Participants completed a temporal bisection task where they first learnt a short (250ms) and a long (750ms) duration and then were presented with a series of comparison durations whilst their hand was immersed in the water. Results showed that comparison stimuli were perceived as more similar to the long duration more often when participants' hand was in painful, cold water (12°C) compared to when their hand was placed in room temperature water (25°C). Accordingly, the pain stimulation also induced a left shift of the point of subjective equality (see 1.1.3.2, page 29), suggesting that pain stimulation lengthened the perceived duration of visual stimuli.

It should be noted, however, that lengthening effects of pain are not universally observed. Evidence of pain shortening perceived time can be found in studies of retrospective timing, where participants are informed about the timing task only after the presentation of the to-be-timed event. In two similar studies, Thorn and Hansell (1993) and later Hellström and Carlsson (1997) asked participants to retrospectively estimate the duration of cold pressor trials where participants' hand was placed in a tank with water at freezing (7°C and 2°C) or room (35°C and 26°C) temperature. Participants underestimated the duration of the trial when the water was painfully cold compared to when it was at room temperature.

Collectively, these studies suggest that in prospective timing studies pain related stimuli are perceived as longer than neutral stimuli. Furthermore, this distorting effect of pain is substantial, producing greater lengthening effects than emotional distortions to time. However, in retrospective studies, pain has a shortening effect on perceived time. Differential effects of pain on pro and retrospective timing support suggestions that the two types of timing are based on different cognitive and neurological processes (Zakay & Block, 2004). Previous studies also compared time

perception between a painful and a non-painful condition, considering pain as a binary experience (pain vs no pain) rather than a single dimension with a spectrum of intensities. Pain intensity is critical for the magnitude of pain effects on cognition (Eccleston & Crombez, 1999) and arousal (Möltner, Hölzl, & Strian, 1990), which is particularly important in clinical settings where patients have a variety of symptoms and pain intensity experience (Somov, 2000).

### *3.2.2 – Clinical studies*

Clinical studies of time experience during pain have typically recruited participants who suffered of pain caused by a medical pathology (e.g., migraine) experienced for long periods (e.g., days) (Zhang et al., 2012). In clinical settings, how long patients report pain to last for forms part of pain's clinical assessment, with longer periods potentially indicating greater treatment need (Somov, 2000). However, numerous studies show that clinical pain affects the perception of time with pain sufferers often reporting time as "dragging" or "standing still" during pain (Somov, 2000). For example, Bilting et al. (1983) asked patients with facial pain and pain free participants to read aloud a list of random letters for a fixed time interval (30 – 120 seconds) and then to retrospectively estimate this interval. Results showed that pain sufferers estimated the time interval as longer than the control group.

Comparable effects are observed in neuropathic pain populations; Somov (2000) observed that people with neuropathic pain overestimated a 3-minute interval whilst completing a series of questionnaires compared to healthy people. The overestimation was predicted by pain intensity, with great pain experience associated with greater time overestimation, and persisted after controlling for the effects that anxiety, depression and perceived control over pain have on time perception. All relationships were also invariant of the type of pain (e.g., back pain or migraine).



However, clinical studies have not always found patients to perceive time longer than healthy people. For example, Zhang et al. (2012) found that pain sufferers (migraineurs) overestimated time compared to healthy controls in a temporal reproduction task when using a 600ms duration, whilst there was no temporal distortion when using a 3- or 5-second duration. Moreover, using a temporal discrimination task in which participants were presented with pairs of stimuli and their task was to decide if the second was longer or shorter than the first, Anagnostou and Mitsikostas (2005) failed to observe a difference between migraineurs and healthy controls. Additionally, Isler et al. (1987) found that patients who suffered of migraine retrospectively underestimated respiratory biofeedback sessions compared to healthy people. The sessions could vary in length (3-42min) and consisted of presenting participants with their respiratory activity, whilst participants were asked to use this feedback to relax as much as possible.

Some of these inconsistencies may result from methodological differences in the studies. Anagnostou and Mitsikostas (2005) gave feedback to participants after every trial of the temporal discrimination task. Giving feedback to participants has been showed to improve participants performance (Failing & Theeuwes, 2016), which may have corrected the time distortion originally present in patients. In contrast, Isler et al. (1987) asked participants to estimate the duration of respiratory and EMG biofeedback sessions, which may have been judged as more pleasant by migraineurs than by healthy people (Van Diest et al., 2014). The difference in pleasantness may have caused the difference in verbal estimation as in Ogden et al. (2015), where a pleasant stimulation is underestimated compared to a neutral or unpleasant stimulation. Patients may have enjoyed the biofeedback session more and dedicated less attention to time than healthy participants, leading to patients' shorter estimates.

Although clinical studies have produced somewhat inconsistent findings, possibly caused by differences in the clinical populations of each study and the methods of temporal assessment, time distortions are increasingly used in clinical settings as a

measure of patient wellbeing. For example, Conev et al. (2018) asked chemotherapy patients to complete the standardized National Comprehensive Cancer Network Distress Thermometer and to produce 1 minute. Results showed that the time production of patients with higher distress was shorter than the time production of patients with lower distress, suggesting that more distressed patients perceived time to go faster. Conev et al. (2018) therefore proposed the implementation of a simple time perception task as an efficient tool to assess patients' distress.

### *3.2.3 – The effect of temporal information on pain experience*

There is evidence to suggest that the relationship between pain and time perception is bidirectional; information about time has the capacity to influence the experience of pain. Pomares et al. (2011) observed that it is possible to reduce the perceived intensity of pain by manipulating its perceived duration. The study required participants to be administered heat pain twice for 30 seconds. During pain stimulation, participants were provided with information about the amount of time the pain would last for in the form of a clock with a moving hand. In one condition, the hand completed an entire revolution; in the other condition, the hand completed only three-fourths of the revolution. After pain stimulation, participants rated the pain intensity. Pain intensity ratings were significantly higher on the former condition than on the latter condition. Similar findings were found by Coldwell et al. (2002) who asked children to take part to nine cold pressor trials of increasing duration (from 10s to 240s) where they were asked to keep their hand in cold water (10°C) while Garfield cartoon character was shown on the screen in front of them. Children were divided in three groups depending on the movements made by Garfield: in the correct feedback group, Garfield was shown descending a series of steps and the cold pressor trial ended once Garfield reached the bottom of the stair. In the misleading feedback group, Garfield randomly jumped from one step to the other of the stair during the trial, and in the no feedback group, Garfield remains static. Results showed that children who were given correct feedback about how long the pain lasted for, also reported reduced pain intensity compared to those who received no or misleading

feedbacks. This evidence therefore indicates that the temporal characteristics of pain is intrinsically related to its intensity and Pomares et al. (2011) suggested that temporal information of pain could affect its perceived intensity.

### **3.3 – Theoretical explanation of pain effects on time**

Experimental studies showed that pain lengthens time perception when using prospective timing tasks and that time distortion induced by pain has greater magnitude than the time distortion induced by emotional stimuli (Ogden, Moore, et al., 2014). The lengthening effect of pain in experimental studies is ascribed by pain ability of increasing arousal and capturing attention, which, in turn, affects the internal clock (in SET), dopamine level (in SBF) and AIC activity (in Craig’s model of awareness). Clinical studies, in contrast, often merely state that time distortion of pain sufferers (e.g., migraineurs) might be due to the comorbidity with other clinical conditions that showed time distortion (e.g., depression), failing to make the link between time distortion due to clinical pain and theories of time perception (Bilting et al., 1983).

#### *3.3.1 – The arousal hypothesis of pain induced distortions to time*

Experimental studies of pain induced distortions to time typically suggest that pain distorts time because it increases physiological arousal which in turn affects the processing of duration (see section 2.3.1, page 47). Clinical studies support this suggestion by observing a direct relationship between patients’ pain intensity and the degree of time distortions they experience (Somov, 2000). In SET, arousal regulates the frequency production of pulses by the pacemaker. At high arousal levels, pulses are generated more frequently, resulting in greater number of pulses stored into the accumulator, leading to longer time estimations. Arousal increases due to pain are therefore thought to lead to longer perceived duration through hastening of pacemaker and increasing of pulses production. In other words, pain increases arousal, which hastens the pacemaker speed, producing a greater number of pulses

later stored into the accumulator, leading to longer estimations. Within SET framework, the arousal modulation hypothesis is also supported by Ogden et al. (2014) and (Fayolle et al., 2015), who found a multiplicative effect (the index of arousal/pacemaker modulation; see section 1.1.3.1.1, page 23) of pain on perceived duration.

In SBF, the thalamus regulates the dopamine level to modulate the oscillatory cortical activity; the higher the dopamine level released by the thalamus the higher the oscillatory activity, leading to longer perceived durations (Matell & Meck, 2004). Nociceptors are directly connected to the thalamus through the pain pathways (Brooks et al., 2002), leading to an increase of dopamine release by the thalamus to the cortex, causing an increase of cortical frequency and subsequently leading to longer perceived durations of pain related events.

In Craig's model of awareness, time distortion is caused by the saliency of the environment; settings that are highly salient (i.e., contains highly important stimuli for the individual survival) increase AIC activity promoting the generation of global emotional moments (Craig, 2009a). A time window where numerous global emotional moments are generated is perceived to last longer than another time window where few global emotional moments are generated. Pain is a highly salient stimulus and has a privileged route to the insula, whose activity is increased during pain (Craig, 2002). Following Craig's model, therefore, a greater number of global emotional moments are generated while experiencing pain, leading to the feeling of time dragging.

For all the major models of timing there are therefore clear biological and neurological pathways between pain experience and time perception which provide plausible explanations for pain-time distortions. However to date, no studies have directly tested some of the explanation proposed. Firstly, no studies have tested whether physiological arousal resulting from pain (e.g.; SNS reactivity) is predictive of the perceived duration of pain. For example, Fayolle et al. (2015) found that an

electrical shock increased physiological arousal (indexed by EDA) and increased perceived duration of visual stimuli concomitantly presented in a temporal bisection task. However, authors did not test whether the pain distortion of time and physiological arousal were related. It is therefore unclear whether arousal attention have *direct* influences on time perception. Secondly, neuroimaging studies are yet to demonstrate how thalamic changes in activity due to nociception are related to time perception. Thirdly, no neuroimaging studies have tested the extent to which AIC activation during pain is correlated to pain-time distortions, and thus testing the homeostatic model of timing. Further work is clearly required to evidence the theoretical explanations proposed.

### 3.3.2 – *The attention hypothesis*

Adequate attention is required for accurate duration processing (see section 1.1.3.1.2, page 26). Recent individual differences studies have also demonstrated that individual differences in working memory, attention, executive function and information processing speed are predictive of duration processing accuracy (Ogden, MacKenzie-Phelan, Montgomery, Fisk, & Wearden, 2018). However, pain impairs attention on ongoing tasks and performance on executive tasks and working memory tasks (see section 3.1.4, page 60). It is therefore plausible that pain may affect time experience because it impairs the cognitive and attentional resources on which timing is dependent. However, it is unclear why this would result in a lengthening of perceived duration, because distraction from timing/inadequate resourcing of timing is typically associated with shorter prospective duration estimates (Brown, 2006).

One possibility is that because pain is attention grabbing it leads to more effective processing of pain duration (Ogden, Moore, et al., 2014). This would manifest in two ways: firstly, pain stimuli are identified quickly, leading to fast switch closure and greater pulses accumulation. Secondly, because pain captures attention from ongoing tasks, the duration of pain is attended to throughout its experience leading to an increase in its perceived duration (i.e., there is no temporal output lost through

an attentional gate fluctuation). Ogden et al.'s (2014) suggestions are supported by their observation that pain produced additive and multiplicative effects, indexes of switch and attentional gate variation respectively (see section 1.1.3.1, page 23).

In SBF, the thalamus is also responsible for resetting the oscillators at stimulus onset (Matell & Meck, 2000, 2004) and the preferential route that pain has to reach it (i.e., the pain pathways; Bear et al., 2007) might lead to faster oscillatory resetting at stimulus onset. If the oscillators' reset occurs earlier for noxious stimuli than for neutral ones, oscillators have more time to desynchronize before the striatum reads their pattern at stimulus offset, leading to longer perceived durations. However, at present there is no neural evidence indicating a shorter reset of cortical oscillators at painful stimuli onset compared to non-pain related stimuli onset.

In Craig's model of awareness, attention does not appear to have a clear effect on time estimations. Whilst a salient environment requires more attentional resources, the model does not predict different outcomes based on where the attention is directed. The saliency of the environment is always directly proportionate to the number of global emotional moments generated by the AIC and to the perceived duration, independently to whether the attention is directed to the to-be-timed stimuli or elsewhere. Following Craig's model, pain should therefore lengthen the perceived duration of the noxious stimuli and of concomitantly neutral stimuli.

### *3.3.3 – Retrospective estimates*

Whilst there is a clear theoretical and biological rationale for pain induced distortions to prospective timing, it is unclear how or why pain shortens retrospective estimates of duration (as in Thorn & Hansell, 1993). Retrospective estimates of duration are often understood in terms of information processing load or contextual change; periods of greater information processing/contextual change result in longer retrospective estimates than periods of low information processing activity/contextual change (Zakay & Block, 2004). Because pain interrupts ongoing

cognition and captures attention from ongoing tasks, it is possible that it reduces information processing. It is also possible that it reduces contextual change because the individual is only focusing on pain and not other stimuli in the environment. These may therefore be the mechanisms by which pain shortens retrospective estimates of duration. However, because pain is intrusive and hard to ignore, it seems likely that information processing relating to pain itself would be high during pain. It therefore remains unclear why pain appears to shorten retrospective estimates.

## Chapter 4

### Aims, equipment and methodology

#### 4.1 – Summary of the literature review and research strategy

From Chapters 2 and 3, it is clear that stimuli associated with changes in arousal (e.g., emotional images, sounds and pain) affect perceived duration. However, the precise mechanisms driving these effects remain unclear. This is due to the stimuli used in previous studies and to the absence of direct tests of the relationship between objective measures of arousal and changes in perceived duration. For example, previous studies investigating the effect of emotions on subjective time have used different physical stimuli in the neutral and arousing conditions (e.g., neutral, positive and negative sounds from the IADS; Noulhiane et al., 2007). However, different properties of the stimuli (e.g., luminance and numerosity) alter their perceived duration (Xuan, Zhang, He, & Chen, 2007). Moreover, Gil and Droit-Volet (2012) suggested that the semantic content of an image could affect the magnitude of the time distortion it generates, even if there is no change in the arousal level. This evidence led Lake et al. (2016) to suggest that future studies should have a broader control over the properties of neutral and arousing stimuli, such as complexity and familiarity. Reducing the physical differences between the neutral and arousing stimuli is therefore necessary to investigate the direct relationship between arousal and perceived duration.

Studies using emotional faces reduced the differences between neutral and arousing stimuli given their structural similarity. However, the neural networks involved in the processing of emotional expressions are different from the neural networks involved in the processing of other emotional stimuli (Kanwisher & Yovel, 2006), limiting the generalisation of the findings. The use of pain-inducing techniques proved to be the most effective way to overcome this issue, given that there are no significant physical differences between painful and non-painful stimuli (Ogden, Moore, et al., 2014). The present thesis therefore used pain-related stimuli



throughout to induce changes in arousal levels and reduce the risk of altering other properties of the stimuli.

A further issue is that previous studies used low and high levels of arousal rather than a range of arousal levels, which limits the supporting evidence for the arousal hypothesis on time perception (see sections 2.2 and 3.2, pages 45 and 61). In fact, arousal hypothesis predicts that increases steps of arousal are associated with increases steps of perceived duration. Testing perceived durations at different arousal levels therefore forms an indirect test of the arousal hypothesis. In addition, studies have primarily investigated the effect of arousal when the to-be-timed stimulus is itself arousing, and little research has been conducted when arousal arises from other sources. Models of timing (SET, SBF and Craig's model) predict that arousal increases affect the central timing mechanism leading to longer perceived duration of all subsequent stimuli, not just of the stimulus that is the source of arousal (see section 2.3.1, page 47). However, the few studies that investigated the effect of arousal when arousal originated from sources other than the to-be-timed stimulus, found contrasting results. For example, a short fearful film induced longer perceived durations in a subsequent temporal bisection task with neutral stimuli (Droit-Volet et al., 2011). However, perceived durations of neutral stimuli were not affected by a concomitant, negatively valenced tactile stimulation (Ogden et al., 2015). Furthermore, physiological activation induced by social stress was associated with the perceived duration of negative stimuli but not with the perceived duration of neutral and positive stimuli in a subsequent temporal reproduction task (van Hedger et al., 2017). It is therefore unclear whether arousal originating from sources other than the to-be-timed stimulus also affect perceived duration.

Previous studies have also failed to directly test the relationship between physiological arousal and perceived duration (see Lake et al., 2016 for discussion). For example, previous studies that tested the effect of arousal on perceived duration used stimuli which experimenters believed to be arousing (e.g., Tipples, 2008) or used self-reported measures of arousal rather than objective measures of arousal.

Furthermore, those studies that recorded physiological measures of arousal rarely related them back to perceived durations. For example, Angrilli et al. (1997), Droit-Volet et al. (2010), Fayolle et al. (2015) and Mella et al. (2011) showed that their arousing stimuli did increase physiological arousal, but they did not relate the changes in physiological arousal back to the changes in perceived duration. The absence of direct testing led Lake et al. (2016) to state that *“Without demonstrating such a relationship between temporal estimates and measures of these emotional dimensions [valence and arousal], it is difficult to conclude that such dimensions, rather than other differences between presented stimuli, are driving temporal distortions.”* (p. 412). It is therefore paramount that future studies relate objective measures of arousal to changes in perceived durations directly.

A further issue in our understanding of arousal modulation of time perception is that research has predominantly investigated the effect of arousal on the “raw” encoding of durations (e.g., the internal clock in SET), meanwhile fewer studies investigated the effect of arousal on following processes (e.g., the memory process in SET). For example, numerous studies assessed the perceived duration of arousing stimuli immediately after their presentation (see sections 2.2 and 3.2, pages 45 and 61), but little research has been conducted to assess how the duration of stimuli of high arousal are remembered over periods of delay. As a result, there is a lack of clarity surrounding the non-immediate effects of arousal on perceived duration. If models of temporal processing are going to comprehensively explain how and why arousal affects timing, then further studies are required to expand their investigation on the processes following the initial “raw” encoding of the stimulus.

Finally, whilst previous studies have consistently demonstrated that arousing stimuli such as pain and fear are relatively overestimated, to date there have been few attempts to prevent this lengthening effect. Pomares et al. (2011) indicated that the perceived duration of pain is related to its perceived intensity. Therefore establishing intervention or mechanisms that reduce the perceived duration of pain could reduce perceived pain intensity with potential benefits to sufferers. Particularly

in a clinical context, the intervention could improve the daily experience of pain sufferers, who have reduced quality of life and wellbeing due to the constant pain experience (Somov, 2000). Coldwell et al. (2002) and Pomares et al. (2011) used inaccurate feedback regarding the duration of experimentally induced pain to reduce its perceived duration and intensity. However, it could be difficult to transpose this methodology into clinical settings, where the intervention could bring the greatest benefits. In clinical settings, pain has biological origins and it is often experienced for long periods of time. It is therefore difficult to give (in)accurate feedback when pain is not controlled by the experimenter and for such long periods. New interventions that could potentially reduce perceived duration of pain are therefore needed.

#### **4.2 – Research aims**

This thesis aims to examine the effect of pain on human temporal perception and the experiments conducted aim to give a clear account of the effect of pain on temporal processing. This will be achieved by examining how different levels and sources of pain affect duration perception and memory for duration, how the pain-induced physiological arousal influences perceived duration, and whether the perceived duration of pain can be reduced using a mindfulness intervention, which has been previously demonstrated to reduce the perceived intensity of pain (Zeidan et al., 2011).

Chapters 5 and 6 aimed to examine the effect of arousal on perceived duration, testing the arousal hypothesis when arousal arises from the to-be-timed stimulus or from other sources. Experiments 1 and 2 (Chapters 5) used stimulations at no pain, low pain and high pain intensity during a verbal estimation task to examine the effect of different arousal levels on perceived duration. Experiment 1 tested the effect of arousal on perceived duration when the to-be-timed stimulus is the source of arousal. Experiment 2 tested the effect of arousal on perceived duration when arousal arises from the background. Experiments 3 and 4 (Chapter 6) also used stimulations at no pain, low pain and high pain intensity during a verbal estimation task and included

physiological measures to directly test the relationship between changes in physiological arousal and changes in perceived duration when arousal arises from the to-be-timed stimulus (Experiment 3) and from the background (Experiment 4).

Chapter 7 aimed to examine the effect of arousal on the processes following the initial “raw” encoding of the stimulus. Experiments 5 and 6 used a modified temporal generalisation task where participants had to remember the standard duration over a period of delay to examine how pain affects the memory for duration. In Experiment 5, participants experience a low pain stimulation during the presentation of the standard duration. In Experiment 6, participants experienced a high pain stimulation during the presentation of the standard duration.

Chapter 8 aimed to establish an intervention to prevent the lengthening effect of pain on duration perception. In Experiment 7, participants completed a verbal estimation tasks before and after practicing 1-week mindfulness intervention to examine whether the intervention could reduce the perceived duration of pain. Experiment 8 also used the experimental design employed in Experiment 7 to examine whether mindfulness intervention could reduce the perceived duration of pain in a chronic pain population.

It is hoped that the findings of this thesis will illuminate the effect of pain on time perception, which will in turn progress our knowledge of human time perception.

### **4.3 – Pain induction techniques**

Two common pain-inducing techniques that previous research has used to affect time perception are electro-cutaneous stimulation (Fayolle et al., 2015) and thermal stimulation (Ogden, Moore, et al., 2014) (see section 3.1.3, page 57). The experiments here presented induced acute and long-lasting pain with different intensities to participants. Electro-cutaneous stimulation was selected to induce acute pain for its high degree of temporal and intensity acuity; electro-cutaneous

equipment controls the duration of the stimulation to the millisecond and microampere range. The same temporal acuity could not be obtained using other means such as thermal stimulation, which requires time to reach the target temperature. However, a state of pain could not be achieved through electro-cutaneous stimulation for safety reasons; electrical stimulation in the range of minutes has high risks of inducing tissue damage. Thermal stimulation was therefore selected to achieve a state of pain for two main reasons: 1) it is possible to maintain thermal painful stimulation for several minutes without risking skin damage; and 2) the state of pain was not required to have a millisecond precision.

The intensity of the pain stimulation varied depending on the study and/or the condition and the intensities were based on the participants' subjective experience rather than the objective intensity of the stimulation. The subjective intensities were therefore selected through an intensity rating task prior to the timing task, where participants rated the pain sensations through a Numeric Rating Scale (NRS, Jensen & McFarland, 1993). Below there is the description of the equipment and of the intensity rating task procedures for the electro-cutaneous and thermal pain.

#### *4.3.1 – Electro-cutaneous pain (Digitimer DS7A Current Stimulator)*

A Digitimer DS7A Current Stimulator (Digitimer Ltd) was used to present the electro-cutaneous stimulation (Figure 4.1). This equipment provides up to 100mA current intensity at up to 400V voltage. In the experiments that required the Digitimer DS7A (Experiments 1, 3, 7 and 8), two lubricated Fukuda standard Ag/AgCl electrodes (1cm diameter) electrodes were placed on the left volar forearm of participants 10cm from the wrist and the stimulation consisted of a train of 2ms pulses at 300V, which were repeated for the desired duration. Current intensity varied depending on the condition and/or study.

The required intensities were selected through an intensity rating task prior the timing task. Participants were informed that their task was to use an 11-point NRS 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. The electro-cutaneous stimulus was then increased at a rate of 0.20mA per trial until participants indicated the scores required by the study on the NRS (e.g., 6), or until the stimulus intensity reached 4.0mA. If at the initial level of intensity participants experienced pain and if the study required a non-painful intensity, 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 participants were asked to rate again the initial stimulus of 0.20mA before increasing the stimulus at a rate of 0.20mA per trial to reach the other required intensities. The task was administered using E-Prime software (<http://www.pstnet.com>).

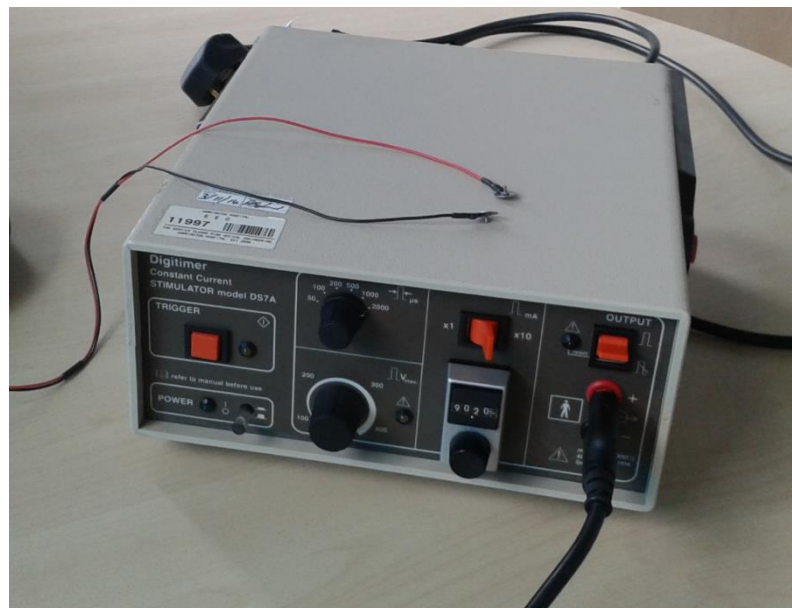


Figure 4.1. Digitimer DS7A Current Stimulator.

#### 4.3.2 – Thermal pain (Method PATHWAY-Advanced Thermal Stimulator)

A Medoc PATHWAY-Advanced Thermal Stimulator was used to present thermal stimulation (Figure 4.2). This equipment is designed for use in clinical and research

settings, and induces thermal stimulation through a thermode placed on the skin. Specialist hardware and software, designed for experimental purposes, delivered and controlled the temperature of the thermode. In the experiments that required the Medoc (Experiments 2, 4, 5 and 6), thermal pain was induced through a 30 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. In all the experiments that required the Medoc, thermal stimulation was induced through a 30 x 30mm Peltier thermode attached to the participants' left volar forearm. Temperature of the thermode varied depending on the condition and/or study.

The required intensities were selected through an intensity rating task prior to the timing task. A search protocol was used to establish subjective intensity levels of stimulation that were then used during the timing task. Participants were informed that their task was to use the 11-point NRS (0 = no pain at all, 10 = worst pain imaginable) to identify the intensities of thermal stimulation required by the study (e.g., 0 and 6). 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 0.1°C occurred. Participants' aim was to increase the temperature until reaching the first intensity required by the study on the NRS (e.g., 0). 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 next target intensity level on the NRS (e.g., 6). 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.



*Figure 4.2. Medoc PATHWAY-Advanced Thermal Stimulator.*

#### **4.4 – Temporal perception assessment**

The studies here reported have a consistent structure: participants completed a timing task where participants estimated the duration of painful stimuli or the duration of neutral stimuli during a state of pain. The verbal estimation task was chosen as the timing task for the majority of the studies in the present thesis. This paradigm consists of presenting stimuli to participants who have to verbally indicate their duration (generally in seconds or milliseconds). The verbal estimation task is one of the first procedures used in the time perception literature (Fraisse, 1964) and it has been later used in numerous studies (Gil & Droit-Volet, 2012; Jones, Poliakoff, & Wells, 2009; Ogden et al., 2015; Ogden, Moore, et al., 2014; Penton-Voak et al., 1996). This technique has the advantage over other techniques (e.g., timing reproduction tasks) of not involving the timing of a motor response that can lead to limitations when estimating very short durations (Wearden, 2015a). Additionally, the verbal estimation task has a lower cognitive load than tasks such as the temporal generalisation task, which could be disrupted by the painful stimulation. This task has been therefore used in the present thesis maintaining the procedure consistent across the studies.

The verbal estimation task was substituted with the temporal generalisation task only in Experiments 5 and 6 (Chapter 7), which required participants to remember



the duration over a period of delay. The verbal estimation task, in fact, is not an efficient paradigm to test the memory for duration over a delay because participants apply a numerical value to the stimulus at its offset and then recall the numerical value after the delay. The temporal generalisation task, in contrast, requires participants to memorize the duration of a stimulus and then to indicate whether a series of comparison stimuli have the same duration. A numerical value therefore cannot help to accomplish the task. The temporal generalisation procedure is described in detail in Chapter 7 (see section 7.1.1.2, page 147).

## **PART II**

### **EXPERIMENTAL STUDIES**

## Chapter 5

### Perceived duration of pain and in a state of pain

Chapters 2 and 3 described the effects of emotion on subjective timing. A consistent finding is that threatening events lengthen perceived duration, whether using images (Gil & Droit-Volet, 2012), facial expressions (Tipples, 2008), sounds (Noulhiane et al., 2007) or pain (Fayolle et al., 2015). These effects are frequently explained through an arousal hypothesis: threatening events lengthen perceived duration because they increase arousal (see section 2.3.1, page 47). The arousal hypothesis, although widely discussed, lacks of appropriate testing (see Lake et al., 2016 for discussion) in terms of the effects of 1) different levels of arousal and 2) different sources of arousal on perceived duration.

A key issue for the arousal hypothesis is that previous studies have used neutral and high arousing stimuli rather than a range of arousal levels. For example, Ogden et al. (2014) showed that people give different verbal estimates for pain and non-pain related events, but did not test whether different pain intensities (e.g., high pain vs low pain) have different effects on perceived duration. The arousal hypothesis predicts that greater arousal levels are related to longer perceived durations. If the arousal hypothesis is correct, we would expect to see different degrees of temporal distortion for different degrees of arousal.

A further issue is that previous research has predominantly investigated the arousal hypothesis using arousing stimuli as the to-be-timed stimuli. For example, participants' task is to report the duration of a negative image or painful stimulus. In these studies, the arousal is task-relevant as it is also the to-be-timed event. Few studies, however, have examined the effect of arousal when it arises from sources other than the to-be-timed event and is therefore task-irrelevant. Arousal hypothesis and other models of timing (SET, SBF and Craig's model) all suggest that *any* change in arousal should influence perceived duration. At present, they do not suggest that arousal needs to be task-relevant to timing to induce temporal distortions. If these

suggestions are correct then task-relevant and task-irrelevant sources of arousal should have comparable effects on perceived duration.

However, the findings of studies investigating the effect of task-irrelevant arousal are often inconsistent. For example, Droit-Volet et al. (2011) found that a fear inducing film led to longer perceived durations of neutral stimuli in a subsequent temporal bisection task; meanwhile, Ogden et al. (2015) found no effect of an unpleasant tactile stimulation on the perceived duration of concurrent neutral stimuli. Furthermore, van Hedger et al. (2017) found that the physiological activation induced by a social stress test was related to the temporal reproductions of negative stimuli, but not to the temporal reproductions of positive and neutral stimuli.

Some of these contrasting findings may be due to methodological issues: in Droit-Volet et al. (2011) and van Hedger et al. (2017) there was an absence of a clear state change between the learning and testing phases on the bisection and reproduction tasks making interpretation of the findings difficult. The use of stimuli with semantic content (e.g., IAPS images) also means that there were differences between the physical properties of the arousing and neutral stimuli, such as complexity and familiarity, which may have influenced the results (Lake et al., 2016). Although many of these issues were overcome in Ogden et al. (2015) through the use of a to-be-timed stimuli with no semantic content that did not change across conditions, they did not assess the arousal change induced by the tactile stimulation that they employed. It is therefore possible that the unpleasant stimulation used did not increase arousal enough to induce temporal distortions. Due to these issues, it is therefore unclear whether the effects of arousal on perceived duration are limited to circumstances in which the source of arousal is the to-be-timed event (i.e., from task-relevant sources), or whether duration perception is affected by any arousal change per se (i.e., from task-relevant and task-irrelevant sources). It is also unclear whether the effects of arousal on perceived duration are limited to emotional stimuli (e.g., negative images), or whether duration perception is also affected by non-emotional stimuli (e.g., vibration). Establishing more precisely the circumstances in which

arousal does and does not affect perceived duration is critical for understanding the mechanisms by which time distorts.

The experiments reported in this Chapter aimed to further our understanding of the effect of arousing stimuli on perceived duration. Specifically, the experiments aimed to establish 1) whether differing levels of arousal produced different distortions to time. 2) Whether changes in the intensity of neutral stimuli distorted time in a comparable way to changes in the intensity of emotional stimuli. 3) Whether task-relevant and task-irrelevant sources of arousal had different effects on temporal perception. Two experiments were conducted. In the first, participants completed a series of verbal estimation tasks in which they were asked to estimate the duration of electro-cutaneous stimuli at no pain, low pain and high pain intensities and vibrotactile stimuli at perceptible vibration, low vibration and high vibration intensities. Arousal in these tasks was therefore task-relevant because it originated from the to-be-timed stimulus. Comparison of the estimates for the different intensities tested whether differing levels of arousal produced different distortions to time. Comparison of the emotional (electrocutaneous) and neutral (vibrotactile) tasks tested whether changes in the intensity of neutral stimuli distorted time in a comparable way to changes in the intensity of emotional stimuli.

In Experiment 2, participants completed a series of verbal estimation tasks in which they were asked to estimate the duration of neutral stimuli while concurrently experiencing thermal stimulation at no pain, low pain and high pain intensities. Here, pain was task-irrelevant because it originated from a source other than the to-be-timed stimulus. Comparison of the findings of Experiments 1 and 2 would establish whether task-relevant and task-irrelevant sources of arousal had different effects on temporal perception. In all tasks measure of mean verbal estimates, estimate accuracy and estimate variability were calculated.

It was expected that intensity increases would be associated with analogous verbal estimate increases; the high vibration intensity was expected to lead to longer verbal

estimates than the low vibration intensity, which in turn was expected to lead to longer verbal estimates than perceptible vibration. Similarly, the high pain intensity was expected to lead to longer verbal estimates than the low pain intensity, which in turn was expected to lead to longer verbal estimates than the no pain intensity. This was expected in both Experiments 1 and 2, confirming arousal hypothesis.

## **5.1 – Experiment 1**

### *5.1.1 – Method*

#### 5.1.1.1 – Participants

Thirty participants (22 females; mean age = 23.03, SD = 4.26) were recruited. Sample size was determined by examining those used in previous research investigating the effect of pain on time perception (Rey et al., 2017; Ogden et al., 2014). The same approach was also applied in the following studies of the present thesis. Participants were required not to be pregnant, not to have a history of epilepsy and not to have chronic pain, heart disease, skin problems (e.g., eczema) 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 £15 for taking part. The study was approved by the Liverpool John Moores University ethics committee and informed consent was obtained from all participants.

#### 5.1.1.2 – Apparatus and materials

*Pain stimulation:* The Digitimer DS7A Current Stimulator (Digitimer Ltd) was used to present the electro-cutaneous stimulation (see section 4.3.1, page 77 for equipment description).

*Tactile vibrations:* A tactile pulse (vibration) was used as the non-noxious stimulus. Tactile vibrations (150Hz square wave vibrations) were produced by sending amplified sound files (using a TactAmp, 4.2, Dancer Design, St Helens, UK), controlled via E-Prime software (Psychology Software Tools, Inc., Pittsburgh, PA, USA), to a tactor. The tactor had a diameter of 18mm (Dancer Design, St Helens, UK) and was

attached to the participants forearm (10cm from the wrist) with a double-sided adhesive pad. The intensity of the stimulus was determined modifying the amplitude (0 to 1) of the tone.

#### 5.1.1.3 – Procedure

Participants completed a health-screening questionnaire to confirm suitability to participate. Each participant then completed two experimental conditions where vibrotactile or electro-cutaneous stimulation were delivered. Conditions were presented to participants in a counterbalanced order. Each condition included an initial intensity rating task, a verbal estimation task and a follow-up intensity rating task (described below). Throughout all tasks, participants listened to white noise through headphones to prevent any auditory feedback influencing performance. All tasks were administered using E-Prime software (<http://www.pstnet.com>).

##### Pain Condition

*Initial intensity rating task:* Participants performed the task with the Digitimer DS7A to select electrical intensities equal to 0, 3 and 6 on the NRS; that is no pain, low pain (Serlin, Mendoza, Nakamura, Edwards, & Cleeland, 1995) and high pain (Khoshnejad, Martinu, Grondin, & Rainville, 2016), respectively (see section 4.3.1, page 77 for task description).

*Verbal estimation task:* The verbal estimation task employed a 3 (intensity: no pain, low pain and high pain) x 5 (duration: 242ms, 455ms, 767ms, 1058ms and 1296ms) repeated measures design. Participants were informed that a series of stimuli would be presented to their forearm, lasting between 50ms and 1700ms, and that their task was to estimate the duration of each stimulus in milliseconds, by typing a numerical value using the keyboard. Participants completed nine blocks of trials in total, with three blocks per intensity (no pain, low pain and high pain). Block order was counterbalanced between participants. Each block contained 16 stimuli; five

standard duration (242ms, 455ms, 767ms, 1058ms and 1296ms) each of which was repeated twice and six 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 five experimental stimulus durations across the experimental blocks. The data from these six additional stimuli were not analysed (as in Ogden et al., 2015). A inter trial interval, the duration of which was selected from a normal distribution between 1500 to 2500 milliseconds, was interposed between trials. The order of presentation of the trials was randomised by E-Prime for each participant. A total of 144 trials were delivered across the entire task. To prevent habituation or hypersensitivity to the stimulation, the location of the electrodes on the forearm (originally placed 10cm far from the wrist) was altered by 1cm between each block.

*Follow-up intensity rating task:* To establish the perceived intensity of the experimental stimuli at the end of the testing session, participants were asked re-rate the three intensity levels (no pain, low pain and high pain) using the 11-point NRS from in the initial rating task. Each intensity was presented five times for 750ms, in a random order, for a total of 15 stimuli.

#### Vibration Condition

*Initial intensity rating task:* Participants selected three vibration intensities (perceptible vibration, low vibration and high vibration) for use in the subsequent verbal estimation task. Participants were asked to rate the intensity of a series of vibrations using an 11-point NRS (0 – no vibration sensation; 10 – the maximum stimulation that the machine was capable of producing). To familiarise participants with a sensation which would score 10, participants were given three, 750ms stimulations at the maximum amplitude (1). Participants were informed that 3 represented a low intensity vibration and 6 a high intensity vibration. An initial stimulus amplitude of 0.05 for 750ms was then presented and stimulus amplitude was then increased at a rate of 0.05 per trial until a score of 6 on the NRS was achieved. The three stimulus intensities selected for the verbal estimation task were:



the last stimulus rated as 1 on the NRS (perceptible), the first stimulus rated as 3 (low vibration), and the first stimulus rated as 6 (high vibration).

*Verbal estimation task and follow-up intensity rating task:* Participants completed the same procedure used in the pain condition. Only the perceptible, low and high vibration intensities were used.

### 5.1.2 – Results

Out of 30 participants tested, data from three participants were excluded from the analysis because one participant withdrew from the experiment because of excessive pain, and two participants appeared to have not been able to detect the stimulus for an entire block of the verbal estimation task. Therefore, it has been reported the results based on data from the remaining 27 participants. To establish the effect of the pain on time perception, three measures of performance were derived for each condition: mean verbal estimates, estimate accuracy and estimate variability (see Wearden, 1999 for further details). Greenhouse-Geisserr correction was applied to ANOVAs when the Sphericity assumption was violated and post-hoc were Bonferroni corrected.

#### Pain Condition

<i>Pain intensity</i>	<i>Electrical current</i>	<i>Follow-up rating</i>	<i>Verbal estimate</i>	<i>Estimate accuracy</i>	<i>Estimate variability</i>
<i>No pain</i>	0.44 (0.38)	0.24 (0.67)	437.00 (196.93)	0.59 (0.05)	0.61 (0.04)
<i>Low pain</i>	1.21 (0.88)	1.47 (1.11)	566.14 (228.43)	0.76 (0.06)	0.41 (0.03)
<i>High pain</i>	2.39 (1.26)	4.27 (1.68)	683.85 (250.55)	0.93 (0.07)	0.34 (0.02)

*Table 5.1. Means (and standard deviations) of electrical current (mA), follow-up pain ratings, mean verbal estimate of all durations (ms), estimate accuracy and estimate variability for the pain condition.*

Table 5.1 shows the intensity current (mA) for the three pain intensities used during the verbal estimation task. Repeated measures ANOVA showed a significant effect of stimulus intensity on intensity current  $F(1.32, 34.24) = 89.80, p < .001, \eta_p^2 = .78$ ; post-hoc tests showed that intensity current values were significantly different in all intensities (all  $ps < .001$ ). Repeated measures ANOVA confirmed that the follow-up pain intensity ratings were also significantly different in all intensities  $F(1.39, 36.07) = 106.58, p < .001, \eta_p^2 = .80, p < .001$  for all post-hoc tests.

### *Verbal estimates*

Figure 5.1 (panel A) shows mean verbal estimates for each stimulus duration, in each pain intensity. Inspection of Figure 5.1 (panel A) and Table 5.1 suggests that the longest verbal estimates were given in the high pain intensity and the shortest verbal estimates were given in the no pain intensity. Moreover, the differences in verbal estimates seem to increase with stimulus durations. A repeated measures ANOVA with pain intensity (no, low, high) and experimental stimulus duration (242ms, 455ms, 767ms, 1058ms, 1296ms) as within subject factors, showed a significant main effect of stimulus duration ( $F(1.28, 33.34) = 88.77, p < .001, \eta_p^2 = .77$ ) and of pain intensity ( $F(1.60, 4.63) = 30.00, p < .001, \eta_p^2 = .54$ ) on verbal estimate. Post-hoc tests showed that estimates were significantly longer in the high pain intensity than in the low pain ( $p = .002$ ) and no pain intensity ( $p < .001$ ). Estimates were also significantly longer in the low pain intensity than in no pain one ( $p < .001$ ). There was also a significant interaction between stimulus duration and pain intensity ( $F(8, 208) = 10.52, p < .001, \eta_p^2 = .29$ ). When each stimulus duration was tested separately with a series of repeated measures ANOVAs, verbal estimates were significantly different across intensities (all  $ps < .05$ ) with the only exception of verbal estimates of the shortest duration (242ms). Verbal estimates of the 242ms duration in the low intensity were not significantly different than estimates in the high intensity ( $p = .14$ ) or in the no pain intensity ( $p = .14$ ). Verbal estimates therefore increased with pain intensity and greater time distortions occurred with longer durations.

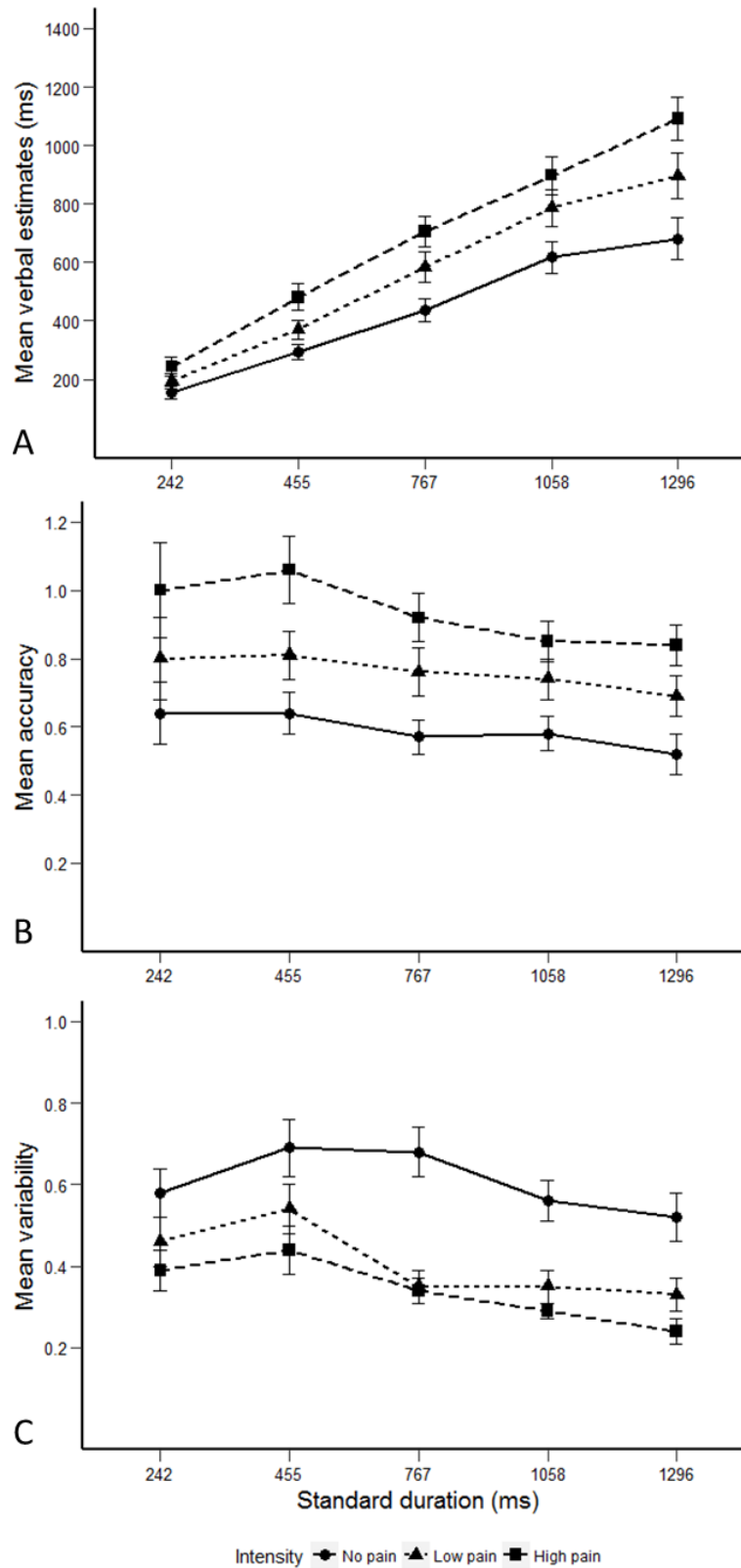


Figure 5.1. Means (and standard errors) of (A) verbal estimates (ms), (B) estimate accuracy and (C) estimate variability for each standard duration in each pain intensity (no pain, low pain and high pain) in Experiment 1.

To explore the interaction between stimulus duration and pain intensity further, slope and intercept of verbal estimates were calculated by conducting individual linear regressions on the mean verbal estimates produced by each participant for each condition. This allowed to examine whether the lengthening effect of pain on verbal estimates was multiplicative (slope) or additive (intercept) (see sections 1.1.3.1.1 and 1.1.3.1.2, page 23 and 26). Inspection of Table 5.2 suggests that pain intensity increases were associated with a steeper slope and greater intercept. A repeated measures ANOVA with pain intensity (no, low, high) as within subject factors confirmed a significant main effect of pain intensity on slope ( $F(2, 52) = 14.22$ ,  $p < .001$ ,  $\eta_p^2 = .35$ ). Post-hoc tests showed that slope was significantly steeper in the high pain intensity than in the low pain ( $p = .014$ ) and no pain intensity ( $p < .001$ ). Slope was also significantly steeper in the low pain intensity than in the no pain intensity ( $p = .034$ ). However, a repeated measures ANOVA with pain intensity (no, low, high) as within subject factors showed no significant effect of pain intensity on slope ( $F(2, 52) = 1.25$ ,  $p = .30$ ,  $\eta_p^2 = .05$ ). Pain intensity increases therefore affected slope but not intercept, suggesting that the lengthening effect of pain on verbal estimates was multiplicative rather than additive.

<i>Pain intensity</i>	<i>Slope</i>	<i>Intercept</i>
<i>No pain</i>	0.55 (0.36)	48.98 (151.38)
<i>Low pain</i>	0.67 (0.39)	60.54 (182.03)
<i>High pain</i>	0.78 (0.35)	90.55 (213.64)

Table 5.2. Means (and standard deviations) of slope and intercept for the pain condition.

#### *Estimate accuracy*

Estimate accuracy was calculated using the following formula: mean verbal estimate/stimulus duration. An accuracy of 1 indicates a correct estimate, below 1

indicates underestimation of duration and above 1 indicates overestimation of duration. Figure 5.1 (panel B) and Table 5.1 suggest that more accurate estimates were given in the high pain intensity condition compared to the no and low intensity. A repeated measures ANOVA with pain intensity and stimulus duration as within subject factors, showed a significant main effect of pain intensity ( $F(1.62, 41.98) = 24.99, p < .001, \eta_p^2 = .49$ ) but no significant effect of stimulus duration ( $F(1.36, 35.39) = 1.73, p = .20, \eta_p^2 = .06$ ) on estimate accuracy. Post-hoc tests showed that estimates were significantly more accurate in the high pain intensity than in the low pain intensity ( $p = .003$ ) and no pain intensity ( $p < .001$ ). Estimates were also significantly more accurate in the low pain intensity than in the no pain intensity ( $p < .001$ ). There was no significant interaction between stimulus duration and pain intensity ( $F(4.25, 110.47) = 1.03, p = .40, \eta_p^2 = .04$ ). Estimate accuracy therefore increased with pain intensity.

#### *Estimate variability*

Estimate variability was calculated using the following formula: standard deviation verbal estimate/mean verbal estimate. The higher the value, the more variable the participants' responses. Figure 5.1 (panel C) and Table 5.1 suggest that verbal estimates had higher variability in the no pain intensity compared to the low and high pain intensities. A repeated measures ANOVA with pain intensity and experimental stimulus duration as within subject factors, showed a significant main effect of stimulus duration ( $F(2.42, 63.00) = 5.04, p = .006, \eta_p^2 = .77$ ) and of pain intensity ( $F(2, 52) = 32.34, p < .001, \eta_p^2 = .55$ ) on variability. Post-hoc tests showed that variability was significantly higher in the no pain intensity than in the low pain ( $p < .001$ ) and high pain intensities ( $p < .001$ ). Variability was not significantly different in the low pain and high pain intensities ( $p = .10$ ). Additionally, there was no significant interaction between stimulus duration and pain intensity ( $F(4.48, 116.54) = 1.51, p = .20, \eta_p^2 = .06$ ). Verbal estimates were therefore more variability in the no pain intensity than in the pain intensities.

Vibration Condition

<i>Vibration intensity</i>	<i>Amplitude</i>	<i>Follow-up rating</i>	<i>Verbal estimate</i>	<i>Estimate accuracy</i>	<i>Estimate variability</i>
<i>Perceptible vibration</i>	0.13 (0.04)	0.56 (0.67)	316.04 (196.32)	0.42 (0.05)	0.71 (0.09)
<i>Low vibration</i>	0.29 (0.08)	1.90 (0.92)	549.45 (239.46)	0.71 (0.06)	0.38 (0.03)
<i>High vibration</i>	0.49 (0.09)	4.62 (2.22)	558.24 (212.43)	0.71 (0.05)	0.36 (0.02)

*Table 5.3. Means (and standard deviations) of vibration amplitude, follow-up vibration ratings, mean verbal estimate of all durations (ms), estimate accuracy and estimate variability during the vibration condition.*

Table 5.3 shows the vibration intensity (amplitude) for the three intensity levels used during the verbal estimation task. Repeated measures ANOVA showed a significant effect of stimulus intensity on stimulus amplitude ( $F(2, 52) = 329.42, p < .001, \eta_p^2 = .93$ ) post-hoc tests showed that amplitude values were significantly different in all intensities ( $ps < .001$ ). Repeated measures ANOVA confirmed that follow-up stimulus intensity ratings were also significantly different in all intensities  $F(1.12, 29.19) = 86.86, p < .001, \eta_p^2 = .77, p < .001$  for all post-hoc tests.

*Verbal estimates*

Figure 5.2 (panel A) shows mean verbal estimates for each vibration intensity. Inspection of the panel and Table 5.3 suggests that the shortest duration estimates were given in the perceptible intensity, but there was no difference in estimates given for the low and high intensities. A repeated measures ANOVA with vibration intensity (perceptible, low and high) and stimulus duration (242ms, 455ms, 767ms, 1058ms, 1296ms) as within subject factors, showed a significant main effect of stimulus duration ( $F(1.41, 36.66) = 117.68, p < .001, \eta_p^2 = .82$ ) and of vibration intensity ( $F(1.26, 32.75) = 36.80, p < .001, \eta_p^2 = .59$ ) on verbal estimates. Post-hoc tests showed that

estimates were significantly shorter in the perceptible vibration intensity than in the low vibration ( $p < .001$ ) and high vibration intensities ( $p < .001$ ). Estimates were not significantly different in the low and high vibration intensities ( $p > .99$ ). There was a significant interaction between stimulus duration and vibration intensity ( $F(3.49, 90.76) = 13.61, p < .001, \eta_p^2 = .34$ ). When each stimulus duration was tested separately with a series of repeated measures ANOVAs, verbal estimates appeared to be shorter in the perceptible intensity compared to the low intensity (all  $ps < .002$ ) and high intensities (all  $ps < .001$ ) which, in turn, did not differ between each other (all  $ps > .23$ ). The only exception were estimates of the shortest stimulus (242ms) which did not differ across vibration intensities ( $F(1.45, 37.64) = 1.32, p = .27, \eta_p^2 = .05$ ). Verbal estimates were therefore longer in the high and low vibration intensities compared to the perceptible vibration intensity. Moreover, the effect of vibration intensity increased with stimulus duration.

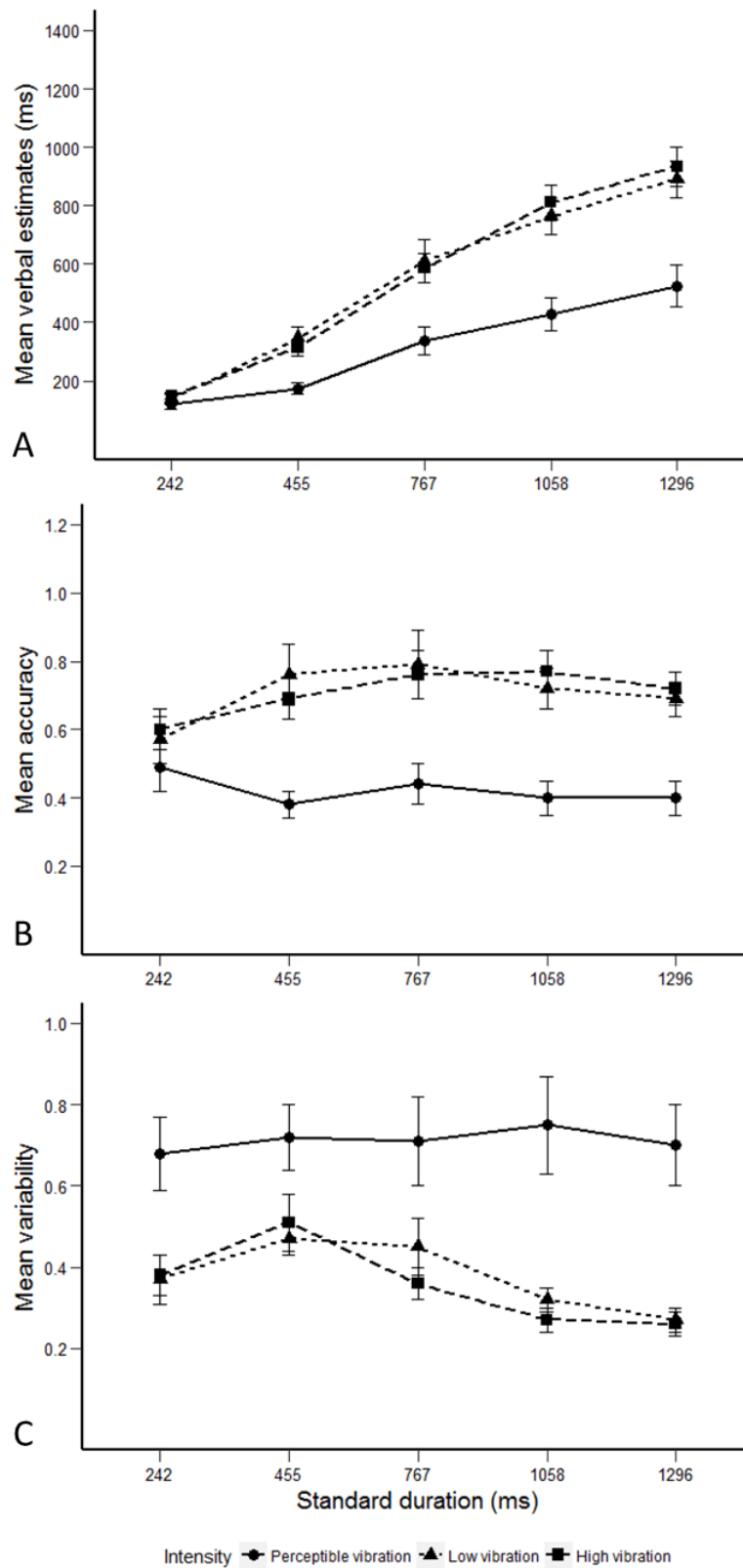


Figure 5.2. Means (and standard errors) of (A) verbal estimates (ms), (B) estimate accuracy and (C) estimate variability for each standard duration in each vibration intensity (perceptible vibration, low vibration and high vibration) in Experiment 1.



### *Estimate accuracy*

Examination of Figure 5.2 (panel B) and Table 5.3 also suggest that more accurate estimates were given for the high and low vibration intensities compared to the perceptible intensity. A repeated measures ANOVA with vibration intensity and experimental stimulus duration as within subject factors, showed a significant main effect of vibration intensity ( $F(1.31, 33.96) = 29.35, p < .001, \eta_p^2 = .53$ ) but no significant effect of stimulus duration ( $F(1.72, 44.77) = 2.22, p = .13, \eta_p^2 = .08$ ) on estimate accuracy. Post-hoc tests showed that estimates were significantly less accurate in the perceptible vibration intensity than in the low vibration ( $p < .001$ ) and high vibration intensity ( $p < .001$ ). Estimate accuracy was not significantly different between the low and high vibration intensities ( $p > .99$ ). There was a significant interaction between stimulus duration and vibration intensity ( $F(4.07, 105.74) = 4.17, p = .003, \eta_p^2 = .14$ ). When each stimulus duration was tested separately with a series of repeated measures ANOVAs, verbal estimates appeared to be less accurate in the perceptible intensity compared to estimates in low intensity (all  $ps < .002$ ) and high intensities (all  $ps < .001$ ) which, in turn, did not differ between each other (all  $ps > .23$ ). The only exception were estimates of the shortest stimulus (242ms) which did not differ between vibration intensities ( $F(1.45, 37.64) = 1.32, p = .27, \eta_p^2 = .05$ ). Estimate accuracy was therefore lower in the perceptible vibration intensity than in the high and low vibration intensities, except for the shortest duration (242ms).

### *Estimate variability*

Figure 5.2 (panel C) and Table 5.3 suggest that estimates had higher variability in the perceptible intensity compared to the low and high vibration intensities. A repeated measures ANOVA with vibration intensity and experimental stimulus duration as within subject factors, showed a significant main effect of stimulus duration ( $F(2.52, 65.43) = 3.58, p = .009, \eta_p^2 = .12$ ) and of vibration intensity ( $F(1.16, 30.17) = 17.38, p < .001, \eta_p^2 = .40$ ) on variability. Post-hoc tests showed that variability was significantly greater in the perceptible vibration intensity than in the low

vibration ( $p = .001$ ) and high vibration intensities ( $p < .001$ ). Variability did not differ between the low and high vibration intensities ( $p > .99$ ). There was no significant interaction between stimulus duration and vibration intensity ( $F(3.50, 90.99) = 1.65$ ,  $p = .18$ ,  $\eta_p^2 = .06$ ). Verbal estimates were therefore more variability in the perceptible vibration intensity than in the low and high vibration intensities.

### 5.1.3 – Discussion

Experiment 1 tested whether different levels of task-relevant arousal produce different distortions to time and whether changes in neutral stimulus intensity distorts time in a comparable way to changes in emotional stimulus intensity. This was achieved by examining the perceived duration of electro-cutaneous and vibrotactile stimulations at different intensities. For both stimulations, it was expected a positive relationship between intensity and perceived duration.

As expected, pain intensity increases were associated with analogous verbal estimate increases; verbal estimates were longer in the high pain intensity than in the low and no pain intensities, and verbal estimates were longer in the low pain intensity than in the no pain intensity. Moreover, the lengthening effect of pain was greater for longer durations than for shorter ones and pain intensity increases were associated with greater slope values. In contrast, pain did not affect intercepts. This suggests that pain affects verbal estimates with a multiplicative effect, which is thought to be the index of the arousal effect on pacemaker in SET (however see Williams et al., 2017). These findings were compatible with the arousal hypothesis and replicate previous work (Fayolle et al., 2015; Rey et al., 2017). Pain intensity increases were also associated with more accurate and less variable estimates. This was compatible with Ogden et al.'s (2014) study showing greater estimate accuracy for pain related stimuli than for neutral stimuli.

As expected, changes in the intensity of a neutral stimulus also affected perceived duration, although this was limited to a difference between the perceptible intensity

condition and low and high intensity conditions; perceived durations were not different between the low and high vibration intensities. Estimate accuracy was also higher and estimate variability was lower in the low and high vibration intensities than in the perceptible vibration intensity but did not differ between low and high vibration intensity. One possibility is that this finding suggests that arousal increases originating from a task-relevant neutral source also have the capacity to distort time, albeit less effectively than pain intensity increases. This finding is compatible with previous studies showing that increasing the intensity of neutral tone lengthens its perceived duration (Matthews, Stewart & Wearden, 2011). Furthermore, given that changes in pain intensity lead to greater arousal variations, it is reasonable that increasing the intensity of a neutral stimulus had a reduced effect on perceived duration compared to increasing the intensity of a painful stimulus.

However, it is also possible that the difference in estimates between the perceptible condition and other conditions is a result of an absence of consistent perception of the entire “just-perceptible” stimuli. Indeed, it should be noted that in the follow-up ratings, the perceptible vibration was rated 0.56, where 0 corresponded to no vibration. It is therefore possible that the perceptible vibration was close to the detection threshold, making the stimulation difficult to detect and causing the less accurate and more variable estimates in this vibration intensity. Furthermore, it is also possible that the difference between the low vibration intensity and the high vibration intensity was simply too little to induce sufficiently different arousal levels, resulting in similar time judgments between the two conditions.

Experiment 1 shows that for task-relevant sources of arousal, greater stimulus intensity is associated with greater temporal distortion. Experiment 2 therefore tested whether comparable effects would be observed with task-irrelevant sources of emotional arousal. In Experiment 2, participants completed three verbal estimation tasks in which they had to estimate the duration of a neutral visual stimulus while experiencing a concurrent constant thermal stimulation on their arm.

The thermal stimulation was set at no pain, low pain and high pain intensity. Measures of verbal estimate, estimate accuracy and estimate variability were calculated. As in Experiment 1, pain intensity increases were expected to lead to longer verbal estimates.

## **5.2 – Experiment 2**

### *5.2.1 – Method*

#### 5.2.1.1 – Participants

Thirty participants (17 females and 13 males; aged between 20 and 35 years old) were recruited. Participants were required not to be pregnant, not to have a history of epilepsy and not to have chronic pain, heart disease, skin problems (e.g., eczema) 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.

#### 5.2.1.2 – Apparatus and materials

*Pain stimulation:* The Medoc PATHWAY-Advanced Thermal Stimulator was used to present the sustained thermal stimulation (see section 4.3.2, page 78 for equipment description).

#### 5.2.1.3 – Procedure

Participants were initially asked to complete a health screening questionnaire to confirm their suitability to participate. Participants then performed an intensity rating task with the Medoc PATHWAY-Advanced Thermal Stimulator to select thermal intensities equal to 0, 3 and 6 in the NRS; that is warm but no pain, low pain

(Serlin et al., 1995) and high pain (Khoshnejad et al., 2016), respectively (see section 4.3.2, page 78 for task description).

Participants then completed a verbal estimation task, which employed a 3 (intensity: no pain, low pain and high pain) x 5 (duration: 242ms, 455ms, 767ms, 1058ms and 1296ms) repeated measures design. Participants were informed that a series of visual stimuli (white squares on a black background, 300x300 pixels, 8x8cm) would be presented on the screen, lasting between 50ms and 1700ms, and that their task was to estimate the duration of each stimulus in milliseconds, by typing a numerical value using the keyboard. Participants were also informed that the thermode on their forearm would be active during the task.

Participants completed nine blocks of trials in total, with three blocks per intensity (no pain, low pain and high pain). The thermode was active during the entire block (about 2 minutes). Block order was counterbalanced between participants. Each block contained 16 stimuli; five standard duration (242ms, 455ms, 767ms, 1058ms and 1296ms) each of which was repeated twice and six 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 five experimental stimulus durations across the experimental blocks. The data from these six additional stimuli were not analysed (as in Ogden et al., 2015). A inter trial interval, the duration of which was selected from a normal distribution between 1500 to 2500 milliseconds, was interposed between trials. The order of presentation of the trials was randomised by E-Prime for each participant. A total of 144 trials were delivered across the entire task.

### *5.2.2 – Results*

Out of 30 participants tested, five chose temperatures too low to be considered painful (i.e., below 40°C as high pain) (Yarnitsky, Sprecher, Zaslansky, & Hemli, 1995) and one gave temporal estimates below 100 or above 1000. Therefore, reported results are based on data from the remaining 24 participants. To establish the effect

of the pain on time perception, three measures of performance were derived for each condition as in Experiment 1: mean verbal estimates, estimate accuracy and estimate variability. Greenhouse-Geisserr correction was applied to ANOVAs when the Sphericity assumption was violates and post-hoc were Bonferroni corrected.

<i>Pain intensity</i>	<i>Temperature</i>	<i>Verbal estimate</i>	<i>Estimate accuracy</i>	<i>Estimate variability</i>
<i>No pain</i>	36.58 (2.28)	558.02 (187.70)	0.74 (0.29)	0.66 (0.16)
<i>Low pain</i>	41.50 (2.59)	535.53 (180.04)	0.70 (0.26)	0.68 (0.16)
<i>High pain</i>	44.47 (1.95)	505.34 (199.47)	0.68 (0.30)	0.70 (0.19)

*Table 5.4. Means (and standard deviations) of temperature (°C), mean verbal estimate of all durations (ms), estimate accuracy and estimate variability in Experiment 2.*

Table 5.4 shows the temperature for the three pain intensities individuated during the initial intensity rating task. Repeated measures ANOVA showed a significant effect of stimulus intensity on temperature ( $F(2, 46) = 200.74, p < .001, \eta_p^2 = .90$ ),  $p < .001$  for all post-hoc tests.

#### *Verbal estimates*

Figure 5.3 (panel A) shows mean verbal estimates for each stimulus duration, in each pain intensity. Inspection of Figure 5.3 (panel A) and Table 5.4 suggests that the shortest verbal estimates were given in the high pain intensity and the similar verbal estimates were given in the no pain and low pain intensity. A repeated measures ANOVA with pain intensity (no, low, high) and experimental stimulus duration (242ms, 455ms, 767ms, 1058ms, 1296ms) as within subject factors was conducted. ANOVA analysis showed a significant main effect of stimuli duration ( $F(1.52, 34.90) =$

134.52,  $p < .001$ ,  $\eta_p^2 = .85$ ) and of pain intensity ( $F(2, 46) = 6.25$ ,  $p = .004$ ,  $\eta_p^2 = .22$ ) on verbal estimates. Post-hoc tests showed that estimates were significantly longer in the no pain intensity than in the high intensity ( $p = .010$ ). Verbal estimates in the low pain intensity were not significantly different than verbal estimates in the no pain ( $p = .17$ ) and high pain ( $p = .26$ ) intensities. There was no significant interaction between stimuli duration and pain intensity ( $F(3.91, 89.99) = 1.40$ ,  $p = .24$ ,  $\eta_p^2 = .06$ ).

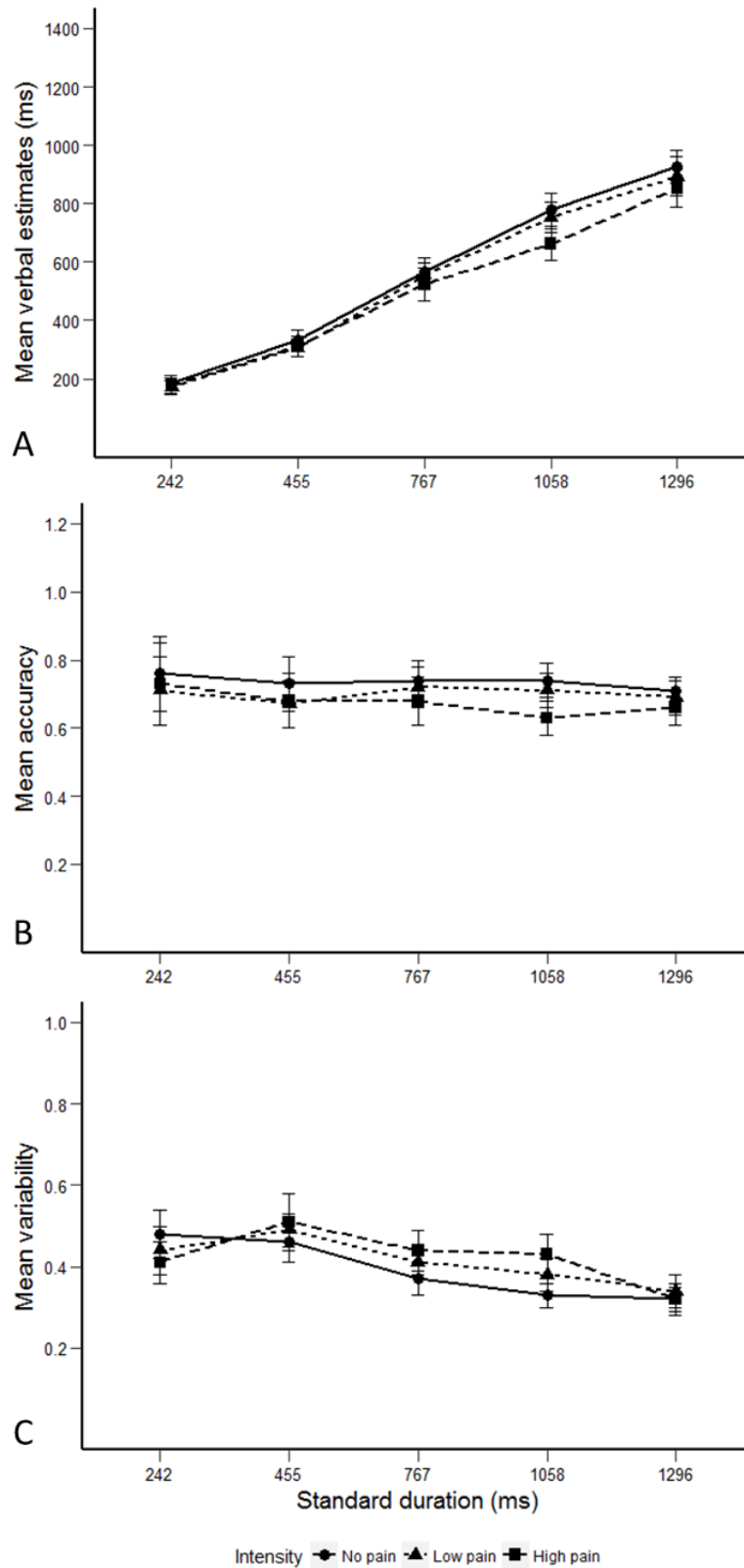


Figure 5.3. Means (and standard errors) of (A) verbal estimates (ms), (B) estimate accuracy and (C) estimate variability for each standard duration in each pain intensity (no pain, low pain and high pain) in Experiment 2.



### *Estimate accuracy*

Figure 5.3 (panel B) and Table 5.4 suggest that more accurate estimates were given in the no pain intensity condition compared to the high pain intensity. ANOVA with pain intensity and stimulus duration as within subject factors, showed a significant main effect of pain intensity ( $F(2, 46) = 3.83, p = .029, \eta_p^2 = .14$ ) but no significant effect of stimulus duration ( $F(1.24, 28.41) = .21, p = .71, \eta_p^2 = .01$ ) on estimate accuracy. Post-hoc tests showed that estimates were significantly more accurate in the no pain intensity than in the high pain intensity ( $p = .048$ ). Estimate accuracy in the low pain intensity was not significantly different from estimate accuracy in the no pain ( $p = .13$ ) and high pain ( $p = .98$ ) intensities. There was no significant interaction between stimulus duration and pain intensity ( $F(8, 184) = .64, p = .68, \eta_p^2 = .03$ ).

### *Estimate variability*

Figure 5.3 (panel C) and Table 5.4 suggest that verbal estimations of the shortest duration had higher variability in the high pain intensity compared to the no pain intensity. A repeated measures ANOVA with pain intensity and experimental stimulus duration as within subject factors, showed no significant main effect of stimulus duration ( $F(2.53, 55.73) = 2.68, p = .065, \eta_p^2 = .11$ ) and pain intensity ( $F(2, 44) = .47, p = .63, \eta_p^2 = .02$ ) on estimate variability. There was also no interaction effect between stimulus duration and pain intensity ( $F(8, 176) = 1.16, p = .33, \eta_p^2 = .05$ ).

### *5.2.3 – Discussion*

Experiment 2 tested whether different levels of task-irrelevant arousal produce different distortions to time. Participants estimated the perceived duration of neutral visual stimuli whilst experiencing concurrent constant thermal stimulation at no pain, low pain and high pain intensity. It was expected that the different levels of pain intensity would produce different effects on time perception, specifically, greater distortion in the high pain than low pain condition.

Contrary to expectations, however, Experiment 2 showed that rather than lengthening perceived duration, task-irrelevant high pain resulted in a shortening of perceived duration. Estimates were also more accurate in the no pain condition than the high pain condition, meanwhile estimate variability was unaffected by task-irrelevant stimulation. These findings contrast with the findings of Experiment 1 and with the arousal hypothesis, which predicts that any increase of arousal should be associated with an increase in the perceived duration (see section 3.3.1, page 67). Task-irrelevant arousal does not therefore appear to be related to perceived duration in the same way as task-relevant arousal. This suggests that the effects of arousal on perceived duration are more complex than suggested.

### **5.3 – Discussion Chapter 5**

Chapter 5 examined whether different levels of arousal produced different distortions to time when arousal arises from 1) a task-relevant emotional stimulus, 2) a task-relevant neutral stimulus and 3) a task-irrelevant emotional stimulus. This was tested in two experiments. Experiment 1 tested the estimate, accuracy and variability of electro-cutaneous and vibrotactile stimulations. Experiment 2 tested the estimate, accuracy and variability of neutral visual stimuli while experiencing constant thermal stimulation. In both experiments, it was expected that increasing the intensity of the stimulation would increase arousal, resulting in longer estimates.

Findings showed that increasing the intensity of electro-cutaneous, vibrotactile and thermal stimulation resulted in different time distortions depending on whether the source of arousal was task-relevant or task-irrelevant. Increases in task-relevant electro-cutaneous intensity were associated with longer, more accurate and less variable verbal estimates, which were different between each pain intensity. Increases in task-relevant vibrotactile intensity were also associated with longer, more accurate and less variable verbal estimates; however, there was no difference between low and high vibration intensity. Finally, task-irrelevant high thermal

intensity was associated with shorter and less accurate verbal estimates of the concomitant neutral stimulus; however, estimate variability was unaffected across pain intensities.

Experiment 1 findings showed that increases in task-relevant electro-cutaneous intensity were associated with longer verbal estimates. This confirmed previous studies showing that pain is an effective method to lengthen perceived duration (Fayolle et al., 2015; Ogden, Moore, et al., 2014). This supports the predictions of the arousal hypothesis, which suggests that increases in arousal due to increases of pain intensity are associated with longer perceived durations. Greater pain intensity was also associated with greater estimate accuracy and reduced estimate variability, again replicating Ogden et al. (2014). These later findings are perhaps surprising because increasing pain intensity has been found to disrupt attention (Moore et al., 2012) and executive functions (Moriarty et al., 2011), which are required for correct timing judgments (Ogden, Moore, et al., 2014). Therefore, rather than impairing timing, pain appears to result in a perceptual advantage for timing. This suggestion will be expanded in the next chapter.

Findings of Experiment 1 also showed that increases in vibration intensity were associated with longer verbal estimates, suggesting that arousal originated from task-relevant, non-emotional sources can affect time perception and supporting the predictions of the arousal hypothesis. However, interpretation of the findings is difficult due to the potential of inconsistent perceptions of the perceptible condition and a lack of sufficient difference between the low and high vibration conditions affecting the findings.

In Experiment 2 however, task-irrelevant pain intensity increases had a shortening effect on the perceived duration of the concomitant neutral stimuli. Estimates of the neutral stimuli were shorter and less accurate while experiencing the high pain intensity than while experiencing the no pain intensity. Comparing the findings of Experiment 1 and 2 suggest that task-relevant and task-irrelevant sources of arousal

have different effects on perceived duration. It is important to note that the differing findings between Experiments 1 and 2 are unlikely to be due to the use of thermal stimulation in Experiment 2, as opposed to the electro-cutaneous stimulation in Experiment 1, because thermal stimulation has been previously found to lengthen perceived duration (Ogden, Moore, et al., 2014).

The arousal hypothesis predicts a linear relationship between arousal and perceived duration; *any* change in arousal level should be associated with an analogous time distortion. Moreover, models of timing do not differentiate between time distortions caused by task-relevant and by task-irrelevant arousal. Any arousal increase leads to faster internal clock in SET, higher DA levels and faster cortical oscillators in SBF, and higher AIC activation in Craig's model of awareness, all resulting in longer perceived durations. Results of Experiment 1 therefore supported these predictions of a simple relationship between arousal and perceived duration, by demonstrating longer verbal estimates for stimuli with higher pain intensity than for stimuli with lower pain intensity. In Experiment 2, however, the absence of a lengthening effect of task-irrelevant pain, suggests that the relationship between arousal and perceived duration is more complex than that proposed by the arousal hypothesis (Angrilli et al., 1997; Burle & Casini, 2001) and perhaps limited to circumstances in which arousal is specific to the event being timed.

One possible explanation for the differing findings of Experiment 1 and Experiment 2 is that time perception was influenced by arousal *and* attention differently in the two tasks. In Experiment 2, it is plausible that pain could have disrupted attention to time, reducing perceived durations. Increasing pain intensity captures attention (Moore et al., 2012) and disrupts executive functions (Eccleston, 2011), impairing performance on ongoing tasks. In addition, attention is modulated by negatively valenced stimuli through bottom-up (i.e., stimulus driven) and top-down (i.e., cognitive driven) mechanisms, leading to faster identification of the emotional stimulus and higher performance on tasks requiring their processing (Vuilleumier, 2005). Both bottom-up and top-down mechanisms were aligned with the electro-

cutaneous stimulus in Experiment 1; meanwhile they were in conflict in Experiment 2, where bottom-up mechanisms directed the attention to the source of pain and the top-down mechanisms directed attention to the ongoing task (i.e., the neutral visual stimuli). This conflict may have taken the attentional resources away from the neutral visual stimuli.

Sufficient attentional resources are required for correct timing; perceived durations are shorter when participants' attention is impaired (Tse, Intriligator, Rivest, & Cavanagh, 2004). In SET, attention modulates the openness of the attentional gate, with reduced attention associated with reduced openness, resulting in shorter perceived durations (Zakay & Block, 1995). In SBF, impairments in attentional processes also results in shorter perceived durations due to disruption of DA release and of reset of cortical oscillators. In Experiment 2, increasing the pain intensity may have therefore distracted participants from the temporal task, leading to reduced attention for the encoding of the neutral visual stimulus, which resulted in shorter perceived durations. This would also explain the reduced estimate accuracy in high pain intensity compared to the other conditions.

It is also possible that the arousal hypothesis is perhaps too general, and that, rather than arousal per se affecting perceived duration, the effect of arousal on perceived duration is perhaps limited to circumstances in which arousal is task-relevant. Pain has also been suggested to reprioritise cognitive processes and affect arousal to react promptly to the threat so to promote survival (Eccleston & Crombez, 1999). A large number of studies have found pain effects on attention (Moore et al., 2013), memory (Buhle & Wager, 2010) and executive functions (Moriarty et al., 2011) compatible with this interpretation. It is therefore possible that pain effects on time perception also aim to promote survival. There were potential benefits to accurately encode the duration of the painful stimulus in Experiment 1; meanwhile there were no evident benefits to accurately encode the duration of the neutral stimulus in Experiment 2. In Experiment 2, greater survival benefits should be given to those who ignored the to-be-timed stimulus and directs attention to the painful, threatening

stimulation. Lake et al. (2016) also suggested that the variety of effects of emotions on time perception may be due to the biological relevance of the target emotion; the greater the stimulus importance for the survival of the individual the greater the time distortion induced by the stimulus. This is compatible with Gil and Droit-Volet (2012), who suggested that the semantic of the stimulus affects time perception even if there is no difference in arousal.

Whilst this survival based explanation addresses the results of this Chapter, it should be taken with caution. Attentional disruptions due to pain are associated with more errors during ongoing tasks (Moore et al., 2013). This should be mirrored with less accurate and more variable estimates in the verbal estimation task. However, in Experiment 2, although estimate accuracy decreased at the high pain intensity, the variability of the verbal estimates did not change across pain intensities. An attentional, survival based explanation might be therefore too simplistic.

In summary, this Chapter found that increasing pain intensity leads to longer, more accurate and less variable estimates when arousal is task-relevant (Experiment 1), meanwhile it leads to shorter and less accurate estimates when arousal is task-irrelevant (Experiment 2). Whilst results of Experiment 1 support the arousal hypothesis, results of Experiment 2 suggest a more complex relationship between arousal and perceived duration. To better understand the circumstances in which changes in physiological arousal are related to perceived duration, a direct test of the relationship between physiological arousal and perceived duration is required.

## Chapter 6

### **The relationship between pain induced autonomic arousal and perceived duration**

Chapter 5 shows that pain affects the perceived duration of event differently depending on whether it is the to-be-timed stimulus, and therefore task-relevant, or, a task-irrelevant source of stimulation. This implies a more complex relationship between arousal and perceived duration than that currently detailed in current models of timing. To further understand how pain induced arousal affects perceived duration, Chapter 6 tested the relationship between the physiological arousal evoked by a stimulus and its perceived duration. This Chapter has been published as an article in the journal *Emotion* with the title: “The relationship between pain induced autonomic arousal and perceived duration” (Piovesan, Mirams, Poole, Moore, & Ogden, 2018).

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 & Droit-Volet, 2012) but also from hypothesised changes in cortical activity as a result of repetitive stimulation (see Droit-Volet, 2010 and 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 verbal estimates themselves. For example, although Angrilli et al. (1997), Droit-Volet et al. (2010), Fayolle et al. (2015) and Mella et al. (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 (Mendes, 2009; Sztajzel, 2004). Increased SNS activity also modulates the electrical activity of the skin resulting in increased sweating (Sztajzel, 2004). 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 (Mendes, 2009; Sztajzel, 2004). 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 et al., 2015; Fung et al., 2017; Meissner & Wittmann, 2011; Pollatos et al., 2014; van Hedger et al., 2017). 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 et al., 1962; Osato et al., 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 et al., 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 Chapter 5, where task-irrelevant arousal shortened perceived duration, and in other studies, where time distortions were observed even if arousal was unaffected (Angrilli et al., 1997; Burle & Casini, 2001; Gil & Droit-Volet, 2012; Mella et al., 2011). 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 et al., 2016). A direct test to clarify the relationship between ANS activity and perceived duration is therefore required.

One effective way of manipulating ANS activity is through the induction of noxious somatosensory stimulation; increasing the intensity of electro-cutaneous and thermal stimulations is positively correlated with both ANS activity and verbal reports of pain experience (Möltner et al., 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 and thermal stimulations are also effective methods of distorting perceived duration (Fayolle et al., 2015; Ogden, Moore, 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 3, 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 4, 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 increasing stimulus intensity would lengthen duration estimates in Experiment 3 and would shorten duration estimates in Experiment 4, replicating findings of Chapter 5. In all conditions of Experiment 3, greater SNS reactivity was expected to be associated with longer perceived durations.

### **6.1 – Experiment 3**

Experiment 3 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 Experiment 1. 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 verbal estimation, as in van Hedger et al. (2017).

### 6.1.1 – Method

#### 6.1.1.1 – Participants

Forty participants (30 females and 10 males; mean age = 26.20, SD = 3.91) were recruited. Participants were required not to be pregnant, not to have a history of epilepsy and not to have chronic pain, heart disease, skin problems (e.g., eczema) 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.

#### 6.1.1.2 – Apparatus and materials

*Pain stimulation:* The Digitimer DS7A Current Stimulator (Digitimer Ltd) was used to present the electro-cutaneous stimulation (see section 4.3.1, page 77 for equipment description).

*Physiological apparatus:* Biopac MP30B-CE was used to record EDA and ECG signals through two separate sets of electrodes. 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. SCL and HF HRV were extracted by EDA and ECG signals, respectively (extrapolation is described below).

### 6.1.1.3 – Procedure

The procedure was similar to the one used in Experiment 1. 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:* Participants performed the task with the Digitimer DS7A to select electrical intensities equal to 0, 3 and 6 in the NRS; that is no pain, low pain (Serlin et al., 1995) and high pain (Khoshnejad et al., 2016), respectively (see section 4.3.1, page 77 for task description).

*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 et al., 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.

#### 6.1.1.4 – 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.

#### *6.1.2 – 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. Greenhouse-Geisserr correction was applied to ANOVAs when the Sphericity assumption was violated and post-hoc were Bonferroni corrected.

<i>Pain intensity</i>	<i>Electrical current</i>	<i>Follow-up rating</i>	<i>Mean SCL</i>	<i>Mean HF HRV</i>	<i>Verbal estimate</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 6.1. Means (and standard deviations) of electrical current (mA), post-timing pain ratings, baseline-corrected Skin Conductance Levels (SCL,  $\mu$ mho), baseline-corrected High Frequency Heart Rate Variability (HF HRV, normalized units) and mean verbal estimate of all durations (ms) in the three intensity level conditions (no pain, low pain and high pain) in Experiment 3.*

Table 6.1 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 electrical currents were significantly different to each other,  $F(1.15, 37.95) = 216.61$ ,  $p < .001$ ,  $\eta_p^2 = .87$ ; confirmed by post-hoc tests (all  $ps < .001$ ). 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 6.1 shows SCL and HF HRV for each stimulus intensity. Examination of Table 6.1 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 electrocutaneous intensity (no pain, low pain and high pain) on SCL  $F(2, 66) = 4.93$ ,  $p = .01$ ,  $\eta_p^2 = .13$ . Post-hoc tests showed that SCL was significantly higher in the high pain condition than in the low pain ( $p = .049$ ) and no pain condition ( $p = .024$ ). SCL was not significantly different between the low pain and no pain condition ( $p = .99$ ). Although examination of Table 6.1 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 6.1 shows mean verbal estimations for each electro-cutaneous stimulus intensity condition. Examination of Figure 6.1 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 verbal estimates. Post-hoc tests showed that estimates were significantly longer in the high pain condition than in the no pain condition ( $p = .01$ ). The difference between the high pain and low pain condition was approaching significance ( $p = .054$ ). Estimates were not significantly different in the low pain and no pain condition ( $p = .91$ ). There was no significant interaction between stimulus duration and pain intensity  $F(5.14, 169.46) = 1.58, p = .17, \eta_p^2 = .05$ .

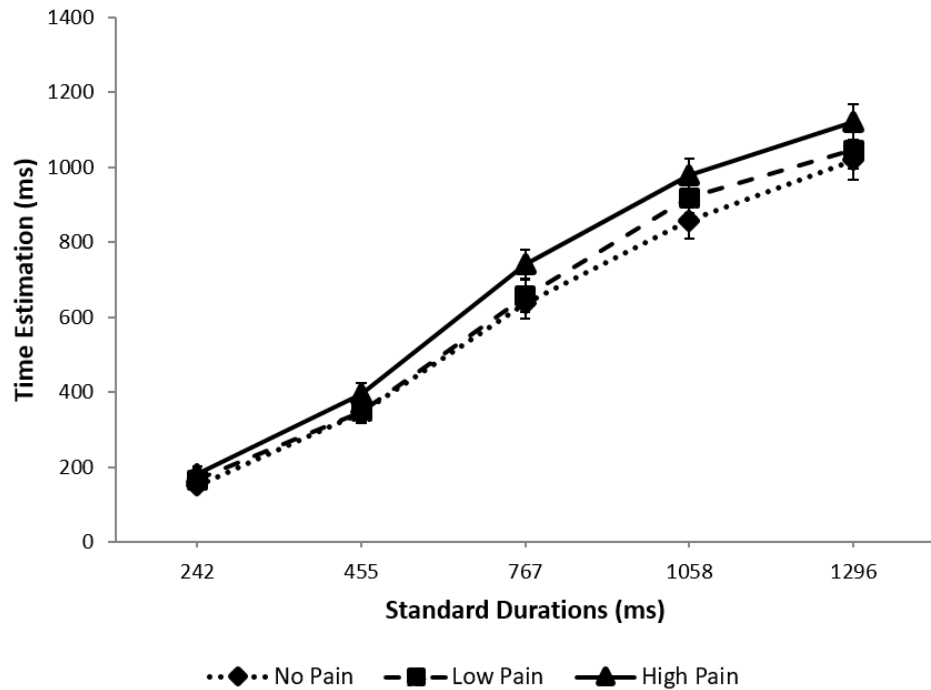


Figure 6.1. Means (and standard errors) of the verbal estimations (ms) plotted against the standard durations and divided by the intensity level conditions (no pain, low pain and high pain) in Experiment 3.

#### 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 6.1). 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 verbal estimate (i.e., whether decreases in HF HRV were associated with increases in duration estimation). Table 6.2 shows this analysis. Examination of Table 6.2 suggests that there were significant positive correlations between SCL and verbal

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 verbal estimate change for the no-low pain condition and the low-high pain condition.

	Changes from no pain to low pain			Changes from no pain to high pain			Changes from low pain to high pain		
	1	2	3	1	2	3	1	2	3
1. Verbal estimate	—			—			—		
2. Skin Conductance Level	<b>.45**</b>	—		<b>.46**</b>	—		<b>.37*</b>	—	
3. High Frequency Heart Rate Variability	<b>-.35*</b>	<b>-.39*</b>	—	-.04	.01	—	<b>-.41**</b>	-.14	—

*Table 6.2. Correlation coefficients between changes of (1) verbal estimate (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 3. \*  $p < .05$ ; \*\*  $p < .01$*

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 ( $\beta = .37$ ,  $p = .04$ ) but HF HRV was not ( $\beta = -.21$ ,  $p = .24$ ). 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 ( $\beta = .46$ ,  $p = .007$ ) but HF HRV was not ( $\beta = -.05$ ,  $p = .78$ ). 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 ( $\beta = -.37, p = .024$ ) and SCL trended towards significance ( $\beta = .32, p = .051$ ).

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 6.2.

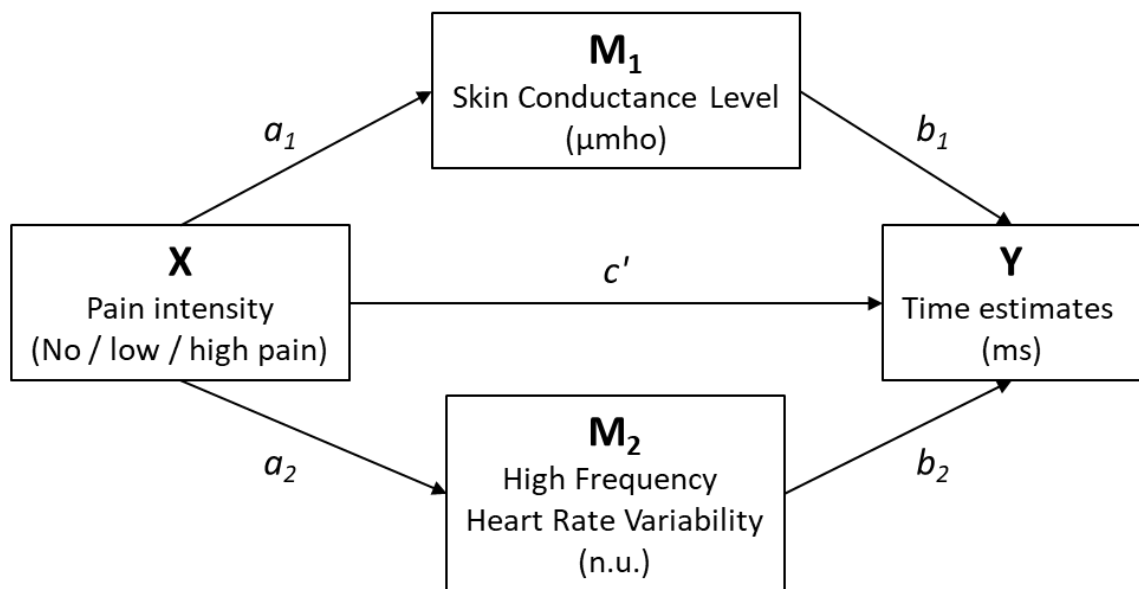


Figure 6.2. Model of the within-participant parallel mediation in path analytic form, showing the effect of pain intensity (X) on verbal estimate (Y) mediated by SCL ( $M_1$ ) and HF HRV ( $M_2$ ).

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 verbal estimate (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 6.3.

Dependent variable, Y	Independent variable, X	Mediating variable, M	Effect of X on M (a)	Effect of M on Y (b)	Indirect effect (ab)	Total indirect effect (ab)	Direct effect (c')	Total effect (c)	Degree of mediation
Time estimation	Pain intensity (0) no pain (1) low pain	SCL	0.24	<b>27.15*</b>	6.46	4.35	20.58	24.94	None
		HF HRV	1.01	-2.08	-2.10				
	Pain intensity (0) low pain (1) high pain	SCL	<b>0.86*</b>	21.91 <sup>‡</sup>	<b>18.78*</b>	10.51	45.25	<b>55.76*</b>	None
		HF HRV	2.86	<b>-2.89*</b>	-8.27				
	Pain intensity (0) no pain (1) high pain	SCL	<b>1.10**</b>	<b>32.09**</b>	<b>35.15*</b>	<b>33.38*</b>	47.32	<b>80.70**</b>	Complete
		HF HRV	3.87	-0.46	-1.77				

Table 6.3. Mediation coefficients of Experiment 3. SCL = Skin Conductance Level ( $\mu\text{mho}$ ). HF HRV = High Frequency Heart Rate Variability (normalized units).  $\ddagger p = .056$ ; \*  $p < .05$ ; \*\*  $p < .001$

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 verbal estimates was either significant, or trending towards significant ( $p = .056$ ).

### 6.1.3 – Discussion

Experiment 3 tested whether perceived duration was related to changes in SNS 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, contrary to Experiment 1. Moreover, pain increases did not have the multiplicative effect on perceived duration as shown in Experiment 1.

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 significantly predicted changes in verbal estimate. 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 3 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.

## **6.2 – Experiment 4**

Experiment 4 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) are also associated with changes in 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 et al., 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 SBF 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. Finally, Experiment 2 found that increasing pain intensity shortened the perceived duration of concomitantly neutral 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 4 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 3, SNS activity, indexed by SCL, was expected to increase with increasing pain intensity and, as in Experiment 2, verbal estimates were expected to decrease with increasing pain intensity. It was therefore expected that changes in SCL and HF HRV would not be associated with changes in perceived durations.

### 6.2.1 – Method

#### 6.2.1.1 – Participants

Thirty-one participants (26 females and 5 males; mean age = 22.23, SD = 4.74) were recruited. Participants were required not to be pregnant, not to have a history of epilepsy and not to have chronic pain, heart disease, skin problems (e.g., eczema) 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.

#### 6.2.1.2 – Apparatus and materials

*Pain stimulation:* The Medoc PATHWAY-Advanced Thermal Stimulator was used to present the sustained thermal stimulation (see section 4.3.2, page 78 for equipment description).

*Physiological apparatus:* As in Experiment 3, 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 3.



### 6.2.1.3 – Procedure

The procedure was similar to the one used in Experiment 2. 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>).

*Initial intensity rating task:* Participants performed the task with the Medoc PATHWAY-Advanced Thermal Stimulator to select thermal intensities equal to 0, 3 and 6 in the NRS; that is warm but no pain, low pain (Serlin et al., 1995) and high pain (Khoshnejad et al., 2016), respectively (see section 4.3.2, page 78 for task description).

*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 3 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°C/second to 1°C above each participant's set threshold. This then oscillated between 1°C above and 1°C below the participant's threshold at 8°C/second for 10 oscillations before returning to the baseline temperature (32°C) at a rate of 8°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.

#### 6.2.1.4 – 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 3. 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.

### 6.2.2 – 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 et al., 1995). Greenhouse-Geisserr correction was applied to ANOVAs when the Sphericity assumption was violated and post-hoc were Bonferroni corrected.

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

*Table 6.4. Means (and standard deviations) of temperature (°C), baseline-corrected Skin Conductance Levels (SCL,  $\mu$ mho), baseline-corrected High Frequency Heart Rate Variability (HF HRV, normalized units) and mean verbal estimate of all durations (ms) in the three intensity level conditions (no pain, low pain and high pain) in Experiment 4.*

Table 6.4 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 6.4 shows SCL and HF HRV of participants for each stimulus intensity. Examination of Table 6.4 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 showed that SCL was significantly higher in the high pain condition than in the low pain ( $p = .013$ ) and no pain condition ( $p = .001$ ). SCL was not significantly different in the low pain and no pain conditions ( $p = .09$ ). 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 6.3 shows mean verbal estimates in each pain intensity condition. Examination of Figure 6.3 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  $F(4.49, 130.09) = 0.83, p = .52, \eta_p^2 = .03$ .

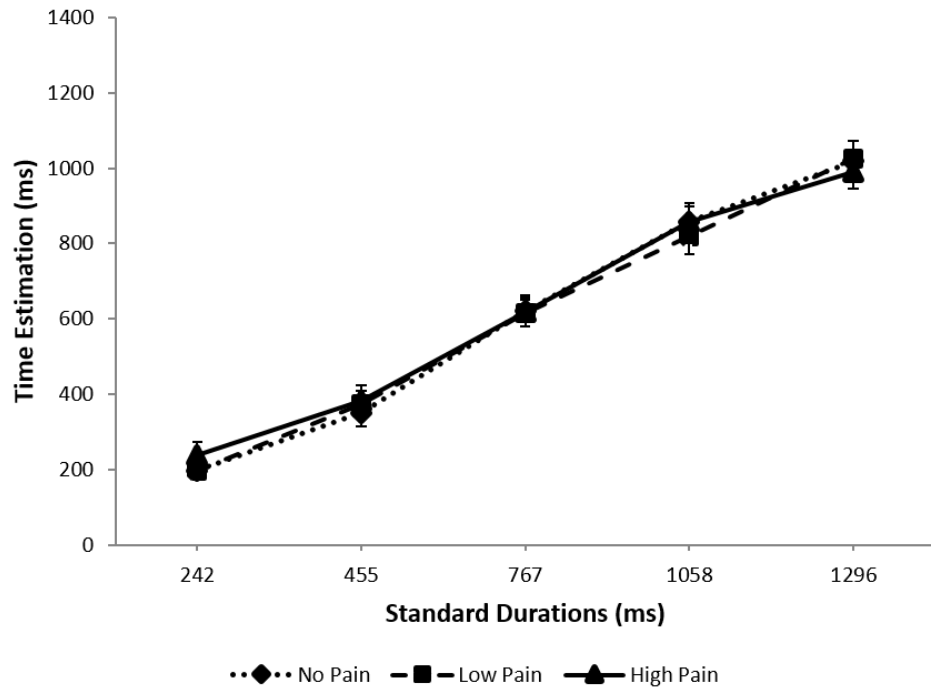


Figure 6.3. Means (and standard errors) of the verbal estimations (ms) plotted against the standard durations and divided by the intensity level conditions (no pain, low pain and high pain) in Experiment 4.

#### ANS activity and perceived duration

As in Experiment 3, 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 verbal estimate and to test whether there was negative correlation between changes in HF HRV and changes in verbal estimate (see Table 6.5). Examination of Table 6.5 shows that there were no significant correlations between ANS reactivity and changes in verbal estimate.

	<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. Verbal estimate	—			—			—		
2. Skin Conductance Level	-.09	—		-.28	—		-.10	—	
3. High Frequency Heart Rate Variability	-.02	.24	—	-.16	-.16	—	-.02	-.10	—

*Table 6.5. Correlation coefficients between changes of (1) verbal estimate (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 4.*

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 verbal 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 ( $0.82 \mu\text{mho}$ ), low pain ( $1.23 \mu\text{mho}$ ) and high pain ( $1.99 \mu\text{mho}$ ) tasks of block 1 than block 2 (no pain:  $0.23 \mu\text{mho}$ ,  $p = .025$ ; low pain:  $0.38 \mu\text{mho}$ ,  $p = .001$ ; high pain:  $0.83 \mu\text{mho}$ ,  $p < .001$ ). 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 verbal estimates (all  $ps > .05$ ).

As in Experiment 3, path analytic mediator analysis was conducted to test whether physiological arousal (indexed by SCL and HF HRV) mediated the effect of pain intensity on verbal estimate. The findings of the mediator analysis are shown in Table 6.6. 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 verbal estimates. 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.

<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>	-11.44	-3.61	-3.33	0.16	-3.17	None
		HF HRV	0.50	0.56	0.28				
	<i>Pain intensity (0) low pain (1) high pain</i>	SCL	<b>0.68**</b>	0.27	0.18	0.76	10.26	11.02	
		HF HRV	-1.54	-0.38	0.58				
	<i>Pain intensity (0) no pain (1) high pain</i>	SCL	<b>0.99**</b>	-33.74	-33.51	-31.63	39.48	7.84	
		HF HRV	-1.04	-1.80	1.88				

*Table 6.6. Mediation coefficients of Experiment 4. SCL = Skin Conductance Level ( $\mu\text{mho}$ ). HF HRV = High Frequency Heart Rate Variability (normalized units). \*  $p < .05$ ; \*\*  $p < .01$*

### 6.2.3 – Discussion

Experiment 4 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. These effects were not mirrored in the changes in duration perception. However, whilst in Experiment 2 verbal estimates were shorter in the high pain intensity, participants of Experiment 4 gave similar estimates across the three conditions (no pain, low pain, high pain).

The correlation and regression analyses did not show any association between ANS activity and verbal estimate. 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.

## 6.3 – Discussion Chapter 6

Chapter 6 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 3, the to-be-timed stimulus itself was arousing and thus arousal was task-relevant. In Experiment 4, 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 3, when the to-be-timed stimulus was itself arousing, high intensity stimuli were perceived as lasting for longer than neutral stimuli, confirming Experiment 1 and previous findings (Fayolle et al., 2015; Ogden, Moore, et al., 2014). However, in Experiment 4, 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 3 is compatible with previous suggestions that “arousal” increases the perceived duration of events (Gil & Droit-Volet, 2012). By examining both SNS and PSNS, Experiment 3 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., 2015) 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). Here, 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. (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 3 suggest a clear and relatively simple relationship between ANS activity and perceived duration, the findings of Experiment 4 suggest that this relationship is unique to certain circumstances. Experiment 4 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. 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 4, task-irrelevant increases in arousal did not affect perceived duration. One possible explanation is that the ANS activation evoked in Experiment 4 was not sufficient to affect perceived duration. A comparison of SCL and HF HRV recorded in Experiments 3 and 4<sup>1</sup> suggests that mean SCL was higher and

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<sup>1</sup>Independent samples t-tests show that SCL was significantly higher in Experiment 3 than in Experiment 4 for the no pain ( $p = .024$ ), low pain ( $p = .043$ ) and high pain ( $p = .022$ ) conditions. HF HRV was significantly higher in Experiment 4 than in Experiment 3 for the no pain ( $p < .001$ ), low pain ( $p = .005$ ) and high pain ( $p = .031$ ) conditions. However, between Experiment 3 and Experiment 4 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 ( $p = .85$ ), from the no pain to the high pain condition ( $p = .82$ ) and from the low pain to the high pain condition ( $p = .66$ ) were not significantly different in Experiment 3 and 4. Changes

HF HRV lower in Experiment 3 than Experiment 4 for all conditions. Thus, SNS activation was greater and PSNS was lower in Experiment 3 than in Experiment 4. 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 3 and 4 suggesting that the condition-condition changes in arousal were similar in both experiments. Moreover, a lower physiological activation due to thermal pain would not explain why the high pain intensity shortened verbal estimates in Experiment 2.

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 et al., 2012). Reduced attention to time can result in shorter perceptions of duration (Zakay & Block, 1995), as is observed in dual task studies (Brown, 1997). In Experiment 4, 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 3. Emotional stimuli, particularly negatively valenced stimuli, affect attention through both endogenous top-down 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 3, both endogenous and exogenous orientation was aligned to the same stimulus (i.e., the electro-cutaneous stimulation), perhaps leading to an

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in HF HRV from the no pain to the low pain condition ( $p = .90$ ), from the no pain to the high pain condition ( $p = .24$ ) and from the low pain to the high pain condition ( $p = .32$ ) were not significantly different in Experiment 3 and 4.

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 4, this explanation should be taken with caution. One effect of the attentional capture of pain is that it increases errors on ongoing tasks (Moore et al., 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 3 and 4 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 et al., 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 3, 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 4 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 the neutral stimuli. Matthews, Stewart and 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 3, the contrast between the stimulus and the background changed from trial to trial. In Experiment 4, 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. However, this explanation would not address findings of Experiment 2, where the high pain intensity shortened verbal estimates.

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, 2009b). 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 (2009a) 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 3, moment-moment salience was high because the electro-cutaneous 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 4 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 (2009a) 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 & Mauguière, 2003). Therefore, in Experiment 3 greater stimulus

estimates may result from increased AIC activation, as suggested by Craig (2009a). 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 4 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.

The experiments presented in this Chapter confirm that there is a relationship between physiological arousal 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.

## Chapter 7

### The effect of pain on memory for duration

Experiments 1 and 3 showed that pain can distort the perception of time. When people estimate the duration of a painful stimulus, duration estimates increase with increasing pain intensity. These findings replicate those of other studies using different experimental paradigms (Fayolle et al., 2015; Rey et al., 2017) and different pain induction techniques (Ogden, Moore, et al., 2014; Rey et al., 2017). A common factor of all of these studies is that they all required participants to make temporal judgments *immediately* after experiencing the stimuli. To date, no studies have investigated how pain affects how duration is remembered over a period of delay.

Understanding how the duration of pain is remembered is important because how patients remember the duration of pain can have clinical implications. Clinical assessments of pain typically require participants to estimate how long pain lasts for (Somov, 2000) and it is therefore possible that inaccuracies in recall may affect treatment. Furthermore, how pain is remembered from previous clinical interventions, such as dental work, can increase pain anxiety and reduce subsequent clinical compliance (Boivin et al., 2008).

Pain may be expected to impair memory for duration because it affects the general cognitive processes upon which temporal processing is reliant (see section 3.1.4, page 60). Accurate temporal processing requires sufficient attention, working memory and executive function (see Ogden, Wearden, & Montgomery, 2014 for discussion). When these resources are exceeded or impaired timing is disrupted, becoming more variable and less accurate (Brown, 1997; Ogden, Salominaite, Jones, Fisk, & Montgomery, 2011), possibly because 1) memory representations of duration are themselves more variable, or because 2) they are more difficult to retrieve from long-term memory when working memory and executive resources are limited (Ogden, Wearden, et al., 2014). It is well established that pain impairs the maintenance of items in memory (Dick & Rashiq, 2007) and recognition accuracy



(Forkmann, Schmidt, Schultz, Sommer, & Bingel, 2016). Leavitt and Katz (2006) suggested that pain affects memory processes possibly because pain functions as a distractor leading to reduced attentive resources dedicated to the experimental task. It is therefore possible that experiencing pain may impair the attentional, memory and executive resources required to encode and maintain duration representations in memory, leading to more fragile representations.

Conversely, however, it is also possible that pain may enhance the accuracy of memory for duration. In general cognition, memories for emotional events are often superior to those for neutral events (e.g., flashbulb memories; Reisberg & Heuer, 2004). Similar effects have also been observed when examining memory for the duration of emotional events; for example, Cocenas-Silva et al. (2012) found that, whilst perceived duration of neutral stimuli was longer after 24h than after immediate presentation, perceived duration of emotional stimuli did not increase after 24h. Furthermore, estimate variability after 24 hours was higher for neutral stimuli than for emotional stimuli. Memory for time is therefore less vulnerable to distortion and decay when emotional than when neutral.

Emotion may enhance memory for time because emotions promote the release of adrenal stress hormones that facilitate memory consolidation by the hippocampus (LaBar & Cabeza, 2006; McGaugh, 2000). This manifests with faster recall of events associated with emotional states and of details related to those events (e.g., location; D'Argembeau & Van der Linden, 2004; Dunbar & Lishman, 1984). Particularly, arousing emotions have been shown to enhance long-term memory for events (Sharot & Phelps, 2004). Given that pain is a high arousing experience and it promotes the release of adrenal stress hormones (see section 3.1.2, page 56 and Bear et al., 2007), it is possible that pain may enhance memory for durations experienced during states of pain.

Experiments 5 and 6 therefore aimed to establish the effect of pain on memory for duration. Using a temporal generalisation paradigm, the effect of pain and delay

to recall were tested. The temporal generalisation task was chosen because it requires a higher memory load compared to other temporal tasks, such as verbal estimation or temporal bisection task (Ogden et al., 2018) and therefore was perhaps more likely to reveal any effects of pain on memory for duration.

In Experiment 5, participants experienced low pain (3 in the NRS), meanwhile participants experienced high pain (6 in the NRS) in Experiment 6. Participants first selected the thermal intensity that considered painful and then completed four temporal generalisation tasks. Each temporal generalisation task was split into two phases, a learning phase and a testing phase. In the learning phase, the participants' task was to memorize the duration of a tone whilst they experienced either 1) painful stimulation on their arm or 2) neutral stimulation on their arm. In the testing phase, the participants' task was to indicate whether a series of comparison durations were the same duration as that presented in the learning stage phase. The testing phase either occurred immediately after the learning phase or following a 15-minute delay. No additional (i.e., painful or neutral) stimulation was experienced during the testing phase. All participants therefore completed four versions of this task (i) no-pain immediate testing (ii) pain immediate testing, (iii) no-pain delayed testing and (iv) pain delayed testing.

Firstly, it was expected that the 15-minute delay would decrease temporal accuracy and temporal discrimination in the no-pain condition, confirming previous studies (Wearden & Ferrara, 1993). Secondly, two potential outcomes were predicted based on the previous arguments for the potential impairing and enhancing effects of pain on memory for duration. The first possibility is that learning a duration in a state of pain will impair memory processing, leading to poorer recognition during immediate and delayed testing. The second possibility is that learning a duration in a state of pain will enhance memory processes, leading to enhanced recall during immediate and delayed testing.

## **7.1 – Experiment 5**

### *7.1.1 – Method*

#### 7.1.1.1 – Participants

Twenty-eight participants (18 females and 10 males; mean age = 25.79, SD = 6.05) were recruited. Participants were required not to be pregnant and not to have chronic pain, skin problems (e.g., eczema) 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 £5 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.

#### 7.1.1.2 – Procedure

Participants were initially asked to complete a health screening questionnaire to confirm their suitability to participate. Participants then performed an intensity rating task with the Medoc PATHWAY-Advanced Thermal Stimulator to establish the thermode intensities to be used during the timing task (see section 4.3.2, page 78 for equipment and task description). Participants were asked to select thermal intensities equal to 0 and 3 in the NRS; that is, 0 a warm but non painful intensity and 3 a low pain intensity.

Participants then completed the four temporal generalisation tasks: (i) no-pain immediate testing (ii) pain immediate testing, (iii) no-pain delayed testing and (iv) pain delayed testing. The order of these tasks was counterbalanced across all participants. The basic task structure was as follows:

*Learning phase:* Participants were told that they would be presented with a standard tone three times and that their task was to remember how long the tone lasted for. The standard was presented as a 500Hz tone and its duration was randomly selected from a normal distribution from 400ms to 800ms. This ensured that participants were presented with different standard durations across the four

tasks, so to avoid learning effect. Each presentation was preceded by an inter-trial interval randomly selected from a 2500-3000ms range.

*Testing Phase:* Participants were informed that they would be presented with a series of comparison tones and that their task was to decide whether each tone was the same length as the standard tone that they previously learnt pressing 'Y' for yes or 'N' for no. At the start of each trial participants were instructed to press the spacebar. A comparison stimulus was then presented in the form of a 500Hz tone and participants indicated whether the comparison tone had the same duration as the standard. Between trials, a delay randomly selected from a 1000-1500ms range was then interposed. On each trial, the duration of the comparison was determined by multiplying the standard by 0.625, 0.750, 0.875, 1 (presented 3 times), 1.125, 1.250 or 1.375. A total of six blocks were presented in each task giving a total of 54 stimuli for task. No performance feedback was given to participants.

In the pain conditions, participants felt the thermode being at the low pain intensity during the learning phase. The thermode started with a baseline temperature of 32°C. At the beginning of the learning phase, the thermode increased its temperature at a ramp rate of 8°C/second until reaching the low pain intensity selected by participant during the intensity rating scale. The standard tone was presented for the first time after 3 seconds from the initial temperature increase of the thermode, allowing the thermode to reach the target temperature. After 15 seconds from the initial temperature increase of the thermode, the temperature decreased at a ramp rate of 4°C/second until reaching again 32°C. No thermal stimulation was presented during the testing phase.

In the no-pain conditions, the procedure was the same used in the pain conditions with the exception of the target temperature; here, the thermode increased its temperature until reaching the no-pain intensity selected by participant in the intensity rating task. No thermal stimulation was presented during the testing phase.

In the delayed testing conditions, a 15-minute delay was interposed between the learning and testing phases. During this 15-minute delay, participants listened to either “The Wizard of Oz” or “The Jungle Book”. The audiobook assignment to the no-pain delayed testing condition or to the pain delayed testing condition was counterbalanced across participants. This task was chosen rather than a cognitive task to avoid interference on the working memory or executive functions (Mirams, Poliakoff, Brown, & Lloyd, 2013).

#### 7.1.1.3 – Data analysis

From the temporal generalisation task, we extrapolated the temporal gradients as the proportion of YES responses (i.e., identification of comparisons as the standard) given by each participant in each of the conditions. In addition, a measure of accuracy and a measure of variability was calculated for each participant in each condition as in Ogden et al. (2018).

*Accuracy:* Accuracy was calculated as the sum of hits and correct rejections divided by 2. Hits corresponded to proportion of YES responses when the comparison’s duration was equal to the standard (i.e., 1). Correct rejections corresponded to proportion of NO responses when the comparison’s duration was not equal to the standard (i.e., 0.625, 0.750, 0.875, 1.125, 1.250 and 1.375).

*Variability:* The mid-three measure used in Ogden et al. (2018) and Wearden et al. (1997) was calculated. Mid-three is an index of response dispersion and was calculated as the sum of proportion of YES responses in the three middle comparisons (i.e., 0.875, 1 and 1.125) divided by the sum of YES responses of all comparisons. Higher mid-three scores indicated that gradients were more peaked around the standard and that participants had greater temporal discrimination.

### 7.1.2 – Results

On average, participants selected 37.63°C (SD = 1.91) as no-pain intensity and 42.77°C (SD = 1.40) as low pain intensity during the initial intensity rating task. Paired-sample t-test indicated that participants selected a significantly higher temperature for the pain conditions compared to the no pain conditions ( $p < .001$ ).

Figure 7.1 shows temporal generalisation gradients depicting the mean proportion of YES responses in the four conditions plotted against comparison/standard ratio. A repeated measures ANOVA with pain intensity (no-pain vs pain), delay (immediate vs delay) and comparison/standard ratio (0.625, 0.750, 0.875, 1, 1.125, 1.250 or 1.375) as within-subject factors was conducted. There was a significant main effect of ratio ( $F(2.02, 54.46) = 32.77, p < .001, \eta_p^2 = .55$ ) on YES responses. There was no significant main effect of pain intensity ( $F(1, 27) = 0.02, p = .90, \eta_p^2 = .001$ ) or delay ( $F(1, 27) = 0.002, p = .96, \eta_p^2 < .001$ ) on YES responses. There were also no significant interaction effects between delay and ratio ( $F(3.03, 81.68) = 2.21, p = .09, \eta_p^2 = .08$ ), between pain intensity and delay ( $F(1, 27) = 0.37, p = .55, \eta_p^2 = .014$ ) or between pain intensity, delay and ratio ( $F(2.31, 62.35) = 0.70, p = .52, \eta_p^2 = .03$ ) on YES responses. There was however, a significant interaction effect between pain intensity and ratio ( $F(2.43, 65.57) = 3.05, p = .045, \eta_p^2 = .10$ ).

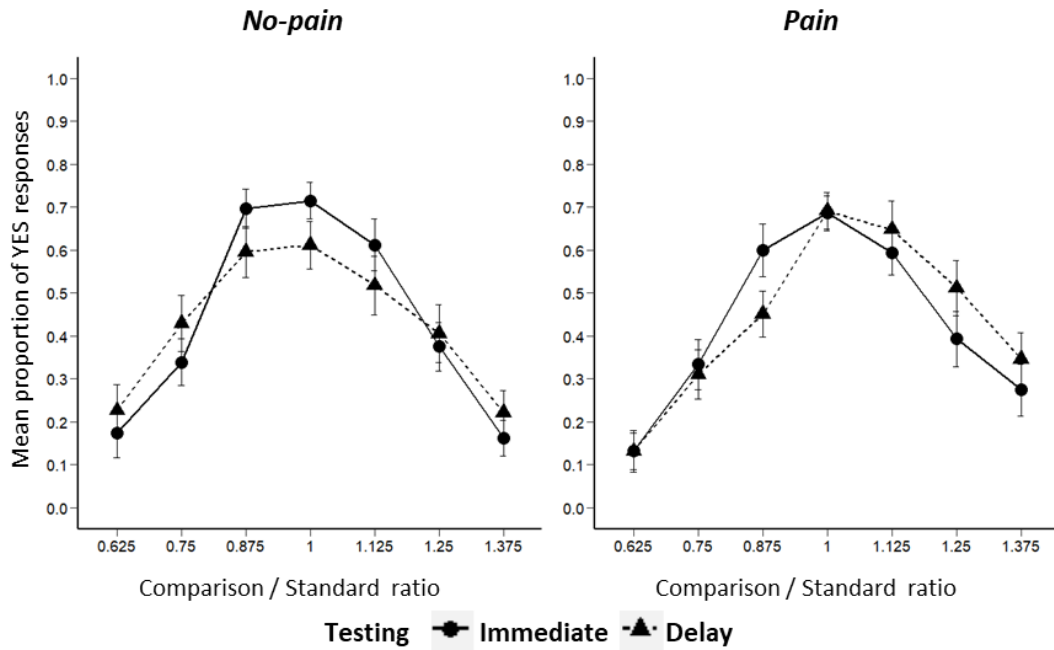


Figure 7.1. Proportion of YES responses plotted against comparison/standard ratio in Experiment 5. YES responses are divided between immediate (solid line) and delay (dotted line), and between no-pain (left panel) and pain (right panel).

To further investigate the interaction between pain intensity and ratio, the proportion of YES responses in the pain conditions was calculated averaging the YES responses in pain immediate and pain delayed testing conditions for each comparisons' duration. Similarly, the proportion of YES responses in the no-pain conditions was calculated averaging the YES responses in no-pain immediate and no-pain delayed testing conditions (see Table 7.1). Visual inspection of Table 7.1 suggests that the proportion of YES responses for the shortest comparison (i.e., 0.625, 0.750 and 0.875) was higher in the no-pain conditions than in the pain conditions; meanwhile the proportion of YES responses for the longest comparison (i.e., 1.125, 1.250 and 1.375) was lower in the no-pain conditions than in the pain conditions. This would suggest that pain related standards were perceived for longer than no-pain related standards. A paired-sample t-test between the no-pain and pain conditions was then conducted for each ratio (0.625, 0.750, 0.875, 1, 1.125, 1.250 and 1.375). To adjust for Type 1 error due to multiple comparisons, the number of comparisons (7) has been taken into account:  $p$ -value should be  $< .0071$  ( $= .05/7$ ) to confirm

significance. Paired-sample t-tests showed no significant difference between YES responses in the pain and no-pain conditions for the 0.625 ( $p = .18$ ), 0.750 ( $p = .29$ ), 0.875 ( $p = .019$ ), 1 ( $p = .48$ ), 1.125 ( $p = .31$ ) and 1.250 ( $p = .17$ ) ratio. Paired-sample t-test showed YES responses for the 1.375 ratio significantly higher in the pain conditions than in the no-pain conditions ( $p = .004$ ). This suggests that standard stimuli in the two pain conditions were perceived as longer than the standard stimuli in the two no-pain conditions.

	<i>Comparison/standard ratio</i>						
	<i>0.625</i>	<i>0.750</i>	<i>0.875</i>	<i>1</i>	<i>1.125</i>	<i>1.250</i>	<i>1.375</i>
<i>No-pain</i>	0.20 (0.23)	0.39 (0.22)	0.65 (0.22)	0.67 (0.19)	0.57 (0.26)	0.39 (0.25)	0.19 (0.20)
<i>Pain</i>	0.13 (0.20)	0.32 (0.26)	0.21 (0.22)	0.69 (0.19)	0.62 (0.22)	0.45 (0.26)	0.31 (0.24)

*Table 7.1. Proportion of YES responses (and standard deviation) averaged across the two No-pain conditions (no-pain immediate and no-pain delay) and across the two Pain conditions (pain immediate and pain delay).*

#### *Temporal accuracy*

Table 7.2 shows temporal accuracy in the four conditions. Examination of Table 7.2 suggests that accuracy was similar in all conditions. A repeated measures ANOVA with pain intensity (no-pain vs pain) and delay (immediate vs delay) as within-subject factors confirmed these suggestions. There was no significant effect of pain intensity ( $F(1, 27) = .57, p = .46, \eta_p^2 = .02$ ) nor delay ( $F(1, 27) = 1.75, p = .20, \eta_p^2 = .06$ ) on accuracy. There was also no significant interaction effect between pain intensity and delay ( $F(1, 27) = 1.49, p = .23, \eta_p^2 = .05$ ). Temporal accuracy was therefore unaffected by pain or delay.



<i>Condition</i>	<i>Accuracy</i>	<i>Mid-three</i>
<i>No-pain immediate</i>	0.66 (0.11)	0.67 (0.14)
<i>No-pain delay</i>	0.61 (0.14)	0.56 (0.14)
<i>Pain immediate</i>	0.65 (0.12)	0.64 (0.17)
<i>Pain delay</i>	0.65 (0.11)	0.59 (0.16)

Table 7.2. Means (and standard deviations) of accuracy and mid-three in the four conditions (no-pain immediate, no-pain delay, pain immediate and pain delay) in Experiment 5.

#### *Temporal variability*

Table 7.2 shows temporal variability (i.e., mid-three) in the four conditions. Examination of Table 7.2 suggests that variability was lower (i.e., gradients were more peaked) in the immediate testing than delayed testing conditions. A repeated measures ANOVA revealed a significant main effect of delay on mid-three ( $F(1, 27) = 8.41, p = .007, \eta_p^2 = .24$ ). Mid-three was significantly higher in the immediate conditions compared to the delayed conditions suggesting that delay disrupts temporal discrimination of both pain and no-pain related stimuli. There was however no main effect of pain intensity ( $F(1, 27) = .001, p = .97, \eta_p^2 < .01$ ) and no significant interaction effect between delay and pain intensity ( $F(1, 27) = .68, p = .42, \eta_p^2 = .03$ ). Therefore, although delay per se increased temporal variability, pain did not affect temporal variability.

#### *7.1.3 – Discussion*

The results of Experiment 5 suggest that memory for the duration was largely unaffected when a low level of pain was experienced during the encoding of temporal information. This was confirmed by the absence of an effect of pain on temporal accuracy and variability. Indeed the only effect of pain was seen when comparing responses to the longest of the comparison stimuli. Memory for duration is therefore unaffected by low pain during encoding. This contrasts with the expectations of the

study: pain neither disrupted the cognitive resources necessary for correct memorization of duration, nor enhanced long-term memory of events, as emotions do (Cocenas-Silva et al., 2012).

Memory for duration was however affected by delay, with significantly more variable responding in the delayed testing conditions than the immediate testing conditions. This replicates previous findings that memory for duration can decay over short delays (Ogden, Wearden, & Jones, 2008) and confirmed that the methodology was appropriate for detecting delay induced changes in responding.

One perhaps unexpected finding of the current study is that generalisation gradients were not systematically skewed by the presence of pain. Previous research shows that painful events are perceived as lasting for longer than neutral events. We may therefore have expected left skewed gradients (i.e., greater proportion of YES responses to durations longer than the standard). Although this was observed to some extent, that is the multiple comparisons showed that 1.375 comparison was recognized more often as the standard in the painful than the neutral conditions, no differences were observed for other comparison durations. One possibility is that pain did not have a clear and systematic effect on responding in this task because the level of pain induced was not intense enough to affect responding. To test this possibility a further experiment was conducted using the same experiment design as Experiment 5 but with a greater pain intensity.

## **7.2 – Experiment 6**

Experiment 6 used the same experimental design as Experiment 5, however the level of pain induced was increased from low to high. Therefore, in the initial intensity rating task, participants were asked to select the thermal intensity that corresponded to high pain (6 in the NRS). As in Experiment 5, two possible outcomes were anticipated: 1) pain could affect the attentional, memory and executive resources necessary for the encoding and maintenance of duration information in memory over

a period of delay; or 2) similarly to emotional durations, durations encoded in a state of pain may be better remembered than those encoded in a neutral state.

### *7.2.1 – Method*

#### *7.2.1.1 – Participants*

Twenty-eight participants (21 females and 7 males; mean age = 24.11, SD = 4.83) were recruited. Participants were required not to be pregnant and not to have chronic pain, skin problems (e.g., eczema) 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 £5 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.

#### *7.2.1.2 – Procedure*

Participants completed the same procedure used in Experiment 5. Only, participants selected thermal intensities equal to 0 and 6 in the NRS (instead of 0 and 3) during the intensity rating task, that is a warm but non painful intensity and a high pain intensity. During the temporal generalisation task, therefore, participants felt the thermode being at high pain intensity during the training phase of the two pain conditions (pain immediate and pain delay). Experimental design and data analysis were as in Experiment 5.

### *7.2.2 – Results*

Participants selected 36.88°C (SD = 1.52) as warm intensity and 43.86°C (SD = 1.54) as high pain intensity during the initial intensity rating task. Paired-sample t-test indicated that participants selected a significantly higher temperature in the pain condition than in the no pain condition ( $p < .001$ ). Independent-sample t-test

indicated that participants selected a significantly higher temperature for the pain condition in Experiment 6 than in Experiment 5 ( $p = .008$ ).

Figure 7.2 shows temporal generalisation gradients depicting the mean proportion of YES responses in the four conditions plotted against comparison/standard ratio. Examination of Figure 7.2 suggests that in the no pain condition, there was no effect of delay on responding. In the pain condition, gradients appear to be shifted to the right following the delay. A repeated measures ANOVA with pain intensity (no-pain vs pain), delay (immediate vs delay) and comparison/standard ratio (0.625, 0.750, 0.875, 1, 1.125, 1.250 or 1.375) as within-subject factors was conducted. ANOVA showed significant main effects of ratio ( $F(2.21, 59.77) = 30.37, p < .001, \eta_p^2 = .53$ ), but no main effect of pain intensity ( $F(1, 27) = 0.25, p = .62, \eta_p^2 = .01$ ) nor delay ( $F(1, 27) = 0.12, p = .73, \eta_p^2 = .005$ ) on proportion of YES responses. There were also no significant interactions between delay and ratio ( $F(1.95, 52.60) = .72, p = .49, \eta_p^2 = .03$ ), pain intensity and delay ( $F(1, 27) = .39, p = .54, \eta_p^2 = .01$ ), pain intensity and ratio ( $F(1.79, 48.26) = 1.75, p = .19, \eta_p^2 = .06$ ) nor between pain intensity, delay and ratio ( $F(1.88, 50.78) = 1.02, p = .37, \eta_p^2 = .04$ ). YES responses were therefore unaffected by pain or delay.

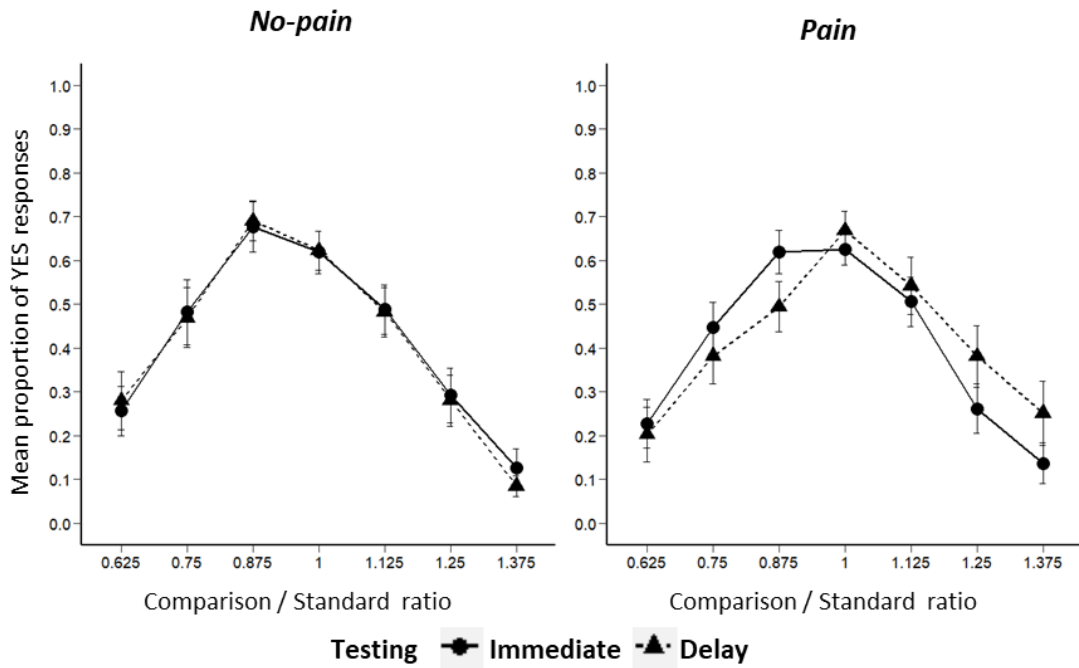


Figure 7.2. Proportion of YES responses plotted against comparison/standard ratio in Experiment 6. YES responses are divided between immediate (solid line) and delay (dotted line), and between no-pain (left panel) and pain (right panel).

#### Temporal accuracy

Table 7.3 shows temporal accuracy in the four conditions. Examination of Table 7.3 suggests that accuracy was similar in all conditions. A repeated measures ANOVA with pain intensity (no-pain vs pain) and delay (immediate vs delay) as within-subject factors confirmed these suggestions. There were no significant effects of pain intensity ( $F(1, 27) = .84, p = .37, \eta_p^2 = .03$ ) nor delay ( $F(1, 27) = .52, p = .48, \eta_p^2 = .02$ ) on accuracy. There was also no significant interaction effect between pain intensity and delay on accuracy ( $F(1, 27) = .10, p = .75, \eta_p^2 < .01$ ). Temporal accuracy was therefore unaffected by pain or delay.

#### Temporal variability

Table 7.3 shows temporal variability (i.e., mid-three) in the four conditions. Examination of Table 7.3 suggests that temporal variability was similar in all

conditions. A repeated measures ANOVA with pain intensity (no-pain vs pain) and delay (immediate vs delay) as within-subject factors confirmed these suggestions. There were no significant effects of pain intensity ( $F(1, 27) = .04, p = .85, \eta_p^2 < .01$ ) nor delay ( $F(1, 27) = .18, p = .68, \eta_p^2 < .01$ ) on mid-three. There was also no significant interaction effect between pain intensity and delay on mid-three ( $F(1, 27) = .59, p = .45, \eta_p^2 = .02$ ). Temporal variability was therefore unaffected by pain or delay.

<i>Condition</i>	<i>Accuracy</i>	<i>Mid-three</i>
<i>No-pain immediate</i>	0.62 (0.13)	0.62 (0.16)
<i>No-pain delay</i>	0.62 (0.13)	0.63 (0.15)
<i>Pain immediate</i>	0.63 (0.11)	0.64 (0.16)
<i>Pain delay</i>	0.65 (0.13)	0.61 (0.18)

*Table 7.3. Means (and standard deviations) of accuracy and mid-three in the four conditions (no-pain immediate, no-pain delay, pain immediate and pain delay) in Experiment 6.*

### 7.2.3 – Discussion

Experiment 6 tested whether a high level of pain intensity during the encoding of duration information would affect subsequent memory for duration. The results showed that temporal responses, temporal accuracy and temporal variability were similar between pain and no-pain conditions, suggesting that high pain had no significant effect on memory for duration. Furthermore, unlike in Experiment 5, there was also no significant effects of delay on responding, contrasting previous studies that have found effects of delay on perceived duration (Wearden & Ferrara, 1993).<sup>2</sup>

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<sup>2</sup> Additional analyses were conducted excluding participants who showed poor temporal sensitivity in the no-pain immediate condition. Results showed no difference from the findings here reported (see Appendix).

### 7.3 – Discussion Chapter 7

Whilst previous studies examined the effect of pain on perceived duration testing participants' perception immediately after the pain presentation (see Chapter 3, page 55), this study was the first to examine the effect of pain on perceived duration when participants are required to remember the duration over periods of delay. Participants completed a temporal generalisation task where they encoded a standard duration while experiencing concurrent neutral or painful somatosensory stimulation. Participants then recalled the standard duration, in the absence of somatosensory stimulation, either immediately or after a 15-minute delay. The effect of pain was tested in two experiments, where participants experienced a low pain intensity (Experiment 5) and a high pain intensity (Experiment 6).

For both pain intensities, when testing occurred immediately after the standard presentation, participants gave similar responses in the pain and no-pain conditions, suggesting that pain did not affect temporal performance. This is in line with Cocenas-Silva et al.'s (2013) study, which showed that emotions also did not affect the temporal performance in a temporal generalisation task when the testing phase occurred immediately after the learning phase.

Participants also gave similar responses in the pain and no-pain conditions when testing occurred 15 minutes after the encoding. This contrasts with the two possible outcomes that were expected. Pain was expected to either 1) decrease temporal accuracy and temporal discrimination because pain disrupts cognitive processes required for memory, or 2) increase temporal accuracy and temporal discrimination because pain facilitates the memory consolidation in the hippocampus. Experiments 5 and 6 showed that temporal performances in the delay conditions were not affected by the painful thermal stimulations, suggesting that pain neither disrupted nor enhanced the memory for duration.

The absence of an effect of pain suggests that, unlike emotion (Cocenas-Silva et al., 2013), pain does not improve the memorization of duration. However, it is possible that these different findings were due to methodological differences. Cocenas-Silva et al. (2013) used a 24-hour delay between learning and testing phase, which reduced the perceived duration of the neutral stimulus and left the perceived duration of the emotional stimulus unaffected. In contrast, the present studies used a 15-minute delay, which reduced the temporal variability of both neutral and pain related stimuli only in Experiment 5. It is therefore possible that a longer retention period would elicit a pain effect not currently evident. However, it should be noted that delay effects were observed in studies with shorted retention periods; for example, Wearden and Ferrara (1993) found that a 10-second delay between learning and testing phases affected temporal performance leading to subjective shortening of the standard duration. Here, 15 minutes was chosen as a sufficient interval delay also because Lechner et al. (1999) showed that the consolidation process is completed after 12 minutes and that the number of errors are not different when recalling a list of words after 12 minutes or after 24 hours. Therefore, there is no evidence suggesting that testing participants after 15 minutes or 24 hours as in Cocenas-Silva et al. (2013) should make a significant difference.

Another possibility is that pain did not affect memory for duration because the pain was not task-relevant. Chapters 5 and 6 showed that task-relevancy is a determinant feature in the pain distortions to time. It is therefore possible that to better remember the duration of a neutral stimulus, even if presented concomitantly to a painful stimulation, might not bring any survival advantage. Similar suggestions were made by Cocenas-Silva et al. (2013), who suggested that emotion effects on memory for duration were greater for threatening stimuli (rather than nonthreatening stimuli) for basic survival reasons. It is therefore possible that pain could affect the memory for duration if it is task related.

In summary, Experiments 5 and 6 showed that pain does not affect memory for duration. Replications of this study could test whether the duration of painful stimuli



themselves, which are overestimated (Experiments 1 and 3), are also overestimated after a period of delay. This could be particularly relevant in clinical contexts, where doctors may want to know how long pain lasted for long after its occurrence.

## Chapter 8

### **Establishing whether mindfulness meditation can reduce the perceived duration of pain.**

The duration and intensity of pain have a bidirectional interaction; increasing the *subjective* duration of pain increases its *subjective* intensity (Coldwell et al., 2002; Pomares et al., 2011) and increasing the intensity of pain increase the *subjective* duration of pain (see Chapter 5 and 6). The lengthening effect of pain has negative impacts on the sufferer, especially in clinical contexts (Somov, 2000) where it is associated with pain anxiety and reduced clinical compliance (Boivin et al., 2008). Therefore, an intervention that attenuates the perceived duration of pain could potentially lead to improved wellbeing and less distress.

To date, beyond pharmacological treatments, attempts to improve sufferers' conditions have focused on psychological interventions which aim to reduce the perceived intensity of pain (Roditi & Robinson, 2011; Turk, Swanson, & Tunks, 2008). These studies have shown that psychophysiological, cognitive-behavioural and psychodynamics approaches, such as biofeedback (Cornel, van Haarst, Schaarsberg, & Geels, 2005), Operant-Behavioural Therapy (Thieme, Turk, & Flor, 2007) and motivational interviewing (Alperstein & Sharpe, 2016), are effective in reducing the perceived intensity of pain (see Adams, Poole, & Richardson, 2006 for review). However, to date, studies have neglected to examine the potential effects of interventions on shortening the perceived duration of pain, despite the fact that perceived duration of pain (i.e., how long it has lasted) forms part of the clinical assessment (Somov, 2000).

Among these psychological interventions, Mindfulness Meditation (MM) has been regularly used for pain management in clinical settings and shows patient benefit (Veehof, Oskam, Schreurs, & Bohlmeijer, 2011; Veehof et al., 2016). MM is a practice originated from the core teaching of early Buddhist traditions (Malinowski, 2017), through which practitioners aim to reach a state of awareness of one's own thoughts,

feelings and surroundings with a non-judging attitude (Bishop et al., 2004; Kabat-Zinn, 1990). This practice has been adapted in a number of intervention programmes, which successfully reduce sufferers' symptoms (Morone, Greco, & Weiner, 2008). Mindfulness-based programs have been found to reduce perceived pain intensity with effect sizes ranging from small to moderate (Lakhan & Schofield, 2013; Veehof et al., 2016), independently of whether patients received the intervention via face-to-face or via videoconference (Gardner-Nix, Backman, Barbati, & Grummitt, 2008). Furthermore, 8 weeks of mindfulness practice improved pain acceptance and physical function in a chronic low back pain population (Morone et al., 2008) and Mindfulness Based Cognitive Therapy (MBCT; Segal, Teasdale, Williams, & Gemar, 2002) reduced pain severity ratings and pain sensitivity to experimental nociceptive stimuli in a clinical population with low back chronic pain (Zgierska et al., 2016).

Mindfulness practice has been also adapted in intervention programmes that target conditions other than pain. A whole range of mindfulness-based programs have been developed for the treatment of clinical conditions, such as mindfulness-based eating awareness training and mindfulness-based childbirth and parenting (see Chiesa & Malinowski, 2011 for review). Among these, Mindfulness-Based Stress Reduction (MBSR; Kabat-Zinn, 1982) and MBCT have become the most common meditation programs used for improving health and wellbeing in clinical settings (Baer, 2003). For example, MBCT is recommended by the UK's National Health Service as a standard treatment for people with previous episodes of depression (NICE, 2009) and Kuyken et al. (2015) found that it is a valid alternative to standard antidepressant treatments.

Mindfulness practice is believed to modulate the pain experience of sufferers (Zeidan et al., 2010) by improving self-regulation of emotions (Hölzel et al., 2011) and emotional cognitive flexibility (Bishop et al., 2004). MM ability of improving self-regulation of emotions is indexed by the physiological changes during MM sessions (Hölzel et al., 2011). Parasympathetic activity was found to increase (Kubota et al., 2001; Wu & Lo, 2008), meanwhile sympathetic activity was found to decrease during

MM (Lush et al., 2009), indicating a greater PSNS dominance and a reduced physiological arousal during MM. Within the mindfulness field, PSNS dominance over SNS is thought to indicate an increase of self-regulation of emotions (Hölzel et al., 2011). MM therefore reduce pain experience in sufferers by reducing physiological arousal, which is associated with the perceived pain intensity (see section 3.1.2, page 56).

MM also improves emotional cognitive flexibility (Malinowski, 2017); for example, MM was found to improve self-regulating attention (Mirams et al., 2013) and cognitive flexibility capacity (Bishop et al., 2004), and MBCT was found to improve the ability to inhibit cognitive automatic responses (Heeren, Van Broeck, & Philippot, 2009) and autobiographical memory recall (Williams, Teasdale, Segal, & Soulsby, 2000). Moore et al. (2012) also showed that MM selectively modulates the electroencephalographic markers (EEG, ERPs) of attentional control. These effects of MM on cognition are thought to promote pain regulation by enhancing the activity of ACC and AIC (Zeidan et al., 2011), neural areas involved in the descending (top down) pathway for pain regulation, resulting in reduced perceived pain intensity (see section 3.1.5, page 60).

MM also affects time perception. Kramer et al. (2013) asked participants to complete a temporal bisection task before and after completing either a MM session or a control audiobook listening exercise. PSE was shifted to the left following MM but was unaffected by the listening exercise, suggesting that MM lengthened perceived duration. This was confirmed by Droit-Volet et al. (2015), who found a left shift of the PSE in a temporal bisection task after that participants had conducted an intensive MM training consisting of a 10-minute session per day for 5 weeks. Interestingly, the self-measured mindfulness disposition of participants also affects their temporal performance on temporal reproduction tasks, with greater mindfulness disposition associated with longer and more accurate reproductions (Wittmann et al., 2014). These studies explained the effect of MM and mindfulness disposition on perceived duration as an attentional effect; mindfulness improve

attentional regulation of participants leading to longer and more accurate perceptions of duration.

Previous studies observing a lengthening effect on time perception with MM, however, have exclusively used neutral stimuli. The effect of mindfulness practice on the perceived duration of pain remains therefore unclear. It is plausible that MM may reduce the perceived duration of pain because it decreases subjective pain intensity (Veehof et al., 2016) and reduces pain-induced physiological arousal (Hölzel et al., 2011), which are predictors of perceived duration (Coldwell et al., 2002; Piovesan et al., 2018). Furthermore, MM shares neural correlates, namely the insular cortex, with pain and time perception (Craig, 2002, 2009a; Young et al., 2018). Mindfulness effects on insular activity are thought to be causal for MM to reduce the perceived intensity of pain (see Bilevicius, Kolesar, & Kornelsen, 2016 for discussion). It is therefore possible that MM could also affect the insular activity resulting in shorter perceived duration of pain.

Experiment 7 tested this possibility by comparing the effects of mindfulness and a control story listening task on verbal estimates of visual, tactile and painful stimuli. In the study, participants completed the three verbal estimation tasks before and after a 1-week mindfulness intervention or a 1-week control story listening exercise. As in previous experiments, participants selected the subjective pain intensity of the electrical shock during an initial task. Following this, participants were allocated to either the mindfulness or control condition. In the mindfulness condition, participants were asked to perform a body-scan exercise, which has previously been shown to enhance somatosensory perception and which involved directing attention toward one's own body, once a day for one week. In the control condition participants listened for an audiobook for the same amount of time. Following this, participants were re-tested on the three verbal estimation tasks.

Mindfulness was expected to have different effects on the perceived duration of painful stimuli than of the visual and vibrotactile stimuli, because of the differing

effects of the stimuli on SNS activity and attentional processing. The mindfulness intervention was expected to decrease physiological arousal and increase self-regulated attention (Hölzel et al., 2011), leading to decreases in verbal estimates and estimate variability of painful stimuli. In contrast, mindfulness intervention was expected to increase verbal estimates of visual and tactile stimuli and to reduce estimate variability, confirming previous studies (Droit-Volet et al., 2015; Kramer et al., 2013). Critically, differences in verbal estimates and estimate variability were expected only with the mindfulness intervention group. No changes in perceived durations were expected with the group practicing the control story listening exercise. These predictions were tested in two groups of participants; 1) healthy people (Experiment 7) and 2) chronic pain patients (Experiment 8).

## **8.1 – Experiment 7**

### *8.1.1 – Method*

#### 8.1.1.1 – Participants

Forty-four participants (31 females and 13 males; mean age = 38.66, SD = 18.88) were recruited. Participants were required not to be pregnant, not to have a history of epilepsy and not to have chronic pain, heart disease, skin problems (e.g., eczema) or any impairment of body sensation. Additionally they were asked not to take any analgesic during the 8 hours prior to the experiment. Regular mindfulness meditators (i.e., those who meditated more than once a month) were also excluded. Participants were reimbursed £15 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.

#### 8.1.1.2 – Experimental design

This experiment replicated the experimental design described by Mirams et al. (2013). Each participant was assigned to either an intervention group, who practiced a mindfulness meditation exercise; or the control group, who practiced a story

listening exercise. Participants were assigned to each group in alternate order based on their participant number. For each participant, active participation in the study took 8 days (see Figure 8.1 for a scheme of the experimental structure). On the first day, participants attended the pre-testing session which involved 1) completing a series of questionnaires including a health screening questionnaire to confirm suitability to participate; 2) the intensity rating task; 3) the verbal estimation task and 4) the assigned intervention exercise (mindfulness meditation or story listening). On days two to seven, participants were asked to complete their assigned exercise in their own time at home and complete a diary recording their experience. On day eight, participants attended a post-testing session, which involved performing their assigned intervention exercise, followed by the questionnaires and verbal estimation task.

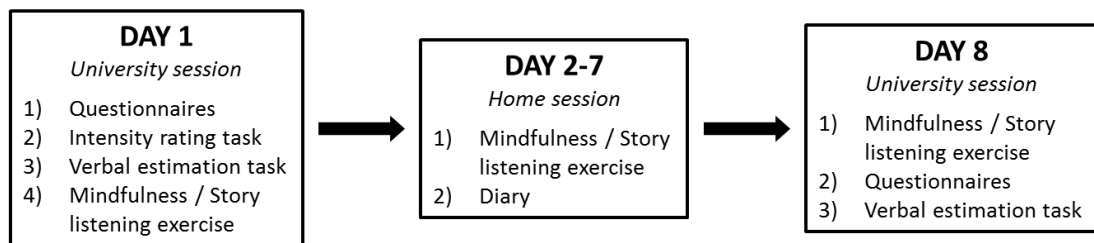


Figure 8.1. Experiment 7 structure.

### 8.1.1.3 – Apparatus and material

*Questionnaires:* Four questionnaires were administered: the Pain Catastrophizing Scale (PCS; Sullivan, Bishop, & Pivik, 1995), the Patient Health Questionnaire-9 (PHQ-9; Kroenke, Spitzer, & Williams, 2001), the Depression, Anxiety and Stress Scale - 21 Items (DASS-21; Lovibond & Lovibond, 1995) and the Mindful Attention Awareness Scale (MAAS; Brown & Ryan, 2003; Carlson & Brown, 2005). The PCS was used to determine the exaggerated negative thoughts and feelings during pain experience. The PHQ-9 was used to determine the depressive feelings of participants. The DASS-21 was used to determine the depressive, anxious and stress feelings. The MAAS was used to determine the mindfulness attitude of participants. Questionnaire scores of the first and the second session were compared to test the efficacy of the

mindfulness intervention. Questionnaire scores were also used to investigate whether catastrophizing, depression, anxiety, stress and mindfulness attitude correlated with verbal estimates.

The PCS consisted of 13 items indicating thoughts and feelings usually associated to the pain experience (e.g., I worry all the time about whether the pain will end). Participants were asked to use a 5-point rating scale to rate the degree of which they have those thoughts and feelings while experiencing pain (0 – Not at all; 4 – All the time). Final scores were divided in Total scores (score range: 0–52) and three subscales: Rumination (score range: 0–16), Magnification (score range: 0–12) and Helplessness (score range: 0–24). Higher scores indicated higher pain catastrophizing, rumination, magnification and helplessness, respectively. PCS has acceptable reliability and validity (Osman et al., 1997).

The PHQ-9 consisted of 9 items indicating thoughts usually associated to depression (e.g., feeling down, depressed or hopeless). Participants were asked to use a 4-point rating scale to rate how often they have been bothered by those thoughts in the previous two weeks (0 – Not at all; 3 – Nearly every day). Final scores ranged from 0 to 27 with higher scores indicating more depressive feelings. PHQ-9 has high reliability and validity (Löwe, Unützer, Callahan, Perkins, & Kroenke, 2004).

The DASS-21 consisted of 21 items indicating thoughts and feelings usually associated to depression (e.g., I couldn't seem to experience any positive feeling at all), anxiety (e.g., I felt scared without any good reason), and stress (e.g., I tended to over-react to situations). Participants were asked to use a 4-point rating scale to rate how much those thoughts applied to them over the past week (0 – Not at all; 3 – Applied to me very much or most of the time). Final scores were divided in three subscales: Depression (score range: 0–56), Anxiety (score range: 0–56) and Stress (score range: 0–56). Higher scores indicated higher depression, anxiety and stress, respectively. DASS-21 has high reliability and validity (Henry & Crawford, 2005).

The MAAS consisted of 15 items indicating daily experience that are associated with being inattentive and opposite to core characteristics of mindfulness (e.g., I rush through activities without being really attentive to them). Participants were asked to rate how often they had these experience on a 6-point Likert scale (1 – Almost always;



6 – Almost never). Final scores ranged from 15 to 90 with higher scores indicating higher mindfulness attitude. The MAAS has acceptable validity and reliability (Brown & Ryan, 2003).

*Tactile vibrations:* A tactile pulse (vibration) was used as the non-noxious stimulus. Tactile vibrations were produced on participants' left arm by using a TactAmp, 4.2 (Dancer Design, St Helens, UK) with identical settings of Experiment 1 (see section 5.1.1.2, page 86). The intensity of the stimulus was with amplitude equal to 1.

*Pain stimulation:* The Digitimer DS7A Current Stimulator (Digitimer Ltd) was used to present the electro-cutaneous stimulation (see section 4.3.1, page 77 for equipment description).

*Intensity rating task:* The procedure was the same as the one used in previous studies and described in the methodology chapter (see section 4.3.1, page 77). For this study, participants were asked to select a single pain intensity rated as a 5 on the Numeric Rating Scale. A 5 indicates a moderate intensity of pain (Aun, Lam, & Collett, 1986). Participants completed this task in the first testing session only and the selected intensity was used for the painful condition of the verbal estimation task during both the pre- and post-testing sessions.

*Verbal estimation task:* The procedure was the same as the one used in previous studies and described in the methodology chapter (see section 4.4, page 80). The task included three conditions, using the following stimulus modality: visual, tactile and painful modality. The visual stimulus consisted of a white square (300x300 pixels, 8x8cm) on a black background. The tactile stimulus consisted of a tactile vibration presented to participants' left arm. The painful stimulus consisted of an electric shock, with the intensity selected during the intensity rating task, presented to participants' left arm. Conditions were presented to participants in randomized order. In each condition, participants completed 3 blocks of 16 stimuli; five standard duration (242ms, 455ms, 767ms, 1058ms and 1296ms) each of which was repeated

twice and six 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 five experimental stimulus durations across the experimental blocks. The data from these six additional stimuli were not analysed (as in Ogden et al., 2015). Each condition therefore contained a total of 48 trials. An inter trial interval, the duration of which was selected from a normal distribution between 1500 to 2500 milliseconds, was interposed between trials. The order of presentation of the trials was randomised by E-Prime for each participant. Throughout the task, participants listened to white noise through headphones to prevent any auditory feedback influencing performance.

*Mindfulness exercise:* Participants were asked to sit, close their eyes and relax whilst listening to an audio recording of a guided mindfulness body-scan exercise which has been used in previous studies (MacIver, Lloyd, Kelly, Roberts, & Nurmikko, 2008; Mirams et al., 2013). Participants were given two 20-minute audio tracks to use during the week. Both tracks started with 2-minute introduction of MM where participants were encouraged to observe the sensations on their body during the session without judgment. Both tracks continued suggesting a comfortable position and asking participants to relax through directing their attention to the breath cycle for 1 minute. Participants were then instructed to direct their attention to different parts of their body in succession, noticing the various sensations they felt in each area. In track one, participants were asked to focus on the bottom part of the body (i.e., legs, feet, toes, ankles, knees, pelvis and hips). In track two, participants were asked to focus on the top part of the body (i.e., chest, upper back, neck, shoulders, arms, fingers, head, face, eyes and jaw). Both tracks then ended asking participants to direct their attention again to the breath cycle and to be aware of the surrounding until opening their eyes. During the 8-day intervention, participants listened to the two tracks in alternate order and half of participants started with track one and ended with track two, and half participants did the opposite. All participants listened to each track four times, completing eight twenty-minute meditation exercise in total. For

the home practice, participants were told to practice meditation at any moment during the day they felt comfortable.

*Story listening exercise:* Participants were asked to sit, close their eyes and relax meanwhile listening to ‘The Jungle Book’ audiobook for 20 minutes. The audiobook was retrieved from the LibriVox recordings (librivox.org) and the first 160 minutes of the story were divided in 8 tracks of 20 minutes each using Audacity software (www.audacityteam.org). Participants listened to one track per day in narrative order.

*Diary:* Every day, after the assigned exercise, participants were asked to rate on a four point scale 1) the difficulty of the exercise (0 – extremely easy; 3 – extremely difficult), 2) the effort taken to pay attention to the audio-track (0 – none at all; 3 – a great deal) and 3) the frequency of distraction (0 – very rare; 3 – extremely often). This diary was used to encourage and assess participants’ adherence during the home sessions and the data obtained with the diary was not analysed.

### 8.1.2 – Results

Out of forty-four participants tested, data from three participants were excluded from the analysis because they did not attend the second session. Therefore, it has been reported the results based on data from the remaining 41 participants, 22 in the mindfulness group and 19 of the story listening group. Participants’ diary reported that there was 94.81% adherence and no participants missed more than two home sessions. To establish the effect of the intervention on time perception three measures of timing were computed: mean estimate, estimate accuracy and estimate variability. Greenhouse-Geisserr correction was applied to ANOVAs when the Sphericity assumption was violated and post-hoc were Bonferroni corrected.

Verbal estimates

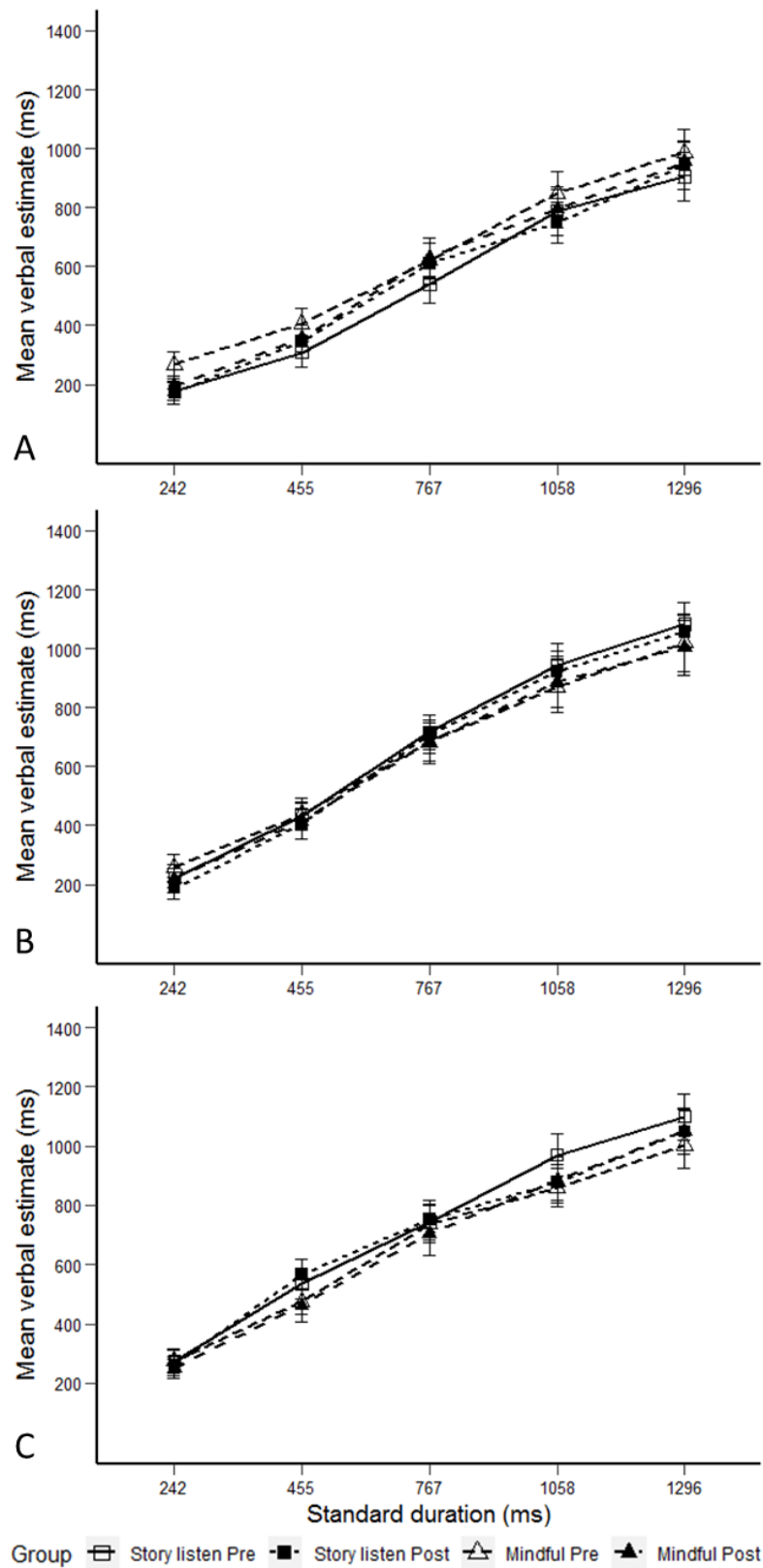


Figure 8.2. Means (and standard errors) of the verbal estimates (ms) plotted against the stimulus duration and divided by group (mindfulness and story listening) and

*by session (pre and post). The three plots show the verbal estimates in the visual modality (panel A), tactile modality (panel B) and painful modality (panel C).*

Figure 8.2 shows the verbal estimates of the visual, tactile and painful stimuli. The three graphs show the verbal estimates of the story listening and MM group in the pre- and post-session separately. A mixed ANOVA with group (MM and story listening) as a between subjects factor and session (pre and post), stimulus modality (visual, tactile and painful) and stimulus duration (242ms, 455ms, 767ms, 1058ms and 1296ms) as within subject factors was conducted. This showed significant main effects of stimulus duration  $F(1.44, 56.15) = 399.35, p < .001, \eta_p^2 = .91$  and stimulus modality  $F(1.74, 68.02) = 8.57, p = .001, \eta_p^2 = .18$  on verbal estimates. Post-hoc tests showed that estimates were significantly shorter in the visual modality compared to the tactile ( $p = .004$ ) and painful modalities ( $p = .003$ ). Verbal estimates were not significantly different between tactile and painful modality ( $p = .717$ ). There was no significant main effect of group  $F(1, 39) = 0.002, p = .960, \eta_p^2 < .001$  nor session  $F(1, 39) = .37, p = .545, \eta_p^2 = .01$ . There was however a significant interaction between stimulus duration and modality  $F(3.60, 140.27) = 2.83, p = .032, \eta_p^2 = .07$  but no other significant interaction was found (all  $ps > .05$ ).

To further investigate the interaction effect between stimulus duration and modality, a repeated measure ANOVA with stimulus modality (visual, tactile and painful) as within subject factors was conducted on each stimulus duration separately (242ms, 455ms, 767ms, 1058ms and 1296ms). ANOVAs showed significant main effect of stimulus modality on verbal estimates of the 242ms duration  $F(2, 80) = 5.18, p = .008, \eta_p^2 = .12$ , the 455ms duration  $F(1.52, 60.72) = 14.96, p < .001, \eta_p^2 = .27$ , the 767ms duration  $F(1.63, 65.23) = 8.50, p = .001, \eta_p^2 = .18$ , the 1058ms duration  $F(2, 80) = 4.34, p = .016, \eta_p^2 = .10$ , and the 1296ms duration  $F(2, 80) = 3.29, p = .042, \eta_p^2 = .08$ . Post-hoc tests showed that verbal estimates were significantly longer in the painful modality compared to the tactile modality for the 455ms duration ( $p < .001$ ), meanwhile the estimates for the 242, 767, 1058 and 1296ms stimuli did not differ (all

$p > .05$ ). Estimates were significantly longer in the tactile modalities than the visual modality for all ( $p < .05$ ) but the shortest stimulus ( $p = .99$ ). Estimates were significantly longer in the painful modalities compared to the visual modality for the 242ms, 455ms and 767ms stimuli (all  $p < .05$ ), but not for the 1058ms and 1296ms stimuli ( $p > .05$ ).

### *Estimate accuracy*

Estimate accuracy was calculated for each stimulus duration of each modality in each session using the same formula used in Experiments 1 and 2: verbal estimate/stimulus duration. An accuracy of 1 indicates a correct estimate, below 1 indicates underestimation of duration and above 1 indicates overestimation of duration.

Figure 8.3 shows the estimate accuracy of visual, tactile and painful stimuli. The three graphs show the estimate accuracy of the story listening and MM group in the pre- and post-session separately. A mixed ANOVA with group (mindfulness and story listening) as a between subject factor and with session (pre and post), stimulus modality (visual, tactile and painful) and stimulus duration (242ms, 455ms, 767ms, 1058ms and 1296ms) as within subject factors was conducted. ANOVA showed significant main effects of stimulus duration  $F(1.28, 50.06) = 5.08, p = .021, \eta_p^2 = .12$  and stimulus modality  $F(2, 78) = 9.84, p < .001, \eta_p^2 = .20$  on estimate accuracy. Post-hoc tests showed that accuracy was significantly lower in the visual modality compared to the tactile ( $p = .024$ ) and painful modalities ( $p = .001$ ). Estimate accuracy was not significantly different between tactile and painful modality ( $p = .128$ ). There was no significant main effect of group  $F(1, 39) = 1.91, p = .174, \eta_p^2 = .05$  nor session  $F(1, 39) = 1.11, p = .299, \eta_p^2 = .03$  on estimate accuracy. There were however significant interactions between stimulus duration and modality  $F(3.08, 120.24) = 4.667, p = .004, \eta_p^2 = .11$  and between stimulus duration and session  $F(1.44, 56.10) = 3.59, p = .048, \eta_p^2 = .08$ , but no other significant interaction was found (all  $p > .05$ ).

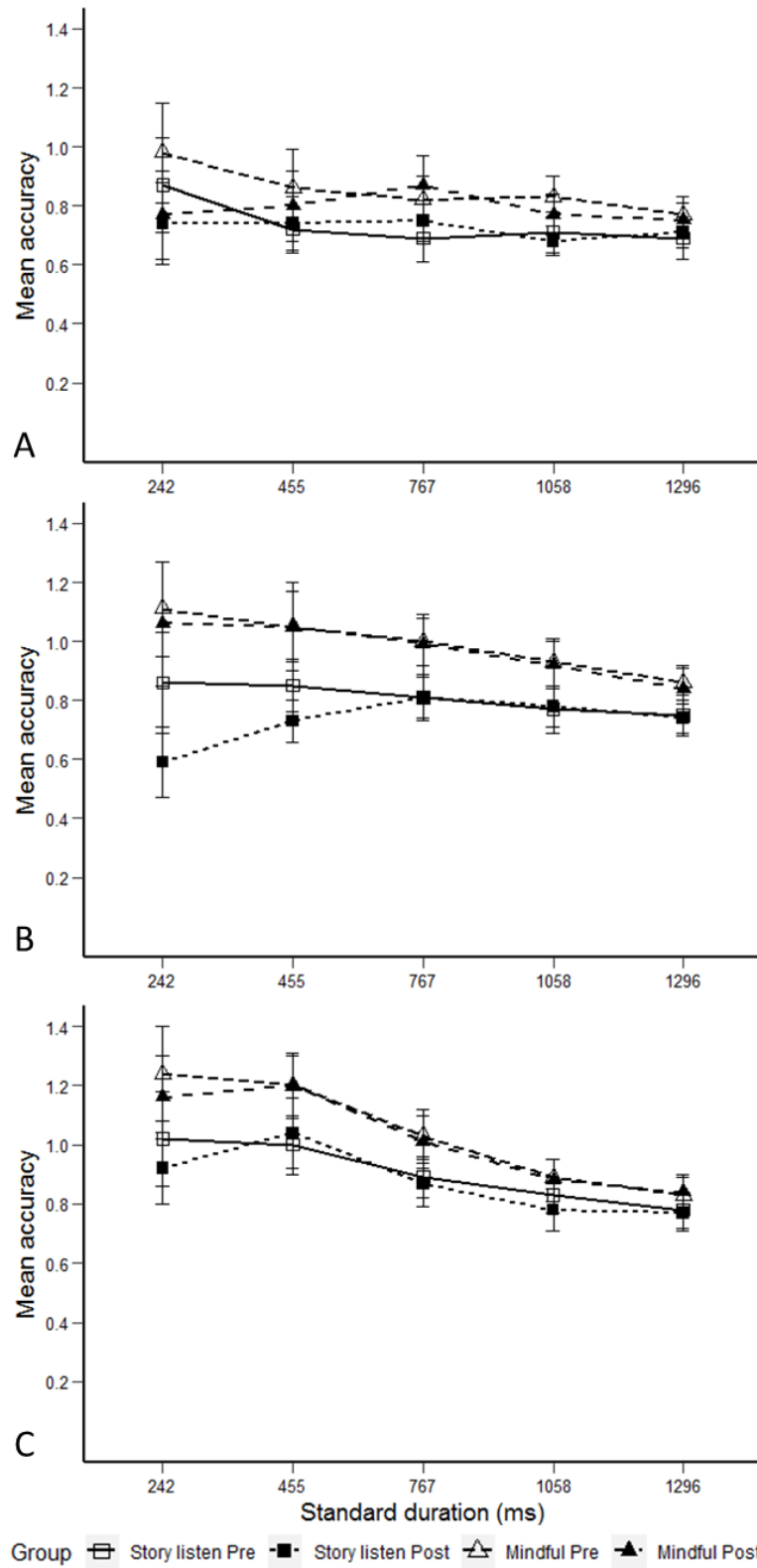


Figure 8.3. Means (and standard errors) of the estimate accuracy plotted against the stimulus duration and divided by group (mindfulness and story listening) and by session (pre and post). The three plots show the verbal estimates in the visual modality (panel A), tactile modality (panel B) and painful modality (panel C).

To further investigate the interaction effect between stimulus duration and modality, a repeated measure ANOVA with stimulus modality (visual, tactile and painful) as within subject factors was conducted per each stimulus duration (242ms, 455ms, 767ms, 1058ms and 1296ms). ANOVAs showed significant main effect of stimulus modality on estimate accuracy of the 242ms duration  $F(2, 80) = 5.19, p = .008, \eta_p^2 = .12$ , the 455ms duration  $F(1.52, 60.85) = 14.90, p < .001, \eta_p^2 = .27$ , the 767ms duration  $F(1.63, 65.30) = 8.48, p = .001, \eta_p^2 = .18$ , the 1058ms duration  $F(2, 80) = 4.32, p = .017, \eta_p^2 = .10$ , and the 1296ms duration  $F(1.75, 69.92) = 3.35, p = .047, \eta_p^2 = .08$ . Post-hoc tests indicated that estimate accuracy was significantly higher in the painful modalities compared to the tactile modality for the 455ms stimuli ( $p < .001$ ), meanwhile estimate accuracy for the 242, 767, 1058 and 1296ms stimuli did not differ (all  $ps > .05$ ). Estimate accuracy was significantly higher in the tactile modalities compared to the visual for all ( $ps < .05$ ) but the shortest stimuli ( $p = .99$ ). Estimate accuracy was significantly higher in the painful modalities compared to the visual modality for the 242ms, 455ms and 767ms stimuli (all  $ps < .05$ ), but not for the 1058ms and 1296ms stimuli ( $p > .05$ ). Whilst verbal estimates were similarly accurate between tactile and painful stimuli, they were consistently more accurate for the tactile stimuli than for the visual ones except the shortest duration; meanwhile verbal estimates were more accurate for the painful stimuli than for the visual ones for the shortest durations.

To further investigate the interaction effect between stimulus duration and session, a paired-sample t-test between the first and second session was conducted per each stimulus duration (242ms, 455ms, 767ms, 1058ms and 1296ms). Paired-sample t-test showed no significant difference between estimate accuracy of the first and second session for the 455ms ( $p = .581$ ), 767ms ( $p = .795$ ), 1058ms ( $p = .421$ ) and 1296ms ( $p = .834$ ) durations, meanwhile the difference of estimate accuracy for the 242ms failed to reach significance ( $p = .073$ ).



### *Estimate variability*

Estimate variability was calculated for each stimulus duration of each modality in each session using the same formula used in Experiments 1 and 2: standard deviation verbal estimate/mean verbal estimate. The higher the value, the more variable the participant's responses.

Figure 8.4 shows the estimate variability of visual, tactile and painful stimuli. The three graphs show the estimate variability of the story listening and mindfulness group in the pre- and post-session separately. A mixed ANOVA with group (mindfulness and story listening) as a between subject factor and with session (pre and post), stimulus modality (visual, tactile and painful) and stimulus duration (242ms, 455ms, 767ms, 1058ms and 1296ms) as within subject factors was conducted. ANOVA showed significant main effects of stimulus duration  $F(1.87, 73.06) = 18.48, p < .001, \eta_p^2 = .32$  and stimulus modality  $F(2, 78) = 4.12, p = .020, \eta_p^2 = .10$  on estimate variability. Post-hoc tests showed that variability was significantly higher in the visual modality compared to the tactile modality ( $p = .010$ ). Estimate variability in painful modality did not significantly differ to tactile ( $p = .541$ ) and visual modalities ( $p = .503$ ). There was no significant main effect of group  $F(1, 39) = .15, p = .697, \eta_p^2 = .004$  nor session  $F(1, 39) = .64, p = .429, \eta_p^2 = .02$  on estimate variability. There were no significant interactions (all  $ps > .05$ ), with the modality\*duration and modality\*exercise interactions that just failed to reach significance ( $ps = .089$ )

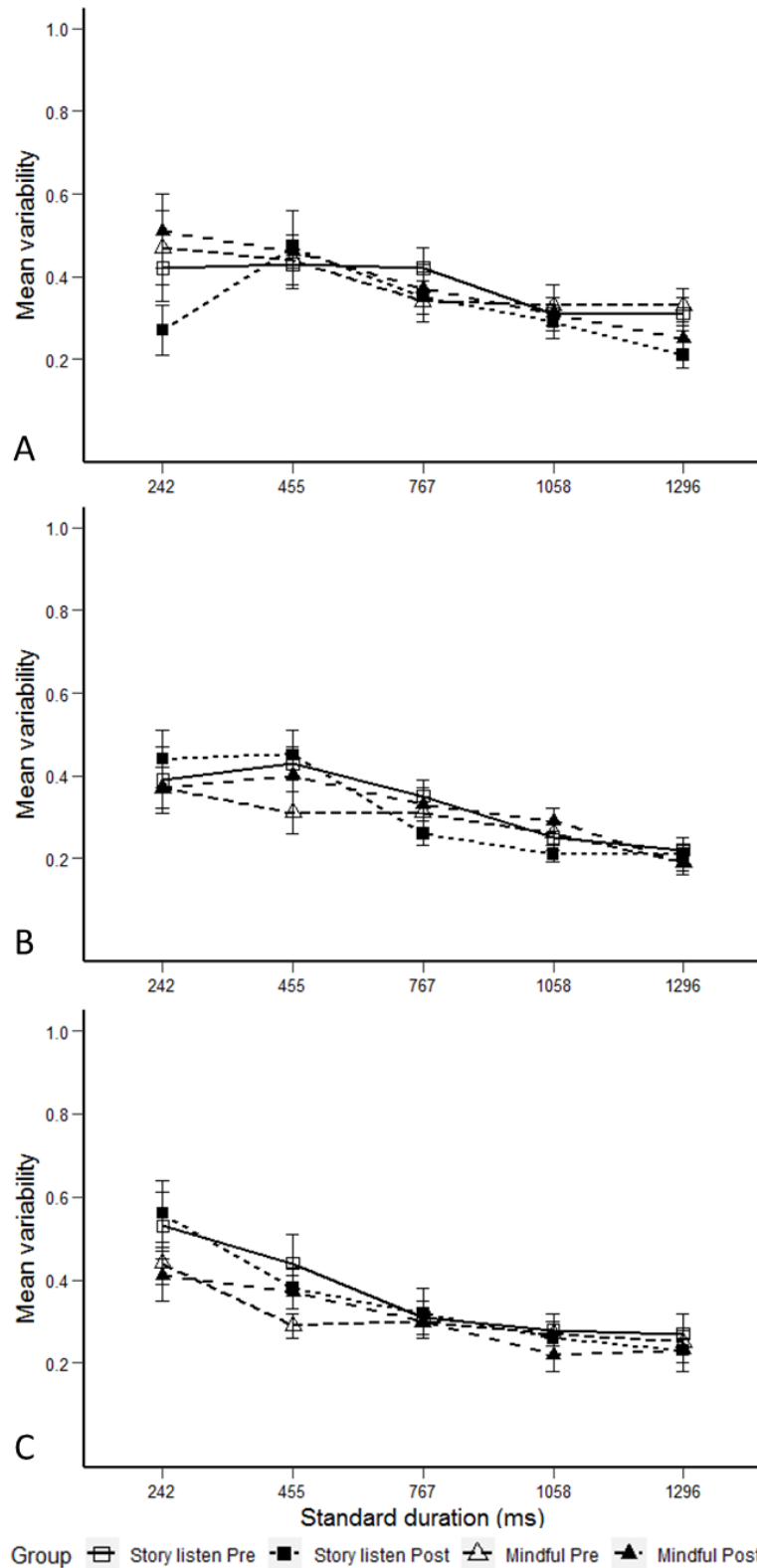


Figure 8.4. Means (and standard errors) of the estimate variability plotted against the stimulus duration and divided by group (mindfulness and story listening) and by session (pre and post). The three plots show the verbal estimates in the visual modality (panel A), tactile modality (panel B) and painful modality (panel C).

Questionnaires

Group	Story listening		Mindfulness	
	Pre	Post	Pre	Post
<i>PCS total</i>	21.63 (13.23)	14.79 (8.53)	12.05 (8.72)	8.55 (8.35)
<i>PCS Rumination</i>	8.00 (4.81)	5.32 (3.50)	4.32 (3.23)	3.91 (3.70)
<i>PCS Magnification</i>	4.63 (2.83)	3.68 (2.33)	2.77 (2.67)	1.73 (1.91)
<i>PCS Helplessness</i>	9.47 (6.26)	6.00 (3.90)	5.23 (4.12)	3.00 (3.78)
<i>DASS Depression</i>	8.42 (6.27)	3.47 (3.58)	7.18 (5.65)	4.00 (4.98)
<i>DASS Anxiety</i>	7.26 (6.15)	4.00 (3.65)	6.09 (6.63)	4.18 (5.52)
<i>DASS Stress</i>	14.32 (10.61)	8.63 (6.67)	11.82 (7.90)	8.27 (6.30)
<i>MAAS total</i>	3.78 (0.93)	4.09 (0.71)	4.01 (0.60)	4.10 (0.59)
<i>PHQ-9 total</i>	6.58 (5.47)	4.74 (3.68)	5.05 (4.10)	4.45 (3.74)

Table 8.1. Means (and standard deviations) of questionnaire scores divided by group (story listening and mindfulness) and by session (pre and post). PCS = Pain Catastrophizing Scale. DASS = Depression, Anxiety and Stress Scale. MAAS = Mindful Attention Awareness Scale. PHQ-9 = Patient Health Questionnaire-9.

Table 8.1 shows questionnaire scores divided by group and session. A mixed ANOVA with group (mindfulness and story listening) as between subjects factor and with session (pre and post) as within subject factor was conducted per each questionnaire and subscale.

ANOVA showed significant main effect of session  $F(1, 39) = 17.65, p < .001, \eta_p^2 = .31$  and group  $F(1, 39) = 7.89, p = .008, \eta_p^2 = .17$  on PCS total score. Participants scored higher in the pre-session than in the post-session and the story listening group scored

higher compared to the mindfulness group. There was no significant interaction between session and group  $F(1, 39) = 1.84, p = .182, \eta_p^2 = .05$ .

ANOVA showed significant main effect of session  $F(1, 39) = 9.38, p = .004, \eta_p^2 = .19$  and group  $F(1, 39) = 5.48, p = .024, \eta_p^2 = .12$  on PCS Rumination. Participants scored higher in the pre-session than in the post-session and the story listening group scored higher compared to the mindfulness group. There was a significant interaction between session and group  $F(1, 39) = 5.08, p = .030, \eta_p^2 = .12$ . To further investigate this interaction, a paired-sample t-test was conducted per each group and an independent sample t-test was conducted per each session. Paired sample t-test showed that story listening group had higher PCS Rumination scores in the pre-session than in the post-session  $t(18) = 3.58, p = .002$  and that scores of the mindfulness group did not change between pre- and post-sessions  $t(21) = .60, p = .554$ . Independent sample t-test showed a significant difference of PCS Rumination scores between story listening and mindfulness group in the pre-session  $t(30.70) = 2.83, p = .008$ , but not in the post-session  $t(39) = 1.25, p = .220$ .

ANOVA showed significant main effect of session  $F(1, 39) = 8.59, p = .006, \eta_p^2 = .18$  and group  $F(1, 39) = 7.68, p = .009, \eta_p^2 = .17$  on PCS Magnification. Participants scored higher in the pre-session than in the post-session and the story listening group scored higher compared to the mindfulness group. There was no significant interaction between session and group  $F(1, 39) = .02, p = .886, \eta_p^2 < .01$ .

ANOVA showed significant main effect of session  $F(1, 39) = 21.88, p < .001, \eta_p^2 = .36$  and group  $F(1, 39) = 7.80, p = .008, \eta_p^2 = .17$  on PCS Helplessness. Participants scored higher in the pre-session than in the post-session and the story listening group scored higher compared to the mindfulness group. There was no significant interaction between session and group  $F(1, 39) = 1.05, p = .313, \eta_p^2 = .03$ .

ANOVA showed significant main effect of session on DASS Depression  $F(1, 39) = 24.32, p < .001, \eta_p^2 = .38$ . Participants scored higher in the pre-session than in the post-session. There was no significant main effect of group  $F(1, 39) = .06, p = .802, \eta_p^2 < .01$  nor interaction between session and group  $F(1, 39) = 1.15, p = .291, \eta_p^2 = .03$  on DASS Depression.

ANOVA showed significant main effect of session on DASS Anxiety  $F(1, 39) = 10.25$ ,  $p = .003$ ,  $\eta_p^2 = .21$ . Participants scored higher in the pre-session than in the post-session. There was no significant main effect of group  $F(1, 39) = .10$ ,  $p = .754$ ,  $\eta_p^2 < .01$  nor interaction between session and group  $F(1, 39) = .70$ ,  $p = .407$ ,  $\eta_p^2 = .02$  on DASS Anxiety.

ANOVA showed significant main effect of session on DASS Stress  $F(1, 39) = 22.14$ ,  $p < .001$ ,  $\eta_p^2 = .36$ . Participants scored higher in the pre-session than in the post-session. There was no significant main effect of group  $F(1, 39) = .39$ ,  $p = .538$ ,  $\eta_p^2 = .01$  nor interaction between session and group  $F(1, 39) = 1.19$ ,  $p = .282$ ,  $\eta_p^2 = .03$  on DASS Stress.

ANOVA showed significant main effect of session on MAAS total score  $F(1, 39) = 5.08$ ,  $p = .030$ ,  $\eta_p^2 = .12$ . Participants scored lower in the pre-session than in the post-session. There was no significant main effect of group  $F(1, 39) = .34$ ,  $p = .563$ ,  $\eta_p^2 = .01$  nor interaction between session and group  $F(1, 39) = 1.66$ ,  $p = .206$ ,  $\eta_p^2 = .04$  on MAAS total score.

ANOVA showed significant main effect of session on PHQ-9 total score  $F(1, 39) = 8.72$ ,  $p = .005$ ,  $\eta_p^2 = .18$ . Participants scored higher in the pre-session than in the post-session. There was no significant main effect of group  $F(1, 39) = .51$ ,  $p = .481$ ,  $\eta_p^2 = .01$  nor interaction between session and group  $F(1, 39) = 2.31$ ,  $p = .137$ ,  $\eta_p^2 = .06$  on PHQ-9 total score.

In summary, the story listening group had higher scores on PCS and subscales compared to the mindfulness group, but MAAS, DASS and PHQ-9 scores did not differ between groups. Furthermore, both mindfulness and story listening exercises appeared to affect questionnaire scores; MAAS scores were lower in the pre-session than in the post-session, meanwhile PCS, DASS and PHQ-9 scores were higher in the pre-session than in the post-session. As only exception, the story listening exercise, but not the mindfulness exercise, decreased the PCS Rumination scores. Overall, these findings suggest that both story listening and mindfulness exercises increased mindfulness disposition and decreased pain catastrophizing, depression, anxiety and stress feelings.

Pearson's correlation was used to establish the relationship between mood, fear of pain and perceived duration. To correlate these items, a mean verbal estimate was calculate for each stimulus modality averaging the verbal estimate of the same stimulus modality across stimulus durations. Table 8.2 shows correlations between mean verbal estimates of visual, tactile and painful stimuli and scores of each questionnaire/subscale. Pearson correlations did not show any relationship between verbal estimates and questionnaire scores. Therefore attitude to mindfulness, mood and pain catastrophizing were not related to perceived duration.

<i>Session</i>	<i>Pre</i>			<i>Post</i>		
	<i>Visual</i>	<i>Tactile</i>	<i>Painful</i>	<i>Visual</i>	<i>Tactile</i>	<i>Painful</i>
<i>PCS total</i>	<i>r = -.13</i> <i>p = .41</i>	<i>r = -.14</i> <i>p = .40</i>	<i>r = .09</i> <i>p = .59</i>	<i>r = -.12</i> <i>p = .46</i>	<i>r = -.20</i> <i>p = .21</i>	<i>r = -.08</i> <i>p = .62</i>
<i>PCS Rumination</i>	<i>r = -.18</i> <i>p = .25</i>	<i>r = -.16</i> <i>p = .31</i>	<i>r = .10</i> <i>p = .52</i>	<i>r = -.11</i> <i>p = .48</i>	<i>r = -.12</i> <i>p = .45</i>	<i>r = -.05</i> <i>p = .75</i>
<i>PCS Magnification</i>	<i>r = -.14</i> <i>p = .39</i>	<i>r = -.08</i> <i>p = .61</i>	<i>r = .07</i> <i>p = .65</i>	<i>r = -.07</i> <i>p = .68</i>	<i>r = -.18</i> <i>p = .27</i>	<i>r = .02</i> <i>p = .92</i>
<i>PCS Helplessness</i>	<i>r = -.05</i> <i>p = .74</i>	<i>r = -.09</i> <i>p = .59</i>	<i>r = .08</i> <i>p = .62</i>	<i>r = -.16</i> <i>p = .31</i>	<i>r = -.29</i> <i>p = .07</i>	<i>r = -.16</i> <i>p = .33</i>
<i>DASS Depression</i>	<i>r = .01</i> <i>p = .94</i>	<i>r = -.05</i> <i>p = .75</i>	<i>r = .05</i> <i>p = .77</i>	<i>r = .03</i> <i>p = .87</i>	<i>r = -.02</i> <i>p = .90</i>	<i>r = .03</i> <i>p = .87</i>
<i>DASS Anxiety</i>	<i>r = .14</i> <i>p = .38</i>	<i>r = .03</i> <i>p = .87</i>	<i>r = -.01</i> <i>p = .95</i>	<i>r = .14</i> <i>p = .39</i>	<i>r = -.06</i> <i>p = .71</i>	<i>r = .10</i> <i>p = .56</i>
<i>DASS Stress</i>	<i>r = .01</i> <i>p = .96</i>	<i>r = -.04</i> <i>p = .80</i>	<i>r = .16</i> <i>p = .31</i>	<i>r = -.03</i> <i>p = .86</i>	<i>r = -.03</i> <i>p = .84</i>	<i>r = .09</i> <i>p = .57</i>
<i>MAAS total</i>	<i>r = -.19</i> <i>p = .23</i>	<i>r = -.04</i> <i>p = .82</i>	<i>r = -.08</i> <i>p = .64</i>	<i>r = -.12</i> <i>p = .46</i>	<i>r = -.10</i> <i>p = .52</i>	<i>r = -.20</i> <i>p = .22</i>
<i>PHQ-9 total</i>	<i>r = -.01</i> <i>p = .98</i>	<i>r = -.13</i> <i>p = .43</i>	<i>r = .06</i> <i>p = .69</i>	<i>r = -.02</i> <i>p = .91</i>	<i>r = -.15</i> <i>p = .36</i>	<i>r = .04</i> <i>p = .81</i>

*Table 8.2. Correlation coefficients between questionnaire score and verbal estimates of visual, tactile and painful stimuli in the pre- and post-session. PCS = Pain*

*Catastrophizing Scale. DASS = Depression, Anxiety and Stress Scale. MAAS = Mindful Attention Awareness Scale. PHQ-9 = Patient Health Questionnaire-9.*

### *8.1.3 – Discussion*

Experiment 7 tested whether a 1-week mindfulness intervention could alter the verbal estimate, estimate accuracy and estimate variability of visual, tactile and painful stimuli. The results indicated that participants gave shorter and less accurate estimates for visual stimuli compared to tactile and painful stimuli, confirming previous studies that tactile stimuli are perceived more accurately than visual stimuli (Jones et al., 2009). Verbal estimates were also more variable for visual than tactile stimuli. However, verbal estimate, accuracy and variability were similar between tactile and painful stimuli, suggesting that pain did not lengthen time perception in comparison with a neutral somatosensory stimulus. The absence of a lengthening effect of pain could be due to the absence of intensity contrast between painful and neutral blocks of trials. Matthews et al. (2011) demonstrated that an evident contrast between two states or stimuli is required for temporal distortion occurrence. In Experiments 1 and 3, participants had intermixed blocks of neutral and painful stimuli. Here, there was one single pain block and one single neutral block. There was therefore a lack of contrast between blocks, which might have contributed to the absence of pain distortions to time.

Verbal estimates were expected to decrease for painful stimuli, and to increase for visual and tactile stimuli, after the mindfulness intervention but not after the audiobook listening exercise. Contrary to expectations, there was no effect of the MM exercise or the listening exercise on duration estimates. MM therefore did not affect the perceived duration in any stimulus modality. This contrasts previous findings showing that a single session (Kramer et al., 2013) and 5 weeks (Droit-Volet et al., 2015) of MM lengthened the perceived duration of visual stimuli in a temporal bisection task.

It is unclear why MM had no effect on the perceived duration of any stimuli. One possibility is that participants in this study were new at mindfulness. Droit-Volet et al. (2015) found the lengthening effect of MM with participants who were already MM practitioners, but not with MM novices. It is therefore possible that MM did not affect perceived duration of visual stimuli in this study because only MM novices were included. Another possibility is that more than 1-week's practice is necessary before MM induces temporal distortions. In clinical settings, mindfulness based interventions frequently consist of several weeks practice (Fjorback, Arendt, Ørnbøl, Fink, & Walach, 2011; Forman, Butryn, Hoffman, & Herbert, 2009; Kabat-Zinn, 1982) and effects on neural activity are only observed after 16 weeks (Moore et al., 2012). Indeed, Droit-Volet et al. (2015) only observed an effect of mindfulness on perceived duration after a 5-week training intervention but not after a single MM session. A longer training period may therefore reveal effects.

Although, Kramer et al. (2013) reported an effect of a single 10-minute mindfulness session on perceived duration it is important to note that this session consisted of a breathing exercise in addition to mindfulness. Droit-Volet et al. (2015) found that a single session of breathing exercise, but not a single session of MM, lengthened the performance in the temporal bisection task. It is therefore possible that in Kramer et al. (2013) the breathing component of the session rather than the mindfulness component had an effect on time perception. Therefore, the use of novice MM practitioners coupled with the absence of an explicit breathing component could have resulted in the absence of an effect of MM in the current study. Indeed, given that disentangling the effect of the mindfulness component of an intervention from other components is a common issue when testing MM effectiveness (see Malinowski, 2017 for discussion), future research should focus on establishing whether controlled breathing can affect the perceived duration of pain.

Another possible explanation for the null effect is that MM might not have reduced physiological arousal enough to modulate time perception. Whilst MM was consistently found to increase PSNS activity (Kubota et al., 2001; Wu & Lo, 2008), MM



effects on SNS are less consistent, with studies showing that MM both decreases (Lush et al., 2009) and increases (Ditto, Eclache, & Goldman, 2006) SNS activity. Experiment 3 showed that time distortions are mediated by SNS changes rather than PSNS changes. It is therefore possible that MM did not decrease SNS activity enough to lead to temporal distortions.

The questionnaires indicated that mindfulness disposition increased and pain catastrophizing, anxiety, stress and depression decreased after the week of MM and story listening exercise. This suggests that MM and the control story listening task both had beneficial effects for the participants. This could be explained by (i) questionnaires re-administration, which could have led to a training effect that induced different self-reports, or (ii) both MM and story listening exercise improving participants' wellbeing. The second explanation is preferred because the used questionnaires have excellent test-retest reliability; for example, PHQ-9 scores are nearly identical after 48 hours (Kroenke et al., 2001). The second explanation is also supported by those studies that found that both MM *and* story listening decrease anxiety, stress and depression (Hofmann, Sawyer, Witt, & Oh, 2010; Warnecke, Quinn, Ogden, Towle, & Nelson, 2011). Although the questionnaire scores changed between pre- and post-sessions, the correlational analysis showed that questionnaire scores did not correlate with verbal estimates, suggesting that time perception is not related to the self-reported mindfulness disposition and mood traits.

Although MM did not affect the perceived duration of pain in healthy people, it is possible that MM could affect time perception in chronic pain patients. There are differences in the way in which non-clinical and chronic pain populations experience pain. Non-clinical groups primarily experience the first phase of pain (the acute, short-lasting sensation); meanwhile clinical groups mainly deals with the second phase of pain (the dull, long-lasting sensation) (see section 3.1, page 55). This leads to chronic higher physiological arousal in chronic sufferers compared to healthy people (Somov, 2000), which is thought to cause the patients' feeling that daily time "drags". We may therefore expect MM to be particularly beneficial to chronic pain

populations because MM has been found to increase emotional self-regulation, inhibiting the negative effects of the second phase of pain, such as reducing the abnormal arousal of chronic sufferers (Hölzel et al., 2011). In fact, mindfulness-based interventions are commonly used in chronic pain management for reducing physiological arousal and pain intensity experience (Gardner-Nix et al., 2008; Zeidan et al., 2010).

Chronic pain is also frequently in comorbidity with mental health problems, such as anxiety, depression and stress reactivity, which are thought to cause the temporal distortions of chronic pain patients (Isler et al., 1987; Somov, 2000). Mindfulness based intervention are frequently used as treatment for these mental health problems; for example, MBCT has the same benefits than drug treatments in depression (Kuyken et al., 2015). Furthermore, mindfulness-based programmes improve quality of life and wellbeing of clinical populations by inducing positive neural changes in brain areas that are involved in stress (Davidson et al., 2003) and by reducing self-reported stress and depression (Davidson et al., 2003; Kuyken et al., 2015). It is therefore possible that MM could reduce perceived duration by reducing depression, stress and anxiety symptoms.

Finally, chronic sufferers have an attitude toward pain which differs from healthy people; chronic pain patients have higher pain catastrophizing, which is defined as an exaggerated negative orientation and fear toward pain experience (Sullivan et al., 1995) and is a significant predictor of suffering and pain severity (Lackner & Quigley, 2005; Picavet, Vlaeyen, & Schouten, 2002). Mindfulness disposition was found to predict pain catastrophizing with higher mindfulness disposition associated with lower pain catastrophizing (Schütze, Rees, Preece, & Schütze, 2010). Mindfulness practice could potentially reduce pain catastrophizing in chronic patients, resulting in reduced pain experience and shorter perceived duration of pain.

Because MM is able to reduce physiological arousal, mental health symptoms and pain catastrophizing, which are abnormal in chronic pain sufferers, MM may be

beneficial in attenuating the perceived duration of pain in chronic patients even if Experiment 7 showed a null effect in healthy people. Experiment 8 therefore pilot-tested whether a 1-week mindfulness intervention could affect the perceived duration of visual stimuli, vibrations and electro-cutaneous shocks in chronic pain patients. The experimental design was a replication of Experiment 7: first chronic pain patients selected the subjective pain intensity of the electrical shock during an initial task and completed three verbal estimation tasks, one for each stimulus modality. Then participants were asked to perform a body-scan exercise once a day for eight days, and finally participants were re-tested on the three verbal estimation tasks. The intervention was expected to increase verbal estimates of visual and tactile stimuli, replicating previous findings (Droit-Volet et al., 2015; Kramer et al., 2013), and to decrease physiological arousal and mental health symptoms, leading to reduced verbal estimates and estimate variability of painful stimuli.

## **8.2 – Experiment 8**

### *8.2.1 – Method*

#### 8.2.1.1 – Participants

Ten participants (4 females and 6 males; mean age = 45.80, SD = 20.33) were recruited. Participants were required not to be pregnant and not to have a history of schizophrenia, diabetes nor skin problems on their left arm (e.g., eczema). Individuals who wore a pacemaker or were regular mindfulness meditators (i.e., meditate more than once a month) were also excluded. Participants were reimbursed £15 in vouchers for taking part. The study was approved by the Liverpool John Moores University and NHS ethics committees and participants were recruited through a primary care centre. Informed consent was obtained from all participants.

#### 8.2.1.2 – Experimental design

The experimental structure was identical to the one used in Experiment 7, except that all participants completed the mindfulness intervention and none were assigned to the story listening exercise. On the first day, participants attended a pre-testing

session which involved 1) completing the questionnaires; 2) the intensity rating task; 3) the verbal estimation tasks and 4) the mindfulness meditation exercise. On days two to seven, participants were asked to complete the mindfulness meditation exercise in their own time at home and complete a diary recording their experience. On day eight, participants attended a post-testing session, which involved performing the mindfulness meditation exercise, followed by the questionnaires and verbal estimation tasks (see Figure 8.5 for an experimental structure scheme).

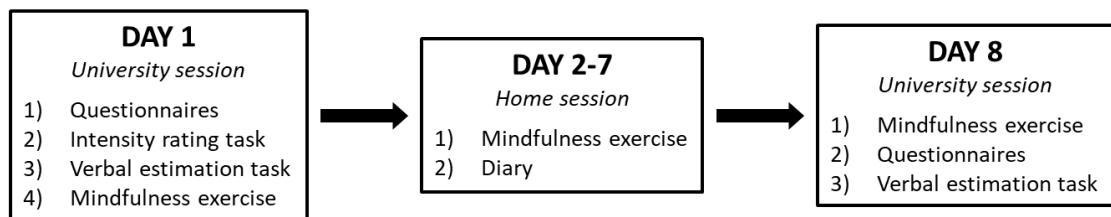


Figure 8.5. Experiment 8 structure.

### 8.2.1.3 – Apparatus and material

Experiment 8 used the same questionnaires (PCS, PHQ-9, DASS-21 and MAAS), visual stimulus, tactile vibration, electro-cutaneous stimulation, mindfulness exercise and diary of Experiment 7. Intensity rating and verbal estimation tasks used the same procedure of the one used in Experiment 7.

### 8.2.2 – Results

Out of ten participants tested, data from two participants were excluded from the analysis because they did not attend the post-session. Therefore, results are reported for the remaining eight participants. Participants' diaries reported that there was 93.75% adherence and no participants missed more than two home sessions. To establish the effect of the intervention on time perception three measures of timing were computed: mean estimate, estimate accuracy and estimate variability. Greenhouse-Geisserr correction was applied to ANOVAs when the Sphericity assumption was violates and post-hoc were Bonferroni corrected.

### *Verbal estimates*

Figure 8.6 shows the verbal estimates of visual, tactile and painful stimuli in the pre- and post-session separately. A repeated measures ANOVA with session (pre and post), stimulus modality (visual, tactile and painful) and stimulus duration (242ms, 455ms, 767ms, 1058ms and 1296ms) as within subject factors was conducted. This showed significant main effects of stimulus duration  $F(1.20, 8.39) = 115.80, p < .001, \eta_p^2 = .94$  on verbal estimates. ANOVA shows no significant main effect of stimulus modality  $F(2, 14) = 2.39, p = .128, \eta_p^2 = .25$  nor session  $F(1, 7) = 1.51, p = .259, \eta_p^2 = .18$  on verbal estimates. There was however a significant interaction between stimulus duration and session ( $F(4, 28) = 6.28, p = .001, \eta_p^2 = .47$ ) but no other significant interaction was found (all  $ps > .05$ ).

To further investigate the interaction effect between stimulus duration and session, a paired-sample t-test between the pre- and post-session was conducted on each stimulus duration separately (242ms, 455ms, 767ms, 1058ms and 1296ms). Verbal estimates were significantly longer in the post-session compared to the pre-session for the 1296ms duration ( $t(7) = 2.46, p = .043$ ), meanwhile the estimates for the 242, 455, 767 and 1058 stimuli did not differ (all  $ps > .05$ ).

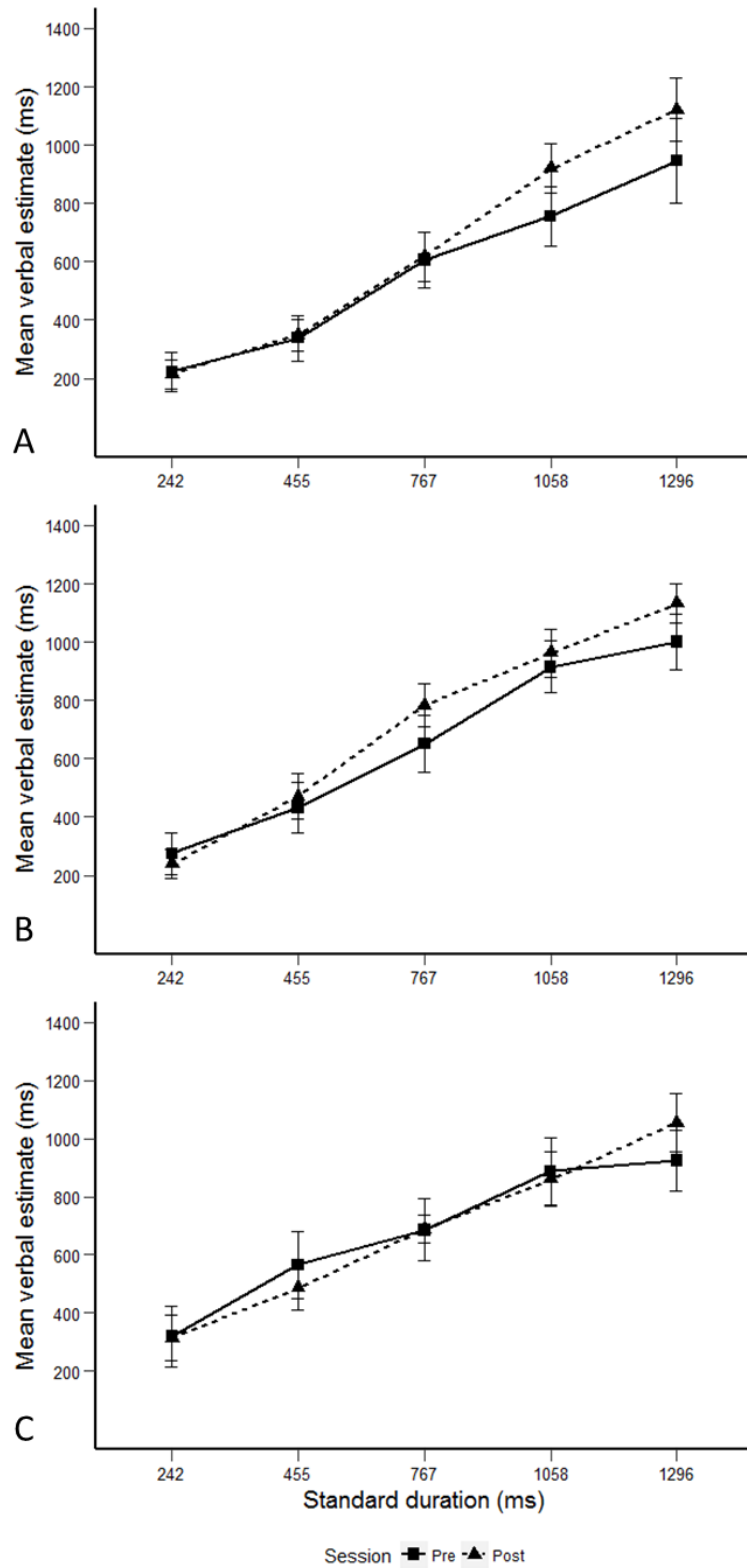


Figure 8.6. Means (and standard errors) of the verbal estimates (ms) plotted against the stimulus duration and divided by session (pre and post). The three plots show the verbal estimates in the visual modality (panel A), tactile modality (panel B) and painful modality (panel C).

### *Estimate accuracy*

Estimate accuracy was calculated for each stimulus duration of each modality in each session using the same formula used in Experiment 7: verbal estimate/stimulus duration.

Figure 8.7 shows the estimate accuracy of visual, tactile and painful stimuli. A repeated measures ANOVA with session (pre and post), stimulus modality (visual, tactile and painful) and stimulus duration (242ms, 455ms, 767ms, 1058ms and 1296ms) as within subject factors was conducted. This showed no significant main effect of duration ( $F(1.03, 7.18) = 1.42, p = .273, \eta_p^2 = .17$ ) and session ( $F(1, 7) = .31, p = .596, \eta_p^2 = .04$ ) on estimate accuracy. There was a significant main effects of stimulus modality  $F(2, 14) = 4.60, p = .029, \eta_p^2 = .40$ . However, post-hoc tests showed that the difference between estimate accuracy in the visual and pain modality failed to reach significance ( $p = .080$ ), meanwhile estimate accuracy in the tactile modality was not significantly different from the visual ( $p = .242$ ) and painful ( $p > .99$ ) modalities.

There was a significant interactions between stimulus duration and session ( $F(2.09, 14.61) = 5.41, p = .017, \eta_p^2 = .44$ ), but no other significant interaction was found (all  $ps > .05$ ). To further investigate the interaction effect between stimulus duration and session, a paired-sample t-test between the first and second session was conducted per each stimulus duration (242ms, 455ms, 767ms, 1058ms and 1296ms). Estimate accuracy was significantly higher in the post-session than in the pre-session for the 1296ms duration ( $t(7) = 2.49, p = .041$ ), meanwhile the estimates for the 242, 455, 767 and 1058 stimuli did not differ between sessions (all  $ps > .05$ ).

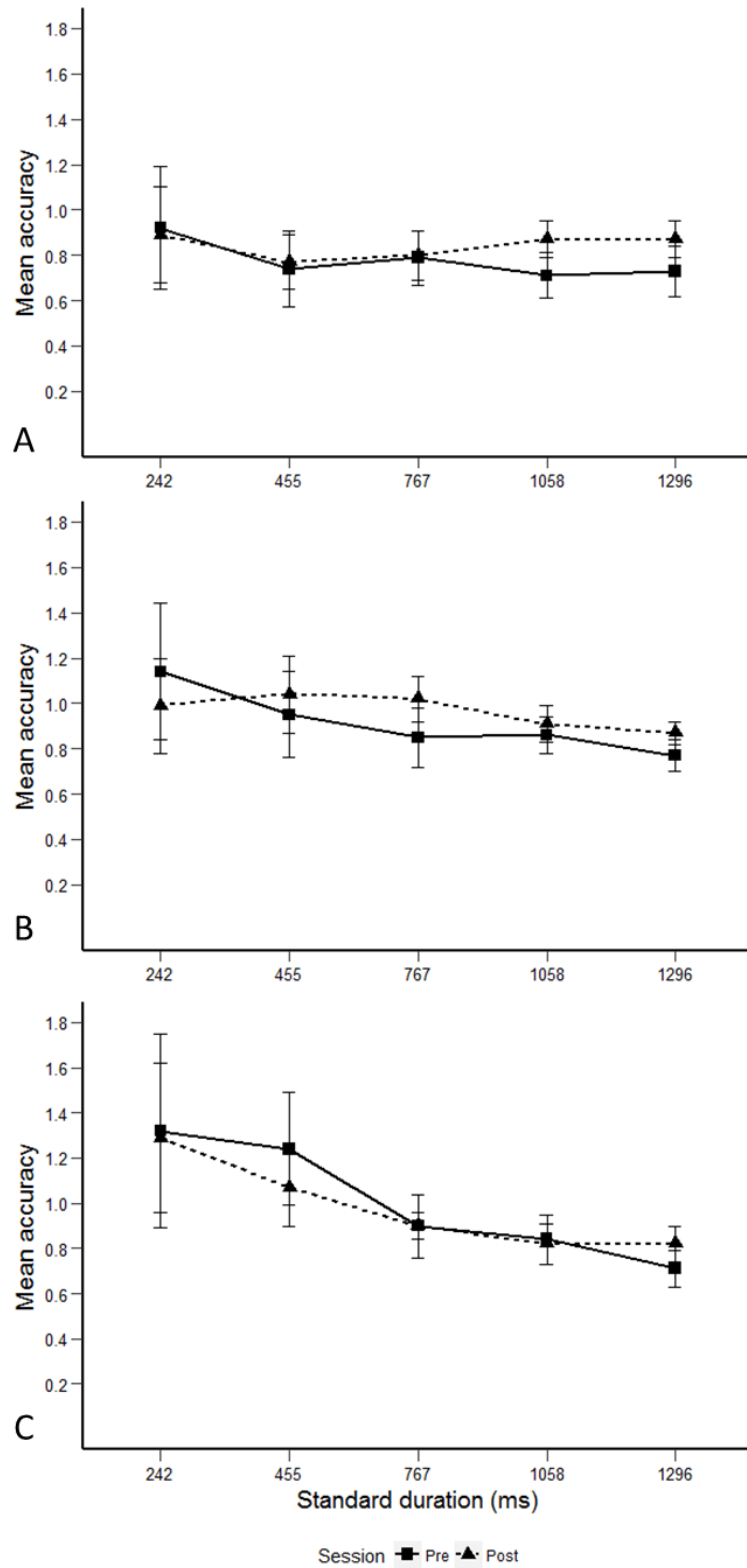


Figure 8.7. Means (and standard errors) of the estimate accuracy plotted against the stimulus duration and divided by session (pre and post). The three plots show the verbal estimates in the visual modality (panel A), tactile modality (panel B) and painful modality (panel C).



### *Estimate variability*

Estimate variability was calculated for each stimulus duration of each modality in each session using the same formula used in Experiment 7: standard deviation verbal estimate/mean verbal estimate.

Figure 8.8 shows the estimate variability of visual, tactile and painful stimuli. A repeated measures ANOVA with session (pre and post), stimulus modality (visual, tactile and painful) and stimulus duration (242ms, 455ms, 767ms, 1058ms and 1296ms) as within subject factors was conducted. This showed significant main effects of stimulus duration  $F(4, 28) = 19.36, p < .001, \eta_p^2 = .73$  and session  $F(1, 7) = 14.11, p = .007, \eta_p^2 = .67$  on estimate variability. Post-hoc tests indicated that estimate variability was higher for the shortest duration (242ms) than for the 1058ms and 1296ms durations ( $ps < .05$ ). Estimate variability was also significantly higher in the pre-session compared to the post-session. There was no significant main effect of stimulus modality on estimate variability  $F(2, 14) = 1.44, p = .269, \eta_p^2 = .17$ . There were also no significant interactions (all  $ps > .05$ ).

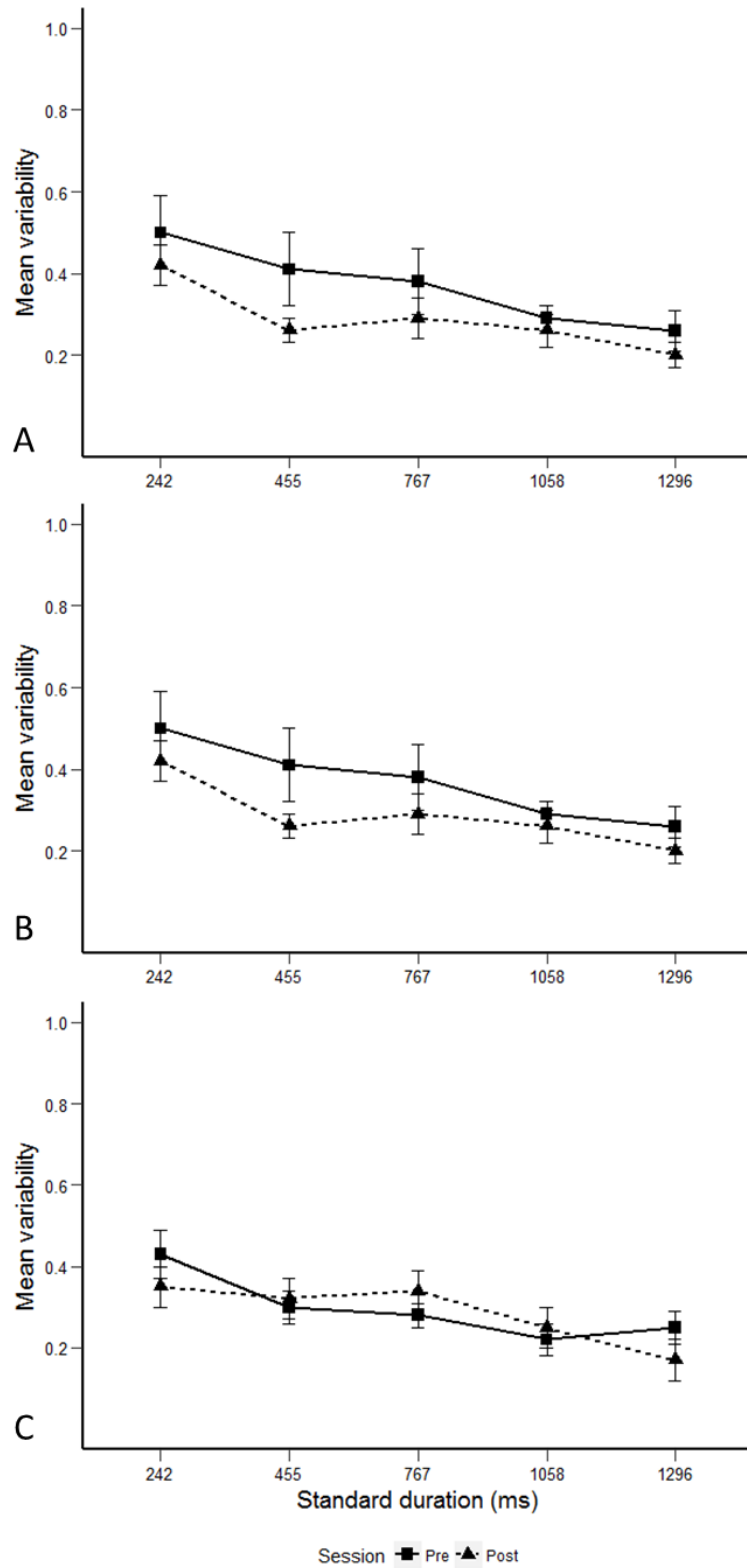


Figure 8.8. Means (and standard errors) of the estimate variability plotted against the stimulus duration and divided by session (pre and post). The three plots show the verbal estimates in the visual modality (panel A), tactile modality (panel B) and painful modality (panel C).

Questionnaires

<i>Session</i>	<i>Pre</i>	<i>Post</i>
<i>PCS total</i>	16.25 (9.21)	13.88 (11.21)
<i>PCS Rumination</i>	5.88 (3.18)	4.63 (3.85)
<i>PCS Magnification</i>	3.50 (2.67)	3.75 (3.99)
<i>PCS Helplessness</i>	6.75 (4.92)	5.75 (5.01)
<i>DASS Depression</i>	8.25 (10.00)	5.00 (3.70)
<i>DASS Anxiety</i>	6.25 (7.74)	4.75 (5.85)
<i>DASS Stress</i>	13.25 (7.70)	11.75 (2.49)
<i>MAAS total</i>	4.11 (0.44)	3.93 (0.68)
<i>PHQ-9 total</i>	6.50 (5.88)	6.13 (4.49)

Table 8.3. Means (and standard deviations) of questionnaire scores divided by session (pre and post). PCS = Pain Catastrophizing Scale. DASS = Depression, Anxiety and Stress Scale. MAAS = Mindful Attention Awareness Scale. PHQ- 9 = Patient Health Questionnaire-9.

Table 8.3 shows questionnaire scores divided by session. A paired-sample t-test was conducted per each questionnaire and subscale. None of the questionnaire scores changed significantly from the pre- to the post-session (all *ps* > .05). Due to the reduced size of the sample, correlational analysis of the relationship between mood, fear of pain and perceived duration was not conducted.

8.2.3 – Discussion

Experiment 8 pilot tested whether a 1-week mindfulness intervention could affect the perceived duration of visual, tactile and painful stimuli in chronic pain patients.

This was also the first study examining how chronic sufferers perceive the duration of stimuli other than visual (Droit-Volet et al., 2015; Kramer et al., 2013). The results indicated that chronic pain patients gave similar verbal estimates across stimulus modality. This contrasts with the results obtained from healthy people in Experiment 7, who gave shorter estimates for visual stimuli than for vibrations and electrocutaneous stimulations, as also shown previous studies (E. A. Williams, Stewart, & Jones, 2009). However, this finding should be taken with caution due to the small sample size and lack of statistical comparison between the two groups.

The MM intervention was expected to increase the perceived duration of the visual and vibrotactile stimuli and to decrease the verbal estimates of painful stimuli. Furthermore, mindfulness was expected to reduce the variability of duration estimates in all stimulus modalities. Results partially confirmed expectations; mindfulness lengthened the perceived duration of the visual and tactile stimuli, although this occurred only for the longest duration (1296ms). MM also reduced the variability of estimates for all modalities. Contrary to expectations, however, MM also increased the perceived duration of the pain stimuli, but again, only for the longest stimulus duration.

The lengthening effect of mindfulness on the perceived duration of visual and tactile stimuli found in this experiment confirmed similar studies using healthy participants (Droit-Volet et al., 2015; Kramer et al., 2013). The effect of the MM intervention on estimate variability is also in line with the findings of Droit-Volet et al. (2015), who found higher temporal sensitivity, indexed by Weber ratio, in a temporal bisection task after a 5-week mindfulness intervention. These effects may occur because MM enhances attentive processes, such as self-regulating attention and inhibition of cognitive automatic responses (Bishop et al., 2004; Mirams et al., 2013). These processes are related to time perception and responses variability, with greater ability to direct attentive resources to the timing task associated with longer estimates and reduced temporal variability (Brown, 2006). It is therefore possible that the mindfulness intervention enhanced attentive processes leading to greater

attention dedicated to the temporal task and resulting in longer perceived duration and reduced temporal variability in visual and tactile stimuli.

MM was expected to decrease physiological arousal and mental health symptoms (e.g., depression), leading to shorter perceived duration for the pain stimuli. However, the results indicated that the longest painful stimulus was perceived longer after the intervention than before. It is possible that the attentive resources enhanced by MM could have led to this lengthening effect, as for the visual and tactile stimuli. It is also possible that the intervention did not shorten verbal estimates because the body-scan exercise did not reduce physiological activity and mental health symptoms. This possibility is supported by studies showing that MM could increase SNS activity (Ditto et al., 2006) and by the questionnaire scores in this experiment, which showed no effect of MM on mental health symptoms. In fact, questionnaire scores in the pre- and post-sessions were alike, suggesting that MM did not decrease anxiety, depression and stress.

The null effect of MM on questionnaire scores contrasts with findings in Experiment 7, which showed that MM decreased questionnaire scores in healthy participants leading to enhanced mindfulness disposition and reduced pain catastrophizing, depression, anxiety and stress. Questionnaire results in Experiment 8 are particularly unexpected because MM was consistently found to improve patients' wellbeing reducing their depression, anxiety and stress symptoms (Davidson et al., 2003; Kuyken et al., 2015).

An evident limitation of Experiment 8 is the small sample size, which reduced the statistical power of the analyses (see Button et al., 2013 for discussion). It is therefore possible that Experiment 8 did not find an effect of MM on questionnaire scores (contrary to Experiment 7) because of the sample size. Additionally, the sample size could explain why the mindfulness intervention lengthened only the verbal estimates of the longest duration. However, small sample sizes can also lead to increased

effects (Button et al., 2013). The promising result that the mindfulness intervention reduced estimate variability could therefore disappear with a greater sample.

In conclusion, this pilot study suggest that MM may affect the way in which chronic pain patients experience the perceived duration visual, tactile and painful stimuli, although not in the expected direction. 1-week body scan exercise lengthened the duration of visual, vibrotactile and electro-cutaneous stimuli and reduced their estimate variability. With results of Experiment 7, this suggests that mindfulness meditation might not be an effective modality to shorten perceived duration and to mitigate the lengthening effect of pain on time perception. However, given the limited chronic pain sample, further research is required to test the effect of mindfulness in clinical populations.

## Chapter 9

### General discussion

#### 9.1 – Review of findings

This thesis aimed to expand our understanding of the effect of pain on human time perception. In particular, this thesis (i) clarifies the conditions under which pain distorts time perception, (ii) directly tests the mediating role of arousal in the lengthening effect of pain on perceived duration, and (iii) tests an intervention for reducing those temporal distortions. This was achieved in a series of studies by examining (i) whether changes in neutral and emotional stimulus intensities induce comparable time distortions; (ii) whether changes in task-relevant and task-irrelevant pain induce comparable time distortions; (iii) the relationship between physiological arousal and perceived duration; (iv) whether pain affects perceived durations when they are recalled over a period of delay; and (v) whether a mindfulness intervention could modulate the perceived duration of visual, somatosensory and painful stimuli.

##### *9.1.1 – The effect of changes in emotional and neutral stimulus intensity on time perception*

Chapter 5 (Experiment 1) tested whether changes in the intensity of a neutral stimulus distorted time in a comparable way to changes in the intensity of an emotional stimulus. This was achieved by examining the perceived duration of perceptible, low and high vibrotactile stimulation and the perceived duration of no pain, low pain and high pain electro-cutaneous stimulation. The results demonstrated that increases in electro-cutaneous intensity were associated with longer, more accurate and less variable verbal estimates, which were different between each pain intensity. Increases in vibrotactile intensity were also associated with longer, more accurate and less variable verbal estimates; however, there was no difference between low and high vibration intensity. Experiment 1 therefore indicated that changing the intensity of a neutral stimulus distorts time in a comparable way, albeit

less effectively, to changing the intensity of an emotional stimulus. This finding perhaps suggests that arousal increases originating from a neutral source have the capacity to distort time in similar ways to that observed with emotional stimuli. This confirms previous studies that found temporal distortions induced by neutral stimuli, such as click-trains (Penton-Voak et al., 1996), visual flicker (Ortega & López, 2008), filled and unfilled durations (Wearden et al., 2007) and modality differences in timing (Jones et al., 2009). Although these findings provide support for the suggestion that changes in arousal can affect the perceived duration of neutral stimuli, it should be noted that there were methodological limitations of the physical properties of the vibrotactile stimulus which caution this interpretation (see section 5.1.3, page 98).

#### *9.1.2 – The effect of task-relevant and task-irrelevant pain on time perception*

Chapters 5 (Experiments 1 and 2) tested whether changes in the intensity of task-irrelevant pain could distort time in a comparable way to changes in the intensity of task-relevant pain. This was achieved by contrasting the temporal distortions observed in two scenarios 1) when pain was the to-be-timed-stimulus and therefore task-relevant and 2) when the pain was not the to-be-timed-stimulus and therefore task-irrelevant. The results demonstrated that, whilst increases in task-relevant pain intensity were associated with longer, more accurate and less variable estimates of duration (Experiment 1), increased task-irrelevant pain intensity was associated with shorter and less accurate verbal estimates of the concurrently experienced visual stimulus (Experiment 2). This contrasts with the prediction of the arousal hypothesis and the theories of timing (SET, SBF and Craig's model of awareness), which suggest that *any* source of arousal should lead to longer perceived durations. The findings of Experiments 1 and 2 therefore indicated that this suggestion is likely to be too simplistic, instead suggesting that lengthening effects of arousal are limited to circumstances in which arousal is relevant to the timing task. This emphasises the need for future work to understand how attention and arousal interact to produce distortions to time.



### *9.1.3 – The relationship between physiological arousal and perceived duration*

Chapter 6 (Experiments 3 and 4) introduced physiological recording equipment to directly test the mediating role of physiological arousal in the lengthening effect of pain on perceived duration. This was achieved by replicating Experiments 1 and 2 and examining the association between changes in perceived duration and changes in physiological activity caused by task-relevant and task-irrelevant pain. The results demonstrated that both task-relevant and task-irrelevant pain increased sympathetic arousal but only task-relevant pain increased verbal estimates. Critically, changes in sympathetic activity were associated with changes in perceived duration only when pain was task-relevant, meanwhile sympathetic activity and perceived duration were unrelated when pain was task-irrelevant. Sympathetic arousal therefore mediated the effect of pain on time perception only when arousal was induced by a task-relevant source. These findings supported the suggestions of Gil and Droit-Volet (2012) that the arousal hypothesis may be too simplistic and that the effect of arousal on time perception is not as automatic as previously described. I therefore proposed a survival based model, in which arousal leads to time distortions exclusively when these distortions promote the best survival chances, that is when pain is relevant (see section 6.3, page 136).

### *9.1.4 – The effect of pain on memory for duration*

Chapter 7 (Experiments 5 and 6) tested the effect of low and high pain on memory for duration. This was achieved by using a temporal generalisation task where participants encoded the duration of a tone whilst experiencing neutral or painful thermal stimulation. Participants then recalled the duration in the absence of thermal stimulation either immediately after learning or after a 15-minute delay. The findings of experiments 5 and 6 were not consistent; 15-minute delay decreased temporal performance of both neutral and pain related durations in Experiment 7, but had no effect on temporal performance of neither neutral nor pain related durations in Experiment 8. In both experiments, however, delay affected neutral and pain related

durations in a comparable way, suggesting that pain does not have any unique effect on the memorization of duration. These findings indicated that unlike emotion (Cocenas-Silva et al., 2012), pain does not enhance the memorization of duration information. The findings also indicated that pain did not disrupt the cognitive functions required for the correct memorization of duration, such as attention and executive functions. Pain therefore neither improved nor disrupted the duration encoding and retrieval from memory. It has been argued that the use of a task-irrelevant source of pain might have limited the effect that pain had on the memorization process (see section 7.3, page 159).

#### *9.1.5 – Reducing the lengthening effect of pain through a mindfulness intervention*

Chapter 8 (Experiments 7 and 8) tested whether a mindfulness intervention could affect the perceived duration of painful, vibrotactile and visual stimuli in healthy people and chronic pain sufferers. This was achieved by asking participants to complete a verbal estimation task before and after practising a body-scan exercise for 20 minutes per day for 8 days. Results showed that mindfulness did not affect the perceived duration of any stimulus in healthy participants (Experiment 7). In contrast, MM had a lengthening effect on the perceived duration of visual, vibrotactile and painful stimuli in chronic pain patients (Experiment 8), although the effect was limited to the longest duration (1296ms) and are difficult to interpret due to the small sample size. MM therefore failed to achieve the intervention's purpose of decreasing the perceived duration of pain. The results of Experiment 7 also contrast with previous findings, which showed that mindfulness lengthened the perceived duration of visual stimuli in healthy people (Droit-Volet et al., 2015; Kramer et al., 2013). It is however possible that the limited effect of mindfulness on time perception in this study might have been due to participants being MM novices and/or the body-scan exercise being ineffective in reducing physiological arousal enough to affect time perception (see section 8.1.3, page 183).

## **9.2 – Methodological and theoretical issues**

### *9.2.1 – Reliability of the intensity rating task*

All experiments described in the present thesis included the intensity rating task in their procedure. This task consisted of participants selecting the required pain intensity based on their subjective experience; each participant had to select the somatosensory stimulation that they think corresponded to a target pain intensity in the Numeric Rating Scale (e.g., 6, which corresponds to high pain). The physical intensity of the electro-cutaneous and thermal stimulations were therefore different across participants and studies, which led to limitations in the interpretation of the findings when comparing different studies. For example, high pain intensity was found to induce temporal underestimation in Experiment 2, but no temporal distortion in Experiment 4 and it was not possible to exclude that the use of different temperatures between the two studies (i.e., 44.47°C in Experiment 2 and 43.60°C in Experiment 4) which may have contributed to the inconsistency between the experiments' findings.

An obvious issue with the intensity rating task, is that some participants may have chosen to select temperatures which were not painful. For example, Rolke et al. (2006) found that few participants in their sample reported 37°C and 29°C, which are within the range of no-pain temperature, as being hot pain and cold pain respectively. Selection of temperatures not typically deemed painful was also evident in Chapters 5 and 6 (Experiment 2 and 4), where participants who selected a temperature below 40°C as high pain were removed from analysis. However, it is not possible to exclude the possibility that other subjects selected an intensity that, although painful, was lower than the one required by the experiment. This might have reduced the effect of pain on physiological activity and perceived duration contributing to some inconsistencies in the findings across chapters.

Despite these known limitations, asking participants to select the stimulus intensity based on their perceived pain is common practice in the pain and time

perception literatures (Fayolle et al., 2015; Moore et al., 2013). It is also preferred to the alternative of using the same physical intensity across participants because the same physical stimulation frequently induces different subjective experience across participants. For example, a thermode at a fixed temperature may be perceived as painful by some participants but non-painful by others (Strulov et al., 2007). This variability across participants is particularly common when using electro-cutaneous stimulations (Rollman & Harris, 1987), resulting in the same physical stimulation inducing different physiological, emotional and cognitive responses across participants, which ultimately affects the ability of the IV (pain) to impact on the DV. In the present thesis, the presentation of a fixed electro-cutaneous (or thermal) stimulation was likely to induce physiological responses and verbal estimates with greater variability across participants than using the intensity rating task, potentially resulting in inconsistent experimental findings. Furthermore, using a fixed intensity might expose people to pain which they experience as unbearable, leading to ethical issues. For these reasons, the intensity rating task was preferred in the present studies rather than using a fixed stimulation. The Numeric Rating Scale was also used due to its higher responsiveness compared to other scales (e.g., Visual Analogue Scale; Ferreira-Valente, Pais-Ribeiro, & Jensen, 2011).

### *9.2.2 – The mechanisms underlying the relationship between arousal and perceived duration*

The mechanisms underlying human time perception are still unclear. Chapter 1 described three popular models of time perception, each of which proposed a different series of processes that would lead to the conscious time experience. Arousal has different roles within these models: arousal is thought to increase the pacemaker speed in SET, the DA levels and cortical activity in SBF and the generation of global emotional moments in Craig's model of awareness (see sections 2.3.1, page 47 and 3.3.1, page 67). Chapter 6 reported the first direct evidence supporting the suggestions of these models that arousal can directly mediate the perceived duration of a stimulus. Whilst this finding supports the basic tenants of the arousal hypothesis,

it remains unclear *how* arousal acts on duration perception. It thus remains unclear whether arousal affects timing by altering the speed of an internal clock, activity in the AIC or neuronal oscillations in striatum.

Chapter 6 did however highlight that arousal has to be task-relevant to distort perceived duration. This contrasts with timing theories' expectation that any arousal modulation should lead to temporal distortions. However, this thesis did not address *why* task-relevancy is determinant for the arousal effect. It has been argued that pain might have distracted participants from the encoding of the neutral stimulus interfering with the arousal effect. In fact, reduced attention is thought to decrease the number of accumulated pulses in SET and the DA level in SBF, resulting in reduced perceived duration, which could cancel the lengthening effect of arousal. However, this argument was not conclusively supported by empirical evidence in this thesis and future studies should be conducted to investigate the interaction effect of arousal and attention on perceived duration.

### *9.2.3 – The contrast effect*

One theory posited in Chapter 6 was that task-irrelevant pain did not lengthen verbal estimates because there was a lack of trial-by-trial contrast between the pain and non-pain conditions; when task-relevant pain was presented, the contrast between the stimulus and the background changed from trial to trial. When task-irrelevant pain was presented, however, the arousing stimulus was presented as a constant background and there was no trial-to-trial contrast, which might have contributed to the absence of a temporal distortion (see section 6.3, page 136). The absence of contrast might also explain why pain did not affect memory for duration in Chapter 7. In Chapter 7, pain was presented in the background whilst participants encoded the neutral tone in the learning phase of the temporal generalisation task. There was therefore no trial-to-trial contrast, which might have led to a lack of temporal distortion. Finally, the absence of contrast might explain why participants did not estimate painful stimuli as lasting longer than neutral somatosensory stimuli

in Chapter 8. In Chapter 5 and 6, short blocks of neutral, low and high intensity pain were inter-mixed. This mean that there was frequent contrast between blocks. In Chapter 8 however, only a single block of pain and a single block of visual and vibrotactile stimuli were delivered. As a result, these blocks were longer than in Chapters 5 and 6. It is therefore possible that the absence of frequent inter-block changes contributed towards the null effects observed.

It should be noted, however, that the absence of contrast could only lead to no temporal distortion and cannot explain why Chapter 5 found that task-irrelevant pain *shortened* the perceived duration of a neutral stimulus. Furthermore, background pain has been constantly found to disrupt memory and attentional processes required in concurrent tasks (Moore et al., 2013). Therefore, although the absence of trial-to-trial contrast might have prevented pain affecting the encoding of the neutral duration (Chapter 7), it is unlikely that it prevented task-irrelevant pain to disrupt the cognitive functions necessary for the correct memorization of the neutral stimulus.

### **9.3 – Directions for future research**

#### *9.3.1 – Why task-relevancy is critical*

This thesis showed that pain lengthened time perception only when pain was task-relevant (Chapters 5 and 6). Multiple suggestions were made to address why the lengthening effect was specific to this circumstance. For example, task-irrelevant pain could have induced cognitive responses and antinociceptive mechanisms, which led to habituation to pain and reduced AIC activity, resulting in the absence of temporal distortions. An additional argument was that experiments testing the effect of task-irrelevant pain on the perceived duration of a neutral visual stimulus used a cross-modal task, which has led to inconsistent findings in previous studies (Ogden et al., 2015). It was also argued that when pain was task-relevant subjects', attention was fully directed toward pain allowing arousal to lengthen perceived duration, but when pain was task-irrelevant, pain reduced people's attention cancelling the lengthening effect of arousal. Finally, it was suggested that there could be some perceptual and

cognitive advantage for survival in lengthening the perceived duration of pain, but this advantage disappears when pain is task-irrelevant (see section 6.3, page 136 for full discussion).

Although these arguments are plausible, this thesis did not directly test these potential explanations and therefore did not address *why* the lengthening effect of pain was specific to task-relevant situations. Future research should therefore examine these suggestions testing whether task-relevancy is determinant for the lengthening effect of pain for survival purposes or due to interference of cognitive responses, antinociceptive mechanisms, cross-modal tasks or attentional disruptions.

### *9.3.2 – The effect of task-relevant pain on memory for duration*

Chapter 7 showed that pain did not affect the memory for duration: pain was not found to improve or disrupt the recall of the duration of a concurrent neutral stimulus after a period of delay. One possible explanation for this finding is that the retention period used (15 minutes) was too short and that a longer delay would elicit a pain effect not currently evident (however see section 7.3, page 159). Another possibility is that pain was task-irrelevant, which was found to alter the effect of pain on perceived duration in Experiments 2 and 4. This possibility could be tested in future studies by replicating Experiments 5 and 6 but using task-relevant pain. For example, in a temporal generalisation task participants could be asked to memorize the duration of neutral and painful electro-cutaneous stimulations and recall it after 15-minute delay. If the painful stimulation were recalled shorter or longer than the neutral stimulation after 15 minutes, this would indicate that pain disrupts or enhances the memory processes. Furthermore, this would be an additional confirmation of the importance of task-relevancy for the pain effects on temporal distortions.

### *9.3.3 – New interventions to reduce the lengthening effect of pain*

Chapter 8 proposed a 1-week mindfulness intervention to reduce the perceived duration of pain. However, Experiments 7 and 8 showed that the intervention failed to affect time perception in healthy participants; meanwhile it had a lengthening effect in chronic pain patients limited to the longest duration. These findings have been attributed to the length of the intervention (8 days), which might have been too short to lead to significant effects, and to mindfulness being effective only with who already has mindfulness experience (see section 8.2.3, page 195). Future studies could test this possibility by replicating Experiments 7 and 8 with a longer mindfulness practice. For example, participants could practice MM for 8 weeks as in the common MBSR and MBCT interventions (Kabat-Zinn, 1982; Segal et al., 2002). However, given that the mindfulness intervention appears to have an effect opposite to its original aim (i.e., lengthening instead of shortening), future studies should also consider new interventions that could have greater beneficial effects in reducing the perceived duration of pain. A breathing exercise would be advised, given that Droit-Volet et al. (2015) and Kramer et al. (2013) found it to affect time perception even after a short practice.

## **9.4 – Conclusions**

The investigations conducted in this thesis advanced our knowledge on the effects of pain on human time perception. We now know that task-relevancy is critical for the lengthening effect of pain on perceived duration: whilst painful stimuli are perceived longer than neutral ones, pain does not increase the perceived duration of concurrent neutral stimuli. Importantly, this thesis also showed that arousal mediates the lengthening effect of pain on perceived duration only in task-relevant situations, meanwhile physiological arousal and verbal estimates are unrelated when pain is task-irrelevant. Furthermore, this thesis showed that pain does not affect the memory processes required to recall the duration of a neutral stimulus after several minutes delay. Finally, this thesis demonstrated that 1-week mindfulness body-scan



intervention is not able to reduce the perceived duration of painful stimuli in healthy people and chronic pain patients.

In summary, this thesis generated new evidence for our understanding of the pain effects on time perception. However, the mechanisms underlying these effects are yet to be explored and required future investigation, which could also deepen our understanding of the processes that lead to the conscious time experience. Furthermore, new interventions need to be tested to reduce the lengthening effect of pain, which could improve the quality of life of clinical populations.

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## Appendix

In Experiment 6, YES responses in the no-pain immediate condition contrasts typical responses of temporal generalisation task, where gradients were more peaked around the standard duration (Wearden, 1992). Here in the no-pain immediate condition the plot showed the peak of YES responses at the 0.875 comparison, suggesting that participants had poor temporal sensitivity. Analysis have been therefore conducted again removing participants who showed poor temporal sensitivity in the no-pain immediate condition. That is, who confused the shortest (0.625) or the longest (1.375) comparisons with the standard more than 50% of the times. With these criteria, we removed 9 participants, reporting the results based on data from the remaining 19 participants.

### *Temporal sensitivity group*

Figure A.1 shows temporal generalization gradients depicting the mean proportion of YES responses in the four conditions plotted against comparison/standard ratio. Examination of Figure A.1 suggests that in the no pain condition, gradients appear to be slightly shifted to the left following the delay. In the pain condition, Figure A.1 suggests that there was no effect of delay on responding. A repeated measures ANOVA with pain intensity (no-pain vs pain), delay (immediate vs delay) and comparison/standard ratio (0.625, 0.750, 0.875, 1, 1.125, 1.250 or 1.375) as within-subject factors was conducted. The ANOVA showed a significant main effect of ratio ( $F(1.93, 34.80) = 33.96, p < .001, \eta_p^2 = .65$ ) on proportion of YES responses. There was however no effect of pain intensity ( $F(1, 18) = 1.34, p = .26, \eta_p^2 = .07$ ) or delay ( $F(1, 18) = 0.11, p = .75, \eta_p^2 = .01$ ). There were also no significant interactions between delay and ratio ( $F(2.45, 44.04) = .53, p = .63, \eta_p^2 = .03$ ), pain intensity and delay ( $F(1, 18) = 2.75, p = .12, \eta_p^2 = .13$ ), pain intensity and ratio ( $F(2.47, 44.47) = .47, p = .67, \eta_p^2 = .03$ ) or pain intensity, delay and ratio ( $F(2.21, 39.81) = .84, p = .45, \eta_p^2 = .04$ ). Therefore, the absence of effects of pain or delay in the previous analysis was not due to the inclusion of participants who responded without

temporal sensitivity. This was further confirmed in the analysis of accuracy and variability.

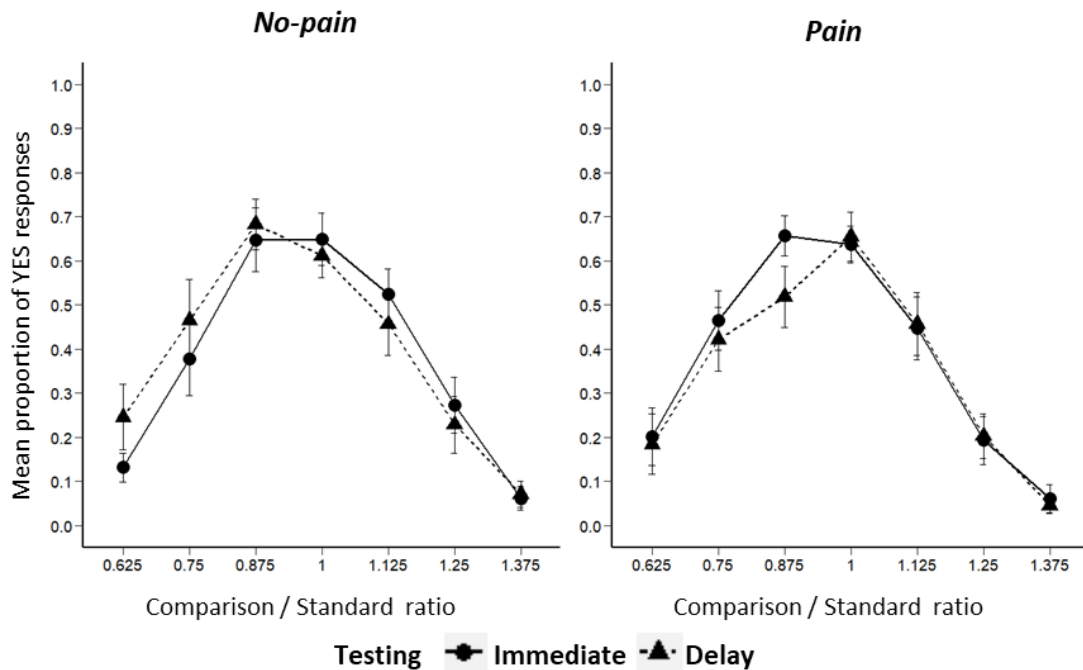


Figure A.1. Proportion of YES responses plotted against comparison/standard ratio in Experiment 6 with only participants who showed temporal sensitivity. YES responses are divided between immediate (solid line) and delay (dotted line), and between No-pain (left panel) and Pain (right panel).

#### Temporal accuracy

Table A.1 shows temporal accuracy in the four conditions. Examination of Table A.1 suggests that accuracy was similar in all conditions. A repeated measures ANOVA with pain intensity (no-pain vs pain) and delay (immediate vs delay) as within-subject factors confirmed these suggestions. There were no significant effects of pain intensity ( $F(1, 18) = .54, p = .47, \eta_p^2 = .03$ ) nor delay ( $F(1, 18) = .01, p = .94, \eta_p^2 < .001$ ) on accuracy. There was also no significant interaction effect between pain intensity and delay on accuracy ( $F(1, 18) = 1.38, p = .26, \eta_p^2 = .07$ ). Temporal accuracy was therefore unaffected by pain or delay.

<i>Condition</i>	<i>Accuracy</i>	<i>Mid-three</i>
<i>No-pain immediate</i>	0.66 (0.13)	0.69 (0.14)
<i>No-pain delay</i>	0.63 (0.11)	0.65 (0.15)
<i>Pain immediate</i>	0.65 (0.11)	0.67 (0.16)
<i>Pain delay</i>	0.68 (0.13)	0.67 (0.19)

*Table A.1. Means (and standard deviations) of accuracy and mid-three in the four conditions (no-pain immediate, no-pain delay, pain immediate and pain delay) in Experiment 6 with only participants who showed temporal sensitivity.*

#### *Temporal variability*

Table A.1 shows temporal variability in the four conditions. Examination of Table A.1 suggests that variability was lower (i.e., gradients were more peaked) in the immediate testing than delayed testing conditions. A repeated measures ANOVA with pain intensity (no-pain vs pain) and delay (immediate vs delay) as within-subject factors confirmed these suggestions. There were no significant effects of pain intensity ( $F(1, 18) = .02, p = .89, \eta_p^2 = .001$ ) nor delay ( $F(1, 18) = .40, p = .53, \eta_p^2 = .02$ ) on mid-three. There was also no significant interaction effect between pain intensity and delay on mid-three ( $F(1, 18) = .36, p = .56, \eta_p^2 = .02$ ).