

**Temporal Binding  
in Causal and Non-Causal  
Event Sequences**

**Gruffydd R Humphreys**

**PhD Thesis**

**School of Psychology**

**Cardiff University**

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## Summary

The malleability of our subjective perception of time has recently received a great amount of empirical attention with respect to the Temporal Binding of intentional actions to their subsequent effects (e.g. Haggard, Clark & Kalogeras, 2002). Given the number of studies utilizing the venerable Libet clock method, this thesis presents several novel methods for the investigation of the Temporal Binding phenomenon. Firstly, five experiments requiring the numerical estimation of intervals between operant action and subsequent effect, as well as intervals between superficially identical (based on stimulus properties) observed sequences showed that participants judged operant intervals to be shorter than relative observed intervals. This effect existed at intervals far longer than those previously reported (Haggard et al., 2002), whilst demonstrating a pattern of results similar to previous studies involving hypothesized changes of internal clock speeds (e.g. Penton-Voak, Edwards, Percival, & Wearden, 1996). This shortening of the reported interval between cause and effect also occurred when numerical estimation was replaced with the reproduction of the inter-event interval.

Having demonstrated a Temporal Binding effect with these novel methods, I then investigate the Causality based explanation of Eagleman & Holcombe (2002), employing keypress timings as an indicator of the perceived timing of the awareness of events. In three experiments, when both conditions involve intentional action, a Binding effect only occurs when this intentional action results in a caused effect. Having thus demonstrated that Causality is an essential pre-requisite of Temporal Binding, two experiments involving the judgment of the length of an object between two classic Michottean launching stimuli show shorter reported lengths for causally related (instantaneous launch) relative to unrelated (delayed launch) trials. I therefore argue that Binding is an online process that influences our perception of the relationship between causal action and effect in *both* temporal and spatial domains.

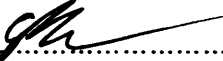
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## Preface

The work for this PhD thesis was conducted at Cardiff University and the following experiments have been accepted or submitted for publication: **Experiments 1-5**, Humphreys, G. R., & Buehner, M. J. (2009). Magnitude estimation reveals temporal binding at super-second intervals. *Journal of Experimental Psychology: Human Perception and Performance*, 35(5), 1542-1549; **Experiment 6**, Humphreys & Buehner. Temporal Binding, a Reproduction Paradigm. (*manuscript submitted*); **Experiments 8 & 9**, Buehner, M. J., & Humphreys, G. R. (2009). Causal Binding of Actions to their Effects. *Psychological Science*, 20(1), 1221-1228; **Experiments 10 & 11**, Buehner, M.J. & Humphreys, G. R. (2010). Causal Contraction: Spatial Binding in the Perception of Collision Events. *Psychological Science*, 21(1), 44-48.

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Cheers!

Gruff Humphreys

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## 0. Overview

How malleable is our perception of events in a temporal stream? An immediate response would be that we probably experience temporal order and relations accurately. However recent evidence disagrees: The perceived onset of an event resultant from an intended action has been shown to shift in time towards the action. This temporal shift was originally hypothesized to facilitate an improved perception of intention between one's own action and its resultant effect: the Temporal Binding phenomenon. A wide range of studies with similar methodologies replicated and furthered understanding of this phenomenon, with respect to this "Intentional Based" explanation. However, to my knowledge, there have been few studies examining an alternate theory, suggesting that Temporal Binding improves the causal percept of the relationship.

Firstly, I will discuss human interval timing with respect to the Scalar Expectancy Theory and how various events and stimulus properties can manipulate how we perceive temporal relations. Next, I will examine how temporal relations between events can give rise to the understanding of complex relations between them such as a causal relationship, before moving on to how a manipulation of inter-event interval (Temporal Binding) appears to help create a sense of agency. Having outlined the Temporal Binding phenomenon, I will attempt in the first part of my Empirical Section to replicate earlier Temporal Binding effects with two novel methods. In the second part of this Empirical Section, I will attempt to dissociate the intentionality/causality dichotomy with several paradigms. In the third part of the Empirical Section, I will attempt to demonstrate a spatial analogue of the Temporal Binding effect. Finally, I will discuss select results and their implication for future research.

# **1. Introduction**

## **1.1 The Measurement of Time**

Since the dawn of time, man has been busy attempting to measure it. From the earliest Egyptian sundials through hourglasses to atomic clocks, the measurement of time has been a consuming passion of humanity. Given the short amount of it that we can possibly experience given our (relatively) short lifespans, it has an inherent importance and worth. Due to the busy nature of a society driven by a 24-hour global economy, the phrase “time is money” perhaps best sums up our attitude to it, and being able to accurately measure time is perhaps more important than ever.

Over long periods of time humans appear to possess an ability to distinguish the general temporal flow via circadian rhythms to identify the approximate time of night and day over 24 hours – in the absence of environmental cues (Takahashi & Zatz, 1982). There is, however, some debate in the timing literature concerning the mechanisms underlying the human timing of short intervals. Indeed, it appears that timing involves the utilization of some amount of cognitive resource: Non-temporal tasks performed in concurrence with temporal production tasks result in a marked interference in the temporal production tasks (Brown, 1997). This interference effect is present in the processing of intervals in the matter of seconds, however it is apparently absent at short intervals from 50ms-100ms (Rammsayer & Lima, 1991). Rammsayer and Lima proposed the existence of two distinct timing mechanisms: At shorter intervals, timing is highly sensory in nature; at longer intervals, interval timing is achieved via a mechanism that utilizes cognitive resources. Support for two timing mechanisms for short versus longer intervals came from a meta-analysis of studies involving the neuroimaging of participants during timing tasks that suggested two

distinct systems: automatic and cognitive (Lewis & Miall, 2003). In a series of experiments Grondin, Meilleur-Wells and Lachance (1999) identified the point at which timing switches from sensory to cognitive mechanisms as occurring for intervals greater than approximately 1.2 seconds in length. However, this dual-system view has been brought into question, as presenting participants with a series of different cognitive interference tasks can result in interference at both short and long intervals (Rammsayer & Ulrich, 2005). A mental arithmetic task performed concurrently with a temporal discrimination task demonstrated a marked effect on the temporal task at both long and short intervals (1,000ms and 100ms respectively). Also, manipulating the loudness of the two target stimuli resulted in worse performance on the discrimination task when participants were presented with incongruent (both tones different loudness) relative to congruent trials across short and long target durations. These findings challenge the dual-system view of interval processing in two ways. Firstly, they demonstrated that cognitive load affects both long and short interval processing. Secondly, while a dual-system view would expect perceptual noise (as induced by incongruent loudness) to affect discriminations only at short intervals (as would be predicted by the dual system's claim that short interval processing is sensory in nature), this was not the case.

Rammsayer and Ulrich (2005) argued that this pattern of results, in which cognitive loads disrupts timing at both long and short intervals, discredits the dual system hypothesis. Rather, they argued, it speaks in favour of a cognitive model of timing that is influenced by attention. A popular and influential model of human interval timing based on the concept of an internal "clock" exists in the form of Scalar Expectancy theory (SET: Gibbon, Church, & Meck, 1984). Originally developed as a model of animal timing (Gibbon, 1977) SET posits that individuals perceive time due to a cognitive mechanism with three parts. Firstly, a "pacemaker" produces pulses at some

rate (these are assumed to be fairly fast, but display a Poisson distribution). The pacemaker is connected to an accumulator via a switch mechanism, which once opened allows the pulses from the pacemaker to flow into the accumulator. The default position of this switch is closed, however at the commencement of the interval that is to be timed, the switch opens, pulses enter the accumulator, and, at the end of the to-be-timed interval, the accumulator totals the number of pulses and in this way infers the length of the interval. Intervals of differing lengths can be distinguished based on the number of accumulated pulses, with more pulses indicating a longer interval. The third component consists of a series of decision processes (including working and reference memory components) by which this can be achieved (Gibbon, Church, & Meck, 1984). Over the years, SET has received a great deal of empirical focus, and in the next section I will discuss some of these studies, as well as applying this model to the results of the studies presented in the Empirical Section.

### **1.1.2 SET and Human Interval Timing**

We would perhaps like to think that we accurately perceive time in the same way a clock measures seconds passing. However, popular idioms such as “a watched pot never boils” suggests that perhaps this notion is a fallacy. It has long been known that stimuli with different properties are perceived to have different lengths. For example, moving stimuli are judged to last longer than stationary stimuli (Brown, 1995), lower intensity sounds are judged shorter than higher intensity sounds (Walker & Scott, 1981), whilst sounds are judged to last longer than lights (Goldstone & Lhamon, 1974; Wearden, Edwards, Fakhri, & Percival, 1998).

Several studies have investigated this illusory perceptual effect with reference to

modulations in two parts of the SET. Firstly, a modulation in the speed at which the pacemaker produces pulses can “slow down” or “speed up” the internal clock by producing more or fewer pulses respectively over a fixed period of time, whilst secondly a temporal shift in the onset of the gate mechanism can result in shorter or longer intervals by opening or closing the gate earlier or later. Further, these two effects do not necessarily occur independently. A study of audio visual timing by Wearden, Edwards, Fakhri and Percival (1998) employing numerous paradigms found a pattern of results suggestive of the pacemaker running slower (generating fewer pulses) for lights than sounds based on an increasing difference over longer intervals.

There is a wealth of data spanning back over nearly a century that investigates how the modulation of certain aspects of the person’s condition, such as body temperature (Hoagland, 1933; Wearden & Penton-Voak, 1995), can speed up or slow down an “internal clock.” Another avenue of research is how subjective arousal can affect the speed by which the pacemaker produces pulses. Penton-Voak, Edwards, Percival, and Wearden (1996), following a similar study by Treisman, Faulkner, Naish, and Brogan (1990), presented participants with 5s click trains immediately prior to auditory stimuli which they believed would result in the participant being more aroused at the onset of the stimulus. With experiments involving the comparison of interval duration to a target presented at the start of a block (temporal generalization), two interval comparison, magnitude estimation, and a duration production paradigm, they demonstrated that preceding a stimulus with a click train resulted in the duration of the stimulus being reported as longer than comparisons, in accordance with the idea that arousal had “sped up” the pacemaker, producing more pulses to enter the accumulator.

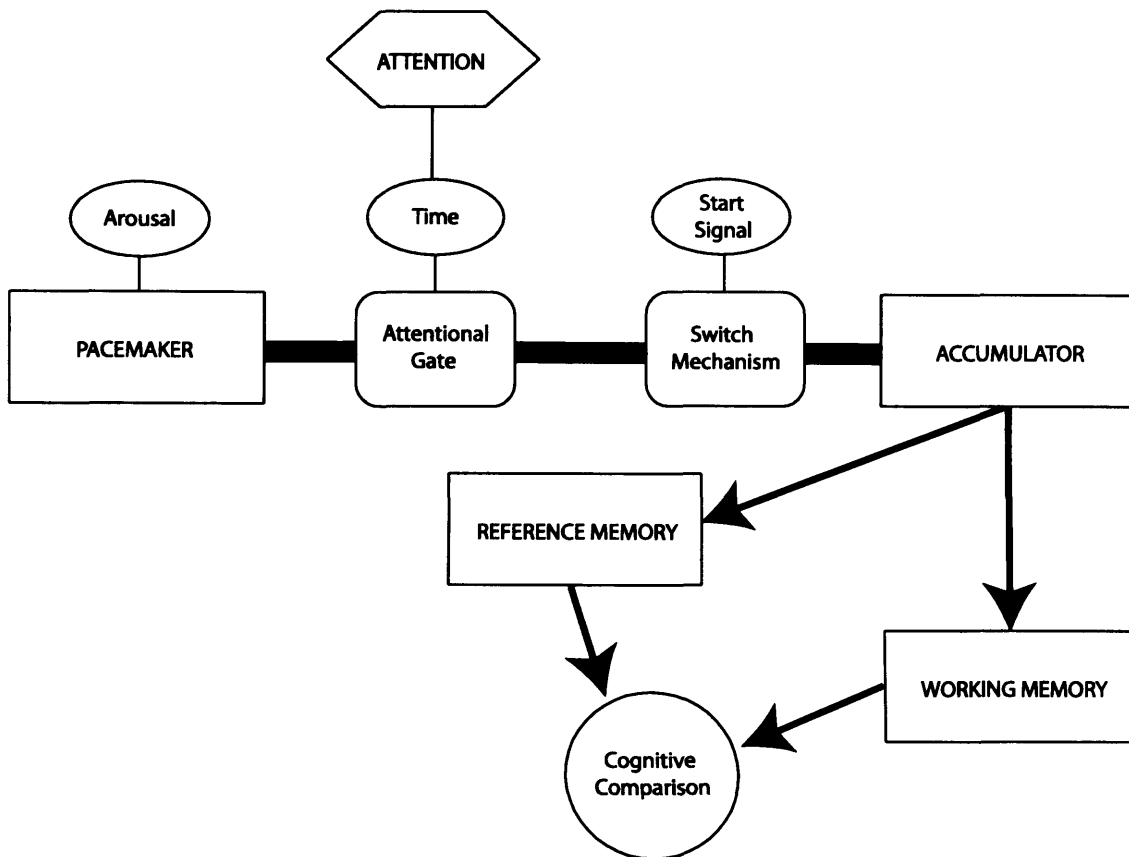
Whilst the arousal state of an individual can be used to speed up the pacemaker it can also be used to slow it down. Wearden (2008) demonstrated that inducing

boredom in participants by interspersing trials with 10s delays (as measured by self-report) resulted in a shorter reported duration for stimuli presented late in the experimental run relative to those presented early, as measured by temporal generalization and magnitude estimation. The general pattern of results was suggestive of a decrease in clock speed (fewer pulses produced by pacemaker) as induced by boredom (see also, Wearden, 1999). Further, differences in the timing of the opening and closing of the gate in relative conditions can also explain why some events are judged as having different durations. For example, a stimulus presented immediately after an eye movement undergoes a subjective lengthening of the perceived duration (Saccadic Chronostasis: Yarrow, Haggard, Heal, Brown, & Rothwell, 2001) and, in a series of experiments, Yarrow, Whiteley, Haggard, and Rothwell (2004) demonstrated that the lengthening of the post-saccadic event was constant across stimuli with varying durations (e.g., 100-300ms). Whilst an arousal (change in clock speed) account would have predicted that the effect would increase as a factor of this interval duration, changes in the onset of the gate mechanism would produce this observed fixed increase (Yarrow, Whiteley, Haggard, & Rothwell, 2004).

Whilst the previous studies investigated the arousal state of the observer as a factor in the modification of the pulse rate of the accumulator, many authors suggest that the amount of attention paid to the passage of time can also modify the perceived duration of it; the popular phrase “Time flies when you’re having fun” being an example of the duration of time appearing shorter due to an individual being engrossed with other activities (and therefore not paying attention to how much time has elapsed). While it has been argued that SET cannot accurately describe human interval timing because it does not incorporate attentional elements (Lejeune, 1998), the Attentional Gate model (AGM) of human timing states that a limited attentional resource must be



split between temporal and non-temporal aspects of a timing task (Zakay & Block, 1997). It consists of a modified SET pacemaker-accumulator mechanism (with a switch that is either open or closed) and working and reference memory components, with the addition of a gate between pacemaker and accumulator that is operated by attention (see Figure 1 for a graphical representation). In effect, Pulses enter the accumulator (and thereby indicate the passing of time), when the attentional gate is open, although this is not an all or nothing process: Attention to time has been compared to a water tap in that while it can be either on or off, the volume of time units passing through varies with how “open” it is (Reeves & Sperling, 1986). The more attention paid to the passing of time, the more “open” the gate becomes, and the more pulses are allowed into the pacemaker (i.e., fewer are “missed”). In effect, the more attention that is paid to the timing of the interval, the fewer pulses enter the accumulator, and the shorter the interval appears; “time flies.”



**Figure 1.** Graphical representation of the Attentional Gate Model. Adapted from Zakay & Block (1997).

There is a deal of debate as to whether the switch mechanism of the SET can encompass variable attentional resources (Lejeune, 1998, 2000; Zakay, 2000). In the unmodified SET model it has been suggested that the gate which, when open, allows pulses to enter the accumulator (and when closed does not) “flickers” in the presence of attention-drawing stimuli: The switch may briefly close at the beginning of the stimulus, or for the duration of the stimulus that is attended to (Lejeune, 1998). This flickering switch removes the need for an additional attention-based component to the SET model as fewer pulses enter the accumulator due to the switch closing for brief periods in which other stimuli, or breaks in the timed stimulus, are present.

While there is some debate as to the validity of this more parsimonious account

(see: Lejeune, 2000; Zakay, 2000) there is a deal of evidence that demonstrates that the distribution of limited attentional resources can warp the perceived duration of an interval, usually by requiring participants to perform two concurrent tasks, one a temporal discrimination or production task, the other a distractor task. Rammsayer and Ulrich (2005) argued that their finding of cognitive tasks eliciting worse performance in temporal discrimination tasks was due to the cognitive task drawing upon attentional resources in line with the Attentional Gate model (and contrary to a dual process timing view- see section 1.1). Macar, Grondin and Casini (1994) had participants count the number of animal names presented within a short period (12s or 18s), and/or estimate the duration of the interval in which the visual stimuli were presented. They were also prompted to proportionally distribute their attention between the two tasks, e.g., 25% on the word discrimination task, 75% on the temporal task. They found that participants' underestimation of the timed interval increased when they attributed more attention to the non-temporal word discrimination tasks. In a neat demonstration of non-task-induced factors influencing perceived duration, high- and low-urge smokers were told that they would be allowed to smoke in a few minutes, then made short interval estimations (Sayette, Loewenstein, Kirchner, & Travis, 2003). Those with high-smoking urges judged the intervals as significantly longer than those with low urges to smoke, presumably because those with a strong urge to smoke were attending more to the passing of time before they were allowed to smoke.

Further, the predictability of events has also been demonstrated to affect interval durations. The introduction of a low probability "oddball" stimulus in a series of high probability stimuli leads to the oddball being judged as longer than the others due to possible arousal increasing pacemaker speed or an increase in attention diverted towards it resulting in a greater number of pulses being "missed" (Tse, Intriligator, Rivest, &

Cavanagh, 2004).

### **1.1.3 Summary**

The previous section discussed how we perceive the passage of time, and how an individual's perception of time can be manipulated by various properties of the objects they are viewing. A currently popular model (SET) involves a pacemaker that is connected to an accumulator by a gating mechanism. While the gate is open, the pacemaker pulses gather in the accumulator; these pulses are then counted, and the interval is estimated. The speed at which the pacemaker produces pulses is variable, leading to differing perceptions of time based on arousal, and the attentional demands of task (according to the Attentional Gate mechanism) may shorten judged duration by taking attention away from the passage of time and "missing" a number of pulses. However, I will discuss in the next section how the temporal relationship between events can help to develop a perceived relationship between events.

## **1.2 Temporal Relations and Perceived Relationships**

In a later section, I will discuss how inter-event intervals can give rise to a perceived relationship between events; however, I will briefly discuss how even unperceived intervals between subconscious events can create a conscious percept of a relationship. Temporal relations have been suggested as a critical factor for the solution of the "Binding Problem" (von der Malsburg, 1981): The question as to how we perceive various properties (e.g., size, colour, texture) as belonging to a single stimulus or event, when these are processed across different perceptual modalities, such as binding the sound of a ball hitting a bat with its visual representation (Roskies, 1999).

Further neuronal studies have suggested the existence of a double dissociation within perceptual streams across cortical areas. Mishkin, Ungerleider, and Macko (1983) suggested that visual processes are divided into two distinct functions: one that assigns meaning to the object being viewed (what it is), and the other that determines its spatial location (where it is). These two processes are located in distinct cortical areas: the former in inferotemporal cortex, the latter in the posterior parietal cortex. In the field of the binding problem, research is currently moving towards the concept of binding representational neurons based on the temporal similarity of their firing patterns. It had previously been suggested that perceptual grouping could be achieved through the formation of cell assemblies. This was furthered by the suggestion that assemblies could ultimately be defined by synchronous firing of neurons (Milner, 1974; von der Malsburg, 1981), in a range of less than 5ms (von der Malsburg, 1985).

This theory solves the problem of binding between processes (e.g., "what" and "where") that are often located in different brain areas by associating neuronal representations of objects (what they are, where they are) based on synchronous firing of cells and cell clusters with latencies of less than 10ms (Singer, 1999). A great deal of empirical work has investigated this hypothesis: Single cell recordings in the cat visual cortex have demonstrated synchronous firings of neuronal populations within the visual cortex to aligned light bars (Gray & Singer, 1989), and these recordings are also correlated with synchronous firing in the Superior Colliculus (Brecht, Singer, & Engel, 1998). Greene (2006) presented human participants with a series of dot pairs that, when combined, formed the outline of an object (e.g., a camel). These were presented at random locations of the "image" near simultaneously. Participants were asked to identify the image, and the introduction of a slight delay (as little as 2ms) between successive dot presentations led to a significant drop in object recognition. Further, an

increase in spatial distance between simultaneously presented dot pairs, led to a reduction in the correct identification of the target (Greene, 2007).

Errors in the correct synchronization of neurons have been approached as a possible explanation for some mental disorders. Autistic spectrum disorder, for example, as a large class of neuro-developmental disorders, is currently being considered as a “connection disorder” (Uhlhaas & Singer, 2007), in which a developmental abnormality leads to an imbalance in communication between distal brain areas, resulting in symptoms such as errors in the integration of sensory information. Schizophrenia has also been suggested to stem from failures in communication between, and the coordination of, disparate brain regions (Ford, Krystal, & Mathalon, 2007). In an empirical analysis of this hypothesis, the synchronization of firing neurons immediately pre-speech in schizophrenic and control participants was compared to post-vocalization activity while participants either spoke or listened to their own speech (Ford, Roach, Faustman, & Mathalon, 2007). It has been hypothesized that the pre-vocalization neural activity related to a dampening forward-mechanism that prepared the CNS for the sensory consequences of the action, dissociating internally and externally generated utterances, and dampening an irrelevant response to internally generated events (Hamada, Miyashita, & Tanaka, 1999). Compared to healthy controls, the schizophrenic participants displayed less pre-speech neural synchrony; this was especially pronounced in those patients who suffered severe auditory hallucinations, suggesting a difficulty in the disassociation of internal and externally generated utterances arising from a failure to correctly synchronize the neurons that constitute a dampening mechanism (Ford, Roach, Faustman, & Mathalon, 2007).

It appears that a near simultaneous firing of neurons across spatially distal brain areas can give rise to a perceived relationship between them. We as individuals do not

perceive these temporal relationships, nor the neuronal activity. Rather, we are aware of the effects of this binding in the stimuli we observe. Therefore, an immediate relationship between subconscious events can structure our perceptions, although it is not always required that these events co-occur. Rene Descartes (1641/1986) outlined his concept of a human being as consisting of two distinct wholes: Firstly, the body acts as a mechanism with the ability to extend and produce motion. Secondly, the conscious mind is a non-physical entity that exists unbound by physics. As such the mind is free to influence the body and cause movement in it. This raises an obvious question: How then, do these two distinct elements interact to create a human as a whole when the conscious mind and the brain are somehow linked?

Libet, Gleason, Wright, and Pearl (1983) directly investigated the relationship between conscious intention to perform an action and neuronal states by observing the disparity between the observed cerebral activity preceding voluntary movements (the Readiness Potential - RP) and a participant's subjective awareness of his/her intention to make the said movement (W-judgment). Participants observed a small clock, marked in 12 intervals of 5, in which a clock hand fully rotated clockwise once every 2.65s. Participants were instructed to make voluntary movements of their hand at a time of their choosing, but were asked to avoid making movements that were dependent upon a pre-decided clock position. The clock hand stopped rotating a random duration after the movement and participants indicated, with reference to the clock hand, when they first became conscious of the desire to act. This subjective indicator of the timing of intentional action was then compared to the objective timings of the readiness potential measured with electroencephalograph recordings.

Participants' neural preparatory signals (Readiness Potentials) began on average 700ms before participants made their simple movement. However, participants only

became consciously aware of a desire to move on average 200ms before movement; unconscious Readiness Potentials preceded Conscious Intention, as reported with the Libet clock position, by a mean of 500ms. Even though these two distinct events (conscious and unconscious) are linked, they are separated by a fairly long temporal interval. These findings contradict the traditional Cartesian notion that our physical actions are subject to our own conscious free will: The "I" as an entity equipped with free will can choose to act or not act, and our brains (and by extension our bodies) follow. Libet and colleagues' findings appear to provide a complete reversal to this view: Our brains choose to act and the "I" follows.

These findings have been understandably controversial. *Prima facie*, it almost suggests the presence of a super-conscious decision-maker over which we have no knowledge. Libet (1985) however defended the concept of free will, proposing two ways in which the "I" controls the action. Firstly, he argued that consciousness may have the power of veto over the unconscious prepared motor command. When participants were instructed to prepare a movement but 100ms-200ms in advance cancel the said movement, they displayed a pre-event potential in the absence of movement, which Libet, Wright, and Gleason (1983) suggested was this process in action (M-veto). Secondly, Libet (1985) suggested an alternative conscious control mechanism, the conscious "trigger," that ultimately green-lights a prepared readiness potential, allowing it to complete and result in an action. In the absence of this conscious trigger, the movement does not occur.

Despite Libet's (1985) defence of conscious free will, his findings have led to great deal of discussion about both his design and the philosophical implications of his results. Firstly, given the requirement of matching internal awareness with external clock positions, it leaves the results vulnerable to the prior entry phenomenon (Haggard,



Newman, & Magno, 1999). In this phenomenon, events occurring on an unattended perceptual stream appear to occur later than those on the attended stream (Sternberg, 1973). From a more philosophical standpoint, Libet's (1999) assertion that the veto has no neural precursor has led his thoughts to be questioned by others (Gomes, 1999; Wood, 1985) as harking back to the immaterial difference between mind and body as opposed to the more modern Compatibilist theory that suggests that the conscious mind arises from a series of pre-determined neuronal processes that is not incompatible with free will. If we think of brain activity as a constant cause-effect chain that follows natural laws, then *all actions* are predetermined by prior causes. A Compatibilist, however, would argue that although events in the world can be reduced at a quantum level to probabilistic relations, we still have free will because our actions are free unless they are *forced* by external events. For example, to choose not to choose, based on the pre-determined nature of existence, is in itself a free choice (Gomes, 1998). Nevertheless, temporally proximal sub-conscious events, as well as temporally distal sub-conscious and conscious events, can apparently be joined together to form a perceived relationship between the two within an individual's physical self. A further question is how the relationships between events outside the body (either independent of, or related to, an individual's action) are dependent on the perceived temporal interval between them.

### **1.2.2 Temporal Relations and Causality**

Outside our own bodies we can frequently understand that events happening at spatially and temporally independent places are related and thus it is possible for us to perceive the world as an ordered and predictable chain of events in certain situations. For example, the understanding and inference of cause-effect relations are essential in

the field of diagnostic medicine and epidemiology (e.g. Vineis, 2003). Arguably in all domains, outside of mathematics and logic, it can be surmised that an accurate understanding of the causal relationship between events is essential for effective reasoning and problem solving (Newsome, 2003).

David Hume (1739/1888) considered the question of how individuals observe two objects or events to develop an understanding that a change in one was caused by the other, and he proposed three causal cues must be present in the “relationship” between events that are necessary for the impression of causality to arise in an observer immediately post cause-effect sequence: that the Effect must always follow the Cause, that the Cause must always have temporal priority over the Effect, and that Cause and Effect must be close together in both space and time.

Of most interest to this thesis is this final assertion that cause and effect must be located closely in both space and time, and how our perception of this interval can manipulate our perception of the relationship. Turning to (a somewhat hackneyed) light-bulb analogy, it seems likely that you would perceive your pressing a light-switch as having caused a bulb to illuminate if both events occurred near-instantaneously. However, this perception of causality seems less likely if the illumination occurred 10 minutes after your action. In a classic scientific example of this effect, Michotte (1946/1963) presented participants with his famous “launching” stimuli. Participants saw two stimuli, circles A and B. Circle B was located motionless at the centre of the field while circle A moved at a constant speed towards it. When A came into contact with B, A stopped, and B began to move. When B commenced its movement immediately after contact with A, participants reported a “launching” effect: Circle A colliding with B *caused* B to move. In this condition, the commencement of the effect (movement of B) occurred instantaneously after the cause (collision of A with B).

However when the two events were temporally separated via the introduction of a slight delay (200ms) between the cessation of the movement of A and the start of B's movement, the causal impression was broken and B was less likely to be reported as having been "launched" by A. The same effect of breaking this launch impression was also observed when A and B were separated by a spatial gap.

Seemingly, an effect occurring immediately after a candidate cause can result in the two being bound together into a causal chain. However experience tells us that the intervals between cause and effect vary depending on the relationships between them. Shanks, Pearson and Dickinson (1989) asked participants to press a button that caused (with a 0.75 contingency) an onscreen stimulus to light up after a set interval. Participants reported a causal relationship between action and effect when the interval between participants' button press and the stimulus change was fairly short, and when this inter-event interval was increased to over 2s, the causal judgments fell.

The intervals over which Shanks et al. (1989) demonstrated a reduction in the causal reports were longer than the short delay that ruined the causal launching of Michotte (1946/1963). It is likely that the influence of injected inter-event delays on causal inference is mediated by assumptions about the temporal relations between the candidate cause and effects (Einhorn & Hogarth, 1986). Either implicit (Buehner & May, 2002), or explicit (Buehner & May, 2003, 2004) expectations of an interval between events can reduce or abolish the detriment of temporal distance to the causal percept. Buehner and May (2002) presented participants with a series of stimuli representative of either a light-switch and bulb or a grenade launcher on a firing range, and participants were instructed to flick the switch and observe the light turn on, or fire the grenade launcher and observe the explosion. Without *explicitly* instructing participants that in a real world situation a delay would be expected between the firing

and detonation of a grenade, participants' causal rating of the launcher causing the explosion were higher than their causal ratings of the switch causing the light to turn on when both causes and effects were separated by a 5s delay.

In a demonstration of how an understanding of the relationship between events appears to effect how temporally distal events are perceived, Schlottmann (1999) presented adult and child participants with a box with two holes (A and B). The experimenter would drop a ball into A, and then a few seconds later would insert a ball into B, which was immediately followed by the ringing of a bell. When participants were asked to indicate which hole the ball being placed into caused the bell to ring, all indicated hole B, which was closest in time to the ringing of the bell.

The experimenter then allowed the participant to inspect the box, and observe two devices that could be placed into the box, so that when the ball was dropped through one hole only it would trigger the mechanism, causing the bell to ring. One mechanism was a "see-saw", which would quickly ring the bell when the weight of the ball was placed upon it, whilst the other was a runway, which would allow the ball to slowly run down it, before it rang the bell. Thus participants learnt that the box could contain devices that rang the bell after either a short or long duration, and they could predict when the bell would ring when they knew what mechanism was contained in the box. The experimenter then showed one mechanism to a participant, and covertly placed it under one of the holes. The experimenter then dropped a ball into hole A, and after a few seconds a ball into hole B; the bell rang immediately after. Although younger participants had some difficulty with this task, adults performed well. When the fast mechanism was in the box adult participants chose the second ball (dropped into B) as having caused the contiguous bell ring. However, when the slow ramp mechanism was in the box, they selected the first, temporally separated, ball as having caused the

bell to ring.

This understanding of causal relations as demonstrated by adults based on knowledge of real world timeframes also allows individuals to reconstruct these relations retrospectively and also from abstract data and temporal intervals that they have not observed. Hagmayer and Waldmann (2002) presented participants with data lists concerning the presence or absence of a mosquito plague per year for 20 years, and whether or not intervening steps had been taken to counter-act it. The cause (intervention) and effect (absence of plague) was presented in each list so that the contingency of cause effect in the same year was negative, yet positive when the effect followed the cause by a year. Participants were given one of two cover stories involving the intervention and asked to rate its effectiveness in preventing the mosquito plague in the same data set. They were either told that the intervention consisted of spraying insecticide over the mosquito breeding grounds, which would be expected to become effective over short period of time, *or* that mosquito-larvae eating beetles had been induced to breed by the planting of special plants, which was expected to take a longer period of time to become effective. Although the temporal relations between intervention and effect were not explicitly stated, participants indicated that the same data set was indicative of the intervention being effective when given the long-term cover story (larvae eating beetles), but ineffective when given the short-term cover story (insecticide).

Although a great deal of empirical effort has focussed on describing the causal relationships based on associative principles and the contingency of effect following cause (e.g. Cheng, 1997), the above studies suggest that an understanding of the mechanism underlying the relationships between events can mediate how we perceive the causal relationship between them. Causal Model theorists (e.g. Waldmann, 1996;

Waldmann & Holyoak, 1992) argue that our perception of causal relationships is based on top-down cognitive processes that encompass prior knowledge of similar relations and properties and can make predictions about relationships based upon these. That is not to say that contingency information is not collected, rather that contingency information is then processed based on this knowledge (Hagmayer & Waldmann, 2002). However, models that assume individuals possess knowledge of causal relationships between events have previously been criticized (Cheng, 1997) for failing to explain how causal models are created without the prior utilization of contingency information.

### **1.2.3 Summary**

In a previous section I discussed how individuals perceive temporal durations between events, and above I discussed how this temporal information could be utilized to understand the underlying relationship between the perceived events. In understanding an important relationship between events, that of causality, studies have shown that whilst simple abstract relations between the movement of two objects can be described as causal if the movement of one begins immediately after the cessation of the other (Michotte, 1946/1963), this relationship can be rendered non-causal when even a short delay is introduced between cause and effect. However, causal inference based on relations of real world time frames can tolerate long delays while still preserving the causal impression (e.g., Buehner & May, 2002; Schlottmann, 1999). In the next section, I will discuss how the perceived onset of events and the interval between them can be manipulated to help construct, rather than comprehend, a relationship.

### 1.3 Timing of Intentional Action and Subsequent Effects

The findings of Libet et al. (1983) question how the intended actions of the conscious mind are bound to the physical to form actions. A further question is how we relate these conscious intentions to their effects outside consciousness: How do I know that “I” made my arm move, and further, how do I know that “I” intended to pick up a cup or push a button. It is certainly true that a disassociation of intention and action can result in fascinating and disturbing beliefs; for example, there is a case documented where an elderly patient had an “alien” hand that had to be restrained by her other hand, lest the “other entity” controlling the left would attempt to choke her with it (Ay, Buonanno, Price, Le, & Koroshetz, 1998).

The above patient highlights an interesting point of how we perceive our own actions; the patient was aware that she herself was experiencing a series of (involuntary) movements, but was not (consciously) aware that she was making them occur. Gallagher (2000) may suggest that she was aware of her “*Sense of Ownership*” of the arm (as it is attached to her and is the object of its abuse), but she did not have a “*Sense of Agency*” (she did not believe that she was making the arm move). Rather this sense of agency was attributed to another individual: “Him” (Ay, Buonanno, Price, Le, & Koroshetz, 1998). It is even possible to create a false sense of agency; healthy participants can be led to believe that they themselves were responsible for the harming of another through a “voodoo hex,” or that they can influence the result of a sporting match (Pronin, Wegner, McCarthy, & Rodriguez, 2006).

In an empirical study of the temporal relationship between intention and subsequent effects Haggard, Aschersleben, Gehrke, and Prinz (2002a) presented participants with a design similar to Libet’s, in which clock- hand movement was initiated from a random start position by a left handed keypress,, and participants were

required to judge the onset of one of six events depending on condition. The first two conditions consisted of single-event baseline trials, in which participants judged the onset time of either a voluntary button press at the time of their choosing, or a 1kHz (100ms duration) tone. The remaining four conditions can be categorized into Reactive and Operant contexts. In Reactive trials, participants responded to a tone (1kHz, 100ms duration presented a random interval after the start of the trial) with a keypress. Participants judged either the timing of the tone or the button press. In Operant conditions, however, participants' voluntary button presses were followed after 200ms by a tone (100ms duration, 1kHz), and again, participants were required to indicate the onset of either the tone or their button press.

Haggard, et al (2002a) calculated a change in judgment error (the perceptual shift), for both motor actions and auditory stimuli in Reactive and Operant conditions by subtracting appropriate baseline values from each participant. Participant's timing judgment for button press in the single event control condition was subtracted from their temporal estimate of voluntary action in operant and reactive conditions. The same occurred for the baseline tone judgment subtracted from tone onset timing judgments in operant and reactive contexts. Action and effect event-timing estimates underwent Temporal Binding: participants' perceived timings of volitional actions and tones were shifted in time relative to baselines, resulting in a shorter interval between the two. Interestingly, the second event in a sequence experienced substantially greater relative shift than the first.

In a second study Haggard, et al (2002a) outlined an experiment in which they expanded their first experiment to include causally unrelated stimuli. The first six conditions were exactly the same as in the first experiment. In the final four however, participants were required to judge the onset time of the first or second tone in a



sequence, which were separated by a 250ms gap, *or* the onset time of the first or second voluntary keypress, which they had been trained to separate by 250ms. They found no significant binding in these sequential contexts and proposed that binding only occurred when action and stimuli were causally related, rather than a series of unrelated stimuli.

Haggard, et al. (2002a) proposed an intentionality based explanation for these results which they termed Efferent Binding. The Efferent Binding process is similar to a predictive “forward model” of movement control (Wolpert, 1997), by which individuals learn the association between intentions and their results in the outside world. Further, Haggard, et al. (2002a) suggested that on a conscious level Efferent Binding could be used to develop a sense of agency between the conscious intention and subsequent movement. When the efferent copy and motor re-afference are identical, the association between the two is strengthened, prompting learning of the relationship between the intended action and the subsequent effect. They argue that this strong relationship should result in a perceptual attraction between the perceived timing of action and effect. This shift, they claimed, aids in attributing a sense of agency between our actions and the subsequent effects based on this stronger association.

Wegner (2002) suggests that a sense of agency, that is to say a feeling of "I made that happen", does not arise from any information contained within our sensory inputs, but must rather be reconstructed after the experience of both action and effect. A key factor required for this postdictive attribution of a sense of agency is that both action and effect must be temporally contiguous: If I press a light switch and a light comes on instantaneously then it is likely that I will attribute myself to be the intentional agent in that particular action event sequence. A long delay between action and effect however would likely result in this sense of agency failing to arise. Haggard, et al. (2002a) follow this logic, arguing that their efferent binding hypothesis prompts

the attribution of agency to oneself ("I made the beep happen") by shortening the perceived interval between two events. Thus in the Sequential conditions, binding did not occur since there weren't intended causal relations between the stimuli and actions (Haggard, Aschersleben, Gehrke, & Prinz, 2002a).

The above experiments suggest that the relationship between intentional action and subsequent effect is subject to an internal process that *binds* internal state and subsequent external state into one intended action that the agent is responsible for; that is to say, the conscious being made a free intended movement that resulted an effect in the outside world, and as such considers itself to be the intentional agent. These studies have led to a series of experiments that investigate the properties of this Binding process, and are ultimately at the root of this thesis.

In an attempt to further investigate the effects of the Binding process, Haggard Clark and Kalogeras (2002) replicated the baseline and operant results of Haggard, et al (2002a), and found that the that mean reported keypress occurred 15ms later than baseline, with the resulting tone on average reported as 46ms earlier with a 250ms inter-event interval. Further, to exclude the possibility that binding occurred because the two events were attracted due to them merely appearing in the same trial (thus strengthening the association between them), Transcranial Magnetic Stimulation (TMS) was used to force an involuntary action, and participants either judged the onset of the involuntary action or the tone following 250ms later. In contrast to previous Temporal Binding results, event timing estimates appeared to be separated by an increased temporal distance (a Temporal Repulsion); that is to say that the reported onset of the motor action was earlier than veridical. Neither was binding found in a further sham TMS condition, in which participants were administered TMS over an area that would not produce movement, and were rather expected to judge the onset of the audible TMS

“click”, or the following tone. Thus, Temporal Binding occurred only when stimuli and actions were causally linked by an intended motor action, suggesting that binding is not a general process of time/sequence perception, but rather dependent on higher level concepts of real world relations, as predicted by Efferent Binding.

Wohlschläger, Haggard, Gesierich and Prinz (2003) presented participants with a design similar to Haggard, et al (2002a), in which a lever press initiates a tone after 250ms: In the first condition the participant’s lever press caused the tone. In the second, the participant viewed the experimenter press the lever, whilst in the third, the lever depressed of it’s own accord. In each trial participants were required to indicate the onset of the button press. They found that perceived time of action was shifted slightly later in the self-generated and experimenter generated conditions. Conversely in the third condition the perceived onset of the machine action appeared earlier than at baseline. Seemingly, rather than being a private mechanism in which only one’s own intention representations are subject to neural modification, Temporal Binding is independent of which agent made the action. They suggested binding arises from part of a social system used both for action generation and action understanding: Mirror Neurons (Gallese & Goldman, 1998), for example, show activation during both the actor’s object orientated actions, and whilst viewing others make similar actions, demonstrating the social properties of certain brain mechanisms. Wohlschläger, et al. also demonstrated that if an action appears to be separate to an intentional agent (when the lever depressed automatically), no binding occurred. This was even true when automatic lever press was replaced with a gloved rubber hand, giving the appearance of humanity, but not the intentionality. A similar finding was reported by Wohlschläger, Engbert, & Haggard (2003) in which the lever depressed automatically when attached to the participants finger, and did not demonstrate a binding effect due to, the authors

argued, a lack of intentionality attributed to the forced movement.

Ultimately there is a great deal of convincing evidence that speaks for the existence of this Binding process, as well as the properties of the relationship between the events and the properties of the stimuli that are required for the appearance of the phenomenon. Soon after the publication of the Haggard et al. (2002b) results an alternative explanation was proposed which moved away from the intentional aspects of the binding phenomenon. With respect to Temporal Binding, Eagleman and Holcombe (2002) suggested that temporal attraction was dependent not necessarily on intention, but rather on the causal relation between the events.

Returning to the previous section on causality, I discussed how the temporal relationship between two events is a vital cue to the attribution of a causal relationship between the two: two causal events must be temporally contiguous for the inference of a causal relationship between them. A light turning on three minutes after the depression of the light-switch is unlikely to be perceived as causally related to each other (without of course previous knowledge of the underlying mechanisms of the relationship; e.g., Buehner & May, 2002). Thus Eagleman and Holcombe argue that the mind utilizes this temporal contiguity cue bi-directionally: Whilst we are more likely to consider two contiguous events as causal, we also are more likely to consider two causally related events as contiguous. Thus when judging the timings of two events which an individual believes are causally related (through prior experience), Temporal Binding that shortens the interval between cause and effect would be an adaptive process that aids in the reconstruction of the relationship between the events (Stetson, Xu, Montague, & Eagleman, 2006). It would be especially useful in situations where an amount of uncertainty of the timing of events (e.g., measurement noise) might mask a causal relationship (Eagleman & Holcombe, 2002).

It should be important to note that a causality based explanation of the Temporal Binding phenomenon is not necessarily in contradiction with the predictions made by the Efferent Binding hypothesis. The button press → tone sequences employed in the above studies are by their very nature causally related (Haggard, Aschersleben, Gehrke, & Prinz, 2002a; Haggard, Clark, & Kalogeras, 2002b) and thus a shortening of the perceived interval between the two would naturally strengthen the inferred causal relationship between the events. As for the shortening of observed experimenter-action effect sequences (e.g. Wohlschläger, Engbert, & Haggard, 2003) the same logic applies given that again these events are causally related.

### **1.3.2 Predictive and Postdictive Processes**

To reiterate, a problem arising with the direct comparison of the two competing hypotheses is that Intentional action and subsequent effect are often causally related, just as Causal actions leading to effects are often intentional sequences. This thesis intends to disassociate Intentional and Causal action and empirically compare both the Haggard et al. (2002a) and Eagleman and Holcombe (2002) intentionality/causality based Temporal Binding hypotheses. As such, it may be interesting to consider when in the action effect sequence the binding occurs.

In a previous section, I outlined a number of studies suggesting that Temporal Binding arose because of an intention to cause the subsequent event. How, then, are intentions and movements related? One explanation involves internal CNS Forward models; these are constructs that encompass the relationship between actions and effects used to predict the consequences of a motor command (Miall & Wolpert, 1996; Wolpert, 1997). A motor command produces an efferent copy of itself that at the moment of movement is processed via a dedicated neural circuit, which predicts the

results of an action before sensory data become available. Previous and accurate knowledge of the relation between movement and effect is critical for prediction, as the internal predicted outcome of the action is compared to the external sensory data. Human inability to tickle oneself has been explained in terms of forward models of motor command accurately predicting sensory feedback, thus the prediction and sensory states cancel each other out (Blakemore, Wolpert, & Frith, 2000). However, when a delay is induced via a robot arm that follows the participants' motor action, there exists a discrepancy between predicted and sensed states, from which arises the sensation of ticklishness (Blakemore, Frith, & Wolpert, 1999). As such, Haggard et al.'s (2002a) Efferent binding falls into this predictive category of relation. The Efferent Copy is produced at the moment of movement, and results in an accurate prediction of the effect, which then strengthens the relationship and produces a binding effect.

An alternative to a predictive process is a postdictive process, in which one observes one's own action and its effects and retrospectively constructs relationships. Wegner's (2002) theory of agency is postdictive, given that all events must have been experienced prior to the construction of a sense of agency. Wegner and Wheatley (1999) demonstrated that a participant could be made to perceive the hand-movement of a confederate as their own by priming them prior to observing the movement with thoughts associated with it: On the basis of the evidence (thinking about a movement and observing it) participants inferred that they must have been the agent responsible for the movement they had just observed.

The Causal Binding hypothesis of Eagleman and Holcombe (2002) suggests that a perceived shift in the timing of events involves postdictive processes. Given the inferential nature of Humean causal cues (Hume, 1739/1888), in a postdictive case, the action and effect must have already been observed before the mind retroactively

reconstructs the relationship given the sequence's adherence to these laws. A reported causal relationship can arise when additional information is conveyed after the observed relationship (Choi & Scholl, 2006) when participants are presented with the classic Michottean stimuli (Michotte, 1946/1963) discussed in section 1.2.2. Previous studies had demonstrated that if a stimulus (A) eclipsed another (B) prior to B starting to move, participants were less likely to consider A as having caused the “launching” of B. However a causal relationship was reported when the eclipsing of B by A was observed in the presence of a separate launching stimulus pair (a “causal capture”: Scholl & Nakayama, 2001). It was later demonstrated that a similar eclipsing of B by A could induce a perceived causal launch (above chance) when presented in the presence of a single stimulus (C) that commenced movement at the same time as the movement of B. This perceived causal relationship between A and B existed even when the movement of C commenced *after* the other launched stimulus began to move.

It appears that this reconstructed causal relationship relies not only on the relationship between objects directly in the cause → effect sequence, but encompasses other events within a short temporal window of approximately 200ms (Choi & Scholl, 2006). The properties of a stimulus that is observed after the moment in which the cause resulted in the effect can affect this relationship as well: The speed of the second launched stimulus has been showed to modify the strength of the reported causal launching (Natsoulas, 1961). Further, the injection of an auditory stimulus has been shown to induce participants to strengthen the reported causal relationship between stimuli in an ambiguous display when an auditory stimulus is presented at the moment of contact, or shortly after. For example, when stimuli A and B moved towards each other from opposite ends of the display, overlapped each other, and continued onwards, an auditory stimulus presented at the moment of overlap resulted in participants

reporting that they had “bounced” against each other, in effect causing them to move apart (Sekuler, Sekuler, & Lau, 1997). This reported causal relationship was present even when the sound occurred 150ms *after* the moment of overlap (Sekuler, Sekuler, & Lau, 1997). Evidently the inference of a causal relationship was not determined solely by events at the moment of causality, when one event causes another, but also takes into consideration the events that occurred after, supporting the assertion that causal inference is a predictive process.

This predictive/postdictive question gives one possible way to approach the question of Efferent or Causal binding. In a direct test of the predictive/postdictive nature of binding, Haggard and Clark (2003) once again employed the Libet clock method and presented participants with blocks of trials in which pressing a button would cause a tone after 250ms. TMS was programmed to occur in each trial and induce a movement in the participant’s hand. If the participants pressed before the TMS, it was cancelled. If they did not, TMS application was followed 270ms later by the tone (250ms as in the previous Haggard et al. (2002a) paradigm, and a 250ms interval was employed with an additional 20ms to account for the delay between TMS and finger movement in healthy participants). Participants were again required to indicate the onset of keypress, TMS, or tone onset. Binding occurred when an intended action caused the tone, but not when intentional build up was cut short by TMS. Haggard and Clark (2003) argued that if Binding is a postdictive process, some amount of relative shift would be expected as both intentions and tones were present. From this, they suggested that a Temporal Binding process would be predictive in nature: As predictive models generate predictions of movement, unintended TMS induced movement would be inconsistent with the efferent copy of an intended movement, resulting in no association between movement and effect.



At first glance, it may be argued that this effect fundamentally undermines the Eagleman and Holcombe analysis as the TMS → Tone event sequence contains apparently causal action and subsequent effect, but no intentional motor action on the part of the participant; thus this evidence supports an intentionality based argument instead. However, as discussed previously, knowledge of the timeframe between causally related events can mediate the perceived causal relationship. I suggest that participants understand the relationship in the trial as both the movement and tone being caused by TMS. Possibly the earlier reported onset of the movement is a Binding shift towards the TMS (or rather the computer program that initiated it), and since previous Binding studies involve linear cause and effect sequences we cannot make predictions as to whether the second or third effect following an action would be shifted towards/away from the primary cause. To clarify this, although I have been discussing causal relationships as linear sequences (a direct cause) it is possible to construct causal relationships based upon multiple causes or effects: For example, in a causal chain, one cause results in an event which then causes another, i.e.,  $A \rightarrow B \rightarrow C$ . In a different causal relationship, A could cause both B and C. Alternatively, both A and B could cause C (Sloman, 2005), and this, I suggest, is how participants may comprehend the relationship between the events in the Haggard and Clark (2003) TMS experiment: TMS → finger movement *and* tone.

A similar question arises in terms of the lack of binding demonstrated by Wohlschläger et al. (2003) in their use of a self-depressing key or rubber hand to simulate a causal yet unintentional action, which also questions the Eagleman and Holcombe (2002) hypothesis. It seems unlikely that participants would consider the movement of the rubber hand or self depressing key as being an independent event (i.e., self initiated) that results in a caused effect. To elaborate, it is clear (and necessary) that

the rubber hand is incapable of intended movement, however participants observe that it moves. How then can the participant conceptualize the observed movement? It seems likely that they would understand that the movement of the rubber hand or key is initiated not by the object itself, but rather by some mechanism. If the participants construct a model of the causal relationship (e.g. Waldmann, 1996; Waldmann & Holyoak, 1992) between the events in the trial, I suggest they will consider the relationship as either Computer Program → Rubber Hand Movement → Tone, or alternatively, Computer Program → Rubber Hand Movement *and* Tone (a *common cause model*: Sloman, 2005). Again, whilst the earlier studies of Haggard et al. (2002a) and Haggard et al. (2002b) involved the Temporal Binding of simple direct cause effect sequences (i.e., Intentional Button Press → Tone sequences), the Wohlschläger et al. (2003) study and Haggard and Clark (2003) studies involved more complex causal relations (Computer Program/Rubber Hand Movement/Tone, and TMS/Movement/Tone respectively). As such, these studies may not provide sufficient evidence to disprove the Eagleman and Holcombe (2002) causality based interpretation.

Recently, the predictive/postdictive nature of binding has been brought into question. By varying the probability of the occurrence of a tone in the simple button press and tone sequence employed by Haggard et al. (2002b), Moore and Haggard (2008) demonstrated that the binding effect is dependent on both predictive and postdictive elements by making reference to a Bayesian framework that weighs predictions and observances based upon its (constantly updated) accuracy (Chater, Tenenbaum, & Yuille, 2006). When the probability of a subsequent tone was low (50% of trials), the binding shift of the intentional action occurred only in trials in which the subsequent tone was present. The authors argued that this indicated that the predictive accuracy of the presence of the tone was poor, and thus Binding shift is postdictively

based on the observed action-tone sequence. However, when the press tone contingency was high (75%), and a predictive process more accurately predicted the presence of a subsequent tone, the binding shift of the action occurred in both tone-present and tone-absent trials. The authors suggest that the predictive process in this case more accurately predicts the tone (given the high probability) and thus implements the shift, even in trials where the tone is not present.

### **1.3.3 Summary**

The above sections consisted of an outline of the phenomenon of Temporal Binding. As with the relations I detailed in earlier parts of this thesis, the temporal relationship between action and effect appears to be crucial for the maximal understanding of the relationship. Here, when an intended causal action results in an effect, there appears to be a perceived shortening of the interval between the two: the Temporal Binding phenomenon (Haggard, Aschersleben, Gehrke, & Prinz, 2002a). This effect still occurs when an experimenter is observed as the agent in the sequence (Wohlschläger, Haggard, Gesierich, & Prinz, 2003), but not, however, when the underlying cause of the sequence is unclear (Haggard & Clark, 2003).

As to the underpinnings of this Binding effect currently two explanations exist: The shortening of the interval between action and effect results in the strengthening of the perceived sense of either the Causal relationship (Eagleman & Holcombe, 2002) or Agency (Haggard, Aschersleben, Gehrke, & Prinz, 2002a) between two events. The temporal position of the process (pre- or postdictive) may offer an avenue for investigation. However, the recent findings of Moore and Haggard (2008) suggest that the temporal onset of the Binding process may not be as clear-cut. Indeed, due to the

difficulty I suggested with differentiating intentionality and causality, it would perhaps be logical to suggest that the Binding effect performs either two functions: the binding of causally and intentionally related sequences, or only one: the binding of an intended AND causally related sequence. It hardly seems unlikely that a Binding effect would serve two masters. The hypothesis that an intended action and effect sequence that is bound together results in the rise of a sense of agency (Haggard, Aschersleben, Gehrke, & Prinz, 2002a), similar to the of Apparent Mental Causation (Wegner & Wheatley, 1999). However, the cues described by Wegner and Wheatley for this sense of agency to become apparent (priority, exclusivity, and consistency) strongly resemble those of Hume's cues for the inference of causality: there is priority of cause over effect, the effect must always follow the cause, and there must be contiguity of cause and effect in space and time (Hume, 1739/1888). Thus the attribution of agency could be considered a special form of causal inference of the relationship between one's own intentional action and effect.

In later Empirical Sections, I will attempt to further investigate an Intentional/Causal distinction, however for now I will attempt to look at one possible problem with inferring the existence of a Binding effect. All the studies mentioned above that directly investigate the Temporal Binding phenomenon employed the Libet clock. Although these studies' reliance on shifts relative to baseline may neutralize any inherent biases (e.g. Flash Lag: Nijhawan, 1994; Haggard, Aschersleben, Gehrke, & Prinz, 2002a), I intend to report a series of studies that involve novel methods that also have the benefit of measuring the perceived inter-event interval, rather than inferring a change in its duration based on the relative timing of events.

## 2 Empirical Sections

The following section is split into three parts. This first Empirical Section deals with attempts to replicate the previous finding of Haggard et al. (2002a: 2002b) with a novel method and further these results. In Experiments 1-6, I will not be attempting to differentiate between causal and intentional explanations of the findings. As such, in this section I will refer to event sequences in which intended action is followed by an event as *Operational*. The alternative is a series of unrelated stimulus sequences, in which the participants are not required to make an action, and will hereafter be referred to as *Observational* trials.

In the second section I will outline a series of experiments in which I used the timing of participant motor actions as a measure of perceived onset of events. These experiments (Experiments 7-9) shared a common rationale: Participants were presented with two conditions, both involving intentional action followed by an event, but in only one of these conditions did this did participants interpret the action as causing the subsequent effect. Through these experiments, I will offer some evidence in support of the causality-based hypothesis. In the final section (Experiments 10-11), I will move out of the temporal domain to that of space, to investigate whether a causal relationship results in an attraction between events in the spatial domain.

## 2.1 Empirical Section 1

### 2.1.1 Experiment 1

The first five experiments in this Empirical Section involved the same basic method: Participants were presented with *Operant* and *Observational* trials and returned numerical judgments of the interval between events in the sequences. In Operant trials, participants pressed a button at a time of their choosing, which resulted in the delivery of a 100ms 1kHz pure tone after a set interval. In Observational trials, participants did not perform any intentional or causal actions; instead, they were presented with an audible click, which was followed by the same 100ms 1kHz pure tone after a set interval. The click in these trials was recorded from the button box used on Operant trials and thus was identical to the noise made by the micro-switch when participants pressed the button in the Operant trials. At the end of each trial, participants had to estimate the length of the interval between their button press (Operant trials) and the tone or the click and the tone (Observational trials) via numerical estimation. This method has previously been used to successfully demonstrate a Binding effect between intended action and subsequent effects, while no binding occurred between unintentional action (e.g., rubber hand movement) and subsequent events (Engbert, Wohlschläger, & Haggard, 2008). However, this study used short (200-300ms) inter-event intervals, while Experiment 1 employed intervals comparable to those of Haggard et al. (2002) in an attempt to replicate the reduced Binding shift over longer inter-event intervals. Since Operant trials involved intentional causal action and Observational trials did not, I hypothesized that participants would judge the inter-event intervals to be shorter in Operant trials than in relative Observational trials, with a greater degree of underestimation at shorter relative to longer intervals.

### **2.1.1.1 Method**

#### *Participants*

Sixteen Cardiff University students (12 female), with a median age of 20, participated for half an hour and received £3 in payment.

#### *Materials and Apparatus*

The experiment was conducted on an Apple iMac, and programmed with Psyscope (Cohen, Macwhinney, Flatt, & Provost, 1993); the psyscope “Button Box” was used for timing of stimulus delivery and as an input device in the Operant trials. Temporal estimates were entered via the keyboard.

Each experimental trial contained two events, separated by an interval. The first was either a button press, or a click in Operant or Observational trials, respectively; the second was a 100ms 1kHz pure tone.

A green or red fixation cross at the centre of the screen denoted Operant or Observational trials respectively. Crosses were removed at the beginning of the first event of each trial.

#### *Design and Procedure*

The factors *Interval* (150, 250, 350, 450, 550 and 650ms) and *Trial Type* (Observational, Operant) were factorially combined in a within-subjects procedure. Operant and Observational trials were blocked so that participants experienced one trial of each interval per block. The order of intervals within a block was random, and Operant and Observational blocks alternated. Each participant worked on 10 Operant and 10 Observational blocks, thus providing 20 separate temporal estimates for each

interval, 10 Operant and 10 Observational trials. The nature of the first block (Operant vs. Observational) was counterbalanced between participants.

Each interval began with a display of a fixation cross at the centre of the screen. The cross was green on Operant and red on Observational trials. On Operant trials, pressing the green button on the response box cleared the fixation cross from the screen and triggered the relevant interval (150, 250, 350, 450, 550, or 650ms), after which the tone was delivered. On Observational trials, the red cross remained on the screen for a random interval between 1500 and 2000ms, after which the click was presented and the cross disappeared; the pure tone was presented after the appropriate interval. The intervals used were the same as in the Operant condition however they were configured so that the interval began at the start of the "click" stimulus. This was done because we did not know when participants would begin subjectively timing the inter-event interval. Although they were instructed to judge the interval *between events*, had they commenced subjectively timing during the click stimulus when the inter-event interval began at the end of the click, it would have resulted in a longer subjective inter-event interval than the objective inter event interval. As such the positioning of the objective inter-event interval at the start of the click stimulus results in a shorter subjective inter-event interval should the subjective timing begin at any point after the start of the click, thus working against the hypothesis. Each trial ended with an on-screen prompt to provide an estimate (between 0 and 999ms) for the inter event interval.

Participants were informed that they would be partaking in a study of time perception. After giving written consent to participate in the study, participants were provided with a general outline of the experimental procedure, followed by written instructions specific to Operant or Observational blocks. At the start of Operant blocks, participants were informed that the appearance of the tone was wholly dependent on

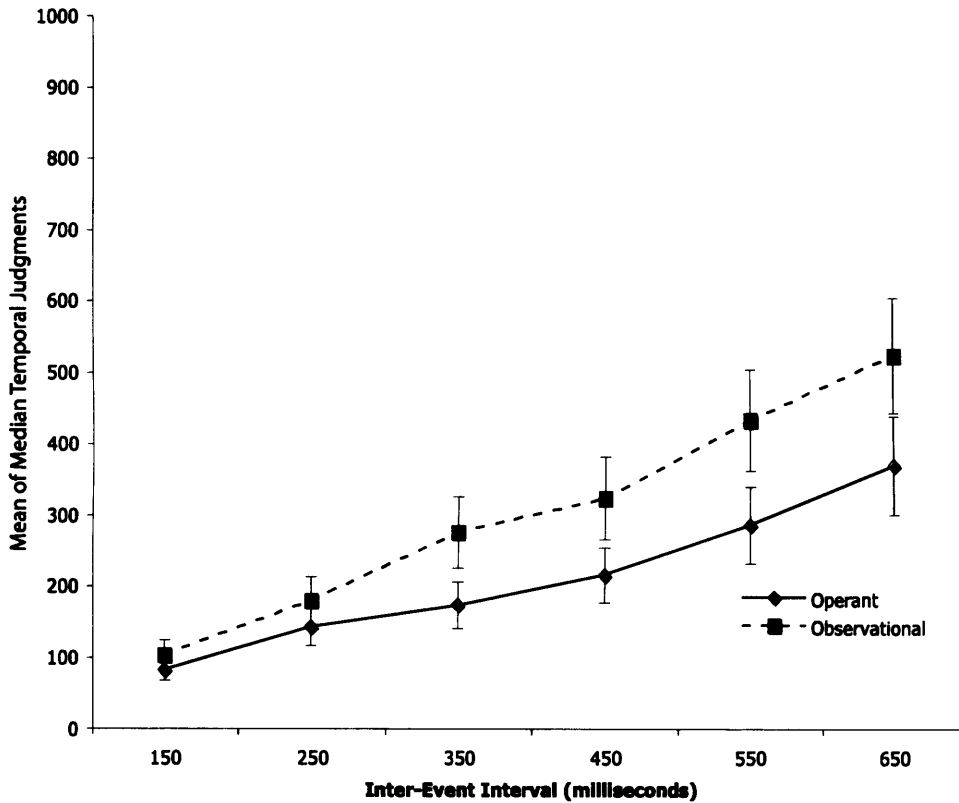


their actions: Pressing the green button on the Button Box would produce a tone after a set interval. Instructions suggested that participants could delay their button press to see that delivery of the tone was wholly dependent on their action.

Prior to Observational blocks, instructions emphasized that participants were not required to press the key, but would rather passively observe two unrelated events. These instructions were presented at the beginning of each block, in order to facilitate clear discrimination between Operant and Observational blocks

### **2.1.1.2 Results and Discussion**

All statistical analyses for this and all subsequent experiments in this Empirical Section adopted a significance level of 0.5. Each participant returned 10 temporal judgements for each Operant and Observational interval. Participant's median judgements for each interval were used as the unit of analysis and are displayed in Figure 2.



**Figure 2.** Experiment 1. Mean Median Temporal Judgment Per Participant. (Error bars show  $\pm 1$  Standard Error)

Inspection of Figure 2 shows that participants clearly distinguished between the various intervals, and suggests that Operant intervals were judged shorter than equivalent Observational intervals. Furthermore, it appears that this difference increases as a function of interval duration. A 2x6 ANOVA found significant effects of *Trial Type*,  $F(1,15)=14.00$ ,  $MSE=31373.09$ , *Interval*,  $F(5,75)=31.66$ ,  $MSE=16129.07$ , and a *Trial Type x Interval* interaction,  $F(5,75)=7.68$ ,  $MSE=3092.34$ .

To investigate the interaction with increasing inter-event intervals, we conducted a Slope Analysis that has previously been used in studies examining the numerical estimation of temporal duration (Penton-Voak, Edwards, Percival, & Wearden, 1996). An individual regression slope across the numerical judgments of Operant and

Observational inter-event intervals for each participant was calculated, and these Operant and Observational slopes were then compared with Wilcoxon Signed Rank tests. The slope of numerical Temporal Judgments were significantly less steep,  $Z=3.411$ , with 15 participants returning a shallower slope in Operant intervals, with one tie.

Experiment 1 established temporal binding as a robust empirical phenomenon that is not specific to the Libet Clock Method, or indirect measures, but can be reproduced when subjective time is measured directly with a magnitude estimation method. However, the finding that the underestimation of operant relative to observational intervals appears to increase with inter-event intervals as a function of interval length (as demonstrated by or Slope Analysis) is at variance with Haggard et al. (2002b). At their maximum interval of 650ms, perceived tone shift had fallen to 16ms, from a 103ms and 40ms shift at 250ms and 450ms respectively. These results, in contrast, suggest that perceptual shifts induced via intentional action are stronger when action and outcome are separated by a 650ms interval than when a 250ms interval is involved. The following experiment was aimed at testing the limits of the timeframe over which binding can occur given this surprising finding.

### **2.1.2 Experiment 2**

The intervals in Experiment 1 overlapped with those used in previous Libet clock experiments (250ms, 450ms and 650ms: Haggard, Clark, & Kalogeras, 2002), and inspired by the steady binding found in Experiment 1, Experiment 2 set out find binding at intervals slightly longer than those previously used, i.e. between 750ms and 1250ms.

### **2.1.2.1 Method**

#### *Participants*

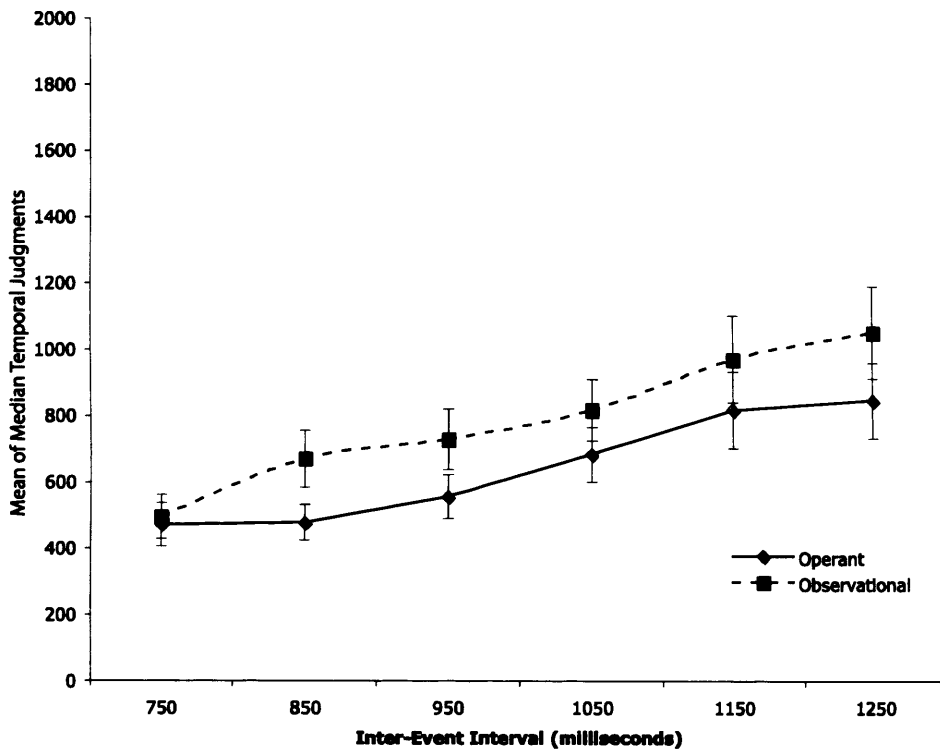
Sixteen Cardiff University Undergraduates (All female) with a median age of 19 participated for half an hour in exchange for course credit.

#### *Design, Procedure and Materials*

The same experimental design as in Experiment 1 was used in Experiment 2, except that the inter-event intervals were increased to 750ms, 850ms, 950ms, 1050ms, 1150ms and 1250ms; the admissible response range provided at the judgment prompt was altered accordingly to 0-1999ms.

### **2.1.2.2 Results and Discussion**

One participant consistently gave responses above the higher limit of the response range (1999ms) and was removed from further analysis. Every participant returned 10 temporal judgements for each Operant and Observational interval. Participants' median judgments for each interval were taken as the basis for the subsequent analysis and are shown in Figure 3.



**Figure 3.** Experiment 2. Mean Median Temporal Judgment Per Participant (Error bars show  $\pm 1$  Standard Error)

As in Experiment 1 the inter-event intervals in Operant trials appear to be considered shorter than the intervals in Observational trials. A 2x6 ANOVA found a significant effect of *Trial Type*,  $F(1,14)=19.58$ ,  $MSE=49923.16$ , *Interval*,  $F(5,70)=24.12$ ,  $MSE=41883.41$  and a *Trial Type x Interval* interaction.  $F(5,70)=3.38$ ,  $MSE=9622.14$ . The Slope Analysis which I employed in Experiment 1 was again conducted, and once more demonstrated a shallower slope for Operant versus Observational estimates,  $Z= 2.54$ , with 12 participants returning a shallower slope, 2 a steeper slope, and one tie, once again indicating an increase in Temporal Binding with increasing intervals.

Overall, the results of Experiment 2 extended the pattern found in Experiment 1: temporal binding appears to be a robust effect, increasing with increased inter-event

intervals. However, visual inspection of Figures 2 and 3 appears to demonstrate practically no difference in the shortest inter event interval between Operant and Observational trials in Experiments 1 and 2. A small shift may be expected at shorter intervals, but not at 750ms that was the shortest interval in Experiment 3. There is no theoretical reason why the effect would cease at 750ms and then return at longer intervals. Instead, it appears more plausible that this result occurred due to a methodological artefact. My speculation is that participants used the shortest interval they experienced as some form of an anchor point for judgments about subsequent, longer intervals.

Prior research indicates that participants' subjective numerical temporal judgments are malleable towards fixed events; Wearden (2006) demonstrated that participants have a tendency to quantize numerical estimates of temporal intervals towards certain "attractor intervals", clustering their interval judgments around values ending in 0 or 50. I suggest that participants in these experiments, having sampled all the intervals, recognize the shortest inter-event intervals in both conditions and assign them similar values. These attractor intervals can then be used as a baseline for subsequent judgments, in that no value falls below this anchor point. This led me to include a 0ms inter-event interval in the subsequent experiments.

### **2.1.3 Experiment 3**

To test the hypothesis that the lack of temporal binding at 750ms was due to an experimental artefact, a "0ms" interval was added into the design. This "instantaneous condition" (second event occurs immediately after the commencement of the first) is not expected to produce any temporal binding, but merely serves as an anchor point. A second purpose of Experiment 3 was to see if I could extend binding into even longer

intervals; thus I adopted a maximum interval of 2000ms. Based on the results from the previous experiments, I predicted that participants would consistently underestimate all Operant intervals, relative to equivalent Observational intervals.

### **2.1.3.1 Method**

#### *Participants*

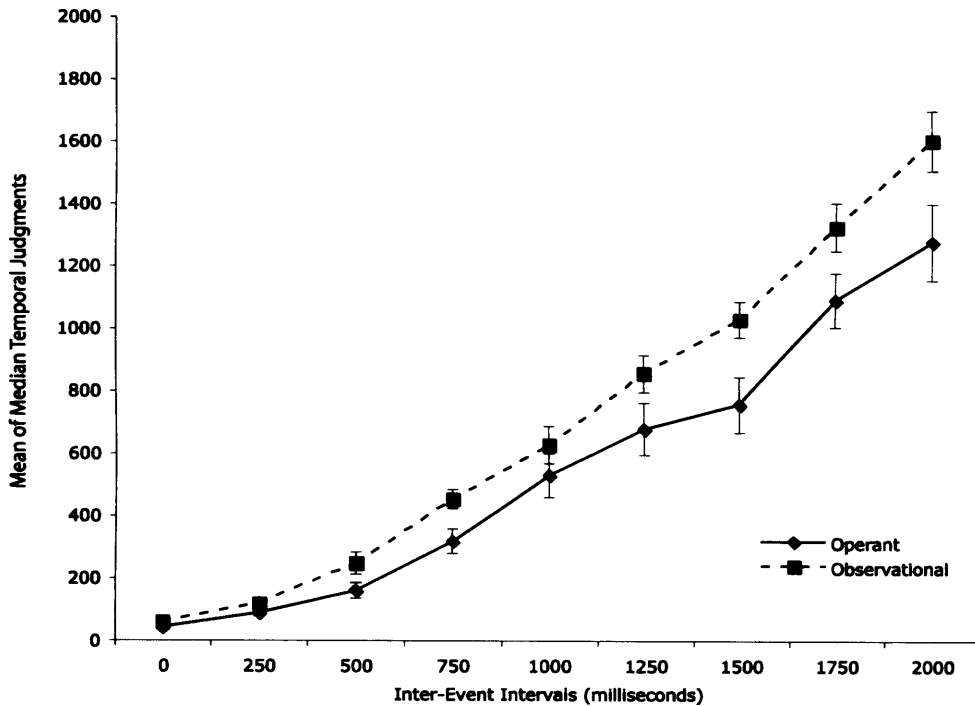
Sixteen Cardiff University Undergraduates (14 female) with a median age of 19 participated for half an hour in exchange for course credit.

#### *Design, Procedure and Materials*

The same experimental design was used as in Experiment 1 and 2. Intervals of 0ms, 250ms, 500ms, 750ms, 1000ms, 1250ms, 1500ms, 1750ms, and 2000ms were employed. Note that 0ms refers to instantaneous within the limitations of the hardware. The response range allowed was 0-2000ms.

### **2.1.3.2 Results & Discussion**

As in the previous two experiments, each participant returned 10 temporal judgements for each Operant and Observational interval, and each participant's median response for each interval was used as the basis of analysis. Figure 4 shows the same pattern of results as in the previous two experiments: Inter-event intervals in Operant trials appear to be considered shorter than intervals in relative Observational trials. Further, the results of Figure 4 describe an apparently stable amount of temporal binding over increasing intervals, as was shown in the previous two experiments.



**Figure 4.** Experiment 3. Mean Median Temporal Judgment Per Participant (Error bars show  $\pm 1$  Standard Error)

A 2x9 ANOVA found a significant effect of *Trial Type*,  $F(1, 15)= 13.66$ ,  $MSE=78674.77$ , a significant effect of *Interval*,  $F(8,120)= 86.7$ ,  $MSE=78041.14$ , and a *Trial Type x Interval* interaction,  $F(8,120)= 3.87$ ,  $MSE=18564.51$ . The Slope Analysis once again demonstrated an increase in the discrepancy between Operant and Observational estimates with increasing intervals,  $Z=3.10$ , with 13 returning a shallower slope, 3 a steeper slope.

The results of Experiment 3 was very surprising given the earlier predictions of a decreasing binding effect with increasing intervals (Haggard, Clark, & Kalogeras, 2002). It appeared to demonstrate a binding effect at intervals over three times longer that we would have expected given these results. I was curious, however, so see how far this effect would continue, and thus ran Experiment 4.



## **2.1.4 Experiment 4**

Inspired by the finding of a robust Temporal Binding effect at 2000ms, I sought to push the boundary even further. This would involve a replication of Experiment 3, but with a maximum interval of 4s and a response range from 0-4000ms. Again I predicted that Operant intervals will be judged as shorter than relative Observational intervals and that this difference will increase with longer inter-event intervals.

### **2.1.4.1 Method**

#### *Participants*

Sixteen Cardiff University Undergraduates (11 female) with a median age of 20 participated for forty minutes. They received a small financial incentive (£4) in exchange for their participation.

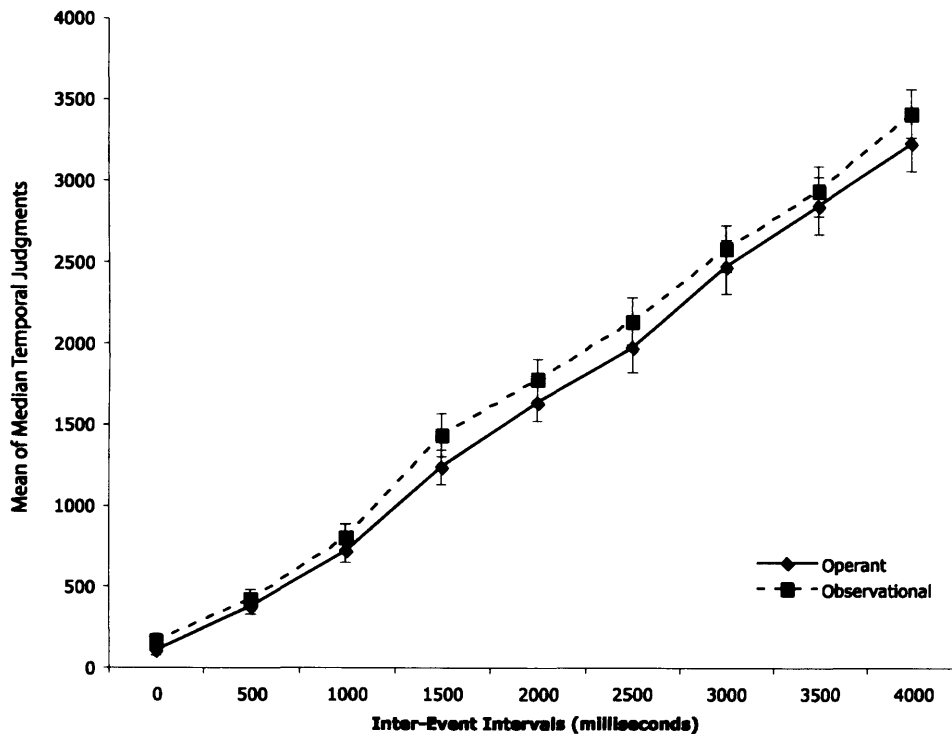
#### *Design, Procedure and Materials*

The experimental design was identical to Experiment 1, but involved the following intervals: 0ms, 500ms, 1000ms, 1500ms, 2000ms, 2500ms, 3000ms, 3500ms and 4000ms. To accommodate this, the participants' response range was changed to 0-4000.

### **2.1.4.2 Results**

Each participant gave 10 responses for each interval. As before, the median responses for each participant was used as the basis for further analysis, and the mean of these is show in Figure 5. 16 data points were considered outliers and removed from the dataset because they were more than 2 standard deviations from the mean; 14 of these

came from the same participant, who was thus dropped entirely from the sample.



**Figure 5.** Experiment 4. Mean Median Temporal Judgment Per Participant (Error bars show  $\pm 1$  Standard Error)

Inspection of Figure 5 suggests that Experiment 4 revealed a similar pattern of results as Experiments 1, 2 & 3: Inter-event intervals in Operant trials appear to be considered shorter than intervals in relative Observational trials. A 2x9 ANOVA found significant effects of *Trial Type*,  $F(1,12)= 5.44$ ,  $MSE=52229.87$ , and *Interval*,  $F(8,96)= 217.38$ ,  $MSE=174139.16$ ), but no significant interaction,  $F(8,96)= 0.55$ , n.s. A Slope Analysis was conducted to see how the Operant and Observational effect changes over increased intervals. However, unlike the previous experiments, it did not find a reliable difference between participants' Operant and Observational slopes,  $Z=1.02$ , with 8 participants returning shallower slopes, 7 steeper slopes, and 1 tie.

The results of Experiment 4 are somewhat mixed. Despite the significant main effects, the lack of both an interaction and a shallower slope for Operant intervals is at variance with the results of Experiments 1-3. At first glance it may appear that we have reached the end of an increase in the binding effect with increasing intervals. A problem with this experiment (as in Experiment 3) is that the maximum available response (2000ms in Experiment 3, 4000ms in Experiment 4) is identical to the maximum inter-event intervals (2000ms and 4000ms respectively). Given that participants' responses may have hit ceiling, I was concerned that this reduced variability may warp their estimations and account for the lack of increase in Experiment 4 (but interestingly not Experiment 3). This was compensated for in the next experiment.

## **2.1.5 Experiment 5**

This experiment was designed to remedy the response range problem outlined in the above discussion that the maximum inter-event interval was the same as the top of the response range in the two previous experiments. I re-conducted Experiment 4, but changed the participants' response range from 0-4000ms to 0-5000ms while keeping the maximum inter-event interval (4000ms) constant.

### **2.1.5.1 Method**

#### *Participants*

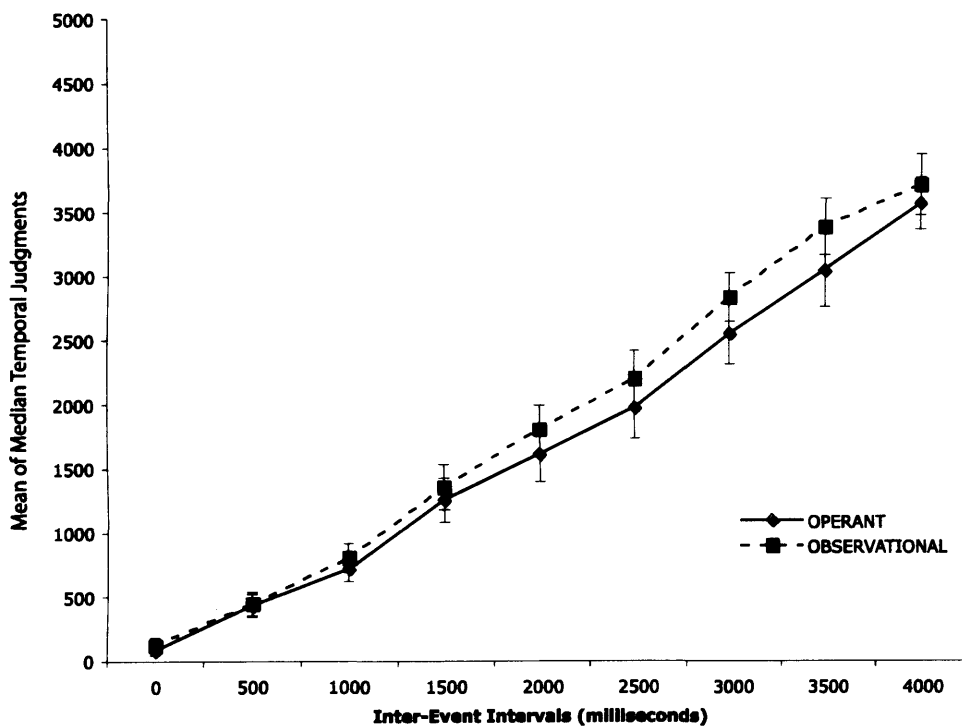
Seventeen Cardiff University Undergraduates participated for forty minutes. They received a small financial incentive (£4) in exchange for their participation.

#### *Design, Procedure and Materials*

The experimental design was identical to Experiment 4, except that the response range was increased from 0-4000ms to 0-5000ms.

### 2.1.5.2 Results and Discussion

Each participant gave 10 responses for each interval. As in Experiments 1-4, the median responses for each participant were used as the basis for further analysis, and the means of which are displayed in Figure 6. Four participants returned data points were considered outliers and removed from the dataset because they were more than 2 standard deviations from the mean.



**Figure 6.** Experiment 5. Mean Median Temporal Judgment Per Participant (Error bars show  $\pm 1$  Standard Error)

Inspection of Figure 6 suggests that Experiment 5 revealed a similar pattern of results as Experiments 1-4: Inter-event intervals in Operant trials again appear to be

considered shorter than intervals in relative Observational trials. A 2x9 ANOVA found significant effects of *Trial Type*,  $F(1,12)= 5.36$ ,  $MSE=113482.62$ , and *Interval*,  $F(8,96)= 187.037$ ,  $MSE=212799.528$ ), but no significant interaction,  $F(8,96)= 0.85$ , n.s. A Slope Analysis, unlike in Experiment 4, demonstrates a reliable difference (one tailed) between participants' Operant and Observational slopes,  $Z=1.70$ , with 11 participants returning shallower slopes, 4 steeper slopes, and 1 tie. The presence of a slope effect here suggests that the absence of a significant slope in Experiment 4 may have somehow been tied to the possible ceiling effect suggested in the previous results section.

Experiment 5 once again demonstrates a significant difference between Operant and Observational trials, which increases with increasing inter-event intervals. In Experiment 1 it was my intention to replicate the findings of earlier Temporal Binding studies with a novel method, however the surprising increase in the degree of Binding with increased inter-event interval drove me to conduct Experiments 2-5 in which I systematically increased the maximum intervals up to 4 seconds. It is tempting to keep this paradigm going by increasing the intervals even more, and perhaps at these longer intervals the Binding effect will cease. However I feel that in terms of this thesis the Magnitude Estimation paradigm has more than fulfilled its purpose and further experimentation would not be particularly informative. There is a possibility that the effects demonstrated above arise from some form of post-dictive bias inherent in a numerical estimation method, such as the “Attractor Intervals” of Wearden (2006), however I will discuss this in greater detail later. For now, given the success of the Magnitude Estimation experiments, I decided to conduct a further experiment with a similar design to Experiments 1-5, but with a novel response method. In Experiment 6 participants experienced both Operant and Observational inter-event intervals as before:

however rather than indicating the duration of the interval by means of a numerical response, participants were asked to reproduce the interval by the depression of a key.

## **2.1.6 Experiment 6**

Experiments 1-5 demonstrated a promising alternative to the Libet Clock method for the study of the Temporal Binding phenomenon at intervals far longer than I would have predicted from previous studies. As such I intended to conduct an experiment with a different method in which participants reproduced the interval between events with a button press, rather than a numerical estimation. To prevent any kind of categorization of the intervals (longest, shortest etc.) that could result in similar biases such as the above anchor point, these inter-event intervals were randomly determined between 1200ms and 1600ms (Experiment 3 demonstrated a Binding effect within this range).

### **2.1.6.1 Method**

#### *Participants*

43 Cardiff University Undergraduates participated for twenty minutes. They received course credit for their participation in this and one other study.

#### *Design, Procedure and Materials*

Experiment 6 was superficially similar to that of Experiments 1-5 in terms of materials employed: In the Operant trials Participants were presented with a Green onscreen cross and their Green button press resulted in a tone occurring after an interval, whilst in Observational trials in which participants observed a red onscreen

cross, a click noise was followed after an interval by a tone.

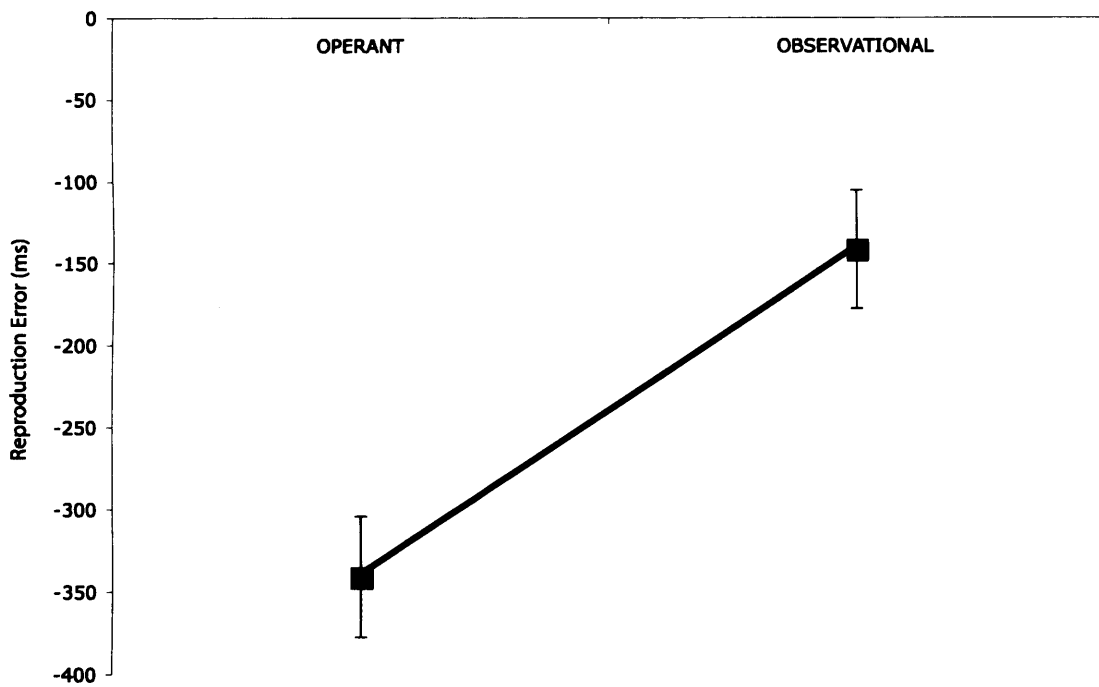
The design was changed in two significant ways. Firstly, having observed either an Operant or Observational sequence, participants did not make numerical judgments of the interval, but were rather required to depress the central (Yellow) key on the button box for a duration equal to that of the perceived interval between the events. Secondly for each trial the interval between events was randomly selected between 1200 and 1600ms. Given that the following analysis is based on the participants' accuracy shift, this design consists of only one factor, *Trial Type*, and all Operant and Observational trials were each presented in individual blocks of 30 trials each. As in previous experiments (see 2.1.1 method section) the inter-event interval in Observational trials began at the start of the "click" noise.

### **2.1.6.2 Results & Discussion**

I calculated a reproduction error by subtracting the randomly determined inter-event interval from the participant's reproduced interval for each trial. Thus with this measure a score of 0 indicates that participants accurately reproduced the duration of the random interval with their keypress on that trial. A positive number indicates that participants' reproduced interval overestimated the interval by that many milliseconds, a negative number indicating an underestimation.

Each participant's mean reproduction error was calculated and then included into the following analysis. The mean reproduction error was used in this experiment rather than the median statistic that was used in Experiments 1-5. When calculating comparisons between median data systematic errors can become apparent when the median statistics are based upon a different numbers of data points (Miller, 1988). In the previous experiments this was not an issue as each participant returned 10 numerical

responses per interval. In Experiment 6 a feature of the experimental program was that a reproduced interval would only be recorded if it occurred when prompted after the end of the second event (beep noise). This interval lasted for an indefinite amount of time and was programmed to end when the response key was released at the end of the reproduction of the interval. However in a number of trials participants appear to have depressed the response key *before* they were prompted to, and so the timing of the depression of the key was not recorded (mean of 2.7 misses per participant). Unfortunately releasing the key during the response phase would have ended it without signalling that their reproduced interval had not been recorded, and so I decided to use mean rather than median statistics for the main analysis. All mean data points falling two standard deviations outside the overall mean were excluded, resulting in 3 data point exclusions. The mean of each participant's mean reproduction error is displayed in Figure 7.



**Figure 7.** Experiment 6. Mean Reproduction Error (Error bars show  $\pm 1$  Standard Error).



Visual examination of Figure 7 indicates that although participants' reproduction of the interval in both Operant and Observational trials are shorter than the veridical random intervals, that shift is greater in the Operant condition. A paired sample T-test indicates that this difference is significant  $t(39)=4.331$ ,  $p<0.05$ . Seemingly participants further underestimate the interval when their intentional button press results in a subsequent effect, relative to when the two events are unrelated. This result appears to go a long way in supporting the previous results of the Operant/Observational paradigm by demonstrating a similar effect when participants are not required to numerically estimate an interval.

### **2.1.7 Empirical Section 1 Discussion**

The first Empirical Section has had some success in achieving its stated goal: the replication of the results of Haggard et al. (2002b) with a novel method. Experiment 1 demonstrated the shortening of the interval between two events when the second event followed a fixed interval after a button press, relative to the same interval between Observed events. Experiments 2-5 replicated these results at intervals up to 4s. Experiment 6 furthered these results with a reproduction method.

The demonstration of a binding effect at these longer intervals (Experiments 1, 2, 3 & 5) as identified by the Slope Analysis came as something of a surprise given the results of Haggard et al. (2002b) in which the amount of binding of the effect reduced with increasing intervals up to 650ms, and following this pattern we would not have expected to see a binding effect at intervals longer than this. The increase in the relative difference between conditions suggested by the Slope Analysis in Experiments 1, 2, 3 and 5 appears to be contrary to the aforementioned Haggard et al. results, suggesting an

increase of the Binding effect with increasing inter-event intervals. Further, given the problems with the Libet clock method that I outlined in the Introduction (section 1.2.1), such as inferring the relationship between events based on relative shifts of events from baseline, it may be likely that the method of directly asking participants to judge the interval is more sensitive to these Binding shifts.

Assuming that the effect demonstrated above arises from a perceptual shifting of the two events closer together in time let us consider the underlying mechanism of this effect with respect to how we internally measure the passing of time. The Scalar Expectancy Theory (SET) is a well respected model of animal timing (Gibbon, Church, & Meck, 1984) which characterizes temporal perception in terms of an internal pacemaker. In its simplest form it consists of three mechanisms. Firstly, the pacemaker is an internal construct that produces a number of pulses interspaced by an interval. Secondly, an accumulator, that receives these pulses. Thirdly, a switch mechanism with two settings: When the switch is “on”, pulses pass from the pacemaker to the accumulator, when the switch is “off”, they do not. According to this model, timing an interval consists of the switch turning from off to on at the start of the interval allowing pulses to flow into the accumulator. The sum of the clicks collected by the accumulator up to the end of the interval to be timed is used as an indication of the interval which has passed. In this model is that the clock speed is not fixed, but rather can be modulated according to certain factors such as arousal (Penton-Voak, Edwards, Percival, & Wearden, 1996), the intensity of a stimulus (Walker & Scott, 1981), and a great deal of research has been conducted to investigate the slowing up/speeding down of these internal clocks. Early examples of this demonstrated the modulation of clock speed by manipulating a participant's body temperature (Hoagland, 1933).

If we apply this model to the results of Experiments 1-5 then we could draw a

fairly straight-forward conclusion: due to some factor underlying the relationship (discussed later) between Operant action and effect, the pacemaker speed was decreased by a fixed factor resulting in a shorter perceived interval in Operant versus Observational trials. This explanation would go some way in explaining the unexpected increase in the Binding effect with increasing inter-event intervals. A fixed decrease in the firing rate of the pacemaker in the Operant condition relative to the Observational condition would result in an increase in the timed duration of both over increasing intervals, but with the difference between the two increasing with the interval. This relationship is borne out by the Slope Analysis in Experiments 1-3 and 5. Further, it is interesting to compare the general pattern of results in Figures 2-7 with the results of other studies that have aimed to demonstrate a discrepancy between the timing of two conditions based on the speeding up or slowing down of the internal clock. For example Wearden (2008, p.p. 9, Figure 2, Lower Panel) and Penton-Voak et al. (1996, p.p. 314, Figure 3, Top Panel) both demonstrate striking similarities to the above pattern of results and are explained in terms of pacemaker modulation.

Without attempting to favour an Intentional/Causal interpretation, as I discussed in the introduction, the inference of a causal relationship is postdictive in nature, which at first glance would appear to be at odds with the sped up/slowed down clock explanation outlined above, given that an increase in the clock speed would have to be triggered before the commencement of the perceived interval. Moore and Haggard (2008) however demonstrated that binding is dependent on both predictive and postdictive processes based on Bayesian predictive reliability. Eagleman and Holcombe (2002) suggested that contiguous events tend to be causally related, and shifting the two together could help cut through measurement noise and safeguard a causal impression: Working from the results of Moore and Haggard (2008), in Experiment 1-6 while an

inferential process occurring after each sequence may be clouded by the variability of the randomly selected inter-event interval, the perfect predictability of the button press → tone relationship may have allowed a predictive process (informed by the constantly updating inferential causal model) to “speed up” the pacemaker thereby reducing the number of pulses for all Operant trials. This would work to undo any error or doubt in the inference of a causal action-effect relationship by shortening the interval between the two, satisfying Hume’s (1739/1888) close temporal contiguity cue, aiding causal inference (Eagleman and Holcombe, 2002) as well as the attribution of agency (Wegner, 2002).

The clock speed modulation explanation of Temporal Binding is supported by the recent findings of Wenke and Haggard (2009). They asked participants to make active (voluntary button press) or passive (automatic lever depression that pulled down a finger) movement, which was followed after an interval (600, 800 or 1000ms) by a tone. In each trial, participants were administered two temporally separated shocks to the index finger, both presented either early in the interval (150ms after movement), late in the interval (150ms before tone), or after the presentation of the tone. The experiment consisted of two trial types: In Interval Estimation (IE) trials participants judged the interval in milliseconds between the movement and subsequent tone; In Temporal Discrimination (TD) trials participants were required to indicate whether the two index finger shocks were simultaneous or successive. The trial type, interval length and shock positions were randomized so that participants were not aware of the trial’s nature until during the trial itself.

Wenke and Haggard (2009) demonstrated that estimates of the interval between action and effect were significantly shorter than interval estimates in the passive trials. This further supports the use of numerical estimation as a paradigm for the investigation

of Temporal Binding (as demonstrated by Engbert et al., 2008), and supports the findings of Experiments 2-5 by demonstrating binding at intervals higher than 650ms. However, Wenke and Haggard (2009) did not demonstrate an interaction between interval and trial type, contrasting with the increase in difference between Operant and Observational trials over increasing intervals in Experiments 1, 2, 3 & 5. The experimenters also demonstrated that the interval between shocks required for participants to indicate that they were successive rather than simultaneous (Temporal Discrimination Threshold) was significantly larger in Active versus Passive trials when they were presented early after movement, but not when the shocks were presented late in the inter-event interval or after the presentation of the tone. This was not true in an additional experiment in which voluntary/passive movement was not followed by a subsequent tone. The authors argue that voluntary action slows down the internal pacemaker in anticipation of the resultant tone. Thus two shocks are more likely to fall between pacemaker pulses, impairing their temporal discrimination. A consistent slowing of the pacemaker speed would result in increasing differences between active and passive interval estimation with increasing interval, as well as increased temporal discrimination threshold when the shocks are presented late in the inter-event interval. Wenke and Haggard (2009) suggests that their pattern of results is consistent with a slowing of the pacemaker immediately post voluntary movement (in expectation of the tone), followed by an increase in the number of pulses produced by the pacemaker to compensate for the earlier modulation. Whilst this does not completely compensate for the original modulation of the pacemaker, resulting in the shortening of voluntary action effect intervals estimations relative to passive action effect sequences, the compensation appears to be constant, as the difference between conditions does not increase with longer intervals.

While the results of Wenke and Haggard (2009) support the notion that Temporal Binding arises from modulations in clock speed, the binding effect does not increase with increasing inter-event intervals. This is in contrast to the results of Experiments 1-3 and 5. As Wenke and Haggard (2009) point out, participants in their study were not aware of the nature of the question (numerical estimation or shock discrimination) until it was presented at the end of the trial. It may be possible then that an increase in arousal during the trial (believed to increase the number of pulses produced by the pacemaker: e.g. Penton-Voak, et al., 1996) is triggered in anticipation of the nature of the upcoming question. In this case, it may be that voluntary movement preparation or action may trigger a modulation in clock speed, which is overridden by an increase in arousal as the participant anticipates the question. A problem with this explanation is that the “arousal” does not happen until some point during the middle of the interval, and appears to provide fixed compensation for the clock speed modulation across the three interval lengths (thus no increase in Binding with increasing intervals). While it would be interesting to see the Wenke & Haggard (2009) experiments modified so that participants are aware of the nature of the trial at the very start, it currently contradicts the clock modulation explanation of Experiments 1-5.

Another possible explanation for the Temporal Binding effect demonstrated in Experiments 1-5 arises from considering the distribution of attentional resources in both Operant and Observational conditions. The Attentional Gate theory (Zakay & Block, 1997) suggests that when sharing attentional resources (which is itself finite, see: Kahneman, 1973) between temporal and non-temporal aspects of a task, the less attentional resource that is paid to the elapsed time then the shorter it appears (by narrowing the width of the attentional gate between pacemaker and accumulator and thereby “missing” some pulses). I had intended to have both Operant and Observational

trials appear identical, and superficially they were: In both trial types a click noise (recorded and resulting from motor action in Observational and Operant trials respectively) was followed by a beep. In terms of attention however, it is possible that the inclusion of a motor action in one may have driven the significant difference between the two. During Operant trials, the first event of a sequence consisted of an intended motor action. It seems likely that the processes involved in motor action planning and execution would involve an amount of attentional resource. With reference then to the Attentional Gate model, the highly salient and attention heavy motor action may have drawn upon the limited attentional resource of the participant, and thereby early on in the inter-event interval (during and immediately post motor action), the attentional gate would not be as open as in the later stages of the interval; preventing some of the generated pulse units from gathering in the accumulator. This would not be the case in the Observational trials, as the motor action is replaced by a “click” noise presented a random interval after the start of the trial. Indeed, given the previous findings of unexpected events appearing to last longer than predicted events (e.g. Pariyadath & Eagleman, 2007) it may be the case that the variable (within 1500-2000ms of start of trial) appearance of the first click event in the Observational trials may have led to them appearing to be longer than veridical, thereby extending the length of the total interval. However, I would argue that this attentional explanation only accounts for a small amount of the difference between conditions. The pattern of results found in Experiments 1-3 and 5, in which the difference between the interval judgments of both conditions increases with longer inter-event intervals, suggests a shortening of the Operant interval by a degree with a small amount of variability, indicative of the number of pulses being reduced by a slowing down of the pacemaker. As a button press in an Operant trial seems unlikely to be more salient, and thereby

draw on more attention, when the inter-event interval is longer than when it is short, any attentional focus directed towards the button press at the start of the trial would likely exert a similar amount of attentional drain across all intervals. While the attention-heavy motor action at the start of the trial may draw attention away from the interval to be timed, resulting in a number of pulses being missed by the accumulator and the inter-event interval appearing shorter, this shortening would be relatively constant for every trial, regardless of the inter-event interval length. However, experiments 1, 2, 3 and 5 demonstrate an increase in the difference between conditions, reminiscent of an arousal-based decrease in clock speed (e.g. Penton-Voak et al., 1996). It is possible that the production of an intended causal action resulted in participants becoming more aroused, and whilst the Temporal Binding of action and effect (Haggard et al., 2002b) was calculated from single event baseline, it seems likely that any change in aroused state would involve either intention or causality.

The results of experiments 1-6 could be interpreted as arising from either a modulation in pacemaker speed, or an attentional effect, in which pacemaker pulses are "missed" due to an attentional resource being focussed on elements other than the passage of time. It is difficult to say whether this demonstrates a dedicated process that shortens inter-event intervals to aid intentional/causal inference, or is a by-product of some element of motor planning. For example, in terms of the Ideomotor theory of action selection (e.g., Greenwald, 1970) in which an intentional action arises in response to an anticipation of its effect, then a dedicated Binding process may consist of a mechanism that modulates internal pacemaker speed in line with the prediction. This would result in a perceptual shortening of the action effect interval and a strengthening of the association between action and effect. This suggests a strong relationship between cognitive, motor and timing mechanisms, in which the process dealing with



intended motor action exerts a top down influence upon those of timing. Alternatively, it may be that a clock speed modulation occurs coincidentally as a by-product of motor action. For example, in terms of forward models of movement control (e.g., Wolpert, 1997) some element of the process prior to action, or in conjunction with movement itself, may modify pacemaker speed, resulting again in this perceptual shortening of action effect interval. The studies by Wenke and Haggard (2009), demonstrating a compensatory increase in clock speed in response to an initial post action slowing, are suggestive that this slowing may be coincidental (though not necessarily detrimental), as another process appears to be trying to correct it.

Whilst clock speed manipulation explanations may suggest either a planned or coincidental process, it seems somewhat unlikely that the attentional gate explanation I discussed above would result from a planned mechanism: Rather it suggest that some element of the motor-action/effect prediction is drawing upon attentional resources to operate. For example, a predictive process involved with the production of the efferent copy (e.g., Haggard, 2002a) or a causal model (e.g. Waldmann, 1996) predicting the result of an action may draw upon this attentional resource to monitor for effects contrary to the predicted outcome of the motor action, and through Bayesian processes update the prediction for the next trial. If any of the above interpretations are correct, it certainly suggests a strong relationship between motor action and timing mechanisms, so that action may have a top down effect upon timing processes, or that these cognitive processes draw upon a resource that they share with timing processes.

A recent study by Wearden, O'Rourke, Matchwick, Min and Maeers (in press) demonstrated the effects of cognitive processes (in terms of task switching) on temporal estimation. They presented participants with a tone, ranging in duration between 77 and 1183ms that they would subsequently be asked to numerically estimate.

The target tone was presented either alone, or was preceded by a numerical task (summing between 2 and 4 numbers presented in sequence) either 500ms or 2,000ms prior to the presentation of the tone. Participants were thus required to switch their attention from the numerical task to the timing task. Further, participants were split into two groups, in which the numerical task was either “easy” (addition of digits from 1-5) or “hard” (digits from 10-15). Participants were required to observe these numbers, then after an interval (500ms, or 2000ms) observe the tone, then indicate the length of the tone in milliseconds, and finally indicate whether a number presented onscreen was the correct sum of the numbers presented at the start of the trial. The experimenters found that the “easy” nontiming task presented 500ms or 2000ms before the timing task did not produce significantly different estimations relative to trials without the nontiming task. However, with the “difficult” nontiming task, participants’ numerical estimation in trials containing the nontiming task was significantly shorter than trials containing only the timing task. This was true when the nontiming task preceded the timing task by both 500ms and 2000ms, and this difference increased with increasing intervals (as measured via Slope Analysis). A second experiment, in which participants judged the duration of a visual stimulus (known to be more difficult to judge than auditory stimuli: Wearden et al. 1998) showed that mean estimates of duration were lower in the task switching trials only in the difficult condition, with an increase in this difference with increased stimulus duration. However in this experiment, the effect of the task switch was weaker in the trials in which the nontiming task preceded the stimulus by 2000ms,

Wearden et al. (in press) dismissed the possibility that their results arose due to attentional demands, suggesting that it seems unlikely that university students would have difficulty attending fully to a single stimulus less than 1s in duration when they are not required to undergo a concurrent task. Rather they explained their results in terms of

a possible link between internal timing and information processing. As they point out, previous studies demonstrate that task switching increases response times (Monsell, 2003), and that click trains (that have been shown to effect pacemaker speed: Penton-Voak et al., 1996) can effect reaction times on information processing tasks that are not usually considered to be based on time judgements (Jones & Wearden, 2008). The experimenters argued that this is suggestive of a connection between timing and cognitive processes, in that a change in the rate of one can influence the rate of the other. They even suggested that the internal clock, whilst apparently used for temporal judgements, may indeed have a more general role in controlling the speed at which other cognitive processes are performed. In terms of the Temporal Binding results of Experiments 1-5 this deep connection between information processing and timing (Wearden et al., 2009) suggests that an increase in clock speed (possibly triggered by action selection processes) could speed up the processing of online processes involved in the prediction of the effect of an action. This could mean that intentional motor processes that predict the outcome of a movement are also capable of modulating the interval between events, in effect making perceived time conform to the temporal prediction. Also, if actions arise in expectation of their sensory results (e.g., Greenwald, 1970; Prinz, 1997) then a modulation in pacemaker speed may also increase the speed at which a cognitive mechanism dealing with the representations of action and effect processes the relevant representations, possibly facilitating faster action selection. In terms of Wenke and Haggard's (2009) findings discussed earlier, the initial increased clock speed could represent the tail end of a quickened action selection process, which is compensated for by a slowing of the clock a few hundred milliseconds after movement. The fixed increase in pacemaker pulses did not seem to return the pacemaker to the speed it was after involuntary action. This could indicate that the

cognitive process involved in the action selection was still working quickly in order to process the results of the action. In that case it would seem that the internal pacemaker holds a powerful position, able to modify not only how we perceive temporal intervals, but also how we process events.

However a more problematic interpretation of the results in this Empirical Section is the possibility that the judgment data of Experiments 1-5 reflects a systematic response bias or heuristic in which participants post-perceptually re-construct the inter-event interval based on the Causal relationship between events, relative to Observational trials. In an example of a systematic shortening of the reported interval between events, Faro, Leclerc and Hastie (2005) demonstrated that participants judged the interval (in years) between pairs of causally related historical events as shorter than the interval between relative unrelated events. This occurs even though participants had not directly observed these inter event intervals, and shows that in experimental settings participants believe that causally related events are closer together in time.

It may be possible that participants in the experiments included in Empirical Section 1 evaluate the Operant inter-event interval, postdictively as shorter than relative Observational intervals purely because they understand that causal intervals tend to be shorter. This post-perceptual shortening of Operant intervals is relative to Observational intervals, and is facilitated by the fact that participants return fixed values as their judgments, and are capable of making future judgments relative to the numerical estimations they previously returned. Given that both Causal and Intentional events tend to be closer together in time (see sections 1.2.2 and 1.3.1 of introduction) it would seem logical that such a post-perceptual bias or heuristic (e.g. Tversky & Kahneman, 1974) would be adaptive for the reporting of intervals between such connected events, but does not constitute a Temporal Binding effect per-se. For example, consider a

hypothetical heuristic that is activated in the numerical consideration of the interval between causally and intentionally related events (the above Operant condition). I mentioned in the introduction that the inferred causal relationship between two events can be facilitated by a short interval between cause and effect, whilst it can be inhibited by a longer duration (e.g. Shanks, Pearson, & Dickinson, 1989). In descriptive terms, this heuristic could be summed up as “Cause and effect are close together in time.”

In terms of Experiments 1-5, imagine a participant having observed and timed the interval between events as being 300ms in duration. If the relationship between two events was deemed to be causal, this heuristic (possibly based on Hume's Close Temporal Contiguity cue: Hume, 1739/1888) prescribes that the original estimation is reduced by a factor of say 20%. This follows the general rule of thumb that “Causally related things are close together in time”. Thus, when numerically reporting the duration of a cause-effect interval, participants would enter 240ms (300ms minus 20%). As the non-causal, non-intentional sequence (the above Observational sequence) does not trigger this heuristic, the original estimation of 300ms would be reported unmodified..

Although similar to the Temporal Binding phenomenon, this heuristic-based interval shortening is drastically different. Whilst the shortening of an interval based on heuristics would consist of a purely post-dictive shortening of the *reported length* of an interval, measured numerically, Temporal Binding of the interval between events would shorten the *perceived* interval. That is to say, having observed two intervals, Operant and Observational, two participants (one “using” only a causality heuristic, the other a Temporal Binding process) would both report that in numerical terms the Operant interval was shorter than the Observational interval. However the participant employing a Temporal Binding process would have perceived the inter-event interval as shorter than veridical, whilst the heuristic based participant perceived the veridical length of the

interval, but reported it as being shorter.

It is not necessarily true however that a Binding process and response heuristics need be independent of each other. The Experiment 1-5 increase in the amount of binding over increasing inter-event intervals (as demonstrated by the Slope Analysis) is suggestive of both a modulation of the speed of an internal pacemaker which would mean that participants were perceiving the time between button press and effect as passing quicker, and also a percentage based reduction in the numerical reported interval. Further the “anchor point” suggestion that participants identify the shortest inter-event interval in both conditions and use it to base their subsequent judgements (in explanation to why there appeared to be little difference between the shortest intervals of 150ms and 750ms in both Operant and Observational conditions of Experiments 1 and 2), and the “attractor intervals” of Wearden (2006) suggest that this magnitude estimation method is prone to some degree of cognitive manipulation. Arguably should the results of Experiments 1-5 arise from a post-perceptual cognitive modulation of the reported rather than perceived interval they would still be of interest to Cognitive psychology as they demonstrate a non-perceptual form of Temporal Binding.

This does not de-value the Magnitude Estimation paradigm as a tool for the investigation of Temporal Binding. The same argument could possibly be extended to the Libet clock method: participants' verbal report of the position of a clock hand is shifted in causal conditions because of a Cognitive, rather than Perceptual shift. Therefore the results of Experiments 1-5 would still be of interest to the field of Causal Cognition as an example of how causality can modulate reported time. Further, as I described in the introduction, recent evidence suggests that Temporal Binding may in fact be driven by pre and postdictive processes (Moore & Haggard, 2008), rather than purely predictive processes (Haggard, et al., 2002a). However as a direct test of

Temporal Binding perhaps these methods may be insufficient to calculate the actual perception of the temporal presentation of events given the possible post-dictive response bias.

The Reproduction experiment was in part designed to deal with this criticism. Experiment 6 demonstrated a binding effect when participants re-produced their experienced interval that was intended to demonstrate an effect by having participants report the perceived interval rather than relying on a somewhat artificial magnitude estimation response method. However given that the measure of the inter-event interval was post-hoc and ultimately relied on an online comparison of two intervals, one of the memory of the interval the other a constantly updating reproduction, even this method cannot escape some degree of criticism given postdictive biases and the malleability of interval timing to cognitive and attentional loads (e.g. Brown, 1997). The following Empirical Section was designed to solve the problem of post-experience biases and errors by using a behavioural paradigm in which the timing of a participant's button press is used to discern the actual perceived onset of events.

## **2.2 Empirical Section 2**

In the previous Empirical Section I discussed a possibility that magnitude estimation as a method for the investigation of temporal binding may be prone to post-perceptual bias resulting from some form of response heuristic that associates causally and intentionally related events as closer together in time. Another question was whether an increase in arousal or attention arising from the participants' button press in the Operant condition lead to a decrease in pulses, or a greater number of pulses being "missed" by the attentional gate, demonstrating differing inter-event interval responses independent of any binding effect. Although the first question was addressed in part by experiment 6, in which participants did not respond through magnitude estimation but rather reproduced the inter-event interval, it did not address the second.

The following Empirical Section aimed to address both concerns with a simple over-arching design: In a two condition experiment participants made the same button-press responses in both conditions, which was used to identify the participants' perceived onset of events. Thus I introduced an online behavioural measure of subjective timings that is not prone to postdictive response heuristics, and required an equivalent attentional focus in both conditions. This allowed me to address one of the central questions of this thesis, the causal or intentional nature of the Temporal Binding phenomenon.

In this Empirical Section each experiment consisted of two conditions. In both participants made intentional button presses, which were followed by a stimulus. In terms of the experimental program, during "Causal" trials the stimulus was an effect of the participant's action, and occurred only if the participant made the intentional action. In the "Non-Causal" trials, the stimulus appeared independently of whether the participant made their intentional action or not.



Assuming that participants made normative responses in both conditions the interval between these button presses and subsequent stimuli were approximately equal in both conditions, and the presentation of the stimulus was perfectly contiguous with the participants' button press. Thus objectively, both conditions were the same: intentional action was followed after an interval by a stimulus. In this case, the causal element that I was attempting to isolate did not occur due to contingency or temporal regularity, as these would be constant across conditions (assuming the null hypothesis).

In these experiments, the causal element was not observable in terms of the relationships between events during experimental trials. Rather, "causality" was defined in terms of each participant's mental interpretation of what will, is and has happened during each trial. To develop this causal element in the mind each participant underwent a number of pre-experiment training trials in which they were encouraged to experiment with their button presses. By withholding button press they learnt the contingencies between action and effect: If button press withheld in a Non-Causal practice trial this had no effect on stimulus presentation (no contingency), whilst the stimulus would not occur in the absence of button press in Causal trials (perfect contingency). By making their button presses earlier or later participants observed that it would increase/decrease the interval between action and effect in Non-Causal practice trials, but have no effect on temporal regularity in Causal practice trials. Further, the causal relationship was explicitly verbalized to the participants in the instructions prior to the training trials.

Whilst both Causal and Non-Causal experimental trials were objectively the same (button press followed by presentation of a stimulus) they varied subjectively in terms of the "causal" interpretations held in the mind of the participant from the prior training trials (experimental trials were blocked according to the causal relationship, each preceded by numerous practice trials representative of the experimental trials).

Thus the experimental trials of both conditions contained intentional action (a button press). However, only in the Causal condition did the participants hold in their mind a belief that their action and the stimulus presentation were causally related. In this way, the following experiments disassociated intentional action and causality.

### 2.2.1 Experiment 7

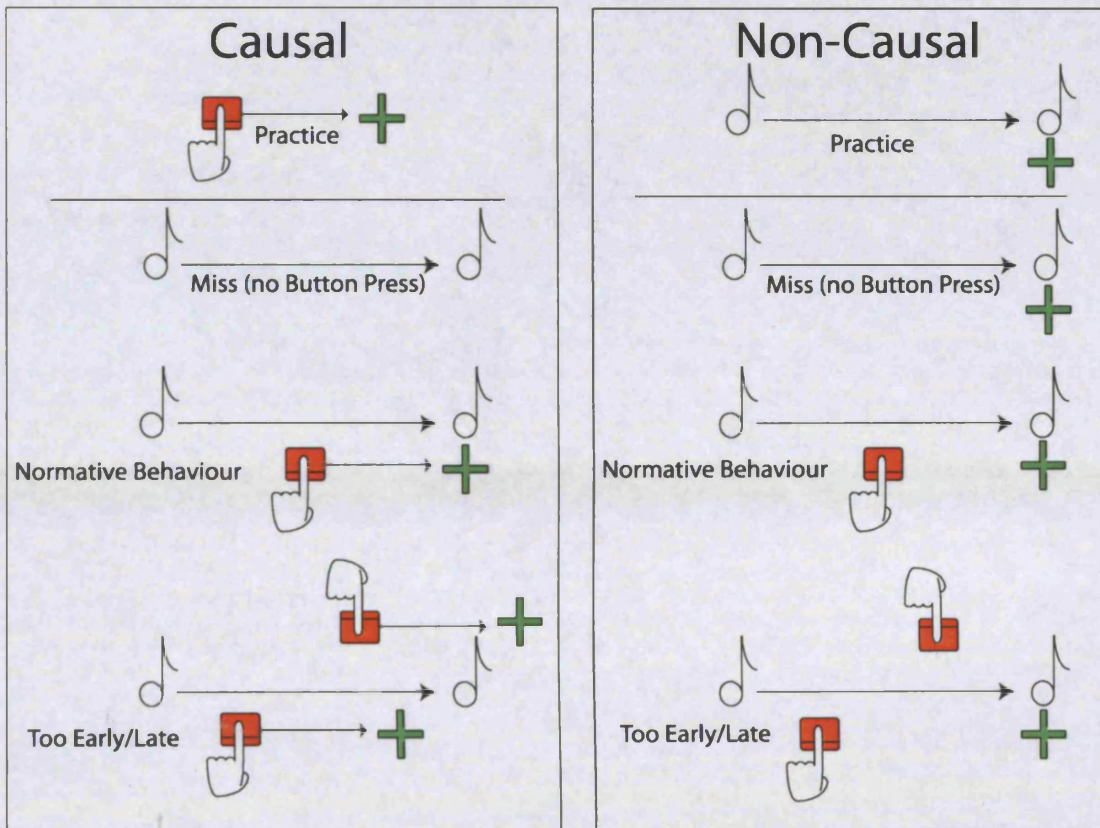
In Experiment 7 I employed a paradigm in which participants were required to bisect the interval between two tones. Waszak, Wascher, Keller, Koch, Aschersleben, Rosenbaum & Prinz (2005) used an interval bisection paradigm to investigate neural mechanisms in two modes of action selection: *Intentional-based* action in which, following Ideomotor theory (e.g. Greenwald, 1970), action occurs in expectation of their sensory effect, and *stimulus-based* action, in which participants make movements in response to a cue. Waszak et al. (2005) presented participants with a continuous series of visual stimuli separated by a fixed inter-stimulus interval of 1200ms. Visual cues appeared on-screen corresponding to one of two keys. In the Intentional-based condition, participants were required to bisect the interval with any key they wished, and the keypress would correspond to the position of the subsequent visual stimulus. In a second, Stimulus-based condition, participants were instructed to bisect the interval by pressing the key corresponding to the position of the last visual stimulus they observed. Actions in a Stimulus-based run were yolked to a previous Intention-based run, allowing comparison between the two runs in terms of action timing and EEG profile.

Waszak et al (2005) observed a Temporal Binding effect, as expected from Haggard et al (2002a), in their behavioural data: Stimulus-based responses were on average earlier than Intentional-based responses (~50ms), suggesting a binding of the intended motor action towards its effect, relative to the stimulus based action, which

was closer to the Stimulus. These results were supported by a later experiment (Keller, Wascher, Prinz, Waszak, Koch, & Rosenbaum, 2006), in which the left/right keypress was not mapped to the position of the stimulus onscreen, but was rather mapped to an arbitrary letter (to further the results of Waszak et al, (2005), with responses that were not spatially tied to the stimuli). They again found a Temporal Binding of Intentional-based action to effect, relative to the Stimulus-based responses, with these responses apparently bound to the stimulus to which they responded. Further, in terms of their EEG recordings, both studies demonstrated a marked difference in the neural preparatory signals preceding action, reflecting stimulus processing and response preparation in the Stimulus based trials, compared to activity indicative of a readiness to act in the Intention-based trials. The authors of both studies argued that their findings are suggestive of two different modes of action planning: One that results in a binding of responses to stimulus, another that involves the binding of action to its subsequent effect. These studies provide support for the idea that binding is a strengthening of the relationship between action and the perception of its effect, which are similarly coded (Prinz, 1997).

Experiment 7 is somewhat similar to the studies of Waszak et al. (2005) and Keller et al. (2006). In this experiment (and all following in this section) the terminology was changed from the previous Empirical Section to reflect the differing nature of the task. In Experiment 7, participants experienced the interval between two tones: one signifying the start of the trial, the other a target. During Causal trials pressing a button caused a visual stimulus to appear onscreen after a set interval. Participants were instructed to press the button at such a time so that the visual stimulus appeared in synchrony with the second, target tone. The interval between button press and effect (250ms, 450ms, 650ms) was exactly half the interval between the start of

both tones (500ms, 900ms, 1300ms). Thus a normative response required the bisection of the inter-event interval. In the Non-Causal condition participants experienced the same two tones present in the Causal condition. However, participants were required to press a button directly between the two tones, with the visual stimuli appearing simultaneously with the second tone independent of the participant's response (see Figure 8).



**Figure 8.** Experiment 7 Comparison of Causal and Non-Causal trials.

The inter-tone intervals were equal in both conditions (500ms, 900ms & 1300ms). However, the interval between causal action and visual stimulus in the Causal condition was exactly half the inter-tone interval (250ms, 450ms, and 650ms respectively) and so Causal trials were analogous in their objectives to Non-Causal trials as accurate bisection would result in perfect synchronization of visual stimulus

and target tone. In contrast, during the Non-Causal trials the participants' responses had no effect on the presentation of the stimuli. Further, whilst the previous experiment of this thesis examined binding in terms of both causality and intention, this experiment provided a distinction between them: Both conditions involve intentional actions (button presses) however only in the Causal condition did this action cause an effect.

Whilst an intentionality based interpretation of Temporal Binding would predict binding in both conditions, I hypothesized that binding would only occur in the Causal condition, with participant responses in the Non-Causal condition remaining fairly accurate. More specifically, I hypothesized that due to binding arising from the causal impressions learnt in the training phase, participants would believe that the interval between their action and its effect was shorter than veridical. Thus participants' underestimation of the interval between their action and its effect would lead them to make a response *later* than accurate during the experimental phase. Whilst this predicts a similar pattern to the results found by Keller, et al. (2006) in terms of intention-based and stimulus-based interval bisection, in this experiment the motor action in Non-Causal trials is not prescribed by the preceding stimulus (i.e. differing stimuli indicate differing buttons to press). Therefore I argue that the Non-Causal button press is a voluntary action that does not result in an effect. In that sense is comparable with the Causal condition as both involve two intentional actions. However in the Causal condition participants understand the relationship between action and effect as "causal".

### **2.2.1.1 Method**

#### *Participants*

Twenty-eight Cardiff University undergraduates with a mean age of 22 (17 female) participated in the following study. 16 participated for half an hour in exchange

for course credit, the remainder for £3.

### *Design and Procedure*

Participants were informed that they would be participating in a study of time perception. An experimental run consisted of undergoing two levels of the factor *Trial Type*, Non-Causal and Causal, presented in alternating blocks. Following a general outline of the study, at the beginning of each block participants were presented with a brief overview of the following trials. Each block was split into two sections: a training section followed by an experimental section. Before a block consisting entirely of Causal trials, participants were informed that pressing a button would cause a green cross to appear onscreen after set interval. They were instructed that having read the instructions they would be presented with a blank screen. Their task in this training period was to press the button at-will to cause the cross to appear. Their task was to learn the interval between cause and effect. The interval was 250ms, 450ms or 650ms in length, and remained constant throughout both practice and experimental trial. If they did not press the button, nothing would happen. It was suggested they try not pressing the button for a while to emphasize the causal nature of their actions.

The practice session lasted for 10 button press-effect sequences. Following this participants were presented with further instructions explaining that each experimental trial consisted of two different tones, a start tone and a target tone, separated by a set interval. The interval between the beginnings of each tone was exactly double the interval between button press and effect (500ms, 900ms, 1300ms). Participants were required to press the button at such a time that the resulting cross would synchronize with the target tone. If they failed to press a button before the end of the second tone the trial would be counted as a miss and a message informing them of this would be

presented onscreen.

Instructions presented at the beginning of Non-Causal blocks emphasized that in the subsequent training trials participants were not to make any inputs and merely experience 10 sequences of start tones followed after a set interval by a simultaneous tone and green cross pairing. They were instructed to learn the interval between the stimuli (500ms, 900ms, 1300ms, as in the Causal condition, kept constant for both practice and experimental trials), and use this knowledge in the following experimental trials to bisect the interval.

Non-Causal and Causal trials were blocked so that the inter-stimulus interval in each trial remained constant. Participants experienced one block of each inter-stimulus duration for the factor *Interval* (500ms, 900ms or 1300ms) in both Non-Causal and Causal conditions. The nature of the first group was counterbalanced. The timing of each button press in the Non-Causal and Causal conditions served as the Dependent Variable. Each block consisted of 30 trials resulting in a total of 180 experimental trials.

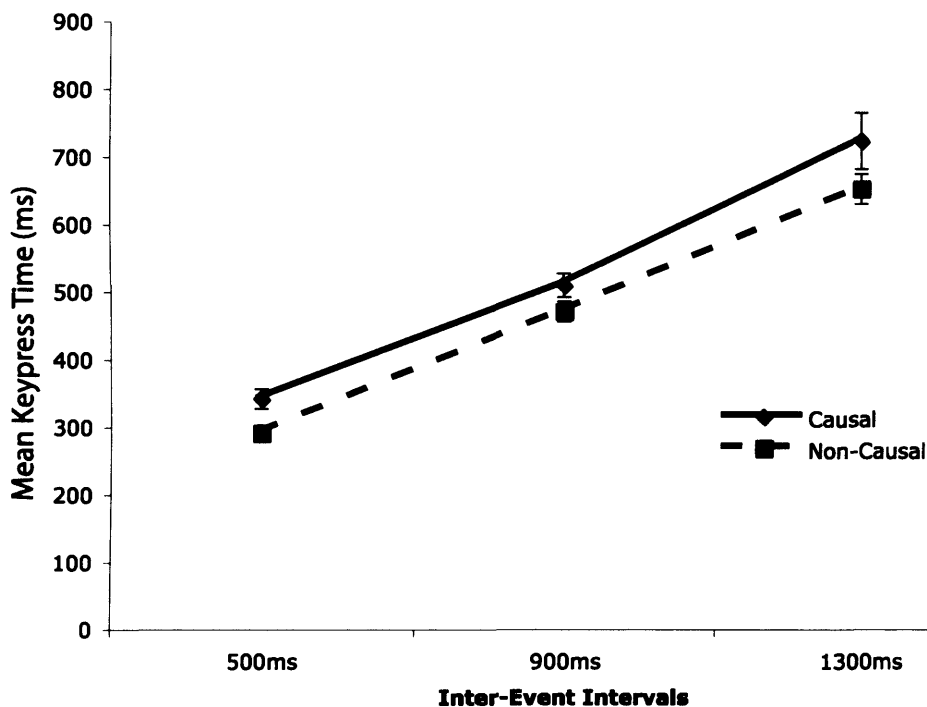
### *Materials and Apparatus*

Experimental runs were designed and presented on iMacs running Psyscope (Cohen, MacWhinney, Flatt & Provost, 1993) experimental design software, connected to a button box for participant input and timing. Both tones presented in experimental trials consisted of 100ms pure tones, the first with a frequency of 600MHz, the second 1 kHz. The visual stimulus was a small green cross, 8cm length, presented at the centre of the screen for 100ms.

### **2.2.1.2 Results & Discussion**

In the Causal conditions Mean keypress times (presented in Figure 9.) appeared

to be later than in Non-causal conditions. A repeated measure ANOVA of these individual means showed a significant main effect of *Trial Type* ( $F(1,27)=8.94$ ,  $MSE=121087.16$ ), and as one would expect given the task a significant effect of *Interval* ( $F(2,54)=202.52$   $MSE=1960000$ ). There was no significant interaction effect ( $F(2, 54)=0.396$ ,  $MSE=3787.08$ , n.s).



**Figure 9.** Experiment 7. Mean Keypress Time in Non-Causal and Causal trials (error bars show  $\pm 1$  Standard Error)

At first glance the results of Experiment 7 appear to support the hypothesis that a causal relationship, rather than intentionality, is the basis of Temporal Binding, as proposed by Eagleman and Holcombe (2002). Participants' causal button presses (which caused the appearance of a green cross) were consistently later than non-causal button presses. I had predicted that this would occur given that during the pre



experimental phase participants would have learnt the interval between their button press and effect was shorter than veridical due to binding in the Causal condition. This reduced interval between action and effect would lead to participants requiring that their button press occurred later than veridical to ensure the synchronization of the effect (green cross) with the second tone. This is what we observe in Experiment 7, in which participants' button presses appear later in Causal versus Non-Causal trials. Since the two tasks were superficially identical, both involving intentional action, it seems possible that the discrepancy arose from Temporal Binding of action and effect in pre-trial training and so button presses occurred later than in Non-Causal trials.

In the experiment's design stage I had considered the possibility that participants would use the discrepancy between visual and auditory stimuli in the causal conditions to correct for the lateness of their button presses. Given the results of Haggard et al. (2002a) in which the second event (the effect) was shifted towards the first, it does seem likely that although the green cross appeared later than the tone, a binding of cause and effect might shift the perceived onset of the green cross towards the button press. It is therefore possible that participants were not perceptually aware that the onset of the green cross when they caused it was later than veridical, rather they may have believed that they were synchronizing the two.

This experiment demonstrates a behavioural method for the investigation of Temporal Binding rather than relying on postdictive reports as in the Libet clock paradigm and the experiments outlined in the previous Empirical Section. However, although objectively the Non-Causal and Causal tasks' normative responses (bisection of the inter-event interval) were identical, subjectively the experimental goals were very different. Arguably the Non-Causal trials consisted of solely the bisection of the interval between two events without reference to extraneous stimuli such as the green cross (a

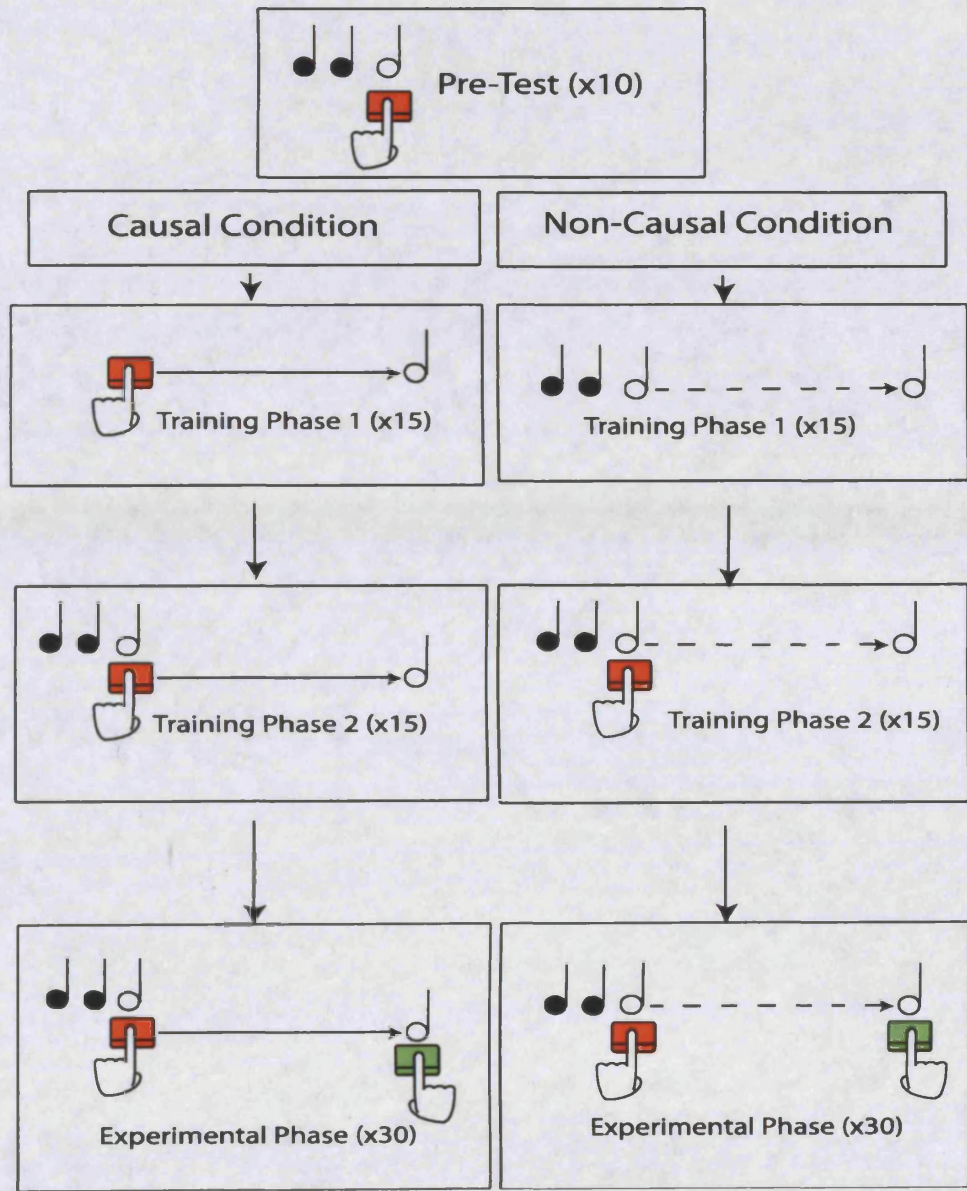
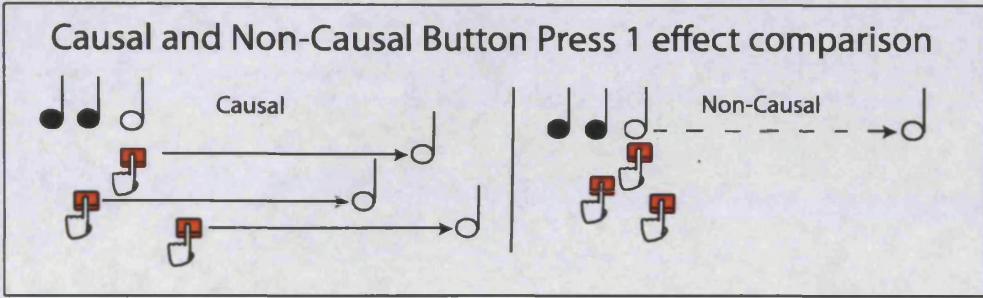
“true” bisection of the interval). This is perhaps not the case in the Causal task, where the participants are required to make their timed response in relation to the external green cross stimulus whilst simultaneously attending to the passed/remaining inter/tone interval. Arguably, the Causal task, requiring the synchronization of the caused visual stimulus (green cross) with the second tone required higher cognitive and attentional load than the true bisection of an interval. The following two experiments attempted to rectify this.

### **2.2.2 Experiment 8**

In this experiment I presented participants with 2 conditions: In the Non-Causal condition, after a sequence of preparatory tones, participants experienced a 100ms, 1kHz target tone, which was followed after an interval of 500, 900 or 1300ms, by a second 100ms 1kHz target tone. In this sequence, participants were required to make a button press in synchrony with the first target tone, and another button press in synchrony with the second. In this Non-Causal condition the participants paced the two tones. In the Causal condition participants were again presented with the preparatory signals followed by the first target tone to which they were required to synchronize a button press. However, in this condition, a second target tone did not automatically appear. Rather the second tone appeared a fixed interval (500, 900 or 1300ms) after the first button press, and participants were required to once again make a second button press in synchrony to the second target tone.

Normative responses in the Causal condition (i.e. accurate synchrony of first button press with the first target tone) should result in participants performing identical tasks in both Non-Causal and Causal conditions when the intervals between Non-Causal tones and between Causal button-press and resultant tone are the same. As such I made

the following prediction from the results of Haggard et al. (2002a): Based on prior understanding of the relationship between button press and subsequent tone in the Causal condition gained in a training phase (where they learn the button press → effect sequence), participants should experience both Action binding of tone towards their action, and Tone binding of their action towards the tone. Thus their second button press should appear earlier in the Causal condition, relative to their second button press in the Non-Causal condition, as in the latter there is no Intentional/Causal relationship between the participant's button press and second tone. Further, given the results of Haggard et al. (2002a), in which their Intended/Causal button press was shifted towards the resultant tone relative to baselines, I also predicted that participants' first button press would also be shifted towards the resultant tone (i.e. Occur later in Causal relative to the Non-causal conditions).



**Figure 10.** Experiment 8: Graphical Comparison of Conditions.

### 2.2.2.1 Method

#### *Participants*

34 Cardiff University Undergraduates (28 female) participated in return for course credit, or £4.

#### *Design and Procedure*

Participants experienced both levels of the factor *Causality*: Non-Causal and Causal. Experimental trials in both conditions were designed to be identical in the case of participant action: Following a sequence of preparatory tones (P1 and P2), participants would observe first target tone (T1), which would be followed after a short interval by a second target tone (T2). Participants were required to make one button (BP1) press in synchrony with T1, and another button press (BP2) in synchrony with T2. This pattern of responses was required for both Non-Causal and Causal experimental conditions, however the experimental manipulation consisted of ensuring that in the Causal condition T2 appeared a fixed interval after BP1, whilst in the Non-Causal condition the second T2 was fixed to T1 (independent of BP1). The dependent variables consisted of the temporal position of BP1 and BP2 relative to T1 and T2, and the inter event interval between button presses. Both BP1 and T2 in Causal and T1 and T2 in Non-Causal were temporally separated by the factor *Interval* with three levels (500ms, 900ms, 1300ms).

As each experimental trial began with the sequence of preparatory tones prior to T1, participants were trained at the start of each experimental run in synchronizing their BP1 with T1. They experienced 10 trials in which two lower frequency tones (P1 and P2) were followed by T1. In each trial participants were instructed to synchronize BP1 with T1. In this pre-experimental phase T2 was never presented.

All levels of the factor interval were grouped together into two blocks and the order of presentation counterbalanced. For each interval participants underwent two training phases prior to the experimental phase trials. In the Non-Causal conditions participants firstly observed a complete sequence of tones (P1, P2, T1 and T2), presented 15 times. They were not required to make any button presses at this stage, but rather learn the interval between T1 and T2. In the second training phase participants again observed the complete sequence of tones, and were required to BP1 in synchrony with T1, for 15 trials. They then underwent a sequence of 30 Experimental trials making both BP1 and BP2 to T1 and T2 respectively.

Prior to each block of Causal experimental trials participants first underwent a free-operant training phase in which no preparatory tones were present. They were requested to press a button (BP1) at the time of their choosing and cause a tone (T2) to appear after a fixed interval. They did this for 15 practice trials, learning the interval between each operant button press and subsequent effect and the causal relationship. In the second Training phase, participants observed the two preparatory tones (P1 and P2) and T1, and were then required to synchronize BP1 with T1, continuing for 15 trials and thus produce T2 after the relevant interval. They were however encouraged to withhold BP1 on occasion, to demonstrate that T2 would not occur unless the participant made BP1. Finally they underwent 30 experimental trials, in which they were required to synchronize BP1 with T1, and BP2 with T2. Throughout the experimental trials, the timing of participants' BP1 and BP2 was recorded. From this I could calculate the relationships between BP1 and T1, and BP2 and T2.

### *Materials and Apparatus*

Experiments were designed and conducted on iMacs running Psyscope (Cohen,

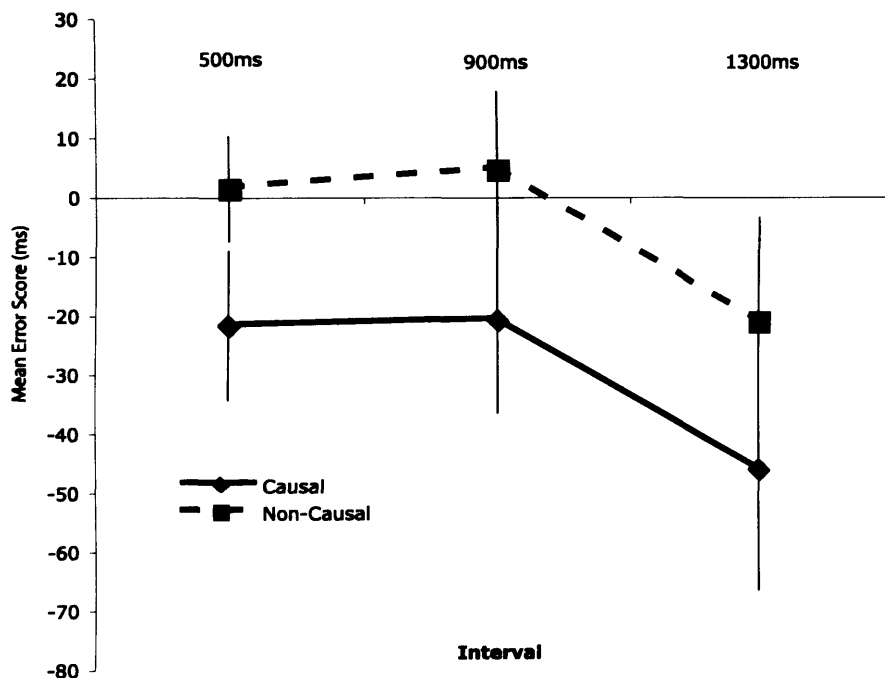
Macwhinney, Flatt, & Provost, 1993), connected to a Psyscope Button Box for participant input and response timing. Preparatory tones consisted of two 100ms 600Hz pure tones (P1 and P2) inter-spaced by 200ms. T1, a 100ms 1kHz pure tone was presented after another 100ms delay. Participants' responses were made on the Button Box, BP1 on the leftmost (red coloured button), BP2 with the rightmost (green button). In Non-Causal conditions, the interval between T1 and T2 was either 500ms, 900ms and 1300ms, and these intervals were used for the interval between BP1 and T2 in the Causal condition.

In Experiment 1 I suggested that when participants observed the relationship between a Non-causal sequence of events (the “click” noise and subsequent tone: see section 2.1.1.1), if the experimental program's timing of the inter-event interval commenced at the end of the ~120ms click noise, and participant's timing of the interval commence at the start of this noise, then the observed interval would be ~120ms longer than the same Operant interval. Therefore all observed intervals in Experiment 1-6 began at the start of the first non-causal event (“click noise”). In the Non-Causal condition of Experiment 8 I also elected to have the experimental program time the inter-event interval from the beginning of the first target tone (T1). This was again a somewhat conservative strategy as the interval between Targets 1 and 2 would likely be shorter than veridical should participants begin timing at any time *during* T1, rather than at the very beginning of it.

### **2.2.2.2 Results & Discussion**

Previously I hypothesized that participants' BP2 would appear earlier in Causal than in Non-Causal trials, whilst BP1 would appear later. This was in response to the findings of Haggard, et al. (2002a), who demonstrated both action binding of tone

towards button press, and effect binding of the onset of button press towards tone. I examined the mean error scores for BP1 and BP2 relative to their respective target tones (subtraction of target timing from button press timing resulted in a score in which a negative value indicates BP too early, positive later, than the start of the Target) for each Interval x Causality condition. Participant's BP2 appears to have occurred earlier in Causal trials relative to B2 in Non-Causal trials (see Figure 11). The effect of *Trial Type* was significant,  $F(1,33) = 11.22$ ,  $MSE=3812.31$ ,  $p<0.05$ . However neither the effect of *Interval*,  $F(2,66)=2.39$ ,  $MSE=7271.16$  n.s., nor the interaction,  $F(2,66)=.31$ ,  $MSE=2619.89$ , attained significance.

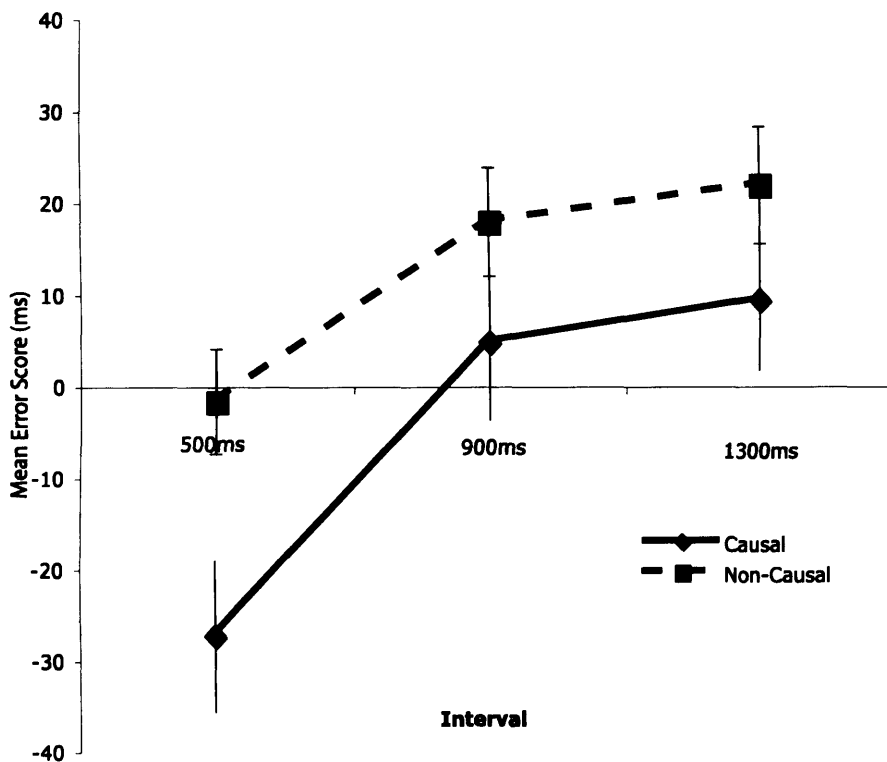


**Figure 11.** Experiment 8. Mean BP2 Error Score Relative to T2 (Error Bars Show  $\pm 1$  Standard Error).

So far the pattern of results supported my hypothesis. This action binding was predicted given the results of Haggard, et al. (2002a): the effect appears to be bound towards the button press. Of note however, this only occurs when the intentional action



causes the second target tone, and not when intentional action is independent of T2. However, examining the timing of BP 1 relative to T1 shows that participants' error score for BP1 across Intervals appears to be *earlier* in the Causal condition than the relative Non-Causal condition (See Figure 12). An ANOVA suggests that the effect of the Causal condition is significant,  $F(1,33)=14.35$ ,  $MSE=1044.99$ ,  $p<0.05$ . The effect of Interval was also significant,  $F(2,66)=17.85$ ,  $MSE=1017.20$ ,  $p<0.05$ , however the interaction was not,  $F(2,66)=1.26$ ,  $MSE=743.41$ , n.s..



**Figure 12.** Experiment 8. Mean BP1 Error Score Relative to T1 (Error Bars Show  $\pm 1$  Standard Error).

At first glance the results of Experiment 8 appear confusing and to contradict my original hypothesis: that BP1 would be shifted away from T1 towards BP2, while BP2 would be shifted away from T2 towards BP1, in the Causal conditions. While BP2 was shifted earlier, towards T1, the finding of a shift in BP1 occurring earlier than T1

(relative to Non-Causal trials) was surprising. Haggard, et al. (2002a) demonstrated both action binding (shift of tone towards intended motor action) and tone binding (shift of action towards tone). However with consideration I suggest that it provides strong evidence for a tone-binding shift in the perceived onset of the participants' action. Firstly, when participants' motor action was in anticipation of a target tone that they had caused (via a previously executed action), their response occurred earlier than motor action to stimuli they had not caused. This anticipation suggests that when participants caused the event, they experienced an earlier subjective awareness of the event. This corresponds to the action binding shown by Haggard, et al. (2002a), in which the participants indicated that the effect caused by an intended motor action occurred earlier relative to a single event baseline condition.

Of particular interest was the finding that BP1 appeared earlier in Causal relative to Non-Causal trials assuming that a Temporal Binding between Cause (BP1) and effect (T2) resulted in participants perceiving BP1 as later, and T2 as earlier (which the positioning of BP2 suggests they did), then had participants made accurate responses to T1, BP1 would have appeared subjectively *later* than veridical to the participants. In effect they would have been aware that they were pressing too late, and possibly also aware that by pressing late, the interval between T1 and T2 was longer than veridical (which would be contrary to both task demands and the notion of Temporal Binding). An earlier BP1 suggests that they modified their behaviour so that they were subjectively making BP1 in conjunction with T1.

Having demonstrated both tone and action shift (the two elements of Temporal Binding Demonstrated by Haggard et al., 2002) in the presses of Causal versus Non-Causal trials I directly compared the shifts in both button presses. I conducted a 2x2x3 ANOVA with the factors *Causality* (2), *Button Press* (2) and *Interval* (3), and found a

significant effect of Causality,  $F(1,33)=10.75$ ,  $MSE=4126.43$ ,  $p<0.05$ , and Button Press,  $F(1,33)=5.15$ ,  $MSE=9189.76$ ,  $p<0.05$ , but no effect of Interval,  $F(2,66)=1.83$ ,  $MSE=3937.8$ , n.s.. There were no significant interactions between Causality and Button Press,  $F(1,33)=0.51$ ,  $MSE=2755.23$ , n.s., nor between Causality and Interval,  $F(2,66)=0.155$ ,  $MSE=2110.88$ , n.s.. There was however a significant interaction between the factors Button Press and Interval,  $F(2,66)=6.11$ ,  $MSE=4028.18$ ,  $p<0.05$ . Finally, there was no Causality x Button Press x Interval interaction,  $F(2,66)=0.38$ ,  $MSE=1655.74$  n.s..

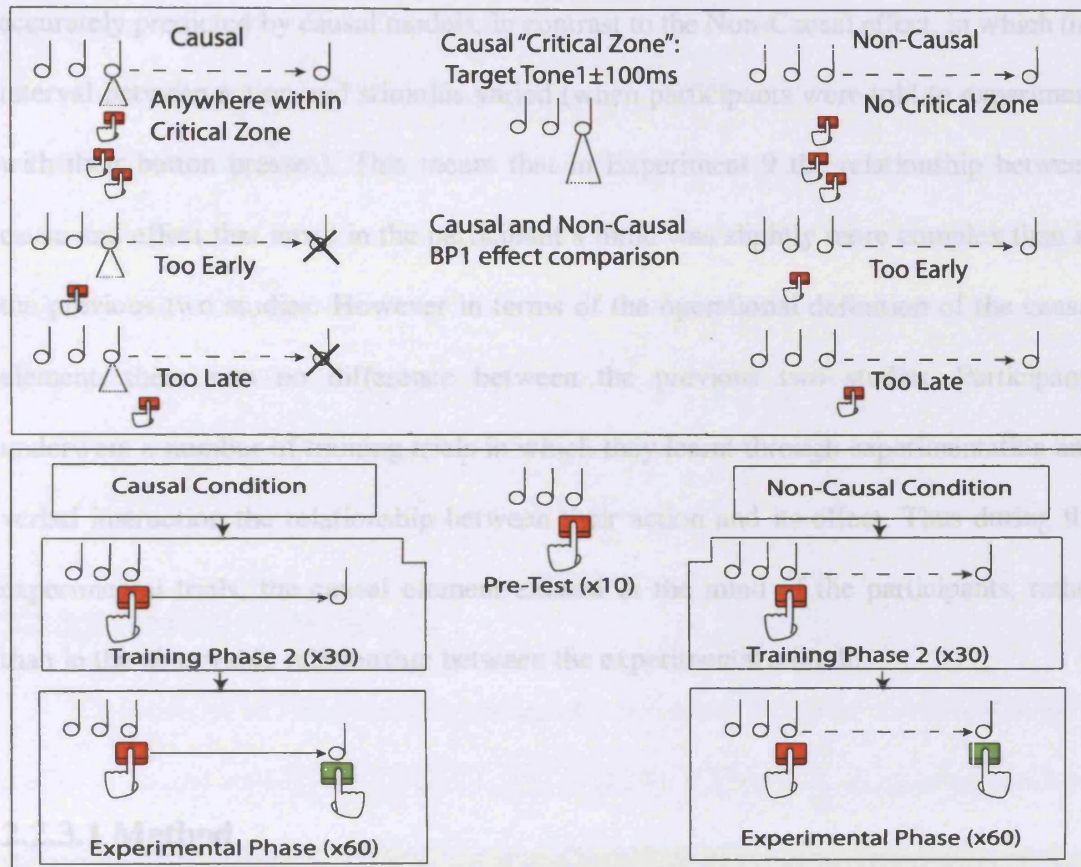
Experiment 8 provides good evidence that for a Temporal Binding of two events to occur, the two must be causally related. Whilst both conditions required participants to make intentional actions, only in the Causal condition did participants interpret their action as having caused the subsequent tone (based on experience from the training trials). The pattern of results is suggestive of the two types of binding (action binding and tone binding) shown by Haggard, et al. (2002a). Participants perceived the onset of their causal action as later than veridical, and thus learnt to initiate it earlier than veridical to subjectively make BP1 and T1 coincide. Further the shift in BP2 occurring earlier in the Causal condition suggests that participants perceived the effect of the causal action as occurring earlier, and were not aware that their button press was not also on target.

### **2.2.3 Experiment 9**

Having conducted the previous Experiment I became aware of a possible criticism of the methodology: In the Causal condition the interval between participants' BP1 and subsequent T2 was constant within each block as T2 was fixed to BP1. This would mean that the interval between BP1 and T2 was highly predictable in Causal

conditions. However, this was not the case in the Non-Causal conditions, as T2 was independent of BP1 (given that BP1 did not result in a caused effect), and was rather tied to T1. The participants' experience of the relationship between BP1 and T2 in Non-Causal conditions would have been more variable than in the Causal conditions.

It is possible that the lack of variability between BP1 and T2 in Causal trials could lead to a stronger association between the two, relative to the more variable BP1-T2 relationship in the Non-Causal trials. As it has been previously suggested that Binding arises from such action-outcome sequences (Haggard, et al., 2002a), then this variability problem could suggest that the above results arise from an association between the less variable Causal BP1-T2 sequence, rather than higher level Causal conceptualizations. To counteract this I modified the experiment so that in the Causal condition, T2 would always appear a fixed interval after T1. To keep the causal relationship between BP1 and T2 we included a "Critical Zone" of 300ms, centred around T1, within which a button press would 'cause' T2 to appear. Should participants press outside this Zone (or not at all) then the presentation of T2 would be cancelled. Participants were encouraged during training to instigate BP1 out of synchrony with T1 to see that T2 would not occur (see Figure 13 for graphical representation). The experiment was further modified by removing the first set of practice trials in both conditions (the free operant button presses and the purely observational trials in Causal and Non-Causal blocks respectively), to counter any possible criticisms that in participants in one condition received more exposure to the preparatory tones (from the Non-Causal observational phase), or made a greater number of button presses (from the free-operant Causal phase).



**Figure 13.** Experiment 9. Comparison of Causal and Non-Causal Conditions

Previously, I defined the causal element that varied between conditions as a mental understanding (gained from participant experimentation in numerous practice trials and verbal instruction) of the relationship between intended action and subsequent effect that could be utilized (perhaps in terms of causal models) to predict and comprehend the events in a trial. In Experiment 9, there was no temporal regularity between intentional action and subsequent effect, since as long as BP1 fell within the 300ms critical zone the second tone appeared a fixed interval after the first. Previously this temporal regularity was one of the ways in which participants developed their causal interpretations of the action effect relationships that would be used in the experimental trials, as during training trials it meant that the onset of the effect could be

accurately predicted by causal models, in contrast to the Non-Causal effect, in which the interval between action and stimulus varied (when participants were told to experiment with their button presses). This meant that in Experiment 9 the relationship between cause and effect that arose in the participant's mind was slightly more complex than in the previous two studies. However in terms of the operational definition of the causal element, there was no difference between the previous two studies. Participants underwent a number of training trials in which they learnt through experimentation and verbal instruction the relationship between their action and its effect. Thus during the experimental trials, the causal element existed in the mind of the participants, rather than in the observable relationship between the experimental stimuli.

### **2.2.3.1 Method**

#### *Participants*

35 Cardiff University Undergraduates (29 female) participated in return for course credit, or £4.

#### *Materials, Design & Procedure*

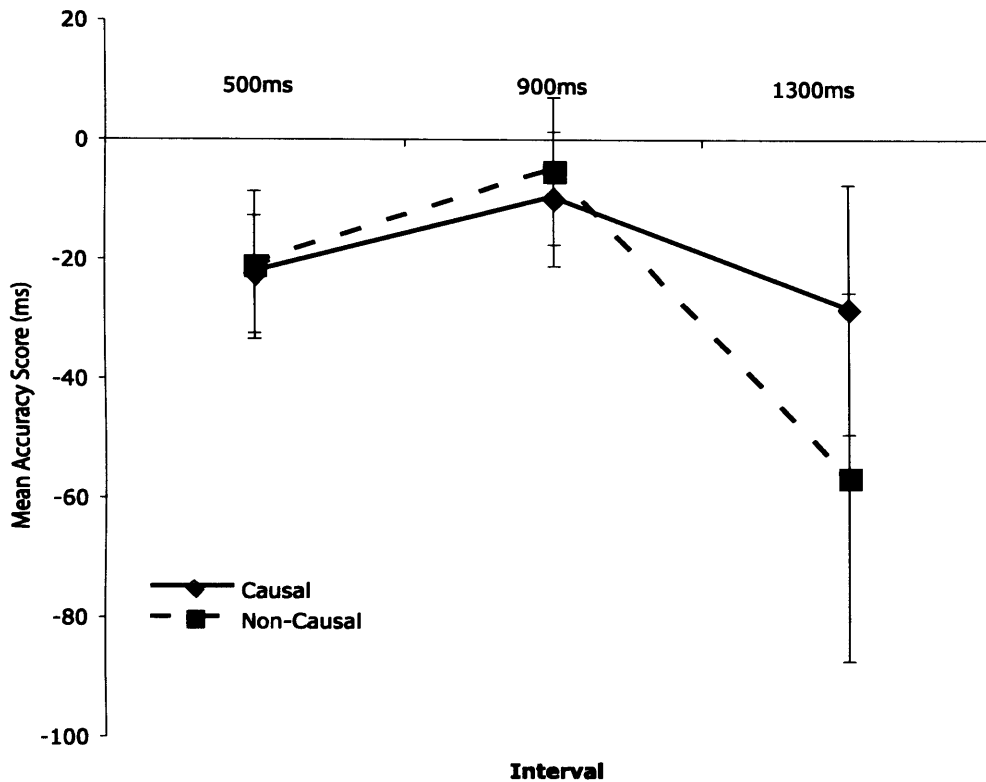
All intervals, materials etc. were kept constant from the previous experiment, with the following changes: The first set of practice trials were removed from both Non-Causal and Causal conditions. These were respectively the trials in which participants observed the full event sequence (making no button presses), and the free operant trials in which the participants made BP1 to cause T2, in the absence of other stimuli. I increased the number of trials to 30 in the second training phase (in which participants observe P1, P2, and T1, and are required to time BP1 in accordance with T1, in both Non-Causal and Causal conditions). There was also an increase in the number of

experimental trials per block, from 30 to 60.

In all Causal trials T2 was linked to the start of T1, so that it would appear after a fixed interval, as it did in the Non-Causal trials. However, to conserve the causal relationship between BP1 and T2, I included a “Critical Zone” around T1 that consisted of an interval 100ms before and 100ms after T1. Making a BP1 response outside of this 300ms zone would cancel the presentation of T2. In effect BP1 still caused T2 to appear, provided that BP1 occurred inside the Critical Zone.

### **2.2.3.2 Results & Discussion**

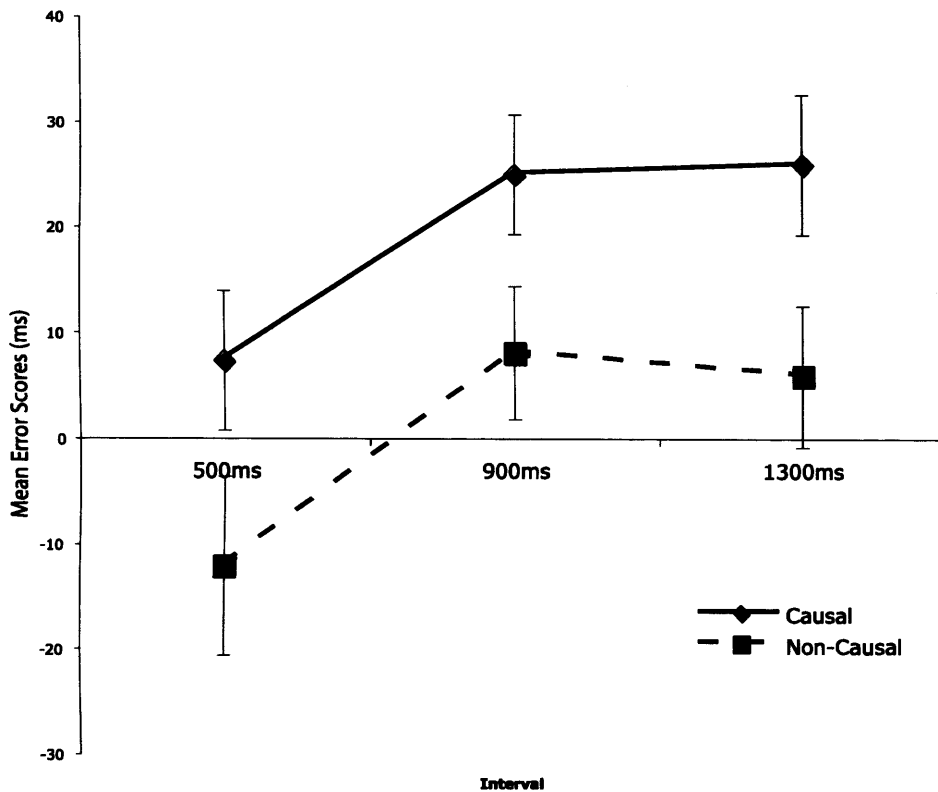
In contrast to Experiment 8 participants no longer to appear to expect Target 2 earlier in Causal versus Non-Causal Trials, as shown in Figure 14. A repeated measures ANOVA demonstrated that there was no significant effect of condition, interval nor an interaction,  $F(1,33)=.46$ ,  $MSE=5666.53$ , n.s. This was in contrast to the previous experiment and somewhat unexpected. At first glance it appears as though the association effect mentioned above may have given rise to the effects shown in Experiment 8, and its removal appears to lead participants to accurately anticipate T2. However the relationship between BP1 and T1 was even more curious.



**Figure 14.** Experiment 9. Mean BP2 Error Score Relative to T2 (Error Bars Show  $\pm 1$  Standard Error).

In contrast to the relationship between T1 and BP1 in Experiment 8, here it seems that participants' timing of BP1 is *later* than that of T1 (see Figure 15) in Causal relative to Non-Causal conditions. A repeated measures ANOVA shows a significant effect of Condition,  $F(1, 34)=16.46$ ,  $MSE=921.36$ ,  $p<0.05$ , and a main effect of Interval,  $F(2,68)=15.11$ ,  $MSE=490.38$ ,  $p<0.05$ , whilst the interaction was not significant.





**Figure 15.** Experiment 9. Mean BP1 Error Score Relative to T1 (Error Bars Show  $\pm 1$  Standard Error).

These results appear to be extremely puzzling but taken in conjunction with the results of Experiment 8 they may offer even more support for a causal interpretation of the binding effect. In terms of the relationship between BP1 and T1, Causal Binding would suggest that BP1 would be perceived as subjectively *later*. Given that in Experiment 8 participants were required to time BP1 to coincide with T1, this would require BP1 to occur *earlier*, negating the difference so that participants subjectively make their button presses in synchrony with T1. However, this pattern of behaviour in Experiment 9 would have led to an objectively longer interval between BP1 and T2 in the Causal condition, as T2 was fixed to T1, rather than BP1. This is obviously in contrast with the Binding Effect. By shifting their BP1 closer to T2, it appears that

participants somehow 'acted out' the Temporal Binding effect.

As before I conducted an ANOVA on the factors Causality, Button Press and Interval. I found a significant effect of Button Press,  $F(1,34)=12.22$ ,  $MSE=10036.98$  and a significant effect of Interval,  $F(2,68)=3.15$ ,  $MSE=4395.85$ , but no effect of Causality,  $F(1,34)=3.971$ ,  $MSE=4519.41$ , n.s. There was a significant interaction between Button Press and Interval,  $F(2,68)=3.887$ ,  $MSE=4102.83$ , but not in the Causality x Button Press,  $F(1,34)=1.38$ ,  $MSE=2534.18$ , n.s., nor Causality x Interval interactions,  $F(2,68)=0.895$ ,  $MSE=3664.63$ , n.s. Finally, there was no significant Causality x Button Press x Interval interaction,  $F(2,68)=0.78$ ,  $MSE=3220.07$ , n.s.

The results of Experiment 9 were not what I had anticipated. Contrary to the original hypothesis, T2 was not significantly earlier in Causal than Non-Causal trials. In terms of the perception of T2 it appears that perhaps participants realized that T2 was tied to T1 and temporally independent of BP1. It may even be that participants understood that while in the Causal condition BP1 was a causal action (in that it facilitated T2), it was not temporally tied to it. This may have led to participants' understanding of T2 as being qualitatively different in terms of it being an effect compared to Experiment 8. The previous experiment involved a linear cause (BP1) → effect (T2), however in Experiment 9 BP1 was not independent of T1 as BP1 would not cause T2 unless it occurred within a fixed interval of BP1. In this case, T1 was an *enabler* (Sloman, 2005): a factor that must be present between two independent events for the first to be able to cause the second; If it is not present, the effect does not occur; BP1 causes T2 only if BP1 co-occurs with T1. Whilst obviously in Experiment 9 T1 did not cause T2, perhaps because there were two factors (BP1 and T1) that had to combine to cause T2, a shift in the perceived onset of T2 would strengthen the inferred causal relationship between BP1 and T2, but also erroneously between T1 and T2. Such a

relationship would go against the understanding participants gained in the training phase as they had been instructed to sometimes withhold BP1 at their will. To preserve a Binding effect, BP1 would have to be shifted towards its caused effect, T2, while T2 would not shift to prevent the inference of a causal relationship between itself and the (Non-Causal) T2. While in Experiment 8, BP2 was shifted towards T1, the lack of shift in terms of BP2 suggests that in Experiment 9, the Temporal Binding effect was driven exclusively by the shift of causal action towards its effect.

### **2.2.4 Empirical Section 2 Discussion**

The rationale of Empirical Section 2 was to move away from inferring the existence of a Temporal Binding effect based on post-hoc interval judgments and introduce an online behavioural method to investigate the perceived onset of events. Further these experiments aimed to disassociate Causal and Intentional actions by requiring intentional actions across conditions, but controlling the participants' beliefs about the causal efficacy of their actions.

In Experiment 7 participants learnt the interval between their motor-actions and caused effect, and were then asked to synchronize these actions with a target. They consistently initiated their Causal actions later than Non-Causal actions, even though both tasks required the same behaviour for normative responses (bisection of interval between tones). These results are consistent with participants having learnt the interval between action and effect as being shorter than veridical, and this mistaken perception being carried on into the experimental phase. However, given that this paradigm likely involved additional cognitive and perceptual processes in the Causal condition, such a comparison of the onset of caused visual stimulus with onset of target, I concede that it is perhaps not as strong a demonstration of the binding effect as I would have hoped.

Experiments 8 and 9 consisted of practically the same designs as each other, with a single modification. In Experiment 8 participants were instructed to make single button presses to two target tones. While in the Non-Causal condition the interval between targets was fixed, during Causal trials participants' first button press caused the second target to appear after a fixed interval. In this condition participants' perceived onset of the second target was shifted towards the first target, suggesting an earlier awareness of the event. Interestingly, this appears to have been strengthened by a shift in the first Causal button press earlier than the relative Non-Causal button press. Participants had been requested to synchronize their first button press with the first target tone, however a Binding shift of the causal motor action (i.e. the subjective awareness of the button press occurring closer to the effect) would lead participants to perceive that their motor action was "too late". As such an earlier response to the first target in Causal relative to Non-Causal trials suggests that participants perceived their actions as later than veridical, and to achieve synchrony with the target shifted their actual button presses earlier than veridical.

An unexpected pattern of results was demonstrated in Experiment 9. During Causal trials a button press was required within a fixed interval around the first target, otherwise the second target would be cancelled. This served to keep a causal relationship between the first button press and second target in the causal condition, but solved the variance problem of Experiment 8 in which the first button press was a perfect predictor of the onset of second target in Causal but not Non-Causal trials. This could possibly have explained the results of Experiment 8 in terms of a stronger association between cause and effect (due to the invariant temporal relationship) rather than higher-level causal relationships. Interestingly however in Experiment 9, there was no shift in the response time of the second button press. It appears that participants

understood that the second target was timed from the first, and as both T1 and BP1 needed to coincide within a narrow temporal window, the causal action BP1 was not independent of T1, which *enabled* BP1 to cause T2. A binding shift in T2 may have erroneously led to participants attributing a causal relationship between BP1/T1 and T2, when T1 did not cause T2 (as demonstrated in the training phase when they were told to make BP1 outside of the critical zone, or not at all, a few times to show that T2 would not occur). The Binding Effect was facilitated in this Experiment by a shift in the onset of Causal Button Press 1 later than that of the relative Non-Causal press.

By using motor actions as an indication of event time I had intended to move away from the reconstructive response methods of the previous Empirical Section. In the previous studies I had speculated that the Temporal Binding effect demonstrated in the magnitude estimation (Experiments 1-5) and reproduction (Experiment 6) paradigms may have arisen from post-perceptual higher level cognitive biases, differences in the attentional requirements of the two conditions leading to a modulation in pacemaker speed, missed pulses or possibly a combination of these. In terms of post-dictive effects I argue that these would not influence the results of Experiments 8-9, as the motor actions are preformed on line and are used as a measurement of the perceived onset of the event, and as such are resultant from predictive processes.

In terms of a modulation of attention it seems unlikely that one condition would differ from the other in terms of the required attentional load between temporal and non-temporal task demands. Given that the conceptualization of Experiments 8-9 involved two event sequences that were identical in their objective aims (synchronization of two button presses with two stimuli) it seems unlikely that one condition would require a differing amount of attention. However the higher-level cognitive processes demonstrated in prior research in which participants observe the

relationship between events and draw upon real world relationship representations (e.g. Buehner & May, 2002) to infer the causal relationship suggests that an amount of cognitive resource may be spent in consideration of the relationship. It is possible that Experiments 8 and 9 would not draw as strongly on these higher-level processes given the simplicity and consistency of the causal relationship here, compared to the grenade launcher and delayed explosion relations of Buehner and May (2002). This is especially true in Experiment 9 in which the second target tone is not temporally linked to the first button press. At that point, in both conditions the relationship between T1 and T2 was fixed, so that attention would not have to be paid to the relationship between BP1 and T2. Therefore, as long as they pressed within the critical zone, as was the goal of the experiment, the Causal relationship was not particularly pronounced (i.e. pressing early or later did not directly effect the onset of T2 making it similar to the Non-Causal condition). Unfortunately the very presence of a causal relationship between button press and effect cannot be explicitly ruled out as having additional attentional demands in the Causal condition. As to whether the mere presence of a causal relationship is enough to drain sufficient attentional resources required to distort timing, I cannot as yet say, however it seems reasonable to conclude that the retrieval or construction of causal models (e.g. Waldmann, 1996) and subsequent effect predictions would require some amount of cognitive or attentional resources.

In terms of the causal versus intentionality question Experiments 9-10 provides evidence that speaks in favour of a causality-based interpretation of the Temporal Binding phenomenon. Given that both Causal and Non-Causal trials involved identical intentional actions, the shifts in the participants' awareness of events in the Causal trial suggests that the Binding of action and effect stems from this causal relationship between the events, over the Intentional relationship. In terms of the mechanism that

drives this it is possible that it depends on higher level representations of the relationship between the button press and the effect, and given the understanding of a causal relationship between the two, drives them closer together. This shift results in participants' awareness of the onset of the cause and effect occurring later and earlier respectively. It is possible to argue that this modulation is driven by a causal relationship between the events drawing on the attention that would otherwise be paid to the interval. As of yet I cannot predict whether the mere presence of a simple causal relationship is sufficient to achieve this. Temporal Binding arising from an attentional drain (rather than say a dedicated process) would not depreciate the empirical support for the phenomenon. Given the results of Experiments 8-9 I feel that it is perhaps time to further investigate a causality-based explanation of the effect.

## 2.3 Empirical Section 3

In previous Empirical Sections I outlined a series of experiments that demonstrated a temporal shortening of the interval between related events, and were motivated by the suggestions of Eagleman and Holcombe (2002) that this Temporal Binding arose from a causal relationship between events. This was based upon David Hume's assertion that two causally related events must necessarily be temporally contiguous. This contiguity of cause and effect however can also occur in the spatial domain.

In an effective demonstration of this requirement of spatial contiguity for the inference of a causal relationship, Michotte (1946/1963), using his now classic stimuli in which a moving object making contact with a stationary one is reported as having caused the latter to move, interjected a small spatial gap (20mm) between the two objects at the moment at which the first object stopped and the second started its movement. This gap dramatically reduced the reported causal relationship between the two. It seems logical to assume that causal attribution between spatially distant events is dependent on an understanding of the mechanistic relationship underlying the relationship (Hubbard, 2004; Hubbard & Favretto, 2000) just as an understanding of the real world timeframe between events can give rise to, or remove, a causal interpretation of a sequence of temporally close or distant events (e.g. Buehner & May, 2002). As such cause and effect do not necessarily have to occupy particularly immediate space for us to understand the relationship between the two as long as we can understand the mechanistic relationship between them: a scientist can remotely control a vehicle on Mars via radio waves from a command room on earth, while the gravitational pull of the Sun causes the Earth to remain within its orbit from a distance of over 93 million miles.

Michottean stimuli have a long history of being employed to study the reported,



and modifiers of, the causal relationships between events, even in situations where a standard numerical judgment of the causal relationships is difficult or impossible such as with six-month old human infants (Leslie & Keeble, 1987) and Pigeons (Young, Beckmann, & Wasserman, 2006). These simple stimuli have also shown to elicit more complex notions than a perception of one object launching the other. One object commencing its movement before other spatially contiguous objects is perceived as “pulling” the other objects along with it (White & Milne, 1997) whilst a single object colliding with, or entering into a group of objects can be reported as disintegrating, or bursting the group (White & Milne, 1999).

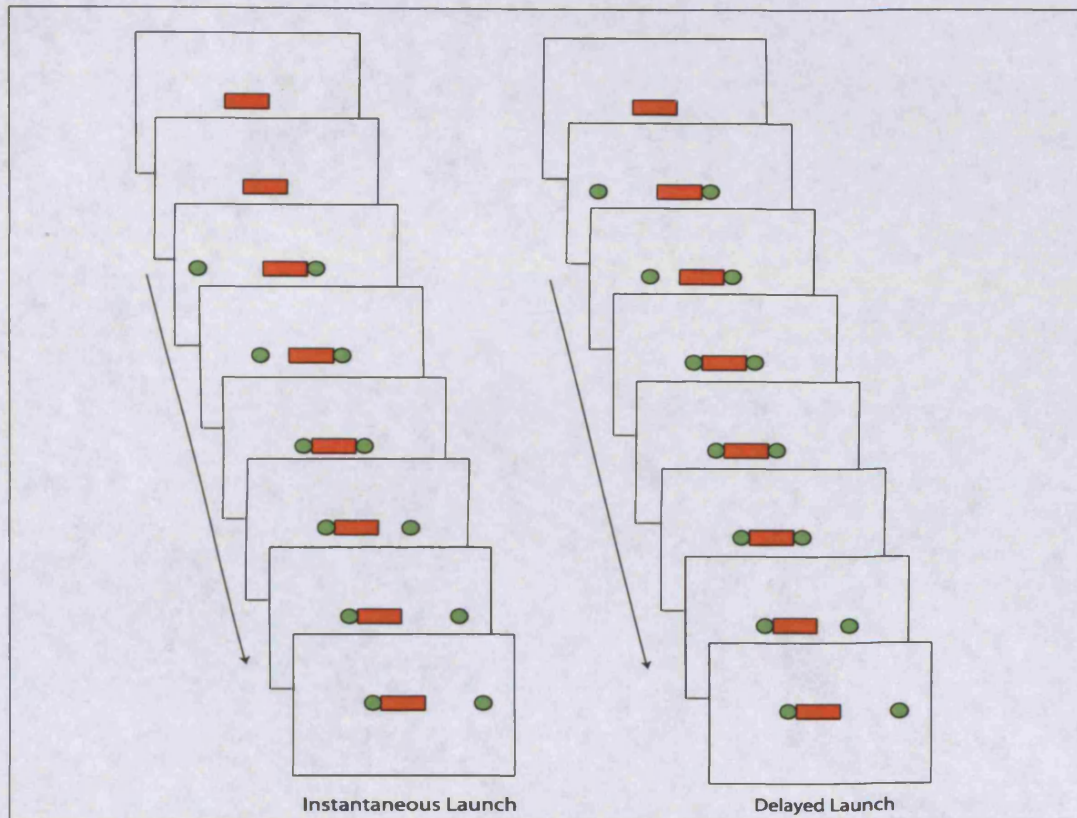
While a spatial gap can destroy the reported launching effect (see above) if the gap is fully, or even partially filled, the launching effect can be restored. Young and Falmier (2008) presented participants with Michottean stimuli that were separated by a gap that was either empty, fully or partially filled: While objects separated by unfilled gaps were judged to be the least causally related, collisions in which the gap was fully filled were judged to be strongly causally related (one having caused the other to move). The authors argued that this demonstrated a cognitive rather than low level perception of the causal relationship, based on a conceptualization of Newtonian physics: That is to say that the impetus of the first stimulus is transferred through the solid object, into the second stimulus causing it to move (Hubbard & Favretto, 2000).

### **2.3.1 Experiment 10**

In this experiment, in order to manipulate whether the participants consider the relationship between two circles presented onscreen was Causal or not, the interval between the end of the first circle’s movement and the start of the second’s varied between trials. In Instantaneous Launch (Causal) trials, the second circle started moving

immediately after the first circle stops moving. This was similar to the trials in which participants perceived “launching” of the second circle by the first reported by Michotte (1946/1963). In the alternative Delayed Launch (Non-Causal) trials, the start of the second circle’s movement was delayed for 600 milliseconds following the end of the first circle’s movement. The interjection of a delay (46ms) between contact and launch has previously demonstrated a marked decrease in reporting of one stimulus having caused the other to move (Michotte, 1946/1963).

In a similar method to that of Young and Falmier (2008), the Experiment 10 paradigm involved two objects that were separated by a bar. Whilst one began the trial in contact with the bar, the other object moved steadily towards this bar, stopping only once it had made contact. The object on the other side of the bar then began to move away along the same trajectory as the original object. With two different conditions, the relationship between these two objects was made to appear either Causal or Non-Causal by manipulating the interval between the first stimulus hitting the bar, and the second stimulus moving away, as either *Instantaneous* (Causal) or *Delayed* by 600ms (Non-Causal: see Figure 16 for graphical representation).



**Figure 16.** Experiment 10. Comparison of Causal and Non-Causal Condition.

Whilst Experiments 7-9 defined the causal element in terms of a mental interpretation of the relationship between two events that the participants learnt from prior training trials, the following two Experiments took a slightly different view. Rather than involving motor action resulting in subsequent effect (or not), participants in these experiments did not make intended motor actions during trials, instead only observed the relationship between stimuli. Participants were also not aware of the nature of the relationship prior to each trial, and had to infer the causal relationship on a trial-by-trial basis. Thus as in Experiments 7-9 the causal element in Experiments 10 and 11 that varied between conditions was a mental interpretation of the relationship between events. In this case, it was the inference that one object did or did not cause the other to move, based on the causal cues of Hume (1739/1888) and possibly the stimuli's

adherence to Newtonian physics (Hubbard and Favretto, 2000).

In both Instantaneous Launch and Delayed Launch conditions required participants to observe one sequence of stimuli and judge the length of the bar between them. Eagleman and Holcombe (2002) hypothesized that a Temporal Binding process or effect that shortens the causal relationship between two events would help strengthen the perceived causal relationship between the cause and effect based on Hume's (1739/1888) assertion that cause and effect should be temporally contiguous. Working back from this, as Hume also discussed spatial contiguity as a cue to causal inference, it was logical to suggest that a causal relationship between events separated in space may also be subject to a shortening of this distance between cause and effect, and thus also strengthen the causal relationship between the two. In Experiment 10 I predicted that participants observing a solid object between two stimuli would report the object as shorter (demonstrating a greater degree of judgment error), when the movement of one object caused the movement of the other.

### **2.3.1.1 Method**

#### *Participants*

18 Cardiff University Undergraduates participated in return for course credit. In a 40-minute session they participated in both this and one other experiment not described here. One participant failed to comply with the instructions and was not included in the analysis.

#### *Materials and apparatus*

The experiment was programmed with Python scripting language (v2.5) with the

Tkinter module and presented on a G3 Power PC iMac (600MHz, 1Gb RAM, 15" CRT display at 1024x768) running Mac OSX. Participants were sat ~44cm away from the screen, without head restraint. In a single experimental trial participants observed 3 onscreen two-dimensional objects: two green coloured circles, each 1.4° in diameter (referred to as circles A and B), and a red bar 1.4° in height and a random length between 1.4° and 6.7°, all presented on a white background. In each trial both circles would move a total of 9.9° each in a rightward direction. The circles travelled at a steady pace approximately 19.8° per seconds. By running the experimental program on one machine this movement rate was kept constant across participants.

The red bar was displayed with its mid-point at the centre of the screen, and had a random length of between 1.4° and 6.7°. Both circles A and B followed fixed horizontal trajectories over a 9.9° distance each. Circle A commenced movement on the left side of the screen (relative to the participant) and moved at a constant rate until it made contact with the red bar. During this movement circle B appeared stationary at the opposite end of the bar, and would not commence its movement until circle A had finished moving. The temporal interval between the end of A's movement and the commencement of B's movement was the experimental manipulation and is discussed in the next section. Circle B's movement occurred at a steady rate (equal to that of circle A) for the first 5.28°, and then began to slow down until it appeared at a standstill.

After each experimental demonstration in which both circles completed their movements all onscreen stimuli were removed. Participants were then presented with a slider object at the bottom of the display and a red probe bar of equal height to the one in the experimental phase. Moving the slider increased and decreased the length of the red bar. This probe bar was centred on the screen with the scroll bar beneath it. The probe bar at the start of each probe phase as set to 0.07° in length, with a width of 1.4°.

### *Design and Procedure*

In each trial of Experiment 11 participants were first shown a sequence of events in the presentation phase, and were required to make a length judgment in the response phase. The experiment consisted of two conditions in which the *Causal relationship* between circles A and B was manipulated via the delay between the end of A's movement and the start of B's. In the *Instantaneous Launch* condition, once circle A ended moving by coming into contact with the red bar, circle B's movement commenced immediately. This condition is similar to the "launching" effects of Michotte's (1946/1963) work in which participants perceived the first circle coming into contact with the second as having caused the movement of the second. In a *Delayed Launch* condition a 600ms delay was interjected between the collision of A with the bar, and the commencement of B's movement. This interval acted to nullify the perception of A having launched B, as demonstrated by Michotte (1946/1963) with a launching paradigm (without an interceding bar) and much shorter inter-event intervals.

In each presentation phase circle A commenced moving immediately, from the left hand side of the screen towards the right. Once A came into contact with the red bar it stopped moving. Following a delay dependent on causality condition (0 or 600ms for Instantaneous Launch and Delayed Launch respectively), circle B commenced moving towards the right of the screen, eventually slowing down and stopping  $9.9^\circ$  after the start of movement. Given that in both conditions circles A and B travelled at a constant rate, then the 600ms delay between impact of A and launch of B would have resulted in the red bar being present onscreen for a longer amount of time per presentation phase in the Delayed Launch condition. To compensate for this a set interval was introduced at the start and end of each trial. At the beginning of a trial, following this delay the circle

stimuli appeared and circle A started moving immediately. Once circle B had finished moving, another delay was enacted in which all stimuli remained motionless onscreen, the end of which consisted of the end of the trial, and the screen was cleared (see Figure 16 for Instantaneous Launch and Delayed Launch representations).

The intervals at the start and end of the presentation phase varied with the condition: In Instantaneous Launch trials, these intervals were 500ms each, whilst in Delayed Launch trials they were shortened to 200ms each to compensate for the 600ms collision/launch delay. This way in both conditions, the amount of time in which the red bar was present in the absence of circle movement as 1000ms.

Following each presentation phase, participants were required to indicate the length of the red bar between circles A and B. Towards this they employed the response method outlined in the above section: Once the stimuli had been cleared from the screen at the end of the presentation phase, participants were presented with a probe bar and an onscreen “slider” which they moved by clicking on it and dragging the mouse. At the beginning of this response phase the slider was set to 0° at its extreme left hand side. Moving the slider right increased the length of the probe bar while moving it back towards the left hand side decreased the length. Participants were instructed to re-size this probe bar so that it matched the length of the bar they had seen in the previous presentation phase. Once they were satisfied with their response, they pressed an onscreen button that stored their response and commenced the next trial. They did not receive any feedback to their responses.

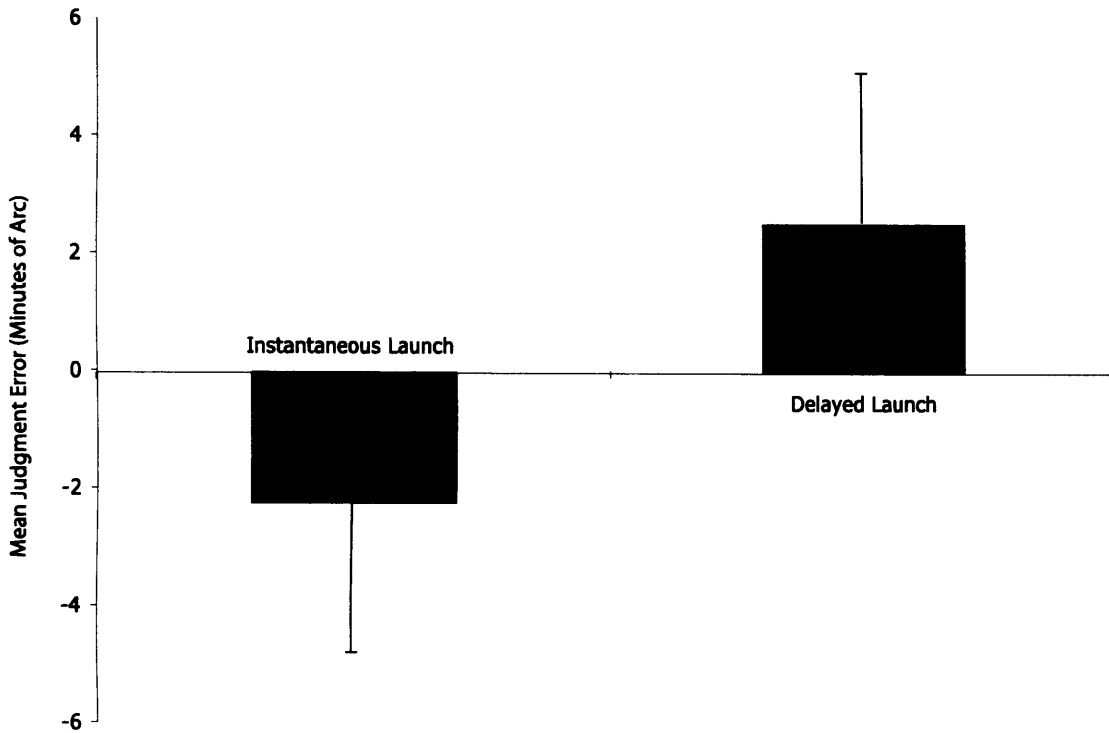
At the beginning of the experiment participants were informed that they were about to undertake a length judgment study. After receiving instructions they observed a Instantaneous Launch and a Delayed Launch presentation phase, as well as one instance of the response method. Once they understood the task they underwent 80 trials (40

Instantaneous Launch, 40 Delayed Launch) in which the presentation order of the trials was randomized. Once they had completed these experimental trials, they received further instructions that they would undergo a small number of Causality Judgment trials. In each of these trials, participants observed an Instantaneous Launch or Delayed Launch sequence (identical to the ones used in the experimental phases) and were then asked to judge how strongly they felt that circle A caused circle B to move. They did this on a 0-100 scale (0= Not at all Causal, 100= Strongly Causal) by moving an onscreen slider that reported the changing Causality Judgment. Once they were satisfied they pressed an onscreen button to move on to the next Causality Judgment trial. In total, they completed 10 Causality Judgments (5 Instantaneous Launch, 5 Delayed Launch), at the end of which the experiment concluded. In total the experiment took little more than 10 minutes, and the participants were fully debriefed.

### **2.3.1.2 Results & Discussion**

Each remaining participant's median Causality judgment for each condition based on a scale between 0 and 100 indicates that the two causality conditions were considered significantly different (Mean Median Instantaneous Launch (67.18) vs. Delayed Launch (30.29),  $t(16)=4.14$ ,  $p<0.05$ ). From the distance judgment data, the length of the red bar in the presentation phase was recorded per trial, as was the participants' reproduced length of the bar. For each trial the observed length was subtracted from the reproduced length to produce a Judgment Error in which a negative score indicates an underestimation of the reproduced relative to the observed length, and a positive number an overestimation. For each participant, the mean Judgment Error from each level of the causality condition was calculated (from a total of 40 trials per condition) and these were entered into the subsequent analysis.





**Figure 17.** Experiment 10. Mean Judgment Error (Error Bars Show  $\pm 1$  Standard Errors)

Figure 17 displays mean of each participant's mean Judgment Error. Evidently participants' mean judgment error in Instantaneous Launch trials was shorter ( $M = -2.19'$  (minutes of arc), or  $-0.04^\circ$ ), than in the Delayed launch condition ( $M = 2.46'$  /  $0.04^\circ$ ), and this difference was statistically significant ( $t(16) = 2.39$ ,  $p < 0.05$ ). This indicates that in the Instantaneous Launch condition participants significantly underestimated the distance between the two objects at the moment of impact, relative to the Delayed Launch condition.

I believe it may be useful to consider why we see such a small shift in the Instantaneous Launch condition. Participants were informed from the beginning that they were required to judge the length of the red bar, and although they were aware of the two circles' movements in both conditions, they did not necessarily have to observe the movement of, or consider the relationship between, the two moving circles. Indeed,

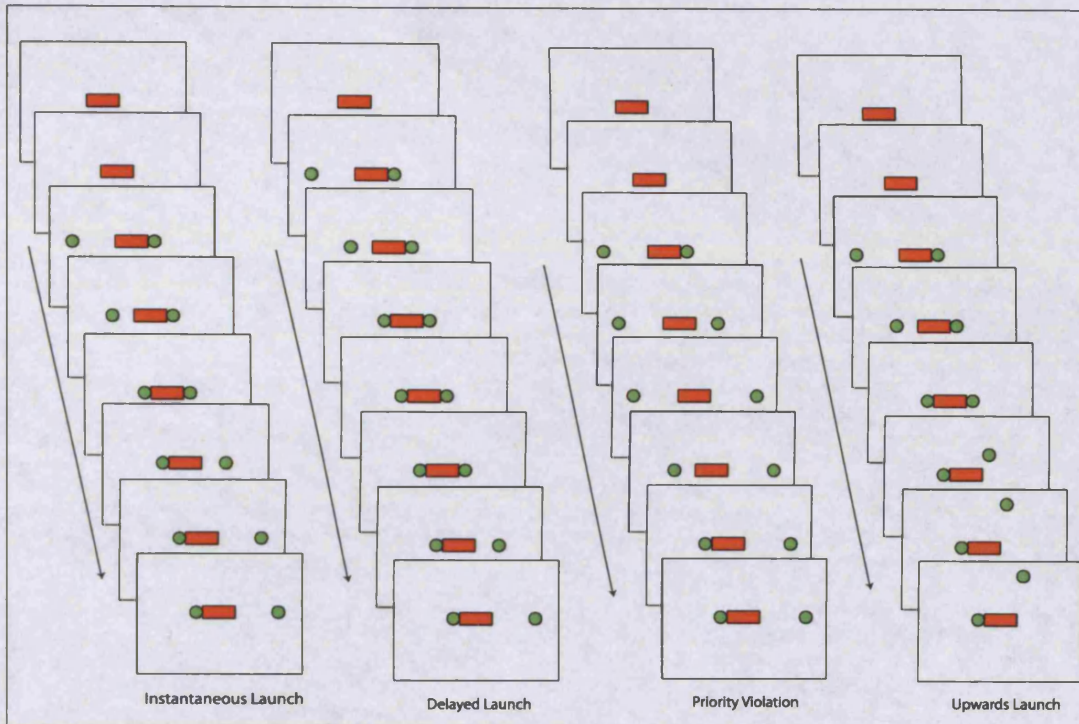
anecdotally in post-test debriefing some participants suggested that they did not particular pay attention to the moving stimuli in the experimental trials. Rather they focussed solely on the length of the red bar, possibly ignoring the Instantaneous Launch sequences. It seems unlikely that they could have wholly ignored the moving visual stimuli, although focussing on the red target bar may have impaired the perceived Instantaneous Launch relationship between the two objects. Although the Instantaneous Launch judgment data indicates that they differentiated the two conditions, these judgments were collected after the distance judgment trials, when participants were aware that they would not be required to judge the length of the bar.

A more problematic interpretation of the results arises when we consider that in the Delayed Launch condition participants observed the length of the bar with both circles adjacent to it than in the Instantaneous Launch condition (i.e. in the Delayed launch condition A and B contact the bar for 600ms, in the Instantaneous launch condition, B moves as soon as A reaches the bar). It is possible that the greater amount of time the circles spent in contact with the bar could have drawn additional focus to the bar itself. Further, it has been suggested that humans direct visual attention in terms of objects, or a group of objects (Duncan, 1984), rather than to the general space it occupies (the famous “attentional spotlight”: Posner, 1978). This lengthened focus period and grouping of bar with stimuli may somehow have lengthened the observed bar by the addition of some of the length of both circles to itself. Although this would explain the difference in terms of a low-level visual process, suggesting a perceived lengthening of the bar in Delayed Launch trials, this would not constitute a Spatial analogy to the Temporal Binding effect which, based on previous results (see Experiments 8-9) is likely dependent on higher level conceptualizations of the relationship.

### 2.3.2 Experiment 11

While experiment 10 demonstrated a robust underestimation of the length of an object separating two Instantaneous Causally related events, Experiment 11 was an advancement of the original paradigm. During the Delayed Launch trials of Experiment 10, in which a delay was injected between the first circle making contact with the bar and the commencement of the movement of the second, both circles appeared connected to the bar for a longer duration than in the Causal (Instantaneous Launch) condition. Obviously had participants been visually tracking the two circles during the trial then their gaze would have alighted on the bar for a longer period of time in these Delayed Launch conditions, and any difference in reported bar size may result from an increase in accuracy due to prolonged exposure or a visual after-effect.

To compensate for this Experiment 11 included two new forms of Non-Causal trial to demonstrate that the underestimation of the Instantaneous Launch bar lengths are not produced solely by a longer focus period on the target red bar. All conditions are displayed graphically in Figure 18. The original Instantaneous Launch and Delayed Launch trials of Experiment 10 were included in Experiment 11. In addition, Priority Violation trials involved the right hand circle B, commencing and completing its movement from the end of the red bar to the right hand side of the screen, *before* the left hand circle A moved (immediately afterwards) towards the red bar. This violation of the priority of cause over effect shows that the movement of circle A could not have caused the movement of the circle B.



**Figure 18.** Experiment 11. Comparison of Causal and Non-Causal Trials.

The second additional trial type, Upwards Launch (Figure 18), began identically to the Instantaneous Launch sequence, with circle A moving towards the central red bar. Once it made contact, circle B began to move. However, contrary to the Instantaneous Launch trials, the second circle moved *upwards* rather than towards the right hand side of the screen. As the effected movement of B did not follow the trajectory of A, I believed that participants would be less likely to have considered A as having caused the movement of B (but see results). Ultimately I predicted that Experiment 11 would replicate the relationship found between Instantaneous Launch and Delayed Launch judgments in Experiment 10. Although to some degree the magnitude of that difference may have been driven by a longer period of fixation on the target red bar in the Delayed Launch trials, I predicted that the experiment would further demonstrate a robust underestimation of the length of the bar in Instantaneous Launch trials relative to the two new Non-Causal trials, although the magnitude of this difference may be smaller.

Experiment 11 was designed to examine the problem in Experiment 10 (outlined above) by comparing the Instantaneous Launch condition with new Non-Causal launches. To ensure that participants considered these novel trials to be “non-causal” I would first perform a manipulation check by directly comparing the mean causal rating of each Non-Causal trial type with the mean causality rating of the Instantaneous Launch trials, and only including in the following analysis those trial types which differed significantly from the mean causal rating of the Instantaneous Launch trials. Once this was complete, I would make direct t-test comparisons between the Instantaneous Launch judgments and the length judgments of the new Non-Causal trial, which would allow direct comparison with Experiment 10.

### **2.3.2.1 Experiment 11 Method**

#### *Participants*

24 Cardiff University Undergraduates participated in return for course credit. In a 30-minute session they participated in both this and one other experiment not described here.

#### *Materials and Apparatus*

Experiment 11 was conducted on the same iMac with the same settings as Experiment 10 to ensure comparability of the duration and stimulus presentation of each trial. All durations, distances and stimuli were kept constant from Experiment 10, as was the response method. Although two new trial types were introduced, properties of the stimuli movement, such as the degree of slow-down towards the end of B’s movement and distance travelled was also kept constant, even when circle B moved before circle A (Priority Violation), or circle B moved upwards (Upwards Launch).

### *Design and Procedure*

The overall presentation of Experiment 11 was identical to that of Experiment 10. I again manipulated the Instantaneous Launch relationship between circles A and B, and as before, an Instantaneous Launch and Delayed Launch condition was included. Instantaneous Launch and Delayed Launch trials were identified by the temporal interval between the end of A's movement and the beginning of B's. Whilst in a Instantaneous Launch trial B moved immediately after A ended its movement in contact with the central red bar, a delay of 600ms was injected between these events in Delayed Launch trials to break the causal impression (see Figure 18). As above, to ensure that the red bar was onscreen for a comparable amount of time in both conditions (given the delay between the movement of A and B in Delayed Launch trials), a short delay was inserted at the beginning and end of the trial: 500ms for Instantaneous Launch, 200ms for Delayed Launch.

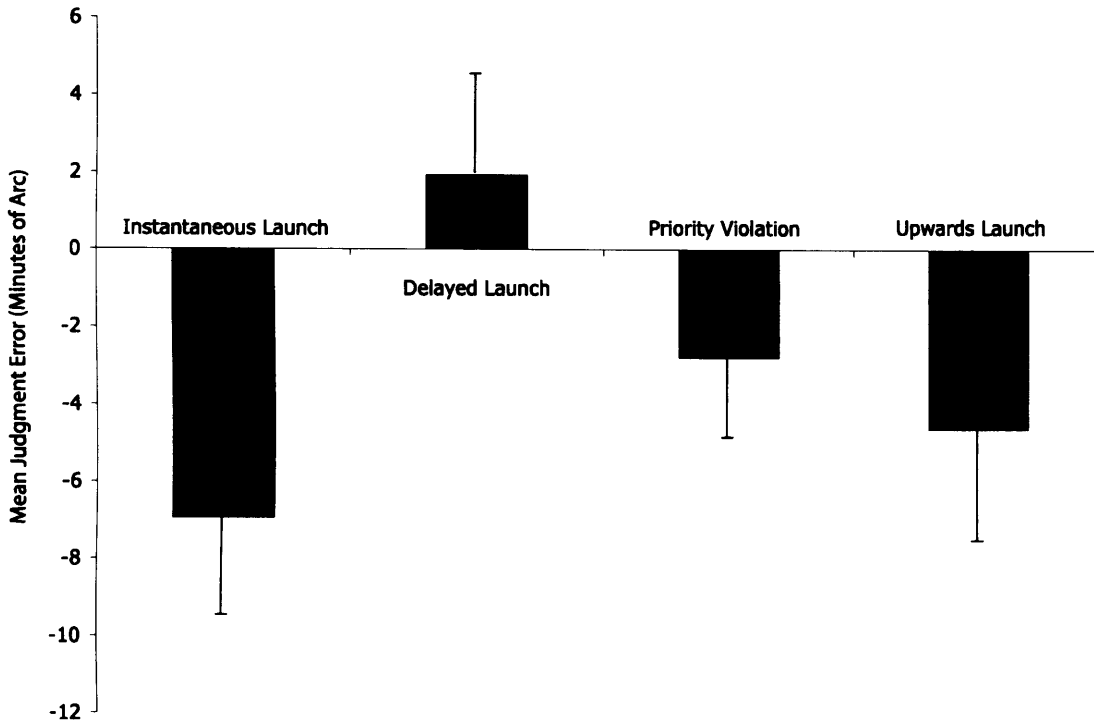
Two new conditions were included in this experiment. As no delay between the movements of A and B was present in either new condition, each trial was preceded and succeeded by a 500ms delay in which the red bar was visible but there was no movement onscreen (the same as Instantaneous Launch trials). Priority Violation trials were similar to Instantaneous Launch trials, but the order in which the circles moved was switched. Contrary to all other conditions circle B began moving towards the right of the screen, and having travelled 9.9°, it stopped. Immediately afterwards circle A began its movement and once it made contact with the red bar, also stopped. The Upwards Launch condition was similar to the Instantaneous Launch condition in that A moved towards the red bar, stopped on contact, and circle B immediately commenced its movement. However, unlike the Instantaneous Launch condition, circle B moved

9.9° upwards, rather than towards the right side of the screen (see Figure 18).

Other than the increased number of trials given the two new conditions Experiment 11 followed the same procedure as Experiment 10. Having received their instructions participants observed one instance of each condition, with the order randomly determined, and one instance of the response probe. Once they indicated that they understood the task they underwent 160 trials in which they judged the length of the red bar (40 trials each of Instantaneous Launch, Delayed Launch, Priority Violation and Upwards Launch). Upon completing this section, they then underwent a number of Causality Judgment trials in which having observed a sequence they were asked to judge how strongly they felt that A caused B to move, on a scale of 0-100 (0=Not at all Causal, 100= Strongly Causal) with an onscreen slider as in Experiment 1. This was done five times for each condition, for a total of 20 Causality Judgments. The experiment took between 15 and 20 minutes, following which participants were fully debriefed.

### **2.3.2.2 Experiment 11 Results and Discussion**

As in Experiment 10 I conducted a manipulation check on the Causality Judgments for all conditions. Instantaneous Launch conditions were judged the most causal ( $M=57.99$ ), followed by Upwards Launch ( $M=44.06$ ), then Delayed Launch ( $M=13.27$ ), and Priority Violation trials were judged the least causal. As the difference between the Causality Judgments of Instantaneous and Upwards launch were not significantly different,  $t(23)=1.9$  n.s. I focused on the Judgment Errors of only the Delayed Launch and Priority Violation trials in relation to the Instantaneous Launch condition (both  $t(23)>8.0$ ). However the Judgment Error for all four conditions are displayed in Figure 19.



**Figure 19.** Experiment 11. Mean Judgment Error (Error Bars Show  $\pm 1$  Standard Errors)

Visual inspection of Figure 19 suggests that as in Experiment 10, participants underestimated the length of the red bar in the Instantaneous Launch condition relative to the two Non-Causal trials. As mentioned previously, this experiment was concerned with comparing Instantaneous launch trials with novel Non-Causal trials. To this end I intended to compare in a similar manner to Experiment 10, Instantaneous Launch trials with the Priority Violation trials (Upwards Launch trials were not significantly different to Instantaneous Launch trials in terms of their Causal Ratings, and were as such removed from subsequent analysis). Whilst not the focus of this experiment Instantaneous Launch trials were also compared with Delayed Launch trials to confirm the results of Experiment 10. All t-test were therefore Bonferroni corrected with a p



value of 0.25.<sup>1</sup>

Reproduced bar length in Instantaneous Launch trials was significantly shorter than in Delayed Launches,  $t(23)=4.83$ ,  $p<0.025$ , as it was relative to bar length in Priority Violation trials,  $t(23)=2.77$ ,  $p<0.025$ , while the Priority Violation trials were judged significantly shorter than the Delayed Launch trials ( $t(23)=3.14$ ,  $p<0.025$ ). As before I demonstrated that participants judge the length of a bar between two objects as shorter in Instantaneous Launch (Causal) trials than in the Delayed Launch (Non-Causal) trials. Previously I suggested the possibility of some form of low level perceptual effect, in which the greater length of time spent observing the bar whilst connected to the two circles could have led to this over-estimation, rather than a higher-level Causality induced shortening. Experiment 11 suggests that this is not necessarily the case, as the Instantaneous Launch bar lengths were judged to be shorter than the Priority Violation bar lengths, in which the two circles were never in contact with the bar at the same time.

### 2.3.3 Empirical Section 3 Discussion

The basic rationale of Experiments 10-11 involved the possible spatial analogue of a causality based Temporal Binding process. Whilst Empirical Section 1 demonstrated that Temporal Binding exists outside of the Libet clock paradigm, Empirical Section 2 provided good evidence that the shifts in subjective awareness arising from the effect occur in the presence of Intentional action, but not in the absence

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<sup>1</sup> For the sake of completeness I also conducted an ANOVA on the three conditions that passed the previous manipulation check, and there was a significant effect of *Trial Type*,  $F(2/46)=15.01$ ,  $p<0.05$  (Upwards Launch trials were not significantly different to Instantaneous Launch trials in terms of their Causal Ratings, and are not included in this analysis).

of Causal action. This suggested that Eagleman and Holcombe (2002) may be correct in their hypothesis that the Temporal Binding process aids in the learning of causal relationships between two events based on the temporal contiguity between the two. Given that Hume (1739/1888) asserted that cause and effect must be close together in space and in time, coupled with the long history of the Michottean launching stimuli in the investigation of how the spatial separation of cause and effect can deter the reported causal relationship, I felt it was timely to investigate the existence of a possible Spatial Binding process.

In Experiment 10 I demonstrated that a red bar separating two circles was judged significantly shorter when the cessation of the first's movement was followed immediately by the movement of the second (Instantaneous Launch), than when the movement of the two was separated by a temporal duration (Delayed Launch). This suggested that a higher-level conceptualization of the relationship between the movement of the two as causally related, led to the shortening of the separating bar, fulfilling Hume's (1739/1888) suggestion that causally related events are likely to be close together in space, and thus helping improve the perceived causal relationship between the two. However I realize it was possible that in the Delayed Launch condition, a lower-level perceptual effect may have lengthened the reproduced length of the bar, in which the image on the retina could have been lengthened by the absorption of some of the surface or edge of the connected circles.

Experiment 11 remedied this by presenting participants with two new Non-Causal trials, however only one, Priority Violation, was considered significantly different from the Causal launch. During the Priority Violation trials the two circles were never in contact with the bar at the same time, and any lengthening of the bar, relative to the Instantaneous Launch trial, could not have involved this lower level

visual effect. Again, Causal trials were significantly shorter than Non-Causal (Delayed Launch and Priority Violation trials) suggesting that a Spatial Binding process, similar to Temporal Binding, does exist.

In previous experiments involving Temporal Binding I suggested a few possible explanations as to how the duration of an interval may be explained, and it is interesting to consider them in relation to Experiments 10-11. Firstly, the pattern of results in Experiments 1-5 closely resembled those of previous studies that investigated duration estimates in which the hypothesized speed of a pacemaker was varied as a factor of arousal (e.g. Penton-Voak et al., 1996). I argued that the causal motor action at the start of the trial may have drawn arousal or attentional resources away from the interval that was to be timed, causing more pulses to be “missed” in Operant versus relative Observational trials. This suggestion that Binding occurred due to salient events drawing attention away from the timed interval was considered in Experiments 8-9. The similarity of both Causal and Non-Causal conditions in Experiment 9 suggested however that the attentional demands of both conditions should be identical. The only difference between conditions was that the Causal button press resulted in an effect, which was not temporally tied to the button-press and therefore should not require more attention or render the sequence different to the Non-Causal sequence. This suggested that the shift of the timing of action in the Causal condition, in terms of an attentional explanation, would likely arise based on a higher-level conceptualization of the Causal relationship drawing upon the attentional resource, rather than a lower level motor or sensory difference between conditions.

As detailed above, in Experiment 10 it was possible that in the Delayed Launch trials the greater amount of time that stimuli A and B were in contact with the red bar (600ms) relative to the Instantaneous Launch condition could have contributed to the

observed difference between conditions. Participants would have spent more time observing the bar in the absence of stimulus movement during Delayed launches, especially had participants been attending to the moving stimuli before and after contact. This may have explained the results of Experiment 10, but Experiment 11 involved new Non-Causal event sequences that did not have this problem. Of interest were the Priority Violation trials in which stimuli A and B were never in contact with the bar at the same time; the reproduced length of the red bar was significantly shorter in the Instantaneous launch than these Priority Violation trials. Arguably the division of attention for both trials was likely to be the same, as had participants been attending to the moving stimuli there would not have been a period in which they observed the two circle stimuli at rest and could attend solely to the red bar. It should be noted that the degree of negative judgment error was greater in the Priority Violation than the Delayed Launch trials. I concede that in the Delayed launch trials, the attention paid to the red bar when both stimuli were at rest (600ms) may have led to the red bar being increased by the appropriation of some of the mass of the two circular stimuli.

However the significant difference between the Instantaneous (Causal) and Priority Launch (Non-Causal) trials suggest the existence of a Spatial Binding process. If the attentional demands of both conditions were the same then it seems unlikely that these results can be dismissed as arising from misdirected attentional resources. As in Experiment 9 however, it is possible that the mere presence of a Causal relationship between two events is sufficient change the attentional demands of a task; perhaps accessing a higher-level schematic understanding of the cause effect relationship is effortful enough to require a certain amount of attentional load.

Further, as I mentioned above, the results of Experiments 1-3 and 5 demonstrated that the degree of relative shortening between Operant and Observational

trials increased with increasing inter event intervals, as measured by verbal estimation. As Experiments 10 and 11 used random bar lengths, it does not show whether the degree of Spatial Binding increased with increasing bar length. In terms of the attentional explanation I discussed in the previous few paragraphs, in which causal inference draws attentional resource away from the processing of the stimulus, it seems unlikely that we would see an increase in Binding with bar length as accessing causal models for both short and long bars would most likely draw a similar amount of attentional load.

It is possible however that Spatial Binding arises from a mechanism dedicated to strengthening a causal relationship between spatially separated events. This could take the form of a fixed shortening (e.g. 2 “measurement units”) of the distance between causally related events. As this would be a smaller shift, in percentage terms, over long relative to short distances, this binding would arguably be less effective in helping strengthen causal relationships at long versus short distances. For example, judging longer probe bars may be susceptible to noise arising from certain perceptual effects (e.g. longer bars requiring longer and more effortful visual saccades to view them in their entirety), which could mask the relatively small fixed binding shift.

In this case I think it more likely that a dedicated Spatial Binding process would involve increasing shifts with longer length judgments, to help lessen the effect of an increase in noise with these longer objects. This could take the form of a mechanism that shortens the reported bar length perceived by the visual system before it is transferred to cognitive processing systems. Alternatively, it may be a top down cognitive process that enacts Spatial Binding in terms of selectively attending to only part of the object, and preventing the rest from being processed by the visual system.

It would be interesting to repeat Experiment 11 with fixed bar lengths and

require participants to make verbal estimations of their lengths (e.g. in millimetres). It seems likely that we would then observe a slope increase in the degree of binding over longer bar lengths as shown in temporal Experiments 1-5. However it is possible that this would result from post-perceptual biases similar to those discussed in section 2.1.7, that result in causally related events being reported as closer together in time (or in this case space) than comparable non-causal events, based on understandings of real-world relationships and causality-based heuristics.

Wearden et al. (in press) suggested that information processing and timing processes were deeply linked. It may be that visual processes are connected in a similar way to timing and information processing systems in terms of inferring relationships between events. For example a purely hypothetical visual system may include mechanisms that detect changes in viewed objects when they occur within short temporal windows. When the system detects such a change it could feed back to the cognitive systems for processing, which in turn instructs the visual mechanism to be more vigilant for subsequent similar changes. Further, when the visual system detects changes in stimuli it could also send a signal to the timing mechanism, which speeds up the internal clock, and thus the cognitive systems that are attempting to process the visual information (and infer a causal relationship) work faster. This, coupled with the attentional explanations discussed above, suggests that this Spatial Binding of objects may have deep rooted cognitive elements, rather than purely perceptual ones.

### **3 General Discussion**

The accurate measurement of time has fascinated many, from the earliest sundials, through the intricate timepieces of the Renaissance to our modern digital and atomic clocks. The Scalar Expectancy Theory describes time perception as involving a pacemaker that produces pulses that are then gathered in an accumulator, from which the amount of time passed is calculated (Gibbon, 1977; Gibbon, Church, & Meck, 1984). The time between events has been shown to help us understand the relationship between these events, even if we are not consciously aware of it. For example, close temporal proximity (within approximately 5ms) between two stimuli processed in different parts of the brain has been suggested to bring them together into a perceptual whole (von der Malsburg, 1981). Indeed it has long been argued that a close temporal proximity between two events must be present for one to have appeared to have caused the other (Hume, 1739/1888), while a long temporal gap between the two can disrupt this inferred causal relationship (e.g. Shanks, Pearson, & Dickinson, 1989).

Given the apparent utility of the perceived interval between events for the understanding of relationships it is perhaps surprising that human time perception can be modulated by factors such as arousal increasing the pacemaker speed (Penton-Voak, Edwards, Percival, & Wearden, 1996) or attentional factors resulting in some of the pacemaker pulses being missed by the accumulator (Zakay & Block, 1997). Recent evidence suggests that a manipulation of the interval between events may aid in the construction and learning of relationships between these events: Haggard, et al. (2002a) demonstrated that participants' reported onset of their intended action and subsequent tone were shifted towards each other in time relative to single event baselines (in which the action did not result in the tone, or the tone appeared automatically). They suggested that Temporal Binding had occurred between intended action and effect: By shortening

the interval between strongly associated actions and effects the process helped the agent infer that their motor action and effect had been the result of a conscious intention to make the movement and produce the effect (Wegner, 2002). There is a large amount of evidence in support of this (e.g. Haggard, Clark, & Kalogeras, 2002b; Wohlschläger, Haggard, Gesierich, & Prinz, 2003), however it has also been suggested that Temporal Binding occurs not to help infer a sense of agency (Wegner, 2002), but rather to help infer a causal relationship between the two (Eagleman & Holcombe, 2002) based on the notion that causally related events tend to be close together in time (Hume, 1739/1888).

The empirical aim of this thesis can be divided into three parts. Firstly, I intended to develop new experimental paradigms for the investigation of a Temporal Binding effect, given a previous reliance on the Libet clock method, with which binding was originally demonstrated (Haggard, Aschersleben, Gehrke, & Prinz, 2002a). Secondly, I intended to investigate the causality and intentionality based hypotheses of Eagleman and Holcombe (2002) and Haggard, et al. (2002a). Finally, I wished to move away from the temporal domain to look for a possible spatial analogue of the Binding effect, as Hume (1739/1888) asserted that proximity of cause and effect must occur in spatial, as well as temporal domains.

### **3.1 Summary of Results**

As I discussed in the introduction most of the prior empirical data supporting the existence of a Temporal Binding phenomenon was gained by the use of the “Libet clock” paradigm in which participants made event time judgments based upon the position of a constantly rotating clock hand. Although I agree that this is an elegant method of calculating the perceived onset of events, it is rather controversial (e.g. Wood, 1985), and I intended to develop new paradigms to see whether the Binding



effect was purely an artefact of the design. This was addressed in Empirical Section 1.

Experiments 1-5 asked participants to numerically judge the length of the interval between their actions and a subsequent event (an Operant sequence), as well as the interval between two unrelated events (Observational sequence). Although superficially identical in terms of auditory and visual stimuli (the first event of the Observational sequence corresponded to the noise produced by the button press in the Operant sequence), Operant trials included an intended action (button press) that caused a subsequent effect. Contrary to the Libet clock method used previously, this paradigm allowed us to directly observe any modulation of the inter-event interval, rather than inferring a shortening based upon the shifting of single events relative to baseline. Based upon previous research (e.g. Haggard, et al., 2002a) I predicted that due to an intentional/causal relationship in the Operant trials, participants would judge the inter-event intervals (in milliseconds) to be shorter than relative inter-event intervals in the Observational sequence.

Experiment 1 supported this prediction: Over intervals from 150-650ms participants consistently underestimated the inter-event intervals in Operant relative to Observational trials. The maximum interval (650ms) was chosen for this Experiment as it corresponded with the maximum interval used by Haggard et al. (2002b), who demonstrated a decrease in the amount of shift in the reported onset of action and effect relative to baseline with increasing inter-event intervals. I had predicted that the amount of difference between Operant and Observational trials would decrease with increasing inter-event intervals, however I found the reverse of this. Although there did not appear to be a difference between Operant and Observational trials at the shortest inter-event interval (150ms), there was an unexpected increase in this difference with increasing inter-event intervals (based on Slope Analysis).

Following Experiment 1 I decided to further investigate the increasing difference in Operant and Observational magnitude judgments by predicting that this increase would continue at longer intervals. By increasing the range of inter-event intervals in Experiment 2 from 750ms-1250ms I found a very similar pattern of results to Experiment 1: Operant trials were judged to be significantly shorter than relative Observational trials with a steady increase in the difference between them and no significant difference between the two conditions at the shortest (750ms) inter-event interval. I conjectured that participants were recognizing the shortest inter-event interval in each block of trials and using it as an “anchor point” upon which to base their subsequent judgments. This was rectified in the following experiments by including trials in which the effect (or second event in the Observational sequence) occurred immediately after the first event of the sequence (the 0ms interval).

With this modification in mind, Experiments 3-5 further examined the increasing difference between the two conditions over increasing inter-event intervals by increasing the longest observed interval. Experiment 3 again demonstrated a similar pattern of results to the two previous magnitude estimation Experiments with an interval range of 0-2000ms. Experiment 4 also showed similar findings, however with no significant increase in difference between conditions, at intervals between 0-4000ms. Experiment 5 was designed to address a flaw in Experiments 3 and 4, in that the maximal inter-event interval corresponded to the maximum possible response in milliseconds. As such, Experiment 5 was identical to Experiment 4, except that the response range (0-5000ms) was greater than the interval range (0-4000ms). Experiment 6 again showed an underestimation of Operant relative to Observational trials. However, unlike Experiment 5, these differences did increase as the length of the inter-event intervals increased.

As I discussed previously I realized that the binding effect demonstrated in Experiments 1-5 could result from a postdictive response heuristic (e.g. Tversky & Kahneman, 1974) that, understanding that intentionally/causally related events tend to be closer in time, could have been biasing the interval duration reports of the participants. As such I conducted Experiment 6 to remove the possibly biased magnitude estimation response method and introduce a reproduction method that is arguably less vulnerable to these effects. In an Experiment similar to Experiments 1-5 participants made intentional Operant actions that caused a subsequent tone, or observed two unrelated auditory stimuli. Following each sequence participants were asked to indicate the length of the interval between the two events by depressing a key for an interval equal to the interval they had just observed. As predicted participants' reproduced intervals were shorter in Operant trials than Observational trials indicating that they perceived the intentionally/causally related sequences as being shorter than unrelated sequences. Unlike Experiments 1-5 however, participants in Experiment 6 judged the interval between randomly determined intervals within a modest range, and so the data does not indicate whether this reported difference between conditions continued with intervals increasing over large ranges. However, given that the range of the randomly determined interval was 1200-1600ms, I still found a binding effect at intervals greater than those used in the Haggard et al. (2002b) study. Despite some possible criticisms Experiments 1-6 demonstrate a binding effect at intervals far longer than would be expected based upon the diminishing shifts of Haggard et al. (2002b).

Whilst Experiments 1-6 were successful in demonstrating a Binding effect outside of a Libet clock paradigm, Empirical Section 2 aimed to disassociate causal and intentional action by having intentional action in both conditions, but only in one condition would this action result in a (caused) effect. In these experiments, the causal

element was not explicitly evident in the experimental trials (assuming normative responses), but appeared prior to these during practice trials in which they experimented with the sequence of events. Thus causality was in the mind of the participant when they made their responses in the experimental phase. Participants in Experiment 7 were required to make a response between two auditory tones and their task in the Non-Causal condition consisted of simply bisecting the inter-tone interval. In the Causal condition their button press resulted in the delivery of a visual stimulus after an interval set to half the inter-tone interval, and their task was to synchronize this visual stimulus with the second tone (achieved by bisecting the interval). I predicted that based upon a perceived shortening of the interval between button press and effect participants would make responses later than veridical in the Causal condition. Although this was the case, I argued that the division of awareness was not equal in both conditions given that both Causal and Non-Causal trials required an awareness of both time past since the tone and remaining until the next tone, whilst Causal trials required additional awareness of the Button-Press → effect interval and a comparison of effect and target.

Experiment 8 remedied this by requiring participants to make 2 responses in conjunction with two target tones. Whilst these targets were a fixed interval apart in Non-Causal trials, during causal trials the second target tone appeared a set interval after the first button press. Having predicted that the first button press would appear later, and the second button press earlier, than their respective targets in the Causal condition, similar to the pattern of results of Haggard, et al. (2002a), participants demonstrated that Causal button presses to the second (caused) target appeared earlier than relative Non-Causal button presses, however the first button press of the Causal condition did not appear later than target. In fact, first button presses in the Causal condition appeared earlier than those of the Non-Causal condition. Participants

anticipated the second target as occurring earlier than veridical in the Causal trials, and the anticipation of the first target resulted in a shorter interval occurring between target tones. I argue that if participants perceived the interval between their button press and the effect as being shorter than veridical, then the shift of the button press towards the effect (i.e. occurring later than the first target tone) would have led to a longer interval between the two tones. Rather, in making their button presses earlier than the first target, participants would be making their first button press subjectively coincide with the first target, whilst also shortening the objective interval between tones. Thus Experiment 8 demonstrated the action and tone binding elements demonstrated by Haggard, et al. (2002a).

A criticism of Experiment 8 arises when considering the predictability of the second target following the first button press. Whilst in Non-Causal trials the interval between the first button press and second target was variable, in Causal trials the first response predicted the second target as occurring a set number of milliseconds afterwards. Experiment 9 dealt with this by introducing a critical zone around the first target in Causal trials. Should a participant press within this zone the second target would appear a fixed interval after the first. However if the button press fell outside this zone (or did not occur) then the second target would be cancelled. In Experiment 10 there was no significant difference between the timing of the second button press in Causal versus Non-Causal trials. However, the shift in button press one in Causal conditions towards the second target suggests participants were reproducing the Binding effect by shifting their button presses (indicative of the perceived onset of the events) closer together in time. At first glance, and in comparison with the results of Experiment 8, these findings are somewhat confusing. I argued that this absence of a perceptual shift in the onset of the second target tone was in fact a beneficial process

given the context of the causal relationship. Whilst in Experiment 8 (and all other experiments in this thesis) the causal trials had a linear cause effect sequence in which the effect was temporally tied to the cause and occurred independently of other factors, in Experiment 9 the effect only occurred if the cause appeared within a fixed interval around the target. This suggests a more complex causal chain, in which the first target acts as an enabler (Sloman, 2005) for the causal button press. Therefore, a Binding shift of the second tone towards both the causal button press and the first target tone would erroneously strengthen a causal relationship between the second and first target. In the case of Experiment 9 the Binding shift is facilitated by the shift of the causal button press towards the second target.

The final Empirical Section demonstrated a spatial analogue to the Temporal Binding phenomenon, by asking participants to judge the length of a bar between two causally or non-causally related stimuli. In the Instantaneous Launch condition, circle B begins the trial resting on the right side of the target bar, whilst Circle A moves from the left of the screen, into contact with the bar, and stops. Once A stops, B commences its movement towards the rightmost side of the screen. These trials resembled the classic Michottean (1946/1963) launching stimuli in which participants report the movement of A as having Caused the movement of B. Michotte reported that a temporal gap between the collision and launch could break this reported causal relationship. In Experiment 10 the alternative to the Instantaneous Launch condition was a Delayed Launch in which the movement of B commenced a short duration (in this case, 600ms) *after* A stopped moving. Having asked participants to rate how strongly they felt that the movement of A caused the movement of B. I found that participants did indeed rate the Instantaneous Launch trials as significantly more causal than the Delayed Launch trials.

Experiment 10 demonstrated that participants judged the length of the bar to be

shorter in Michottean Instantaneous Launch (causal) trials than in trials in which the Causal impression was destroyed by the introduction of a 600ms delay between movement phases (Delayed Launch trials). An unforeseen criticism of Experiment 10, that participants observed the two moving stimuli in contact with the red bar for longer in the Delayed versus Instantaneous launch conditions, thereby perhaps leading to greater accuracy or the absorption of some amount of the surface of the two moving stimuli into that of the bar, was dealt with in Experiment 11.

Whilst retaining the original two conditions (Instantaneous and Delayed Launch), I included two new Non-Causal trials. These aimed to remove the longer focal period on the red bar during the rest period of the two moving stimuli in the Delayed trials, while still appearing Non-Causal (i.e. the collision of one stimulus with the bar did not result in the movement of the other). The first, Priority Violation, involved circle B, commencing its movement in contact with the red bar and moving towards the rightmost side of the screen. Once it came to a halt, the until-then stationary stimulus A moved from its starting position on the left of the screen, and collided with the red bar. The other novel Non-Causal condition included in Experiment 11 closely resembled the Instantaneous Launch condition, in that stimulus A moved from the left of the screen into contact with the red bar, and B, positioned in contact with the right side of the bar, began to move immediately. However, in this Upwards Launch condition, B moved towards the top of the screen, rather than to the right (see Figure 18 for a graphical representation of all the conditions used in Experiment 11).

Having again asked participants to rate the causal relationship between the two stimuli we found that participants judged the Instantaneous Launch condition as the most causal, with both Delayed Launch and Priority Violation trials significantly less causal. However the Upwards Launch trials were not significantly different to the

Instantaneous Launch condition in the reported strength of the causal relationship, and as such were removed from further analysis. Again in Experiment 11 participants reported the length of the bar in the Instantaneous Launch condition as shorter than both the Delayed Launch and Priority Violation condition. Given that the reported length of the bar in the Priority Violation trials were shorter than in the Delayed Launch trials (both Non-Causal), I suggested that increased attention to the red bar may have indeed led to a lengthening of the red bar as detailed above. However, as the reported length of the bar was significantly shorter in the Instantaneous Launch than the Priority Violation trials, I argue that a Spatial Binding effect led to participants perceiving the distance between causally related stimuli as shorter than between unrelated ones.

### **3.2 Implications for Temporal Binding**

Whilst the previous section outlined the results of the Empirical Sections that demonstrate several instances of Temporal Binding, some consideration is required as to what these findings tell us about the Binding effect, and the underlying mechanism. Firstly, the number of studies in which I have apparently demonstrated a Binding effect leads me to conclude that this phenomenon is not merely an artefact of the Libet clock paradigm but an adaptive online mechanism that modifies how we consider the temporal relationship between events. As this was one of the main aims of the thesis I consider this a success.

In the Introduction I discussed how differing interpretations of Temporal Binding suggested differing critical times at which the mechanism was required to act. Whilst an Intentional account predicted that an efferent copy produced at the onset of movement to accurately predict the effect of movement was required for the subsequent shift in event timing to occur, the causality based interpretation suggested that



individuals needed to observe the relationships between events before inferring a causal relationship and then producing a shift. Recently Moore and Haggard (2008) have blurred this distinction by suggesting that both pre and postdictive elements are required for the rise of the binding effect. The results of the experiments in this thesis support the role of a predictive mechanism in Temporal Binding. For example, the SET pacemaker modulation explanation of the results of Experiments 1-5 suggests that in Operant trials (involving an intentional causal action) a slowing of pacemaker speed occurred prior to, or at the moment of, the gate mechanism opening at button press. Further the Causality conditions of Experiments 7-9 involve shifts in motor action that by nature cannot occur post-hoc. Arguably however, if the results of Experiments 1-5 are the result of a post perceptual bias this would obviously demonstrate a post-dictive effect based upon an understanding that causally related events tend to be closer together in time. Although not a Binding effect per-se, as it involves the reported manipulation of the perceived duration after the fact, rather than an actual manipulation of the perceived temporal interval, should this effect arise from a postdictive bias it would still be of interest to the field of causal cognition as an example of how we interpret events based on a reconstructed relationship.

I would however argue that perhaps the role of inferential causality in the binding effect is not necessarily one that re-constructs a temporal relationship after concluding whether it is causal or not, but rather informs the Binding process that the previous relationship was causal (via a Bayesian framework as suggested by Moore & Haggard, 2008), before commencing the next trial. By observing trials and concluding that certain stimuli such as a button press in Experiments 1-6 result in an effect (or even just being told that the following trials are causally related in the absence of disconfirming evidence), an inferential process can trigger, or help inform, a predictive

Binding process in the subsequent trial. Assuming that Spatial Binding is similar in nature to Temporal Binding, Experiments 10-11, unlike the other experiments, did not indicate to the participant the causal relationship between the stimuli prior the observation of the relationship (impact with a bar). This may demonstrate a purely postdictive inferential binding effect, however given the short temporal relationship between collision and launch and the arguably perceptual nature of causal launching (e.g. Michotte 1946/1963), then this may also be incorrect, and that the Binding process triggers at the moment of launch (i.e. the moment when cause results in effect). Perhaps it would be better to abandon pre and postdictive processes as a way to disassociate Cause and Effect given the results of Moore and Haggard (2008) and the above studies. Rather it may be a continuous online mechanism or series of mechanisms that monitor the relationships between events by constructing action-effect predictions in the presence of other stimuli.

Another possible interpretation of the results arises when we consider the effect that attentional task demands can have on temporal relationships. The Attentional Gate model of human interval timing (Block & Zakay, 1997; Zakay & Block, 1997) is based upon the SET model, but with the addition of a “gate” mechanism that opens wider when more attentional resource is paid to it. By attending to the passage of time (rather than extraneous stimuli), fewer pulses are “missed” and the interval appears longer than if another event or stimulus is attended to. I will now briefly review the above experiments with reference to how attentional demands may have driven the results.

Experiments 1-5 involved two conditions that were objectively identical: a click (either a recorded noise or actual causal button press in the Observational and Operant conditions respectively) was followed after an interval by a beep and participants were required to indicate the length of the interval between the events in milliseconds.

Subjectively it is possible that the demands of planning and making the causal button press in the Operant condition drew upon cognitive resources that may have included attention. This would not have been the case in the Observational condition, in which no motor action was present, and so in the Operant trials, due to some amount of attention being drawn away from the attentional gate to focus on the motor-action, the gate would not have been open as wide as in the Observational trials. Fewer pulses would have been able to pass through the gate (in effect some were “missed”), so that when the accumulator calculated the interval between the events, participants would have perceived the interval as being shorter than the same Observational interval. Experiment 6 was essentially the same as the previous experiments but with the magnitude estimation response method replaced by a reproduction paradigm, in an attempt to eliminate any shortening of the reported interval by some causality based heuristic or bias, however it did not address the attentional problem arising from the Operant condition’s use of a motor action.

Experiment 8 addressed this discrepancy by requiring participants in both Causal and Non-Causal sequences to make objectively identical intended motor actions. They were asked to make button presses in conjunction with two target tones. In the Causal conditions, the second target tone occurred a fixed interval after the first button press, while in the Non-Causal trials the second target tone appeared a fixed interval after the first target tone. Participants’ second button presses (that were meant to synchronize with the second target tone) in the Causal condition were significantly shifted towards the first target tone relative to the Non-Causal condition, suggesting the participants perceived that the caused effect occurred earlier. While based on previous Temporal Binding results (e.g., Haggard et al., 2002a) I expected the first button press to be shifted towards the second button press, doing so in this experiment would have

led to a longer inter-target tone interval. Instead, the shift of the first button press earlier in the Causal versus Non-Causal condition is suggestive of the participants having perceived their button press as occurring later than veridical, and having shifted their button press so that it subjectively appears in conjunction with the first target tone.

In both of these conditions participants made intentional motor actions, and so it seems unlikely that one can consider the Causal condition actions as being attended to more than their Non-Causal counterparts. However it remains to be seen whether the presence of a causal relationship is enough to make the cause and effect more salient than comparable unrelated events. If participants understand that their actions will result in a caused effect they may construct, or make predictions based on pre-constructed causal models (Waldmann, 1996). This could take the form of a retrieval of the memory of past relations which may exert some amount of cognitive load, or as part of a predictive motor action planning sequence (e.g. Wolpert, 1997) that draws attention to the action. Experiment 11 further supported this attentional based explanation of the Binding effect by demonstrating that the length of a red bar is shortened when it links two causally related (Instantaneous Launch) stimuli, than when the two stimuli are never in contact with the bar at the same time, nor is the movement of one caused by the other (Priority Violation condition). Arguably during both these trials the attentional demands of the task is the same and devoid of motor action, but the relative shortening of the bar between causally related stimuli suggests that this causal relationship is driving the shortening effect.

What implications would this Causal-Attention based explanation have on our understanding of Temporal Binding? Engbert (2005) demonstrated that higher intensity tones resulting from intended button presses produced greater Binding shifts than lower intensity tones relative to baselines. Arguably, given the Causal-Attention hypothesis,

this suggests that at the end of the interval the more salient high-intensity tones drew more attentional focus that resulted in some time-pulses being missed by the accumulator. This would however mean that the Temporal Binding may indeed help construct causal relationships by drawing attention away from time in the presence of causal action, and also infer causal relationships between events by shortening the inter-event interval, it would do so as a quirk of consciousness, not as an independent dedicated process.

While the question of the Binding phenomenon's attentional basis is still very much open, a few experiments in this thesis appear to cast doubt upon it. Firstly, in Experiments 1-5 I argued that an intended causal motor action in the Operant sequence may have drawn attentional focus away from the interval to be timed, relative to the Observational sequence. However, Experiments 1, 2, 3 and 5 demonstrated an increase the relative difference between conditions with increasing inter-event intervals. I do not see why the motor action would draw more attentional focus at longer intervals than at short ones, and an attentional explanation would account for some fairly constant difference between the conditions over all intervals, but not the observed increase. Instead the pattern of results was suggestive of a decrease in pacemaker speed. Previous studies investigated pacemaker modulations as a factor of arousal (e.g. Penton-Voak, 1996), and it is possible that the presence of the causal action was enough to increase the arousal state of the participants, thus decreasing the pacemaker speed. Alternatively, it may be that an increase in arousal is initiated by a Binding process, or that the pacemaker speed is modulated by a Binding process independent of arousal, but this remains to be investigated.

Experiment 9 provided in my opinion very strong evidence that Temporal Binding is not a simple attentional focus based phenomenon. While similar to

Experiment 8 in that participants were required to make intended button presses in conjunction with two target tones, unlike its sister study in Experiment 9 the second target tone was not temporally tied to the first button press. Rather, the second target tone appeared a fixed interval after the first target as long as the first button press appeared within a fixed zone around the first target. Unlike Experiment 8, the second button press was not shifted towards the first, which would have indicated that participants perceived the second target as occurring earlier in the Operant versus Observational trials. Instead in Experiment 9 the first button press was shifted towards the second in the Operant trials, whilst In Experiment 8 the first button press was shifted earlier than the first tone in the Operant condition, compared to the Observational button press.

I believe that a shift in the onset of the Operant second tone in Experiment 9 would have been detrimental to the inferred causal relationship between button press and tone. In this experiment the first button press had to occur within a 300ms window around the first tone, so the button press and tone were not independent. The presence of the first tone was required to enable the button press to cause the second tone. However participants were aware that the appearance of the first tone in the absence of the first button press would not cause the second tone. A shift in the second tone would have erroneously helped strengthen the causal relationship between the effect and an event that was know to not result in the effect. Instead, the shift in the causal button press away from the first target tone suggests that participants enacted a Temporal Binding of causal action and effect by shifting their motor action later in the Operant condition towards the second tone, which they knew was caused by the first button press. This Temporal Binding shift goes beyond anything that can be explained in terms of an allocation of attentional resources. Evidently some mechanism understood that the

shift of the second target towards its cause would also bring a mistaken impression that the first target had also caused it, and prevented its shifting. This allowed the Temporal Binding to be initiated solely by the shift in the first causal button press.

I intended to disassociate causality and intentionality to investigate what property of event relationship resulted in the Binding effect with respect to Haggard et al. (2002a) and Eagleman and Holcombe (2002). I believe that Experiments 8 and 9 successfully demonstrated that a mental interpretation of a relationship between action and effect as being causal is required for the demonstration of shifts in the perceived timing of events that shortens the relative interval between them: In these experiments participants made intentional actions in both conditions, and in one condition this action resulted in a caused effect. However Experiments 8-9 do not demonstrate a binding effect with a causal relationship independent of intentional action. As such it is too early to state that causality in the absence of intentionality would be sufficient to produce the Temporal Binding phenomenon, given the possibility of a requirement for both. The efferent explanation of Haggard, Aschersleben, Gehrke, & Prinz (2002a), in which an intentional action produces a prediction of the effect, necessarily requires a causal relationship between the two, and given the complex intertwining of both factors then an intentional causal relationship may be one solution.

The above experimental findings support a very recent Libet clock experiment, showing that causal relationships are essential for the appearance of Temporal Binding by demonstrating that a shift in the onset of action depended on the contingency between button press and tone (Moore, Lagnado, Deal, & Haggard, 2009). Participants were split into groups that varied the probability (high or low) that their button press would result in a tone, and both groups performed two conditions in which the contingency of the tone (whether or not it occurred without the press) varied. The

authors showed that in the low tone probability group, binding was increased by a strong contingency between action and effect only when the button press resulted in the tone. However in the high probability group, a stronger contingency increased binding in trials in which the tone did not occur. Moore et al. argued that a contingency dependant increase in binding in the low tone probability group during trials in which the tone occurred demonstrated that the shift occurred postdictively. Conversely, the increased shift brought on by strong contingency in the high probability group only when the tone was expected, but did not occur, shows a predictive element to the Temporal Binding phenomenon (Moore, Lagnado, Deal, & Haggard, 2009). While the authors showed that contingency between action and effect, one of Hume's cues to causal inference (Hume, 1739/1888), is an essential element of binding, they discuss their findings in terms of the attribution of agency. Whilst the work of this thesis has been geared towards the investigation of Temporal Binding as a mechanism for aiding causal inference (Eagleman & Holcombe, 2002), the possibility that the causality-based Temporal Binding process does indeed aid in the attribution of agency as part of a complex interaction between causality, action and perception cannot as yet be answered.

### **3.3 Further Directions**

The experiments outlined in the above empirical provided a glimpse at various facets of the Temporal Binding phenomenon, and yet several questions remain. Firstly, the Magnitude Estimation paradigm of Experiments 1-5 will most likely be of great use for the future investigation of the Temporal Binding phenomenon. In the studies outlined in the above Empirical Sections I attempted to investigate a binding between intentional causal actions and their subsequent effects, however the question remains whether Binding is a low-level process that modifies Intentional/Causal action effect



sequences based their likely temporal closeness, or whether this process employs higher level concepts that contain real world time-frame information. The TMS results of Haggard and Clark (2003) to some degree suggests that Binding is capable of more than a simple attraction effect by demonstrating a repulsion of the reported onset of action and effect when the action is involuntary. Whilst previous research has indicated that a complex higher level understanding of the relationship between events had been shown to mediate the reported causal relationship between the two (e.g. Buehner & May, 2004) it remains to be seen whether an understanding of such relationships can mediate the direction of a temporal shift, possibly by either slowing down or increasing the SET pacemaker speed.

The previous experiments reported here and those outlined in the Introduction tend to involve somewhat abstract button press electronic tone sequences in which participants may not be aware of the relationship between the two. It is logical to assume that most if not all participants have some form of experience with similar electronic relationships where an action (e.g. keyboard press, mouse click) results in a relatively sudden response (such as a document opening or a sound file playing), and given the electronic nature of these experimental action-effect relationships an assumption may be that the button presses ought to result in an immediate or near immediate effect. In this case a shortening of the inter-event interval would appear to be the best course of action in terms of the intention/causality based Binding process. However, suppose that participants were aware that the action effect sequence involved some form of mechanical action that tended to take a longer period of time to resolve? At that point a shortening of the reported action event sequence would to some degree be detrimental to the causal inference as it would violate the expected relationship between the two events. Instead, a lengthening of this reported relationship would better

reflect the higher-level conceptualization of the temporal relationship. In this case, whilst giving the participants some form of cover story in the Libet clock paradigm may be of interest, the Magnitude Estimation and Reproduction paradigms offers the chance to implicitly and explicitly manipulate the expected action effect sequence between two abstract stimuli with the use of cover stories that identify these stimuli in terms of real world objects. For example the comparison of the interval between changes in the appearance of two onscreen shapes (assuming that the Binding process employs higher order relations) would result in shorter estimates of the duration (relative to changes in unrelated stimuli) when participants expected that a change in one would result in a quick change in the other when implied by a cover story. However, should this story imply a longer (e.g. mechanical) relationship, then arguably the same interval could be reported as longer to reflect this expected temporal relation.

In terms of identifying the underlying Causal or Intentional nature of the Binding effect in the above Empirical Sections I demonstrated that the effect does not occur in the absence of a Causal relationship (see Experiments 8-9). However, as of now I can only state that causality in the presence of an intentional action is required to demonstrate a Binding effect, and cannot as yet say whether it occurs independently of intentional action. Indeed, Moore and colleagues (2009) have demonstrated the importance of contingency in pre and postdictive Temporal Binding shifts, while stating categorically that these results do not mean that motor signals have no influence on the experience of action.

A new series of experiments is required to comprehensively answer this question, by removing intentionality from the action-effect relationship. Take for example a device that pushes a button after a random interval, and this button press then causes a tone. This would completely remove intentionality from the button press-effect

relationship, as the machine itself would not have an intentional state. However, the button press and resultant tone would still be causally related, and a Causal Binding explanation predicts a reported shortening of the interval between cause and effect in this case, despite the absence of an intentional agent. While previous studies have done something similar to this (Wohlschläger, Engbert, & Haggard, 2003; Wohlschläger, Haggard, Gesierich, & Prinz, 2003) I argued in the Introduction that these non-intentional relationships, such as a self depressing key, may not be considered as causing the tone. Rather both key depression and tone may be considered as resulting from unseen cause, such as a computer program. The above device would provide a more explicit cause-effect sequence than these studies, as the lever hitting a button would cause the tone, and participants would be required to judge the interval between these events.

Such a device could quite easily be used in a Magnitude Estimation experiment (such as Experiments 1-5), as well as a Reproduction experiment (Experiment 6). In both cases one would predict that if Intentional action (in the presence of a Causal relationship) is required for the manifestation of the Binding shift, then the reported inter-event intervals would not be significantly different from a superficially similar Non-Intentional/Non-Causal event sequence given that neither condition involves an intentional agent. However, should causal beliefs underpin the Binding process, then due to the causal relationship between button press and effect, participants should rate the button press resulting from the device's action and the subsequent caused tone (Causal/Non-Intentional sequence) as shorter than a similar unrelated event sequence. Moving towards the empirical origins of Temporal Binding, the application of such a device to Haggard, et al.'s (2002a) original Libet Clock experiment may finally resolve the intentionality/causality question. In this study, the demonstration of an identical

pattern of results to the original Libet Clock experiment could resolve this issue. As such, I believe the removal of intention from the cause-effect sequence should be the next step in the investigation of the Temporal Binding phenomenon.

### **3.4 Conclusions**

The above thesis outlined and added to the ever-increasing amount of evidence that supports the existence of a Temporal Binding of action and effect with several novel methodologies that can be expanded still further to better understand the phenomenon. In terms of the intentionality versus causality based underpinnings of the Binding effect the experiments in which intentional action was kept constant between conditions (Experiments 8-9), and in which we observed Binding, suggests that indeed we should now begin to consider the shifts in terms of the causal relationship. However the degree to which this Temporal Binding is driven by attentional resource allocation, or dependent upon concurrent intended action to develop a sense of agency (Moore, Lagnado, Deal, & Haggard, 2009) or causality (Eagleman & Holcombe, 2002) is still an open question that will doubtless be of great interest to cognitive psychologists in the years to come.

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