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**UNIVERSITY OF SOUTHAMPTON**

FACULTY OF MEDICINE, HEALTH AND LIFE SCIENCES

School of Psychology

**Awareness in Ageing**

by

**Cassandra Richardson**

Thesis for the degree of Doctor of Philosophy

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UNIVERSITY OF SOUTHAMPTON

ABSTRACT

FACULTY OF MEDICINE, HEALTH AND LIFE SCIENCES

SCHOOL OF PSYCHOLOGY

Doctor of Philosophy

AWARENESS IN AGEING

by Cassandra Richardson

Deficits in awareness are found in diseases of ageing, and with acute and traumatic brain injury. Despite investigations of awareness in ageing patient populations, little is known about any potential effects of normal ageing on awareness. The Hierarchies of Processing model (Stuss, Picton & Alexander, 2001) provided a theoretical framework for an investigation of different types of awareness in healthy ageing. Four empirical studies are reported in this thesis.

An investigation of sensory processing using ERP components found that older adults had reduced attentional capture of auditory stimuli and allocated less attention to processing target stimuli. However, behavioural performance was equivalent across groups, indicating that the underlying differences found in sensory processing did not significantly impact on functioning. Age-related differences were also found in ERP components associated with performance monitoring: error detection; error processing; and, in reaction times. However, again, behavioural performance was similar, and indicated that older adults were able to compensate for underlying brain changes. In the third study, there were no age differences in any of the measures of awareness specifically focusing on current functioning and abilities, which suggested that awareness of abilities, did not alter as a function of healthy ageing. The final exploratory study found that the different levels of awareness were related, and, that the pattern of relationships was similar for younger and older adults.

Normal healthy ageing was associated with subtle differences in some processes underlying different types of awareness, but without any functional impairment. It was concluded that older adults may adapt to underlying brain and cognitive changes occurring during later life.

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## DECLARATION OF AUTHORSHIP

I, Cassandra Richardson, declare that the thesis entitled

Awareness in Ageing

and the work presented in the thesis are both my own, and have been generated by me as the result of my own original research. I confirm that:

- this work was done wholly or mainly while in candidature for a research degree at this University;
- where any part of this thesis has previously been submitted for a degree or any other qualification at this University or any other institution, this has been clearly stated;
- where I have consulted the published work of others, this is always clearly attributed;
- where I have quoted from the work of others, the source is always given. With the exception of such quotations, this thesis is entirely my own work;
- I have acknowledged all main sources of help;
- where the thesis is based on work done by myself jointly with others, I have made clear exactly what was done by others and what I have contributed myself;
- none of this work has been published before submission.



**Signed:** .....

**Date:**.....17<sup>th</sup> October 2008.....

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## Overview of Thesis.

Awareness can be affected by diseases of ageing (e.g. Alzheimer's disease: AD; Clare, 2004a, and Parkinson's disease: PD; Leritz, Loftis, Crucian, Friedman & Bowers, 2004), but, despite development in our understanding of awareness as a psychological concept (Chapter 1), the degree of change in awareness with normal ageing is not established. This calls into question the extent to which pathology constrains awareness over and above any normal age-related changes. Indeed, irrespective of pathology, ageing has been shown to be a time of significant neurological and cognitive change (Chapter 2). Impairments in awareness may, thus, be one result of the normal ageing process. Moreover, evidence from the metacognitive literature has indicated that different processes involved in awareness are differentially affected by age.

The empirical studies presented herein were designed to address two main issues. Firstly, the extent to which our understanding of awareness might be improved by the application of cognitive neuroscience techniques. Secondly, the degree to which awareness changes with age. Thus, traditional theory and modern neuroscience approaches are combined to offer a perspective on the investigation of awareness in normal ageing. The third chapter describes, in detail, the method employed to this end. Traditional neuropsychological techniques were applied, but also extended, to allow greater understanding of the underlying brain mechanisms involved in awareness. The aim was to replicate and extend understanding of different types of awareness in a younger and older sample.

Event-related potentials (ERPs) were introduced as a newer approach to investigating sensory awareness and performance monitoring. ERPs associated with the processing of basic sound or visual stimuli (stimulus-locked) reflect the moment at which attention is captured (Chapter 4). More complex cognitive functions are reflected in response-locked components, for example, the error-related negativity, elicited by a choice-response paradigm (Chapter 5). The third study (Chapter 6) utilised traditional metacognitive and clinical approaches to the study of awareness. The relationships between the traditional and neuroscientific measures of awareness were subsequently explored (Chapter 7). The final chapter discussed the utility of this approach, the contribution of the present data to existing models, and the possible implications of the findings for clinical populations.

## Chapter 1. Introduction.

If the fact of subjectivity runs counter to a certain definition of 'science', then it is the definition and not the fact which we will have to abandon.

Searle (1991, p. 25).

### 1.1. Introduction.

Awareness is a complex and multidimensional construct, inextricably linked with consciousness. Whilst the aim of this thesis is not to investigate consciousness per se, any discussion of awareness should consider consciousness. It is not proposed that consciousness and awareness are synonymous, even inter-changeable terms, but rather that the development of these concepts has a shared history. Indeed, the concepts of consciousness and awareness have long been a focus for discussion by philosophers, theorists and psychologists (e.g. Chalmers, 1996; Damasio, 1999; Dennett, 1993; Duval & Wicklund, 1972; Searle, 1991, 2000; Tulving, 1985; Zeman, 2001, 2006). Consequently, a number of psychological, neuropsychological and neurological theories and models exist, some of which will be reviewed in this chapter. The conceptualisation of levels of awareness found in some neurological models of consciousness is particularly influential (e.g. Prigatano & Johnson, 2003; Stuss, Picton & Alexander, 2001). The basic tenet is that there are different types of awareness, and that these types are hierarchically organised, and dependent on the functioning and inter-connectivity of neural assemblies in the brain. The development of brain imaging technology has facilitated investigation of candidate neural correlates of consciousness and awareness (e.g. Damasio, 1999; Frith, Perry & Lumer, 1999; Koch & Crick, 2000). The neuroscience community is thus moving towards empirical examination of the ancient and long-debated concepts of consciousness and awareness, where previously these concepts were considered too nebulous for scientific enquiry. An unavoidable challenge is first to operationalise awareness for objective study. As suggested by Searle (1991), rather than avoiding subjective concepts, a re-examination of definitions may indicate a way in which to facilitate modern neuroscience enquiry. The approach of this chapter is to offer an historical perspective into the evolution of the

definitions of consciousness and awareness. This review is focussed rather than exhaustive, with the aim being to demonstrate how the subjective may be objectively tested.

## 1.2. Philosophical Approaches to Consciousness.

A definitive description of consciousness does not exist, but this has not limited debate (see Blackmore, 2005). There is some consensus, however, that consciousness incorporates the multiple concepts of subjectivity, intentionality, qualitiveness, unity and awareness (e.g. Chalmers, 1996; Searle, 2000); still these are broad concepts and not a definition, per se. Describing consciousness has a long history in philosophy. Indeed, the relationship between consciousness and the physical body is one of the prominent issues in philosophy, popularly referred to as the ‘mind-body problem’. Early philosophers acknowledged the existence of a subjective thought process, and attempted to delineate its relationship with the body. During the Age of Enlightenment over a thousand years later, this theme was revisited by philosophers such as Descartes, whose reflections on the nature of consciousness promoted a separation of mind and body. Some present day philosophers (e.g. Dennett, 1993; Searle, 1991) have actively engaged with the neuroscience literature, and modified opinions about consciousness to include consideration of the fact that consciousness, awareness and brain function are inextricably linked.

### 1.2.1. Ancient Philosophy: Attempts to Demystify Subjective Experience.

To strive for an understanding and awareness of the self has been a feature of human existence for thousands of years, evidenced by the maxim “know thyself” which is often attributed to Socrates (c470-399 BC, cited in Warburton, 2006). Although the early Greek philosophers did not explicitly address the concepts of consciousness and awareness, they provided an early form of dualism which speculated upon the relationship between the soul and the body, and between the person and the universe. In his *Theory of Forms*, Plato (c427-347 BC, cited in Warburton, 2006) posited that experience is an illusion, and only the eternal is real. Forms are the ideal for experiences by which the world is made possible. The observable world and physical bodies were merely imperfect ‘copies’ of Forms. The goal of human intelligence is to reach a higher understanding of this relationship

(Robinson, 2003). Plato's theory was developed by Plotinus (205-270 AD, cited in Stokes, 2006) who described a trilogy of: 'the one', which is the source of our reality; 'the intellect', the knowledge by which 'the one' may contemplate itself (and the basis for Plato's Forms); and the highest level, 'the soul'. The soul has two types, one type is concerned with the body, whilst the other is concerned with rational thinking; the individual may focus on one or the other (Stokes, 2006). One of the obvious criticisms levelled at Plato, among other early philosophers, is his failure to explain the connection between a given body and soul (Robinson, 2003). However, Aristotle (c384-322 BC, cited in Warburton, 2006) argued against Plato's Forms and proposed that the soul was the very nature and property of the body, indeed, *linking* the soul and the body. Aristotle proposed that although intelligence was part of the soul, without a bodily organ it did not have material limits, hence it was immaterial. Intelligence was therefore not constrained by the properties of an organ, rather it was capable of receiving and reflecting upon all forms of information (Robinson, 2003). These are some of the earliest conceptualisations of a human characteristic, for example intelligence, being 'outside' and separate from the physical domain, a philosophical legacy that held for many years.

### 1.2.2. Cartesian Dualism: Separation of Mind and Body.

Perhaps the most influential form of dualism was provided by Descartes (1596-1650, cited in Warburton, 2006), who wrote *Meditations* during the seventeenth century, a time of significant conflict between religion and science. Descartes undertook a process of calling into doubt all of his own beliefs. This process culminated in the discovery of his only certainty, namely that he existed, and this led to the statement "I think, therefore I am". The belief that thinking and existence were inseparable, led Descartes to support a conceptual separation between the mind and the body. Descartes argued that the mind was an immaterial substance whose property was thought, whereas the body was non-thinking, therefore the mind was substantially distinct from the body (Robinson, 2003; Warburton, 2006). Cartesian dualism, nevertheless, held that although the mind and body were different substances, they causally interacted. In fact, Descartes believed that the mind communicated with the body via the pineal gland, located between the cerebral hemispheres. One of the challenges posed by this conceptualisation is in explaining how the immaterial mind communicated with the pineal gland. Interestingly, Damasio (2005)

later noted that the pineal gland is located very close to the hypothalamus, the brain stem and limbic system, all regions which are involved in the integration of physiological regulation and emotion. The social consequence of Descartes' division was the belief that the mind came under the jurisdiction of religion, whereas the physical, material aspects of the person came under the auspices of science (Searle, 2000); herein is reflected the separation of the science of medicine from psychology. The logical conclusion of this line of thought is that the body may be scientifically investigated, but not necessarily the functioning of the mind. Descartes' legacy: "I think, therefore I am" had popular appeal, but the lesser known rebuttal from the Materialists': "*I am, therefore I think*" (Gibson, 2004), suggests the alternative definition, namely that subjective thought is in fact *constrained* by physiological processes.

### 1.2.3. Development of Consciousness.

Other philosophers writing during and since the seventeenth century have been concerned with the origins of thought, and the relationship between thought and reality. Locke (1632-1704, cited in Warburton, 2006) posited that, at birth, an individual possesses no innate knowledge and that only experience provided knowledge, via sensory information. Locke believed that ideas were derived from experience, and that the perception of an object incorporated both sensation and an idea, which is a mental representation of the object (Warburton, 2006). As ideas are derived from experience, ideas would therefore be modified over time; a causal theory of perception (Stokes, 2006). For Locke this had implications for the stability of mental function and, more generally, for personal identity, which he associated with consciousness. Locke supported the view that without input from an 'external' reality, the mind would not be able to generate ideas, which may lead to an unstable personal identity (Warburton, 2006). However, Berkeley (1685-1783) argued against aspects of this philosophy with the proposition that an individual only has access to their mind, and therefore only the mind exists; that there is no evidence for an external world (Warburton, 2006). In other words, a vast amount of sensory information is perceived, and it is the mind that organises this information into coherent schemas of the external objects, thus giving them 'existence'. Awareness of an idea is not caused by perception, it *is* perception: "To be is to be perceived" (Berkeley, 1685-1783).

Hume (1711-1776, cited in Warburton, 2006) was influenced by Locke to the extent that he believed that the contents of the mind were derived from experience with the external world. He relied on observation and introspection to support his conceptualisations, bringing philosophical views under scientific assessment. Hume expanded on Locke's conceptualisation of ideas, in that he employed the term perceptions to incorporate the contents of experience, and separated perceptions into impressions and ideas. Impressions were sensory experiences, whereas ideas were copies of impressions and comprised the content of memories. Hume suggested a number of relationships between ideas that underlay thinking; resemblance, contiguity (timing), and cause and effect. Hume posited that his conceptualisation of perceptions and the relationships between ideas accounted for the conscious mind (Warburton, 2006). Although Hume did not directly address the concept of consciousness, conscious experience can be viewed as the incorporation of incoming sensory experience and the relationships between ideas. More importantly for the present discussion, Hume and Locke offer another reason for believing that the subjective mind is related to the body. If a process must undergo development, then the process is more consistent with the human body than with an ethereal, unchanging soul.

#### 1.2.4. Levels of Consciousness.

In the nineteenth century, the conceptualisation of levels of consciousness was a focus for the emerging field of psychology. Indeed, Nietzsche (1844-1900, cited in Warburton, 2006) described an extra-moral stage of human evolution, promoting the recognition of unconscious rather than conscious motivations for human behaviour (Warburton, 2006). Nietzsche's focus on unconscious drives was particularly influential on Freud's thinking (Warburton, 2006). One of the prominent features of Freud's psychoanalytic theory was the role of unconscious processes in motivating behaviour (Freud, 1946, cited in Warburton, 2006). Freud (1856-1939, cited in Warburton, 2006) proposed that the personality was composed of the Id, Ego and Superego and that a balance between the three constructs was necessary for mental health. Freud was concerned with the unconscious mind, as he believed that negative experiences were repressed, which led to anxiety and conflict. In his view, defence mechanisms were the unconscious strategies employed by the Ego to relieve this anxiety and conflict (Freud, 1946, cited in Warburton, 2006). Freud employed hypnosis, free association and dream interpretation as methods for

assessing the unconscious mind. A critical assumption is that an individual may be aware of some levels of conscious thought, but not others. The 'inaccessible' levels of consciousness (e.g. the Id and Superego), nevertheless exert influence over behaviour, and may become accessible through examination of their behavioural manifestations, for example, obsessive-compulsive behaviour in an individual with neurosis. This theory suggests that there are levels of consciousness of which we are not aware, but about which the individual may *become* aware.

#### 1.2.5. Contemporary Philosophy: Mind-Body Association.

Contemporary philosophers have incorporated the increased knowledge and understanding of the processes involved in brain functioning into their theories of consciousness (e.g. Chalmers, 1996; Dennett, 1993; Searle, 1991). Dennett (1993), in his Multiple Drafts model of consciousness, proposed that at any one point numerous brain processes are interpreting incoming sensory information and that this stream of information is continuously being edited. Experience and consciousness are the products of these ongoing processes (Dennett, 1993). The many "editorial processes" (Dennett, 1993, p. 112) occurring at any one time contribute to an explanation of the subjective experience of consciousness; different points in the stream of information being processed will elicit different "narratives" (Dennett, 1993, p. 113). In contrast, Chalmers (1996) has argued that consciousness is a fundamental property of the world, and cannot be wholly explained in physical terms and relationships.

Searle (1991) has also argued for acceptance that consciousness is a feature of the brain. However, he describes the relationship between consciousness and underlying brain function in terms of a system's properties: mental states are caused by and realised in the micro-structure and functioning of neurons. Searle (2000) advocated the investigation of neural correlates of consciousness, because "[...] the way, in short, to dispel the mystery is to understand the processes." (Searle, 1991, p. 23). The processes of awareness are a feature of consciousness (Searle, 2000), with Chalmers (1996) conceptualising awareness as "the psychological correlate of consciousness" (p. 220).

The combination of philosophical and neuroscientific approaches to the study of consciousness has been viewed as progressive (Searle, 1991), but could also be overtly reductionist (cf. Wittgenstein, included in Rosen, 2000). Francis Crick stated that “philosophers often ask good questions, but they have no techniques for getting the answers” (Crick in *Conversations On Consciousness*, p. 74, Blackmore, 2005), but that is not necessarily the purpose of philosophy.

### 1.3. Introduction to Psychological Theories of Awareness.

This chapter began by saying that the terms consciousness and awareness are inextricably linked. Indeed, contemporary philosophers (e.g. Chalmers, Searle) consider awareness to be a central construct in consciousness. However, some psychologists have distinguished between these concepts: “awareness presumes consciousness, but consciousness does not imply awareness” (Wheeler, Stuss & Tulving, 1997, p. 335). This statement is quite profound, and requires consideration. Firstly, the proposition that ‘awareness presumes consciousness’ may be true for many cognitive processes such as language comprehension, but it may also be argued that there are some types of pre-conscious awareness. For example, physiological regulatory mechanisms transmit awareness of needs (e.g. hunger and thirst) into consciousness in order for that particular drive to be satisfied (Damasio, 2005). Arguably, these are forms of awareness. Moreover, Broadbent (1958) theorised that all environmental stimuli are processed on some level, with only those judged to be most significant passing through an attentional filter. Recent research into error processing also indicates the existence of a performance-monitoring system, operating within about 100ms of an action (see Falkenstein, Hohnsbein, Hoorman & Blanke, 1991; Gehring, Coles, Meyer & Donchin, 1993). This system monitors for the presence of an error and may alert the brain to take remedial action; the brain may thus be *aware* that an error has occurred before the individual is consciously aware. The second part of the statement states that ‘consciousness does not imply awareness’. In other words, an individual may be conscious, but without being aware of certain aspects of the environment. This proposition will now be discussed in more detail.

Modern philosophy has considered that consciousness and awareness may be constructs of the brain, but a major challenge for psychology is the relationship between these concepts, with some definitions of awareness being easier to test than others. Psychologists have responded by identifying components of awareness that may be individually tested. Social psychological theories of self-awareness have, for example, identified attention as a critical cognitive process producing self-awareness. Thus, an important aim of the next section is to demonstrate that awareness is not a unitary construct and that different aspects of awareness lend themselves to empirical research. An additional aim is to show that these facets may be differentially affected by ageing.

### 1.3.1. Metacognition.

Metacognition is the study of self-reflected aspects of cognition; that is how an individual reasons about their own thinking and memory, as well as the executive processes that facilitate the selection of strategies and control of the allocation of processing resources (Wellman, 1983). Awareness has been conceptualised as a metacognitive process, in that monitoring abilities and awareness are analogous (Hertzog & Dixon, 1994). Metamemory, in particular, has been a focus for research. Metamemory is a multidimensional construct that is comprised of knowledge of, and strategies for, memory functioning, awareness of one's own memory, and self-referent beliefs about memory (Hertzog & Dixon, 1994). The metamemory literature typically does not employ the term awareness; rather, studies have investigated memory monitoring, which has been defined as the mechanism facilitating the revision of memory knowledge (Bjerman-Copland & Charness, 1994). Nelson and Narens (1990) provided a framework for the empirical assessment of the monitoring and control processes involved in memory and learning. The model identified four types of monitoring, each defined by the judgments involved at different points in the course of performance in a memory task: ease-of-learning; judgements-of-learning and feeling-of-knowing judgements in the initial prediction of performance phase of the task; feeling-of-knowing; and confidence judgements in the test phase of the task.

### 1.3.2. Social Psychological Theories of Self-Awareness.

The relationship between self-awareness and self-knowledge has been extensively debated within the social psychology literature. This may be partly due to the assumption that the relationship is complex and difficult to investigate empirically. A review by Silvia and Gendolla (2001) explored the moderating role of self-focused attention on self-awareness and argued that the process of introspection may function to alter the view of the self. In other words, requesting that a person considers their self-awareness can fundamentally change the process under observation. In essence, the person may become more self-aware. The role and consequences of self-focused attention underpins each of the main social psychological theories of self-awareness (see Silvia & Gendolla, 2001). Some theories are considered in greater detail below due to their important contribution to our understanding of self-awareness.

Before the self-awareness theories can be considered, however, it is necessary to understand the definitions of self-awareness on which they are based. The consensus of opinion appears to be behind the definition of self-awareness presented by Duval and Wicklund (1972): “when attention is directed inward and the individual’s consciousness is focused on himself” (p. 2). This is where the consensus ends as there are two approaches to the actual study of self-awareness based on a distinction between situational self-awareness and dispositional self-awareness, otherwise termed self-consciousness. Although both research traditions view self-awareness as an independent variable, the situational literature manipulates self-awareness by assigning participants into either a control condition or to a condition that elicits ‘high’ self-awareness, for example, through the use of mirrors, video cameras or reminders of the self (e.g. Duval & Wicklund, 1972; Macrae, Bodenhausen & Milne, 1998). In contrast, the dispositional literature focuses on individual differences in self-consciousness, which is typically measured by self-report of self-symbolic thoughts. A median split on the Private Self-Consciousness subscale (Fenigstein, Scheier & Buss, 1975) is commonly employed to create high and low self-consciousness groups (e.g. Hull, Sloane, Meteyer & Matthews, 2002).

The objective self-awareness theory (Duval & Wicklund, 1972; Silvia & Duval, 2001) posited two types of self-awareness: ‘objective self-awareness’ which arises from self-focused attention, and ‘subjective self-awareness’ which occurs through the experience of

activity. The theory states that when attention is focused in on the self (self-focused attention), objective self-awareness may be elicited. This can lead to a comparison between the self, defined as knowledge held about the individual, and a standard defined as “a mental representation of correct behaviour, attitudes, and traits” (Duval & Wicklund, 1972, p. 3). Any discrepancy found between the self and standards from this self-evaluation could lead to negative affect, which could in turn motivate the person to reduce the discrepancy, either by modifying their standards or by avoiding introspection. Since the original theory was published, research has focused on elucidating those processes potentially involved in reducing any discrepancy, for example, attributions involved in explaining the discrepancy (Silvia & Duval, 2001).

Two later theories of self-awareness also have the concept of an automatic internal comparison with a standard as a central tenet of self-regulation and behaviour (Carver & Scheier, 1981; Gibbon, 1990). These later theories however, differ in their conceptualisation of standards. Carver and Scheier’s (1981) model incorporated the view that self-focused attention could be towards either public or private aspects of the self; the external environment or the internal. Additionally, they proposed that mental representations of standards were hierarchically arranged. There are two critical points: firstly, if the standard chosen during the evaluative process does not correspond with behaviour in the current evaluation this can lead to dissonance; secondly, lower level standards can be accessed leading to the self-regulation of behaviour (Carver & Scheier, 1981). Gibbon (1990) also presented a multi-level model suggesting that at the experiential level, attention is focused on arousal and bodily sensations; at the behavioural level, a consideration of standards regarding correct behaviours occurs; and at the evaluative level, there is a comparison with the ‘ideal’ self. In summary, the automatic, internal focusing of attention was considered to provide accurate information necessary for self-regulation of behaviour.

Failure of self-focused attention and self-regulatory processes (processes that facilitate regulation of behaviour) have been linked with psychopathology (e.g. Ingram, 1990; Pyszczynski & Greenberg, 1987). Intense self-focus can elicit hyper-sensitivity to psychopathological symptoms which, in turn, may interact with other processes such as catastrophic thoughts and rumination, which can compound the problem. Pyszczynski and Greenberg (1987) presented a ‘self-awareness theory’ of reactive depression, proposing

that self-focused attention exacerbated negative affect and a perception of negative outcomes, which in turn created a “depressive self-focusing style” (Pyszczynski & Greenberg, 1987, p. 127), a style of focusing attention to depressive symptomatology. Similarly, Ingram (1990) proposed that this relationship between self-focused attention and self-focusing style was central to the majority of psychopathological conditions (e.g. depression, anxiety, alcohol abuse, vulnerability and schizophrenia). Again, a limitation of these theories is the difficulty in testing them, in this case from an ethical perspective. It may be problematic and unethical to manipulate a depressive self-focusing style, and in separating illness symptomatology from a self-focusing style.

In contrast to the theories of self-awareness that are based on self-focused attention, Hull and Levy (1979) proposed that self-awareness involves the automatic encoding of self-relevant information from the environment. This refers to the sensitivity of self-relevant environmental cues. The point of this theory is that any information that is processed as self-relevant may influence behaviour, regardless of the applicability of the information to the participant. More recently, in support of Hull and Levy’s (1979) earlier theory, Hull, Slone, Meteyer and Matthews (2002) used implicit and subliminal primes to investigate self-consciousness and its relationship to behaviour. Hull et al. (2002) employed a median split on the Private Self-Consciousness subscale and found that high, private self-consciousness (high awareness of internal, self-symbolic thoughts) was associated with behaviours influenced by the primes presented, e.g. walking more slowly after presentation to an elderly prime (elderly stereotype: Studies 1a and 1b), and increased blood pressure to an angry prime (Study 4). These results suggest that individuals high in private self-consciousness have greater sensitivity to environmental cues that may be processed as self-referent. One challenge for the Hull and Levy (1979) theory, in particular, is in accounting for behavioural consistency. That is, the degree to which an individual may maintain ‘high’ self-awareness, for example, the extent to which this is a trait rather than is state, is not known.

In summary, the social psychological theories have defined self-awareness as either situational or dispositional. In other words, self-awareness either arises from self-focused attention or is a personality characteristic. Whilst investigators have focused on the relationships between self-awareness and the self-regulation of behaviour, none address

possible developmental influences on self-awareness or the influence of experience on the development and maintenance of standards.

### 1.3.3. Psychological Models of Awareness.

Cavanaugh (1989) presented a theoretical framework accounting for different perspectives of awareness in memory, drawing heavily from the metamemory literature. The model incorporates several variables identified as important to memory functioning namely; experience, task demands, knowledge, personality, beliefs, and motivation. Three types of awareness are distinguished: (i) systemic awareness, defined as knowledge and beliefs about memory functioning; (ii) epistemic awareness, which represents the ability to evaluate the knowledge base; and, (iii) on-line awareness, which refers to the monitoring of processing during memory tasks. Each type of awareness may be tested using different methodologies. Systemic awareness can be assessed with metamemory questionnaires, epistemic awareness with feeling-of-knowing (FOK) judgements, and on-line awareness with processes of recollection. The model predicted pathways between systemic awareness and the knowledge base, knowledge evaluation and epistemic awareness, and executive processing and on-line awareness. However, the different types of awareness are not connected. A search of the available literature did not find any studies empirically assessing this model. Memory monitoring studies have, nevertheless employed FOK judgements and predictions across trials (e.g. Bieman-Copland & Charness, 1994; Perrotin, Isingrini, Souchay, Clarys & Taconnat, 2005; Souchay & Isingrini, 2004; Souchay, Isingrini & Espagnet, 2000) and, it can be argued that these processes also represent on-line awareness. Although Cavanaugh's (1989) model does not account for the dynamic relationship between awareness and memory functioning, it does provide testable hypotheses about awareness and its underlying processes. For example, investigating relationships between knowledge evaluation and FOK judgements may contribute to an assessment of epistemic awareness.

The relationship between awareness and memory is also a central tenet of one of the most influential psychological models of awareness. Tulving (1985) introduced the concept that different types of awareness corresponded with retrieval from different types of memory. In his seminal paper, Tulving (1985) linked semantic memory with noetic (knowing) awareness, and episodic memory with auto-noetic (self-knowing) awareness. Tulving

(1985) developed the remember/know paradigm empirically to assess the concepts of auto-noetic and noetic awareness. These concepts are operationalised by retrieval from episodic memory. The experience of 'reliving' the memory (remember), corresponds to auto-noetic awareness, whilst knowing, retrieval without an experiential component, corresponds to noetic awareness. There is an extensive literature regarding these concepts (see Yonelinas, 2002, for a review), and they have been shown to be dissociable processes with differing neural substrates; converging evidence suggests that remembering and knowing are associated with activation in separate regions of the medial temporal lobes (Yonelinas, 2002).

#### 1.3.4. Clinical Approaches to Awareness.

The clinical literature has focused on deficits in awareness of specific functions found in a number of patient populations: Alzheimer's disease (Clare, 2004a, 2004b), acquired and traumatic brain injury (Prigatano & Schacter, 1991), Parkinson's disease (Leritz et al., 2004) and schizophrenia (Medalia & Lim, 2004). Babinski (1914) first introduced the term anosognosia (*a*, without; *noso*, disease; *gnosia*, knowledge) to describe a lack of awareness of hemiplegia in stroke patients. The term is now more commonly used to indicate a lack of awareness of impaired neurological and/or neuropsychological functions (McGlynn & Kaszniak, 1991). There has been increased interest in anosognosia in Alzheimer's disease (AD) in particular, over recent years (e.g. Clare, 2004a, 2004b). One consistent finding is that anosognosia is a common feature of AD and is present in the early stages of the illness (Correa, Graves & Costa, 1996; Dalla Barba, Parlato, Iavarone & Boller, 1995; Derouesne et al., 1999; Vogel et al., 2004). A lack of awareness of deficits in AD may delay self-referral to health care professionals (Carr, Gray, Baty & Morris, 2000), reduce the effectiveness of cognitive rehabilitation interventions (Clare, Wilson, Carter, Roth & Hodges, 2004; Koltai, Welsh-Bohmer & Schmechel, 2001), contributes to caregiver burden (DeBettingnies, Mahurin & Pirozzolo, 1990; Seltzer, Vasterling, Yoder & Thompson, 1997), and may lead to the continued performance of potentially dangerous behaviours (e.g. poor car driving; Rizzo, Reinach, McGhee & Dawson, 1997).

A variety of conceptualisations, operationalisations and methodologies has been employed to measure anosognosia and may, in part, account for inconsistent findings. Several

methods have been employed in the clinical literature for investigating awareness of cognitive functioning: clinician ratings; questionnaires; and, combinations of these methods. Discrepancies between patient and carer ratings and between prediction and performance are also frequently employed as indices of awareness (see Clare 2004b, for a review).

Impaired awareness of numerous domains has been identified: memory functioning (Eslinger et al., 2005; Vasterling, Seltzer, Foss & Vanderbrook, 1995), cognitive and behavioural functioning (Migliorelli et al., 1995; Starkstein, Sabe, Chemerinski, Jason & Leiguarda, 1996), activities of daily living (DeBettignies, Mahurin & Pirozzolo, 1990), naturalistic action errors (Giovannetti, Libon & Hart, 2002) and metacognition (Gil et al., 2001). These findings indicate that deficits in awareness appear to be domain specific rather than generalised. Behavioural correlates of anosognosia have also been found in relation to organic disease severity ( $r = .60$ ; Kashiwa et al., 2005), apathy ( $R^2 = .48$ ; Derouesné et al., 1999), social skills and behaviours ( $r = .52$ ; Seltzer, Vasterling, Yoder & Thompson, 1997) and frontal lobe functioning ( $r = .70$ ; Michon, Deweer, Pillon, Agid & Dubois, 1994). However, there are highly divergent results, in particular with regard to depression and frontal lobe functioning (e.g. Barrett, Eslinger, Ballentine & Heilman, 2005; Dalla Barba, Parlato, Iavarone & Boller, 1995). The relationship between anosognosia and depression is complex, as some studies fail to find a relationship between anosognosia and depression (e.g. Barrett et al., 2005; Michon et al., 1994), whilst others have found negative correlations indicating that depression decreases as anosognosia increases (e.g. Kashiwa et al., 2005; Smith, Henderson, McCleary, Murdock & Buckwalter, 2000). These results suggest that greater insight into the condition may be of therapeutic benefit. It is also conceivable, however, that greater knowledge about a condition may lead to more negative mood.

Similar discrepancies are found in the literature investigating frontal lobe functioning and anosognosia. Michon et al. (1994) created a frontal score from performance on the Wisconsin Card Sorting Test, verbal fluency and a graphic series task and found that greater anosognosia was significantly associated with worse performance on these tasks. A similar finding between verbal fluency and awareness of memory abilities ( $r = .56$ ) was found by Dalla Barba et al. (1995), however, no other relationships were found between anosognosia and other frontal lobe tasks (Modified Card Sorting Test, cognitive estimates

and Graphic Sequences). In contrast, Barrett et al. (2005) required participants to predict their performance pre- and post-testing on a range of neuropsychological tasks assessing multiple domains of function (naming, visuo-spatial skill, memory, limb praxis, attention and generative behaviour). Barrett et al. (2005) calculated an anosognosia ratio for each group (controls and AD) by (i) subtracting estimated from actual performance percentages and (ii) adding estimated and actual performance percentages, then dividing these two numbers (i/ii) to provide a ratio score between  $\pm 1$ , with 0 representing perfect awareness. The authors found significant differences between the ratios of the groups on pre-test visuo-spatial skill prediction and post-test memory postdicton. Interestingly, Derouesné et al. (1999) failed to find relationships between anosognosia and the Wechsler Memory Scale, verbal fluency or WAIS similarities. Although Derouesné et al. (1999) found that those with anosognosia presented with more behavioural signs consistent with frontal lobe dysfunction, there was no significant difference in awareness in those who reported one or more behavioural signs and those who reported none.

#### 1.3.4.1. Neuropsychological Models of Awareness.

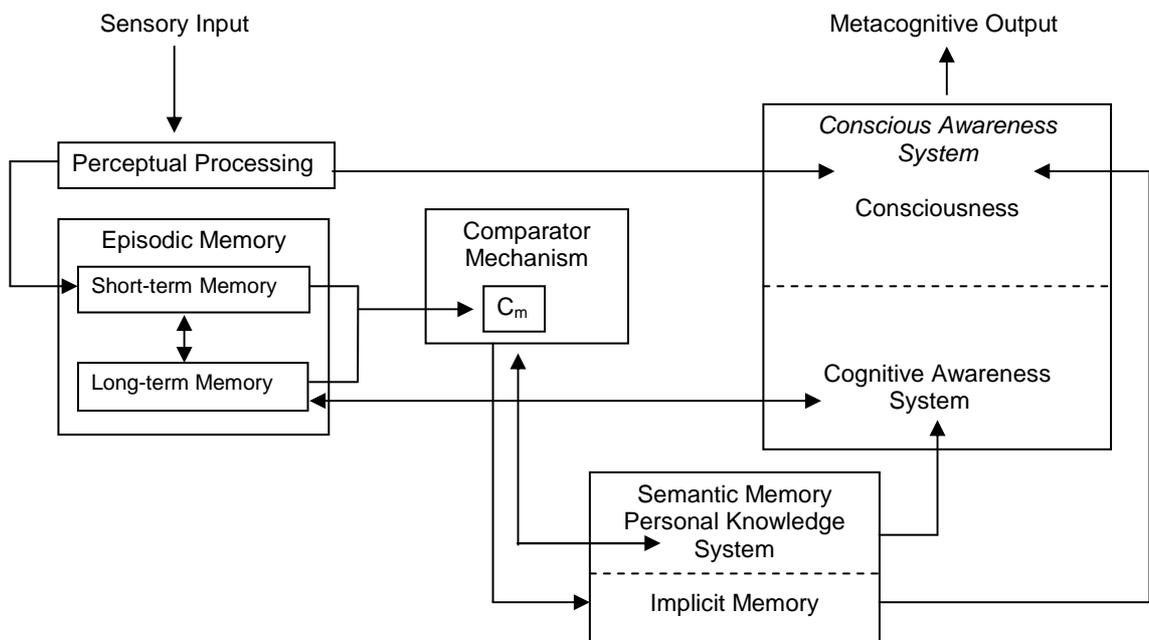
Neuropsychological models of awareness have been developed from the clinical assessment of patients presenting with deficits in awareness of level of cognitive functioning, particularly those with dementia. Neuropsychological assessment is undertaken to measure the degree to which cognitive domains are affected by neurodegenerative diseases.

##### 1.3.4.1.1. Neuropsychological Models of Awareness in Alzheimer's Disease.

Agnew and Morris (1998) reviewed the literature on anosognosia in AD and developed a cognitive model to account for the heterogeneous presentation of anosognosia (see Figure 1.1). This model suggested possible mechanisms involved in memory functioning, and identified ways in which deficits may influence metacognition. The authors proposed that deficits at various, crucial points within the model could result in three types of anosognosia. Mnemonic anosognosia refers to failure to revise the personal knowledge base (PKB): a mismatch concerning functioning is detected, but the mismatch is not encoded in the PKB. However, due to the links between the comparator mechanism ( $C_m$ )

and consciousness via implicit memory, the mismatch may be encoded into implicit memory; this implicit knowledge of impaired functioning may be exhibited behaviourally (e.g. by avoiding tasks relying on the impaired function). Executive anosognosia refers to the perception of an error, but due to a failure of the  $C_m$ , a comparison with the PKB does not occur; a mismatch signal is not created and, therefore, there is no output from the comparator mechanism such that there is no communication about a deficit into memory. Primary anosognosia is global unawareness of impaired cognitive functioning due to a deficit in the Conscious Awareness System, despite communication of impairments from the  $C_m$  and the PKB. However, negative affect may be experienced due to the link between implicit memory and consciousness. The model has only recently been directly tested (see Ansell & Bucks, 2006; Graham, Kunik, Doody & Snow, 2005), with limited support being found for mnemonic anosognosia; in that the revision of the PKB does appear to be affected with AD. However, Ansell and Bucks (2006) found that older adults were able to update their memory predictions, indicating that this type of awareness deficit was specific to AD.

Figure 1.1. A Cognitive Model of Anosognosia of Memory Impairment (adapted from Agnew & Morris, 1998).



Clare (2004b) presented a biopsychosocial model of awareness in AD in her review and included neurological, neuropsychological, psychiatric and psychosocial perspectives. Clare (2004b) proposed that the self was “the unifying context for experience over time” (p. 169), a cognitive schema, and socially constructed, with age, personality, coping styles, norms and experience influencing the self. The model described biological, psychological and social dimensions of awareness, each of which included factors to be considered when examining an individual’s level of awareness. For example, the biological dimension included: domain-specific unawareness; executive unawareness; mnemonic, executive and primary anosognosia; damaged and/or disconnected Conscious Awareness System; and, general metacognitive deficit. The model posited that the biological and social dimensions influenced psychological aspects of awareness, with each of the dimensions interacting with different aspects of the self. For example, Clare (2004b) argued that AD threatened the sense of self, and combined with personality, coping styles and beliefs would influence the psychological dimension of awareness, which included the concept of denial. A dynamic interaction was posited between the social self and the social dimension of awareness, which included interactions with significant others, and opportunities for participation in activities and social roles. The model has integrated a wide range of concepts from diverse literatures and whilst comprehensive, has not been empirically assessed, probably due its complexity. Although the model was developed from existing literature, it does not offer testable hypotheses about awareness; rather it suggests possible relationships between different dimensions of awareness in AD.

#### 1.3.4.1.2. Neuropsychological Models of Awareness in Brain Injury.

Deficits in awareness alongside neuropsychological deficits are found in other neurological conditions (e.g. acquired and traumatic brain injury), lending validity to the close association between awareness and brain functioning. Whilst the previous neuropsychological models may be applied to multiple neurological conditions, they have been specifically developed from the literature regarding deficits in awareness in AD. There has been little comparison of deficits in awareness in differing patient populations (however, see Eslinger et al., 2005, for a comparison of awareness deficits in different types of dementia). Crosson et al. (1989) presented the Pyramid Model of Awareness, which was developed to account for deficits in awareness found in individuals with brain

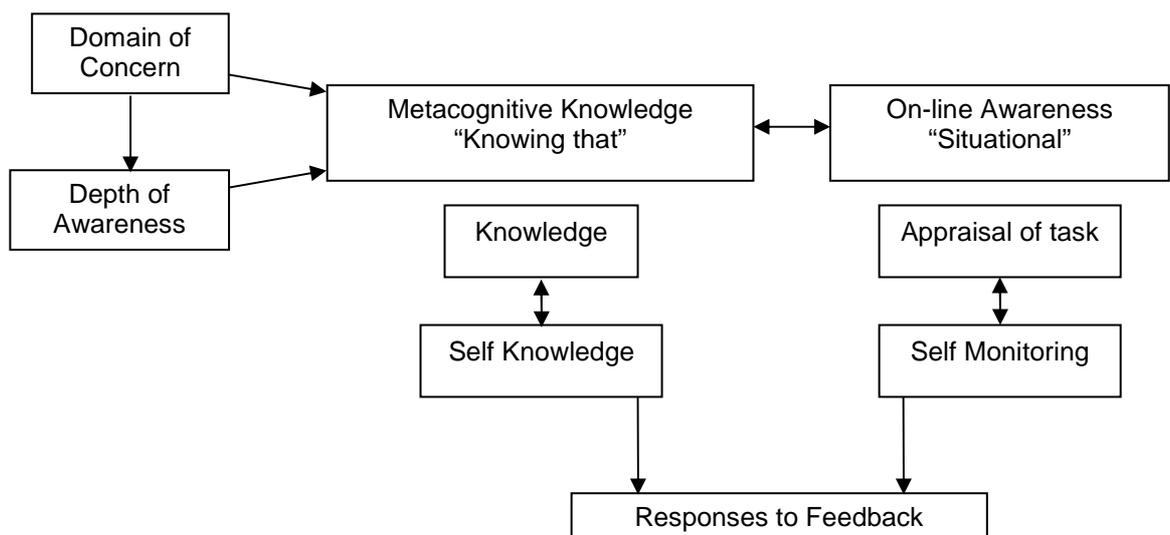
injury. The model describes three levels of awareness hierarchically arranged, with higher levels being dependent on lower levels. Specific deficits in awareness were outlined for each of the levels. Intellectual awareness at the bottom level, represents the awareness that there is impairment in some domain. Emergent awareness, at the middle level, is awareness that the impairment is a problem. At the top level, anticipatory awareness is the understanding that the impairment is causing the problem.

Little support has been found for the Pyramid Model of Awareness. For example, Abreu, et al. (2001) compared patients' predicted and actual performance as assessed by clinicians, on basic activities of daily living (ADL), meal planning and money management. Questions regarding their prediction (intellectual level), performance (emergent level) and assessment of performance effects on independent living (anticipatory level) were employed. The study found significant differences between the patients' different assessments and their actual performance for each of the tasks, with the exception of performance of basic ADL and their assessment of their actual performance. Two groups were created on the basis of their response to a question regarding their awareness of changes in abilities. Crosson et al. (1989) predicted in the Pyramid Model of Awareness that aware patients would have greater accuracy in their assessments. However, the groups (aware/unaware) in the Abreu et al. (2001) study were not significantly different on the discrepancy between the patients' predictions and the clinicians' ratings of performance. Both unaware and aware patients significantly overestimated their abilities.

Toglia and Kirk (2000) argued against the hierarchical structure of the Pyramid Model of Awareness and expanded the model to incorporate the dynamic relationships between awareness and performance (see Figure 1.2). The model separates metacognitive knowledge and on-line awareness, with the former relating to general knowledge existing prior to task engagement, whilst the latter is situational and activates with task performance. The Domain of Concern influenced the Depth of Awareness concept, as well as prior knowledge of task performance and associated strategies. Depth of Awareness influenced self knowledge and beliefs (intellectual awareness). The model proposed a dynamic interaction between prior knowledge and self knowledge. These constructs, in turn, interact with situational, on-line awareness, which was linked to anticipatory awareness (the understanding that the impairment is causing the problem), task experience, monitoring of cognitive state (emergent awareness) and self-evaluation. On-line awareness

was also influenced by cognitive deficits, fatigue, motivation and social influences. The model posited that self knowledge, the constructs of on-line awareness and the other factors, impacted on the individual's responses to feedback. Although the model does provide possible hypotheses about awareness and processes involved in awareness during task performance, the multitude of variables and interactions makes any empirical investigation difficult. However, O'Keefe, Dockree, Moloney, Carton and Robertson (2007) investigated Toglia and Kirk's (2000) model, and employed a multidimensional approach to assess awareness in individuals with traumatic brain injury. This study examined different types of awareness, using a variety of measures to operationalise each type, with 31 adults with traumatic brain injury (mean age 28.74, SD 8.52 years) and a control group of 31 adults, matched on sex, age and level of education. This study found that metacognitive knowledge about personal abilities was not related to either emergent or anticipatory awareness. However, there was a highly significant relationship between emergent and anticipatory awareness ( $r = .72$ ), indicating that better monitoring of errors during a sustained attention task was associated with more accurate prediction of performance. These findings indicate a separation between metacognitive knowledge and on-line monitoring, supporting Toglia and Kirk's (2000) conceptualisations of these different types of awareness.

Figure 1.2. Model of Awareness. Adapted from Toglia and Kirk (2000).



In summary, each of the neuropsychological models reviewed above has attempted to conceptualise different types of awareness into a framework and has incorporated existing metacognitive evidence to delineate processes that may underlie deficits in awareness of various domains. However, these models have been developed to account for evidence of awareness deficits in patient populations, and have rarely been empirically tested, limiting conclusions that can be drawn about their utility to an investigation of awareness in normal ageing. An investigation of these models considering both age and disease, particularly as AD is a disease of ageing, would contribute to an understanding and separation of the effects of neurological diseases on awareness.

#### 1.3.4.2. Neurological Models of Awareness.

The advent of brain imaging techniques has provided opportunities directly to investigate functional neuroanatomy. The following section describes two prominent neurological models of awareness, both of which have been developed from brain imaging studies indicating that injury is associated with deficits in different types of awareness. The value of the neurological models is in the linking of different types of awareness with underlying neural architecture. Although the neuropsychological models provide frameworks to account for the presentation of deficits in awareness, they do not specify the associated neuroanatomy.

Most of the neuroimaging work has been conducted since the mid 1990s. Wheeler et al. (1997) reviewed positron emission tomography (PET) studies, which suggest areas of relative metabolic activation in the brain, providing evidence for the significant role of the frontal lobes in autonoetic awareness (awareness associated with experiential remembering). Stuss, Picton and Alexander (2001; described in more detail in Section 2.6.2, Chapter 2) developed a model to account for the deficits in awareness often found in patients with brain lesions, incorporating current knowledge of neural systems and their functioning. The authors suggested that awareness arose from “processes that construct models of the world” (Stuss et al., 2001, p. 101). Different types of awareness are associated with processing at different neural levels, which are hierarchically arranged. The concept of mental representations (models) is similar to that proposed by the social psychological theories of self-awareness and, again, is a vague explanation of neural

processing that does not lend itself to empirical testing. The value of Stuss et al. model, however, is in the separation of different types of awareness, and the description of the processes involved in each type of awareness, which can be more easily tested, and the association of these components with functional neuroanatomy.

The lowest level of the Hierarchies of Processing model (HoP; Stuss et al., 2001) is Arousal and relates to from coma to waking state. The second level, Sensorimotor Awareness results from the processing of sensory information in the posterior regions of the brain. The final two levels of the model involve processing within the frontal lobes. Consistent Consciousness, at the third level, relies on executive functioning and the ability to organise sensory information underlying behaviour. Self-Awareness is the highest level in the model, and involves accurate monitoring of the self and personal abilities.

The second neurological model of awareness is more simplistic than that of Stuss et al. (2001). Based on the review of consciousness by Zeman (2001), Prigatano and Johnson (2003) proposed a hierarchical model of consciousness to account for deficits in self-awareness found in traumatic brain injury. These authors, similarly to Stuss et al. (2001), proposed that the neural circuits underlying different types of consciousness interact. Vector 1 of this model represents the waking state, the second vector relates to the phenomenological sense of self and the third corresponds with theory of mind; insight into the self and an understanding of others' mental states. Prigatano and Johnson (2003) identified specific structures and their interconnectivity underlying the different vectors. The brain stem and reticular formation and the thalamus underpin the waking state. The anterior and posterior cingulate, the prefrontal, posterior parietal, and lateral temporal cortices underlie the experience of being a 'self' and theory of mind. Whilst both models describe how neurological damage is involved in deficits of awareness, Prigatano and Johnson (2003) also suggested possible avenues of rehabilitation: medication can elevate energy levels during the waking state, thus improving engagement with tasks; teaching of coping strategies and performance feedback may assist those with some degree of accurate self-awareness; and, cognitive therapy may assist those with deficits in theory of mind.

In summary, the neurological models of awareness were developed to account for evidence that focal brain lesions produce deficits in different types of awareness, probably due to disruption to neural networks. The main contribution of the neurological models is the

suggestion that different types of awareness are hierarchically arranged and that neural processing at different levels can be level specific but can also impact on the interaction of processing at higher levels in the model. Despite the HoP model being developed to account for deficits in awareness found with brain injury, the descriptions of the processes underlying each type of awareness provide testable hypotheses appropriate for healthy ageing. Although there has been some investigation of two of the neuropsychological models (Agnew & Morris, 1998; Toglia & Kirk, 2000), each of the neuropsychological models reviewed previously (Agnew & Morris, 1998; Clare 2004b; Toglia & Kirk, 2000), assumes the presence of deficits in awareness associated with neuropathology (e.g. AD) or brain injury. Whilst ageing is associated with neural and cognitive changes, there is little evidence of impairments in awareness with increasing age. Moreover, only Ansell and Bucks (2006) and Graham et al. (2005) included healthy older adult control groups. Of particular note, Ansell and Bucks (2006) found that older adults were able to update their personal knowledge base, which indicated that ageing was not associated with mnemonic anosognosia, the failure to revise the knowledge base (Agnew and Morris, 1998). Critically, the definitions of awareness provided by Stuss et al. (2001) move from the generalised, for example, “when attention is directed inward and the individual’s consciousness is focused on himself” (Duval & Wicklund, 1972, p.2.), and somewhat problematic to assess empirically, to the specific. By accepting that awareness is composed of a number of hierarchically organised components, empirical investigation is facilitated by the specification of processes underlying types of awareness.

Although Stuss et al. (2001) have indicated a candidate neuroanatomy for the different types of awareness, the nature of on-line awareness has not been confirmed, partly due to the limited temporal resolution of most brain imaging techniques. The electroencephalogram (EEG), however, provides a measure of the electrical activity of the brain and has excellent temporal resolution, allowing for the investigation of brain functioning underlying processing of sensory information and cognitive functions in ‘real’ time. There is some discussion that event-related potential (ERP) components may be associated with awareness, for example, the error positivity (Pe) has been linked with the conscious awareness of an error (Nieuwenhuis, Ridderinkhof, Blom, Band & Kok, 2001; see also Chapter 5). In addition, other stimulus-locked ERP components reflect perceptual sensory processing and attentional engagement, which represent Stuss et al. (2001) second level of Sensorimotor Awareness, while other response-locked ERP components reflect

on-line performance monitoring and are associated with the third level of the Stuss et al. (2001) model.

#### 1.4. Summary.

This chapter began with a quote by Searle (1991) suggesting that if a definition of a process was not useful, the definition, not the existence of the process should be challenged. In retrospect, early philosophical approaches to consciousness and awareness appear ‘mystical’; however, they initiated a long tradition of debate into the nature of these subjective phenomena. Separation of mind and body was promoted in later years, and while this was based on a process of introspection and self-reflection, it effectively limited opportunities to develop testable definitions of consciousness and/or awareness. The beginnings of a testable account of awareness may be found in the Materialists’ response to Descartes: I am, therefore I think (Gibson, 2004).

It is important to reiterate that there are no definitive definitions within the literature, distinguishing consciousness from awareness. A popular primary definition of consciousness is ‘the state of being conscious’ (Oxford English Dictionary, 1989), whereas to be aware is to have ‘knowledge or perception of a situation or fact’. In other words, awareness implies perhaps a greater degree of cognitive involvement than simply being conscious, which may be more popularly used within a medical-context. Within the scientific community, attempts to distinguish between consciousness and awareness have also proved challenging. For example, Zeman (2006) discussed different meanings of consciousness: the waking state; self-consciousness, including self-monitoring and theory of mind; narrow and broad consciousness, the former relating to awareness involved in self-report, the latter to the acquisition of knowledge; inner and outer consciousness, the former being private; and the easy and hard questions of consciousness. Some contemporary philosophers and scientists (e.g. Chalmers, 1996; Searle, 2000; Wheeler et al., 1997) take the view that awareness is an aspect of consciousness, and seek no further clarification. Indeed, Koch and Crick (2000) studied visual awareness in their investigation of neural correlates of consciousness, apparently showing greater concern to emphasise that visual awareness was amenable to scientific investigation and provided a starting point for research into the neural correlates of consciousness, than to seek to distinguish between

consciousness and awareness. Based on this convention, the view taken in this thesis is that awareness is a feature of consciousness, that awareness is multidimensional, and that different aspects of awareness can be investigated through identifying and testing definitions of the processes underlying awareness (e.g. Stuss et al., 2001). Otherwise stated, rather than aim to provide new definitions of awareness or consciousness, it is the author's intention to review definitions of awareness applicable and appropriate for empirical assessment within healthy ageing, and to select a model within which the interpretation of empirical data may be explored. If it is assumed that consciousness and awareness are to a degree synonymous, then the use of the term 'awareness' rather than 'consciousness' becomes an arbitrary choice.

The critical underlying assumption for this thesis is that awareness is a function of the brain (cf. Koch & Crick, 2000; Searle, 2000), meaning that assessment of brain function will facilitate assessment of awareness. Moreover, social and experimental psychology has indicated that awareness may be separated into levels or component parts, such as error recognition. These two conceptualisations are the foundation of contemporary models of awareness (e.g. Stuss et al., 2001), and the basis for the interpretation of data presented in this thesis.

## Chapter 2. Neural and Cognitive Development Across the Lifespan.

“Age is simply a surrogate index of maturation and experience.”

Mabbott, Noseworthy, Bouffet, Laughlin and Rockel (2006, p. 937).

Brain development involves numerous processes that begin during the early gestational period and continue throughout the lifespan. There has been much focus on early development and later senescence, but less is known about the trajectory of brain development during the period between childhood and older age. The neural changes found in later life should not be viewed in isolation of earlier processes, as the division of the lifespan into distinct age bands can be misleading. Indeed, older adulthood represents an accumulation of the dynamic interaction between neural change and experience. It is for this reason that this discussion of brain and cognitive function in later life is set within the context of brain and cognitive development across the lifespan (see Sections 2.1 and 2.2). The purpose of this review is to demonstrate that any changes in awareness in older age occur against a background of more general brain and cognitive change. As awareness is defined as a multi-level brain function (Stuss et al., 2001), the extent to which change occurs in one or more of its component structures or functions may be explored.

### 2.1. Gross Neuroanatomy of the Brain.

There are five major divisions within the brain: the cerebral hemispheres; the thalamus and hypothalamus; the midbrain; the pons and cerebellum; and, the medulla. The latter four divisions collectively form the brain stem. The cerebral hemispheres are symmetrical and are linked by the corpus callosum, a large tract of white matter facilitating communication in the form of neural impulses between the hemispheres. The cerebral hemispheres are comprised of the cortex (outer layer), myelinated axons and subcortical structures and systems. The limbic system surrounds the thalamus and incorporates the cingulate cortex, the fornix, the hippocampus, the septum and the amygdala. The basal ganglia motor system is situated either side of the thalamus and includes the globus pallidus and the striatum which is in turn composed of the caudate and putamen (Pinel, 2003). Each hemisphere is subdivided into four lobes: frontal, parietal, temporal, and occipital.

## 2.2. The Cortex.

The gross laminar (columnar) organisation and function of the cortex is highly similar across individuals and is considered to be genetically determined (Rakic, 2002). The cortex is typically formed of six layers of cells, which provide the structure of the grey matter. Layer I is closest to the scalp and is mainly composed of axons and dendrites. Pyramidal cells form the majority of the cortex (layers II, III, V and VI) and are the output projection neurons to the spinal cord, brainstem and other cortical regions. Stellate cells are a second major group of neurons found in the cortex with shorter axons and an inhibitory function via neurotransmitters. There are regional variations in the cortical layers dependent on the function of the area. Layer V of the motor cortex is the largest, due to the pyramidal cells in this layer projecting from the cortex to the brain stem and spinal cord, allowing for motor function (Pinel, 2003). Some subcortical structures are also considered to be grey matter structures, due to the laminar organisation of the cell layers, for example the thalamus and basal ganglia (Alexander & Crutcher, 1990; Sullivan, Rosenbloom, Serventi & Pfefferbum, 2004; Xiao & Barbas, 2004).

## 2.3. Brain Connectivity.

The cortical regions do not function independently. Neural pathways allow for communication between the cortex and other subcortical structures including the limbic system, the basal ganglia, the midbrain, and the brain stem, to process sensory information, and store memories. This facilitates the high level of neural communication required for cognitive functioning and behaviour. White matter allows for the rapid transmission of neural impulses between brain structures. More specifically, axons provide the connectivity between neurons, and large numbers of myelinated axons provide the neural pathways between structures, as in the corpus callosum. A number of pathological and, more recently, neuroimaging studies has investigated brain connectivity in order to further the understanding of the relationship between structure and function (e.g. Alexander, DeLong & Strick, 1986; Behrens et al., 2003; Johansen-Berg et al., 2004; Parker et al., 2005).

Alexander et al. (1986) proposed that five neural circuits connect the cortex and the basal ganglia. Each circuit forms a feedback loop between specific regions of the cortex through specific regions of the striatum, pallidum, substantia nigra and the thalamus. These circuits are hypothesised preferentially to subserve certain functions. The motor circuit is involved in movement and may be further subdivided into more specific pathways for different limbs. The oculomotor circuit is involved in vision and eye movement. At the time of Alexander et al.' (1986) review, the functions of the prefrontal circuits were less well understood compared to the other circuits. However, it was suggested that the dorsolateral prefrontal circuit may be involved in spatial memory, whilst, based on primate lesion studies, the lateral orbitofrontal circuit was linked with perseveration. The anterior cingulate circuit includes projections from various regions of the limbic system; however, a function was not specified.

More recent functional neuroimaging studies have further elucidated the functions of the prefrontal circuits. The dorsolateral pathway has been linked with working memory, and is activated in conditions of high cognitive demand (Rympa & D'Esposito, 2003). The orbitofrontal cortex has been linked with the processing of reward and punishment, and, in a recent review, the lateral orbitofrontal circuit was specifically associated with the evaluation of punishment stimuli (Kringelback & Rolls, 2004). Both imaging and electrophysiological studies have indicated that the anterior cingulate cortex may be involved in performance monitoring processes (Carter, Braver, Barch, Botvinivk, Noll & Cohen, 1998; van Veen & Carter, 2002). The anterior cingulate cortex has been implicated in both cognitive and emotional processing, but isolating specific functions for this circuit is complicated due to its inter-connectivity with numerous other brain regions (Bush, Luu & Posner, 2000).

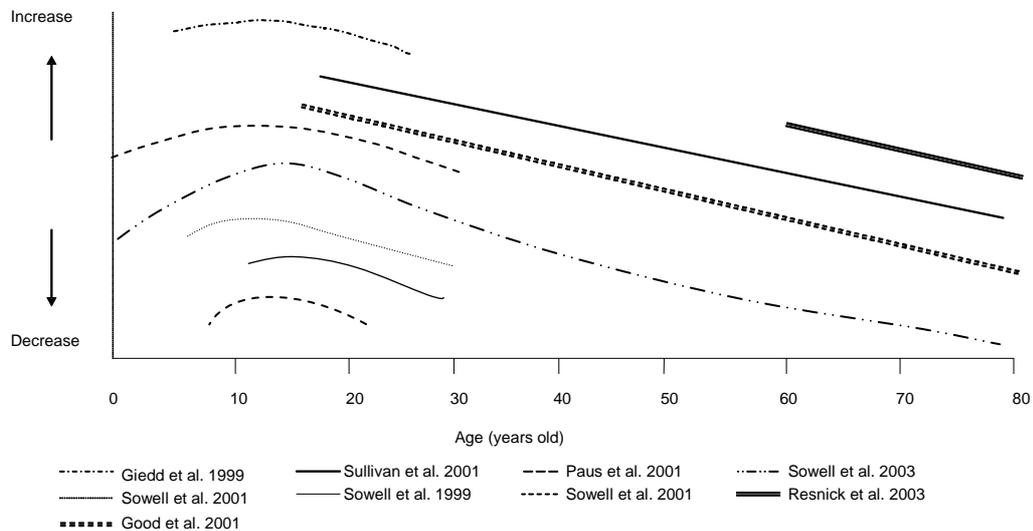
#### 2.4. Overview of the Human Brain across the Lifespan.

At birth, the infant brain appears similar to that of an adult. However, a number of processes begin during gestation and continue into the postnatal period to modify the maturing brain: the growth of axons and dendrites (cell differentiation), the establishment of inter-connectivity with other cells (synaptogenesis), the pruning of redundant synapses and the formation of myelin along the axons (Joseph, 2000; Webb, Monk & Nelson, 2001).

The gestational and childhood period is characterised by overabundant production of cells and synapses, but also by the subsequent pruning of connections that are less optimal than others. As development progresses, neural pathways are moulded by the input of sensory experiences (e.g. Hebb, 1948), suggesting a dynamic relationship between the genetically determined cortical structure (Rakic, 2002) and the environmentally influenced connectivity between and within cortical regions. The neural pathways that are strengthened are those which transmit neural impulses most efficiently, and are dependent on incoming sensory stimuli (Forssberg, 1999; Grubb & Thompson, 2004; Katz & Shatz, 1996). Synaptic plasticity is an essential process in the continuing development of the human brain (Martin, Grimwood & Morris, 2000) and for adapting to the dynamic demands of the environment. The processes involved in plasticity underlie the development of higher cognitive functions; the ability to maintain and restructure cortical networks is fundamental to learning, memory and self-awareness, and continues into later life (Arendt, 2001; Martin et al., 2000).

Grey matter volumes continue to increase well into adolescence, however these increases are regionally specific. Giedd et al. (1999) found that grey matter volumes were maximal at age 12 for frontal and parietal cortices, age 16 for the temporal cortex and age 20 for the occipital cortex. Following adolescence there are regionally specific grey matter volume decreases (Giedd et al., 1999; Paus et al., 2001; Sowell et al., 2003; Sowell, Thompson, Holmes, Jernigan & Toga, 1999; Sowell, Thompson, Tessner & Toga, 2001; Sowell, Trauner, Gamst & Jernigan, 2002; Sullivan, Rosenbloom, Serventi & Pfefferbum, 2004). This is illustrated in Figure 2.1. This schematic is intended as a representation of published data, as different volumetric measures were used in each study. As grey matter has differential rates of decline, the schematic presents data from various cortical regions. Although the decreases in grey matter are regionally specific, and grey matter volumes change in a non-linear pattern (see Gogtay et al., 2004), there is an overall decline in grey matter from adolescence.

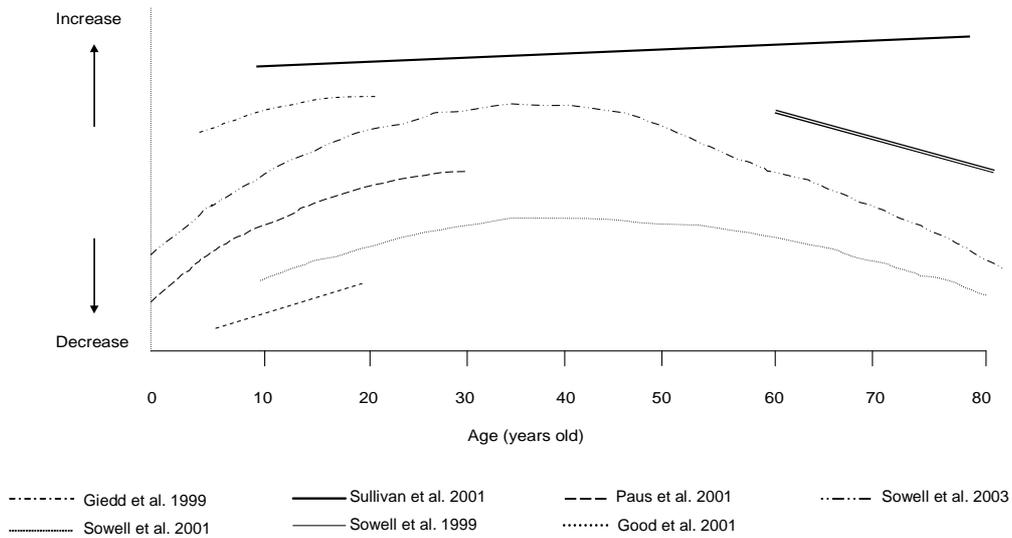
Figure 2.1. Schematic of grey matter changes over the lifespan.



Conversely, white matter density (myelin) increases in a linear pattern until middle age and then declines (Giedd et al., 1999; Paus et al., 2001; Sowell et al., 2003; Sowell et al., 2002; Sullivan et al., 2004). This is illustrated in Figure 2.2; as with the previous figure, the schematic is a representation of the data. There have been variable findings with regard to white matter changes in later life. Numerous variables can influence studies: imaging techniques; age range of the sample; and, regions of interest. For example, the three studies including wide age-ranges (18-79 years; Good et al., 2001; 7-87 years; Sowell et al., 2003; 20-85 years; Sullivan et al., 2004) did not indicate how many participants were in each age group, thus it is not known how many participants feature in the later decades.

Resnick, Pham, Kraut, Zonderman and Davatzikos (2003) found annual volume decreases of  $2.4 \pm 0.4$  and  $3.1 \pm 0.4$  cm<sup>3</sup> for grey and white matter, respectively, over a five year period in a sample aged 59–85 years. Sohmiya, Tanaka, Aihara, Hirai and Okamoto (2001) found that the volume of the midbrain decreased with age (age range 12-94 years). Good et al. (2001) found, in a sample of 465 participants (age range 18-79 years) using magnetic resonance imaging (MRI), significant reductions in grey matter with ageing ( $R^2 = .49$ ). Although Good et al. (2001) found that overall white matter volumes did not decrease as a function of age, regional losses were found in frontal white matter and in the posterior

Figure 2.2. Schematic of white matter changes over the lifespan.



internal capsule. However, again it is not known how many participants feature in the later decades. Salat et al. (2005) employed diffusion tensor imaging (DTI) to assess white matter as decreases in myelin and changes in its structure are found with ageing. Salat et al. (2005) found a strong relationship between fractional anisotropy, a measure of the structure of white matter derived from the pattern of flow of water molecules along tracts, and ageing ( $r = -.75$ ), with significant differences in white matter microstructure between younger and middle aged and older adults. Supporting the findings of Good et al. (2001), regional differences in myelin degeneration were found in the frontal white matter, posterior internal capsule and the genu of the corpus callosum (Salat et al., 2005).

Widespread neuronal loss has been a prevailing explanation for grey matter losses in later life (e.g. Coleman & Flood, 1987). However, Morrison and Hof (1997) reviewed the evidence for this hypothesis and concluded that ageing was not characterised by significant neuronal loss. In addition, Scheff, Price and Sparks (2001) focused on synaptic density in the superior-middle frontal cortex and did not find a decrease in synapses between the ages of 20-89 years. Scheff et al. (2001) further investigated the relationship between synaptic density and the later age groups (50-89 years) and, again, did not find a significant decrease in synaptic volumes in these older age groups. Nevertheless, there is some consensus that one of the features of ageing is a decrease in total brain volume (Raz, 2000,

2004; Resnick et al., 2003; Sohmiya et al., 2001). Raz (2004) reviewed MRI volumetric studies and extended a previous extensive review (Raz, 2000) by including data from participants < 30 years and > 60 years. He found that the prefrontal cortex (PFC) displayed the greatest reductions in grey matter with ageing ( $r = -.58$ ), followed by the striatum ( $r = -.47$  for the caudate,  $r = -.44$  for the putamen), the temporal lobes ( $r = -.35$ ), the hippocampus ( $r = -.34$ ), the cerebellar hemispheres ( $r = -.33$ ) and the parahippocampal gyrus ( $r = -.27$ ). Interestingly, the anterior cingulate gyrus, which has been linked with a number of cognitive functions including performance monitoring (e.g. Gehring & Knight, 2000), showed little change with ageing ( $r = -.17$ ). Increases in cerebrospinal spaces have also been found with ageing (Good et al., 2001; Raz, 2000; Resnick et al., 2003; Sohmiya et al., 2001), which may in part account for the inconsistent findings of age-related structural changes.

There are numerous possible factors which may contribute to an explanation of these neural changes. The distinction between grey and white matter in MRI scans is not straightforward, as there are a number of cellular components. For example, Paus (2005) suggested that as much as one third of grey matter 3-D pixels ('voxels') will show a white matter signal. Secondly, the reduction in grey matter earlier in the lifespan is considered to correspond with the normal, adaptive process of synaptic pruning rather than the processes of cell death (Paus, 2005; Sowell et al., 2003; Sowell et al., 1999; Sowell et al., 2001; Sowell et al., 2002). An alternative explanation for the decreases in grey matter in late adolescence is that the axons within layers V and VI of the cortex become myelinated, thus white matter proportionally increases (Paus, 2005; Sowell et al., 2003; Sowell et al., 1999; Sowell et al., 2001; Sowell et al., 2002). Although some studies (e.g. Scheff et al., 2001) suggest that there is not widespread synaptic and neuronal loss in later life, gross structural atrophy does appear to be a feature of ageing, and may be related to cell shrinkage and myelin degeneration (Raz, 2004). However, drawing such conclusions from cross-sectional studies is not without difficulties; age and cohort effects, and variable methodologies, measurement techniques, and statistical approaches may all confound interpretation. Cross-sectional studies provide percentage differences between different age groups, but cannot inform on age-related changes. Conversely, longitudinal studies place high demand upon participants, and are subject to high rates of attrition, reducing statistical power. Longitudinal study participants may also represent a select group within society (Raz, 2004).

In summary, the brain continues to develop and change from gestation throughout the lifespan. Grey matter increases in a non-linear, regionally specific pattern until adolescence and then decreases. Conversely, white matter continues to increase in a linear manner and is maximal during middle age after which it decreases. In addition to the gross structural changes found in ageing, a number of neuropathologies are also features of ageing; white matter hyperintensities (WMH; Deary, Leaper, Murray, Staff & Whalley, 2003), neuritic plaques and neurofibrillary tangles (Anderton, 2002; Morrison & Hof, 1997, 2003), and argyrophilic grains (Keller, 2005). However, the presence of neuropathologies significantly varies within the older adult population. Raz (2004) suggested that other neurochemical, hormonal and disease factors should also be considered, as the interaction between the various factors may contribute to the cognitive performance declines found in later life, particularly as the gross structural changes account for a small proportion of the variance in cognitive performance (Raz, 2004). More recent evidence reviewed by Raz and Rodrigue (2006) found that aerobic exercise had a protective effect on cognition in ageing, whereas estrogen which had been thought to be a protective hormone, may interact with inflammatory processes and lead to poorer memory functioning and smaller hippocampal volumes (Raz & Rodrigue, 2006). This literature is still in its infancy and time will undoubtedly lead to a greater clarification of the possible moderating roles of ageing related systemic illness (e.g. cardiovascular disease and hypertension), life style, and medications on neural and cognitive functioning in ageing.

## 2.5. Overview of Cognitive Development over the Lifespan, and its relation to Brain Development.

The development of cognition is accelerated in childhood, remains relatively stable throughout adult life, with progressive, but gradual decline with senescence (Craik & Bialystok, 2006). However, as with neuropathology, there is significant inter-individual variation in cognitive abilities, particularly in later life. Although a number of theorists have posited that cognitive development is closely related to the development of the brain in childhood (e.g. Fischer & Silvern, 1985; Hebb, 1949, Luria, 1973), the exact neural basis of cognition is not established, and any relationship between different brain structures and cognitive functions is likely to be highly complex. In older adults there is little

evidence to relate gradual decline in neural function with that of cognition. Hebb (1949) stated that larger neural networks are required in childhood to process the vast amount of information that has to be learned efficiently during this time. Thus, greater IQ may be expected to correlate with brain size. However, by maturity, the brain is composed of a smaller number of more efficiently working networks, so brain volume is not necessarily the foundation of greater IQ in adults; this may partly account for the lack of strong correlations between brain structure and cognitive function in older adults. Studies utilising structural MRI data are reviewed first, followed by studies of functional brain imaging (e.g. functional magnetic resonance imaging: fMRI; positron emission tomography: PET).

### 2.5.1. Studies based on Structural Brain Imaging.

Reiss, Abrams, Singer, Ross and Denckla (1996) found that IQ increased with cerebral volume ( $R^2 = .21$ ) during late childhood, reached a plateau and thereafter decreased: the largest volumes were found in those aged  $10.6 \pm 2.9$  years and  $10.7 \pm 2.8$  years (females and males, respectively). After controlling for age and gender, only prefrontal grey matter volume was a significant predictor of IQ (Reiss et al., 1996). Sowell et al. (2004), using a cross-sequential design with a sample aged 5 - 11 years with a 2 year interval, found improvements in vocabulary were significantly related to grey matter thinning in the left lateral dorsal frontal and left lateral parietal regions. Whilst Nagy, Westerberg and Klingberg (2004) found significant relationships between white matter and visuo-spatial working memory in the left superior fronto-parietal region ( $r = .54$ ), left inferior frontal region ( $r = .55$ ), corpus callosum ( $r = .58$ ), and left temporo-occipital region ( $r = .58$ ) in a sample aged 7.8 - 18.5 years. White matter in the left temporal region significantly correlated with reading ability ( $r = .44$ ). Nagy et al. (2004) suggested that the maturation of white matter increases conduction speed: functional improvements are dependent on increased efficiency of neural transmissions. More recently, Mabbott, Noseworthy, Bouffet, Laughlin and Rockel (2006) employed hierarchical regression analyses to separate the effects of age and white matter maturation, as measured by fractional anisotropy, on information processing speed in a sample of 17, 6-17 year olds. These authors found white matter maturity in the right frontal, right temporal and frontal-parietal regions uniquely contributed to visual-spatial searching ( $R^2 = .81, .81, .79$ , right frontal, right frontal-parietal and right temporal regions, respectively), indicating that white matter maturity rather than

age per se was associated with improvements in processing speed. Hogan, Vargha-Khadem, Kirkham and Baldewag (2005) suggested that task performance was more dependent on the increasing *efficiency* of neural networks during adolescence than on gross structural changes which were likely to be limited by this time. It is likely that increased efficiency is related to the increases in myelination, a process that allows for improved connectivity and communication between neural assemblies (Paus, 2005), suggesting that the functional integrity of white matter contributes to cognitive functioning (see also Mabbott et al., 2006). Of note, Menon, Boyett-Anderson and Reiss (2005) found a significant reduction in activation in the left medial temporal lobe (MTL) with age ( $R^2 = .52$ ), and significant increases in white matter connectivity between the left entorhinal cortex and the left dorsolateral PFC ( $R^2 = .57$ ), supporting the suggestions of Nagy et al. (2004) and Hogan et al. (2005).

Research into age-related changes in cognition has typically focused on childhood, adolescence and later life, due to the dynamic cognitive and neural changes occurring particularly during these ages. Although there are cognitive and neural changes in middle age (e.g. Salat et al., 2005; Schroeder & Salthouse, 2004; see also Figures 2.1 and 2.2), it is not a period of great change. It is possible that in order to obtain significant correlations, many authors have focused on the periods in which there are considerable cognitive and neural changes, namely childhood, adolescence and later life; and perhaps that a lack of positive correlations may have limited the drive for publication of data from the middle age groups. However, there are some data available, for example, Schroeder and Salthouse (2004) found that memory, spatial awareness and reasoning were relatively stable until age 35 and then decreased, whereas vocabulary increased with age, in an aggregated sample of 5391 participants. Annual rates of change (standard deviation units) of  $-.02$  and  $-.02$  for memory and spatial awareness/reasoning respectively and an increase of  $.05$  for vocabulary were found. Schroeder and Salthouse (2004) suggested that increased knowledge may counteract the declines found in other aspects of cognition. However, it was not clear how this might work, and the relationship with underlying brain function was neither investigated nor implied.

Although a range of cognitive functions may be constrained with age, there is also a significant proportion of older adults who do not display decline in memory or other cognitive abilities (e.g. Collie et al., 2001; Silver, Newell, Brady, Hedley-White & Perls,

2002; Snowdon, 1997). Raz, Gunning-Dixon, Head, Dupuis and Acker (1998) investigated the neural bases of cognition in a sample aged 18 - 77 years, and found that perseverative errors and responses on the Wisconsin Card Sorting Task (WCST) increased with age ( $r = .40$  and  $.40$ , respectively), and performance on the other measures (e.g. listening span, spatial relations test, paired associate learning, line patterns recognition test and word-stem completion) decreased (range  $r = -.49$  to  $-.21$ ). The increases in WCST perseveration were correlated with volume decreases in the PFC, limbic and visual cortices ( $r = -.42$ ,  $-.23$ ,  $-.29$ , respectively), volume decreases in these regions also correlated with nonverbal working memory ( $r = .29$ ,  $.21$ ,  $.33$ , respectively). Better explicit, nonverbal memory performance was associated with larger PFC and visual cortex volumes ( $r = .20$  and  $.21$ , respectively), and verbal working memory performance with PFC, limbic cortex and inferior parietal lobe volumes ( $r = .27$ ,  $.21$ ,  $.20$ , respectively). In summary, all measures suggest a relationship between poorer performance and reduced volume of brain structures. In a follow-up analysis with participants aged 60-77 years, limbic cortex volume was positively associated with explicit verbal and nonverbal memory performance ( $r = .44$  and  $.66$ , respectively), again suggesting that better performance was associated with larger brain volume. In another study, Van Petten et al. (2004) did not find any correlations between tests of executive function and frontal grey matter; although white matter hyperintensities (WMH; micro lesions) predicted 9% of the variance in executive functioning, and this is compatible with the view that changes in the brain are associated with changes in cognitive function. Nevertheless, other factors undoubtedly impact on executive functioning as indicated by the large amount of variance not accounted for. For example, there may be decline in cognitive domains that contribute to executive functioning, such as memory. Interestingly, however, Van Petten et al. (2004) found negative correlations between memory scores and the temporal cortex ( $r = -.33$ ) and the middle frontal gyrus bilaterally ( $r = -.42$ ), indicating that smaller volumes were associated with *better* performance. These authors suggested that the older adult brain is an accumulation of developmental processes and neural changes, and memory ability in later life may be more dependent on the effectiveness of this lifetime process, than on the current structure of the brain. Van Petten et al. (2004) further suggested that white matter changes may be more related to cognitive decline, due to reductions in cortical connectivity. In support, O'Sullivan et al. (2001) found that ageing was associated with disruptions in white matter structure, and that the structural integrity of white matter in the anterior regions was significantly correlated with attentional set-shifting and executive

functioning ( $r = .61$ ) and the mid-brain regions with verbal fluency ( $r = .61$ ). Moreover, these correlations based on white matter volume ( $>.6$ ) are generally stronger than those based on grey matter volume (described above: mostly  $<.5$ ).

More recent studies of white matter in ageing have used diffusion tensor imaging (DTI) to offer a measure of mean diffusivity, which indicates the degree of water diffusion in any direction, and complements fractional anisotropy (FA), a measure of the directionality of the flow of water along white matter tracts. Higher diffusivity values and lower FA values may reflect greater atrophy (e.g. Kochunov et al., 2007). Charlton et al. (2007) found that between the ages of 50 and 90 ( $n = 99$ ), FA significantly decreased in anterior, middle and posterior white matter, whilst mean diffusivity significantly increased. Working memory, executive function and processing speed were also found to decrease with increasing age. Each measure of white matter integrity in each region significantly correlated with each of the cognitive domains. However, once age and premorbid IQ had been controlled for, only the correlations with working memory remained significant (FA:  $r = .29, .20$ , middle and posterior regions respectively; mean diffusivity:  $r = -.30, -.24, -.26$ , anterior, middle and posterior regions, respectively).

Charlton et al. (2007) suggested that their findings support the “disconnection” hypothesis, the view that age-related changes and disruptions in white matter structure and tracts contribute to cognitive decline. Investigation of this hypothesis has been further facilitated by multiple measures of white matter integrity. For example, Deary et al. (2006) included FA, mean diffusivity and magnetisation transfer ratio (MTR), a measure of the degree of saturation of the water molecules, further to assess the relationship between white matter and cognition. The sample ( $n = 40$ , mean age 83) were a subgroup of participants from the Scottish Mental Survey of 1932, an initiative cognitively to assess all Scottish school children at age 11. This study, therefore, assessed an older adult population for whom information about level of cognitive function during childhood was available. Participants repeated the original cognitive tests alongside more recently standardised neuropsychological assessments; thus the study was partly longitudinal and partly cross-sectional. The study also found reduced FA and MTR and increased mean diffusivity with age, corresponding with the study by Charlton et al. (2007). Interestingly, only letter-number sequencing and verbal fluency were correlated with mean diffusivity, and choice reaction time variability (standard deviation: SD) with FA in the frontal regions. However,

mean diffusivity and FA in the centrum semiovale were related to a wider range of cognitive functions: letter-number sequencing, verbal fluency and general cognitive ability with mean diffusivity; and age 11 IQ, NART, MMSE, Raven Matrices, letter-number sequencing, verbal fluency, digit symbol, general cognitive ability, simple choice reaction time and SD and choice reaction time correlated with FA. No correlations were found between any measure of cognitive ability and MTR. The authors concluded that the coherence and organisation of the white matter tracts (as measured by mean diffusivity and FA) was more associated with cognitive functioning than volume of myelin (as measured by MTR). Furthermore, a path analysis was conducted investigating possible factors influencing cognitive ability at age 83. A direct relationship was found between IQ at age 11 and mental ability at age 83. Processing speed mediated the relationship between FA in the centrum semiovale and cognition at age 83, indicating that white matter integrity influences the efficiency of cognitive processing, which in turn improves cognitive function. These findings offer support for the “disconnection” hypothesis, and also have the potential to provide a specific explanation for the deficit in processing speed commonly found in older adults.

The relationship between WMH and cognitive functions has received greater attention, probably as these lesions may be seen on a MRI brain scan and do not require the sophisticated sequences of DTI. In a review, Gunning-Dixon and Raz (2000) found that a greater number of WMH correlated significantly with poorer performance on tests of executive function, ‘global’ functioning, processing speed, delayed memory and immediate-recent memory ( $r = .30, .22, .22, .20, .12$ , respectively), but not with fluid and crystallised intelligence, or motor function. The strength of association was small ( $r < .3$ ), suggesting that other factors may also account for level of cognitive function (e.g. educational level may be a better predictor than WMH: Soderlund et al., 2006), but it has been robustly demonstrated. For example, De Groot et al. (2000) also found that periventricular WMH were significantly correlated with decreases in memory, cognitive functioning and psychomotor speed in a large sample of 1077 participants aged 60-90 years. Interestingly, subcortical WMH were not found to influence cognition (De Groot et al., 2000). Periventricular WMH disrupt the main tracts between different cortical regions, whereas subcortical WMH affect shorter connections linking grey matter regions within the basal ganglia, and also affect short and long distance connections between cortical to subcortical regions, indicating that cognition may utilise widespread cortical circuits. Even

within cortical regions, differential effects of WMH on cognition have been found. Deep (medial) WMH were correlated with decreases in sequencing abilities and semantic memory ( $r = .29$  and  $-.29$ , respectively), and periventricular WMH with decreases in sequencing abilities and abstract reasoning ( $r = .33$  and  $-.33$ , respectively; Cook et al., 2002). WMH in both deep and periventricular regions were associated with reduced connectivity in rolandic networks, with negative impact on attention and processing speed (Cook et al., 2002).

In summary, significant age-related changes in brain structures are related to cognitive performance. In older adulthood, volume reductions in the PFC and limbic cortex have been specifically associated with memory declines (Raz et al., 1998). Conversely, smaller volumes of the temporal cortex and middle frontal gyrus bilaterally have also been linked with better memory performance (Van Petten et al., 2004). This indicates considerable complexity in the relationship between brain function and behaviour when age is taken into account. White matter lesions have also been associated with decline in several cognitive domains, providing support for the view that cognition is also supported by the connectivity between different neural regions.

### 2.5.2. Studies based on Functional Brain Imaging.

Functional neuroimaging studies allow for a direct association between structure and function. Such studies have found different patterns of activation between older and younger adults in a range of cognitive domains (see Raz, 2000, for a review), suggesting that brain regions and networks are used differently by younger and older adults. Perhaps the most frequently cited example of this is in relation to frontal lobe memory function. Neuroimaging studies of episodic memory in younger adults have found lateralised activations: encoding is associated with left PFC activation and retrieval with right PFC activation (Nyberg, Cabeza & Tulving, 1996). However, in older adults, there is symmetrical activation of the PFC during episodic memory tasks. In an update to the hemispheric encoding/retrieval asymmetry (HERA) model (Nyberg et al., 1996), Cabeza (2002) presented the hemispheric asymmetry reduction in older adults (HAROLD) model to account for the evidence that patterns of activation in older adults in a range of cognitive tasks are less lateralised than in younger adults.

A lack of lateralisation of function is also a feature of childhood cognition (see Hogan, Kirkham & Issacs, 2000), suggesting that late in life, as in early life, there is less division of function between the two hemispheres. However, there is no evidence to suggest that this is due to a similar process in each age group. There are a number of possible explanations for this finding in older adults, including global re-organisation of networks, the activation of compensatory mechanisms and/or a de-differentiation of functioning. Indeed, recruitment of additional neural networks may be necessary to compensate for increasingly limited attentional resources, reductions in processing speed and the breakdown of inhibitory processes (Cabeza, 2002). With regard to the HAROLD model, Tisserand, McIntosh, van der Veen, Backes and Jolles (2005) found that older adults had activation in the same areas during recognition compared to younger adults (prefrontal, premotor, lateral and medial temporal regions) to support encoding, but to the detriment of recognition task performance. Rajah and D'Esposito (2005) also found similar regions of activation during episodic memory tasks in both older and younger adults. These authors concluded that, despite regionally specific age-related changes, global PFC functioning remained relatively stable with ageing. It is possible that those areas of the brain that are particularly important for maintaining higher cognitive function are preferentially 'spared' at the expense of other areas. In support of this hypothesis, Persson et al. (2006) found a negative correlation ( $r = -.39$ ) between the structure of the anterior corpus callosum and activation in the right ventral PFC during an episodic memory task, in 20 older adults with stable memory performance ( $M$  age 66.1, 5.7 SD) and 20 older adults with memory decline, as assessed using longitudinal data ( $M$  age 65.3, 7.1 SD). This correlation indicated that lower FA in the genu (indicative of a reduction in the integrity of white matter) was related to increased activation in the PFC, which suggested that the increase in the signal may be a response to the structural changes found in the corpus callosum. Furthermore, Persson et al. (2006) found that the older adults with memory decline compared to older adults without memory decline had increased areas of activation in the left PFC during semantic categorisation. This provides further support for the proposition that greater frontal activation may be a response to structural changes; in the case of the Persson study greater frontal activation may have been a reaction to decreased hippocampal volume (measured by manual tracing) and decreased anterior corpus callosum volume (measured by fractional anisotropy on diffusion tensor imaging) (Persson et al, 2006). Taken together, the results from the functional imaging studies indicate that,

despite age-related changes in the structure of the brain, there is evidence of greater functional recruitment of brain areas, which may be compensatory (cf. Cabeza, 2002).

### 2.5.3. Summary of the Effects of Ageing on Brain and Cognition.

The considerable variation in cognitive performance and neuropathologies in later life contributed to the development of the cognitive reserve hypothesis, the proposition that there is a variable 'cognitive reserve', and that greater reserve has a protective function against cognitive decline in later life (see Stern, 2003). The increases in functional activation in ageing have been suggested to be compensatory (Cabeza, 2002), and may provide the neural correlates of cognitive reserve. However, it can be problematic to operationalise cognitive reserve, and a number of different methods have been employed such as: reading ability; early life idea density; and, the National Adult Reading Test (Richards & Sacker, 2003; Riley, Snowden, Desrosiers & Markesbury, 2005; Spitznagel & Tremont, 2004). More recent evidence has challenged the cognitive reserve hypothesis. Christensen et al. (2007) examined brain atrophy, WMH, levels of education, intelligence, creativity, and estimated cognitive decline in a sample of 446 adults aged 60-64 years. The study did not find any evidence that estimated cognitive decline was related to atrophy and WMH, nor that education, intelligence or creativity provided protection from estimated cognitive decline in those older adults with significant atrophy.

Ageing is associated with decline in a number of cognitive domains; however, there is variability in the presentation and degree of such 'deficit'. The cognitive domains that decline with age are those considered to rely most heavily on the frontal lobes (Moscovitch & Winocur, 1995; Fuster, 1989), suggesting that decline in cognition may in part be explained by alteration in the functional integrity of the frontal lobes (see West, 1996). The limited strength of the relationships between cognition and indices of frontal lobe change nevertheless indicate that other factors undoubtedly contribute to the deficits found in ageing. A decline in the integrity of white matter tracts has been linked with cognitive deficits (O'Sullivan et al., 2001), and WMH have been found to affect cognitive domains, such as processing speed and executive functioning (Gunning-Dixon & Raz, 2000). White matter degeneration and WMH have been found to correlate with reductions in processing speed, and the HAROLD model suggests that the recruitment of additional regions in task performance may counteract the reduction in processing speed found in older adults.

Reductions in the volumes of other neural structures and systems have also been linked with ageing. For example, the volume of the caudate declines with age, and Rubin (1999) suggested that the resulting disruption of the frontal-striatal network may account for the declines in inhibitory processes, processing speed and executive functions. Moreover, Erixon-Lindroth et al. (2005) found that cognitive decline in ageing was mediated by reductions in the striatal dopamine transporter system, supporting the view that the integrity of neural networks is fundamental to cognitive functioning. A number of cognitive factors have been found to account for significant age-related variance in task performance, for example, processing speed (see Salthouse, 1996) and general intellectual function (see Rabbitt, Lowe & Shilling, 2001). Although the neuroanatomical correlates of general intellectual function have not been widely investigated, it is highly probable that such a complex construct will involve the interaction of numerous neural assemblies and pathways. Deary et al. (2003) found that WM and periventricular lesions accounted for 14.4% of the variance in general cognitive ability in his sample of older adults.

In summary, cognition across the lifespan is dependent on the functional integrity of neural assemblies and their connective networks. The development of cognition in childhood and adolescence depends on the development and modification of neural networks and changes in the structure of cortical regions (e.g. Sowell et al., 2004). Cognitive ageing is associated with multiple structural and functional changes and it is likely that the combined effects of these changes, in addition to alterations in neurotransmitter systems, may contribute to the deficits in cognition found in later life (Buckner, 2004).

## 2.6. Awareness and Ageing.

In the previous chapter a range of conceptualisations, models and definitions of awareness were presented. One of the limitations was that there had been little empirical investigation of these models. Particularly important for the present discussion of possible change in awareness with ageing, is that few studies have included older adult control samples (but see Eslinger et al., 2005). It is clear, however, that there is significant decline in both brain and cognitive function with ageing. The metamemory literature has employed age as a quasi-experimental variable, and a large body of literature exists investigating processes involved in awareness and ageing. Following the review of this metamemory literature, will be a return to the Hierarchies of Processing model (HoP; Stuss et al., 2001), in order to

explore the extent to which awareness might continue to function, despite change in one or more of its component processes and/or levels.

### 2.6.1. Metamemory and Ageing.

The metacognitive literature has predominantly focused on metamemory in ageing, as memory functioning is one of the cognitive domains which has been shown to decline with age. An extensive literature investigating metamemory and ageing has examined the hypothesis that age-related differences in metamemory underlie age-related changes in memory performance. However, there has been little empirical support for this proposition (see Hertzog & Hultsch, 2000, for a review), indicating that the relationship between the metamemory dimensions, memory functioning and ageing is complex. The literature has identified a number of processes involved in awareness that are less effective in ageing. The findings of these studies provide support for the proposition that different types of awareness are differentially affected with age.

#### 2.6.1.1. Feeling-of-Knowing and Ageing.

Recent studies have employed the feeling-of-knowing (FOK) paradigm to investigate memory monitoring and its accuracy in ageing. The FOK paradigm requires participants to make a judgement about the likelihood that they will recall study stimuli prior to a recognition task. The accuracy of the FOK judgement is typically assessed with a gamma correlation; the strength of the relationship between the FOK judgement and actual recognition performance. The gamma correlation is an infrequently applied statistical tool, defined as a simple symmetric correlation that does not correct for tied ranks ([www.nyu.edu/its/statistics/Docs/correlate.html](http://www.nyu.edu/its/statistics/Docs/correlate.html)). The greater the gamma correlation, the closer the judgement of recall is to actual performance. Therefore, the gamma correlation is also a measure of the accuracy of the FOK judgement. Studies have found that the accuracy of FOK judgements and actual performance was reduced in older adults (62 older adults aged 61-89 years, 40 younger adults, aged 20-30 years; Perrotin et al., 2005; Souchay & Isingrini, 2004; 41 older adults aged 60-98 years, 20 younger adults aged 20-32 years; Souchay et al., 2000). These results indicate that older adults are less accurate in

their estimations of their ability to recognise study stimuli. Accuracy of judgements is related to awareness of memory functioning, and this has been shown to be reduced with ageing. However, executive functioning was found to mediate the age-related differences in FOK judgements (Perrotin et al., 2005; Souchay et al., 2000), whilst processing speed mediated the age-related differences in recall (Perrotin et al., 2005). Souchay and Isingrini (2004) investigated the role of self-paced study time on FOK judgements and recall performance with 24 older adults aged 56-96 years and 23 younger adults aged 20-31 years. The design of the study provided two recall test phases, one with the FOK judgements and one after a self-paced study stage; the same word pair stimuli were employed for both recall tasks. Both groups had improved performance after self-paced study time. Younger adults, however, had significantly greater recall than older adults for the second recall task. Although there was no difference between the groups in the amount of self-paced study time, there was an age-related difference in the amount of time given to the study of non-recalled words in the first recall task, indicating that older adults were less effective in their use of self-paced study time to improve their performance. The authors concluded that older adults had reduced awareness of how to utilise self-paced study time to improve performance.

#### 2.6.1.2. Judgements-of-Learning and Ageing.

The judgements-of-learning (JOL) paradigm is similar to the FOK paradigm in that participants are required to make a confidence judgement about their likelihood of recalling stimuli. Connor et al. (1997) investigated age-related effects on JOL in three experiments involving immediate and delayed JOL, cued by either a single stimulus word from a paired-associate task, or from the stimulus-response word pair (older adults:  $n = 34$ , mean age 71.1 years,  $n = 78$ , mean age 69.4 years and  $n = 34$ , mean age 65.5 years, younger adults:  $n = 60$ , mean age 20.2 years,  $n = 80$ , mean age 21.0,  $n = 30$ , mean age 20.1 years, experiments 1, 2 and 3, respectively). Additionally, they recorded global predictions and item-by-item predictions of recall performance. The second and third experiments involved minor changes in design: two exposures of the word pairs and instruction of strategy use for recall (Study 2), and stimulus only cues for the JOL, instruction for strategy use and postdictions of performance (Study 3). In all three studies older adults had significantly lower recall. Recall was greater for items with a delayed JOL, and older

adults predicted lower recall before study, with both groups lowering their predictions after study. However, only older adults significantly lowered their predictions after study. Younger adults were more accurate in their global predictions, whilst the older adults overestimated their recall performance. Mean JOL ratings found that younger adults accurately predicted their higher proportion of correct recall. However, in Study 2, there were no age differences in global predictions, which may be due to the repetition of the word pairs in this study, in that prediction of performance may have been influenced by their confidence of recall. In Study 3, older adults again made initial lower global predictions of performance, and again lowered their predictions after study, whereas the younger adults provided the same predictions before and after study, but increased their postdictions to correspond with actual performance. The correlations between actual recall performance and postdiction were significant (.68, .95, .92, .92, .80 and .80, older and younger adults for experiments 1, 2 and 3), and did not differ between age groups. In summary, the findings indicate that awareness as indexed by accuracy of mean JOL ratings did not differ between age groups. JOL were used to indicate the correspondence between confidence of recall and actual recall. Simply stated, older adults were able to revise their predictions after study indicating an awareness of their assessment of their memory capabilities. Notwithstanding this generally positive outcome, some effects of ageing were found indicating a degree of inaccuracy in their awareness of their level of memory functioning.

#### 2.6.1.3. Memory Monitoring and Ageing.

The metamemory literature has also employed predictions of performance as an index of memory monitoring in ageing (e.g. Ansell & Bucks, 2006; Bieman-Copland & Charness, 1994; Hertzog, Dixon & Hultsch, 1990; Rabbitt & Abson, 1991). Bieman-Copland and Charness (1994) found differential age-related effects on memory monitoring in two trials of word recall tasks with three types of experimental cues (letter, rhyme and meaning) in a sample of 36 older adults aged 59-79 years and 36 younger adults aged 18-28 years. There were no significant age differences in the predictions for both of the trials of each of the cue types. However, there were age-related differences in the accuracy of the predictions: older adults significantly overestimated their performance with letter and rhyme cues, but accurately predicted their performance with meaning cues, indicating that overestimation

only occurs for some stimuli in older adults. Whilst younger adults also significantly overestimated their performance with rhyme cues, they underestimated their performance with meaning cues and accurately predicted their performance with letter cues. Interestingly in the second trial, although older adults significantly reduced their predictions on all cue types, their performance was still significantly worse for the letter and rhyme cued word recall. Younger adults were increasingly accurate in their predictions of performance in the second trial, decreasing their rhyme cue prediction and increasing their prediction of meaning cued recall. These findings indicate that older adults are able to monitor their performance and adjust predictions on the basis of previous test experience; however, their predictions are still less accurate indicating less effective monitoring abilities. Ansell and Bucks (2006) gave three 10 word lists to healthy older adult controls ( $n = 18$ , aged 61-89 years) and individuals with AD ( $n = 18$ , aged 66-88 years), and compared predictions of performance prior to list viewing, post list viewing and actual performance. Although the study investigated awareness in Alzheimer's disease, it found that the healthy older adult controls underestimated their performance in the first list, and then overestimated their performance in the second and third lists. However, the magnitude of the difference between predictions and actual performance was very small ( $M = -.61, .53$  and  $.36$ , for the first, second and third list, respectively), indicating that older adults largely were accurate in their predictions and that the magnitude of the difference in their accuracy decreased over the lists.

The word lists utilised by Bieman-Copland and Charness (1994) were much longer than in the Ansell and Bucks (2006) study (54 and 45 words in list 1 and 2, respectively), indicating that accuracy of predictions may be influenced by task demands. Moreover, these data suggest that the updating of knowledge about memory functioning is less effective in older adults.

#### 2.6.1.4. Metamemory Beliefs and Ageing.

Self-referent beliefs about memory have also been examined. Self-referent beliefs are beliefs about ageing, memory functioning and attitudes towards memory change with age. Such beliefs are typically assessed by metamemory questionnaires, for example the Metamemory In Adulthood questionnaire (MIA; Dixon, Hultsch & Hertzog, 1988), and

relationships with age and with predicted and actual performance on cognitive tasks may be explored. Hertzog et al. (1990) investigated beliefs, performance predictions of three trials of word list and text recall tasks, with actual performance in a cross-section of 422 adults aged 22 -79 years. The study found that older adults predicted lower performance for both word list and text recall than younger adults, and had significantly reduced performance, however, all groups underestimated their performance. The MIA subscales Change and Capacity and the Frequency of Forgetting subscale of the Memory Functioning Questionnaire (MFQ; Gilewski, Zelinski & Schaie, 1990) converge to form a memory self-efficacy factor, which was significantly related to each of the trial predictions for both of the tasks. The study found that age differences in predictions were mediated by age differences in memory self-efficacy. Importantly, all age groups changed their predictions towards their actual performance across the trials for the word recall task; there were no age related differences in the accuracy of predictions. This study showed that older adults were able to monitor their performance equivalently to younger adults in the word recall task, and therefore had an awareness of their memory performance across the trials. Repetition of tasks may contribute and particularly benefit older adults in the monitoring of their performance; improvements in predictions over trials were found in both Ansell and Bucks (2006) and Bieman-Copland and Charness (1994).

Self-referent beliefs of memory may also be assessed by measuring subjective reports of memory problems. Rabbitt and Abson (1991) found in a sample aged 50-79 years that reports of functioning as measured by the Cognitive Failures Questionnaire (CFQ; Broadbent, Cooper, FitzGerald & Parkes, 1982) and MFQ were not related either to prediction or actual performance on a number of cognitive tasks. Older adults underestimated their performance on some cognitive tasks (picture recognition, digit span and cumulative learning), while overestimating their performance on others (free recall; Rabbitt & Abson, 1991). The finding of overestimation of word recall is in contrast to the Hertzog et al. (1990) result, but there were methodological differences between the studies. In the Hertzog et al. (1990) study, participants were informed of a typical, normative performance, which may have influenced predictions. Nevertheless, both studies included 30 words in the free recall task, and the predicted scores were similar (12.2 and 14, Rabbitt & Abson, 1991, and Hertzog et al., 1990, respectively). Older adults in Rabbitt and Abson (1991) recalled 8.32 words, whereas the average recall performance in Hertzog et al. (1990) was 17 words. Again, this difference may be partly explained by methodology. In

contrast to the Rabbitt and Abson (1991) study, the Hertzog et al. (1990) word lists were composed of 6 words from five semantic categories, possibly facilitating recall. These studies are therefore important because they suggest that age-related differences may be a product of methodology employed. Moreover, the value of the Hertzog et al. (1990) study is in the evidence that, regardless of age, task experience influenced predictions. Specifically, prediction accuracy increased across trials, suggesting increasing awareness of performance with task familiarity.

One potential criticism is that these studies did not cognitively screen their samples, which is now the convention in ageing studies (e.g. Deary, Bastin, Pattie, Clayden, Whalley, Starr & Wardlaw, 2006). It is therefore reasonable to speculate that memory impairments in the Rabbitt and Abson (1991) sample may have accounted for the low rate of recall. However, although Weaver-Cargin, Maruff, Collie and Masters (2006) found mild memory impairment in 28% of a community based sample of older adults (mean age 69.2 years), there were no differences between groups (memory impaired and non-impaired) on subjective reports of memory problems as measured by the CFQ. This suggests that memory complaints are not always related to *actual* memory impairments. In other words, there may be a separation between how older adults feel about their cognitive capacities and their assessment and understanding of their actual performance. In support, Rabbitt and Abson (1991) found that affective factors, such as depression and self-regard are more related to predictions of cognitive functioning in older adults, than their beliefs about their memory.

The metamemory studies have extended our understanding of the processes involved in awareness, but are limited by being cross-sectional. The opportunity to examine how awareness evolves or declines with age is limited within such studies. Ageing has been shown to be a time of progressive cognitive decline (see Section 2.5, Chapter 2), and longitudinal studies provide the opportunity to investigate any change in awareness over time. McDonald-Miszczak, Hertzog and Hultsch (1995) published two longitudinal studies of the stability and accuracy of metamemory in ageing. The first study was conducted over a two year period with 231 adults aged 22-78 years and the second included 234 adults aged 55-86 years over a six year period. Both studies found that metamemory beliefs as measured by metamemory questionnaires (MIA and MFQ) were stable across time. The first study did not find any decline in memory performance as assessed by word and text

recall tasks. Interestingly, the second study found that memory as measured by word, fact and text recall tasks declined over six years and that this decline was related to the Change subscale of the MIA, which measures perceptions of change (.16, .26 and .21 for word, fact and text recall, respectively). The longer duration of follow-up may have been necessary to reveal significant change. Although these findings suggest that changes in actual memory performance are related to perceived changes in memory; the correlations, albeit significant, are small (range:  $r = .13$  to  $.28$ ). Hertzog and Hultsch (2000) concluded in a later review that self-referent beliefs may influence “subjective well-being and everyday behaviour in cognitively demanding situations” (p. 459), rather than being linked to actual performance.

In conclusion, there are some differential age-related effects on processes involved in metamemory. The literature has provided evidence that older adults do differ in some processes involved in monitoring, suggesting that some aspects of awareness change with ageing. The critical point may be that older adults are less accurate in their assessment of their abilities; although there is evidence of updating of self-assessment through experience, studies have found that revisions are still less accurate in older adults compared to younger adults. A number of variables has been identified that may mediate age-related differences in predictions, self-assessment and performance, namely executive functions, processing speed and memory self-efficacy. It remains unclear, however, whether deficits in awareness processes found in older adults have any significant influences on their everyday functioning.

### 2.6.2. Hierarchies of Processing Model and Ageing.

As reviewed in Chapter 1, there are a variety of models of awareness developed from different perspectives; however, there were limitations inherent in each of the models reviewed. The psychological models (Cavanaugh, 1989; Tulving, 1985) exclusively considered awareness of memory functioning. Whilst Tulving’s (1985) conceptualisations of auto-noetic and noetic awareness have been widely tested using behavioural and neuroimaging techniques, both types of awareness also focus on memory, in this case memory retrieval. As shown in the metamemory literature, there are other types of awareness, for example, performance monitoring, which has been shown to be less

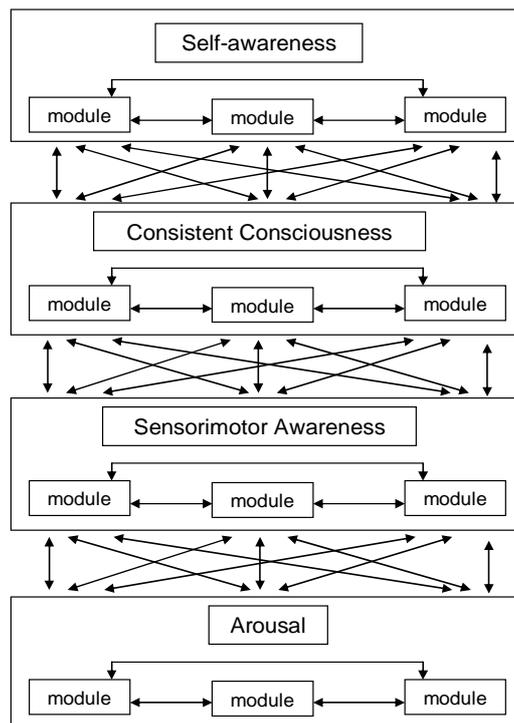
effective in older adults (Bieman-Copland & Charness, 1994). Although testing separate conceptualisations of awareness based on a number of models is one way of investigating potential age-effects on awareness, the HoP model by Stuss was selected for its potential to generalise to memory and performance-monitoring, and its focus on higher cognitive (executive) functions. Additionally, some models can be overly complex (e.g. Clare, 2004b); include vague descriptions of awareness (e.g. Prigatano & Johnson, 2003); and/or do not describe interactions between different types of awareness (e.g. Cavanaugh, 1989). Two neuropsychological models of awareness (Agnew & Morris, 1998; Toglia & Kirk, 2000) have been tested (see Ansell & Bucks, 2006; Graham et al., 2005; O'Keefe et al., 2007). However, both of these models assumes the presence of clinically significant deficit in awareness, and are therefore better at explaining diseases of awareness (e.g. dementia) than normal, healthy ageing. Although the Stuss et al. (2001) model was developed to account for deficits in awareness in patients with brain lesions, it fundamentally differs in that there is no inherent presumption of impaired awareness. The HoP model also provides specific definitions of the processes underlying different types of awareness, and promotes the concept of interaction between levels of awareness. This made the HoP model particularly suitable for an investigation of healthy ageing.

The HoP model (Stuss et al., 2001; see Figure 2.3) identified neural regions and networks involved in different types of awareness, with a particular focus on the fundamental role of the frontal lobes and their limbic system connections. The functioning of frontal lobe networks is proposed to underlie Consistent Consciousness and Self-Awareness, and has been linked with executive functions. A significant body of literature has shown that ageing is associated with a reduction in the volume of the frontal lobes (bilateral frontal grey and white matter, see Raz, 2000), and an increased number of white matter lesions (Gunning-Dixon & Raz, 2000), causing structural alterations and network disruptions. For this reason, it is plausible to assume that some aspects of awareness will be affected by ageing.

Stuss et al. (2001) further proposed that each level of the model contained a number of modules whose functioning supported the processes involved in the different types of awareness. The processes within each level can function independently of other levels, however, the ability of the modules to transmit and receive input, to and from, higher and lower levels, provides the connectivity between the different levels (see Figure 2.3). This

inter-connectivity explains how deficits at one level can be circumvented: other modules within that level transmit neural activity to modules at higher and lower levels. The description of neural pathways throughout the model accounts for the possibility of level-specific deficits in awareness, whilst other types of awareness and their associated processes remain functional. However, deficits within each level may also constrain the functioning of processes at other levels. One important caveat to be acknowledged with the HoP model is that the description of the inter-connectivity of the model provides an explanation of how deficits may be both transmitted and circumvented. This poses a challenge for the empirical assessment of the model, as both null and significant results of relationships between the levels are explained by the model.

Figure. 2.3. The Hierarchies of Processing Model (adapted from Stuss et al., 2001).



As shown in Figure 2.3, the lowest level of awareness in the model is Arousal, located within the brain stem (reticular formation) and relates to the dimension from coma to waking state. The second level, Sensorimotor Awareness, results from the processing of sensory and bodily information in the posterior regions (association cortices) of the brain. The final two levels of the model involve processing within the frontal lobes. Consistent

Consciousness, at the third level, relies on executive functions and the ability to organise sensory information underlying behaviour. At the highest level, accurate monitoring of the self and personal abilities facilitates Self-Awareness, and is dependent on the inter-connectivity of the frontal lobes with the limbic system. The following section will discuss evidence for assuming age-related impairments in Arousal, Sensorimotor functions, Consistent Consciousness, and Self-Awareness.

The Arousal level of awareness is related to ‘basic’ consciousness, from coma to waking state. Between 30-50% of older adults report sleep disturbances (Ohayon, 2002). In a large study investigating normative sleep patterns in a community-based sample of 1026 participants aged 60 years and over, Ohayon and Vecchierini (2005) found that 5% of the sample had  $\leq 4\frac{1}{2}$  hours sleep per 24 hour period, whilst 25% had fewer than 6 hours sleep. Of this 5%, only 54.2% were satisfied with their sleep, and regression analyses found higher levels of insomnia and excessive daytime sleepiness were linked with  $\leq 4\frac{1}{2}$  hours sleep. Poor cognitive functioning as measured by the Cognitive Difficulties Scale was associated with  $< 6$  hours sleep per 24 hour period. In contrast, in another community-based sample of 375 participants aged 75-85 years, Schmutte, Harris, Levin, Zweig, Katz and Lipton (2007) found that sleep onset latency ( $> 30$ mins), rather than sleep duration, after controlling for the effects of age, was associated with poorer performance on the Wechsler Adult Intelligence Scale (WAIS) subtests of Information, Vocabulary and Block Design. Converging evidence for a relationship between sleep and cognition has been found using polysomnography. Reduced delta activity in the PFC has been found in older adults, indicative of poor recovery from sleep and cortical re-organisation, and was related to decreased nonverbal planning and verbal fluency abilities (Anderson & Horne, 2003). These results indicate that sleep problems in older adults impact on daytime cognitive functioning, providing support for the HoP model, in that problems at lower levels can be transmitted to higher levels. More pertinently for the current thesis, these findings suggest that some aspects of awareness may be compromised in some older adults who are assumed to be healthy.

The ‘cholinergic hypothesis’ proposes that the cognitive and behavioural symptoms of AD are a result of reductions in acetylcholine and basal forebrain cholinergic neurons (Muir, 1997). Cholinergic neurons are located in the basal forebrain, and project throughout the cortex, the hippocampus and amygdala, and are also found in the pontomesencephalon,

part of the reticular formation, projecting to the basal forebrain and thalamus (Woolf, 1997). There has been debate regarding the function of the cholinergic system in arousal, attention, and consciousness, due to its prominence throughout the brain (e.g. Coull, 1998, Perry & Perry, 1995; Perry, Walker, Grace & Perry, 1999; Woolf, 1997). Woolf (1997) suggested that the vast connectivity of cholinergic and monoaminergic neurons throughout the brain underlies different states of arousal, from waking through to higher cognitive functions. In support, scopolamine, a muscarinic receptor antagonist, has been used temporarily to block the cholinergic system, resulting in cognitive deficits in healthy adult participants (Vitiello et al., 1997). Scopolamine has also been employed to induce states of altered consciousness (see Perry & Perry, 1995). PET studies of acetylcholinesterase (AChE), a marker of cholinergic uptake, in individuals with AD, have found significant reductions of AChE compared to controls, without significant differences in the size of the nucleus basalis of Meynert (Herholz et al., 2004). Significant reductions of AChE throughout the entire cerebral cortex, hippocampus and amygdala have also been found in individuals with early-onset AD (Shinotoh et al., 2000). Dournaud, Delaere, Hauw and Epelbaum (1995) found that reduced choline acetyltransferase (ChAT) activity in the middle frontal gyrus, supramarginal gyrus and the superior temporal gyrus was associated with lower 'global' cognitive function as measured by Blessed test scores. These results indicate that cholinergic deficits contribute to the cognitive decline found in AD. However, the extent of cholinergic system change with healthy ageing is not clear. Most studies do not demonstrate decrease in cholinergic activity with normal ageing, however, they do report a loss of cholinergic receptors (e.g. Winblad, Hardy, Backman & Nilsson, 1985). Nevertheless, in support of the HoP model, the evidence from individuals with AD indicates that impairments in the Arousal level of awareness, as measured by cholinergic deficits, can impact on higher cognitive processes. Of interest is the threshold over which losses of acetylcholine become symptomatic.

In a series of studies, Falkenstein and colleagues (Kolev, Falkenstein & Yordanova, 2006; Falkenstein, Yordanova & Kolev, 2006; Yordanova, Kolev, Hohnsbein & Falkenstein, 2004a) investigated age-related slowing of information processing and reaction times, with simple and choice-reaction speeded tasks in auditory and visual modalities, and the associated electrophysiological components of stimulus processing (P1, N1 and P3), response selection (lateralised readiness potential: LRP) and motor-related processing (motor-related potential: MRP). Although these studies do not directly relate to the HoP

model, the separation of sensorimotor performance into a simple and a more complex task facilitates an investigation of sensory processing and the organisation of information underlying behaviour, which correspond with Levels 2 and 3 of the model (Stuss et al., 2001). The results of the studies were similar in that older adults (all studies:  $n = 14$ ,  $M$  Age 58.3,  $SE \pm 2.1$ ) had slower RTs in the more complex choice-reaction task (CRT) compared to the younger adults (all studies:  $n = 13$ ,  $M$  Age 22.5,  $SE \pm 1.5$ ). However, in the simple-reaction task (SRT) both groups had comparable response times, with equivalent error rates in both paradigms (Kolev et al., 2006; Falkenstein et al., 2006; Yordanova et al., 2004a). Early sensory processing, as indexed by the N1 and P1, was not affected by age. However, the P3 in the CRT (P3 not present in SRT waveforms), was both attenuated and delayed in the older adults. The latency of the P3 peak was also longer than RT, indicating a distinction between the cognitive and motor aspects of complex task performance. Again, there were no age differences in the LRPs, indicating that response selection was equivalent across groups. Of note, was the significant increase in MRP amplitudes in older adults in the CRT, but not the SRT, with longer MRP latency compared with younger adults. These results indicate that the behavioural slowing commonly found in older adults originates in the generation of the motor response (Yordanova et al., 2004a), and that age-related slowing may be specific to complex tasks, rather than differences in sensory processing (Kolev et al., 2006). With regard to the HoP model, these results indicate that while some aspects of Sensorimotor Awareness may be intact (e.g. initial sensory processing of information) with ageing, processing at a higher level may be constrained (e.g. attentional processing and motor response generation).

The third and fourth levels of the model rely on the functional integrity of the frontal lobes (Stuss et al., 2001), with Consistent Consciousness being associated with executive functions and Self-Awareness with monitoring of the self and personal abilities. As discussed in Section 2.5 of this chapter, executive function impairments and frontal lobe changes can be a feature of increasing age, suggesting that Consistent Consciousness may be affected by ageing. The metamemory literature (see Section 2.6.1), has also indicated age-related impairments in some aspects of self-awareness, for example, overestimation of cognitive abilities indicates a deficit in self-monitoring. A diverse but converging literature has indicated support for an application of the HoP model (Stuss et al., 2001) to investigating different types of awareness in ageing. Ageing may be related to deficits in Arousal, as indexed by quality of sleep, but with intact Sensorimotor Awareness, as

indicated by Falkenstein and his colleagues. However, Consistent Consciousness and Self-Awareness may also be impaired with age, possibly due to the structural and functional changes found within the frontal lobes with increasing age. The description of the inter-connectivity of the modules involved in each level of the model provides an explanation for how lower level deficits may influence higher levels, but also how impairments can be circumvented (see Figure 2.3). At the same time, such a description makes it hard to test the model empirically. For example, if deficits at one level are related to deficits at a higher level, support is found for inter-connectivity of the model. However, if deficits are not related to higher level measures, the model may also be supported because it proposes circumvention by unaffected intra-connected modules (i.e. not all modules within a level are damaged). Moreover, there is a lack evidence to relate these levels of awareness to brain function. Such studies are necessary to provide empirical support that the ageing brain is associated with alterations in awareness.

The previous section has argued for the applicability of the HoP model to an investigation of different types of awareness in healthy ageing. Age differences in the processes underlying three types of awareness were assessed in this thesis. It was assumed that Arousal would be functional in awake participants; therefore this level was not experimentally assessed. The second level of awareness (Sensorimotor Awareness) was operationalised using a novelty auditory oddball ERP paradigm. The rationale was that this type of awareness involves the processing of basic sensory information. The oddball paradigm employed elicits ERP components shown to be sensitive to initial attentional capture (N1) and of later, more controlled, attentional processing (P3). These components are well-documented measures of sensory information processing in the brain (see Chapter 4). Consistent Consciousness refers to the ability to organise sensory information underlying behaviour and relies on executive functions. The term executive functioning encompasses a broad range of cognitive functions including attention, working memory, cognitive control and conflict processing. The performance of complex tasks depends on the interaction and organisation of such cognitive (executive) processes and incoming sensory information. Therefore, electrophysiological and behavioural measures of performance monitoring were employed to operationalise Consistent Consciousness. von Cramon and Ullsperger (2002) emphasised that the performance monitoring process incorporates numerous subcomponents that occur at different time points. Electrophysiological measures of performance monitoring include pre-response conflict

(N2); allocation of attention (P3b); response-conflict priming (N4); error detection (CRN-ERN); and, error processing (Pe). In addition, behavioural measures of remedial actions were included: error correction and post-error slowing. Postdiction report of performance accuracy provided a metacognitive measure of the individual's performance monitoring abilities. It is of interest to consider the possibility that ERP correlates of performance monitoring reflect the 'consistent consciousness' level of awareness, in consort with the lower levels. This is due to the fact that performance-monitoring requires more than simple stimulus-response activity, it requires the individual consciously to monitor that sensory-input and motor-output activity and amend behaviour accordingly. It is important to acknowledge that the HoP model rests on philosophical and theoretical psychology foundations, rather than direct scientific-empirical evidence, but in this respect it does not differ from other psychological models that, with testing, have been widely accepted in mainstream cognitive neuroscience, for example, the working memory model of Alan Baddeley. Mapping of cognitive neuroscience techniques to a theoretical psychological model provides a basis for interpreting empirical data, and perhaps an indication as to how a model may be updated to be more useful, but does not provide definitive support for that model.

This chapter has presented considerable evidence that the brain undergoes physical change in normal ageing, and there is concomitant albeit subtle change in cognitive function. Therefore, it is plausible to assume that the neural and cognitive changes found in ageing may have some effect on awareness.

## Chapter 3. Recruitment & Overview of Methodology.

The purpose of this chapter is twofold. Firstly, the recruitment of younger and older adult participants is described. Specific consideration is given to good clinical practice in the assessment of older adult volunteers. Secondly, this chapter provides an overview of the methodologies used to study awareness. All participants were assessed using the same methodologies; while each chapter that follows is based on empirical data obtained using different awareness measures, the participants were drawn from the same cohort.

### 3.1. Recruitment of Participants.

Approval for the study was granted by the Ethics Committee of the School of Psychology, University of Southampton, Hampshire, UK (Appendix 1).

#### 3.1.1. Participants.

Thirty older adult (OA) participants aged 60 years and over were recruited from the 'Exploring the Mind' Older Adult Volunteer Panel created by Dr. Romola Bucks, University of Southampton. Twenty-one younger adult (YA) participants were recruited opportunistically, and received course credits in return for their participation. It was decided a priori to recruit in excess of 20 participants into each age group. This number was determined based on a number of factors. The total testing time was approximately 4 hours per person, across 2 sessions, therefore, participants were asked for a considerable time commitment. Additionally, the sample size is commensurate with published ERP studies with older adults, for example: Falkenstein et al. (2001) obtained data from 11 younger and 11 older adults. Finally, power to detect an effect was considered and is addressed in Chapter 8.

All participants were healthy at the time of testing, had no self-reported history of neurological or psychiatric conditions, normal or corrected-to-normal vision and hearing and, if on medication, had been taking stable dosages for the preceding three months. Colour vision was also assessed, in order to check that the participants could differentiate between the red and green arrows in the 4-CRT ERP paradigm. As auditory perceptual

awareness was examined (Chapter 5), the majority of participants underwent a hearing assessment in accordance with British Audiometry Society guidelines (British Society of Audiology, 1986) prior to testing. Access to audiometry equipment was limited; therefore not all participants received a hearing assessment (numbers are given in Section 4.2.1, Chapter 4). Where there was mild-to-moderate hearing loss in one or both ears (25-45 dB, and 45+ dB, respectively), the sound pressure level was adjusted accordingly.

All participants were cognitively screened using the Mini Mental State Examination (MMSE; Folstein, Folstein & McHugh, 1975: range 0-30; abnormal score  $\leq 26$ ) and the Montreal Cognitive Assessment (MoCA; Nasreddine et al., 2005: range 0-30; abnormal score  $\leq 26$ ). Data from these two measures were explored using the Shapiro Wilk test, and the MoCA was found to be normally distributed, however, the MMSE was not. The mean or median, as appropriate, scores for both OA and YA groups (Table 3.1.) were within the normal range. No individual participant obtained a score indicative of clinically significant abnormality on either measure, and the groups were statistically equivalent (MMSE and MoCA, both  $p > .1$ ).

Estimated premorbid IQ was calculated from National Adult Reading Test (NART; Nelson & Willison, 1991) scores. The NART is a reliable and valid method of estimating premorbid IQ (Crawford, Deary, Starr & Whalley, 2001). It assesses knowledge of irregular word pronunciation. A major assumption of the NART is that greater accuracy in word pronunciation reflects greater crystallised intellectual function. As shown in Table 3.1, the OA participants had significantly higher estimated premorbid IQ,  $t(44) = 7.00, p < .001$  than the YA participants. Conversely, the YA participants had more years of education,  $t(47) = -5.14, p < .001$ . This pattern of results may reflect a change in teaching practice alongside the development of new language and usage since the original publication of the NART. It is also important to acknowledge that a University education may have been more accessible to participants in the YA group. The finding of reduced estimated premorbid IQ in YA participants compared to that obtained from OA has previously been found (e.g. Bunce & Macready, 2005).

Table 3.1. Demographic Data obtained from younger and older adults.

<i>M (SD)</i> or Median ( <i>IQR</i> )	<u>Younger adult participants</u> <i>n</i> = 21	<u>Older adult participants</u> <i>n</i> = 30
Age	22.1 (4.1)	69.4 (6.7)
Gender (M : F)	5 : 16	10 : 20
MMSE <sup>1</sup>	29 (1.0)	29 (1.0)
MoCA	28.1 (1.4)	28.2 (1.1)
NART predicted IQ	105.4 (7.8)	119.0 (5.4) †
Years of Education	15.5 (2.1)	12.3 (2.1) †

*Note.* †  $p < .05$ ; <sup>1</sup> Data were analysed with the Mann-Witney U test; MMSE = Mini Mental State Examination, Folstein, Folstein & McHugh, 1975; MoCA = Montreal Cognitive Assessment, Nasreddine et al., 2005; National Adult Reading Test, 2<sup>nd</sup> edition, Nelson & Willison, 1991.

### 3.2. Rates of Attrition.

The complete assessment battery took a total of four hours. The assessment was, therefore, split into two sessions for each participant. Despite the need for two assessment visits, the attrition rate was very low. Only one OA (3.3%) and one YA (4.8%) chose not to participate in the second laboratory-based session of the study. Two YAs (9.5%) chose to participate in the laboratory-based assessments, but failed to complete the questionnaire battery. Overall, 92.2 % of the cohort completed all assessments associated with this study, and their demographic data are presented in table 3.1.

### 3.3. Further Information about the Recruitment of Older Adults.

Forty-one potential OA participants were identified using the following criteria: aged 60 and over; prepared to come to the University for research participation; and, ERP technique-naïve (i.e. had not participated in an earlier study of ERP components in normal ageing). The identified volunteers were sent an invitation letter (Appendix 2) and information sheet (Appendix 3), and requested to return the reply slip expressing interest in participation. On receipt of the reply slip, the OA volunteer was telephoned by the

researcher, and the TELE (Gatz et al., 1995: score  $\geq 15$  is considered normal. OA  $M 18.9 \pm 1.2$ ), and the Participant Demographic Form were administered as preliminary screening measures. Additionally, 4 volunteers responded to an article about the study in the Volunteer Panel newsletter, and a further 7 volunteers responded to information packs available at a Volunteer Panel Open Day. The study had a response rate of 73.2% from the mailings; ineligibility was due to central nervous system medication, neurological exclusion criteria (e.g. stroke, head injury) and failure to pass the cognitive screening (see Table 3.2). The OAs were requested to provide an informant, as the clinical awareness questionnaires (DEX questionnaire from the Behavioural Assessment of the Dysexecutive Syndrome battery: BADS; Wilson, Alderman, Burgess, Emslie & Evans, 1996; Memory Awareness Rating Scale: MARS; Clare, Wilson, Carter, Roth & Hodges, 2002) included informant versions, and discrepancy scores can be calculated to assess any difference in reports between the OA and their informants. YA were not required to provide informants, as the likelihood of students having long-term close relationships with others in Southampton was considered to be low. Twenty-four OA informants gave written consent and additionally participated in the study. An informant was a person (e.g. spouse, significant other, or close friend) whom the OA participant consented to being approached.

Table 3.2. The numbers of older adults and participation decisions from identified Panel volunteers.

<u>Participation Decision</u>	<u>Numbers of Older Adults</u>
Contacted:	<b><u>52</u></b>
Mailing	41
Responded to newsletter/Open Day	11
Did not participate	<b><u>22</u></b>
Ineligible	8
Declined to participate	4
Withdrew from Panel	1
No response	9
Participated	<b><u>30</u></b>

### 3.4. Cognitive Screening in Older Adult Research.

Memory decline is considered to be a feature of increasing age, and there has been growing investigation of the concept of a pre-clinical phase of AD, termed mild cognitive impairment (MCI). The First Key Symposium of MCI (Stockholm, Sweden, 2nd–5th September, 2003) considered the clinical features of MCI, and concluded that the variable outcome (e.g. stability in some, progression to AD in a proportion, and recovery in others) suggested that MCI should be considered as more than a simple ‘preclinical phase of dementia’ (Winblad et al., 2004). The Symposium further concluded that MCI represented neither dementia nor normal ageing (Winblad et al., 2004). In support, there are conflicting data on the prevalence of MCI in the normal population, with rates ranging from 5.3% (Hänninen, Hallikainen, Tuomainen, Vanhanen & Soininen, 2002) to 16.2% (Zanetti et al., 2006). Differential diagnostic criteria for MCI may account for the variable findings. For example, other nomenclature related to MCI have been introduced into the literature, such as ‘age-associated memory impairment’ (AAMI), ‘age-associated cognitive decline’ (AACD), and, ‘benign senescent forgetfulness’ (Collie & Maruff, 2000), to describe the range and degree of impairment found in older adults. Although the exact nature and measurement of these diagnoses remains under debate, one of the challenges for research into healthy ageing is to demonstrate that any given older adult sample does not present with any significant, and potentially confounding, cognitive impairment.

One of the most frequently used questionnaires to screen for cognitive impairment is the MMSE (described above: section 3.1.1), which provides a widely accepted and understood measure of global cognitive function. However, this questionnaire was designed to screen for dementia, rather than as a generalised cognitive screening instrument, and was developed prior to rigorous debate about MCI. Furthermore, MMSE scores may be influenced by level of education (Crum, Anthony, Bassett & Folstein, 1993; Tombaugh & McIntyre, 1992). Despite the wide acceptance of and reliance on the MMSE, other questionnaires focussing on the assessment of MCI have been developed, e.g. the Montreal Cognitive Assessment (MoCA; Nasreddine et al., 2005), which is proposed to have greater sensitivity and specificity than the MMSE (Nasreddine et al., 2005).

All younger and older adult participants in this thesis were administered both the MMSE and the MoCA. Using the normal cut-off of  $\leq 26$ , the MoCA detects 90% of MCI and

100% of AD, with a specificity of 87% (Nasreddine et al., 2005). To the author's knowledge similar data are not available for the MMSE. Both questionnaires were included for the purpose of cognitive screening, mainly because the MMSE is a more widely accepted tool. As the MoCA is a recent addition to the literature, it has been more recently standardised than the MMSE and although it may therefore be a more valid measure of general cognitive function in the current sample, it is less well established. A brief telephone screening questionnaire for dementia (TELE; Gatz, Reynolds, Nikolic, Lowe, Karel & Pedersen, 1995), which correlates well with the MMSE ( $r = -.71$ ; Jarvenpaa et al., 2002), was also administered at the initial telephone contact with the older adult participants to try and prevent any unnecessary demand on their time. The MMSE and MoCA take longer to administer and were thus the second line of screening.

### 3.5. Issues of Acceptability.

Table 3.3 documents the factors that were considered to ensure minimal discomfort, particularly for OA participants. The ERP testing required the use of an Easy-Cap<sup>TM</sup> for data collection. This is similar to a close-fitting bathing cap. Participants were monitored closely throughout testing to ensure that the process was acceptable to them. They were also offered a hair wash at a nearby hairdressing salon on completion of the ERP study, as the ERP testing involved the use of conductive gel spread widely over the scalp. When participants expressed concern over their performance, they were informed that tasks were designed to be challenging. Protocols were in place if participants expressed further concern over their performance (Appendix 4). Each testing session was designed to incorporate breaks and refreshments in order to avoid unreasonable demands on concentration and lengthy testing. The order of the tests and questionnaires was designed to avoid repetition of similar items and to reduce boredom. The initial ERP paradigm battery was comprised of the auditory novelty oddball, followed by two blocks of a two choice-reaction time task (2-CRT; Hogan et al., 2005) and two blocks of a four choice-reaction time task (4-CRT; Hogan et al., 2005). The CRT tasks were designed to elicit errors; in fact this is an important design feature of the performance monitoring ERP study, as errors are necessary in order to obtain an error-related negativity ERP component (ERN). Both younger and older participants had exemplary performance on the 2-CRT (>95%), so this task was excluded to reduce the length of the testing session, and, in most

instances, participants consented to additional blocks of the 4-CRT to compensate. The 4-CRT was perhaps the most challenging task, so it was decided to conclude the study with the more familiar ‘everyday behaviour’ tasks to ensure that the session was ended with a positive experience.

Table 3.3. Factors to be considered in the design of studies with older adult participants.

<u>Factor</u>	<u>Details</u>
Distress	Is testing likely to cause the participant any distress or have any negative consequences?
Familiarity with surroundings	Will the option of a home visit be available, if applicable to the study?
Fatigue Effects	What is the length of testing? Is there a high degree of repetition?
Physical Factors	Have physical factors, such as mobility, hearing and vision been taken into consideration?
Time of Day	Does the participant have a choice regarding time of testing?

### 3.6. Neuropsychological Measures of General Intellectual Function, Memory, Affect and Awareness of Performance.

Neuropsychological measures of general non-verbal intellectual function and memory were administered to provide data on the functional capabilities of the YA and OA groups.

#### 3.6.1. Neuropsychological Measures:

i) Ravens’ Standard Progressive Matrices Sets A to D (RSPM; Raven, Raven & Court, 2000). The RSPM provides a measure of fluid (non-verbal) intelligence to compliment the estimated premorbid IQ provided by the NART. There is a progressive increase in difficulty across sets A-D<sup>2</sup>.

<sup>2</sup> In the present study set E was excluded as it can lead to fatigue through lengthy testing (Caffarra, Vezzadini, Zonato, Copelli & Venneri, 2003).

ii) Pyramids and Palm Trees (PPT; Howard & Patterson, 1992). The PPT test assesses semantic knowledge by asking participants to match a target image with one of two possible stimuli. There are 52 items to match. The test has been validated in an older adult population and used with individuals with AD (Hodges & Patterson, 1995). These data are not reported in this thesis as there were ceiling effects in both groups.

iii) Autobiographical Memory Interview (AMI; Kopelman, Wilson & Baddeley, 1990). The AMI is a semi-structured interview providing a standardised measure of autobiographical memory validated in 18-80 year olds and in patient populations, e.g. Alzheimer's disease (AD). The interview is separated into 10 sections regarding different times during the lifespan, providing scores for semantic and episodic memories from early childhood, early adulthood and recent times. In the current study, the interview was tape-recorded to allow for an assessment of inter-rater reliability. These data are not reported as the YA group were not forthcoming in episodic memories; their scores, therefore, erroneously indicated significant episodic memory deficits.

### 3.6.2. Measures of Affect.

Measures of affect were administered in order to control for the possible confounding of negative mood on self-report of functional abilities, metamemory perceptions and event-related potentials (ERPs).

i) Rosenberg Self-Esteem Scale (R-SES; Rosenberg, 1965). The questionnaire is composed of 10 items with a six-point rating scale. Although originally designed with a four point rating scale, the dimensionality can be altered to provide a greater range of values (e.g. DeCremer & Sedikides, 2005; Greenberger, Chen, Dmitrieva & Farrugia, 2003). The R-SES has high internal consistency ( $\alpha = .88$ ; Greenberger et al., 2003). Higher scores indicate higher levels of self-esteem.

ii) Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) is composed of 14 items and provides measures of anxiety and depression. Widely used with wide-ranging age groups and patient populations, both scales have high internal consistency;  $\alpha = .80$  and  $.76$  (anxiety and depression, respectively; Mykletun, Stordal & Dahl, 2001). In the present study the HADS was given as a self-report measure. Higher scores indicate higher

levels of anxiety and depression, with clinically significant thresholds exceeding a score of 11 for both anxiety and depression.

iii) Positive and Negative Affect Schedule (PANAS; Watson, Clark & Tellegen, 1988). The PANAS is a 20 item self-report measure of positive and negative affect using a five point rating scale. The PANAS was used to assess the effect of the ERP-study experience on the individual, and as such was administered pre- and post-ERP testing. Higher scores indicate higher levels of affect (positive and negative affect). Clinically significant scores are found in the lower percentiles for positive affect and higher percentiles for negative affect (Crawford & Henry, 2004). However, exact scores indicating clinical levels of affect were not provided. Discrepancy scores indicate a degree of change in affect and the discrepancy values can be positive, indicating an increase in positive or negative affect, or negative, indicating a decrease in positive or negative affect.

iv) Shortened Marlowe-Crowne Social Desirability Scale (M-C 1; Strahan & Gerbasi, 1972). The M-C 1 is a shortened version of the Marlowe-Crowne Social Desirability Scale (M-C SDS; Crowne & Marlowe, 1960) using a dichotomous rating scale of true or false on 10 statements reflecting highly desirable or undesirable properties. The M-C SDS has high internal consistency ( $\alpha = .88$ ; Crowne & Marlowe, 1960), and the shortened version is highly correlated with the original version ( $\alpha = .80$ ; Strahan & Gerbasi, 1972). The M-C 1 was administered to all participants and the OA informant. It was administered in order to determine if self-report of metacognitive ability or awareness measures might be related to social desirability.

v) Cornell Scale for Depression in Dementia (CSDD; Alexopoulos, Abrams, Young & Shamoian, 1988). The interview consists of 19 items and has high internal consistency (0.84). The CSDD was administered to the OA informant and required their assessment of the behaviour of the participant in the week prior to testing. These data are not reported, due to floor effects.

vi) Rating Anxiety In Dementia (RAID; Shankar, Walker, Frost & Orrell, 1999). The interview consists of 18 items and has high internal consistency (0.83). The RAID was administered to the OA informant and as above with the CSDD required their assessment

of the participant in the two weeks prior to testing. These data are not reported, due to floor effects.

### 3.6.3. Measures of Awareness.

Awareness has frequently been assessed using self-report questionnaires (see Clare, 2004b). It was considered important, therefore, to incorporate such scales in this study in order to offer a degree of validation of the more novel electrophysiological (ERP) measures of awareness.

i) Metamemory In Adulthood (MIA; Dixon, Hultsch & Hertzog, 1988). The MIA is a 108 item self-report questionnaire investigating self-referent beliefs. The questionnaire provides 7 subscales shown in Table 3.4. The MIA subscales have high internal consistency, with  $\alpha$  values ranging from .71 to .93 (Dixon et al., 1988). Discriminant validity analyses, conducted by the authors, found that the MIA subscales (except MIA Anxiety) were not related to generalised locus of control, state or trait anxiety, or depression. The MIA Anxiety subscale was related to state and trait anxiety (Dixon et al., 1988).

ii) Memory Awareness Rating Scale (MARS; Clare, Wilson, Carter, Roth & Hodges, 2002) was specifically designed to assess memory awareness in dementia. It contains self and informant versions of a 10 item questionnaire regarding the management of the individual in different situations dependent on memory abilities. The scale has high internal consistency ( $\alpha = .94$ ) and a high test-retest reliability (.91; Clare et al., 2002). The participant version was administered as an interview, whereas the informant version was given as a questionnaire. OA informants were administered the informant version. Discrepancy scores are calculated and can also be either positive; participant scores are higher than informants', or negative; participant scores are lower than informants'. Lower scores represent greater problems managing in the given situations, and therefore better awareness of functioning.

Table 3.4. The Dimensions of the Metamemory In Adulthood (MIA) Instrument.

<u>Dimension</u>	<u>Description</u>
Achievement (range 16-80)	Perceived importance of having a good memory and performing well on memory tasks (higher scores indicate greater achievement).
Anxiety (range 14-70)	Feelings of stress related to memory performance (higher score indicate greater anxiety).
Capacity (range 17-85)	Perception of memory capacities as evidenced by predictive report of performance on given tasks (higher scores indicate greater capacity).
Change (range 18-90)	Perception of memory abilities as generally stable or subject to long-term decline (higher scores indicate greater stability).
Locus (range 9-45)	Perceived control over remembering abilities (higher scores indicate greater internal control).
Strategy (range 18-90)	Knowledge and use of information about one's remembering abilities such that performance in given instances is potentially improved (higher scores indicate greater use).
Task (range 15-75)	Knowledge of basic memory processes (higher scores indicate greater knowledge).

*Note.* Based on Dixon and Hultsch (1983).

iii) DEX questionnaire from the Behavioural Assessment of the Dysexecutive Syndrome battery (BADS; Wilson, Alderman, Burgess, Emslie & Evans, 1996). The BADS has been validated in 16-87 year olds and is sensitive to frontal lobe functioning (Wilson et al, 1996). The self and informant versions of the DEX have 20 items regarding emotional or personality change, motivational, behavioural and cognitive changes. Reliability and validity data are not provided by the BADS manual. However, a significant relationship was found between informant DEX scores and total BADS score ( $r = -.62$ ; Wilson, Evans, Emslie, Alderman & Burgess, 1998), indicating that informants' assessments of problems were associated with worse performance on the BADS by the participant. OA informants were administered the informant version. Discrepancy scores are calculated and may be positive: participant scores are lower than informants', or negative: participant scores are higher than informants'. Higher scores are indicative of greater everyday executive function difficulties.

(iv) Performance-Monitoring in a Naturalistic Task: Four tasks were administered to assess the quality of performance-monitoring in naturalistic situations, potentially requiring planning and execution of strategies: making a cheese and lettuce sandwich; wrapping a gift; preparing a letter for posting (taken from Forde & Humphreys, 2000); and, the Action Program Test from the BADS, a test of practical problem solving that was more novel (using water to float a cork out of a tube without touching the apparatus). Each task was divided into five stages that needed to be completed sequentially to succeed on the task. These data are not reported, due to ceiling effects in both groups.

In summary, whilst the focus of this thesis is awareness in normal ageing, a number of measures were administered to control for potential confounding variables such as reduced intellectual function, negative affect, socially desirable responding and low self-esteem. The measures of awareness selected enabled comparison with previously published literature (e.g. metamemory questionnaires). Such assessments allow inferences to be drawn about the quality of underlying brain function associated with awareness, but this is an indirect association. Thus, these assessments are useful but necessarily limited. It was anticipated that a more complete understanding of brain function associated with awareness may be obtained using imaging techniques. In particular, ERPs, derived from stimulus and/or response-related brain electrical activity, may allow investigation of the power and timing of neural correlates of 'awareness'.

### 3.7. Introduction to Event-Related Potentials.

Brain imaging techniques (e.g. MRI; fMRI; DTI; and PET) focus on neural structures, and areas of activation with associated sensory, cognitive and motor function. Although these techniques have good spatial resolution, the temporal resolution is poor, due to the time taken to create an image. For example, the blood oxygenation level dependent (BOLD) fMRI signal is a measure of hemodynamic response to neural changes, and this can take several seconds to register. However, sensory processing occurs within milliseconds (ms) and, as such, imaging techniques are not able to record all activity associated with this type of processing.

Electroencephalography (EEG) is a technique that records the electrical activity of the brain by detecting voltage changes over time and has excellent temporal resolution (within ms of a stimulus/event), allowing for the investigation of ‘on-line’ processing. The technique is non-invasive and particularly amenable with populations who may not cope with other brain imaging techniques (e.g. MRI), such as the very young, very old or particularly anxious. The following sections provide an overview of the origin and processes by which the electrical activity of the brain can be measured at the scalp with an EEG. Electrophysiological components (ERPs), which are derived from the EEG and related to specific events, are subsequently described.

### 3.7.1. Neural Generators of the Electroencephalogram.

As the cortex develops, so does the electrical activity of the brain. Each neuron has a semi-permeable membrane. This facilitates chemical stimulation which leads to changes in the voltage at the cell membrane. An action potential is the term used to describe this rapid change in voltage (Kolb & Whishaw, 1996). Action potentials travel along the cell’s axon, with larger axons conducting faster potentials. The process is facilitated by the flow of sodium ions (depolarization) through the semi-permeable cell membrane, at the initial segment part of the axon and at regular intervals along the axon (nodes of Ranvier). Myelination facilitates conduction of the action potential. Otherwise stated, the action potential travels along the axon facilitated by the nodes of Ranvier which are gaps along the axon between each glial (myelin) cell (Kolb & Whishaw, 1996). When the action potential reaches the axon’s terminals (terminal bouton), neurotransmitters are released, and, dependent on their function (inhibitory or excitatory), the voltage of the post-synaptic membrane is changed (Kolb & Whishaw, 1996). A dipole is a division of voltage within the extracellular space, created by a change in the voltage (positive/negative) in the extracellular space caused by the movement of ions into the post-synaptic cell (Nelson & Monk, 2001). The dipole disperses through the extracellular space to the surface of the scalp, aided by the columnar organisation of pyramidal cells (Nelson & Monk, 2001) which are prevalent in the cortex.

### 3.7.2. The Electroencephalogram.

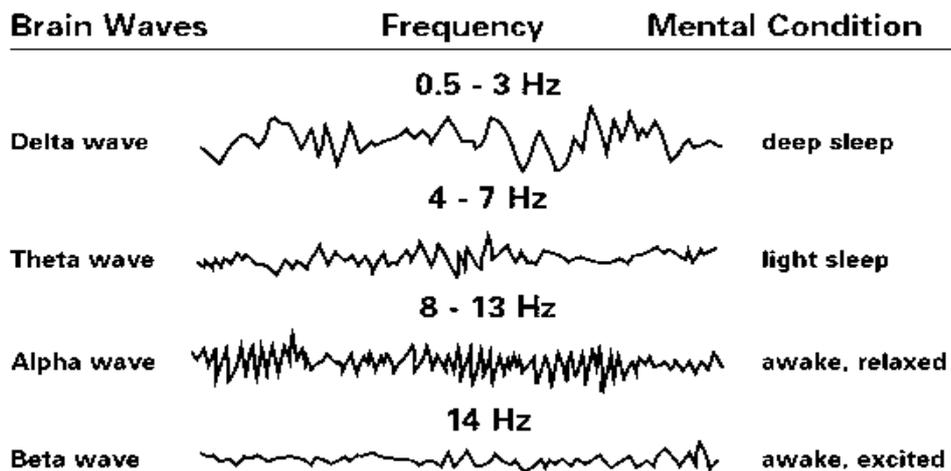
When stimulated simultaneously, groups of pyramidal neurons produce a positive/negative pattern of activity, the direction of which is dependent on the orientation of the dipole. The voltage of the ions closest to the recording site (scalp) produces the deflections that are identifiable as an EEG trace. This trace is recorded by placing electrodes on the scalp and amplifying the signal so that it is readable. Therefore, the EEG records the summed electrical activity from neuronal populations with columnar organisation. Not all neural activity is recorded by the EEG; the non-columnar organisation and distance from the scalp of sub-cortical structures means electrical activity from these areas may not be detected (Coles & Rugg, 1995).

An EEG signal has three dimensions: frequency, time and amplitude. The frequency of the EEG is measured in Hertz (Hz), which refers to the number of oscillations per second. The frequency bands of the EEG are alpha, beta, theta and delta (see Figure 3.1.). Alpha band activity (8 – 13Hz) is found in adults in a relaxed state with their eyes closed; beta band activity (13 – 30Hz) is considered to be normal wakefulness; theta band activity (4 – 8Hz) is ‘slow activity’ and is found in wakeful children into early adolescence and in sleeping adults; delta band activity (0.5 – 4Hz) is the slowest frequency, found in children up to one year old and in sleep stages 3 and 4 (Niedermeyer, 1998a; Niedermeyer, 1998b).

A number of processes may contribute to changes in frequency of electrical activity across the lifespan. Myelination of axons is important for efficient transmission of inter- and intra-hemispheric neural impulses, and this process continues into the third decade (Sowell et al., 2003). Studies have also suggested that the frequency of the electrical activity can influence the rate of myelination, although little is known about the mechanisms underlying this process (Demerens et al., 1996; Stevens, Tanner & Fields, 1998). The low frequencies found in neonates and in children may be partially accounted for by the continued development of cortical structures and neural circuits. For example, development of ‘EEG coherence’ throughout the first few years of life has been described (Thatcher, Krause & Hrybyk, 1986), and the development of adult patterns of sleep cycles has been linked to the maturation of the cortex (Joseph, 2000). Interestingly, the amplitude of the EEG has been found to decrease over the lifespan (Dustman, Shearer & Emmerson, 1999), and the authors suggest that age-related changes in cortical structure are the cause.

In summary, the changes in the EEG over the lifespan are considered to reflect the changes that occur in the CNS (Dustman et al., 1999).

Figure 3.1. The Frequency Bands of the EEG. From: brain.web-us.com/images/theanine.fig.1.jpg.



### 3.7.3. Event-Related Potentials.

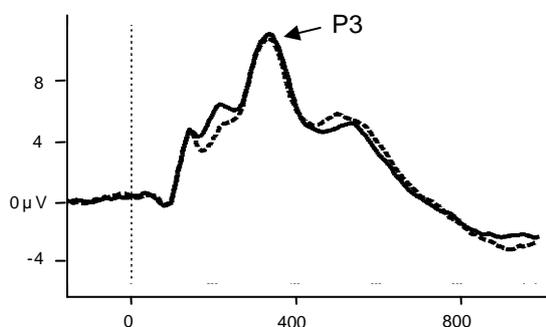
An ERP is the voltage change in the EEG signal in response to an event (stimulus/response) that is identified in a time-locked segment of an EEG recording (Coles & Rugg, 1995). Such EEG segments (epochs) are isolated from the EEG for display and analytic purposes. Due to the conductive properties of the brain, the activity at the scalp may not be from the neural assemblies closest to the recording sites, resulting in low spatial resolution (Coles & Rugg, 1995). Nevertheless, scalp distributions of ERPs (topographical maps) provide an assessment of gross topographical information, such as lateralisation and anterior/posterior orientation. However, ERPs have excellent temporal resolution and, as such, can provide information about the timing of sensory, motor and cognitive processes involved in experimental manipulations.

As ERPs are only a part of the entire EEG recording, these data have to be processed to extract the ERP (signal) from the EEG (noise) recording. Each time-locked segment (epoch) to an event (stimulus/response) within the entire EEG recording is averaged across

a number of trials to provide a single measure of the activity of that type or category of event (Coles & Rugg, 1995). The grand average waveform represents a number of overlaid EEG-epochs associated with the same kind of stimulus or response, thus it does not provide a direct measure of on-line activity per se, but an indication of how the brain typically responds to a particular event (Coles & Rugg, 1995). It is important to appreciate, however, that there can be great variation in the amplitudes and latencies of the individual trials. The benefit of averaging is that it allows an ERP component to be more easily identified.

There has been much debate about what constitutes an ERP component (Coles & Rugg, 1995; Luck, 2005; Otten & Rugg, 2005). Any chosen peak or trough within a data set can provide the component of interest, which is defined by its polarity and latency. For example, the P300 (often abbreviated to 'P3') is a positive component occurring around 300ms. The amplitude and latency of the component (measured from the grand average for each individual) provides the indices that are statistically examined. At present there are no definitive methods for identifying ERP components (Overbeek, Nieuwenhuis & Ridderinkhof, 2005), for example, the P300 may occur between 200 and 600ms (see Figure 3.2). The researcher must therefore be guided by their group grand average and by previous literature.

Figure 3.2. Example of a stimulus-locked waveform from the younger adult group recorded from Cz: Compatible condition (solid line) and incompatible condition (dashed line) elicited by the 4-CRT.



In addition to the reporting of the amplitude and latency, a component may also be described by its topographic distribution and/or by the cognitive process it represents

(Coles & Rugg, 1995). For example, the P300 associated with target (expected) stimuli is usually largest over central-parietal regions, while the P300 associated with novel (unexpected) stimuli is more often detected over central-frontal regions. More generally, a distinction has been made between ERP components that occur within about 200ms of the stimulus presentation and those that occur after that time. The early (exogenous) components are related to the sensory aspects of a stimulus and the later (endogenous) components are those more related to cognitive factors, such as categorisation of that stimulus and memory updating (Coles & Rugg, 1995). Although a distinction has been drawn between the sensory and cognitive aspects of ERP components, the relationship is dynamic; cognitive processes can influence earlier sensory components (e.g. directed attention can modulate N1 amplitude; Chao & Knight, 1997a) and later cognitive processing can be influenced by the modality of the stimulus (e.g. in divided attention conditions, cognitive processing occurs later in the auditory modality; Falkenstein, Hohnsbein, Hoormann & Blanke, 1991).

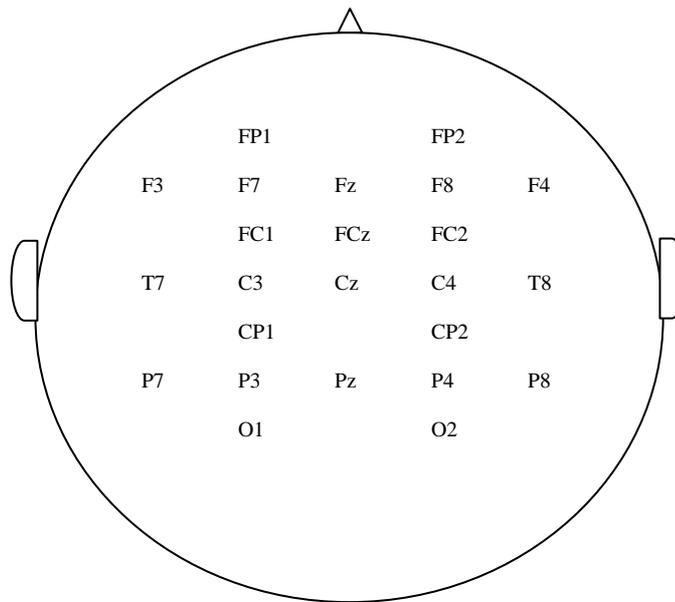
#### 3.7.4. EEG acquisition and ERP processing in the current study.

The EEG was recorded using an Easy-Cap<sup>TM</sup> fitted with 24 electrodes positioned over midline and lateral sites according to the International 10-20 system (see Figure 3.3: Jasper, 1958). The continuous EEG was recorded at a sampling rate of 500Hz (band-pass of 0.05 to 70 Hz: SynAmps 2 Amplifiers, NeuroScan, UK) using a linked-mastoid reference with a ground lead placed on the forehead (FP1). Vertical ocular activity (blinks), the EOG, was recorded from bipolar leads placed at FP2 and below the right eye, and horizontal EOG (lateral eye movements) from bipolar leads situated at the outer canthi of each eye. Impedance was kept below 15k $\Omega$ , checked at the start and end of the session.

ERP processing was carried-out offline using NeuroScan and Brain Vision Analyzer software. The EEG recording was low-pass filtered (20Hz) and blink artefacts were reduced using a standard algorithm (Semlitsch, Anderer, Schuster & Presslich, 1986). The EEG was divided into epochs centred on stimuli or responses, baseline corrected (each epoch is overlaid at a fixed time interval prior to time 0 ms). Variable time intervals were used in this thesis according to the component of interest: please refer to Chapters 4 and 5), movement artefact rejected ( $\pm 75\mu$ : each waveform that exceeds  $\pm 75\mu$  is excluded from

analysis, to reduce any potential confound of movement artefacts on the amplitude of the waveform) and averaged according to stimulus or response type. The amplitude and latency of the components of interest were measured and entered into a SPSS (version 13) file.

Figure 3.3. The montage of the lead placement following the International 10-20 system.



*Note.* Labels ending in a 'z' represent the midline (Frontal, Central, Parietal). Labels ending with an odd number represent activity recorded from the left hemisphere (e.g. T7 – left temporal lobe, O1 – left occipital lobe), and labels ending with an even number represent activity recorded from the right hemisphere. Labels with two letters represent activity recorded from midway between two main sites (e.g. FCz, represents fronto-central midline activity).

### 3.7.5. Overview of Event-Related Potential Paradigms Used in the Present Thesis:

#### i) Sensory Processing (Novelty Auditory Oddball Paradigm).

Participants were requested to sit as still as possible and look at a static image (small watercolour landscape) on the screen. They were asked to press a mouse button upon hearing target (high) tones which were randomly interspersed in a stream of standard (low) tones, but were not informed about novel (noise) stimuli which occurred with equal probability as the target tones. This paradigm assessed processing of simple auditory

sensory information. It was included as a measure of basic sensory awareness. The main components of interest were the stimulus-locked N1 and P3.

ii) Performance-Monitoring (4-Choice Response Task):

Participants were requested to respond to the direction of the arrows. Green and red arrow stimuli pointing left and right were presented randomly with equal probability. At the presentation of a green arrow, participants were instructed to press the mouse button corresponding to the direction of the arrow (compatible condition). For red arrows, participants were instructed to press the opposite mouse button in response to the direction of the arrows (complex, incompatible condition). The purpose of this paradigm was to examine brain activity occurring immediately after a response has been initiated: post-response evaluation. Specifically, it was of interest to see if brain activity was different for trials on which an error occurred compared to correct trials. Greater magnitude of difference between the amplitude of error/correct response-locked components (~50-150ms post-response) was hypothesised to reflect greater error-recognition, and subsequent slow wave activity (Pe: ~400ms) has been associated with awareness of having made an error (e.g. Nieuwenhuis, Ridderinkhof, Blom, Band & Kok, 2001).

### 3.8. Procedure.

A script for the procedure was followed to maintain consistency (Appendix 4). With the exception of one OA participant, the OA participants were visited at their homes for the first appointment. The YA participants and one OA participant were interviewed in the ERP laboratory at the University of Southampton. Written consent was obtained from each participant. The OA participants were requested to provide an informant and written consent was also obtained from the OA informants. The order of the questionnaire battery was constant across participants (Table 3.5). The informant was given the informant versions of the DEX, the MARS and the M-C 1 questionnaires to complete and at the end of the visit was interviewed with the Cornell Scale and the RAID. After the questionnaire battery was completed, the participant's colour vision was assessed, a skin test with the EEG cleaning gel was conducted to test for possible reactions (e.g. redness or itching), and head circumference measured in order to prepare the EEG cap prior to the ERP session. At the next visit, preparation of the participant for the EEG recording took approximately 30 minutes. The EEG recording commenced with a baseline EEG (2 minutes eyes closed and

2 minutes eyes open) and was followed by the practice trials for the auditory oddball, and the novelty oddball task. Four blocks of the 4-CRT were administered after the auditory oddball. If necessary, in order to increase error rate, an additional one or two blocks of the 4-CRT were administered. On completion of each block, participants were requested to provide an estimated percentage for their perceived accuracy of performance. The behavioural tasks were performed at the end of testing and were video recorded for scoring offline.

Table 3.5. Order of Assessments.

Telephone Screening (OA only)	TELE Participant Demographic Form (OA only)
Visit 1	Participant Demographic Form (YA only) Mini Mental State Examination Montreal Cognitive Assessment Rosenberg Self-Esteem Scale Hospital Anxiety and Depression Scale Marlowe-Crowne Social Desirability Scale National Adult Reading Test Metamemory In Adulthood questionnaire Pyramids and Palm Trees DEX Questionnaire Memory Assessment Rating Scale Ravens' Standard Progressive Matrices Rating Anxiety In Dementia scale Cornell Scale for Depression in Dementia
Visit 2	Hearing Assessment Positive and Negative Affect Schedule Baseline EEG Auditory Novelty Oddball 4-CRT Positive and Negative Affect Schedule Everyday Behavioural Tasks

### 3.9. Statistical Analyses.

Please refer to each empirical chapter (Chapters 4-7) for details of the statistical analyses performed in each study.

## Chapter 4. Sensory Awareness and Novelty Processing.

Adaptive cognitive functioning depends on the ability to detect, locate and process important stimuli from the ‘noise’ present in the environment at any one time. In other words, while many everyday events do not engage the individual, other events may be novel or unexpected, and therefore require further processing (Friedman, Cycowicz & Gaeta, 2001). Due to the potential significance of such stimuli (e.g. for learning or to signal danger), the early capture of attention by novel or unexpected events may be an automatic process (Friedman et al., 2001). Once attention is captured, it is likely to lead to further processing in terms of categorisation of stimulus-type and memory updating (Polich, 1996). With increasing experience of the same stimulus, there is habituation (Courchesne, Hillyard & Galambos, 1975). A study was designed to assess these basic attentional processes in younger and older adults, with the aim of exploring the extent to which the power, timing and topography (pattern of activity at the scalp) of brain function associated with Sensorimotor Awareness, Level 2 of the Hierarchies of Processing model (HoP; Stuss et al., 2001, see Section 1.4.3.2. Chapter 1) is altered by the normal ageing process.

### 4.1. Introduction.

#### 4.1.1. Overview of Cognitive and Neuroscience Approaches to Attention.

The focusing of attention is considered to be intrinsic to self-awareness (see Section 1.3.2., Chapter 1), and has been described as the process controlling access to conscious experience (Baars, 1988). The empirical investigation of attention has distinguished between selective (or focused) and divided attention. Selective attention can be voluntarily focused on a particular stimulus type or can be automatically captured by distracting or novel stimuli. Divided attention refers to the processing of different streams of information and/or the performance of different tasks. Selective attention is typically assessed by presenting two or more categories of stimuli with the requirement that attention is focused only on one type of stimulus. In contrast, divided attention is assessed by the performance of two or more concurrent tasks, usually a combination of perceptual and motor tasks. These conceptualisations of attention have led to the development of numerous theories involving different processes and components underlying attention. One of the prominent

early theories of selective attention suggested that all incoming sensory information was initially registered, and then filtered (Broadbent, 1958). The role of this filter is to prevent overloading of short-term memory, which has limited capacity. Specifically, the filter allows some information through to short-term memory, whilst holding the remainder of the information for later processing. In a similar, but amended version of Broadbent's (1958) model, Treisman (1964) proposed that all information was processed; however, the processing of unattended information was attenuated. This is contrary to Broadbent's (1958) view that the incoming, unattended information was held at a constant level of activation for later processing. However, the functional distinction between delayed and attenuated processing is not clear. Deutsch and Deutsch (1963) also amended the model proposed by Broadbent (1958). They argued that all incoming information was processed equivalently, however, only the most important or relevant to the goal (response) was transmitted to short-term memory. This suggests that there must be an executive mechanism exerting some 'top-down' control, in order to distinguish the relevant from the irrelevant. Consistent with this, more recent theorists have described the role of the frontal lobes in directing attention (e.g. Shallice & Burgess, 1996: Supervisory Attention System). Each of these theories incorporates the concept of an attentional bottleneck; as short-term memory has a limited capacity, the bottleneck functions to restrict incoming information. These models differ, however, in the degree of processing that unattended stimuli receive.

Since Broadbent's seminal paper in 1958, there has been a move in the literature away from the concept of a filter in attentional processing to a conceptualisation of automatic information processing. Early proponents of this view were Shiffrin and Schneider (1977) who proposed a distinction between controlled and automatic processes: controlled (top-down) processes required attention and were flexible, whilst automatic (bottom-up) processes did not require attention and were inflexible. Norman and Shallice (1986) expanded on the distinction between automatic and controlled processing and argued that there were three types of information processing: fully automatic processing by schemas, which occurred with little conscious awareness; partial automatic processing, which resolved any conflict arising from competing automatic schemas, and which could involve some degree of awareness; and deliberate control by a supervisory attentional system, suggested to be located in the frontal lobes, which supported decision making and was involved in responding to novelty. The automatic processing theories also posited, similar

to Treisman (1964), and Deutsch and Deutsch (1963), that all incoming information is processed.

The neuroscientific investigation of selective attention is important because it facilitates an understanding of how the multitude of incoming sensory information is processed in the brain. Cabeza and Nyberg (2000) reviewed the functional imaging (fMRI and PET) literature and found that selective attention, assessed by target detection, was associated with widespread, increased activation in parietal, temporal and occipital regions, with the profile of activation being dependent on the modality of the stimuli (e.g. auditory, visual). Frontal regions were additionally activated in tasks requiring detection of stimulus feature discrepancies. Such widespread activation may constrain our ability to define a functional neuroanatomy of selective attention, but it is important to appreciate the limitations of fMRI and PET. Attentional processing is likely to occur on a faster (~ms) time scale than may be measured by fMRI (~sec), and thus the widespread activation may mask sequential involvement of different neural regions.

The cognitive theories of selective attention discussed above, all suggest that the processing of unattended stimuli was a critical process to be explained. Cabeza and Nyberg (2000) reviewed a study (Rees, Frith & Lavie, 1997, cited in Cabeza & Nyberg, 2000) indicating that irrelevant stimuli were attended to (and had associated neural activity) but only when target detection required little attention. In other words, novel stimuli capture attention when attention does not have to be *fully* focussed on the task of target identification. More recently, Kincade, Abrams, Astafiev, Shulman and Corbetta (2005) investigated the neural regions associated with the orienting of automatic (stimulus-driven) and voluntary attention. These authors manipulated attention using a cue-target paradigm with 20 adults aged 18-30 years, and found that voluntary orienting of attention was associated with the dorsal fronto-parietal network, particularly the intraparietal sulcus and frontal eye field. There was some overlap with activation for automatic orienting: in the frontal eye field and occipito-temporal regions. Thus, by manipulating task demands and/or conditions, it is possible to identify more specific candidate neural regions associated with automatic and voluntary orienting of attention. However, a limited capacity for temporal resolution in fMRI studies means that it is important additionally to consider other neuroscience techniques. In particular, the sensitivity of event-related potentials to

the temporal aspects of cognitive functioning provides an opportunity to investigate the timing of these processes.

#### 4.1.2. Event Related Potential Correlates of Auditory Sensory Processing.

Event related potentials (ERPs) provide electrophysiological correlates of attentional capture, as they permit investigation of early brain activity associated with stimulus processing (within a few hundred milliseconds of stimulus presentation), and with further processing associated with more conscious cognitive assessment of the event (e.g. >300ms). Two components are of particular interest in the study of auditory stimuli, which is the focus of this chapter: the N100 (N1) and the P300 (P3).

The N1 has been associated with the monitoring of changes in auditory stimuli and, more generally, with attentional capture (e.g. Näätänen & Picton, 1987; Näätänen, 1988; Hillyard, Hink, Schwent & Picton, 1973). It appears as a negative peak between 50 and ~150ms after stimulus onset. However, the N1 component is actually associated with a number of negative peaks occurring within this time frame, defined by their latency and topography; for example, the N1b and N1c (Näätänen & Picton, 1987). The sites of maximal N1 recording are over the temporal lobes and midline regions. More specifically, the N1b is the term sometimes used to describe the N1 that is evident over central regions, whereas the N1c is used to describe the N1 appearing over temporal regions (Näätänen & Picton, 1987). Others simply refer to the N1, prefixed by an indication of location, e.g. central N1 (Senkowski, Linden, Zubrägel, Bär & Gallinet, 2003), or simply to the N1 (Jääskeläinen et al., 2004).

Halgren et al. (1995) examined data obtained using depth electrodes in patients with epilepsy, in an attempt to localise the neural generators of ERP components associated with auditory processing. These authors found activation in the posterior superior temporal plane corresponding to the amplitude and latency of the N1. More recently, Senkowski et al. (2003) used dipole localisation to confirm that the neural generators of the N1 were within the temporal regions. They described signal propagation upward to central regions and laterally to the temporal regions, a finding that is consistent with earlier topographical descriptions of the 'N1b' and 'N1c', respectively (e.g. Näätänen & Picton, 1987).

It has been suggested that the sensitivity of the N1 to changes in auditory stimuli reflects mechanisms (e.g. a 'pre-attentive gating') that direct novel stimuli into awareness and initiate the orienting of attention to that stimulus (Jääskeläinen et al., 2004). Interestingly, the amplitude of the N1 appears to be associated with the salience of the stimulus (Näätänen & Picton, 1987), such that novel stimuli elicit greater amplitudes than other types of stimuli. This may reflect the potential importance of such stimuli for learning, and indeed for survival if the stimulus indicates danger (e.g. car horn) (Friedman et al, 2001). It may only be inferred, however, that greater novelty N1 amplitude leads to greater capture of attention. Although a distinction has been made between the sensory and cognitive aspects of ERP components, the relationship is dynamic; this may be evidence supportive of the proposition that cognitive processes can influence earlier sensory processing (i.e. directed attention can also modulate N1 amplitude; Chao & Knight, 1997a).

The second component of interest is the P3, which is linked with more intensive information processing; when attention is involved in updating mental representations of stimuli (Donchin & Coles, 1988). The P3 is a positive peak, with maximal amplitude over midline sites. The latency of the P3 varies between 300 and 900ms, and its magnitude has been related to the probability of the stimulus, with greater amplitude for rarer stimuli (Coles & Rugg, 1995). As the P3 is elicited in paradigms that require a response to a rare (target) stimulus it has been associated with controlled attention (Segalowitz & Davies, 2004), but others have noted that it may be elicited independent of task relevance if the stimulus is novel (Courchesne, Kilman, Galambos & Lincoln, 1984). Like the N1, it has been suggested that the P3 is not a single component, rather it may represent a number of sub-processes reflecting the activity of inter-connected neural circuits (Coles & Rugg, 1995). Picton (1992) reviewed the literature and described three overlapping positive components within the P3 time-frame: the P3a (~250ms), P3b (~350ms), and a less specific 'positive slow wave'. While these subcomponents are often difficult to separate, they may also be considered on the basis of their eliciting stimuli. For example, the 'P3a' is elicited by rare stimuli presented in a three-stimulus task<sup>3</sup>, and the 'novelty P3' is elicited in a novelty paradigm when a third novel stimulus is introduced but about which the

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<sup>1</sup> Typically in a standard oddball task, one stimulus is presented at high frequency, and the other at low frequency, with an instruction to respond to the low frequency or 'target' stimuli and ignore the high frequency stimuli in the active version of the task. The target stimuli elicit the P3b.

individual is not informed (Simons, Graham, Miles & Chen, 2001). Of note, the term P3a is often used to indicate the P3 to novel stimuli in studies employing a novelty oddball paradigm (e.g. Katayama & Polich, 1998); while others advocate different nomenclature to represent potentially separate underlying cognitive processes (Simons et al, 2001)<sup>2</sup>. It has been reported that the P3a is present whether or not the participant is attending to the task (Picton, 1992), but there is also evidence that the P3 varies with the degree of attention directed towards the stimulus (Opitz, Mecklinger, Friederici & von Cramon, 1999), with greater magnitude associated with increased attention. Importantly, the P3 may be functionally differentiated on the basis of stimulus characteristics (e.g. target P3, and novelty P3).

In general, the P3a and novelty P3 are believed to represent immediate attention to the stimulus, but not memory processing per se (Friedman et al., 2001; Segalowitz & Davies, 2004). Memory updating, after initial attentional capture by the stimulus, is associated with the later P3b component which has been related to information processing and indexes the allocation of attentional resources to updating mental representations of stimuli (Polich, 1996). The term P3b is also used to describe the P3 to target stimuli which is maximal at parietal regions (Polich, 2004), as the timing and associated function is similar. The region of maximal amplitude of the P3b has been found to increase from anterior to posterior regions in younger adults (Fabiani & Friedman, 1995), which was interpreted by the authors to represent the maintenance of memory traces of the target stimuli.

#### 4.1.2.1. Functional Neuroanatomy of Auditory Sensory and Novelty Processing.

The novelty P3 and P3a are maximal at frontal sites and are generated by widespread neural regions, including the temporo-parietal junction and the prefrontal cortex (PFC) (Friedman et al., 2001). In support, using depth electrodes implanted in the brains of patients undergoing monitoring for epilepsy, Halgren and colleagues (Halgren et al., 1995; Halgren, Marinkovic & Chauvel, 1998) found widespread activation associated with the orienting of attention. Activation in the dorsolateral PFC and the supramarginal gyrus was associated with the P3a in a standard two-tone auditory oddball, whereas activation in the

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<sup>2</sup> In this study the term P3 will be used, with the prefixes 'standard', 'target' or 'novelty' where relevant (c.f. Friedman, Cycowicz & Gaeta, 2001).

ventrolateral PFC, superior temporal sulcus and the hippocampus was associated with the P3b.

Novelty processing, in general, has been linked with the PFC and its connections with the limbic system, specifically the hippocampus (see e.g. Yamaguchi, Hale, D'Esposito & Knight, 2004). One event-related fMRI study suggested the extent of neural involvement in P3 generation: target processing activated bilaterally the anterior superior temporal gyrus, inferior and superior parietal lobules, anterior and posterior cingulate, thalamus, caudate and the amygdalae/hippocampal complex, whereas novel stimuli activated bilaterally the inferior frontal gyrus, insula, inferior parietal lobule, and the inferior, middle and superior temporal gyri (Kiehl, Laurens, Duty, Forster & Liddle, 2001). This suggests that there are differences as well as similarities in the neural pathways underpinning target (voluntary selective attention) and novelty (involuntary attentional capture) processing. Convergent information has been obtained from adults with brain injury. An ERP study using a visual novelty oddball paradigm in individuals with frontal lobe damage found reductions in the amplitude of the novelty P3 and reduced attention to novel stimuli (Daffner et al., 2000)<sup>3</sup>. The authors suggested that infarctions in the PFC disrupted attentional neural networks, and that the reduced novelty P3 component reflected attenuated allocation of attention to novel stimuli. Taken together, these studies suggest that the P3 is the result of widespread activation of the frontal and association cortices, and that the novelty P3 may be particularly dependent on efficient frontal lobe functioning.

Novelty processing involves both detection of novel stimuli and habituation, the process by which novel stimuli become familiar (learned). Electrophysiological studies have shown that there is greater activity over frontal regions with the first presentation of a novel stimulus compared to activity at this region with repetition of the same novel stimulus, indicating evidence for dynamic neural 'learning'.

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<sup>3</sup> Auditory and visual oddball paradigms activate frontal and parietal regions with overlap (Stevens, Skudlarski, Gatenby & Gore, 2000).

#### 4.1.2.2. Habituation to Novelty.

In one of the earliest studies investigating orienting and habituation to novel stimuli, Courchesne et al. (1975) found that P3 amplitude to repeated novel stimuli increased from frontal to parietal regions with increasing time spent on task. When novel stimuli were presented only once, P3 amplitude was significantly increased at frontal and central sites, but this amplitude decreased with increasing experience of novel stimuli. Courchesne et al. (1975) interpreted the P3 elicited by single novel stimuli as representing an initial orienting evaluation: “what is it?” (Courchesne et al., 1975, p. 141). The authors further suggested that a ‘continuum of recognition’ influenced P3 amplitude, with greater recognition of stimuli being associated with greater novelty P3 amplitude over parietal regions. Cycowicz and Friedman (1997) also investigated dynamic habituation to novel stimuli during the course of a testing session, and found that P3 amplitudes over frontal regions indeed decreased with repeated exposure to novel stimuli, with step-wise amplitude reductions between children aged 9-11 years, adolescents and adults aged 22-28 years. A decrease in P3 amplitude at fronto-central regions with repetition of novel stimuli was also found in a later study by Cycowicz and Friedman (1999), however, no P3 amplitude decrease was found at parietal sites with habituation to repeatedly presented novel stimuli. Based on these findings, Cycowicz and Friedman (1999) suggested that the novelty P3 was composed of two aspects: a frontal aspect representing initial orienting to novelty, and a parietal aspect associated with the categorisation of stimuli.

Debener, Kranczioch, Herrmann and Engel (2002) assessed the effect of repetition on both the novelty P3 and the target P3 elicited by an auditory novelty oddball task in 14 adults aged 25-36 years, in two separate testing sessions seven days apart. In the first session, novelty P3 amplitude was maximal over central regions within 230-360ms, whilst the target P3 was maximal at parietal sites at longer latencies (400-580ms). The novelty P3 was found to decrease between the first and second half of the first recording session, but did not differ between sessions (seven day interval). Conversely, the target P3 was reduced in the second recording session compared to the first session, but did not differ between the first and second blocks of either session. Debener et al. (2002) suggested that the reduction in the novelty P3 from the first to second half of the first session represented habituation and initiation of automatic bottom-up processing, as novel stimuli captured less attention over time. The target P3 decrease was also associated with a reduction in the allocation of

attentional resources. However this does not fully explain the pattern of results. It is possible that there was fast habituation to novel stimuli, but that this represented short-term learning (within the session) rather than longer term memory updating. Either trial-unique novel stimulus take longer to form a memory trace because of their variability within the stimulus category 'novel', or some forms of novel stimuli do not lead to habituation, because it is not adaptive for them to become automatically processed, such as the case with stimuli alerting of danger. The target P3 amplitude decrease in the second recording session may have been due to the memory trace of the target stimuli requiring only a small amount of attention to update the mental representation. The behavioural performance of the task was near ceiling and did not differ between sessions (all error rates <2.4%). These findings suggest that task experience may have provided episodic recollection of the ease of the task, which may have influenced the amount of controlled processing required to perform the task.

The habituation of target stimuli has been of interest to a number of other investigators. For example, Segalowitz, Wintink and Cudmore (2001) examined the effect of task familiarity in 39 undergraduate students using a working memory task. This study found evidence of habituation, namely, a linear decline in amplitude of the target P3 at frontal regions over time. The reduction in the target P3 amplitude was suggested to indicate familiarisation with the task. Fabiani and Friedman (1995) also investigated the effect of repetition on target stimuli with a subgroup of younger ( $n = 6$ ) and older adults ( $n = 7$ ), and found that target P3 amplitude decreased over time. Furthermore, in the younger adults, the target P3 amplitude decreased at fronto-central sites with habituation, with similar target P3 amplitudes over parietal regions at all times. However, this effect was absent in older adults.

Taken together, these results indicate that habituation can occur with both novel and target stimuli in younger adults, and that habituation is associated with reductions in amplitude at fronto-central locations. It is logical to conclude that habituation to novel stimuli (typically, unexpected complex sounds) may take longer than habituation to target stimuli (typically, the same deviant tone repeated) due to their greater complexity, and the fact that novel stimuli are, by definition, unexpected. This may be the reason for the greater interest in novelty compared to target habituation in the literature. In partial support, Fabiani and Friedman (1995) found that target P3 amplitude in older adults did not significantly differ

in location, indicating that although some habituation was found, as evidenced by the reduction in target P3 amplitude with time, this was not associated with amplitude decreases at fronto-central sites. Of greater interest to the current study, this pattern of results suggests that the formation and revision of memory traces, as indexed by the target P3 (Polich, 1996) and perhaps also the novelty P3, requires more widespread frontal activity in older adults (Fabiani & Friedman, 1995). In support, studies investigating the effect of age on habituation to novelty have consistently found, using both auditory (Friedman et al., 1997; Friedman & Simpson, 1994) and visual (Weisz & Czigler, 2006) novelty oddball paradigms, that frontal novelty P3 amplitudes show reduced decline with repeated exposure of the stimuli with ageing indicating that, within the confines of ERP studies, older adults do not habituate to novelty as much as younger adults. It is important, therefore, to consider carefully the more general evidence for attentional-ERP activity in older adults.

#### 4.1.3. Event-Related Potential Correlates of Sensory Processing in Normal Ageing.

The N1 and P3 components may be examined to investigate age-related cognitive changes (Polich, 1996), facilitating our understanding of age effects on attention, sensory perception and awareness. For example, older age has been associated with ERP evidence of orienting to irrelevant stimuli (i.e. distractibility), whilst a latency deficit has been discussed in terms of speech perception problems (e.g. Ostroff, McDonald, Schneider & Alain, 2003).

Significant decline in grey matter volumes in the frontal and temporal lobes (Raz, 2000) and white matter degeneration in the corpus callosum and frontal regions (Salat et al., 2005) have been associated with increasing age. It is conceivable, therefore, that this may affect the ability to detect and process stimuli by reducing the strength of signals along neural networks, slowing the speed at which these networks function, and by decreasing the ability of gating mechanisms in the sensory and association cortices to inhibit irrelevant stimuli (see Hasher & Zacks, 1988; Jääskeläinen et al., 2004; Raz, 2000; Salthouse, 1996). These gross brain changes may also alter the power of the underlying EEG (Dustman et al., 1999), and therefore the ERP components elicited. Age-related differences in ERP

components have been found using both standard and novelty oddball paradigms, and some of the findings are now discussed.

#### 4.1.3.1. Standard Two-Tone Auditory Oddball Task.

The standard two-tone auditory oddball paradigm has been used to investigate age-related changes in attentional processes. This task may be administered passively during which participants ignore the standard (high probability) and deviant (low probability) tones, or actively where participants respond to the deviant (target) tone; the latter task is likely to require more top-down focusing of attention (see e.g. Debener et al., 2002). Of note is that the ERP components elicited by passive and active manipulations with children have been found to be similar in morphology, amplitude and latency (Zenker & Barajas, 1999). To the author's knowledge, a similar comparison has not been conducted with adults, but would be informative.

There are some inconsistencies with regard to age-related changes in the amplitude of the N1 elicited by the two-tone oddball paradigm. Anderer, Semlitsch and Saletu (1996) assessed a sample of 172 participants aged 20-88 years, separated into 6 groups (20-29, 30-39, 40-49, 50-59, 60-69, 70+ years), and found that standard N1 amplitudes at midline sites increased with age ( $-0.03 \mu\text{V}/\text{year}$  at Fz and Cz, and  $-0.02 \mu\text{V}/\text{year}$  at Pz). The latency of the standard N1 was also found to increase with age ( $0.12 \text{ ms}/\text{year}$  at Pz), indicating that older adults had slower, yet enhanced attentional capture by irrelevant stimuli. In support, Amenedo and Diaz (1998) also found that standard tones produced an increase in N1 amplitude with ageing in a sample aged 20-86 years, separated into 3 age groups (20-39, 40-59, 60-86 years). However, no latency differences were found between the groups, indicating that the timing of attentional capture did not differ with age in this study. These authors speculated that older adults may attend to task-irrelevant stimuli to a greater degree than younger adults, because older adults may experience even standard stimuli as more ambiguous, and thus more 'novel'. However, neither of these studies described the target N1, which would have provided an interesting comparison for the standard N1 data. Nevertheless, in support of the proposition that older adults attend to irrelevant stimuli to a greater extent than younger adults, Chao and Knight (1997b), using a different ERP paradigm (delayed matching-to-sample task), found that N1 amplitudes to all stimuli were larger in older adults. This finding may be partly explained by the task requirements of the

delayed matching-to-sample task, in that each stimulus required a response, whereas oddball paradigms only require participants to monitor and sometimes respond to target stimuli with a low probability of presentation.

Other studies have investigated the N1 elicited by standard and target stimuli, using a variant of the active oddball manipulation (e.g. requiring a response to targets). Iragui, Kutas, Mitchiner and Hillyard (1993) did not find any age effects on either standard or target N1 amplitudes, in a sample of 71 participants aged 18-82 years, separated into 3 age groups (18-39, 40-59, 60-82 years). These authors additionally found that the topography of the N1 for targets and standards was similar across groups, with maximal recordings at central regions. In support of Anderer et al. (1996), standard N1 latency increased with age. However, no age differences were found on target N1 latency. In a sample of 120 participants aged 18-80 years, separated into similar age groups as Anderer et al. (1996), Polich (1997) employed both visual and auditory oddball paradigms, and also did not find an age-effect on the target N1 amplitude. However, a main effect of group was found for N1 amplitude to standard tones; as actual data were not presented the degree of an age effect on standard N1 amplitude is not clear. No age-related differences in latency were found either for standard or target N1. Despite the general null finding of age effects on the target N1, a negative correlation between age and auditory target N1 amplitude was found, suggesting that initial attentional capture of auditory targets may be sensitive to increasing age.

Behaviourally, the older adults performed the oddball tasks equivalently to younger adults (Amenedo & Diaz, 1998; Anderer et al., 1996; Polich, 1997). Reaction times in the oddball paradigm requiring a response to target stimuli were also similar across groups (Iragui et al., 1993; Polich, 1997). This is an important consideration, as a difference in behavioural performance can have implications for the interpretation of ERP data (see Hogan et al, 2005, for a discussion). The finding of decreased behavioural target detection in one study of older adults (Iragui et al., 1993) cannot be explained by methodological issues, as this was the only study both cognitively to screen their sample with the Mini Mental State Examination (Folstein, Folstein & McHugh, 1975) and assess the ability to discriminate between the stimulus tones. The other oddball studies reported above did not assess the hearing levels of their older adult participants, and Polich (1997) did not report the use of any cognitive screening measures. Although the lack of hearing assessments may be

considered a limitation, the equivalent behavioural performance of the older and younger participants in the majority of studies argues against any impact impaired hearing may have had on performance.

In summary, studies have generally found that the N1 amplitude to standard stimuli increases with age (Amenedo & Diaz, 1998; Anderer et al., 1996). However, the timing (latency) of this attentional capture by irrelevant stimuli has been found to differ between studies. It has been suggested that older adults are less able to discriminate between stimulus types requiring attention and may treat each stimulus as 'new' (Amenedo & Diaz, 1998). Neither study focussing on the target N1 found any age-related differences in amplitude or latency (Iragui et al., 1993; Polich, 1997), suggesting that the timing and the degree of initial attention to target stimuli were similar across age groups. However, the correlation reported by Polich (1997) indicates that there may be a relationship between increasing age and decreasing N1 amplitude. The most conservative conclusion is that these data suggest that older adults may process some aspects of auditory stimuli differently to younger adults, and that this may be associated with normal brain changes in ageing.

#### 4.1.3.2. Novelty Auditory Oddball Task.

The novelty auditory oddball is a modification of the standard oddball paradigm that allows for the investigation of attentional capture by unexpected, novel (e.g. potentially threatening) stimuli. Controlling for the probability of novel stimuli and target tones (both are equally infrequent compared to standard stimuli), the novelty oddball allows for the investigation of novel versus anticipated (e.g. target) 'deviant' stimuli. This type of novelty oddball paradigm also provides greater ecological validity as the novel stimuli (e.g. dog bark, car horn) may be more meaningful to the individual in terms of potential threat.

Few studies have used this task to explore the ageing brain, and those data that are available are as variable in terms of age-related differences in amplitude and latency of ERP components as those obtained from the standard oddball task. In such tasks, younger and older adults have comparable N1 amplitudes and latencies to standard stimuli (Fabiani & Friedman, 1995; Friedman, Simpson & Hamberger, 1993); unfortunately, Fabiani and

Friedman (1995) did not report on the N1 elicited by target or novel stimuli in the same task. In their sample of 10 younger adults (21-28 years), 10 middle-aged (43-55 years) and 10 older adults (65-74 years), Friedman et al. (1993) additionally found that standard N1 topography was similar across age groups (see also Iragui et al., 1993), showing greatest negativity over fronto-central regions. Similar to Amenedo and Diaz (1998) and Anderer et al. (1996), Friedman et al. (1993) did not report the N1 for target and novel stimuli. Fabiani and Friedman (1995) found decreasing amplitudes of the standard (central) N1 from frontal to parietal regions in younger adults ( $n = 8$ , age range 22-28 years), whereas the older adults ( $n = 8$ , age range 65-88 years) showed no significant differences across the midline regions. This suggests that the older adults had a more equal distribution of negativity associated with the standard N1.

Similar to ageing studies using the standard oddball task, studies utilising the novelty oddball task have not found age effects on RT (Friedman et al., 1993) or accuracy of identification of the target tone (Fabiani & Friedman, 1995). Interestingly, however, Friedman et al. (1993) found that older adults provided more false alarms (erroneous manual responses) to novel stimuli. Although no age difference was found in response bias, discrimination (a measure of sensitivity to the stimuli) decreased with age for the novel stimuli, indicating that the novel false alarms were due to the older adults being less sensitive to the difference between the stimuli (Friedman et al., 1993). Both studies screened for hearing thresholds and excluded participants with significant hearing impairments ( $>35\text{dB}$ ; and/or difference between ears  $>20\text{dB}$ ; and/or difference between best and worst threshold  $>30\text{dB}$ ), and increased the volume for participants who had hearing impairments which were not severe enough to reach the exclusion criteria (borderline cases). It is therefore unlikely that the decrease in sensitivity to novel stimuli, as measured by the discrimination index, was due to hearing deficit in a proportion of the participants. However, it would be of interest to investigate the possibility that false alarms are confined to novel stimuli (Friedman et al., 1993; Weisz & Czigler, 2006), as this might indicate a degree of 'startle', potentially influenced by individual level of anxiety, or if false alarms also occur in association with standard stimuli (e.g. Friedman et al., 1993), in which case performance may be considered to be generally more impulsive and/or less inhibited. Of note, Hogan, Butterfield, Phillips and Hadwin (2007) found that anxiety, within the normal range, modulated the N1 in children, with higher amplitudes in those with higher levels of anxiety. Another limitation of this literature is that studies do not

consistently describe/compare the N1 elicited by the different stimulus types (standard, target and novel). Notwithstanding these concerns, the N1 amplitude age-differences that are described typically occur in the presence of normal task behaviour, indicating that differences in initial sensory perception may have little effect on subsequent behavioural performance. In other words, whilst it may be considered that older people process stimuli slightly differently, their behavioural responses are similar to those in younger people.

The investigation of the later P3 component has provided more consistent findings with regards to age-related changes. Anderer et al. (1996), in a relatively large sample of 172 participants aged 20-88 years, found that the amplitude of the target P3 decreased with age at the midline sites (-0.12 and -0.15  $\mu\text{V}/\text{year}$  Cz and Pz, respectively) and the latency increased (0.97, 0.92, 0.92 ms/year Fz, Cz and Pz, respectively). Indeed, the finding of age-related reductions in target P3 amplitude and increases in P3 latency has been shown in numerous studies (Amenedo & Diaz, 1998; Chao & Knight, 1997b; Fabiani & Friedman, 1995; Friedman et al., 1993; Iragui et al., 1993; Polich, 1997; Weisz & Czigler, 2006). Friedman et al. (1993) also described different P3 topography with ageing. For the target P3, older adults ( $n = 10$ , age range 65-74 years) had a predominantly frontal activation whereas the younger ( $n = 10$ , age range 21-28 years) and middle aged ( $n = 10$ , age range 43-55 years) groups had more parietal distributions. Novel stimuli were associated with an increased frontal compared to posterior activation in all age groups.

In one of the most recent studies of older adults using a novelty visual oddball paradigm, Daffner et al. (2006) found that high functioning older adults ( $n = 16$ ,  $M$  Age 73.0,  $SD$  4.9), categorised by performance in the top third percentile on a range of neuropsychological tests, had greater novelty P3 amplitudes compared to older adults with average performance ( $n = 15$ ,  $M$  Age 70.1,  $SD$  4.3). Furthermore, better performance on tests of attention and executive function was correlated with increased P3 amplitude over the midline ( $r = .44$ ). This suggests the importance of controlling for level of cognitive function, as well as hearing level.

Habituation to novelty in ageing has also been investigated using the P3 elicited by novel and target stimuli. As described above (p. 100), Fabiani and Friedman (1995) found some evidence of target P3 habituation in a subsample of older adults. Others have investigated habituation to novelty using a visual oddball paradigm (Weisz & Czigler, 2006), and an

auditory oddball paradigm (Friedman, Kazmerski & Cycowicz, 1998), and found little evidence of habituation to novelty in older adults. However, the discrepancies between these studies with regard to age-related differences in the amplitude of the novelty P3 overall, indicates that further investigation of habituation in ageing is warranted. Another reason for this is the interesting differences in P3 topography emerging from some studies. For example, there is evidence that novel stimuli are associated with a more frontal orientation in younger and older adults (e.g. Friedman et al., 1993). There are age-related differences in the topographical distribution of target P3 activity over the same frontal regions. Younger adults typically show a more parietal compared to frontal distribution of activity in association with target stimuli, which may represent fast habituation to a simple type of stimuli. In contrast, older adults have been found to show more widespread distribution of activity at frontal and parietal regions to the same target stimuli (Fabiani & Friedman, 1995). This pattern of electrical activity at the scalp suggests that older adults require additional neural resources to process even basic target stimuli and to support task performance, although this cannot be assessed with ERP components and topographical maps alone, and/or that they fail to habituate to stimuli as efficiently as younger adults. In support, a link has been hypothesised between increased P3 latency and a reduction in dendrites and myelin in the frontal regions (Amenedo & Diaz, 1998). The reported increased latency of the target P3 (e.g. Amenedo & Diaz, 1998; Chao & Knight, 1997b; Fabiani & Friedman, 1995; Friedman et al., 1993; Polich, 1997), may indicate that the process of initiating memory updating is constrained in ageing; in other words it is less likely that an individual will habituate to something that they have not had the opportunity fully to process<sup>4</sup>. Alternatively, Fabiani and Friedman (1995) suggested that older adults are less able to inhibit processing of irrelevant stimuli and need the recruitment of frontal regions to help them in this task, possibly due to increased memory decay and difficulties in maintaining mental representations. This is also indicated by increased false alarms to novel stimuli (Friedman et al, 1993; Weisz & Czigler, 2006). Such hypotheses may only be tested by analysis of the degree to which older adults, compared to younger adults, habituate to novel stimuli during the course of the task, and by investigating topographic alongside amplitude data.

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<sup>4</sup> Polich (1996) suggested that P3 latency was associated with the timing of stimulus evaluation.

#### 4.1.4. Aims of the Study.

With regard to the Stuss et al. (2001) HoP model, deficits in Sensorimotor Awareness may exist, with potential to impact on higher levels of awareness, associated with frontal lobe (executive) functioning. One of the aims of the study was to investigate how the N1 and P3 components associated with basic auditory stimuli may clarify the degree to which this level of awareness is affected by the ageing process. Specifically, it was considered that any significant behavioural or ERP difference between younger and older adults, associated with the processing of basic auditory stimuli, may indicate a deficit in Sensorimotor Awareness, with potentially adverse consequences for higher levels of awareness. The novelty auditory oddball paradigm was administered to allow for simultaneous investigation of online habituation to novel stimuli, a function potentially reflective of the importance of interacting sensorimotor and executive function levels of awareness (Levels 2 and 3 in the Stuss et al., 2001 HoP model). In order to address these aims, the following hypotheses were tested.

1. Novel stimuli would elicit a larger N1 and P3 response compared to both standard and target stimuli.
2. Older adults would have attenuated and delayed ERP components compared to younger adults.
3. Older adults would show more widespread orientation (pattern) of activity compared to younger adults.
4. Younger adults would show reduced frontal activation with repeated exposure to novel stimuli, but this pattern of activity would be reduced or absent in older adults.
5. Higher level of anxiety would be associated with higher N1 and P3 amplitudes to target and novel stimuli.

## 4.2. Method.

### 4.2.1. Participants.

Twenty-six older adults (OA: *M* Age 69.3, *SD* 6.7) and 17 younger (YA: *M* Age 21.9, *SD* 4.4) were administered the novelty auditory oddball paradigm. However, due to insufficient numbers of trials for the target and novel waveforms (<15 trials in each), one YA and five OA were excluded from the analysis. Further, problems with access to audiometry equipment resulted in failure to obtain hearing levels in a proportion of the remaining sample (31.1%), and thus the sound pressure level could not be correctly determined in these individuals. Exploratory statistical analysis revealed that data were not normally distributed; those without hearing assessment featured in the tail of the distribution. It was, therefore, decided to limit analyses to those OA and YA who had had a corrected sound pressure level. The final sample was composed of 14 OA (*M* Age 69.1, *SD* 7.1) and 13 YA (*M* Age 20.3, *SD* 3.6); this sample size is larger than those included in earlier ERP studies of ageing using a novelty oddball paradigm (e.g. Fabiani & Friedman, 1995; Friedman et al., 1993). Importantly, the OA and YA subgroups were comparable in terms of general intellectual ability as measured by the Raven's Standard Progressive Matrices (Raven, Raven & Court, 2000): OA: *M* = 39.7, *SD* 3.2; YA: *M* = 39.8, *SD* 3.1;  $t(24) = .095, p = .925$ .

### 4.2.2. Measures.

#### i) Novelty Auditory Oddball Paradigm.

Permission to use this paradigm was given by Dr. Torsten Baldeweg, Developmental Cognitive Neuroscience Unit, UCL Institute of Child Health, auditory stimuli were presented via headphones. Sound pressure levels of stimuli (65dB: level 1 = <25 dB loss; 75 dB: level 2 = 25-40 dB loss; 85dB: level 3 = <40 dB loss) were set for each participant depending on their hearing threshold level (OA: level 1  $n = 11$ ; level 2  $n = 2$ ; level 3  $n = 1$ , YA: level 1  $n = 13$ ). Stimuli (200ms, 5ms rise and fall time) were pure sinusoidal tones: standard tone (1 kHz, 0.8 probability) and target tone (1.5 kHz, 0.1 probability), and

computer generated novel sounds (e.g. dog bark, drum beat, car horn, 0.1 probability<sup>5</sup>), with stimulus-onset-asynchrony of 900 ms. During the task, participants were requested to sit as still as possible and look at a static image (small watercolour landscape) on the screen. They were asked to press a mouse button upon hearing the target tone, but were not informed about the novel stimuli. A practice trial of 8 standard tones randomly interspersed with 4 target tones was administered to ensure that

participants could discriminate between the tones and understood the instructions. All participants were able to perform the practice trial successfully on the first attempt.

ii) The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) (described in Chapter 3).

i) anxiety subscale

ii) depression subscale, with higher scores reflective of more negative affect.

4.3. ERP Acquisition and Processing (see Section 3.7.4, Chapter 3 for detailed description).

The EEG was recorded using NeuroScan SynAmps<sup>TM</sup> amplifiers at a sampling rate of 500Hz (band-pass 0.05-70Hz) from 24 leads located over lateral and midline sites, using a ground lead situated at Fp1, and referenced to linked mastoids. Impedance was maintained at less than 15K $\Omega$ . Vertical (right eye) and lateral ocular electrodes enabled offline blink reduction according to a standard algorithm (Semlitsch et al., 1986). EEG data were epoched at -200 to 1000 ms centred on presentation of stimuli, baseline corrected at -200 to 0 ms, and artefact rejected at  $\pm 75\mu\text{V}$ . Data were averaged according to stimulus type

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<sup>5</sup> The novelty auditory oddball paradigm included potentially identifiable (meaningful) and non-identifiable novel stimuli. Research has shown that meaningful novel stimuli may additionally elicit the N400, associated with semantic categorisation of stimuli (e.g. Mecklinger, Opitz & Frederici, 1997; Opitz et al., 1999). Whilst the current study was designed to assess sensory attentional processing, rather than semantic processing, it would be of interest to investigate overlapping N400 activity to determine if some novel noises were more meaningful, and thus perhaps elicited greater *awareness*, than other stimuli.

(target, standard and novel). The two components of interest (lateral N1; central N1<sup>6</sup>; P3) were identified and defined on the basis of the group grand average as the maximum peak within a specified time frame: lateral and central N1 (70 – 250 ms) and P3 (200 – 520 ms).

#### 4.4. Habituation Analysis.

Fourteen different novel stimuli were presented, and each was repeated up to four times during the paradigm, facilitating an exploration of habituation to novel stimuli in ageing. Novel 1 represents the P3 amplitude from the grand-average ERP derived from the first presentation of the novel stimuli, Novel 2, the second presentation, Novel 3, the third presentation and Novel 4, the fourth presentation. This factor was labelled ‘time’.

#### 4.5. Statistical Analysis.

Shapiro-Wilk tests of normality were conducted to assess fit with the normal distribution. The percentage of hits and false alarms to these stimuli was calculated in addition to the target RT, and analysed using Independent Samples *t* tests. ERP data were analysed using mixed-design ANOVA for amplitude and latency of the N1 and P3 components separately: stimulus (x3: low, target and novel), lead (for lateral N1, x2: T7 and T8; for central N1<sup>7</sup> and P3, x3: Fz, Cz and Pz) and group (x2: OA and YA). Significant interactions were explored using ANOVA and Independent Samples *t* tests. For the examination of novelty habituation, data from Fz and Pz were subjected to a mixed-design ANOVA model with group as the between subjects factor, and time (x4: Novel 1 {1<sup>st</sup> presentation}, Novel 2, Novel 3, Novel 4) and location (x2: Fz, Pz) as the within-subjects factors. Planned (a priori) tests were also conducted to explore age differences in habituation of novel stimuli at the frontal (Fz) location.

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<sup>6</sup> The reader is reminded that the N1 has two subcomponents (Senkowski et al., 2003): a lateral component that is maximal over the auditory cortex, and a central component that is maximal over the fronto-central midline regions. Both components are generated in the auditory cortex but the lateral component is orientated radially (outward), whereas the central component is orientated tangentially (upward).

<sup>7</sup> A separate analysis of the central and lateral N1 was conducted to measure these subcomponents of the N1 (see previous footnote). Furthermore, Hogan et al. (2007) found that anxiety modulated the lateral N1, and not the central N1.

## 4.6. Results.

### 4.6.1. Behavioural Responses.

Mean scores and standard deviations are presented in Table 4.1. This reveals that target detection and reaction times to targets were equivalent across groups. However, OAs responded erroneously to standard stimuli more often than YAs,  $t(25) = 2.63, p = .014$ . There was no group difference in the rate of false alarm responses to novel stimuli.

Table 4.1. Mean (*SD*) for target hits, target RT, novel false alarms and standard false alarms.

	<u>Younger</u> <i>n</i> = 13	<u>Older</u> <i>n</i> = 14
Target hits (%)	96.3 (5.1)	92.7 (9.8)
Target RT (ms)	427.3 (40.9)	451.1 (56.9)
Novel false alarms (%)	4.3 (6.1)	6.0 (5.2)
Standard false alarms (%)	0.3 (0.3)	1.5 (1.7) †

*Note.* †  $p < .05$ .

### 4.6.2. ERP Components.

#### 4.6.2.1. Lateral N1 Component.

Amplitudes and latency means are presented in Table 4.2. Only a stimulus main effect was found for the amplitude of the lateral N1 over the auditory cortex,  $F(2, 50) = 44.69, p < .001$ . Post hoc analyses found that novel N1 amplitude was greater than standard N1 amplitude ( $p < .001$ ), and that the target N1 amplitude was greater than that of the standard N1 ( $p < .001$ ). There was no difference between target and novel N1 amplitude, suggesting that the lateral N1 may have been sensitive to stimulus probability, but not to stimulus novelty. The lack of group main effects on either the amplitude or the latency of the lateral N1 indicates equivalent magnitude and timing of brain activity associated with auditory sensory processing in the temporal lobes in YA and OA (see Table 4.2.).

#### 4.6.2.2. Central N1 Component.

##### 4.6.2.2.1. Amplitude.

Amplitudes means are presented in Table 4.2, and, for clarity due to multiple significant values, the results of the ANOVA model are presented in Table 4.3. This shows that main effects of stimulus, location and group were found for the central N1 amplitude. Post hoc analyses confirmed that, irrespective of group and location, the greatest amplitude was associated with novel stimuli compared to that elicited by both target stimuli ( $p = .043$ ), and standard stimuli ( $p < .001$ ). Target N1 amplitude was also significantly larger than that of the standard N1 ( $p < .001$ ). Further analysis of the location main effect revealed that the location of maximal N1 amplitude was Cz compared to Fz and Pz (both  $p < .001$ ). The N1 amplitude at Fz was significantly greater than that at Pz ( $p < .001$ ), as suggested in Figure 6.1. The main effect of group indicated that, regardless of stimulus type or location, the OAs had significantly lower N1 amplitudes compared to YAs. However, as indicated in Table 4.3, there were no significant interactions between group and either stimulus type or location.

##### 4.6.2.2.2. Latency.

Latency means are presented in Table 4.2, and the results of the ANOVA model are presented in Table 4.4. A main effect of stimulus was also found for the central N1 latency, explained by longer novel N1 latency compared to both target N1 latency ( $p = .009$ ) and standard N1 latency ( $p = .009$ ). As shown in Table 4.2, in the YA group, irrespective of location, N1 latency was shortest for target stimuli, whereas for the OA group, N1 latency was shortest for standard stimuli. This resulted in a significant interaction between stimulus and group (see Table 4.4). Further analyses revealed that this was driven by a group effect for standard stimuli only,  $F(1, 25) = 7.93$ ,  $p = .009$ . A location by group interaction was also found (Table 4.4) indicating topographical differences in the timing of the N1 between the groups, irrespective of stimulus type. There was a main effect of group only over the frontal regions (Fz),  $F(1, 25) = 7.72$ ,  $p = .010$ . It was of interest to investigate the possibility of a significant group difference confined to standard stimuli at Fz, and this was indeed found (see Table 4.2.). In summary, these data suggest a frontal orientation associated with the processing of standard stimuli in older adults.

Table 4.2. Mean (*SD*) peak amplitudes and latencies of the central N1 and lateral N1 components for younger and older adults.

	<u>Younger</u>	<u>Older</u>	<u>Younger</u>	<u>Older</u>
	Amplitude ( $\mu$ V)		Latency (ms)	
<u>Lateral</u>				
T7 Novel	-4.1 (2.0)	-4.6 (2.8)	154.5 (23.7)	164.3 (27.1)
T8 Novel	-4.1 (2.6)	-3.5 (2.7)	164.0 (33.0)	150.9 (19.5)
T7 Target	-4.5 (1.5)	-4.5 (3.5)	165.2 (31.6)	174.4 (42.7)
T8 Target	-4.4 (2.1)	-3.3 (2.3)	168.6 (24.0)	158.9 (25.7)
T7 Standard	-1.8 (1.0)	-1.4 (0.9)	159.2 (24.5)	143.4 (27.3)
T8 Standard	-2.3 (1.1)	-1.2 (1.0)	177.1 (18.3)	151.3 (24.5)
<u>Central</u>				
Fz Novel	-7.7 (2.0)	-6.5 (2.7)	165.1 (30.9)	156.7 (20.6)
Cz Novel	-9.7 (2.5)	-8.4 (5.3)	156.5 (14.7)	171.0 (37.3)
Pz Novel	-5.7 (2.1)	-5.8 (3.7)	143.5 (18.7)	168.4 (34.8)
Fz Target	-8.2 (2.0)	-5.5 (2.3) †	156.9 (38.6)	141.6 (14.8)
Cz Target	-7.9 (2.0)	-5.8 (3.1) †	151.4 (42.5)	146.3 (31.7)
Pz Target	-5.1 (2.1)	-4.0 (2.4)	133.5 (18.1)	148.4 (32.8)
Fz Standard	-3.4 (1.8)	-2.3 (1.4)	177.7 (49.4)	133.4 (10.3) †
Cz Standard	-3.9 (2.0)	-2.7 (1.6)	152.0 (33.6)	136.1 (10.4)
Pz Standard	-3.2 (1.3)	-2.7(1.6)	142.6 (17.7)	138.0 (12.6)

Note. †  $p < .05$  (Post hoc Independent Samples  $t$  tests).

Table 4.3. Results from the ANOVA model for the central N1 amplitude.

		df	<i>F</i>	<i>p</i>
Main Effects	<b>Stimulus</b>	2, 50	34.70	< <b>.001</b>
	<b>Location</b>	2, 50	25.12	< <b>.001</b>
	<b>Group</b>	1, 25	4.24	<b>.050</b>
Interactions	<b>Stimulus X Location</b>	2, 50	15.35	< <b>.001</b>
	Stimulus X Group	2, 50	0.69	.509
	Location X Group	4, 100	2.45	.096
	Stimulus X Location X Group	4, 100	0.40	.811

*Note.* Significant effects in **bold** type; Stimulus = standard, target, or novel stimuli; Location = Fz, Cz, or Pz; Group = Older or younger adults.

Table 4.4. Results from the ANOVA model for the central N1 latency.

		df	<i>F</i>	<i>p</i>
Main Effects	<b>Stimulus</b>	2, 50	5.10	<b>.010</b>
	Location	2, 50	2.98	.060
	Group	1, 25	0.50	.486
Interactions	Stimulus X Location	2, 50	0.93	.453
	<b>Stimulus X Group</b>	2, 50	5.29	<b>.008</b>
	<b>Location X Group</b>	4, 100	9.52	< <b>.001</b>
	Stimulus X Location X Group	4, 100	0.36	.839

*Note.* Significant effects in **bold** type; Stimulus = standard, target, or novel stimuli; Location = Fz, Cz, or Pz; Group = Older or younger adults.

#### 4.6.2.3. P3 Component.

##### 4.6.2.3.1. Amplitude.

Amplitude means are presented in Table 4.5. As shown in Table 4.6, there were main effects of stimulus and location for P3 amplitude. The stimulus main effect is examined

first. Post hoc examination revealed that novelty P3 amplitude was significantly greater than that of target P3 (see Figure 4.1), and standard P3 (both  $p < .001$ ) amplitudes. Target P3 amplitude was also greater than that for standard stimuli ( $p < .001$ ). The main effect of location (Table 4.6.) was explained by the fact that P3 amplitude at both Cz and Pz was significantly greater than that recorded at Fz (all  $p < .001$ ).

The stimulus by group interaction (Table 4.6.) indicated that there were group differences in P3 amplitude for different stimulus types, and a three way interaction with location suggested the presence of topographical differences. These interactions were explored in a series of post-hoc analyses and by examining the results of the planned group comparisons (Table 4.5). A main effect of group was found at Fz,  $F(1, 25) = 7.55, p = .011$ , and stimulus by group interactions were found at Cz,  $F(2, 50) = 6.20, p = .004$  and Pz,  $F(2, 50) = 6.85, p = .002$ . The results of planned comparisons confirmed that OA had higher standard P3 amplitudes at Fz, Cz and Pz, compared to YA (see Table 4.5 and Figure 4.2), and lower target P3 amplitudes at Pz, but higher amplitudes at Fz compared to YA (see Table 4.5 and Figure 4.3).

#### 4.6.2.3.2. Latency.

Latency means are presented in Table 4.5, and the results of the ANOVA model are presented in Table 4.7. There was a main effect of stimulus type on P3 latency. Standard P3 latency was found to be significantly shorter than both target P3 latency ( $p = .004$ ) and novelty P3 latency ( $p = .019$ ); a difference between target and novelty P3 latency fell just short of statistical significance ( $p = .053$ ). A stimulus by group interaction (Table 4.7) was explored and main effects of group were found for standard stimuli,  $F(1, 25) = 9.05, p = .006$ , and novel stimuli,  $F(1, 25) = 19.91, p < .001$ , supported by group differences at each location on planned comparisons (see Table 4.5). Interestingly, standard P3 latency in the OA group was shorter compared to YA, whereas the opposite was found for novel stimuli, indicating that the processing of standard stimuli was fastest in OA, and the processing of novel stimuli was fastest in YA.

Figure 4.1. Grand average waveforms for target and novel stimuli at frontal and parietal regions in younger and older adults.

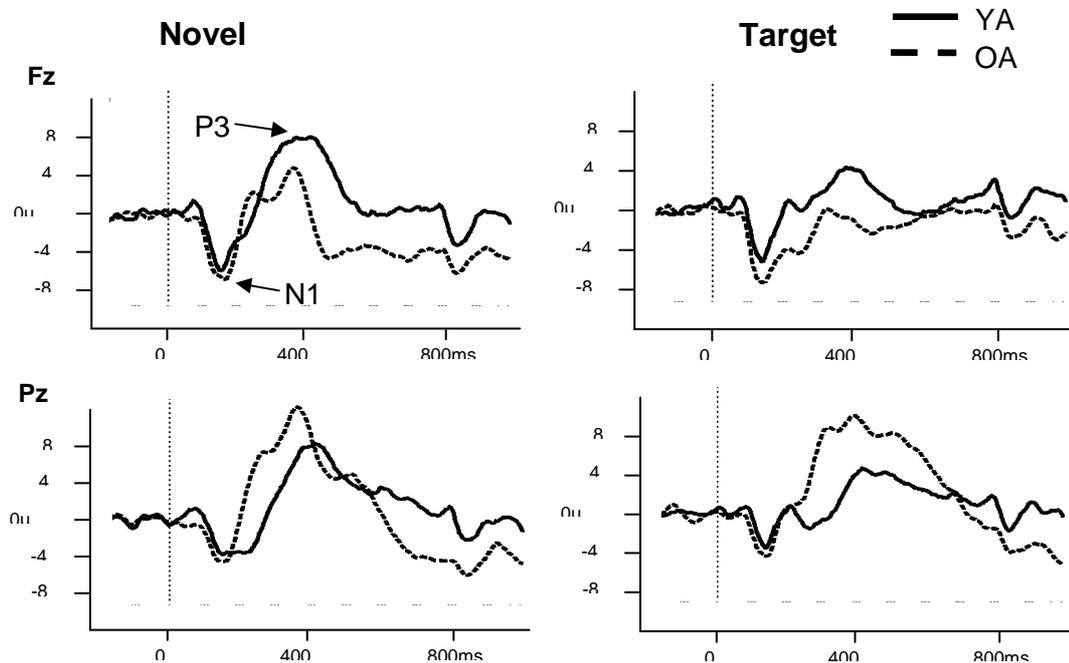


Table 4.5. Mean (*SD*) peak amplitudes and latencies of the P3 component for younger and older adults.

	<u>Younger</u>	<u>Older</u>	<u>Younger</u>	<u>Older</u>
	Amplitude ( $\mu$ V)		Latency (ms)	
Fz Novel	7.2 (4.3)	10.3 (5.0)	318.3 (59.9)	387.9 (63.7) †
Cz Novel	13.1 (5.7)	10.5 (5.7)	295.1 (61.6)	395.6 (53.5) †
Pz Novel	13.1 (5.3)	10.4 (3.7)	366.5 (19.0)	410.9 (52.7) †
Fz Target	3.1 (3.5)	6.3 (4.2) †	362.2 (69.1)	377.9 (78.4)
Cz Target	8.3 (5.1)	4.9 (6.0)	370.0 (85.0)	413.7 (68.4)
Pz Target	12.1 (5.3)	6.6 (6.1) †	393.4 (75.0)	412.6 (69.0)
Fz Standard	0.0 (1.0)	2.4 (1.7) †	379.7 (89.4)	270.3 (74.0) †
Cz Standard	-0.4 (1.1)	2.6 (1.6) †	362.6 (100.1)	281.4 (80.1) †
Pz Standard	-0.6 (0.9)	1.2 (1.2) †	347.5 (97.8)	273.6 (74.7) †

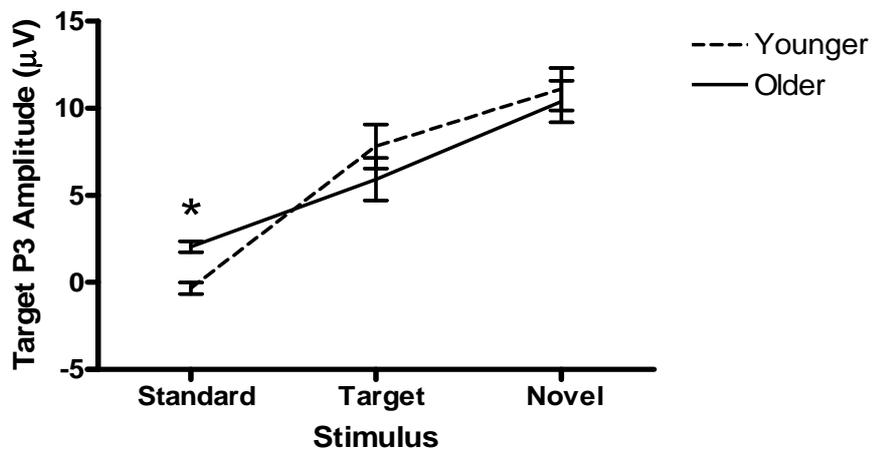
Note. †  $p < .05$  (Post hoc Independent Samples  $t$  tests).

Table 4.6. Results from the ANOVA model for the P3 amplitude.

		df	F	p
Main Effects	<b>Stimulus</b>	2, 50	72.89	< .001
	<b>Location</b>	2, 50	11.26	< .001
	Group	1, 25	0	.950
Interactions	<b>Stimulus X Location</b>	2, 50	17.78	< .001
	<b>Stimulus X Group</b>	2, 50	3.56	.036
	<b>Location X Group</b>	4, 100	14.71	< .001
	<b>Stimulus X Location X Group</b>	4, 100	10.77	< .001

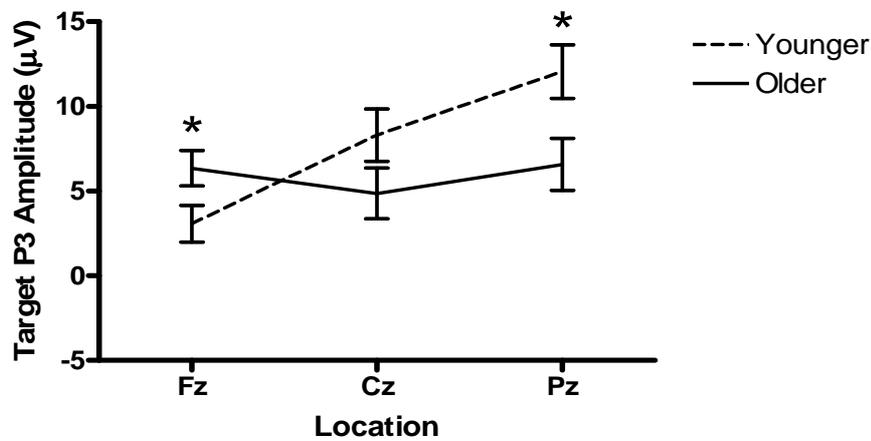
*Note.* Significant effects in **bold** type; Stimulus = standard, target, or novel stimuli; Location = Fz, Cz, or Pz; Group = Older or younger adults.

Figure 4.2. Target P3 Amplitude: Stimulus by Group Differences.



*Note.* Bars = Standard error; \* =  $p < .05$ .

Figure 4.3. Target P3 Amplitude: Location by Group Differences.



Note. Bars = Standard error; \* =  $p < .05$ .

#### 4.7. Results from the ANOVA model for the P3 latency.

		df	<i>F</i>	<i>p</i>
Main Effects	<b>Stimulus</b>	2, 50	7.92	< <b>.001</b>
	Location	2, 50	3.13	.053
	Group	1, 25	0.05	.820
Interactions	<b>Stimulus X Location</b>	2, 50	4.96	<b>.001</b>
	<b>Stimulus X Group</b>	2, 50	11.01	< <b>.001</b>
	Location X Group	4, 100	2.10	.133
	Stimulus X Location X Group	4, 100	1.32	.267

Note. Significant effects in **bold** type; Stimulus = standard, target, or novel stimuli; Location = Fz, Cz, or Pz; Group = Older or younger adults.

#### 4.6.3. Habituation to Novel Stimuli.

The amplitude of the P3 in relation to time of novel stimuli presentation can be found in Table 4.8. As shown in table 4.9, there was a main effect of location, and an interaction between location and group for the P3 amplitude. Together, these findings indicate that,

consistent with the stimulus comparison data presented above, Pz was the location of maximal P3 amplitude in YA, whereas OA had increased frontal (Fz) orientation, irrespective of time spent on task. The interaction was driven largely by a main effect of group at Fz,  $F(1, 25) = 6.92, p = .014$ .

Of particular interest was a significant interaction between group and time,  $F(1, 25) = 8.02, p = .009$ . As revealed in Figure 4.4, this interaction represents a decrease in novelty P3 amplitude with time in the YAs, and an increase in OAs. Planned comparisons revealed group differences at the third and fourth presentation of the novel stimuli at Fz, and at Pz for the first presentation (Table 4.8). Thus, OAs had significantly increased P3 amplitudes for the third and fourth presentations of novel stimuli at Fz compared to YA.

Table 4.8. Mean (*SD*) peak amplitudes of the P3 component to novel stimuli presentations for younger and older adults.

	<u>Younger</u>	<u>Older</u>
	Amplitude ( $\mu$ V)	
Fz Novel 1	9.6 (5.0)	9.9 (5.3)
Fz Novel 2	8.6 (5.3)	10.7 (5.2)
Fz Novel 3	6.4 (5.9)	13.4 (7.9) †
Fz Novel 4	6.6 (7.4)	12.2 (6.1) †
Pz Novel 1	15.1 (6.4)	10.5 (5.1) †
Pz Novel 2	13.6 (6.4)	9.5 (5.8)
Pz Novel 3	14.7 (7.9)	11.9 (5.7)
Pz Novel 4	14.1 (6.3)	13.1 (6.3)

*Note.* †  $p < .05$  (Planned comparison, Independent Samples *t* tests).

Table 4.9. Results of the ANOVA model for the habituation of the novelty P3.

		df	<i>F</i>	<i>p</i>
Main Effects	Time	3, 75	0.52	.668
	<b>Location</b>	1, 25	12.07	<b>.002</b>
	Group	1, 25	0.04	.851
Interactions	Time X Location	3, 75	0.79	.503
	<b>Time X Group</b>	3, 75	3.15	<b>.030</b>
	<b>Location X Group</b>	1, 25	14.50	<b>.001</b>
	Time X Location X Group	3, 75	0.90	.444

*Note.* Significant effects in **bold** type; Stimulus = standard, target, or novel stimuli; Location = Fz, Cz, or Pz; Group = Older or younger adults.

#### 4.6.4. Planned comparisons between level of anxiety and N1 and P3 components.

As shown in Table 4.10, there were no relationships between level of anxiety and the amplitudes of the N1 and P3 components elicited by target and novel stimuli in either group. These results indicate that anxiety did not modulate either initial attentional capture or later attentional processing of stimuli requiring a response or of unexpected novel stimuli.

Figure 4.4. Topographical maps of the effect of habituation on scalp distribution of electrical activity for younger and older adults.

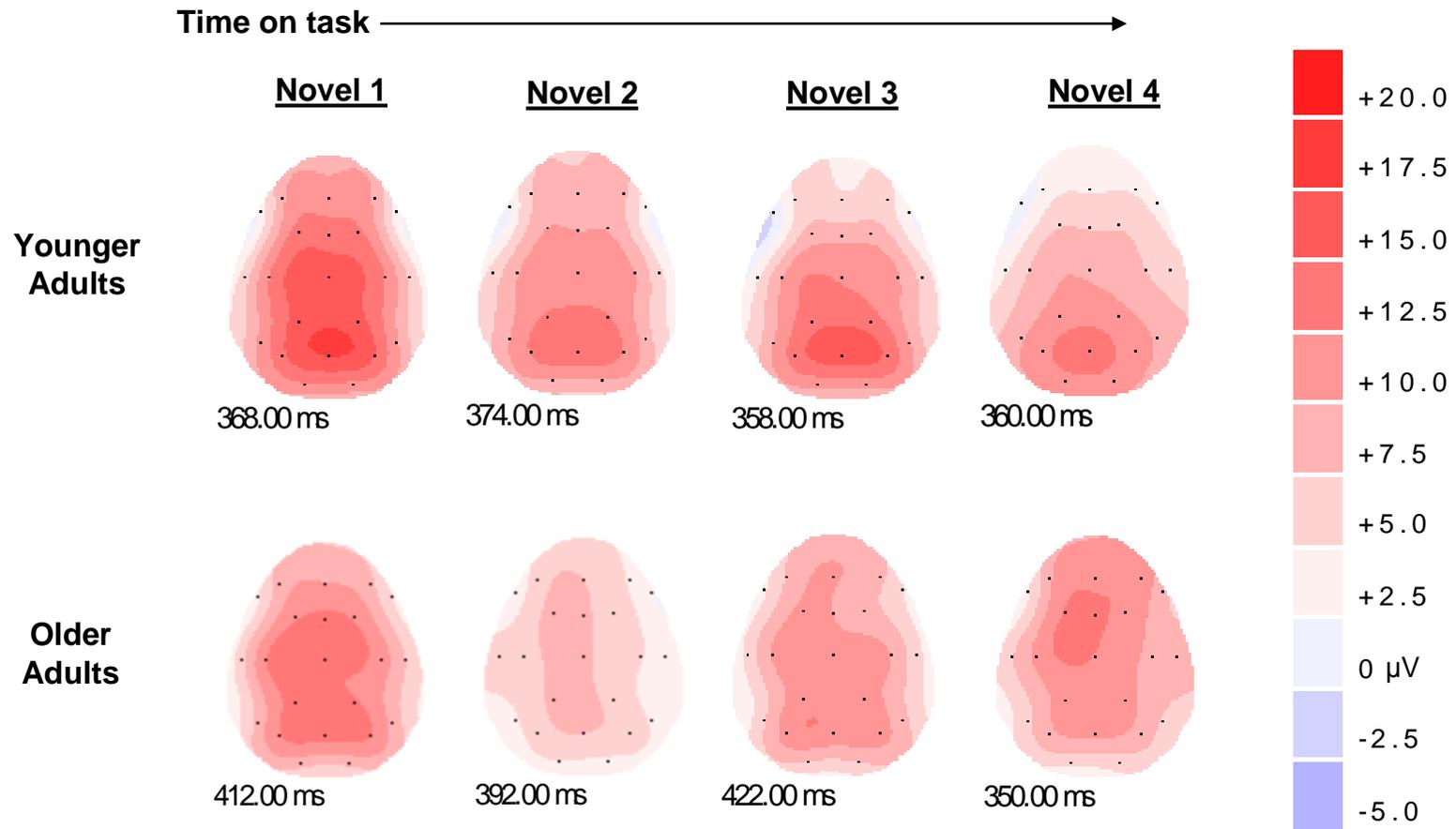


Table 4.10. Pearson product-moment correlations between anxiety and N1 and P3 amplitudes in younger and older adults.

		Target							Novel					
		Anxiety	N1			P3			N1			P3		
			Fz	Cz	Pz	Fz	Cz	Pz	Fz	Cz	Pz	Fz	Cz	Pz
Target	Anxiety													
	N1 Fz	.18 (.03)												
	N1 Cz	.13 (.02)	.81 (.66) †											
	N1 Pz	-.12 (.02)	.35 (.12)	.50 (.25)										
	P3 Fz	-.17 (.03)	.32 (.10)	.21 (.04)	-.20 (.04)									
	P3 Cz	.02 (.00)	.22 (.23)	.34 (.12)	-.08 (.00)	.70 (.49) †								
Novel	N1 Fz	.23 (.05)	.56 (.31) †	.46 (.21)	.51 (.26)	.30 (.09)	.34 (.12)	.44 (.19)						
	N1 Cz	.32 (.10)	.43 (.18)	.28 (.08)	-.10 (.01)	.13 (.02)	-.04 (.00)	-.25 (.06)	.35 (.12)					
	N1 Pz	-.11 (.01)	.12 (.02)	.13 (.02)	.23 (.05)	.05 (.00)	.11 (.01)	-.13 (.02)	.43 (.18)	.70 (.49) †				
	P3 Fz	.05 (.00)	-.04 (.00)	.23 (.05)	.28 (.08)	.30 (.09)	.37 (.14)	.22 (.05)	.53 (.28)	.30 (.09)	.55 (.30)			
	P3 Cz	.10 (.01)	-.04 (.00)	.28 (.08)	.25 (.06)	.18 (.03)	.63 (.40) †	.49 (.24)	.44 (.19)	.02 (.00)	.33 (.11)	.80 (.64) †		
	P3 Pz	.11 (.01)	.02 (.00)	.36 (.13)	.18 (.03)	.15 (.02)	.69 (.48) †	.81 (.66) †	.31 (.10)	-.39 (.15)	-.14 (.02)	.40 (.16)	.73 (.53) †	

Note. OA ( $n = 13$ ) data above the line; YA ( $n = 12$ ) data below the line; †  $p = .05$ ;  $R^2$  values in parentheses.

#### 4.7. Discussion.

The present study investigated Sensorimotor Awareness in ageing. It was hypothesised that a novelty auditory oddball paradigm would provide a means to explore the effects of ageing on Sensorimotor Awareness, the second level of awareness in the HoP model described by Stuss et al. (2001). As predicted, the amplitude of the N1 and P3 were greatest for novel stimuli irrespective of age, but only the P3 significantly distinguished novel from infrequent target tones. Additionally, and as predicted, longer N1 and P3 latencies were found for novel than for target and standard stimuli. Although this study did not find consistent amplitude attenuation and delayed latency in the older adult group, some subtle age-related effects were evident: central N1 amplitude was attenuated in the older adults, in particular for target stimuli; N1 latency to standard stimuli was shorter; standard P3 amplitude was increased; and, the latency was shorter in OA compared to YA. It was also hypothesised that OA would show more widespread distribution of electrical activity compared to YA, and this was partly confirmed by an increased frontal orientation of activity in relation to target stimuli, and in relation to novel stimuli in the novelty habituation analysis. Finally, this study tested the hypothesis that habituation to novelty would be altered by the normal ageing process, and this was indeed the case. Importantly, these results were not confounded by possible decline in hearing thresholds, level of anxiety, or by reduced intellectual function, evident in the comparable Raven's SPM scores. The implications of these findings are that the ageing brain is able to distinguish between stimuli, and may thus be considered to have basic sensory awareness compatible with intact Sensorimotor Awareness, but subtle differences, particularly in latency and topography, suggest that more widespread neural networks are required to facilitate this processing (e.g. Cabeza, 2002). This conclusion is now discussed in greater detail.

The behavioural performance of the older adults in the current study replicated earlier, consistent findings of equivalent target detection across age-groups (Amenedo & Diaz, 1998; Anderer et al., 1996; Fabiani & Friedman et al., 1995; Friedman et al., 1993; Polich, 1997; Weisz and Czigler, 2006). Previous studies have generally found that reaction times in oddball paradigms do not differ between age groups (Fabiani & Friedman et al., 1995; Friedman et al., 1993; Iragui et al., 1993; Polich, 1997), and this was also found in the present study. Critically,

this suggests that ERP latency differences occur in the absence of increased behavioural response times. One possibility is that as the response time for both groups typically exceeded 400ms and the N1-P3 complex amplitude maximums occurred prior to 400ms, there was time to compensate for any early sensory processing discrepancy between groups allowing normal initiation of the motor response.

The findings from the current study replicate and extend knowledge about attentional processing in younger and older adults. In line with previous studies (Amenedo & Diaz, 1998; Friedman et al., 1993; Iragui et al., 1993), the topographical distribution of the N1 was similar across groups, with maximal recordings at central regions for each stimulus type; this was despite the overall reduced N1 amplitude in older adults. In contrast to the previously reported finding that older adults have increased N1 amplitude to standard stimuli in standard oddball tasks (Amenedo & Diaz, 1998; Anderer et al., 1996), and a lack of age-related standard N1 amplitude differences in the novelty auditory oddball (Fabiani & Friedman, 1995; Friedman et al., 1993), the present study found that older adults had reduced N1 amplitudes to all stimuli. Increased, standard N1 amplitude in older adults in the standard oddball was interpreted to indicate attentional capture by irrelevant stimuli (Amenedo & Diaz, 1998; Anderer et al., 1996), and it was proposed that older adults may have a tendency to treat all stimuli as 'new' (Amenedo & Diaz, 1998; Friedman & Simpson, 1994). The lack of age-related differences in the standard N1 in the novelty paradigm (Fabiani & Friedman, 1995; Friedman et al., 1993) may indicate that both younger and older adults disregard standard stimuli to an equal extent, when presented in the context of additional novel stimuli that demand greater capture of attention. Thus, the differences between the present study and that of Amenedo and Diaz (1998) may be more methodological than real. However, the N1 for target and novel stimuli (note Amenedo and Diaz administered a standard oddball not a novelty oddball task) was not reported in any of these studies limiting any more meaningful comparison with the present study. It may simply be concluded that the overall reduction in N1 amplitude presented herein suggests that automatic attentional processing of all stimulus types in an auditory novelty oddball task is attenuated in older adults.

The latency of the standard N1 did not differ with age in previous novelty oddball studies (Fabiani & Friedman, 1995; Friedman et al., 1993), nor in the majority of the published standard oddball studies (Amenedo & Diaz, 1998; Iragui et al., 1993; Polich, 1997). However, Anderer et al. (1996) did find that standard N1 latency increased with age. Conversely, the present study found that standard N1 latency *decreased* with age, indicating a degree of faster attentional capture by what are essentially background stimuli in the older adults. This finding is compatible with an earlier proposition that older adults have an automatic selection bias for irrelevant standard stimuli (e.g. Amenedo & Diaz, 1998; Anderer et al., 1996).

The topographical distributions of the target and novelty P3 in younger and older adults in the current study replicates those already reported in the literature. Studies have consistently found that the target P3 is associated with a centro-parietal (Cz-Pz) maximal distribution in younger adults, whereas older adults have been shown to have an additional frontal orientation (Amenedo & Diaz, 1998; Fabiani & Friedman, 1995; Friedman et al., 1993; Iragui et al., 1993). The topography of the novelty P3 is more frontally orientated (Fz) in all age groups; however, this frontal distribution is greater in older adults (Fabiani & Friedman, 1995; Friedman et al., 1993). Another consistent finding in the literature is that the target P3 is both attenuated and delayed in older adults (Amenedo & Diaz, 1998; Chao & Knight, 1997b; Fabiani & Friedman, 1995; Friedman et al., 1993; Iragui et al., 1993; Polich, 1997; Weisz & Czigler, 2006). Although the current study did not find a main effect of group on P3 amplitude, post hoc analyses found that target P3 amplitude at parietal regions was attenuated in older adults supporting earlier studies. Results diverge, however, when it is noted that target P3 amplitude was, by contrast, significantly greater at frontal regions in older compared to younger adults. The greater parietal orientation of activity to target stimuli in younger adults has been interpreted to indicate the delegation of ‘attentional work’ to association cortices, whilst the greater frontal orientation found in older adults may be indicative of an impairment in the ability to construct and sustain mental representations of stimuli (Fabiani & Friedman, 1995); essentially, even target (recognisable) stimuli may have been treated as ‘novel’ by the older adults.

The current study did not find any age differences in the novelty P3 amplitude, contrary to the findings of Fabiani and Friedman (1995), but similarly to the findings of Weisz and Czigler (2006), indicating that immediate controlled attention to stimuli, as indexed by the novelty P3 (Friedman et al., 2001; Segalowitz & Davies, 2004), was equivalent across groups. The standard P3 is not typically reported in the literature, possibly because a specific higher cognitive function has not been assigned to this component. However, if it is assumed that even frequent, background stimuli may elicit some degree of attentional processing, then it is possible to conclude that older adults attend to standard stimuli to a greater extent than younger adults. The current study indeed found significantly increased P3 amplitudes to standard stimuli in older adults, suggesting that there is increased allocation of attentional resources to updating mental representations of such stimuli. In other words, older adults appear to be working harder just to process basic stimuli.

A significant delay of the target P3 in older adults has been consistently reported (Amenedo & Diaz, 1998; Fabiani & Friedman, 1995; Friedman et al., 1993; Iragui et al., 1993; Polich, 1997; Weisz & Czigler, 2006), indicating that the timing of controlled attention for the formation and maintenance of memory traces of targets is slower in older compared to younger adults. Contrary to previous studies, the current study found that the latency of the target P3 did not differ between older and younger groups, such that memory updating occurred within a similar time frame. The present study also found a strong trend for the timing of the novelty P3 to be faster than the target P3, and this is in line with previous studies (Fabiani & Friedman, 1995; Friedman et al., 1993). Moreover, this is consistent with interpretations of the functional significance of the novelty P3, namely immediate attention to the stimulus, but not memory updating per se (Friedman et al., 2001; Segalowitz & Davies, 2004). Of particular interest here, however, was the finding that older adults attended to standard stimuli more quickly than younger adults. This may be interpreted as further evidence of a selection bias for standard stimuli, and/or slower immediate controlled attention to novel stimuli compared to younger adults. Only two previous studies (Friedman et al., 1993; Weisz & Czigler, 2006) found that older adults gave significantly more false alarms to novel stimuli, interpreted as more impulsive responding and/or less inhibition. The current study did not find any age-related differences in false alarms to novel stimuli. Nevertheless, false alarms to

standard stimuli were increased in the older adults, a finding shared with Friedman et al. (1993). Alongside evidence already described that the older adults in the current sample might have a bias towards standard stimuli, this suggests a deficit in the inhibition of attention and responses to standard stimuli. The longer novelty P3 latency in older adults may also represent impaired disengagement of attention to irrelevant, novel stimuli (Weisz & Czigler, 2006). This finding is of importance, as novel stimuli can represent potential danger requiring an immediate response. One implication of this finding is that older adults are slower to process and respond to these novel types of stimuli, for example, a delay in processing a warning car horn may result in an accident.

In summary, the current and previous studies have found an attenuation of the target P3 in older adults, suggesting that the allocation of attentional processing, and updating of memory traces of target stimuli is reduced. Taken together, the equivalent target detection and reaction times of older adults, suggests that this attentional impairment did not have an impact on behavioural performance. An alternative interpretation is that reduced attentional capabilities in older adults do not negatively impact on performance, due to the simple requirements of the task, and the potential for older adults to compensate for longer processing latencies before the response is executed. A more difficult oddball paradigm with increased attentional load, for example, by including a fourth category of stimuli (e.g. stimuli with a similar tone to targets), may make the discrimination of targets harder, facilitating the exploration of possible effects of age-related attentional impairments on target detection, but this would also make greater demands on working memory and thus be less focussed on attentional processing per se. Perhaps more pertinent, it may not be assumed that these ERP findings reflect the quality of cognitive functioning in everyday life, and few studies have attempted to validate such claims by looking for associations between ERP components and, for example, behavioural indices of memory and awareness.

The current study also investigated habituation of the novelty P3 in younger and older adults and found, in line with previous studies (Cycowicz & Friedman, 1997; 1999; Friedman et al., 1997; Friedman & Simpson, 1994; Weisz & Czigler, 2006), that younger adults had a reduction in novelty P3 amplitude at frontal regions with repeated exposure to novel stimuli.

Novelty P3 amplitude at parietal regions did not differ with time in younger adults, in line with Cycowicz and Friedman (1997; 1999). Older adults showed a different pattern of activity over time. Specifically, there was no evidence of habituation of the novelty P3, rather an increase in frontal orientation was found with increasing time spent on task. Although there are already consistent findings for a lack of habituation to novelty in older adults (Friedman et al., 1997; Friedman & Simpson, 1994; Weisz & Czigler, 2006), the present study also makes some new observations. For example, the increase in frontal activity with time contrasts with the report of Weisz and Czigler (2006), who found that novelty P3 amplitude in older adults did not significantly change with time at any location. The differentiation of activity at frontal and parietal regions in the younger adults is compatible with the proposition that the novelty P3 is composed of two aspects: a frontal aspect representing initial orienting to novelty, and a parietal aspect associated with the categorisation of stimuli (Cycowicz & Friedman, 1999). Indeed, Courchesne et al. (1975) suggested that the shift from frontal to parietal orientation with repeated novel stimuli was influenced by recognition and learning. The results of the present study therefore suggest that younger adults rapidly habituate to novel stimuli, providing evidence of learning in this group. The contrasting increase in frontal novelty P3 amplitude and orientation in older adults, together with the findings of their selective bias for attending to frequent stimuli, suggest instead that there is a failure to inhibit the processing of all types of irrelevant stimuli, and that each stimulus is treated more as 'new' than as 'familiar' (cf. Amenedo & Diaz, 1998). Thus, this may reduce older adults' capacity for habituation and learning to which Courchesne and colleagues referred. Importantly, the increase in frontal activity to novel stimuli in older adults is also unlikely to be explained by an increase in their startle response, as there were equivalent, low rates of false alarm responding to novel stimuli across groups (YA = 4.3%; OA = 6.0%). As novel stimuli may also represent a threat, the perception and awareness of such stimuli may be influenced by emotional factors, such as anxiety. However, there were no significant associations between anxiety, as measured by the HADS, and the N1 and the P3 amplitude in either group. While this suggests that the current findings are unlikely to be due to an exaggerated startle response in older adults, it would be of considerable interest to investigate this possibility more methodologically, perhaps using other autonomic nervous system measures of startle such as eye-blink.

In conclusion, the lack of consistent amplitude reductions and delayed latencies in the older adults, suggests that there is not necessarily a global reduction in underlying EEG power. The neural generators of the N1 have been located in the superior temporal plane (Halgren et al., 1995), with dipole localisation identifying lateral and tangential signal orientations (Senkowski et al., 2003). The similar central N1 topography and lack of group effects for the lateral N1 suggest that the neural generator of the N1 may not be altered as a function of age. The reduction in amplitude at fronto-central regions may be due to a reduction in the propagation of this signal, for example, by myelin degeneration (Bartzokis, 2004); cell shrinkage (Raz, 2004); and/or increases in cerebrospinal spaces (Good et al., 2001; Raz, 2000). The lack of response time differences (reflecting processing speed) argues against any functional significance of the former hypothesis. However, signal propagation differences may also account for the different amplitude and latency differences found for the P3 components, particularly as the neural generators of the P3 components are more widespread. Such conclusions are currently limited by the lack of structural and functional imaging data for the older adults in this study.

Frontal orientation of target P3 amplitude in older adults has been interpreted to represent reduced frontal lobe functioning (Fabiani & Friedman, 1995; Fabiani et al., 1998); on the basis that an *efficient* frontal lobe would permit fast habituation associated with a shift in the management of simple target detection to the association (parietal) cortex. The increase in frontal activity in older adults has been hypothesised to represent the need for continued updating of mental representations of target stimuli, due to greater working memory decay and/or impairment in the maintenance of these memory traces (Fabiani & Friedman, 1995). Indeed, previous studies have found relationships between age-related deficits in tests of frontal lobe functioning and reduced P3 amplitudes (Daffner et al., 2006; Fabiani & Friedman, 1995; Fabiani et al., 1998), supporting the hypothesis that the frontal orientation of the target P3 indexes diminished frontal lobe functioning. Although the current study did not find any age-related differences on the Ravens' Matrices, a test of abstract reasoning and problem solving which are, arguably, frontal lobe functions, the study did find evidence of subtle distractability/non-selectivity (response to standard stimuli). Such a relationship may be

further elucidated by the inclusion of a more rigorous frontal-lobe targeted neuropsychological test battery.

#### 4.7.1. Summary.

Sensorimotor Awareness is at the second level of the Stuss et al. (2001) HoP model, and is proposed to result from the processing of sensory information in the posterior regions of the brain. The current study found differential effects of age on Sensorimotor Awareness; there were no consistent reductions in amplitude or delays in latencies of the N1 and P3 components. Initial attentional capture (indexed by the early N1 component) by all types of stimuli was reduced in older adults, with evidence found of a selective bias for standard stimuli, and topographical data suggested deficits in the maintenance of memory for targets. The reduction of frontal lobe habituation in the older adults also suggested that their ability for dynamic learning was altered. Later, controlled top-down processes may compensate for earlier (e.g. N1) deficits in sensory perception (e.g. Alain, McDonald, Ostroff & Schneider, 2004), and the increase in the frontal orientation of target and novel P3 found in the older adults provided support for this proposition, particularly as there were no behavioural differences in target detection or reaction times.

## Chapter 5. Performance Monitoring.

The ability to monitor behaviour, recognise errors and take remedial actions when necessary is fundamental to effective and adaptive cognitive and behavioural performance. These skills are described as components of performance monitoring (e.g. Rabbitt, 1966). A fundamental assumption of this chapter is that performance-monitoring requires the ability to hold information on-line (in working memory) about the rules for correct task performance, to focus and switch attention, initiate and co-ordinate physical responses to compatible and incompatible stimuli, and have awareness about the quality of performance in order to make moment-by-moment adjustments to behaviour. There are similarities between these functions and those proposed by Stuss et al. (2001) to comprise the third level of the HoP model, namely executive function; a function necessary for organisation of sensory information.

In earlier research into performance monitoring, error correction and post-error slowing provided behavioural measures of error detection and compensatory actions (Rabbitt, 1966). More recently, electrophysiological (ERP) correlates of performance monitoring have indicated the existence of an error monitoring mechanism in the brain. The discovery of an ERP component associated with the occurrence of an error has been particularly important to this field of study: the error related negativity (originally called the 'Ne': Falkenstein, Hohnsbein, Hoorman & Blanke, 1991, but more widely known as the ERN: Gehring, Coles, Meyer & Donchin, 1993). The ERN is a negative deflection that occurs after an erroneous response, and has been interpreted as a response-monitoring mechanism (Falkenstein et al., 1991; Gehring et al., 1993). Error processing involves the detection of errors through the comparison of the actual response with the on-line representation of the correct response; more specifically, the ERN reflects this comparison process (Falkenstein et al., 1991; Falkenstein, Hoormann, Christ & Hohnsbein, 2000; Gehring et al., 1993). The study presented in this chapter was designed to assess performance monitoring and its associated ERP and behavioural correlates in normal ageing.

## 5.1. Introduction.

### 5.1.1. The Error Related Negativity.

The ERN occurs at the same time or very soon after an erroneous response, and is maximal at fronto-central sites (Falkenstein, Hoorman, Christ & Hohnsbein, 2000). The onset of the ERN is concurrent with electromyographic (EMG) activity associated with the execution of an incorrect response (Coles, Smid, Scheffers & Otten, 1995). Source localisation and fMRI studies have indicated that an important neural generator of the ERN is the anterior cingulate cortex (ACC: Carter, Braver, Barch, Botvinivk, Noll & Cohen, 1998; Gehring & Knight, 2000; Herrman, Rommler, Ehliis, Heidrich & Fallgater, 2004; Yeung, Botvinivck & Cohen, 2004). However, patient lesion studies have also implicated the lateral PFC in the generation of the ERN signal (Gehring & Knight, 2000; Ullsperger, von Cramon & Muller, 2002). More recently, evidence from patients with frontal lobe white matter damage, but with intact ACC and lateral PFC grey matter, suggested that the combined functioning of these two regions is important to the generation and propagation of the ERN (Hogan, Vargha-Khadem, Saunders, Kirkham & Baldeweg, 2006).

The ERN is typically elicited in speeded choice response tasks, but may represent a neural error detection signal that is similar across a wide range of paradigms (Gehring et al., 1993). There has been much debate about the functional significance of the ERN. Early opinion sought to categorise published evidence into that supporting an ‘error-detection’ interpretation, and that supporting a ‘conflict-monitoring’ view. This division appears to have outlived its usefulness, as more recent studies have investigated how functions associated with each model may be examined within one paradigm (e.g. Hogan et al., 2006). Nevertheless, it is important briefly to consider the theoretical assumptions underlying each approach. The error-detection model states that the ERN represents a monitoring process: if there is a mismatch between the actual and the intended correct response then an error signal will be produced (Coles, Scheffers & Holroyd, 2001; Falkenstein et al., 1991; Falkenstein et al., 2000; Gehring et al., 1993; Scheffers & Coles, 2000; Scheffers, Coles, Bernstein, Gehring & Donchin, 1996). The conflict-monitoring model states that the ERN is a reflection of increased stimulus-response

conflict (Botvinick, Braver, Barch, Carter & Cohen, 2001; Carter et al., 1998). There is, however, little evidence to support the proposition that the ERN is differentially sensitive to conflict (however, see Gehring & Fencsik, 2001). Such dismissal of the conflict-monitoring interpretation of the ERN relies on the interpretation of the *timing* of conflict processing in a stimulus-response task. In other words, monitoring of response conflict may be expected to occur on seeing the incompatible stimuli (e.g. the word blue in red ink in a Stroop-like task) rather than only at the post-response (ERN) stage. Thus, ERP components associated with conflict-processing may be found across the stimulus as well as response-locked waveforms. Indeed, a stimulus-locked component termed the N2 has been shown to be sensitive to the degree of pre-response conflict (Kopp, Rist & Matler, 1996). The N2 was found to have early (N2b) and late (N2c) subcomponents, with the latter being associated with response priming<sup>1</sup> (Kopp et al., 1996). Interestingly, van Veen and Carter (2002) found that a single dipole model accounted for both the N2 and ERN, suggesting that conflict detection may actually underlie both negative components.

In summary, performance monitoring is likely to involve both response conflict and error detection processes. Therefore, it is important to investigate the response-locked ERN within the context of stimulus-locked components such as the N2 and N4, to provide a more comprehensive account of performance monitoring. Importantly for the present study, there is agreement that the ERN provides a measure of internal performance monitoring, and that some degree of awareness of executive function may be inferred by its presence; in order to adapt performance it is necessary to recognise that errors have occurred. In order to examine the relationship between performance monitoring and awareness in more detail, and following the lead of more recent ERN investigation, a combination of ERP assessment with self-report measures of performance was employed (e.g. Scheffers & Coles, 2000).

### 5.1.2. The Correct Response Negativity.

A negative deflection, similar to the ERN, occurring in the response-locked waveforms associated with correct response trials has been found in numerous studies. This negativity has

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<sup>1</sup> In the current study, following the nomenclature of Hogan et al. (2006), the terms N2 and N4 will be used.

been termed the correct-response negativity (CRN). A number of explanations have been offered to account for the CRN: as evidence for a response evaluation process (Vidal, Hasbroucq, Grapperon & Bonnet, 2000; Vidal, Burle, Bonnet, Grapperon & Hasbroucq, 2003); as an emotional process (Falkenstein et al., 2000; Vidal et al., 2000), influenced by personality and mood (Hajcak, McDonald & Simons, 2004; Luu, Collins & Tucker, 2000); and as a result of stimulus uncertainty (Pailing & Segalowitz, 2004). The CRN may also represent a small amount of conflict associated with incongruent stimuli when presented alongside congruent stimuli. For some authors, the magnitude of the difference between the CRN and the ERN is considered to be the most important measure, representing the “true Ne, which is dependent on response correctness” (Falkenstein, 2004, p. 7). The difference between the CRN and ERN indexes the functional integrity of the error detection system (Falkenstein, 2004).

### 5.1.3. The Error Positivity.

A third response-locked component has been linked to performance monitoring, namely a later, positive peak occurring after the ERN, termed the error positivity (Pe). The Pe is maximal at centroparietal sites and peaks ~300ms after an error response (Falkenstein et al., 2000; Nieuwenhuis, Ridderinkhof, Blom, Band & Kok, 2001). Some authors have investigated the possibility that the Pe is a reflection of ongoing (stimulus-locked) P3 activity (Falkenstein et al., 2000; Davies, Segalowitz, Dywan & Pailing, 2001). For example, Davies et al. (2001) suggested that “the Pe is a P3 response to the internal detection of errors” (p. 191), due to the timing and morphology of the waveforms. However, Luu et al. (2000) found that the topographical distribution of the Pe differed slightly from the P3, indicating separate components, and Falkenstein et al. (2000) found that the Pe and P3 were not similar components as they varied within participants. Specifically, in some participants a Pe was absent, whilst a clear P3 was present. In addition, the Pe did not differ between stimulus modalities, whereas the P3 was larger after visual stimuli.

More importantly for the study of awareness, the Pe has been suggested to represent conscious awareness of an error (Nieuwenhuis et al., 2001; Vidal et al., 2000). This hypothesis is contentious. For example, Falkenstein et al. (2000), arguing strongly against this interpretation, have suggested instead that the Pe reflects later error-processing activity that is independent of the processing linked to the ERN. In support, Herrmann et al. (2004) reported different generators for each component, concluding that they represented different features of error processing. In a review of the functional significance of the Pe, Overbeek, Nieuwenhuis and Ridderinkhof (2005) outlined three hypotheses: the affective-processing hypothesis; the behaviour-adaptation hypothesis; and the error-awareness hypothesis. The affective-processing hypothesis posited that error monitoring included an emotional component, evidenced by modulations of the ERN and Pe with negative affect (e.g. Hajcak et al., 2004). The behaviour-adaptation hypothesis stated that remedial actions were dependent on the amplitude of the Pe (Nieuwenhuis et al., 2001), whilst the error-awareness hypothesis posited that the Pe was modulated by awareness that an error has occurred (Nieuwenhuis et al., 2001). With regard to the behaviour-adaptation hypothesis, there is inconsistent evidence for a relationship between the ERN/Pe and compensatory behaviours (e.g. Falkenstein et al., 2000; 2001; Gehring et al., 1993; Gehring & Fencsik, 2001); although Nieuwenhuis et al. (2001) found that longer post-error slowing was associated with increased Pe amplitudes, this result was interpreted as evidence for the error-awareness hypothesis.

#### 5.1.4. Experimental Manipulations of Response Monitoring Components.

Numerous experimental paradigms have elicited the ERN: Stroop, Flanker, choice reaction-time tasks, Go/No Go, picture-name matching, source memory, antisaccade and mental rotation (reviewed in Overbeek et al., 2005; see also Falkenstein et al., 2000), indicating that the ERN may represent a generic feature of internal error detection.

In one of the original studies, Gehring et al. (1993) employed a flanker task with three conditions emphasising speed or accuracy or both, with financial rewards and losses for speed and errors, respectively. The sample was composed of 6 students aged 18-26 years. ERN

amplitude was greatest for the accuracy-emphasised condition, indicating that the ERN may be modulated by motivational factors, such as performance accuracy and/or financial gain. Interestingly, the greater the amplitude of the ERN, the greater the likelihood the error would be corrected. Larger ERN amplitudes were also associated with post-error slowing (response slowing on the next trial). This suggests an intriguing relationship between error detection and compensatory actions. Similar to Gehring et al. (1993), Hajcak, Moser, Yeung and Simons (2005) also found that the ERN amplitude was associated with motivational significance; the amplitude of the ERN was greater when the monetary value of errors was high and also when participants believed that their performance was being evaluated.

There has also been a focus on different types of errors, e.g. those that are committed deliberately compared to those that result from true behavioural 'slips'. Stemmer, Witzke and Schönle (2001) found that deliberate errors did not elicit an ERN or Pe, indicating that the ERN is an internal measure of an unintended error response, or a momentary lapse in attention. In support, Gehring and Fencsik (2001), using a paradigm with hand/foot responses, found that ERN amplitudes were greater when the error and correct responses were similar, for example, using the incorrect hand when a hand response was required. In addition, whilst post-error slowing was found in the analysis of reaction time (RT) data, this was not related to ERN amplitude. This is in contrast to the results of Gehring's earlier study (Gehring et al., 1993), but the authors suggest that contradictory results were due to the use of a hand-foot manipulation in the later study.

An important caveat was provided by Herrmann et al. (2004) who found that ERN amplitudes were reduced with a greater number of errors. This suggests that as the individual experiences greater ambiguity about the accuracy of their performance (reflected in an increased error response) the ERN decreases. The ERN may be dependent on the individual being aware of task rules, and if that level of awareness declines, either through poor understanding of the task initially, poor working memory, and/or lapses in attention, then the magnitude of the ERN will decrease, but that this decrease may not necessarily be due to a failure of the error-detection system per se. An alternative, but not inconsistent interpretation could be that the ERN is modulated by habituation to making errors, in other words, the individual may become

resigned to their poor performance. However, there may also be a methodological explanation for the Herrmann et al. (2004) finding that is equally valid. ERP convention dictates that single error trials are averaged together to create an error waveform, but variability in the latency of the ERN in each single trial can reduce the amplitude of the ERN in the averaged waveform (termed ‘latency jitter’ in the literature: Picton et al., 2000). For example, consider four individual ERN components occurring at 50, 100, 150 and 200ms in one person, and another four individual ERN components occurring at 47, 48, 49 and 50ms in a second person. When averaged together, the grand average waveform in the first person will appear to be of longer latency and have lower amplitude than the grand average waveform in the second person, which will look more like a ‘sharp peak’. Greater ERN latency variability in the single trials, perhaps due to increased numbers of error trials, may have led to the reduction in the ERN amplitude reported by Herrmann et al. (2004).

#### 5.1.5. Associations between ERN/Pe, Remedial Actions and Measures of Awareness.

By accepting that the ERN represents an internal signal of error detection, it is possible to consider a relationship with awareness of online performance. Postdiction of performance<sup>2</sup> has been employed as an index of working memory. It may also reflect the integrity of the individual’s awareness, as a degree of awareness about performance is necessary to facilitate the individual’s ability to comment on their performance when asked to do so (see Section 2.6.1, Chapter 2). Scheffers and Coles (2000) found that greater ERN amplitudes were related to greater perceived inaccuracy in a sample of 15 adults. On a trial-by-trial basis, participants were required to rate their performance on a five-point scale ranging from ‘sure correct’ to ‘sure incorrect’. Although these authors did not calculate discrepancy scores between perceived and actual performance, which is typical in the psychology postdiction literature, they did find that correct trials were associated with greater perceived accuracy (‘sure correct’), indicating that the sample had “awareness of the inaccuracy of behaviour” (Scheffers & Coles, 2000, p. 149). This is one of the few studies to make explicit a link between ERN amplitude and awareness of performance. Another study suggested that the Pe,

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<sup>2</sup> Postdiction is the individual’s assessment of their performance *after* they have performed the task.

rather than the ERN, was more related to awareness. Nieuwenhuis et al. (2001) investigated awareness in a sample of 15 students aged 18-23 years and found that ERN amplitude did not differ with degree of error awareness in an antisaccade task. This task required participants to direct their gaze to the opposite direction to a cue. On completion of each trial, subjective ratings of error performance were made. In contrast to the lack of a relationship between ERN amplitude and awareness, Pe amplitude was greater for recognised errors. Moreover, contrary to the report of Gehring et al. (1993), post error slowing was found only for perceived errors. Post-error slowing may reflect a conscious decision to slow down in order to prevent the occurrence of further errors; the individual may purposefully attempt to improve performance on subsequent trials. It follows that if an error is not perceived then the individual may not know to slow down. This, however, remains speculative. All that may currently be concluded is that there is likely to be a complex relationship between the ERN and compensatory behaviour, particularly as there were no ERN amplitude differences between perceived and unperceived errors (Nieuwenhuis et al., 2001). Error correction also provides an indirect measure of error awareness, but this occurs very quickly after the error (typically <800ms) and may reflect concomitant activation of the correct response hand at the same time as the incorrect response hand, rather than a true correction of an error<sup>3</sup>. Indeed, Falkenstein et al. (2000) investigated individual ERN and Pe amplitudes for errors that were subsequently corrected and those that were not. They found similar ERN and Pe amplitudes for corrected and uncorrected errors. This finding suggests that error correction may not solely be dependent on either error detection or error processing as indexed by the ERN and Pe.

Whether or not an error is compensated for in terms of immediate correction or post-error slowing, it has the potential to elicit an emotional response. Few studies, however, have considered a relationship between performance-monitoring ERPs and autonomic nervous system activity, which is also regulated by the ACC. Hajcak, McDonald and Simons (2003) found that ERN amplitude did not correlate with heart rate change, skin conductance response or post-error slowing. In contrast there were correlations between Pe amplitude and skin

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<sup>3</sup> De Jong, Coles, Logan and Gratton (1990) found that there was a point between the motor cortex and the hand before which the original incorrect response signal may be intercepted and changed (in which case the response will look like a normal correct one), however, if the corrective signal intercepts at a point after the threshold, then the incorrect response will be immediately followed by the correct response, but it is not an error correction per se.

conductance response ( $r = .55$ ), and post-error slowing ( $r = .48$ ). Based on these findings, the authors extended the proposition of Nieuwenhuis et al. (2001) by suggesting that the Pe represented error awareness. Specifically, they emphasised a “visceral” component to error detection (Hajcak et al., 2003, p. 901), but this was related more to compensatory actions than the ERN. Also of note, the ERN was reduced with greater error rates, predating the influential findings of Herrmann et al. (2004).

In summary, ERN amplitude has been found to be influenced by motivational factors, such as performance accuracy and monetary gain (Gehring et al., 1993; Hajcak et al., 2005).

Individual differences in affect in non-clinical samples have also been found to modulate ERN amplitudes (Hajcak et al., 2004; Luu, Collins & Tucker, 2000), with greater ERN and reduced Pe amplitudes in those classified as having high negative affect (Hajcak et al., 2004). ERN amplitudes can be influenced by salience (Bernstein et al., 1995; Falkenstein et al., 2000), error rate (Hajcak et al., 2003; Herrmann et al., 2004), response-conflict (Gehring & Fencsik, 2001), and greater perceived inaccuracy (Scheffers & Coles, 2000). The Pe has been linked to conscious recognition and awareness of an error (Nieuwenhuis et al., 2001), but not to the type of error (Falkenstein et al., 2000). Findings of relationships between the ERN and Pe and compensatory error-correction/post-error slowing are mixed, indicating that the processes involved in electrophysiological correlates of performance monitoring may not necessarily contribute to the processes underlying remedial actions.

#### 5.1.6. Performance Monitoring Components and Ageing.

There have been few studies investigating the effect of ageing on ERP correlates of performance monitoring. However, there is evidence in the metacognitive literature to indicate that older adults have comparable performance monitoring abilities to younger adults (see Section 2.6.1.3, Chapter 2). With regard to behavioural performance, Falkenstein et al. (2000) found in two studies comprised of 12 younger adults, aged 19-25 years, and 12 older adults, aged 55-65 years, that older adults had longer RTs and increased post error slowing but comparable error rates and error correction rates to younger adults. However, older adults had significantly reduced ERN and Pe amplitudes in comparison to younger adults in both a four-

choice response (4CR) and flanker task. Falkenstein et al. (2000) failed to find consistent amplitude reductions in stimulus-locked components (P3; the N2 and N4 were not measured), indicating that the ERN and Pe amplitude reductions were not simply reflective of a global power reduction in the underlying EEG. Moreover, as comparable error rates were found between the groups, the reduction in ERN and Pe amplitudes was unlikely to be due to increased error rates in older adults (cf. Herrmann et al., 2004). Similar findings were found by Mathewson, Dywan and Segalowitz (2005) in that ERN and Pe amplitudes elicited by both a flanker and source memory task were reduced in their older adults, and also by Band and Kok (2000) using a mental rotation task. The sample reported by Mathewson and colleagues consisted of 16 younger adults aged 19-26 years and 16 older adults aged 61-85 years. In support of Falkenstein et al. (2000), older adults were slower to respond in both tasks, but, conversely, the older adults had a greater number of errors in the flanker task and a greater number of false positives in the source memory task. Mathewson et al. (2005) also investigated the effect of RT variability on the amplitude of the ERN and Pe, and found that although older adults had significantly increased RT variability compared to younger adults, no relationships were found with the amplitude measures for either group. Moreover, ERN amplitude was also not related to error-rate in either task. However, after controlling for age, higher Pe amplitudes were associated with fewer errors. An alternative interpretation of this finding is that individuals with higher error rates may become habituated to making errors.

In a later study with 11 younger participants aged 19-25 years and 11 older adults aged 54-65 years, Falkenstein, Hoormann and Hohnsbein (2001) again found ERN amplitude reductions in older adults in 4CR and flanker tasks. However, no effect of age was found on CRN amplitude. To the author's knowledge this is the only published study that has conducted systematic, single-trial analyses on the younger and older adult data to investigate the possible effects of latency jitter. The single-trial analyses found that older adults had generally lower ERN amplitudes and longer latencies, and greater standard deviation (SD) of ERN amplitude and latency. These findings, nevertheless, further indicate that error detection is both attenuated and delayed in older adults. The significant contribution of the study by Falkenstein and colleagues is that ERN amplitude reduction found in older adults is not purely an artefact of the averaging process; although increased latency SD suggests some amplitude reduction by

single-trial latency jitter. The decreased amplitude SD indicates that the ERN amplitude reduction is not due to a greater absence of the ERN in some of the single trials recorded from older adults, but rather due to an actual amplitude reduction in the ERN in the older adults (Falkenstein et al., 2001).

Few studies have investigated performance monitoring in ageing and Alzheimer's disease (AD), although it is clearly of interest to see if performance-monitoring is affected by a disease associated with deficits in awareness (see Section 1.3.4, Chapter 1). Mathalon et al. (2003) conducted a study with 12 patients with AD (*M* Age 76.2, *SD* 5.7), 10 younger (*M* Age 21.2, *SD* 2) and 10 older adults (*M* Age = 75.3, *SD* 5.1). In contrast to Falkenstein et al. (2000; 2001), these authors found CRN and ERN, but not Pe, amplitude reductions in older adults compared to younger adults. However, it is important to note that Mathalon et al. (2003) employed a picture-name matching task, a paradigm rarely used to elicit the ERN. This task relies on language and semantic memory, whereas the ERN is typically elicited in speeded choice response paradigms that elicit 'slips' of action (Stemmer et al., 2001). It may be that errors associated with semantic retrieval deficits result in performance-monitoring components that differ in morphology to those elicited by other paradigms. Mathalon et al. (2003) also investigated stimulus-locked components associated with the processing of the stimulus prior to the occurrence of the response. They did not find any differences between younger and older adults in either N1 or P3 amplitudes; ERP latencies were not reported. In summary, the small number of published studies limits the conclusions that can be drawn about the functional significance of the ERN, CRN and Pe in ageing. There is, however, evidence that performance monitoring and error detection are attenuated with ageing.

#### 5.1.7. Aims of the Study.

The third level of the Stuss et al. (2001) Hierarchies of Processing (HoP) model, Consistent Consciousness, relies on executive functioning and its ability to organise sensory information underlying and supporting behaviour. Deficits at this level of awareness can result in an incoherent and unrealistic representation of the person and their world, which may in turn

impact on self-awareness at the highest level of the model (Stuss et al., 2001). The term executive functioning encompasses a broad range of cognitive functions, including attention, working memory, cognitive control and conflict processing. The performance of complex tasks depends on the interaction and organisation of such cognitive (executive) processes and incoming sensory information. There are consistent findings within the literature of subtle, but specific, executive function deficits in older adults (see Section 2.5, Chapter 2). More specifically, increased RTs and intra-individual variability in performance of cognitively demanding tasks are related to age (Mathewson et al., 2005; Salthouse, Nesselroade & Berish, 2006; Shammi, Bosman & Stuss, 1998). Little is known, however, about specific alterations in brain activity that potentially underlie such changes. The aim of the present study was to investigate performance monitoring in healthy ageing, as a means of exploring the integrity of Stuss et al.' (2001) third level of awareness in older adults. A secondary aim was to explore the possibility that brain function changes with ageing, perhaps in relation to underlying structural changes.

The performance monitoring ERP paradigm employed in this study (Hogan et al., 2005) has two equally probable conditions: compatible and incompatible stimulus-response. The incompatible condition manipulates task complexity/response conflict, henceforward 'task complexity'. The paradigm provides measures associated with performance monitoring at the level of stimulus-processing: pre-response conflict (N2); allocation of attention (P3b and SW<sup>4</sup>) and response-conflict priming (N4); response-monitoring: error detection (CRN-ERN); and, error processing (Pe). In addition, behaviour was investigated in terms of error rate and RT, and remedial actions: error correction and post-error slowing. The inclusion of postdiction report of performance accuracy provided a metacognitive measure of the individual's performance monitoring abilities.

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<sup>4</sup> The positive slow wave (SW) was described by Picton (1992) see p. 96, Chapter 4. However, a specific function has not been assigned to this component. Hogan et al. (2006) also measured two positive peaks (P3a and P3b), however different nomenclature was used in the current study, due to the debate over the functional significance of the P3a and P3b (see p. 96, Chapter 4).

The following hypotheses were tested:

1. Older adults have attenuated response-locked ERN and Pe amplitudes, and a reduced CRN-ERN difference compared to younger adults.
2. Older adults have attenuated and delayed stimulus-locked P3 and SW components.
3. Stimulus-locked N2 and N4 components have increased amplitude in the incompatible compared to compatible condition in both younger and older adults (reflecting conflict processing), but the magnitude of this increase is reduced in older adults.
4. The accuracy of postdiction of performance increases with task experience; however the magnitude of discrepancy between postdiction and actual performance is consistently greater in the older adults.
5. The relationship between ERN and Pe amplitudes and the number of errors, postdiction of performance, rate of error correction, post-error slowing and affect is different in older compared to younger participants.
6. ERN, CRN and Pe amplitudes significantly increase when the potential confound of latency-jitter is addressed, but the magnitude of this increase will be greater in older participants potentially due to greater pre-analysis latency-jitter.

## 5.2. Method.

### 5.2.1. Participants.

Twenty-nine older (OA: *M* Age 69.2 years, *SD* 6.8) and 18 younger (YA: *M* Age 22.3 years, *SD* 4.2) adults participated in this study. However, due to insufficient numbers of trials for the error waveforms (<5 trials), 7 OA and 2 YA were excluded from the analysis. ERN, CRN and

Pe amplitudes were the main variables of interest in the study, and exploratory analyses found that one older adult had >50% outliers and was therefore excluded; their performance was considered to be at chance-level. The final sample was composed of 21 OA (*M* Age 68.7 years, *SD* 6.3; 7 male and 14 female) and 18 YA (*M* Age 21.6 years, *SD* 3.7; 3 male and 15 female); this sample size exceeds that in earlier ERN studies of ageing (e.g. Falkenstein et al., 2000; Falkenstein et al., 2001; Mathalon et al., 2003; Mathewson et al., 2005).

### 5.2.2. Measures.

i) 4-Choice Response ERP Task (4-CRT; Hogan et al., 2005).

Stimuli were four horizontal arrows (17 cm length and 5 cm width) presented horizontally on a black background, with a duration of 150 ms and a stimulus-onset-asynchrony of 1500 ms. Participants were requested to cup the mouse in their palms and to use their thumbs to respond to the direction of the arrows. Green and red arrow stimuli pointing left and right were presented randomly with equal probability (green arrow .5 probability, red arrow .5 probability). At the presentation of a green arrow, participants were instructed to press the mouse button corresponding to the direction of the arrow (compatible condition). For red arrows, participants were instructed to press the opposite mouse button in response to the direction of the arrows (incompatible condition). Practice trials were not administered, however, careful explanation and checking of participants' understanding of the instructions and use of the mouse, suggested that all participants understood what was required. Speed and accuracy were equally emphasized, and participants were informed that they could correct their errors to prevent them asking for permission to do so during the actual task<sup>5</sup>. Four blocks of 100 stimuli were administered to the participants with a short break between each block. Two extra blocks were given, as necessary, to increase the error rate which was monitored by the examiner on-line<sup>6</sup>. On completion of each block, participants were requested to provide an

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<sup>5</sup> Piloting of the paradigm had revealed that participants would interrupt the task to ask whether they could correct errors if this was not explicitly addressed at the outset.

<sup>6</sup> Additional blocks were administered to some participants to increase the number of error trials available for ERP averaging. Behavioural error rate was calculated as a percentage to account for variation in number of blocks administered. However, the median number of blocks administered to younger ( $n = 4$ ) and older ( $n = 4$ ) groups was comparable ( $p > .1$ ).

estimated percentage of their perceived accuracy of performance.

ii) The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) (described in Chapter 3).

i) anxiety subscale

ii) depression subscale, with higher scores reflective of more negative affect.

iii) The Positive and Negative Affect Schedule (PANAS; Watson, Clark & Tellegen, 1988).

The PANAS is a 20 item self-report measure of positive and negative affect using a five point rating scale. The PANAS was used to assess the effect of testing and was administered pre and post ERP testing. Higher scores indicate higher levels of affect. Positive discrepancy scores indicate an increase in positive or negative affect, negative discrepancy scores indicate a decrease in positive or negative affect.

5.2.3. ERP Acquisition and Processing (see Section 3.7.4, Chapter 3 for detailed description).

This task was administered in the same session as the novelty auditory oddball paradigm (Chapter 4). In all cases, the 4-CRT was administered after the Novelty Auditory Oddball. Thus, the EEG recording was similar to that reported in Chapter 4. Briefly, the EEG was recorded using NeuroScan SynAmps<sup>TM</sup> amplifiers at a sampling rate of 500Hz (band-pass 0.05-70Hz) from 22 leads located over lateral and midline sites, using a ground lead situated at Fp1, and referenced to linked mastoids. Impedance was maintained at less than 15K $\Omega$ . Vertical (right eye) and lateral ocular electrodes enabled offline blink reduction according to a standard algorithm (Semlitsch et al., 1986).

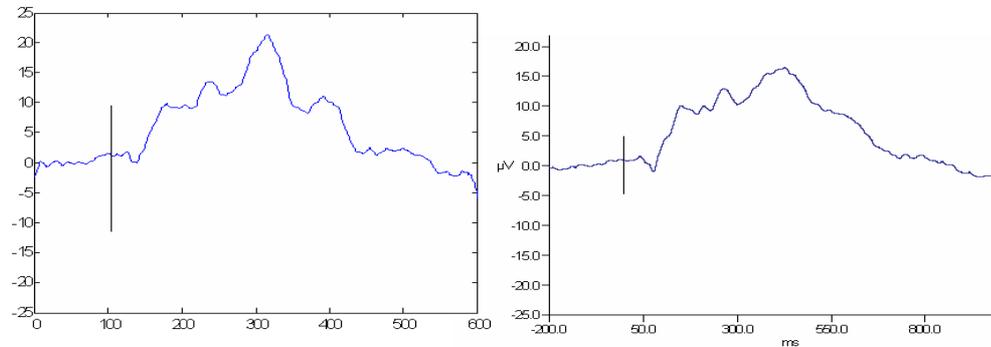
For stimulus-locked waveforms (N2, P3b, N4, SW), EEG data were epoched at -200 to 1000 ms centred on presentation of arrow stimuli, and baseline corrected at -200 to 0 ms. For response-locked waveforms, EEG data (CRN, ERN, Pe) were epoched at -500 to 500 ms, and baseline corrected at -200 to -100 ms. Epochs were averaged by stimulus type (green and red

arrows), correct trial status (correct green arrows, error green arrows, correct red arrows and error red arrows) and response type (green arrow error, red arrow error, green arrow correct and red arrow correct). The components were identified as the maximum peak within the specified time frames: N2 at Fz (190 – 310 ms: most negative), P3b at Pz (250 – 450 ms: most positive), N4 at Fz (380 - 520ms: most negative), SW (450 – 800 ms: most positive), and Pe (250 – 450 ms: most positive). The ERN and CRN components were calculated from the peak preceding the response (-100 to 0 ms) to the highest negative peak after the response (0 – 200 ms) to provide a measure of the magnitude of each component (a ‘peak-to-peak’ measure).

#### 5.2.3.1. Woody Filter.

The Woody Filter was applied to examine the potential confound of latency jitter (re: Hypothesis 6). Each single trial is composed of a number of data points which correspond with the sampling rate of the EEG recording. In the EEG studies reported herein, the sampling rate was 500Hz. Each data point is an amplitude measurement with the position of the data point providing the latency measurement. In the conventional averaging process the data points in each single trial are averaged to create the final waveform. One of the potential concerns with the conventional method is that the latency of the peaks of interest may vary considerably between trials. The averaging process results in a smaller and broader component and may not accurately represent that component as it is found in the single trials. Woody (1967) proposed an adaptive filter based on a cross-correlation process. The Woody filter averages the single trials by aligning the signals (peaks) found in the trials, rather than by a time-locked stimulus or response code. Each single trial provides a row of data points, with the columns providing the amplitude measurement at that time point. The Woody filter uses a template of the data points to which each trial is aligned; each trial is shifted until the waveforms are most highly correlated. A matrix is created containing the cross-correlations for each of the combinations of data in the columns. Once these data points are correlated a plot of the waveform is created (see Figure 5.1).

Figure 5.1. Stimulus-locked waveforms provided by an older adult averaged by the Woody filter process (left) and the conventional process (right).



*Note.* The X axis scale differs between the figures as the NeuroScan waveform (right) provides the timing of the epoch (1200 ms), whereas the Woody waveform (left) is created from the data points in the epoch (600). The horizontal line represents stimulus presentation in both waveforms.

#### 5.2.4. Statistical Analysis.

Behavioural responses made  $< 150$ ms after stimulus presentation were excluded from the analysis as they were unlikely to represent a 'true' response to the stimulus. The percentages of errors, errors corrected and corrects made incorrect, RT for errors and correct responses, post-error slowing and the number of missed trials was calculated for each participant. Post-error slowing was calculated by subtracting the mean RT for trials following an error from the mean RT for correct trials. The behavioural responses and ERP data were explored using the Shapiro-Wilk test of normality to explore their distribution (all  $p > .05$ ); this test is appropriate for sample sizes  $< 50$ . Three variables (compatible errors corrected, compatible corrects incorreced, and incompatible corrects incorreced) were not normally distributed and were therefore analysed using Mann Whitney U tests. The number of missed trials was compared between compatible and incompatible conditions using Independent Samples  $t$  tests.

ERP behavioural data were analysed with mixed-design ANOVA models: condition (x2: compatible, incompatible stimuli) and group (x2: younger, older). ERP components (amplitude and latency) were also analysed using ANOVA models: condition (x2: compatible,

incompatible) and group (x2: younger, older) for the N2, P3b, N4, SW and Pe. ANOVA models for amplitude and latency were conducted to assess the CRN-ERN simultaneously (to assess the magnitude of the CRN-ERN difference; cf. Falkenstein, 2004): condition (x2: compatible, incompatible), component (x2: CRN, ERN) and group (x2: younger, older). Planned t-tests were also conducted for each of these amplitude and latency measures. The Woody filtered, response-locked ERP data (CRN, ERN and Pe) were also analysed with ANOVA models as described previously. Mixed-design ANOVA models were employed to investigate the magnitude of the amplitude difference between conventional and Woody filter analyses: analysis (x2: Woody, conventional) and group (x2: younger, older).

Finally, planned, within-group correlations were conducted to assess relationships between the response-locked components and variables found in the literature to be related to the ERN and Pe amplitude: affect, compensatory actions and perceived accuracy. Bonferroni adjusted alpha values were not calculated, since a priori predictions between the variables had been specified. Discrepancy scores for postdiction accuracy were calculated by subtracting postdiction accuracy percentages from the percentage of correct responses for each block of 4-CRT trials. These values were then averaged to produce a mean discrepancy score expressed as a percentage. Speed-accuracy trade-off was also explored with correlations between error rate and correct and error RT.

### 5.3. Results.

#### 5.3.1. Behavioural Responses.

Mean scores and standard deviations of the behavioural measures are presented in Table 5.1, with the results of the ANOVA models presented in Table 5.2. The error rates were comparable between groups. The OA, however, responded significantly more slowly compared to the YA for both correct and error responses. The rates of error correction and post-error slowing were also equivalent, indicating that both groups undertook similar compensatory actions. However, the OA displayed additional within trial responding in both

conditions; e.g. altering correct responses to errors,  $U(18) = 101.0, p = .020$  and  $U(20) = 86.5, p = .005$ , compatible and incompatible conditions, respectively. The OA also missed out more trials than the YA,  $t(36) = 2.37, p = .023$ . With the exception of correct response RT, there were no significant main effects or interactions with condition, indicating that both groups managed the increase in task complexity equivalently. As shown in Table 5.1, RT to incompatible stimuli was longer compared to the RT to compatible stimuli, and the magnitude of this increase in RT was greater in OA compared to younger adults for correct trials (Table 6.2: condition X group interaction). Post-error slowing was also significantly reduced for red arrows, possibly due to the increased time taken correctly to respond to incompatible stimuli. There were no significant correlations between error rate and correct and error RTs for either group for either condition, suggesting that there was no evidence of a speed-accuracy trade-off (see Table 5.3).

In summary, the OA responded to fewer trials, and were generally slower at responding compared to YA. However, they had an equivalent error rate, and responded equivalently to an increase in task complexity in the majority of variables examined. However, a higher rate of correct trials ‘incorrected’ suggests a degree of uncertainty, indicated also by the higher number of missed trials. Remedial actions (correction of errors and post-error slowing) were compatible between groups. It may be assumed, therefore, that both age groups understood the task and were able to adjust to the complexity manipulation.

Table 5.1. Error rate, error RT, correct RT, percentage of errors corrected, percentage of correct responses made incorrect, post-error slowing and total number of missed trials in younger and older adults.

<i>M (SD)</i>	<u>Condition</u>	<u>Younger</u> <i>n</i> = 18	<u>Older</u> <i>n</i> = 20 <sup>a</sup>
Error (%)	Compatible	10.6 (7.0)	9.2 (8.3)
	Incompatible	12.8 (7.3)	8.9 (5.0)
Error RT (ms)	Compatible	445.6 (98.9)	550.6 (104.8) †
	Incompatible	428.5 (78.7)	570.7 (121.1) †
Correct RT (ms)	Compatible	483.8 (77.0)	637.9 (96.0) †

	Incompatible †	512.8 (71.8)	695.4 (110.7) †
Errors Corrected (%)	Compatible	27.6 (29.4)	39.6 (34.5)
	Incompatible	24.4 (33.3)	33.1 (31.9)
Corrects Incorrected (%)	Compatible	0.3 (0.7)	1.6 (2.4) †
	Incompatible	0.2 (0.5)	1.4 (1.6) †
Post-error slowing (ms)	Compatible	63.0 (46.6)	72.4 (84.0)
	Incompatible †	25.6 (54.1)	29.6 (81.0)
Missed trials (number)	Combined	2.5 (3.7)	12.3 (17.1) †

*Note.* <sup>a</sup> One OA behavioural data set was lost; †  $p < .05$ , from the planned  $t$ -test.

Table 5.2. ANOVA models simultaneously assessing the effect of task complexity (Condition) and age (Group) for behavioural data.

			df	$F$	$p$
Error %	Main Effects	Condition	1, 36	0.53	.473
		Group	1, 36	1.11	.298
	Interaction	Condition X Group	1, 36	2.04	.161
Error RT (ms)	Main Effects	Condition	1, 36	0.01	.931
		<b>Group</b>	1, 36	18.39	< .001
	Interaction	Condition X Group	1, 36	1.22	.277
Correct RT (ms)	Main Effects	<b>Condition</b>	1, 36	50.42	< .001
		<b>Group</b>	1, 36	33.87	< .001
	Interaction	<b>Condition X Group</b>	1, 36	5.46	.025
PES (ms)	Main Effects	<b>Condition</b>	1, 36	5.28	.028
		Group	1, 36	0.30	.587
	Interaction	Condition X Group	1, 36	0	.958

*Note.* PES = post-error slowing; Condition = compatible or incompatible stimuli; Group = Older or Younger adults; significant effects in **bold** type.

Table 5.3. Pearson product-moment correlations investigating speed-accuracy trade-off in younger and older adults.

	G_Error %	R_Error %	G_Correct RT	R_Correct RT	G_Error RT	R_Error RT
G_Error %		.34 (.12)	.05 (.00)	-.27 (.07)	.20 (.04)	-.24 (.06)
R_Error %	.50 (.25)		.36 (.13)	.35 (.13)	.42 (.18)	.04 (.00)
G_Correct RT	.23 (.05)	-.19 (.04)		.92 (.85) †	.56 (.31) †	.46 (.21) †
R_Correct RT	.19 (.04)	-.19 (.04)	.92 (.85) †		.57 (.32) †	.54 (.29) †
G_Error RT	.34 (.12)	.08 (.01)	.46 (.21)	.41 (.17)		.61 (.37) †
R_Error RT	.43 (.18)	.17 (.03)	.71 (.50) †	.69 (.48) †	.31 (.10)	

*Note.* G = Compatible 4-CRT condition; R = Incompatible 4-CRT condition; RT = reaction times. OA ( $n = 20$ ) data above the line. YA ( $n = 17$ ) data below the line.  $R^2$  values in parentheses; †  $p < .05$ .

### 5.3.2. Stimulus-locked components.

The stimulus-locked waveforms can be viewed in Figure 5.2. Amplitude and latency means and standard deviations are presented in Table 5.4. The results from the ANOVA models are given in Table 5.5 and 5.6.

#### 5.3.2.1. P3b Component.

As shown in Tables 5.4, 5.5 and 5.6, there was a significant effect of group on the amplitude and the latency of the P3b, indicating that OA have attenuated and delayed P3b peaks for both compatible and incompatible conditions. Task complexity did not affect the P3b, indicating that the age effect was not influenced by the complexity manipulation (Table 5.5: lack of condition X group interactions for amplitude and latency)

#### 5.3.2.2. SW Component.

Only the latency of the SW component was delayed in OA (see Tables 5.4 and 5.6), consistent with the significant slowing of the immediately preceding P3b. The lack of condition effects, again, indicates that the later processing of compatible and incompatible stimuli was of equivalent power and timing.

Figure 5.2. Stimulus-locked waveforms recorded from Cz: Compatible condition (solid line) and incompatible condition (dashed line) group grand averages.

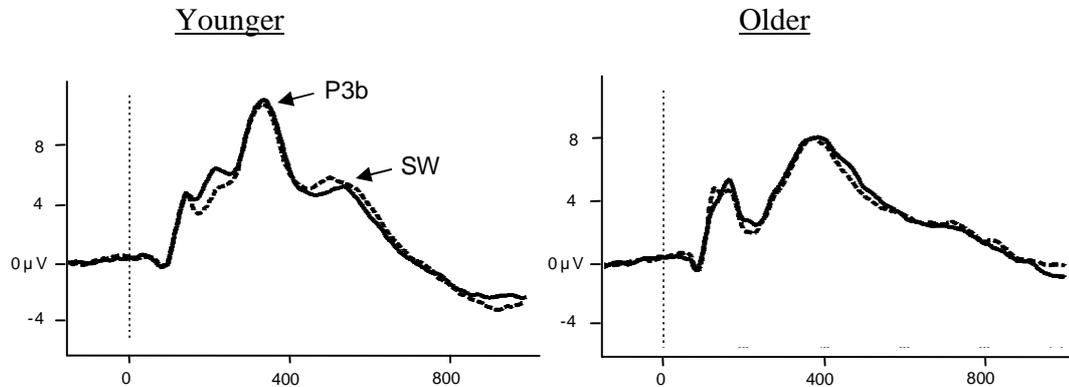


Table 5.4. Peak amplitudes and latencies of stimulus-locked components for younger and older adults.

		<u>Younger</u>	<u>Older</u>	<u>Younger</u>	<u>Older</u>
		<i>n</i> = 18	<i>n</i> = 21	<i>n</i> = 18	<i>n</i> = 21
<i>M</i> ( <i>SD</i> )		Amplitude ( $\mu$ V)		Latency (ms)	
P3b	Compatible	15.1 (5.1)	9.4 (6.2) †	322.2 (28.0)	366.4 (46.3) †
	Incompatible	15.5 (5.2)	9.1 (6.3) †	335.9 (40.7)	360.5 (54.1) †
SW	Compatible	5.6 (4.1)	4.6 (6.0)	541.3 (44.0)	666.2 (104.4) †
	Incompatible	6.7 (4.1)	4.2 (6.5)	529.8 (56.9)	676.3 (81.7) †

Note. P3b at Pz; SW at Cz; †  $p < .05$ , planned comparison using *t* test.

Table 5.5. ANOVA models assessing amplitude of the stimulus-locked components.

			df	<i>F</i>	<i>p</i>
P3b	Main Effects	Condition	1, 37	0.01	.946
		<b>Group</b>	1, 37	10.72	<b>.002</b>
	Interaction	Condition X Group	1, 37	1.03	.317

SW	Main Effects	Condition	1, 37	0.73	.399
		Group	1, 37	1.04	.315
	Interaction	Condition X Group	1, 37	3.56	.067

*Note.* Condition = compatible or incompatible stimuli; Group = Older and Younger adults; significant effects in bold.

Table 5.6. ANOVA models for the latencies of the stimulus-locked components.

			df	<i>F</i>	<i>p</i>
P3b	Main Effects	Condition	1, 37	0.33	.569
		<b>Group</b>	1, 37	7.70	<b>.009</b>
	Interaction	Condition X Group	1, 37	2.10	.156
SW	Main Effects	Condition	1, 37	0	.961
		<b>Group</b>	1, 37	46.28	<b>&lt; .001</b>
	Interaction	Condition X Group	1, 37	0.55	.465

*Note.* Condition = compatible or incompatible stimuli; Group = Older and Younger adults; significant effects in bold.

### 5.3.2.3 Correct Trial Analyses: Investigation of Pre-Response Conflict.

Amplitude and latency means and standard deviations are presented in Table 5.7 (see also Figure 5.3). The results from the ANOVA models are given in Tables 5.8 and 5.9.

#### 5.3.2.3.1. N2 Component.

There was a main effect of condition on the amplitude of the N2, indicating that the incompatible condition elicited larger negative amplitudes. This finding is consistent with the proposition that the N2 component represents pre-response conflict, as greater response conflict is produced by the incompatible stimulus-response condition. The lack of any group effect indicates that the power and timing of the pre-response conflict signal was equivalent across groups.

#### 5.3.2.3.2. P3b Component.

The P3b was attenuated and slower in the OA, regardless of condition. There were no effects of condition, indicating that this component was not sensitive to stimulus-response compatibility.

#### 5.3.2.3.3. N4 Component.

The N4 component was significantly reduced and delayed in OA. There were no effects of condition, indicating that this component was not sensitive to stimulus-response compatibility.

#### 5.3.2.3.4. SW Component.

The SW component was significantly delayed in the OA. The lack of condition effects indicates that this component was also not sensitive to stimulus-response compatibility.

Figure 5.3. Stimulus-locked waveforms recorded from Cz: Compatible condition (solid line) and incompatible condition (dashed line) group grand averages.

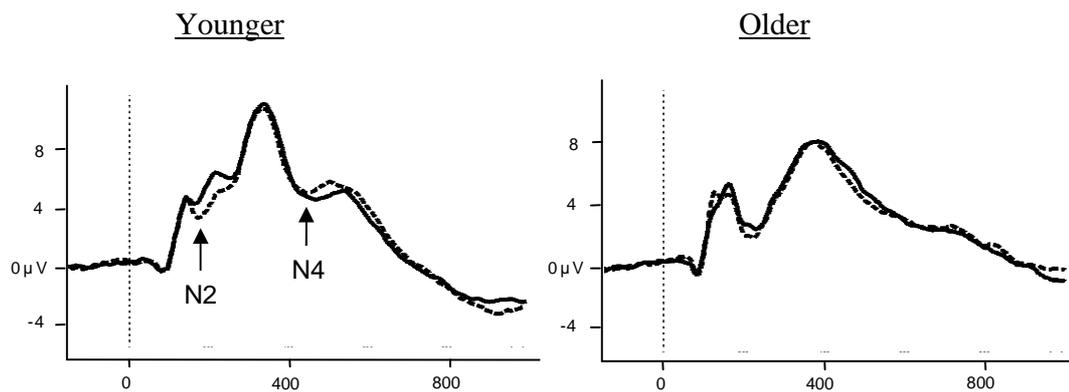


Table 5.7. Mean (standard deviation) peak amplitudes and latencies of correct trial stimulus-locked components for younger and older adults.

		<u>Younger</u>	<u>Older</u>	<u>Younger</u>	<u>Older</u>
		<i>n</i> = 18	<i>n</i> = 21	<i>n</i> = 18	<i>n</i> = 21
<i>M</i> ( <i>SD</i> )		Amplitude ( $\mu$ V)		Latency (ms)	
N2	Compatible	-0.9 (3.0)	0.2 (4.7)	226.2 (34.3)	228.8 (30.4)
	Incompatible †	-1.9 (3.3)	-0.0 (4.8)	230.8 (36.6)	233.1 (30.8)
P3b	Compatible	15.4 (5.1)	9.0 (6.2) †	324.8 (28.4)	365.9 (44.7) †
	Incompatible	15.3 (5.1)	8.8 (6.2) †	329.4 (45.6)	368.4 (45.6) †
N4	Compatible	-2.0 (4.6)	2.9 (4.8) †	456.3 (37.0)	503.0 (37.6) †
	Incompatible	-1.4 (4.2)	2.9 (5.0) †	456.1 (42.9)	503.5 (34.7) †
SW	Compatible	5.7 (4.3)	5.5 (7.1)	538.9 (51.9)	647.6 (96.5) †
	Incompatible	6.8 (4.1)	5.3 (7.3)	528.9 (56.5)	632.2 (92.2) †

Note. N2 and N4 at Fz; P3b at Pz; SW at Cz. †  $p < .05$ , planned comparison using  $t$  test.

Table 5.8. ANOVA models for amplitude of the correct trial stimulus-locked components.

			df	<i>F</i>	<i>p</i>
N2	Main Effects	<b>Condition</b>	1, 37	9.00	<b>.005</b>
		Group	1, 37	1.31	.259
	Interaction	Condition X Group	1, 37	3.49	.070
P3b	Main Effects	Condition	1, 37	0.13	.720
		<b>Group</b>	1, 37	12.50	<b>.001</b>
	Interaction	Condition X Group	1, 37	0.05	.822
N4	Main Effects	Condition	1, 37	0.92	.344
		<b>Group</b>	1, 37	9.64	<b>.004</b>
	Interaction	Condition X Group	1, 37	0.87	.357

SW	Main Effects	Condition	1, 37	0.56	.461
		Group	1, 37	0.21	.649
	Interaction	Condition X Group	1, 37	1.37	.249

*Note.* Condition = compatible or incompatible stimuli; Group = Older and Younger adults; significant effects in **bold** type.

Table 5.9. ANOVA models for the latency of the correct trial stimulus-locked components.

			df	F	p
N2	Main Effects	Condition	1, 37	2.74	.103
		<b>Group</b>	1, 37	0.06	.812
	Interaction	Condition X Group	1, 37	0	.974
P3b	Main Effects	Condition	1, 37	0.29	.591
		<b>Group</b>	1, 37	11.55	<b>.002</b>
	Interaction	Condition X Group	1, 37	0.03	.869
N4	Main Effects	Condition	1, 37	0	.980
		<b>Group</b>	1, 37	22.41	<b>&lt; .001</b>
	Interaction	Condition X Group	1, 37	0	.956
SW	Main Effects	Condition	1, 37	0.80	.377
		<b>Group</b>	1, 37	25.90	<b>&lt; .001</b>
	Interaction	Condition X Group	1, 37	0.04	.850

*Note.* Condition = compatible or incompatible stimuli; Group = Older and Younger adults; significant effects in **bold** type.

#### 5.3.2.3.5. Summary.

Only the N2 component was sensitive to stimulus-response compatibility. Consistent with previous studies, this component was more negative for incompatible stimuli. There was,

however, no interaction with group. Group effects (decreased amplitudes and increased latencies) were only present for the later P3b, N4 and SW components.

### 5.3.3. Response-locked components.

The response-locked waveforms can be viewed in Figure 5.4. Amplitude and latency means and standard deviations are presented in Table 5.10. The results from the ANOVA models are given in Tables 5.11 and 5.12.

#### 5.3.3.1. CRN-ERN Components.

As indicated in Figure 5.4, ERN amplitude was significantly greater than CRN amplitude across stimulus type and group, indicating that the error detection system was functional in both YA and OA (Table 5.11: main effect of component). However, a main effect of group (see Table 5.11) indicated that OA had significantly attenuated amplitudes across conditions and components. The results of planned  $t$  tests showed that the greatest amplitude differences between YA and OA were for the incompatible CRN and ERN (also supported by a trend towards an interaction between condition and group:  $p = .077$ , Table 5.11). As shown in Table 5.10, there were no latency differences between the groups, indicating that the timing of the response-locked components was equivalent across groups (a trend towards an interaction between condition and group fell just short of statistical significance:  $p = .057$ , Table 5.12). Note that the CRN and ERN are response-locked components meaning that they are not sensitive to actual RT. A main effect of component was found for latency, suggesting that the CRN occurs faster than the ERN.

#### 5.3.3.2. Pe.

The Pe was significantly attenuated in OA as shown in Tables 5.11 and 5.12 (see also Figure 5.4), indicating that, regardless of condition, OA had significantly lower Pe amplitudes

compared to YA. A lack of a main effect of condition suggests that this feature of later error processing in older participants was equivalent for compatible and incompatible stimuli.

Figure 5.4. Response-locked waveforms recorded from electrode FCz: Compatible condition (top figures) and incompatible condition (bottom figures) group grand averages.

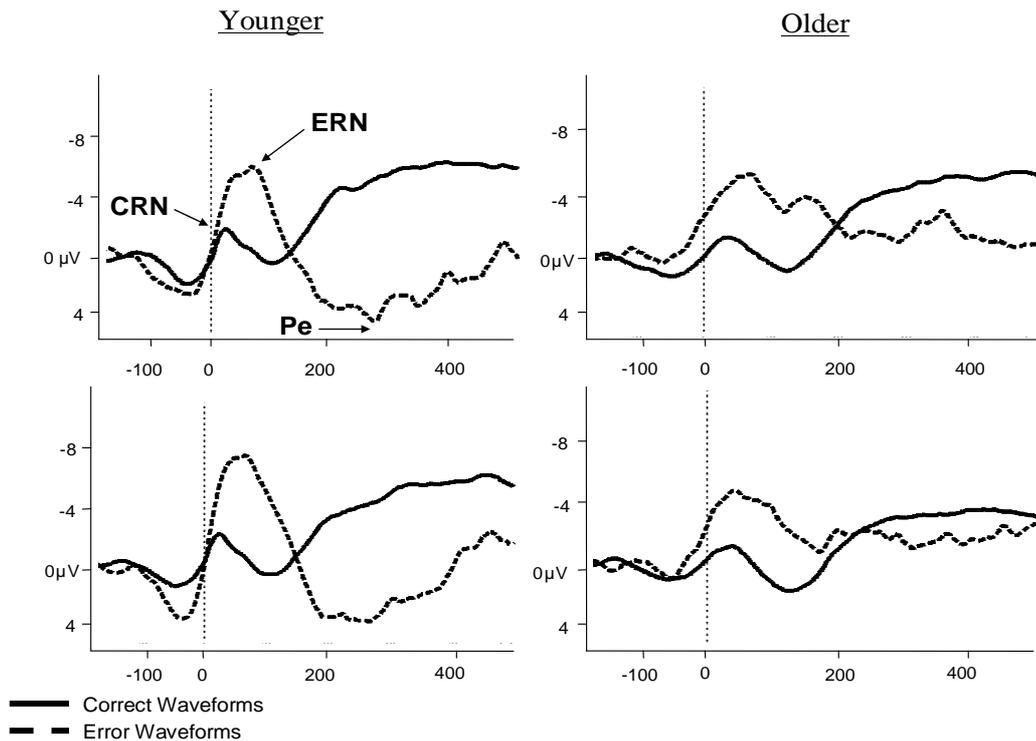


Table 5.10. Peak amplitudes and latencies of response-locked components for younger and older adults.

		<u>Younger</u>	<u>Older</u>	<u>Younger</u>	<u>Older</u>
		<i>n</i> = 18	<i>n</i> = 21	<i>n</i> = 18	<i>n</i> = 21
<i>M</i> ( <i>SD</i> )		Amplitude ( $\mu$ V)		Latency (ms)	
CRN	Compatible	5.7 (3.5)	3.8 (2.7)	35.3 (27.8)	44.3 (27.6)
	Incompatible	5.7 (3.1)	3.6 (2.6) †	33.0 (27.4)	39.0 (27.9)
ERN	Compatible	13.8 (7.1)	10.5 (6.8)	60.3 (31.8)	73.4 (42.8)
	Incompatible	15.4 (7.1)	9.4 (6.5) †	65.3 (33.9)	55.6 (42.7)
Pe	Compatible	7.8 (8.0)	1.7 (6.1) †	313.7 (62.0)	327.1 (56.3)

Incompatible	5.1 (6.9)	1.3 (4.6)	323.2 (51.1)	350.3 (48.3)
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*Note.* Condition = compatible or incompatible stimuli; Group = Older and Younger adults; †  $p < .05$  planned comparison using a t-test.

Table 5.11. Results for the amplitude of the response-locked components: CRN-ERN complex and the Pe.

			df	<i>F</i>	<i>p</i>
CRN- ERN	Main Effects	<b>Component</b>	1, 37	77.43	< <b>.001</b>
		Condition	1, 37	0.07	.798
		<b>Group</b>	1, 37	6.00	<b>.019</b>
	Interactions	Component X Group	1, 37	2.32	.136
		Condition X Group	1, 37	3.32	.077
		Component X Condition	1, 37	0.17	.686
		Component X Condition X Group	1, 37	2.71	.108
Pe	Main Effects	Condition	1, 37	2.30	.138
		<b>Group</b>	1, 37	7.60	<b>.009</b>
	Interaction	Condition X Group	1, 37	1.36	.251

*Note.* Condition = compatible or incompatible stimuli; Group = Older and Younger adults; significant effects in **bold** type.

Table 5.12. Results for the latencies of the response-locked components: CRN-ERN complex and the Pe.

			df	<i>F</i>	<i>P</i>
CRN- ERN	Main Effects	<b>Component</b>	1, 37	26.01	< <b>.001</b>
		Condition	1, 37	2.42	.128
		Group	1, 37	0.32	.577
	Interactions	Component X Group	1, 37	0.33	.569
		Condition X Group	1, 37	3.86	.057
		Component X Condition	1, 37	0.13	.720
		Component X Condition X Group	1, 37	1.89	.178

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Pe	Main Effects	Condition	1, 37	2.35	.134
		Group	1, 37	2.14	.152
	Interaction	Condition X Group	1, 37	0.41	.528

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*Note.* Condition = compatible or incompatible stimuli; Group = Older and Younger adults; significant effects in **bold** type.

In summary, although there were consistent delays in the processing of stimuli in OA, perhaps consistent with their longer RTs, there were no significant latency differences between the groups for any of the response-locked components. This suggests that the timing of the error processing system, once activated by the initiation of a motor response, does not differ with age.

#### 5.3.4. Postdiction Accuracy.

Postdiction measures of perceived accuracy were recorded for four blocks of the 4-CRT from 18 OA and 15 YA participants. In order to assess whether each group had accurately monitored their performance, discrepancy values were calculated by subtracting the postdiction accuracy percentage from the actual correct performance. The means and standard deviations of the performance and discrepancy scores over time are provided in Table 5.13, and the ANOVA models are shown in Table 5.14.

Firstly, performance accuracy over the duration of the task is considered. A block by group interaction (see Table 5.14) reflected a small increase in performance accuracy with increasing experience in OA and a small decrease in performance accuracy with experience in YA (see Table 5.13). Thus, performance in OA improved over time, whilst that in YA declined. A lack of any significant main effects or interactions for postdiction discrepancy scores suggested, however, that this pattern of responding had little effect on the individual's perceived accuracy.

Table 5.13. Correct performance and postdiction discrepancy over time for younger and older adults.

		<u>Younger</u>	<u>Older</u>
<i>M (SD)</i>		<i>n = 15</i>	<i>n = 18</i>
Correct %	Block 1	89.7 (7.2)	85.0 (12.5)
	Block 2	87.6 (8.0)	87.1 (10.5)
	Block 3	87.3 (6.1)	90.0 (7.6)
	Block 4	86.2 (8.4)	90.6 (6.0)
Postdiction Discrepancy %	Block 1	22.3 (14.4)	24.6 (18.9)
	Block 2	18.9 (10.4)	21.4 (20.4)
	Block 3	18.4 (11.0)	22.2 (21.9)
	Block 4	14.7 (11.4)	22.5 (21.8)

Table 5.14. ANOVA models for the perceived accuracy analyses.

			<i>df</i>	<i>F</i>	<i>p</i>
Correct %	Main Effects	Block	3, 93	.60	.616
		Group	1, 31	0.03	.872
	Interaction	<b>Block X Group</b>	3, 93	5.27	<b>.002</b>
Discrepancy	Main Effects	Block	3, 93	2.04	.114
		Group	1, 31	0.55	.462
	Interaction	Block X Group	3, 93	0.83	.482

*Note.* Condition = compatible or incompatible stimuli; Group = Older and Younger adults; significant effects in **bold** type.

### 5.3.5. Association between ERN/Pe, measures of affect, remedial actions and perceived accuracy.

There were no significant relationships (all  $p > .1$ ) between ERN or Pe amplitude and any of the measures of affect (HADS: anxiety and depression; PANAS: pre-test positive affect; post-test positive affect; discrepancy positive affect; pre-test negative affect; post-test negative affect; discrepancy negative affect; see also Tables 5.15 and 5.16) in either the YA or OA groups.

As shown in Table 5.17, the study also failed to find any relationships (all  $p > .1$ ) between ERN or Pe amplitudes and remedial actions of correcting errors or post-error slowing in the younger adults. However, a relationship was found between incompatible error detection and post-error slowing in the older adults, indicating that greater ERN amplitudes were associated with longer post-error slowing.

Further, perceived accuracy did not correlate with either ERN or Pe amplitudes in the OA. However, for the YA group, there were significant negative correlations between the perceived accuracy discrepancy measure and compatible and incompatible ERN amplitudes, suggesting that a greater difference between perceived accuracy and actual correct performance was associated with attenuated compatible and incompatible ERN amplitudes (see Table 5.16). Greater self-perceived inaccuracy was associated with lower ERN amplitudes in YA, but not in OA.

Table 5.15. Pearson product-moment correlations between the ERN, Pe, Anxiety and Depression in younger (below the line) and older adults (above the line).

	G_ERN	R_ERN	G_Pe	R_Pe	Anx	Dep
G_ERN		.81 (.66) †	-.09 (.01)	-.46 (.21) †	-.10 (.01)	-.15 (.02)
R_ERN	.78 (.61) †		.25 (.06)	-.28 (.08)	-.02 (.00)	-.23 (.05)
G_Pe	-.11 (.01)	.11 (.01)		.22 (.05)	.07 (.00)	-.40 (.16)
R_Pe	.02 (.00)	-.04 (.00)	.73 (.53) †		.16 (.03)	-.09 (.01)
Anx	.11 (.01)	-.05 (.00)	-.29 (.08)	-.22 (.05)		-.33 (.11)
Dep	.38 (.14)	.35 (.12)	-.32 (.10)	-.43 (.18)	.31 (.10)	

*Note.* G = Compatible 4-CRT condition; R = Incompatible 4-CRT condition; Anx = Anxiety; Dep = Depression; OA ( $n = 21$ ) data above the line; YA ( $n = 15$ ) data below the line;  $R^2$  values in parentheses; †  $p < .05$ .

Table 5.16. Correlations between the ERN, Pe and PANAS scales for younger (below the line) and older adults (above the line).

	G_ERN	R_ERN	G_Pe	R_Pe	Pre_PAS	Post_PAS	Disc_PAS	Pre_NAS	Post_NAS	Disc_NAS	Post_Avg
G_ERN		.81 (.66) †	-.09 (.01)	-.46 (.21) †	-.57 (.32)	-.28 (.08)	.25 (.06)	-.21 (.04)	-.38 (.14)	-.26 (.07)	-.07 (.00)
R_ERN	.78 (.61) †		.25 (.06)	-.28 (.08)	-.58 (.34)	-.45 (.20)	-.07 (.00)	-.18 (.03)	-.40 (.16)	-.28 (.08)	-.05 (.00)
G_Pe	-.10 (.01)	.11 (.01)		.22 (.05)	.03 (.00)	.24 (.06)	-.08 (.01)	.12 (.01)	.11 (.01)	.13 (.02)	-.09 (.01)
R_Pe	.02 (.01)	-.04 (.00)	.73 (.53) †		-.04 (.00)	-.01 (.00)	.09 (.01)	.26 (.07)	.21 (.04)	.06 (.00)	.05 (.00)
Pre_PAS	.10 (.01)	.01 (.00)	.16 (.03)	.35 (.12)		.70 (.49) †	-.24 (.06)	.05 (.00)	.47 (.22)	.39 (.15)	-.41 (.17)
Post_PAS	.14 (.02)	.20 (.04)	.32 (.10)	.48 (.23)	.86 (.74) †		.53 (.28)	.31 (.10)	.22 (.05)	.18 (.03)	-.38 (.14)
Disc_PAS	.08 (.01)	.38 (.14)	.32 (.10)	.25 (.06)	-.26 (.07)	.27 (.07)		.36 (.13)	-.10 (.01)	-.23 (.05)	-.56 (.31)
Pre_NAS	.26 (.07)	.04 (.00)	.15 (.02)	.26 (.07)	.02 (.00)	.03 (.00)	.01 (.00)		-.10 (.01)	-.47 (.22)	.06 (.00)
Post_NAS	.28 (.08)	-.20 (.04)	-.16 (.03)	.07 (.00)	-.06 (.00)	-.15 (.02)	-.18 (.03)	.86 (.74) †		.92 (.85) †	.24 (.06)
Disc_NAS	.09 (.01)	-.46 (.21)	-.57 (.32) †	-.32 (.10)	-.15 (.02)	-.34 (.12)	-.37 (.14)	-.09 (.01)	.44 (.19)		-.94 (.88) †
Post_Avg	-.54 (.29) †	-.58 (.34) †	-.13 (.02)	-.40 (.16)	-.31 (.10)	-.53 (.28)	-.41 (.17)	-.11 (.01)	-.04 (.00)	.11 (.01)	

*Note.* G = Compatible 4-CRT condition; R = Incompatible 4-CRT condition; Pre\_PAS = pre-test Positive Affect Scale; Post\_PAS = post-test Positive Affect Scale; Disc\_PAS = PAS discrepancy between pre and post-testing; Pre\_NAS = pre-test Negative Affect Scale; Post\_NAS = post-test Negative Affect Scale; Disc\_NAS = NAS discrepancy between pre and post-testing; Post\_Avg = average postdiction performance %; OA ( $n = 11$ ) data above the line; YA ( $n = 13$ ) data below the line; †  $p < .05$ ;  $R^2$  values in parentheses.

Table 5.17. Correlations between components of error detection, error processing and remedial actions in younger (below the line) and older adults (above the line).

	G_ERN	R_ERN	G_Pe	R_Pe	G_EC	R_EC	G_PES	R_PES
G_ERN		.81 (.66) †	-.09 (.01)	-.46 (.21) †	.19 (.04)	-.03 (.00)	.25 (.06)	.46 (.21) †
R_ERN	.78 (.61) †		.25 (.06)	-.28 (.08)	.30 (.09)	.03 (.00)	.10 (.01)	.47 (.22) †
G_Pe	-.10 (.01)	.11 (.01)		.22 (.05)	-.23 (.05)	-.35 (.12)	.13 (.02)	.12 (.01)
R_Pe	.02 (.00)	-.04 (.00)	.73 (.53) †		-.17 (.03)	-.05 (.00)	-.16 (.03)	-.36 (.13)
G_EC	.22 (.05)	.06 (.00)	-.30 (.09)	-.17 (.03)		.74 (.55) †	-.29 (.08)	.13 (.02)
R_EC	.14 (.02)	-.03 (.00)	-.25 (.06)	.06 (.00)	.89 (.79) †		-.36 (.13)	.10 (.01)
G_PES	-.03 (.00)	-.18 (.03)	.05 (.00)	.31 (.10)	.23 (.05)	.26 (.06)		-.12 (.01)
R_PES	-.16 (.03)	-.26 (.07)	-.09 (.01)	-.08 (.01)	-.04 (.00)	-.02 (.00)	.02 (.00)	

*Note.* G = Compatible 4-CRT condition; R = Incompatible 4-CRT condition; EC = error correction; PES = post-error slowing; OA ( $n = 20$ ) data above the line; YA ( $n = 17$ ) data below the line; †  $p < .05$ ;  $R^2$  values in parentheses.

### 5.3.6. Woody filtered response-locked components.

Mean and standard deviations of the amplitudes and latencies of the CRN, ERN and Pe response-locked components from the Woody filter process are presented in Table 5.18. The results from the ANOVA models are given in Tables 5.19 and 5.20.

#### 5.3.6.1. CRN-ERN Components.

In general, the results from the conventional averaging process were replicated (see Section 5.3.3) with the Woody filter. As shown in Table 5.19, there were main effects of component and group on CRN-ERN amplitude indicating that the error processing system was functional in both groups, albeit reduced in the OA. However, using the Woody filter, both a component by group interaction and a condition by group interaction were revealed (see Table 5.19). The component by group interaction is considered first. This reflects a greater difference between CRN and ERN amplitudes in YA compared to OA, irrespective of condition. The interaction between condition and group is due to a slight fall in both CRN and ERN amplitudes with increasing task complexity (compatible vs. incompatible stimuli) in OA, compared to minimal change in YA. In support, post-hoc tests found that the condition difference in CRN and ERN amplitudes was not significant for the younger adults ( $p > .1$ ), however there were trends in the OA group, in that the incompatible condition elicited lower CRN and ERN amplitudes:  $t(20) = 2.07, p = .051$ ;  $t(20) = 1.90, p = .071$ , CRN and ERN respectively. The latency of the CRN was also found to be shorter than the ERN, consistent with the results from the conventional averaging process (see Section 5.3.3), indicating that the CRN occurred faster than the ERN.

#### 5.3.6.2. Pe Component.

The Woody filtered Pe component was also found to be significantly reduced in the OA (see Table 5.19), again replicating the finding from the conventional averaging process.

### 5.3.6.3. Direct Comparison between Conventional Averaging and the Woody Filter.

A series of mixed model ANOVAs investigated the comparison between the two methods (see Table 5.23), with main effects of analysis type indicating that the Woody filtering process significantly increased the amplitudes of the ERN and CRN for both conditions and the Pe for the incompatible condition. Group interactions were only found for the ERN (both conditions), indicating that the YA actually had greater ERN amplitude increases using the Woody filtering process compared to the OA. As shown in Table 5.21, within-group planned comparisons revealed that the Woody filter produced significantly increased CRN and ERN amplitudes for both conditions and increased Pe amplitude for the incompatible condition in younger adults. However, in the older adults, significant amplitude increases were only found for the ERN for the compatible condition and the CRN for both conditions.

Table 5.18. Peak amplitudes and latencies of Woody filtered response-locked components for younger and older adults.

		<u>Younger</u>	<u>Older</u>	<u>Younger</u>	<u>Older</u>
		<i>N</i> = 18	<i>n</i> = 21	<i>n</i> = 18	<i>n</i> = 21
<i>M</i> ( <i>SD</i> )		Amplitude ( $\mu$ V)		Latency (ms)	
CRN	Compatible	10.3 (6.5)	8.0 (5.2)	43.7 (20.8)	49.0 (42.3)
	Incompatible	9.9 (5.3)	5.9 (5.1) †	41.1 (30.6)	51.0 (36.6)
ERN	Compatible	21.0 (8.6)	15.0 (9.4) †	67.9 (30.0)	80.2 (48.3)
	Incompatible	22.3 (8.6)	11.5 (6.9) †	77.3 (40.7)	64.1 (49.8)
Pe	Compatible	10.3 (12.5)	2.6 (12.6)	335.6 (59.2)	337.3 (40.2)
	Incompatible	9.7 (11.8)	2.4 (11.5)	336.4 (57.1)	328.6 (43.8)

Note. †  $p < .05$ .

Table 5.19. ANOVA models for the amplitude of the Woody filtered response-locked components.

			df	<i>F</i>	<i>p</i>
CRN -ERN	Main Effects	<b>Component</b>	1, 37	56.37	< <b>.001</b>
		Condition	1, 37	3.01	.091
		<b>Group</b>	1, 37	11.88	<b>.001</b>
	Interactions	<b>Component X Group</b>	1, 37	4.87	<b>.034</b>
		<b>Condition X Group</b>	1, 37	5.88	<b>.020</b>
		Component X Condition	1, 37	0.01	.919
		Component X Condition X Group	1, 37	0.98	.329
Pe	Main Effects	Condition	1, 37	0.06	.810
		<b>Group</b>	1, 37	4.58	<b>.039</b>
	Interaction	Condition X Group	1, 37	0.01	.907

*Note.* Condition = compatible or incompatible stimuli; Group = Older and Younger adults; significant effects in **bold** type.

Table 5.20. ANOVA models for the latency of the Woody filtered response-locked components.

			df	<i>F</i>	<i>p</i>
CRN- ERN	Main Effects	<b>Component</b>	1, 37	17.89	< <b>.001</b>
		Condition	1, 37	0.14	.716
		Group	1, 37	0.17	.679
	Interactions	Component X Group	1, 37	0.42	.521
		Condition X Group	1, 37	1.14	.292
		Component X Condition	1, 37	0.10	.749
		Component X Condition X Group	1, 37	2.53	.120
Pe	Main Effects	Condition	1, 37	0.16	.690
		Group	1, 37	0.06	.813

Interaction	Condition X Group	1, 37	0.24	.626
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*Note.* Condition = compatible or incompatible stimuli; Group = Older and Younger adults; significant effects in **bold** type.

Table 5.21. Peak amplitudes of response-locked components from the Woody filtered process and from the conventional averaging process for younger and older adults.

		<u>Younger</u>		<u>Older</u>	
		<i>n</i> = 18		<i>n</i> = 21	
<i>M</i> ( <i>SD</i> )		Conventional	Woody	Conventional	Woody
CRN	Compatible	5.7 (3.5)	10.3 (6.5) †	3.8 (2.7)	8.0 (5.2) †
	Incompatible	5.7 (3.1)	9.9 (5.3) †	3.6 (2.6)	5.9 (5.1) †
ERN	Compatible	13.8 (7.1)	21.0 (8.6) †	10.5 (6.8)	15.0 (9.4) †
	Incompatible	15.4 (7.1)	22.3 (8.6) †	9.4 (6.5)	11.5 (6.9)
Pe	Compatible	7.8 (8.0)	10.3 (12.5)	1.7 (6.1)	2.6 (12.6)
	Incompatible	5.1 (6.9)	9.7 (11.8) †	1.3 (4.6)	2.4 (11.5)

*Note.* † *p* < .05.

Table 5.22. Peak latencies of response-locked components from the Woody filtered process and from the conventional averaging process for younger and older adults.

		<u>Younger</u>		<u>Older</u>	
		<i>n</i> = 18		<i>n</i> = 21	
<i>M</i> ( <i>SD</i> )		Conventional	Woody	Conventional	Woody
CRN	Compatible	35.3 (27.8)	43.7 (20.8) †	44.3 (27.6)	49.0 (42.3)
	Incompatible	33.0 (27.4)	41.1 (30.6) †	39.0 (27.9)	51.0 (36.6)
ERN	Compatible	60.3 (31.8)	67.9 (30.0)	73.4 (42.8)	80.2 (48.3)
	Incompatible	65.3 (33.9)	77.3 (40.7)	55.6 (42.7)	64.1 (49.8)
Pe	Compatible	313.7 (62.0)	335.6 (59.2)	327.1 (56.3)	337.3 (40.2)
	Incompatible	323.2 (51.1)	336.4 (57.1)	350.3 (48.3)	328.6 (43.8)

*Note.* † *p* < .05.

Table 5.23. ANOVA models for the comparison between conventional averaging and Woody filtering for the amplitude of the response-locked components.

			df	<i>F</i>	<i>P</i>
Compatible CRN	Main Effects	<b>Analysis</b>	1, 37	50.22	< <b>.001</b>
		Group	1, 37	2.33	.136
	Interactions	Analysis X Group	1, 37	0.08	.783
Incompatible CRN	Main Effects	<b>Analysis</b>	1, 37	31.66	< <b>.001</b>
		<b>Group</b>	1, 37	6.22	<b>.017</b>
	Interactions	Analysis X Group	1, 37	2.92	.096
Compatible ERN	Main Effects	<b>Analysis</b>	1, 37	81.68	< <b>.001</b>
		Group	1, 37	3.42	.072
	Interactions	<b>Analysis X Group</b>	1, 37	4.47	<b>.041</b>
Incompatible ERN	Main Effects	<b>Analysis</b>	1, 37	29.19	< <b>.001</b>
		<b>Group</b>	1, 37	14.65	< <b>.001</b>
	Interactions	<b>Analysis X Group</b>	1, 37	8.45	<b>.006</b>
Compatible Pe	Main Effects	Analysis	1, 37	1.67	.204
		<b>Group</b>	1, 37	5.36	<b>.026</b>
	Interactions	Analysis X Group	1, 37	0.36	.554
Incompatible Pe	Main Effects	<b>Analysis</b>	1, 37	6.04	<b>.019</b>
		<b>Group</b>	1, 37	4.15	<b>.049</b>
	Interactions	Analysis X Group	1, 37	2.35	.134

*Note.* Condition = compatible or incompatible stimuli; Group = Older and Younger adults; significant effects in **bold** type.

### 5.3.7. Summary of Results.

With the exception of reaction time and number of missed trials, there were no behavioural differences between the YA and OA groups. Both groups managed the increase in task complexity equivalently, with older adults performing better over time. The incompatible condition elicited greater N2 amplitudes, associated with conflict detection, which were similar across groups. However, group differences were found for subsequent stimulus-locked components. The P3b and N4 were both attenuated and delayed in the older adults, and the SW was significantly delayed. In both conventional and Woody filter analyses of response-locked components, there were age-related reductions in the ERN and the Pe, indicating an impoverished error detection system. However, the integrity of the error detection system may also be judged by the presence of a significant difference between the CRN and ERN. There was no significant interaction between component (CRN-ERN) and group in the conventional analysis, suggesting that irrespective of reduced ERN power in OA, the error detection system was operational. However, a component by group interaction was revealed in the Woody filter analysis, due mainly to the filtering process improving YA amplitudes, and this provided further evidence of an age-related reduction in the error detection system. The study did not find any relationships between the ERN/Pe and affect in either group, nor with remedial actions in the younger adults. However, greater error detection in the incompatible condition was associated with increased post-error slowing in the older adults, suggesting a link between internal performance monitoring and compensatory behaviour. Moreover, more accurate perception of performance was associated with greater ERN amplitudes in both conditions in the younger adults, suggesting a link between the ERN and awareness of performance in this group.

### 5.4. Discussion.

The current study investigated Consistent Consciousness, a type of awareness involving executive functioning and its ability to organise sensory information underlying behaviour (Stuss et al., 2001) in healthy older and younger participants. It was hypothesised that an

investigation of performance monitoring would provide a means to assess this type of awareness. As predicted, and in line with previous studies (Band & Kok, 2000; Falkenstein et al., 2000; Falkenstein et al., 2001; Mathalon et al., 2003; Mathewson et al., 2005), older adults had reduced ERN and Pe amplitudes. While this did not affect the CRN-ERN difference, a marker of the integrity of the error-detection system, using conventional analysis, there was a significant interaction when data were corrected for latency jitter (Woody filter). Specifically, this correction amplified the younger adult waveforms such that the smaller CRN-ERN difference in the older adults became, by comparison, significantly smaller than the CRN-ERN difference in younger adults. Importantly, there were no behavioural differences between the groups with regard to the most influential factor on the ERN and Pe, namely error rate. Remedial actions (error correction and post-error slowing) were equivalent between groups, and both groups managed the increase in task complexity (compatible to incompatible stimuli) equivalently. However, the older adults had significantly slower responses, failed to respond to more trials and made additional unnecessary responses (altering correct responses to errors). As performance monitoring also involves the sub-process of response competition (Ullsperger & von Cramon, 2001), the study investigated conflict and found, in line with Kopp et al. (1996), that the N2 amplitude was influenced by stimulus-response compatibility. Of note is that the detection and timing of this marker of pre-response conflict was equivalent across groups. The N4, however, did not differ with level of conflict, contrary to Hogan et al. (2006)<sup>7</sup>, but was both attenuated and delayed in older adults suggesting that response priming was altered with age.

ERP components provide an on-line measurement of the processes underlying cognition and behaviour. In the current study, Consistent Consciousness was conceptualised by performance monitoring which requires the integration of numerous processes: conflict detection; allocation of attention; response priming; error detection; and error processing. The results indicated that age was associated with differential effects on brain processes underlying performance monitoring. Whilst markers of conflict detection did not differ with age, the N4 was attenuated and delayed. As early stimulus processing (N2) was normal it is possible that older adults

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<sup>7</sup> Hogan et al. (2006) included 3 groups of participants aged 11-23.7 years (sickle cell with frontal lobe infarctions), 13.6-25.0 years (sickle cell without infarcts), and 12.2-21.0 years (sibling controls). Therefore, there is some overlap in age ranges between Hogan et al. (2006) study and the younger adult group in the current study.

begin to process information similarly to younger adults, but that this ability declines as a response is initiated. Interestingly, the former process reflects activity of the association cortices and the latter the additional involvement of the frontal lobe (Hogan et al, 2006). Compatible with this, response-locked error components occurring very shortly after the P3b/N4/SW complex in real time, and probably overlapping to a degree, were also affected in the older adults. These results are, therefore, consistent with the view that the ageing process exerts its greatest effect on frontal lobe networks (e.g. Moscovitch & Winocur, 1995; Raz 2000; 2004; West, 1996).

Only two previous studies (Falkenstein et al., 2000; Mathalon et al., 2003) have investigated age effects on stimulus-locked components in performance monitoring. However, these studies differed with regard to P3 amplitude; Falkenstein et al. (2000) found reductions with age, whilst Mathalon et al. (2003) did not. In support of Falkenstein et al. (2000), in this study P3b amplitude was significantly reduced in the older adults, perhaps indicating that attentional allocation to stimulus processing was altered in this group. It would be of interest to investigate, on a trial-by-trial basis, fluctuations in P3b component amplitude, alongside fluctuation in other stimulus-locked components, in order to explore possible dynamic changes in attention allocation during the course of the task. Fluctuations in online stimulus-processing between younger and older adult groups may offer further insight into altered performance-monitoring components. To the author's knowledge, no such study has yet been undertaken to explore performance-monitoring in ageing.

One of the prominent caveats in the literature regarding factors that modulate the ERN amplitude was provided by Hajcak et al. (2003) and Herrmann et al. (2004). Namely, that ERN amplitude was reduced with increased error rate. The current study found equivalent rates of error performance between groups, and within-group correlations failed to find any relationships between error rate and ERN and Pe amplitudes. These findings are of particular importance to interpretations that follow with regard to the attenuated ERN and Pe in the older adults.

Individual differences in affect in non-clinical measures using the Positive and Negative Affect Scale (PANAS) have also been found to modulate the ERN and Pe (Hajcak et al., 2004). Whilst the present study also utilised the PANAS, in addition to the HADS, no relationships were found between these measures and the ERN and Pe amplitudes in either group. This suggests that affect did not confound the altered ERN amplitude found in older adults. However, in the current study data were treated as continuous, in contrast to Hajcak et al. (2004), in which PANAS scores were used to categorise the sample into high and low negative affect groups. In addition, the range of negative affect scores in the current study (YA = 10-16, OA = 10-15) differed from Hajcak et al. (2004) in that high negative affect was classified as  $M = 21.1$ ,  $SD = 3.27$ . Thus it must also be considered that the lack of significant associations in the present study may have been due to methodological differences and/or the sample not having scores representative of high negative affect.

When performance is accurately monitored, behaviour can be adapted to meet the task demands of correct performance. Compensatory actions include immediate corrective behaviours (error correction and post-error slowing) and strategy changes, for example response speed (Ullsperger & von Cramon, 2001). The present study found that immediate remedial actions were similar across groups, however, the older adults consistently responded significantly slower, and responded to fewer trials, suggesting that older adults utilised a different response strategy compared to the younger adults. It may not be assumed, however, that one strategy was better than the other. For example, the response strategy adopted by the older adults may also have contributed to the significant improvement in their performance over time, in contrast to the younger adults, whose performance declined over time. This suggests that slower reaction times may have facilitated surer performance. A complimentary interpretation is that the older adults had up-regulated activity in attention and cognitive control circuits to compensate for impairments in information processing (cf. Band & Kok, 2000); anecdotally, it was noted that they were highly motivated to perform the task well. Converging confirmatory evidence for this proposition comes from Sharp, Scott, Mehta and Wise (2006). In a PET study with 8 younger (37-54 years) and 8 older (60-83 years) adults, Sharp et al. (2006) investigated the hypothesis that reduced performance capabilities in ageing were nevertheless associated with increased activity in the ACC and the lateral PFC, both

regions supporting cognitive control. As predicted, the study found age-related deficits in decision-making accuracy, and this was associated with increased activation in the ACC. However, contrary to this interpretation is the possible reduction in underlying EEG power suggested by the fact that most, but not all, components were attenuated in older adults. It would be of interest to investigate the effect of underlying EEG power in alpha, beta, theta and delta ranges on ERP components in order to address this hypothesis further. However, spectral analyses are conducted on EEG data recorded during a period of time (e.g. 2 minutes) with eyes open and eyes closed rather than during performance of a task. Moreover, the epochs for spectral analyses are longer than in ERP analyses (e.g. 8 seconds vs. 1 second), with ERP epochs specified by an event (stimulus/response) rather than a segment of time. Therefore, it can be problematic to extrapolate to the relationship between the EEG and ERP components, as the challenge is in linking voltage changes to events to underlying oscillations in electrical activity. However, there has been recent development of techniques that permit an investigation of the underlying spectral power alongside conventional ERP analyses (see Hanslmayr et al. 2008; Yordanova, Falkenstein, Hohnsbein & Kolev, 2004).

Despite performance monitoring deficits indicated by the ERP components, the behavioural adaptations (e.g. post-error slowing) of the older adults to errors were equivalent to those of the younger adults. The implications of these findings are that ageing is associated with impairments in some aspects of the ability to organise sensory information as it initiates executive functions. This may be interpreted as consistent with a deficit in Consistent Consciousness, as defined by Stuss et al. (2001), but such a relationship is not confirmed. Importantly, a positive interpretation may also be offered: effective and adaptive remedial actions and response strategies were found; suggesting that performance monitoring was functional albeit attenuated in the older adults, and that residual function and as yet unknown compensatory mechanisms may supersede deficits that occur earlier in the monitoring process.

The investigation of compensatory behaviours precedes ERP studies of performance monitoring (e.g. Rabbitt, 1966). Error correction and post-error slowing are immediate remedial actions, occurring within a few seconds of the error, and relationships with the ERN and Pe have been explored with mixed results. Although Gehring et al. (1993) found that ERN

amplitude increased with the probability of error correction and post-error slowing, these results have not been consistently replicated (Falkenstein et al., 2000; Falkenstein et al., 2001; Gehring & Fencsik, 2001; Hajcak et al., 2003). Pe amplitude elicited by a flanker task has been statistically associated with post-error slowing (Hajcak et al., 2003), in that higher Pe amplitudes were associated with increased post-error slowing, but did not differ between errors corrected and those uncorrected (Falkenstein et al., 2000). Using an antisaccade task to investigate perceived and unperceived error performance, Nieuwenhuis et al. (2001) only found post-error slowing for perceived errors. ERN amplitude did not differ between perceived and unperceived errors; however, the Pe amplitude was significantly greater for perceived errors. Nieuwenhuis et al. (2001) argued that the covariation of the post-error slowing and Pe amplitude results (see also Hajcak et al., 2003) supported the view that the Pe represented further conscious error processing and response adjustment. However, no statistical analyses were reported investigating the relationship between Pe amplitude and post-error slowing. With regard to the relationship between the ERN, Pe and compensatory behaviours in ageing, Falkenstein and colleagues (Falkenstein et al., 2000; Falkenstein et al., 2001) found greater post-error slowing in older adults, but similar rates of error correction to younger adults (although numerically lower in the older adults). One possible explanation for the contradictory results between these studies may be due to statistical analyses employed. Gehring et al. (1993) calculated quartiles of ERN size based on the probability of an error being present in a random subset of correct and error trials. Other studies have separated and subsequently averaged trials on the basis of the variable of interest: errors corrected and errors uncorrected (Falkenstein et al., 2000); trials with and without post-error slowing (Gehring and Fencsik, 2001); post-error slowing for perceived and unperceived errors (Nieuwenhuis et al., 2001); post-error slowing calculated from error trials and a subset of correct trials matched to equivalent number and RT in error trials (Hajcak et al., 2003). The analyses conducted by Gehring et al. (1993) differed from other studies in that the ERN amplitudes were calculated by error probability in a subset of trials rather than error trials separated by error correction or post-error slowing. The findings of relationships with error correction and post-error slowing to ERN amplitude may be due to these behaviours not being performed in correct trials, and the lack of consideration of these behaviours in all error trials.

The current study found equivalent rates of error correction and post-error slowing between older and younger adults. Both measures were calculated from behavioural data. The rates of error correction in the current study (YA = 27.6% and 24.4%; OA = 39.6% and 31.1%, compatible and incompatible conditions, respectively) were lower than those reported by Falkenstein et al. (2001): 65% and 59%, younger and older adults, respectively for the 4CR task; and 63% and 43%, younger and older adults, respectively for the flanker task. However, the interstimulus-interval (ISI) in the Falkenstein et al. (2001) study was greater than in the current study (1800ms vs. 1500ms); the increased ISI may have provided a greater opportunity for error correction, even with only a 300ms difference. The current study also failed to find any relationships between corrective measures and ERN and Pe amplitudes in the younger adults. However, an association between increased ERN amplitudes in the incompatible condition and longer post-error slowing in older adults was found, indicating a link between internal error detection and compensatory behaviour as a function of increased task complexity. Increased task-complexity is associated with up-regulation of frontal lobe activity (Hogan et al., 2006). This further suggests that the frontal lobes are of critical interest in the investigation of attenuated performance-monitoring in normal ageing. It would be of considerable interest to conduct functional imaging studies based on the performance-monitoring ERP paradigms to confirm this theory. The CRN-ERN magnitude difference was also calculated to provide a measure of the functional integrity of the error detection system. Again, within-group correlations with remedial actions were not significant.

In summary, these findings suggest that neither ERN nor Pe amplitudes, nor the CRN-ERN difference are related to immediate corrective behaviours in younger adults. Despite different statistical analyses, the findings in the younger adults are in line with previous results of a lack of relationships between ERN and Pe amplitudes and post-error slowing and error correction (Gehring & Fencsik, 2001; Falkenstein et al., 2000; Falkenstein et al., 2001). Falkenstein et al. (2001) suggested that the reduction in the ERN in older adults may not have been sufficient significantly to influence error correction. Nevertheless, the finding of a relationship between ERN amplitude and post-error slowing in older adults in the present study extends existing evidence of relationships between the ERN, Pe and corrective behaviours (Gehring et al., 1993; Hajcak et al., 2003; Nieuwenhuis et al., 2001). Further investigation is required to

understand the significance of such relationships for the functional neuroanatomy of performance-monitoring in normal ageing. The most conservative conclusion is that a complex relationship is indicated between electrophysiological and behavioural correlates of performance monitoring, and that this relationship may change slightly over the normal life-span.

Error correction and post-error slowing have been described as indirect indices of error awareness (Rabbitt, 1966). With regard to the aim of this study, which was to investigate the Consistent Consciousness level of awareness, it may be concluded, on the basis of behavioural performance, that error awareness did not change with age. However, as the Pe has also been associated with awareness and with error recognition and post-error slowing (Hajcak et al., 2003; Nieuwenhuis et al., 2001), and this component was significantly attenuated in older adults, it may alternatively be concluded that there is some ERP support for decline in awareness; the fact that behavioural performance was normal suggests perhaps that the decline has not reached a threshold for *functional* impairment in awareness. Finally, another possible explanation for the lack of evidence for relationships between error detection and processing as indexed by the ERN and Pe, and corrective behaviours in the present study, was provided by Bernstein et al. (1995): “the process by which errors are detected [is] not necessarily available to awareness” (p.1312). In other words, the error-detection system as indexed by the ERN may be very fast and unconscious, while awareness of having made an error, albeit initiated by this detection system, occurs over a longer period of time and involves other processes, such as working memory, affect and motivation, in ways not assessed by the present study.

Postdiction of performance is utilised within the metamemory literature as a measure of memory monitoring, conceptualised as analogous to awareness. Studies have found that older adults are less accurate in their monitoring of their abilities; although there is evidence of updating of self-assessment through experience, revisions are still less accurate in older adults compared to younger adults (e.g. Bieman-Copland & Charness, 1994). The current study investigated age differences in postdiction of performance, and found that both groups had similar underestimation of their performance and that neither group revised their postdictions

with experience. The underestimation of performance was not influenced by affect in either group, and previous metamemory studies (e.g. Ansell & Bucks, 2006; Bieman-Copland & Charness, 1994) utilised tasks with fewer stimuli to monitor, suggesting that monitoring of performance in the current study, may have been more difficult due to the greater length of task performance.

Scheffers and Coles (2000) included a postdiction measure of performance accuracy, a five-point rating scale from sure correct to sure incorrect, after each trial and found that greater ERN amplitudes were related to greater perceived inaccuracy. Interestingly, in support of Scheffers and Coles (2000), ERN amplitudes were associated with awareness of performance accuracy; more accurate perception of performance was linked with increased ERN amplitudes in both compatible and incompatible conditions, but only in the younger adults. The current study differed from Scheffers and Coles (2000) in that postdictions were required after each block of 100 trials rather than after each trial. Furthermore, discrepancy scores, the difference between perceptions of performance and actual performance, were calculated and correlated with ERN amplitudes. However the present study, and that of Scheffers and Coles (2000), provides limited but converging evidence, albeit using different methodologies, that the ERN is associated with awareness of performance in younger adults. No relationships were found between discrepancy scores and corrective measures, indicating that these different measures of awareness may not be related, but this may need to be confirmed in studies with larger sample sizes. The results also indicate that prediction of accuracy of performance, as indexed by larger discrepancy scores, is associated with reduced ERN amplitudes, providing support for Pailing and Segalowitz (2004), who found an attenuation of the ERN amplitude with uncertainty of performance in younger adults. Gehring and Fencsik (2001) concluded that the ERN may represent an evaluative process, and the results from the younger adults in the current study provide some support for this proposition. Data obtained from the older adults are not easily explained and argue against a relationship between the ERN and awareness of performance, as there were no group differences in discrepancy scores or postdictions, indicating that awareness of performance did not differ with age. However, the standard deviation of the older adult discrepancy scores is almost double that of the younger adults,

indicating that the discrepancy scores were more variable in the older adults, which may account for the lack of a relationship between ERN amplitude and awareness of performance.

This study was unique in the application of the Woody filter to explore possible effects of latency jitter on response-locked components. To the author's knowledge there is only one published study conducting single trial analyses on older adult electrophysiological ERN data (Falkenstein et al., 2001). The results of those analyses suggested some amplitude reduction by single-trial latency jitter in the increased latency standard deviation. More recently, Walhovd, Rosquist and Fjell (2008) employed the Maximum Likelihood Estimation (MLE) correction technique to assess latency jitter in the P3a and P3b elicited in a three-stimuli visual oddball paradigm in a sample of 133 adults, aged 20-88 years. The study found that P3a and P3b amplitudes were significantly increased with the MLE technique compared to conventional analyses. The authors correlated age with the amplitudes and latencies of the P3a and the P3b, and found that the correlations from conventional analyses and the MLE technique were not significantly different. These results indicate that adjusting for latency jitter in stimulus-locked waveforms does not alter the findings of reduced amplitudes and longer latencies in the P3a and P3b in ageing. Similarly to Walhovd et al. (2008), the results of the Woody filter in the current study replicated the findings of the conventional analyses, in that older adults had significantly reduced ERN and Pe amplitudes compared to younger adults. Moreover, group interactions with component and condition found that the magnitude of the difference between the CRN and ERN was greater in the younger adults compared to the older adults, and that older adults had attenuated CRN and ERN amplitudes elicited by the incompatible condition compared to the compatible condition. The Woody filter increased the amplitude in response-locked components in both groups, which indicated that latency jitter occurred in both younger and older adult waveforms from the conventional averaging process. Contrary to the expectation that variability in older adult trials would lead to greater latency jitter in the averaged waveforms compared to younger adults, the study found that latency jitter was greater in the younger adults. Despite different paradigms, ERP components investigated and latency jitter techniques, both the current study and that of Walhovd et al. (2008) have shown that latency jitter is present in both younger and older adult waveforms. Taken together, the results from these studies suggest that latency jitter influences the

amplitudes of components in all age groups. However, this difference does not impact on the results from both types of analysis. In summary, using the Woody filter to account for latency jitter yielded an interaction between component and group that was not present in the conventional analysis, but this was due to the correction of greater ‘jitter’ in the younger compared to older adults. These analyses, which are highly novel, address the concern that age-related amplitude reductions in response-locked components are artefacts of the conventional averaging process. The current study indicates that the attenuated error processing components in older adults, consistent with previous studies (Band & Kok, 2000; Falkenstein et al., 2000; Falkenstein et al., 2001; Mathalon et al., 2003; Mathewson et al., 2005), are genuine age-related reductions, rather than artefacts of the averaging process.

The four choice reaction time paradigm utilised in the current study provided both sufficient error rates and facilitated an investigation of two predominant theories of the functional significance of the ERN. The error-detection model (Falkenstein et al., 1991; Gehring et al., 1993) posits that the ERN is a signal elicited by a mismatch between the representation of the correct response and the actual (erroneous) response. Whereas the conflict-detection model (Carter et al., 1998; van Veen & Carter, 2002) contends that the ERN represents a measure of increased stimulus-response conflict. The incompatible condition in the current study provided an opportunity to measure response conflict. ERN amplitude did not differ between conditions in either group, indicating that the incompatible condition did not elicit greater amplitudes, supporting the view that the ERN is a generic error detection signal. That the N2 was modulated by pre-response conflict, but without any concurrent effect on the ERN, suggests that conflict detection was less influential on the ERN than previous studies suggest (e.g. van Veen & Carter (2002)). The current study was unable to contribute to the debate over the functional significance of the Pe. Specifically, the study failed to find any relationships between Pe amplitude and corrective behaviours, awareness of performance, or affect in either group. Studies have suggested that the Pe represents conscious recognition of error performance and is related to remedial post error slowing (Hajcak et al., 2003; Nieuwenhuis et al., 2001). The null results in the current study may have been due to the methodology for assessing awareness of performance; a postdiction measure of the perception of performance. A more valid measure of awareness may be recorded by requesting the participants to report

on performance after each response or the number of errors after each block of 10 trials. However, it is not known what effect such measures might have on task performance or on corrective behaviours, since this might interrupt the flow of responses during the task.

Studies have also indicated that the ERN can be modulated by manipulating the salience of the error (Bernstein et al., 1995; Falkenstein et al., 2000; Gehring et al., 1993). Although errors in the incompatible condition might be considered as more salient, the lack of condition effects and the equal probability of compatible and incompatible stimuli argue against this view. Habituation to the occurrence of errors was offered as a possible explanation for findings of reduced ERN amplitude with increased error rates (Hajcak et al., 2003; Herrmann et al., 2004). Data from the present study from a subset of younger ( $n = 4$ ) and older adults ( $n = 5$ ) with sufficient error rates ( $>5$ ) in each block of the paradigm were explored in order to investigate this, but significant differences in the ERN amplitude with time were not found, indicating that habituation to making errors was not influencing the amplitude (Appendix 5). As the error rate did not differ between groups, nor were there significant correlations between error rate and ERN or Pe amplitude, reduction in the amplitudes in the older adults was not due to error rates.

The age range of the older adults in the current study (60-83 years) is comparable to other studies of performance monitoring in ageing (Falkenstein et al., 2000; Falkenstein et al., 2001; Mathalon et al., 2003; Mathewson et al., 2005). It is clear that ageing is associated with significant neural and cognitive change (see Sections 2.4 and 2.5, Chapter 2). For example, Resnick et al. (2003) found annual volume decreases of 2.4 SD 0.4 and 3.1 SD 0.4 cm<sup>3</sup> for grey and white matter, respectively, over a five year period in a sample aged 59–85 years. None of the previous studies has considered the heterogeneity of the neural changes in older adult samples. In a supplementary analysis (Appendix 6), the older adult group was divided into two groups: young-old (age range 60-69 years) and old-old (age range 70+ years), based on a median split. No differences were found between the two older adult groups on ERN or Pe amplitudes for either condition. Additionally no behavioural differences between the older groups were found. However, and more importantly, an ANOVA model found similar results to those reported (main effects of component and group) with significant group differences

between the younger adults and the 'old-old'. These findings suggest that the age-group differences reported within the literature may be driven by significant amplitude decreases (perhaps *accelerating* decreases) in adults aged over 70 years. The ERN and Pe may be sensitive to maturational change, and thus may provide predictive markers of future cognitive decline. A longitudinal study of ageing would be a worthwhile avenue for future investigation; no ERN studies of ageing have utilised this approach to date, but such a technique would be particularly informative to track change in healthy ageing, the progression from mild cognitive impairment to AD, and from mild to moderate disease severity in older adults with dementia.

The ability to monitor performance requires the integration of sub-processes; when errors are detected it is conceivable that the system signals the requirement for remedial actions, otherwise the purpose of an error-correction system would be unclear. The current study found that older adults had similar timing and detection of pre-response conflict, partially reduced and delayed attention to stimulus-processing, and response priming. The SW component was delayed, but not attenuated. A component with similar morphology is found within the task switching literature. This component is associated with intentional set switching; a change in the selection of motor responses. The component is maximal at central sites and occurs between 520-1080ms (Rushworth, Passingham & Nobre, 2002; 2005). The SW did not differ between conditions and, as the stimuli have equal probability, the SW in the current study may represent the preparation for the switch between condition responses. The similar amplitude to younger adults indicated that the process underlying this component, although delayed, was not attenuated with age. Although the older adults may have had a degree of attentional deficit, as indexed by the P3b, plus reduced and delayed response priming, performance was equivalent. The ERN and Pe were also significantly reduced, indicating that the error detection and processing system were impoverished, yet functional. These attenuations cannot be explained by a global reduction in amplitude in older adults, as there are not consistent findings of amplitude reductions in the stimulus-locked components. It may be considered that neural changes within the frontal lobes, particularly affecting frontal lobe white matter connectivity (Hogan et al, 2006), impact negatively on the signal transfer from lateral PFC to ACC. van Veen and Carter (2002) located a similar dipole for the N2 and the ERN, and the lack of age effects on N2 amplitude suggests that the ERN reduction may not be fully

accounted for by factors influencing signal propagation: myelin degeneration (Bartzokis, 2004); cell shrinkage (Raz, 2004); or increases in cerebrospinal spaces (Good et al., 2001; Raz, 2000). However, the lack of structural data limits such conclusions, as it is not known whether the older adult sample had any significant neural change. That there were no consistent relationships between the ERN, Pe and remedial actions in both groups, suggests that other processes are involved in signalling the necessitation of immediate corrective behaviours. The current study also found some evidence of response strategies in older adults; reducing response speed and responding to fewer trials ('taking small breaks'). Of interest, the improvement in performance over time in the older adults indicated that their response strategy was effective.

In conclusion, there is evidence to suggest an alteration of neural activity underlying performance-monitoring, but this does not reach a threshold for functional impairment. Assuming that there is a relationship between awareness (internalised unconscious and/or overtly conscious) and ERP correlates of performance-monitoring, it may therefore be concluded that there is also evidence of altered Level 3 – Consistent Consciousness in normal ageing. Importantly, the slower response strategy adopted by older adults may be viewed as compensatory; different but not necessarily abnormal. In other words: slower may facilitate surer.

#### 5.4.1. Summary.

The current study investigated Consistent Consciousness (Stuss et al., 2001), a type of awareness dependent on the ability to organise sensory information and executive functioning underlying effective and adaptive cognition and behaviour. Performance monitoring was proposed to provide a conceptualisation of this type of awareness. Ageing was associated with some deficits in Consistent Consciousness: attention (stimulus processing), error detection and error processing. A possible explanation for similar performance is that age-related attenuations in ERP correlates of performance monitoring are not sufficient negatively to impact on functioning (Falkenstein et al., 2001). However, effective and adaptive remedial

actions and response strategies were found, providing evidence of compensatory mechanisms for impaired underlying processes in older adults.

## Chapter 6. Awareness Assessed by Measures of Metamemory and Self-Report of Functioning.

Metacognition is the study of self-reflected aspects of cognition; the reasoning and consideration of one's own thinking and memory, as well as the executive processes that facilitate the selection of strategies and control the allocation of processing resources (Wellman, 1983). Metacognitive research has traditionally focused on three methods of measurement: self-evaluation of memory functioning off-line; self-evaluation of memory functioning during task performance (on-line); and, monitoring of memory performance (Kausler, 1991). Off-line, self-evaluation of memory includes the participant's perceptions of the efficiency and general functioning of their memory abilities and is typically assessed using questionnaires. This includes assessment of metamemory beliefs. This type of metacognitive performance is closely related to awareness; indeed, Hertzog and Dixon (1994) argue that they are analogous - that evaluation of performance is likely to involve the focussing of attention combined with awareness of one's abilities. In this regard, this type of metacognitive ability is similar to Self-Awareness, the highest level of the Hierarchies of Processing (HoP) model defined by Stuss et al. (2001). A number of studies has reported changes in self-perceptions of memory abilities with age. Older adults reportedly perceive reduced memory capabilities, more decline and less internalised control over their memory (Dixon & Hultsch, 1983; Hertzog, Dixon, Schulenberg & Hultsch, 1987; Hultsch, Hertzog & Dixon, 1987).

The aim of this study was to assess Self-Awareness (cf. Level 4 of the HoP model, Stuss et al., 2001), and to extend current knowledge of metamemory beliefs in ageing by using multiple self-report measures of abilities, focusing on memory knowledge and capabilities, memory functioning in demanding situations, and cognitive and behavioural changes. Awareness and reporting of personal abilities may be influenced by level of cognition, socially desirable responding, and/or affect (e.g. Derouesné et al., 1999; Kashiwa et al., 2005; Rabbitt & Abson, 1991), and the current study examined the effect of these potentially confounding variables.

### 6.1. Introduction.

#### 6.1.1. Assessment of Metamemory in Ageing.

The measurement of metamemory beliefs has a long history in the metacognitive literature, and typically takes the form of questionnaires. The Metamemory In Adulthood questionnaire (MIA; Dixon et al., 1988) was designed as a multidimensional measure of self-referent beliefs about memory functioning and knowledge of memory processes (see Table 6.1). There are consistent findings of age-related differences in some of the MIA subscales: Capacity, Change, and Locus (Dixon & Hultsch, 1983; Hertzog, Dixon, Schulenberg & Hultsch, 1987; Hultsch, Hertzog & Dixon, 1987), indicating that older adults perceive that they have reduced memory capabilities, more memory decline over time, and less internalised control over their memory. However, there are mixed results with regard to the Strategy subscale. Only Hultsch et al. (1987), for example, found that older adults reported employing more strategies to support their memory performance than younger adults. Dixon and Hultsch (1983) identified two subcomponents of the Strategy subscale: Retrieval (internal mnemonic strategies) and Physical Reminders (external memory aids), and observed numerical differences in that older adults used Physical Reminders to a greater extent and Retrieval to a lesser extent compared to younger adults. However, McDonald-Miszczak et al. (1995), who also split the Strategy subscale into its subcomponents, did not find any age differences in either component. Although Hultsch et al. (1987) found that older adults employed more strategies to support their memory, the Strategy subscale was not separated into its subcomponents, limiting knowledge about specific strategy use in older adults. The authors suggested that further investigation of strategy use in older adults was warranted.

Despite the work described above, there has been little further investigation of metamemory beliefs in ageing in the last decade (however see Verhaeghen, Geraerts & Marcoen, 2000). Arguably, these beliefs might have changed given that engagement in cognitively demanding activities has been shown to protect against cognitive decline in older age (Coyle, 2003; Hultsch, Hertzog, Small & Dixon, 1999), and there has been increased public awareness of the benefits of a healthy lifestyle. Increased knowledge of protective factors may influence general beliefs about cognition and ageing, termed implicit theories (Hertzog & Hultsch, 2000), which may impact on metamemory beliefs. It was, therefore, of interest to examine whether self-perceptions of memory have changed since the MIA studies reported by Hultsch

and colleagues, as it is possible that implicit theories of ageing and cognition have changed since that time.

Table 6.1. The Dimensions of the Metamemory In Adulthood (MIA) Instrument.

<u>Dimension</u>	<u>Description</u>
Achievement (range 16-80)	Perceived importance of having a good memory and performing well on memory tasks (+ = high achievement).
Anxiety (range 14-70)	Feelings of stress related to memory performance (+ = high anxiety).
Capacity (range 17-85)	Perception of memory capacities as evidenced by predictive report of performance on given tasks (+ = high capacity).
Change (range 18-90)	Perception of memory abilities as generally stable or subject to long-term decline (+ = stability).
Locus (range 9-45)	Perceived control over remembering abilities (+ = greater internal control).
Strategy (range 18-90)	Knowledge and use of information about one's remembering abilities such that performance in given instances is potentially improved (+ = high use).
Task (range 15-75)	Knowledge of basic memory processes (+ = high knowledge).

*Note.* Based on Dixon and Hultsch (1983). + = Higher scores.

Dixon and Hultsch (1983) employed three samples to validate the MIA. The first sample was composed of 60 younger and 60 older adults (18-37 yrs and 50-81 yrs, respectively). Sample 2 included 50 younger, 50 middle aged and 50 older adults (21-39 yrs, 40-58 yrs, and 60-84 yrs, respectively); and, Sample 3 included 36 younger, 36 middle aged and 36 older adults (21-39 yrs, 40-58 yrs, and 60-74 yrs, respectively). There were no significant differences in the demographic data provided for each of the groups and samples; for example, years of education and vocabulary scores on the Vocabulary Test 1 and the Advanced Vocabulary Test from the Kit of Factor-Referenced Cognitive Tests were equivalent. Participants rated their overall health, eyesight and hearing as 'moderately good' or 'better', and the groups did not

significantly differ on self-reported health status. The results from Sample 1 are considered first. Younger adults had greater knowledge of memory processes (Task), perceived their memory capabilities to be better (Capacity) and their memory function as stable (Change) compared to older adults. A trend ( $p < .07$ ) for an age difference in perceived control over memory (Locus) was also found. Scores obtained from Sample 2 replicated the findings of age-related differences in Capacity and Change subscales between younger and older adults and indicated, further, that older adults perceive more memory decline with time compared to middle aged and younger adults. Consistent with these findings, an age difference between younger and older adults (Sample 2) was found on the Locus subscale, indicating that older adults perceived less internalised control over their memory. Significant effects of age were also found on the Task, Capacity and Locus subscales in Sample 3. The relatively consistent findings indicated that the age differences were robust. Dixon and Hultsch (1983) reported that the Capacity and Change subscales were positively correlated. They suggested that older adults had an awareness of possible memory decline with age and perceived this outcome as a given, which is compatible with the perception of reduced control over memory in this population.

In a later validation study, Hultsch et al. (1987) included 360 participants from Victoria (British Columbia, Canada) aged 20-78 years, separated into four age groups (20-26, 55-61, 62-68, 69-78), and 415 participants from Annville (Pennsylvania, USA) aged 20-78 years divided into seven age groups (20-33, 34-40, 41-47, 48-54, 55-61, 62-68, 69-78). Data from the Victoria sample were analysed using multivariate analysis of variance and revealed that older adults perceived significantly less memory capability, and less control over their memory, at the same time as they perceived significantly more memory decline and reported greater strategy use compared to younger adults. The younger adults reported significantly greater perceptions of memory capacity and less change in memory functioning compared to all of the older adult groups. The younger adults also reported lower strategy use than the 55-61 year old group, and higher control over memory than the 69-78 year old group. The only difference between the older adult groups was that the oldest group reported less control over memory than the 55-61 year olds. As the Annville sample was composed of a continuum of chronological age, a hierarchical regression analysis was conducted with year of birth to

identify age-related trends. The results from the Annville sample replicated the previous findings from the Victoria sample, in that older adults perceived reduced memory capacity and more decline, with a trend for older adults to perceive reduced control over their memory. Although there were no group differences in education, vocabulary or self-reported health in either sample, the younger adults in the Annville sample were recruited from the community and did not have a University education, indicating that age differences in Capacity, Change and Locus perceptions were not driven by educational status. Hultsch and colleagues concluded that the greatest age-related metamemory differences were between younger University students and older community-based adults. There were generally no significant differences between the older age groups in the Victoria sample, but linear age-related trends were found in the Annville sample. To explore this difference in greater detail, the authors conducted a similar, hierarchical regression analysis with year of birth from the older adults aged 55-78 years in the Victoria sample. This analysis failed to find any linear trends in metamemory beliefs, indicating that findings of significant metamemory differences in this age range may be variable. A possible interpretation is that metamemory perceptions do not differ between the young-old and old-old, suggesting that these perceptions become entrenched at a relatively early age (~ 55 yrs) and may remain stable over time (see also McDonald- Miszczak et al., 1995). In support, McDougall (1994) did not find any relationships between age and metamemory beliefs in a community-based sample of 169 adults aged 55 years and over. However, older adults aged 65-79 years reported greater use of strategies to support their memory performance compared to the older adults aged 55-65 years (McDougall, 1994).

#### 6.1.2. Confounding factors.

Differences in metamemory perceptions in ageing have consistently been found (e.g. Dixon & Hultsch, 1983; Hertzog et al., 1987; Hultsch et al., 1987), but there has been little consideration of potentially confounding factors which may contribute to self-assessment of cognitive functioning. A notable exception is the study by Verhaeghen et al. (2000), which found that anxiety about memory, as measured by the MIA Anxiety subscale, mediated the

relationship between self-reported memory complaints and coping behaviour. Moreover, McDougall (1995) found that depression was related to perceptions of greater change in memory over time. In support, Rabbitt and Abson (1990, 1991) found that self-assessment of functioning using the Cognitive Failures Questionnaire (CFQ) was positively correlated with Beck depression scores ( $r = .33$  and  $r = .15$ ; Rabbitt & Abson, 1990, 1991, respectively). However, the CFQ differs from the MIA in that the former focuses on the occurrence and frequency of cognitive problems and failures, whereas the MIA requires assessment of memory knowledge and abilities. Rabbitt and Abson's (1990; 1991) work is, nevertheless, important, as it emphasises the necessity of screening for level of fluid intelligence, affect and self-regard, which are all variables that may contribute to self-assessment of functioning.

In summary, previous studies have found age-related differences in the MIA subscales of Capacity, Change, Locus and Strategy (Dixon and Hultsch, 1983; Hertzog et al., 1987; Hultsch et al., 1987). The Strategy subscale has been shown to have two subcomponents (Dixon & Hultsch, 1983; internal mnemonic strategies and external memory aids) with younger adults relying more on internal strategies, whereas older adults utilise external aids to a greater extent. There is some evidence that affect may contribute to self-assessment of functioning (Rabbitt & Abson, 1990, 1991) and it has been suggested that awareness deficits may be related to self-esteem (Clare, 2004a).

### 6.1.3. Clinical Approaches to Assessing Awareness of Functioning.

The clinical literature has focused on impairments in awareness of specific functions, such as memory functioning (Eslinger et al., 2005; Vasterling et al., 1995), and cognitive and behavioural functioning (Migliorelli et al., 1995; Starkstein et al., 1996). One of the methods commonly employed is the calculation of discrepancy scores between patients and their informants. For example, Vasterling, Seltzer, Foss and Vanderbrook (1995) utilised a general questionnaire with items assessing memory, health, self-care, anxiety, depression and irritability. Anosognosia questionnaires have also been developed focusing on memory, cognitive and behavioural deficits (e.g. Migliorelli et al., 1995). Larger discrepancy scores are

interpreted as indicating awareness deficits in the patient. Discrepancy scores can either be positive or negative: positive discrepancies may be obtained when the informant reports greater impairment than the patient (anosognosia), whereas negative discrepancies reflect the converse - the patient reporting greater deficit than their informant (“hypergnosia”: Michon, Deweer, Pillon, Agid & Dubois, 1994, p. 807). Informant reports can be influenced by factors such as carer burden (DeBettingnies, Mahurin & Pirozzolo, 1990; Seltzer, Vasterling, Yoder & Thompson, 1997), tolerance and understanding of the effects of illness; and patients’ overestimation of impairments may be related to depression (Michon et al., 1994). In addition, it may not always be assumed that limited awareness is a sign of deficit, as an individual may adopt limited awareness as a coping mechanism that prevents them from having to contend with something they would rather avoid. Indeed, Clare (2004a) indicated that impairments in awareness may serve a protective function to the self-esteem of the patient and found that denial of deficits was common in individuals with AD. Self-esteem was not described with regard to the biopsychosocial model that Clare (2004b) developed, rather it was presented as a possible explanation for why patients may deny impairments in awareness. Clare (2004b) suggested that denial of deficits may serve to protect self-esteem; that is, there would be no need for the individual to adjust their self-perceptions if they had not acknowledged that they were experiencing any problems. Although impairments in awareness are found in individuals with diseases of ageing (e.g. AD and Parkinson’s disease), and with brain injury (Section 1.3.4., Chapter 1), it is not clear the proportion of ‘deficit’ in awareness that is related to ageing, level of cognitive function, and the individual’s own coping strategy. It may also be considered that these factors are inter-related and that it is not helpful to try and separate them. Perhaps of greater importance is the lack of studies with appropriate control groups. Few studies employ older adult control samples (however, see e.g. Eslinger et al., 2005); therefore, there is a lack of evidence identifying patterns of discrepancy scores on measures of awareness in healthy ageing.

#### 6.1.4. Aims of the Study.

The study aimed to investigate Level 4 – Self-Awareness of the HoP model (Stuss et al., 2001) and explored age-related changes in awareness in three complementary ways. Using the MIA

as a measure of metamemory beliefs, the study aimed to replicate previous findings of age-related differences in the Capacity, Change, Locus and Strategy subscales. As the Strategy subscale of the MIA can be separated into two subcomponents, and there is some evidence to suggest that there are age-related differences in types of strategies used to support memory, internal strategies and external aids were also investigated. Furthermore, two additional self-report measures of awareness of functioning were administered. The Memory Awareness Rating Scale (MARS; Clare, Wilson, Carter, Roth & Hodges, 2002) assesses awareness of the functioning of memory in demanding situations. The DEX questionnaire from the Behavioural Assessment of the Dysexecutive Syndrome battery (BADS; Wilson, Alderman, Burgess, Emslie & Evans, 1996), addresses both cognitive and behavioural changes. Both the MARS and the DEX have informant versions. The aim was to determine whether changes in metamemory were related to awareness of changes in memory more generally, and to changes in general cognitive and behavioural functioning, and to explore how these related to informant reports in older adults.

The literature suggests that awareness of function is related to anxiety, depression and self-esteem (Clare 2004a; Rabbitt & Abson, 1991), and relationships between these variables and the awareness questionnaires were explored in greater detail. The potentially confounding variables of cognitive function, self-esteem and socially desirable responding were also considered. Discrepancy scores between the older adults and their informants were calculated for the DEX and the MARS to examine patterns of awareness of functioning in healthy older adults. In order to assess Self-Awareness (cf. Level 4 of the HoP model; Stuss et al., 2001), which is supported by the monitoring of personal abilities, age-effects on awareness of functioning were examined. Specifically, the following hypothesis was tested:

1. Older and younger adults differ in awareness functioning as assessed by MIA subscales, and by the MARS and DEX, and this may be related to differences in anxiety, self-esteem and/or level of cognitive function.

In order to explore the relationship between domains of awareness functioning assessed by the MIA, and possible differences in this profile between older and younger adults, a number of more specific hypotheses were tested:

2. Age-related differences occur in the Capacity, Change, Locus and Strategy subscales of the MIA, but not in the Achievement or Anxiety subscales.
3. A significant, positive association would be found between perception of change in memory functioning (MIA Change) and perception of general memory capacity (MIA Capacity).
4. Perception of change in memory functioning (MIA Change) and self-reported strategy use (MIA Strategy) would be negatively correlated.
5. The MIA Anxiety subscale would correlate negatively with: (i) perceptions of memory capacity (MIA Capacity), and, (ii) perceptions of *change* in memory capacity (MIA Change), in the older but not younger adults.

## 6.2. Method.

### 6.2.1. Participants.

Thirty older adults (OA: *M* Age 69.4 years, *SD* 6.7; 10 male and 20 female) and nineteen younger adults (YA: *M* Age 21.9 years, *SD* 4.3; 4 male and 15 female) completed the questionnaire battery. The OA were requested to provide an informant and 24 OA informants additionally participated in the study. An informant was a person (e.g. spouse, significant other, or friend) that the OA participant consented to being approached.

In the current study, the age range of the older adult sample exceeded two decades, thus, based on findings of metamemory differences between older adult groups (see Hultsch et al., 1987), the older sample was divided into young-old (60-69 years) and old-old (70-84 years) groups.

## 6.2.2. Measures.

### 6.2.2.1. Cognitive Measures:

i) Ravens' Standard Progressive Matrices Sets A to D (RSPM; Raven, Raven & Court, 2000). The RSPM provides a measure of fluid intelligence. Sets A to D were administered in order and the participants were advised that the sets would increase in difficulty. As in other studies, Set E was excluded due to time constraints (e.g. Caffarra, Vezzadini, Zonato, Copelli & Venneri, 2003). The maximum possible score is 48 (Sets A-D).

### 6.2.2.2. Affective Measures:

i) Rosenberg Self-Esteem Scale (R-SES; Rosenberg, 1965). The questionnaire is composed of 10 items with a six-point rating scale. The R-SES has high internal consistency ( $\alpha = .88$ ; Greenberger et al., 2003). Higher scores indicate higher levels of self-esteem, with a maximum score of 60.

ii) The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) is composed of 14 items and provides measures of anxiety and depression. It is widely used with healthy and patient populations. Both the Anxiety and Depression scales have high internal consistency:  $\alpha = .80$  and  $.76$ , respectively (Mykletun, Stordal & Dahl, 2001). In the present study, these scales were completed by the individual without assistance from the examiner. Higher scores indicate higher levels of anxiety and depression, with a maximum score of 21 per scale. A score of 0-7 is considered to be within the normal range, clinically significant scores are 11+.

iii) Shortened Marlowe-Crowne Social Desirability Scale (M-C 1; Strahan & Gerbasi, 1972). The M-C 1 is actually a shortened version of the Marlowe-Crowne Social Desirability Scale (M-C SDS; Crowne & Marlowe, 1960). It uses a dichotomous rating scale of true or false for

10 statements reflecting highly desirable or undesirable properties, e.g. 'I like to gossip at times'. The M-C SDS has high internal consistency ( $\alpha = .88$ ; Crowne & Marlowe, 1960), and the shortened version is highly correlated with the original version ( $r = .80$ ; Strahan & Gerbasi, 1972). The M-C 1 was administered to the younger and older participants, and to the OA informants. By having informants complete the scale it was also possible to assess the degree to which they gave socially desirable responses on the informant questionnaires. There has been little consideration of psychological factors that may potentially influence informant's reports of functioning. This scale had a maximum score of 10, with higher scores indicating greater socially desirable responding.

#### 6.2.2.3. Self-Report Measures of Awareness:

i) Metamemory In Adulthood (MIA; Dixon, Hultsch & Hertzog, 1988). The MIA is a 108 item self-report questionnaire investigating self-referent beliefs. The questionnaire provides seven subscales (see Table 4.1). The MIA subscales have high internal consistency, with  $\alpha$  values ranging from .71 to .93 (Dixon et al., 1988).

ii) DEX questionnaire from the Behavioural Assessment of the Dysexecutive Syndrome battery (BADS; Wilson, Alderman, Burgess, Emslie & Evans, 1996). The BADS has been validated in 16-87 year olds, and is considered to be sensitive to frontal lobe functioning (Wilson et al, 1996). The self and informant versions of the DEX have 20 items regarding emotional or personality change, motivational, behavioural and cognitive changes (score range 0-80). Reliability and validity data are not provided in the BADS manual. However, a significant relationship was found between informant DEX scores and total BADS score ( $r = -.62$ ; Wilson, Evans, Emslie, Alderman & Burgess, 1998), indicating that informants' assessments of problems were associated with worse performance on the BADS by the participant. OA informants were administered the informant version. Discrepancy scores are calculated and may be positive, indicating that participant scores are lower than informants', or negative, indicating that participant scores are higher than informants'. Larger positive scores are indicative of greater difficulties with awareness of deficit in the participant, but

such scores must be interpreted cautiously due to the possibility that the informant's rating is influenced by carer-burden. Larger negative scores may also suggest altered awareness of deficit, or overestimation of deficit based on other factors such as low self-esteem. DEX discrepancy scores, nonetheless, provide important information related to individual awareness.

iii) The Memory Awareness Rating Scale (MARS; Clare, Wilson, Carter, Roth & Hodges, 2002) was specifically designed to assess memory awareness in dementia. There are self and informant versions of a 10 item questionnaire investigating performance in different situations dependent on memory abilities (score range 0-52). Responses relate to management in the given situations, for example, 'you have made an appointment and you need to remember to go along' and range on a scale from always to never. Lower scores represent greater problems managing in the given situations, and perhaps therefore better awareness of functional limitation. The scale has high internal consistency ( $\alpha = .94$ ) and a high test-retest reliability ( $r = .91$ ; Clare et al., 2002). The participant version is administered as an interview, whereas the informant version is given as a questionnaire. OA informants were administered the informant version. Discrepancy scores are calculated and can also either be positive: participant scores are higher than informants', or negative: participant scores are lower than informants'. Larger positive and negative scores are indicative of greater difficulties with awareness in the participant, although the same considerations as those described for the DEX undoubtedly apply.

### 6.2.3. Procedure.

The questionnaire battery was administered alongside the demographic questionnaires (see Chapter 3). These data were collected prior to the ERP session. With the exception of one older adult, the older adults were assessed in their homes and the younger adults at the School of Psychology, University of Southampton. The order of the questionnaires is given in Section 3.8 Chapter 3 and took approximately 2 hours to complete. One older adult did not complete the MIA.

#### 6.2.4. Statistical Analysis.

Outliers were identified with box-plots and scores outside of the normal range were excluded from the analyses, as such, one OA was excluded from the RSES, one YA from the HADS Anxiety scale, and one YA from the HADS Depression scale. Shapiro-Wilk tests were then conducted on all variables to assess fit with the normal distribution. All data were normally distributed ( $p > .05$ ). In addition, testing of the measures of awareness did not reveal any violations of linearity, homogeneity of variance or multicollinearity. The RSPM, RSES, HADS Anxiety and Depression scales were analysed with Independent Samples t-tests, as were the DEX and the MARS between the OA and their informants. Social desirability between the two groups and the OA informants was investigated using a one-way analysis of variance. A series of MANOVAs was conducted comparing age groups on the measures of awareness (MIA subscales, DEX and MARS). MANOVAs were also performed on the subscales of the Strategy scale, and between the young-old and old-old groups on the awareness measures. As depression was found to differ between the groups, a MANCOVA was conducted to assess the effects of depression and group on the awareness measures. Anxiety and self-esteem were also included in MANCOVA models to assess the effects these variables had on awareness. Bivariate correlations were performed between the MIA subscales for each group, between the awareness measures and the social desirability scores for each group, and between the RSPM scores, a measure of fluid intelligence, and the measures of awareness. Bonferonni corrections were used to control family-wise error rate for unplanned, post hoc comparisons following the analyses of variance. Corrections were not used for exploratory correlations given the increased risk of Type 2 error (Perneger, 1998; Rothman, 1990).

### 6.3. Results.

#### 6.3.1. Measures of Cognition and Affect.

Mean scores and standard deviations are presented in Table 6.2. There was a significant difference between the groups on the HADS depression scale:  $t(44.5) = -3.45, p = .001$ , indicating that younger adults reported lower levels of depression. However, the ranges of HADS depression scores were similar; 2-8 (YA) and 3-9 (OA). None of the scores were within the clinical range.

#### 6.3.2. Measures of Awareness.

A MANOVA was conducted to investigate age differences in self-report of awareness of functioning (MIA subscales, MARS, DEX). There were significant differences between OA and YA on the combined dependent measures of awareness: Wilks'  $\Lambda = .48$ , multivariate  $F(9, 37) = 4.54, p < .001$ , partial  $\eta^2 = .53$ . Univariate tests indicated significant age differences on three of the MIA subscales: Change,  $F(1, 45) = 21.24, p < .001$ ; Locus,  $F(1, 45) = 5.38, p = .025$ ; and Strategy,  $F(1, 45) = 5.30, p = .026$ , but not on the MARS or DEX. These findings indicate that OAs perceived their memory to decline over time, reported less internalised personal control and reported utilising more strategies to improve their memory performance. The Strategy subscale was divided into the subcomponents Internal strategies and External aids further to investigate these age differences. As there were no specific hypotheses about age differences in the subcomponents, due to inconsistencies in previous findings, the alpha level was adjusted ( $p = .025$ ). There was a significant difference between the groups: Wilks'  $\Lambda = .70$ , multivariate  $F(2, 45) = 9.71, p < .001$ , partial  $\eta^2 = .30$ . The External subcomponent was found to differ between the groups:  $F(1, 46) = 19.17, p < .001$ . This result indicates that older adults utilised more external memory aids compared to younger adults to support their

Table 6.2. Mean scores (standard deviation) for questionnaire battery obtained from younger and older adults.

<u>Measures</u>		<u>Younger</u>	<u>Older</u>
		<i>N</i> = 19	<i>N</i> = 30
Cognitive	RSPM	40.5 (3.5)	39.4 (4.2)
Affect	RSES	49.6 (7.8)	49.9 (5.4)
	HADS Anxiety	7.0 (2.2)	7.8 (2.7)
	HADS Depression	4.3 (1.4)	6.0 (2.0) <sup>a</sup>
	MC-1	5.3 (2.3)	5.4 (1.9)
	MC-1 Informant	-	6.4 (1.1)
Awareness	MIA: Achievement	58.5 (8.5)	58.6 (6.3)
	MIA: Anxiety	42.3 (7.5)	40.9 (9.1)
	MIA: Capacity	54.7 (7.3)	51.6 (8.7)
	MIA: Change	60.4 (8.1)	46.1 (11.4) <sup>b</sup>
	MIA: Locus	30.3 (4.0)	27.7 (4.4) <sup>b</sup>
	MIA: Strategy	58.9 (7.2)	64.7 (7.5) <sup>b</sup>
	MIA: Internal	39.7 (5.5)	40.4 (5.0)
	MIA: External	19.3 (4.2)	24.3 (3.7) <sup>b</sup>
	MIA: Task	57.7 (5.8)	57.3 (4.9)
	DEX	21.6 (9.1)	20.8 (7.9)
	DEX Informant	-	13.2 (8.7) <sup>a</sup>
	DEX Discrepancy	-	-7.7 (10.5)
	MARS	40.6 (6.2)	42.0 (7.5)
	MARS Informant	-	41.1 (8.7)
	MARS Discrepancy	-	1.5 (10.2)

*Note.* <sup>a</sup>  $p < .05$  (Independent Samples t-tests): YA vs. OA and OA vs. Informant. Informant  $n = 24$ ; <sup>b</sup>  $p < .05$  (MANOVA); RSPM = Ravens' Standard Progressive Matrices; Raven, Raven & Court (2000); R-SES = Rosenberg Self-Esteem Scale; Rosenberg (1965); HADS = Hospital Anxiety and Depression Scale; Zigmond & Snaith (1983); MC-1 = Shortened Marlowe-Crowne Social Desirability Scale; Strahan & Gerbasi (1972); MIA = Metamemory In Adulthood; Dixon, Hultsch & Hertzog (1988); DEX questionnaire from the Behavioural Assessment of the Dysexecutive Syndrome battery; Wilson, Alderman, Burgess, Emslie & Evans (1996); MARS = Memory Awareness Rating Scale; Clare, Wilson, Carter, Roth & Hodges (2002)

memory performance, and this difference was driving the finding of group differences on the overall Strategy subscale. The MANOVA investigating age-related differences within the OA group on the measures of awareness was not significant: Wilks'  $\Lambda = .68$ , multivariate  $F(9, 18) = .92$ ,  $p = .529$ , partial  $\eta^2 = .32$ , indicating that there were no significant differences between the young-old and old-old participants across the combined measures of awareness.

#### 6.3.2.1. Influence of mood and self esteem.

One of the aims of the study was to explore the potential effects of self-esteem and anxiety on awareness. The MANCOVA examining the effects of self-esteem was not significant (Wilks'  $\Lambda = .67$ ), multivariate  $F(9, 35) = 1.95$ ,  $p = .076$ , partial  $\eta^2 = .33$ , suggesting that self-esteem was not related to self-reported awareness of functioning. However, the multivariate main effect of group remained significant, with univariate analyses replicating the group effects reported above, indicating that self-esteem was not related to the age differences found on the Change, Locus and Strategy subscales. The MANCOVA investigating the effect of anxiety revealed a consistent main effect of age group and an effect of the covariate HADS Anxiety: Wilks'  $\Lambda = .47$ , multivariate  $F(9, 35) = 4.35$ ,  $p = .001$ , Wilks'  $\Lambda = .54$ , multivariate  $F(9, 35) = 3.28$ ,  $p = .005$ , partial  $\eta^2 = .46$ , group and anxiety, respectively. This indicates that anxiety was significantly related to self-report of awareness and suggests that HADS Anxiety and age independently influenced MIA subscale scores. The univariate analyses of group effects replicated the findings above indicating that, after controlling for anxiety, the group differences found in Change, Locus and Strategy were not related to anxiety. The univariate tests revealed relationships between anxiety and three of the MIA subscales and the DEX: Anxiety,  $F(1, 43) = 23.76$ ,  $p < .001$ ; Capacity,  $F(1, 43) = 7.85$ ,  $p = .008$ ; Change,  $F(1, 43) = 7.83$ ,  $p = .008$ ; and DEX,  $F(1, 43) = 5.88$ ,  $p = .020$ . Higher levels of anxiety were related to increased levels of stress about memory, the perception of more decline over time, reduced memory capacities and more problems with behavioural and cognitive functioning. Despite adjusting the alpha level for multiple comparisons ( $p = .006$ ), the relationship between anxiety and scores on the MIA subscales of Anxiety, Capacity and Change remained significant.

As level of depression differed between the age groups, a MANCOVA was also performed to investigate the effects of depression and age on the awareness measures, with depression as a covariate. Multivariate tests found that there were significant effects of both HADS Depression and group on the awareness measures: Wilks'  $\Lambda = .59$ , multivariate  $F(9, 35) = 2.76$ ,  $p = .015$ , partial  $\eta^2 = .42$ , Wilks'  $\Lambda = .52$ , multivariate  $F(9, 35) = 3.54$ ,  $p = .003$ , partial  $\eta^2 = .48$ , depression and group, respectively. This result indicates that after controlling for the effect of depression, age differences in the self-report of awareness remained. This also suggested that HADS Depression and age independently influenced MIA subscale scores. Univariate tests revealed significant differences between the groups on the Change subscale,  $F(1, 43) = 12.23$ ,  $p = .001$  and the Strategy subscale,  $F(1, 43) = 6.30$ ,  $p = .016$ . The age difference in the Locus subscale was no longer significant once the effect of depression was controlled, indicating that the age difference in control over memory functioning could be explained by the greater level of depression in the older adults. The univariate tests for depression found relationships with several MIA subscales: Anxiety:  $F(1, 43) = 11.26$ ,  $p = .002$ ; Capacity:  $F(1, 43) = 10.56$ ,  $p = .002$ ; Change:  $F(1, 43) = 9.30$ ,  $p = .004$ , the DEX:  $F(1, 43) = 6.43$ ,  $p = .015$  and the MARS:  $F(1, 43) = 6.56$ ,  $p = .014$ . Higher levels of depression were related to increased levels of anxiety/stress about memory, the perception of more decline over time, reduced memory capacities, more problems with memory abilities in demanding situations, and behavioural and cognitive functioning. These results also indicated that age-related differences in the DEX and MARS only became apparent when depression was controlled for. As before, despite adjusting for multiple comparisons ( $p = .006$ ), the relationship between age group, controlling for depression, and scores on the MIA subscales of Anxiety, Capacity and Change remained significant. However, the DEX and MARS age group differences were no longer significant.

### 6.3.3. Correlations.

Exploratory, bivariate correlations performed between each of the MIA scales are shown in Table 4.3. In the OA sample, the only significant correlations were between Anxiety and Change, and Capacity and Change (see Table 6.3). The Anxiety and Change correlation was

also significant in the YA group. These results indicate that in both YA and OA participants, as anxiety/stress over memory performance increased, the perception of memory stability over time decreased, and in the OA group, the perception of increased memory capabilities was also related to the belief that memory remained stable. Although the correlations between Anxiety and Capacity, and Change and Strategy were not significant, perhaps due to an insufficient sample size ( $n = 29$ ), the strength of each relationship was moderate ( $r = -.46$  and  $r = -.38$ , respectively), and accounted for 21% and 14% of the shared variance, respectively. These results indicate that as stress and anxiety over memory performance increased, the belief that memory is stable over time decreased. In the OA participants, greater reported use of strategies was related to the belief that memory declined over time.

The DEX and MARS were not related, and no relationships were found between the DEX and any of the MIA subscales in either group. However, the MIA subscale Capacity was related to the MARS in both groups:  $r(19) = .79, p < .001$  and  $r(29) = .56, p = .002$ , YA and OA groups, respectively. These results indicate that perceptions of better memory capabilities were associated with better self-reported performance in memory-demanding situations.

Post hoc bivariate correlations were conducted to assess the relationship between the perception of Change and the use of Internal mnemonic strategies and External aids in OA. The correlation between Change and the Internal subcomponent was significant:  $r(29) = -.39, p = .039$ , however, there was no significant relationship between Change and External aids in OA. That is, participants who perceived themselves as having experienced greater change in memory functioning over time also reported using more internal memory strategies.

Fluid intelligence was significantly related to Strategy and DEX scores in the older adults,  $r(29) = .40, p = .031$  and  $r(30) = -.49, p = .006$ , respectively, suggesting that higher levels of fluid intelligence were associated with greater strategy use, and with fewer reported problems with cognitive and behavioural functioning.

As socially desirable responding in each group of participants (YA, OA and OA informants) was a potential threat to the validity of the study, correlations with the measures of awareness

were also performed. Social desirability was not related to any of the awareness measures in either group.

#### 6.3.4. Patterns of Discrepancy Scores on Awareness of Functioning.

Independent t-tests were conducted on the DEX and the MARS, between the OA and their informants, to explore whether there were any differences between these groups in the reporting of cognitive and behavioural functioning. The means and standard deviations are provided in Table 6.2. A significant difference was found between the reporting of functioning on the DEX,  $t(52) = 3.39, p = .001$ , indicating that the OA reported more problems with cognitive and behavioural functioning than were provided by their informants. No differences were found with the MARS.

Table 6.3. Pearson product-moment correlations between the MIA subscales for older and younger adults.

	Achieve	Anxiety	Capacity	Change	Locus	Strategy	Task
Achieve		.38 (.14)	.15 (.02)	-.27 (.07)	.34 (.12)	.12 (.01)	.24 (.06)
Anxiety	.06 (.00)		-.46 (.21)	<b>-.65</b> † (.42)	-.01 (.00)	.20 (.04)	.26 (.07)
Capacity	.28 (.09)	-.54 (.29)		<b>.59</b> † (.35)	.36 (.13)	-.26 (.07)	-.08 (.01)
Change	-.05 (.00)	<b>-.59</b> † (.35)	.48 (.23)		.34 (.12)	-.38 (.14)	-.26 (.07)
Locus	.31 (.10)	.19 (.04)	.13 (.02)	.04 (.00)		-.09 (.01)	.04 (.00)
Strategy	.02 (.00)	.05 (.00)	-.23 (.05)	-.23 (.05)	-.13 (.02)		.35 (.12)
Task	.58 (.34)	.15 (.02)	.23 (.05)	.04 (.00)	.58 (.34)	.30 (.09)	

*Note.* OA ( $n = 29$ ) data above the line; YA ( $n = 19$ ) data below the line; \*  $p < .007$  (Bonferroni adjusted for multiple comparisons);  $R^2$  values in parentheses.

#### 6.4. Discussion.

The present study investigated Self-Awareness, the highest level of the HoP model (Stuss et al., 2001), in healthy older and younger adults using multiple measures of awareness of functioning. Awareness was assessed using self-report measures of memory knowledge and abilities (MIA), awareness of memory in demanding situations (MARS) and awareness of cognitive and behavioural changes (DEX). As predicted, differences were found in the Change, Locus and Strategy subscales of the MIA, supporting previous studies indicating that older adults perceive more memory decline over time, less internalised control over their memory and report utilising more strategies to support memory performance (Dixon & Hultsch, 1983; Hertzog et al., 1987; Hultsch et al., 1987). Furthermore, the current study found that older adults relied upon external memory aids to a greater extent than younger adults, providing support for the numerical trend indicated by Dixon and Hultsch (1983). Although perceptions of Capacity and Change were positively correlated in the older adults, in that greater self-reported memory capabilities were related to greater reported stability of memory functioning, the study failed to find an age-related difference in perceptions of memory capacity. It was expected that a perception of greater change in memory abilities over time would be related to increased use of strategies to help memory functioning in older adults. Although the correlation was not significant, possibly due to the small sample size, the relationship was modest and accounted for 14% of the variance. The G\* Power 3 calculation program (Faul, Erdfelder, Lang & Buchner, 2007) revealed that 12 additional participants, to provide a sample size of 41 older adults, would have been needed to find a relationship with an alpha level of .05, power .80.

Similar to Hultsch et al. (1987) and McDougall (1994), the current study failed to find any age-related differences in metamemory perceptions within the older adult sample. Despite equivalent awareness of cognitive and behavioural functioning to younger adults, the study found evidence of hypergnosia in the older adults, as they reported more cognitive and behavioural problems than were reported by their informants. This discrepancy between participant and informant was not related to depression in the older adults, or to socially desirable responding in the older adult informants. By contrast, the age group difference in

perceptions of control over memory could be accounted for by the higher levels of depression in the older than in younger adults. Additionally, general anxiety and depression, as measured by the HADS, were related to increased anxiety and stress over memory, reduced memory capabilities and more perceived change over time in memory functioning. Higher anxiety and stress about memory and greater perceptions of change in memory functioning were highly significant and positively correlated in both the older and younger adults. Modest correlations were also found in both groups between anxiety about memory and perceptions of memory capacity, such that lower perceptions of capacity were associated with greater anxieties about memory. In summary, these convergent findings indicate that there is an emotional component to awareness of memory functioning, providing support for Abson and Rabbitt (1990; 1991) and McDougall (1995).

Despite the relationship between affect (anxiety and depression) and metamemory beliefs, self-esteem and social desirability were not related to awareness of functioning in the current study. There were, however, modest correlations between greater fluid intelligence in the older adults and strategy use, and with fewer reported problems with cognition and behaviour, suggesting that level of cognition is related to the use of strategies to support memory functioning and awareness of cognitive and behavioural functioning. This is consistent with Verhaeghen et al. (2000) who found that anxiety and stress over memory mediated coping behaviours.

The present investigation extends our knowledge of metamemory in ageing in three important ways. Firstly, this study is one of the first to provide empirical evidence of the use of different memory strategies in healthy older adults; Hultsch et al. (1983) only found numerical trends for different types of strategy use with age. Secondly, although the older adults perceived greater memory decline with time, they reported similar memory capabilities as the younger adults. Thirdly, the age-related difference in perceived control over memory was explained by increased depression in the older adults.

The current study replicated the finding that older adults report utilising memory strategies to a greater extent than younger adults, and extended knowledge by providing evidence that older

adults rely on external memory aids more than younger adults. Dixon and Hultsch (1983) observed a trend for younger adults to utilise internal mnemonic strategies more than older adults. However, this finding was not supported with the present data, as the scores on this subcomponent were similar between groups. The expectation that the perception of memory decline over time would be related to increased use of strategies in older adults was supported by a moderate correlation. Further investigation of the Strategy subcomponents found that the perception of stability in memory abilities over time in older adults was related to the increased use of internal mnemonic strategies and not to external memory aids. These results indicated a relationship between a belief in memory stability with age and mental techniques, for example elaboration and rehearsal, to support memory functioning in older adults. This association warrants further investigation as it suggests, despite the greater reliance of the older adults on external memory aids compared to younger adults, that the use of mnemonic (internalised) techniques was related to positive perceptions of memory abilities over time. One implication of this finding is that the teaching of mnemonic strategies to older adults may contribute to more optimistic perceptions of memory functioning. It would also be of interest to assess whether the teaching of such skills influenced perceptions of control in older adults.

The replication of previous findings on the Change subscale suggests that perceptions of memory decline with age are prevalent in older adults. The current study also supports the proposition that perceptions of change in memory functioning may become entrenched at a relatively early age and may remain stable over time (e.g. Hultsch et al., 1987: Annville sample; McDonald-Miszczak et al., 1995; McDougall, 1994). However, contrasting results have been found by Hawley, Cherry, Su, Chiu and Jazwinski (2006). These authors, using the Knowledge of Memory Aging Questionnaire (KMAQ), found that older adults aged 80+ years, endorsed stereotyped views of normal memory ageing more often than did middle age adults (40-59 years), and young-old adults (60-79 years). However, the discrepant results may be methodological and could have arisen from differences between the focus of the questionnaires; stereotypical beliefs in the KMAQ and assessment of memory knowledge and abilities in the MIA. It would be of interest to explore any relationships between the two questionnaires, to assess potential overlap or whether the factors are complementary.

Nevertheless, the current study's consistent results with those of previous studies using the MIA, indicate that perceptions of memory decline with time in older adults prevail.

These findings may be viewed as providing some support for the influence of implicit theories of cognition and ageing on metamemory perceptions in ageing (see Hertzog & Hultsch, 2000). For example, the perception of memory decline may be related to an implicit belief that ageing is associated with decline; however, it may also be related to perceptions of *actual* change within the individual. As the current study was designed to assess awareness in ageing, rather than the *accuracy* of awareness of functioning in ageing, no measures of prediction and postdiction (traditionally used to assess accuracy: Section 2.6.2., Chapter 2) were administered. However, previous longitudinal studies (e.g. McDonald-Miszczak, Hertzog & Hultsch, 1995) have found a weak relationship between actual change in memory functioning and perceptions of memory decline with a longer duration of follow-up (six years).

It is striking that the older participants in this study did not perceive their memory capabilities (Capacity) as being poorer than those reported by the younger adults. Previous studies have found that older adults perceive their memory capabilities to be lower compared to younger adults (Dixon & Hultsch, 1983; Hultsch et al., 1987). There are a number of possible explanations for this discrepancy. The divergent results in this study indicate that whilst the older adults perceived that their memory will change over time, at the time of the assessment they rated their current abilities similarly to younger adults, suggesting a separation of beliefs relating to the present and the future in the older participants. The current younger and older adult samples were smaller than those previously reported, hence, the study may have been underpowered to detect any age differences in perceptions of memory capabilities. However, previous studies did not account for confounding variables. The current study found that anxiety and depression covaried with the MIA Capacity subscale, indicating that affect influenced perception of capabilities. Divergent results may, therefore, be due to previous studies not controlling for levels of affect. In addition, Stevens, Kaplan, Ponds, Diederiks and Jolles (1999) found that self-reported high levels of social and physical activity predicted the perception of better memory capabilities (Capacity). This provides support for the importance

of accounting for the possible confounding effects of more than just psychological factors on metamemory beliefs.

There were no age differences in any of the awareness measures specifically focusing on current functioning and abilities (Capacity, DEX and MARS), suggesting that awareness of abilities does not alter as a function of healthy ageing. As little is known about deficits in awareness in healthy ageing, the present study also contributes to this body of knowledge. This finding was not due to the groups failing to report any difficulties on any measure as both groups reported some problems with cognitive and/or behavioural functioning; suggesting that both younger and older adults were aware of limitations in their abilities. Furthermore, no positive discrepancies, presumed to reflect impaired awareness, were found between the older adults' reports and the assessments provided by their informants. In fact, the converse was found on the DEX questionnaire, in that the informants reported that the older adults presented with fewer problems with cognition and behaviour than the older adults perceived in themselves. This finding is not easily explained. Initially it was thought that socially desirable responding by the informants influenced their assessments; perhaps also a degree of denial that there might be any problem with their relative. However, social desirability was assessed within this group and these scores were not indicative of socially desirable response bias. It would be of interest to assess the prevalence of hypergnosia in normal ageing, particularly as studies have found that older adults underestimated their performance (Abson & Rabbitt, 1991; Ansell & Bucks, 2006). However, given that awareness in the current study was measured by self-report questionnaires, future studies would benefit from determining the accuracy of these perceptions by measuring prediction and postdiction of objective task performance. It would be of particular importance to involve informants in such predictions so as to be able to determine the degree to which any deficits in prediction accuracy might be a general function of poor judgement rather than a specific age-related deficit in self-awareness (Trosset & Kaszniak, 1996).

Hertzog and Hultsch (2000) suggested that metamemory beliefs in older adults may be related to "subjective well-being" (p. 459). Awareness, as indexed by metamemory perceptions, may contain an emotional component, and the present study has provided evidence for this

proposition. Firstly, depression in the older adults accounted for the age-related difference in perceived control over memory functioning. Previous studies using the MIA have rarely investigated the possible influence of affect on beliefs (however, see McDougall, 1995); therefore the current study extends knowledge regarding the variables that may contribute to metamemory perceptions in ageing. The study also offers support for Rabbitt and Abson (1990; 1991) who found that depression was related to perceptions of cognitive problems in older adults. Secondly, anxiety, as measured by the HADS, was related to increased levels of anxiety and stress about memory, the perception of more decline over time, and reduced memory capacities. Clare (2004a), based on an exhaustive review of anosognosia in AD, suggested that impairments in awareness may serve a protective function for self-esteem, in that patients may deny the existence or extent of problems to avoid having to accept and/or acknowledge decline. The present study found similar ratings of self-esteem between the groups, and that self-esteem was not related to the measures of awareness. However, the study was not designed to assess the protective effect of self-esteem, and an exploration of the relationship between self-esteem and awareness is restricted within the current study as ageing was not found to be associated with deficits in awareness of functioning, as measured by participant-informant discrepancy scores.

#### 6.4.1. Summary.

The current study investigated Self-Awareness (Stuss et al., 2001), a type of awareness dependent on the ability to monitor the self and personal abilities. These data suggest that ageing is not associated with significant impairments in awareness of functioning and level of ability, despite the older adults' perception of decline in memory function over time, which may be related more to societal expectation than based on significant levels of actual decline. Perhaps as may have been expected, the study has also shown that expressions of awareness have an emotional component. More generally, these findings contribute to the documentation of awareness in healthy normal ageing.

Chapter 7. A Multidimensional Assessment of the Hierarchies of Processing Model (Stuss et al., 2001).

This chapter builds on the studies reported in Chapters 4 to 6 by exploring the relationships between the different methods and measures used to assess awareness. As detailed in Chapter 2, it takes as its conceptual framework the Hierarchies of Processing (HoP) model developed by Stuss et al. (2001). Each level of the model contains a number of modules, the integrated functioning of which underlies the processes involved in the different types of awareness. Specifically, Stuss and colleagues (2001) argued that: i) the processes within each level can function independently of other levels (intra-connectivity); ii) the ability of the modules to transmit and receive input to and from higher and lower levels, provides the connectivity between the different levels (inter-connectivity). This description poses a challenge for the empirical assessment of the model, as it provides an explanation for evidence of relationships, but also for any null findings. A fundamental assumption of the present study is that higher levels of awareness are dependent, to a degree, on the functioning of lower levels; a view that is compatible with Stuss and colleagues' model (2001). The aim of this chapter is to consider this relationship by investigating associations between the measures of the different levels presented in previous chapters. By doing so it is hoped to address the proposed inter-connectivity in the model.

There is some precedence in the literature for exploring inter-connectivity between levels of awareness. An earlier study that utilised a multi-dimensional approach to assess awareness in adults with traumatic brain injury reported significant impairments in different types of awareness compared to controls (O'Keefe et al, 2007).<sup>1</sup> Of interest to the current study are the correlational analyses conducted to explore such relationships. O'Keefe et al. (2007) found that metacognitive knowledge about personal abilities was not related either to emergent or to anticipatory awareness. However, there was a highly significant relationship between emergent and anticipatory awareness ( $r = .72$ ), indicating that better monitoring of errors during a sustained attention task was associated with more accurate prediction of performance.

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<sup>1</sup> This study is introduced, alongside that of Togliola and Kirk (2000), in Chapter 1, page 34.

Each of the studies presented thus far in this thesis (Chapters 4, 5 and 6) has investigated different types of awareness in ageing. Tests were used that were operationalised to be related to the different levels of awareness in Stuss and colleagues' model. Although evidence was found of subtle differences between age groups (Table 7.1), results were interpreted to suggest intra-connectivity (within-level functioning) in the HoP model was generally similar in healthy younger and older adults. Importantly, this does not preclude statistical exploration of the inter-connectivity between Levels 2-4 of the HoP model within the two age-groups. Given that the participants were awake and interacting normally during testing, it was assumed that Level 1 – Arousal (a dimension from coma to waking state) was functional such that relationships with Level 1 were not explored. Simply stated, it was of additional interest to consider the possibility that the inter-connectivity between HoP levels differs between younger and older adults.

#### 7.1. Introduction.

The previous three chapters investigated Levels 2-4 of the HoP model (Stuss et al., 2001). The excellent temporal resolution, and sensitivity to experimental manipulations, of event-related potentials (ERPs) provided on-line measurement of the timing of cognitive processes. Although the spatial resolution of ERPs is poor, due to the volume conductance properties of the brain, intra-cranial recording of electrical activity using depth electrodes (e.g. Halgren et al., 1995), and dipole localisation (e.g. Senkowski et al., 2003; van Veen & Carter, 2002) studies have identified neural generators of the components measured at the surface of the scalp. Stuss and colleagues (2001) specified the neural candidature, and the cognitive processes involved in different types of awareness. The experimental ERP paradigms utilised to operationalise Levels 2 and 3 of the model, were considered to provide behavioural data corresponding to the processes within each level, and review of the literature indicated that the selected ERP components were associated with neural generators located in regions specified by the model for these levels.

Sensorimotor Awareness, at Level 2, relies on sensory processing within posterior regions (association cortices) of the brain, and was operationalised by the N1 (Chapter 4). More specifically, the N1 has been linked to initial, attentional capture of stimuli, and its neural generator has been located in the superior temporal plane (Halgren et al., 1995). The P3 component has been associated with the allocation of attention to the updating of pre-existing mental representations (Polich, 1996) and is generated by more widespread activity in the temporal lobes and prefrontal cortex (Halgren et al., 1995; Halgren et al., 1998). The novelty auditory oddball paradigm provided measurements of the degree and timing of attentional processing of sensory (auditory) information, including target recognition and novelty processing.

Consistent Consciousness, at Level 3, involves the ability to organise sensory information underlying behaviour and executive functioning, and was operationalised by assessing performance monitoring (Chapter 5). The ERP correlates of performance monitoring were the N2, ERN, CRN and Pe components, the neural generators of which are located within the frontal lobes: anterior cingulate cortex (van Veen & Carter, 2002), pre-supplementary motor area (Ullsperger & von Cramon, 2006), lateral prefrontal cortex (Gehring & Knight, 2000; Ullsperger & von Cramon, 2006), and white matter tracts connecting these regions (Hogan et al, 2006). Stimulus processing in the context of performance monitoring was also investigated using the P3b, generated by the ventrolateral prefrontal cortex, superior temporal sulcus and the hippocampus (Halgren et al., 1995; Halgren et al., 1998); due to the presence of 'Stroop-like' conflicting information in the 4-CRT paradigm, this P3 component represents more complex cognitive activity than that reflected in the novelty auditory oddball P3 component. In addition, the performance monitoring paradigm provided behavioural measures of reaction time, correct and error performance, and the remedial actions of error correction and post-error slowing.

The highest level of the model, Self-Awareness, Level 4, arises from the monitoring of the self and personal abilities (Chapter 6). This was assessed using self-report measures of awareness of cognitive and behavioural functioning, and metamemory perceptions. Chapter 6 also examined the influence of affect on metamemory perceptions. Interestingly, the HoP model

Table 7.1. Measures of awareness with age-related differences.

Level	Description	Chapter and task		Operationalised by	Age group differences			
1	Arousal	Presumed functional as all individuals awake.						
2	Sensorimotor Awareness	Chapter 4: Novelty Auditory	N1	Standard Target	N			
		Chapter 4: Oddball	P3	Standard	Y (Fz/Cz/Pz)			
				Target	N			
				Novel	Y (Fz/Cz/Pz)			
3	Consistent Consciousness	Chapter 5: 4-CRT	P3b	Compatible	N			
				Incompatible	Y			
			ERN	Compatible	N			
				Incompatible	Y			
			Pe	Compatible	Y			
				Incompatible	N			
			4	Self-Awareness	Chapter 6: Self-Report Questionnaires	MIA	Achievement subscale	N
							Anxiety subscale	N
Capacity subscale	N							
Change subscale	Y							
Locus subscale	Y							
Strategy subscale	Y							
Internal Strategy	N							
External Strategy	Y							
DEX	Task subscale	N						
	MARS	N						

*Note.* Y = Yes; N = No; Fz/Cz/Pz = location of observed age-group differences in amplitude; 4-CRT = four choice reaction time task; MIA = Metamemory In Adulthood, Dixon, Hultsch & Hertzog, 1988; Achievement = perceived importance of having a good memory and performing well on memory tasks; Anxiety = feelings of stress/anxiety related to memory performance; Capacity = perception of memory capacities as evidenced by predictive report of performance on given tasks; Change = perception of memory abilities as generally stable or subject to long-term decline; Locus = perceived control over remembering abilities; Strategy = knowledge and use of information about one's remembering abilities such that performance in given instances is potentially improved; Internal Strategy = use of mnemonic strategies; External Strategy = use of external memory aids; Task = knowledge of basic memory processes; DEX questionnaire from the Behavioural Assessment of the Dysexecutive Syndrome battery (BADS; Wilson, Alderman, Burgess, Emslie & Evans, 1996); MARS = Memory Awareness Rating Scale (Clare, Wilson, Carter, Roth & Hodges, 2002).

proposed that Self-Awareness is supported by the inter-connectivity between the frontal lobes and limbic system. The limbic system is the term used to describe a number of structures lying at the border of the cerebral cortex and hypothalamus, including the cingulate, parahippocampal gyri, hippocampal formation, amygdalae, and anterior thalamic nucleus (Snell 2006), and although tracts arising from the cingulate cortex predominantly connect with the hippocampus (Snell 2006), the amygdala is also connected with these structures, and plays an important role in emotion processing (Gallagher & Chiba, 1996). In summary, connectivity between the frontal cortex and limbic system is proposed to facilitate interaction between cognitive and emotional aspects of Self-Awareness. As shown in Table 7.1, there were differential age-related effects on the measures of awareness at each level.

#### 7.1.1. Aims of the Study.

The aim of this study was to investigate if the different types of awareness (HoP levels) were similarly correlated with each other in younger and older age groups. Significant correlations between measures associated with the different levels would provide support for the proposition that each level is inter-connected. A different pattern of correlation in younger and older adults would suggest functional differences at the level of interconnectivity of awareness in normal ageing.

These aims were addressed using correlation analyses. In addition, given that affect was found to influence metamemory perceptions, but was not related to any of the ERP components (see Chapters 4 and 5), separate partial correlations, controlling for anxiety and depression, were conducted (Level 3 – Consistent Consciousness and Level 4 – Self-Awareness). The following hypotheses were explored.

1. Operationalisations of Sensorimotor Awareness and Consistent Consciousness (Levels 2 and 3) would be related to each other within each age group.

2. Operationalisations of Consistent Consciousness and Self-Awareness (Levels 3 and 4) would be related to each other within each age group.

3. The pattern of correlations investigated in the first two hypotheses would differ between younger and older age groups.

## 7.2. Method.

### 7.2.1. Participants.

Data obtained from 30 older adults (OA: *M* age 69.4 years, *SD* 6.7) and 21 younger adults (YA: *M* age 22.1 years, *SD* 4.1) were included in this exploratory study. Four participants were excluded (OA: *n* = 1; YA: *n* = 3) due to the fact that they did not complete both experimental sessions. Data were also excluded from participants who did not complete sufficient trials to enable the averaging of ERP components and /or due to lack of access to audiometry equipment (OA: *n* = 12; YA: *n* = 4). Numbers and descriptive details of participants included for each of the main analyses are given in Table 7.2. All groups, for any given pair of HoP levels, had equivalent cognitive functioning (MMSE, MoCA and Raven SPM, all  $p > .1$ ).

Table 7.2. Demographic data for the younger and older adults for each pair of levels.

Correlation	Younger participants		Older participants	
	<i>n</i> : M:F	Age: <i>M</i> ( <i>SD</i> )	<i>n</i> : M:F	Age: <i>M</i> ( <i>SD</i> )
Level 2 with Level 3	2:11	20.3 (3.6)	1:11	67.8 (6.2)
Level 3 with Level 4	2:14	21.3 (3.9)	6:14	69.0 (6.3)

*Note.* M:F = number of males and females

### 7.2.2. Variables Included in Analyses.

1) Novelty Auditory Oddball Paradigm (described in Chapter 4).

- i) N1 amplitude from standard stimuli at Fz/Cz/Pz
- ii) N1 amplitude from target stimuli at Fz/Cz/Pz
- iii) N1 amplitude from novel stimuli at Fz/Cz/Pz
- iv) P3 amplitude from standard stimuli at Fz/Cz/Pz
- v) P3 amplitude from target stimuli at Fz/Cz/Pz
- vi) P3 amplitude from novel stimuli at Fz/Cz/Pz

2) 4-Choice Response ERP Task (Hogan et al., 2005) (described in Chapter 5).

- i) N2 amplitude (compatible and incompatible conditions)
- ii) P3b amplitude (compatible and incompatible conditions)
- iii) N4 amplitude (compatible and incompatible conditions)
- iv) SW amplitude (compatible and incompatible conditions)
- v) CRN amplitude (compatible and incompatible conditions)
- vi) ERN amplitude (compatible and incompatible conditions)
- vii) Pe amplitude (compatible and incompatible conditions)

3) Metamemory In Adulthood (MIA; Dixon, Hultsch & Hertzog, 1988) (described in Chapters 3 and 6).

- i) Achievement (perception of a good memory and its importance)
- ii) Anxiety (anxiety over memory performance)
- iii) Capacity (perception of memory capabilities)
- iv) Change (belief regarding memory abilities over time)
- v) Locus (perception of control over memory)
- vi) Strategy (use of strategies for memory performance); divided into External and Internal (external memory aids and internal mnemonic) strategies

vii) Task (knowledge of memory processes)

4) DEX questionnaire from the Behavioural Assessment of the Dysexecutive Syndrome battery (BADs; Wilson, Alderman, Burgess, Emslie & Evans, 1996) (described in Chapter 3 and 6).

5) Memory Awareness Rating Scale (MARS; Clare, Wilson, Carter, Roth & Hodges, 2002) (described in Chapter 3 and 6).

6) Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) (described in Chapter 3).

i) anxiety subscale

ii) depression subscale.

### 7.2.3. Statistical Analyses.

The distribution of each measure did not significantly differ from normality (Shapiro Wilk tests, all  $p > 1$ ). Each outlying value was identified using boxplots and was excluded on a participant by participant basis. Pearson product-moment correlations were used to test for relationships between variables in each level comparison<sup>2</sup>. Bonferroni adjusted alpha levels were not calculated for multiple comparisons due to the exploratory nature of this study (see Perneger, 1998; Rothman, 1990)<sup>3</sup>. Anxiety and depression were controlled for in the Level 3 and 4 correlations, as these measures covaried with metamemory perceptions (see Section 6.3.2.1, Chapter 6). As affect was not found to be related to any of the ERP components representing either Sensorimotor Awareness (Level 2) or Consistent Consciousness (Level 3),

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<sup>2</sup> One of the caveats of this study is the risk of making type 1 errors due to the size of the correlation tables. However, this likelihood is reduced by the reporting of results based on the strength of the correlations following the suggestions of Tabachnick and Fidell (2001).

<sup>3</sup> However, the alpha value would be  $p = .0025$  when Bonferroni corrected for multiple comparisons and, therefore, all of the results reported in this chapter would have been non-significant.

these partial correlations were not conducted for testing of the first hypothesis. Due to the small sample sizes, there were few significant correlations, therefore only correlations accounting for 20% or more ( $-.45 \geq r \geq +.45$ ) of the shared variance are reported as noteworthy relationships. The G\* Power 3 calculation program (Faul et al., 2007) revealed that each group needed to consist of 36 participants to find relationships of this magnitude with an alpha level of .05, power .80. In order to assess whether the pattern of correlations differed significantly between groups, the correlations for each group were compared using Fisher's Z test (Howell, 2002).

### 7.3. Results.

#### 7.3.1. Comparison between Level 2 - Sensorimotor Awareness and Level 3 - Consistent Consciousness.

The correlations between the measures of awareness operationalising Levels 2 and 3 are presented in Tables 6.3 and 6.4, for older and younger adults, respectively. As indicated, there were significant and moderate to strong relationships between the measures at each level in both groups. In the older adults, higher N1 amplitudes to target (at Cz) and novel (at Fz) stimuli were related to increased N2 amplitudes in both conditions, suggesting a link between initial attentional capture and conflict monitoring. Increased P3 amplitudes at parietal regions for target and novel stimuli were also related to higher P3b amplitudes in both conditions of the 4-CRT in the older adults, indicating that the allocation of attention to processing stimuli in both paradigms was similar.

There were fewer relationships between the measures at each level in the younger adults. However, consistent with the older adults, increased novelty P3 amplitude was related to P3b amplitudes in both conditions, indicating that greater attentional processing of unexpected novel stimuli was associated with increased attentional processing of stimuli in the 4-CRT. In addition, higher target P3 amplitudes at parietal regions were related to increased P3b amplitudes in the incompatible condition in the younger adults. This association suggests that

younger adults allocate a similar amount of attention to stimuli requiring a response in a simple sensory processing paradigm and to stimuli with greater stimulus-response incompatibility in the 4-CRT.

Contrary to the prediction that there would be group differences in the pattern of correlations between groups in each level comparison, there were no significant differences between groups in the magnitude of any of the correlations. These results indicate that, despite the correlations suggesting a different pattern of relationships between measures of awareness in each group, the groups were not significantly different in the magnitude of any of the associations.

### 7.3.2. Comparison between Level 3 - Consistent Consciousness and Level 4 – Self-Awareness.

As indicated in Tables 6.5 and 6.6, older and younger adults, respectively, measures of awareness at Level 3 were related to measures at Level 4 in both groups. In the older adults, better conflict monitoring of incompatible stimuli, as indexed by higher N2 amplitude, was related to increased perceptions of memory capabilities (Capacity). Error detection in the compatible condition of the 4-CRT was also related to perceptions of control over memory functioning (Locus). In addition, better error processing in the compatible condition, as indexed by Pe amplitude, was associated with strategy use (Strategy), and mnemonic internal strategies (Internal Strategy). Relationships between remedial actions after errors and measures of Self-Awareness were also found in the older adults. Higher rate of error correction in the incompatible condition was associated with greater knowledge of memory processes (Task). Longer post-error slowing after errors in the compatible condition was linked with perceptions of greater memory capabilities (Capacity), more control over memory (Locus) and fewer problems in memory demanding situations. Longer post-error slowing in the incompatible condition was related to the perception of greater stability in memory over time (MIA Change). With regard to relationships with affect in the older adults, higher level of depression, as measured by the HADS, was related to increased feelings of stress and anxiety

over memory functioning (Anxiety), the perception of reduced memory capabilities (Capacity), and a belief in greater decline in memory over time (Change).

In the younger adults, increased conflict monitoring in the incompatible condition, again measured by N2 amplitude, was associated with perceptions of more control over memory (Locus), and fewer reports of cognitive and behavioural problems, as measured by the DEX. In addition, the increased allocation of attentional processing of both compatible and incompatible stimuli, as indexed by P3b amplitudes, was related to perceptions of better memory capabilities (Capacity), more control over memory functioning (Locus), and fewer reported problems in memory demanding situations, as measured by the MARS. The P3b amplitude in the incompatible condition was also associated with the perception of greater memory stability over time (Change). Similarly to the older adults, better error detection in the compatible condition was related to perceptions of greater control over memory functioning (Locus), greater internal strategy use (Internal Strategy), and knowledge of memory processes (Task). Increased error detection in the incompatible condition was also related to strategy use (Strategy), particularly internal mnemonic strategies (Internal Strategy), and reduced feelings of stress and anxiety over memory functioning (Anxiety). Better error processing, as indexed by Pe amplitude, in the compatible condition was related to the use of external aids to support memory performance (External Strategy). Relationships between remedial behaviours and metamemory perceptions were also found in the younger adults. Higher rate of error correction in the compatible condition was associated with perceptions of better memory capabilities (Capacity). Whilst longer post-error slowing in the compatible condition was linked to greater strategy use to support memory functioning (Strategy), the use of internal strategies (Internal Strategy), and more knowledge of memory processes (Task). As with the older adults, relationships were found with affect and measures of Self-Awareness. Increased level of anxiety, as measured by the HADS, was related to greater feelings of anxiety and stress over memory (Anxiety), reduced perceptions of memory capabilities (Capacity), belief in greater change in memory over time (Change), and more reported cognitive and behavioural problems, as measured by the DEX. Higher level of depression, also measured by the HADS, was associated with more feelings of stress and anxiety over memory functioning (Anxiety), and more reported cognitive and behavioural problems.

As with the previous level comparison, a different pattern of correlations between the measures of awareness at each level was found within each group. However, only a small number of these associations differed in magnitude between the age groups. In the younger adults, perceptions of better current memory capabilities were significantly related to lower levels of anxiety, but this association was not found in the older adults, and the *Z* test revealed that this difference was significant ( $p = .029$ ). Better attentional processing of incompatible stimuli was also related to perceptions of greater memory capabilities in the younger adults, but not in the older adults ( $p = .015$ ). Significant correlations between error detection and post-error slowing in the compatible condition and the Task subscale were found in the younger adults, but not in the older adults ( $p = .012$  and  $p = .025$ , error detection and post-error slowing, respectively). Thus, even though older and younger adults reported similar levels of knowledge of memory processes, older adults' knowledge of memory processes was not influenced by their performance monitoring abilities in the compatible condition of the 4-CRT.

Table 7.3. Pearson product-moment correlations between the measures of awareness for Level 2 – Sensorimotor Awareness (rows) and Level 3 – Consistent Consciousness (columns) for older adults.

		Compatible							Incompatible							
		N2	P3b	N4	SW	CRN	ERN	Pe	N2	P3b	N4	SW	CRN	ERN	Pe	
Standard	N1	Fz	.35 (.12)	.31 (.10)	.27 (.07)	.16 (.03)	-.05 (.00)	.39 (.15)	.27 (.07)	.33 (.11)	.23 (.05)	.05 (.00)	.06 (.00)	<u>.55 (.30)</u>	.24 (.06)	<u>-.53 (.28)</u>
		Cz	.28 (.08)	.31 (.10)	.38 (.14)	.29 (.08)	.09 (.01)	.36 (.13)	.18 (.03)	.26 (.07)	.24 (.06)	.22 (.05)	.16 (.03)	<u>.58 (.34)</u>	.28 (.08)	-.42 (.18)
		Pz	.06 (.00)	.18 (.03)	.42 (.18)	.29 (.08)	.23 (.05)	.22 (.05)	-.00 (.00)	.07 (.00)	.13 (.02)	.32 (.10)	.13 (.02)	<u>.57 (.32)</u>	.19 (.04)	-.21 (.04)
	P3	Fz	.37 (.14)	.28 (.08)	.24 (.06)	<u>.53 (.28)</u>	-.06 (.00)	<u>.62 (.38)</u>	-.06 (.00)	<u>.50 (.25)</u>	.35 (.12)	.35 (.12)	<b>.69 (.48) *</b>	.35 (.12)	<u>.56 (.31)</u>	<b>-.64 (.41) *</b>
		Cz	<u>.58 (.34)</u>	<u>.51 (.26)</u>	<u>.52 (.27)</u>	<u>.60 (.36)</u>	-.12 (.02)	.30 (.09)	-.15 (.02)	<b>.70 (.49) *</b>	<u>.62 (.38)</u>	<u>.63 (.40)</u>	<b>.78 (.61) *</b>	.07 (.00)	.28 (.08)	-.35 (.12)
		Pz	.34 (.12)	.40 (.16)	<u>.49 (.24)</u>	<u>.45 (.20)</u>	.03 (.00)	.10 (.01)	-.18 (.03)	<u>.47 (.22)</u>	<u>.50 (.25)</u>	<u>.55 (.30)</u>	<u>.61 (.37)</u>	.14 (.02)	.19 (.04)	-.12 (.02)
Target	N1	Fz	.37 (.14)	.35 (.12)	.38 (.14)	.21 (.04)	<u>-.48 (.23)</u>	-.00 (.00)	<u>.57 (.32)</u>	.43 (.18)	.30 (.09)	.11 (.01)	.05 (.00)	-.08 (.01)	-.01 (.00)	.04 (.00)
		Cz	<u>.49 (.24)</u>	<u>.55 (.30)</u>	<u>.61 (.37)</u>	<u>.61 (.37)</u>	-.29 (.08)	-.09 (.01)	.42 (.18)	<u>.51 (.26)</u>	<u>.52 (.27)</u>	.40 (.16)	<u>.45 (.20)</u>	.10 (.01)	.07 (.00)	-.02 (.00)
		Pz	.40 (.16)	<u>.46 (.21)</u>	<u>.58 (.34)</u>	<b>.68 (.46) *</b>	-.12 (.02)	.15 (.02)	.27 (.07)	.43 (.18)	<u>.45 (.20)</u>	<u>.46 (.21)</u>	<u>.54 (.29)</u>	.28 (.08)	.21 (.04)	-.17 (.03)
	P3	Fz	-.00 (.00)	.01 (.00)	-.01 (.00)	.13 (.02)	-.05 (.00)	<u>.62 (.38)</u>	.05 (.00)	.16 (.03)	.01 (.00)	.09 (.01)	.10 (.01)	.10 (.01)	<u>.54 (.29)</u>	-.13 (.02)
		Cz	<b>.67 (.45) *</b>	<b>.71 (.50) *</b>	<b>.63 (.40) *</b>	<b>.64 (.41) *</b>	-.36 (.13)	.12 (.02)	.18 (.03)	<b>.76 (.58) *</b>	<b>.70 (.49) *</b>	<u>.57 (.32)</u>	<u>.57 (.32)</u>	-.14 (.02)	.09 (.01)	-.07 (.00)
		Pz	<u>.62 (.38)</u>	<b>.75 (.56) *</b>	<u>.56 (.31)</u>	<u>.49 (.24)</u>	-.23 (.05)	.09 (.01)	.27 (.07)	<b>.64 (.41) *</b>	<b>.67 (.45) *</b>	.36 (.13)	.37 (.14)	.11 (.01)	.08 (.01)	-.13 (.02)
Novel	N1	Fz	<b>.74 (.55) *</b>	.41 (.17)	.34 (.12)	.42 (.18)	<b>-.75 (.56) *</b>	-.15 (.02)	.39 (.15)	<b>.80 (.64) *</b>	<u>.56 (.31)</u>	.28 (.08)	<b>.65 (.42) *</b>	<u>-.54 (.29)</u>	-.10 (.01)	-.06 (.00)
		Cz	<u>.60 (.36)</u>	<u>.50 (.25)</u>	<u>.55 (.30)</u>	<b>.64 (.41) *</b>	-.43 (.18)	-.28 (.08)	.08 (.01)	<b>.66 (.44) *</b>	<u>.63 (.40)</u>	<u>.60 (.36)</u>	<b>.75 (.56) *</b>	<u>-.50 (.25)</u>	-.16 (.03)	.19 (.04)
		Pz	.43 (.18)	.42 (.18)	<u>.49 (.24)</u>	<u>.47 (.22)</u>	-.18 (.03)	-.36 (.13)	-.23 (.05)	<u>.47 (.22)</u>	<u>.54 (.29)</u>	<b>.65 (.42) *</b>	<u>.58 (.34)</u>	<u>-.55 (.30)</u>	-.27 (.07)	.36 (.13)
	P3	Fz	.17 (.03)	.03 (.00)	-.14 (.02)	-.10 (.01)	.01 (.00)	<u>.49 (.24)</u>	-.04 (.00)	.22 (.05)	.10 (.01)	.03 (.00)	.04 (.00)	-.14 (.02)	.43 (.18)	-.31 (.10)
		Cz	<u>.49 (.24)</u>	.36 (.13)	.24 (.06)	.05 (.00)	-.31 (.10)	.09 (.01)	.16 (.03)	<u>.54 (.29)</u>	.43 (.18)	.25 (.06)	.13 (.02)	-.43 (.18)	.07 (.00)	.00 (.00)
		Pz	.39 (.15)	<u>.45 (.20)</u>	.36 (.13)	-.12 (.02)	-.17 (.03)	-.25 (.06)	.14 (.02)	.42 (.18)	<u>.46 (.21)</u>	.25 (.06)	-.11 (.01)	-.39 (.15)	-.18 (.03)	.34 (.12)

Note. OA ( $n = 10$ ); shaded areas = measures with age-related differences in the actual test scores; \*  $p < .05$ ; underlined values =  $-.45 \leq r \leq +.45$ ,  $R^2$  values in parentheses.

Table 7.4. Pearson product-moment correlations between the measures of awareness for Level 2 – Sensorimotor Awareness (rows) and Level 3 – Consistent Consciousness (columns) for younger adults.

			Compatible							Incompatible						
			N2	P3b	N4	SW	CRN	ERN	Pe	N2	P3b	N4	SW	CRN	ERN	Pe
Standard	N1	Fz	-.39 (.15)	.03 (.00)	-.44 (.19)	<u>-.53 (.28)</u>	.09 (.01)	.25 (.06)	<u>-.48 (.23)</u>	-.33 (.11)	.01 (.00)	<b>-.64 (.41) *</b>	<u>-.55 (.30)</u>	.17 (.03)	.12 (.02)	<u>-.50 (.25)</u>
		Cz	-.24 (.06)	.10 (.01)	-.38 (.14)	<u>-.53 (.28)</u>	.15 (.02)	.19 (.04)	<u>-.49 (.24)</u>	-.22 (.05)	.03 (.00)	<b>-.61 (.37) *</b>	<b>-.56 (.31) *</b>	.11 (.01)	.15 (.02)	<b>-.61 (.37) *</b>
		Pz	-.10 (.01)	-.07 (.00)	-.22 (.05)	<u>-.46 (.21)</u>	.06 (.00)	.10 (.01)	-.40 (.16)	-.16 (.03)	-.17 (.03)	<u>-.48 (.23)</u>	-.44 (.19)	-.07 (.00)	.15 (.02)	<u>-.55 (.30)</u>
	P3	Fz	-.13 (.02)	.03 (.00)	.09 (.01)	.18 (.03)	.27 (.07)	<b>.60 (.36) *</b>	.12 (.02)	-.10 (.01)	.15 (.02)	.17 (.03)	.25 (.06)	.28 (.08)	.20 (.04)	.39 (.15)
		Cz	-.04 (.00)	.19 (.04)	.08 (.01)	.35 (.12)	.36 (.13)	<u>.51 (.26)</u>	.21 (.04)	-.06 (.00)	.34 (.12)	.26 (.07)	.43 (.18)	.25 (.06)	.19 (.04)	.36 (.13)
		Pz	.11 (.01)	.27 (.07)	.02 (.00)	.20 (.04)	.35 (.12)	.29 (.08)	.12 (.02)	-.02 (.00)	.38 (.14)	.17 (.03)	.31 (.10)	.15 (.02)	.17 (.03)	.09 (.01)
Target	N1	Fz	-.43 (.18)	-.11 (.01)	-.15 (.02)	-.12 (.02)	.25 (.06)	.25 (.06)	-.37 (.14)	<u>-.53 (.28)</u>	.04 (.00)	-.22 (.05)	-.19 (.04)	.16 (.03)	-.17 (.03)	-.26 (.07)
		Cz	-.30 (.09)	.20 (.04)	-.42 (.18)	-.34 (.12)	<b>.57 (.32) *</b>	.25 (.06)	<u>-.51 (.26)</u>	-.43 (.18)	.31 (.10)	<u>-.46 (.21)</u>	-.36 (.13)	.43 (.18)	.06 (.00)	<u>-.49 (.24)</u>
		Pz	.05 (.00)	-.09 (.01)	-.13 (.02)	-.35 (.12)	.22 (.05)	-.02 (.00)	<b>-.67 (.45) *</b>	-.40 (.16)	-.09 (.01)	-.21 (.04)	-.31 (.10)	-.13 (.02)	-.29 (.08)	<u>-.55 (.30)</u>
	P3	Fz	-.35 (.12)	.17 (.03)	-.02 (.00)	.25 (.06)	.28 (.08)	.31 (.10)	.44 (.19)	-.23 (.05)	.17 (.03)	.12 (.02)	.33 (.11)	.35 (.12)	.33 (.11)	<u>.47 (.22)</u>
		Cz	-.44 (.19)	.40 (.16)	-.44 (.19)	.11 (.01)	.43 (.18)	.27 (.07)	.13 (.02)	-.38 (.14)	<u>.47 (.22)</u>	-.21 (.04)	.29 (.08)	.32 (.10)	.23 (.05)	.21 (.04)
		Pz	-.27 (.07)	.42 (.18)	-.34 (.12)	-.01 (.00)	.42 (.18)	.13 (.02)	-.16 (.03)	-.41 (.17)	<u>.47 (.22)</u>	-.13 (.02)	-.05 (.00)	.29 (.08)	-.06 (.00)	-.09 (.01)
Novel	N1	Fz	-.05 (.00)	.38 (.14)	-.25 (.06)	-.10 (.01)	<b>.56 (.31) *</b>	.44 (.19)	<u>-.45 (.20)</u>	-.34 (.12)	.41 (.17)	-.20 (.04)	.00 (.00)	-.01 (.00)	-.04 (.00)	-.37 (.14)
		Cz	-.39 (.15)	.24 (.06)	-.36 (.13)	-.41 (.17)	.32 (.10)	.44 (.19)	-.28 (.08)	-.31 (.10)	.16 (.03)	<u>-.54 (.29)</u>	-.39 (.15)	.28 (.08)	.32 (.10)	-.36 (.13)
	P3	Pz	-.32 (.10)	.38 (.14)	<u>-.52 (.27)</u>	<u>-.51 (.26)</u>	<u>.45 (.20)</u>	.33 (.11)	-.38 (.14)	-.38 (.14)	.28 (.08)	<b>-.58 (.34) *</b>	-.34 (.12)	.23 (.05)	.28 (.08)	-.37 (.14)
		Fz	.12 (.02)	<b>.60 (.36) *</b>	-.41 (.17)	-.17 (.03)	<b>.70 (.49) *</b>	.34 (.12)	-.15 (.02)	.04 (.00)	<u>.49 (.24)</u>	-.39 (.15)	.03 (.00)	.17 (.03)	<u>.49 (.24)</u>	-.38 (.14)
		Cz	-.05 (.00)	<u>.49 (.24)</u>	<u>-.45 (.20)</u>	.03 (.00)	<b>.57 (.32) *</b>	.22 (.05)	-.15 (.02)	-.13 (.02)	<u>.54 (.29)</u>	-.35 (.12)	.26 (.07)	.04 (.00)	.22 (.05)	-.16 (.03)
		Pz	.04 (.00)	<u>.53 (.28)</u>	-.38 (.14)	.13 (.02)	<u>.50 (.25)</u>	.04 (.00)	-.09 (.01)	-.01 (.00)	<b>.64 (.41) *</b>	-.14 (.02)	.23 (.05)	.14 (.02)	.01 (.00)	-.09 (.01)

Note. YA ( $n = 13$ ); shaded areas = measures with age-related differences in the actual test scores; \*  $p < .05$ ; underlined values =  $-.45 \leq r \leq +.45$ ,  $R^2$  values in parentheses.

Table 7.5. Pearson product-moment correlations between the measures of awareness for Level 3 – Consistent Consciousness (rows) and Level 4 – Self-Awareness (columns), and between affect (rows) and Level 4 – Self-Awareness measures for older adults.

		Metamemory In Adulthood Subscales										
		Achievement	Anxiety	Capacity	Change	Locus	Strategy	Internal	External	Task	DEX	MARS
Compatible	N2	-.03 (.00)	.17 (.03)	-.41 (.17)	-.19 (.04)	-.31 (.10)	.42 (.18)	.42 (.18)	.32 (.10)	-.41 (.17)	-.06 (.00)	-.05 (.00)
	P3b	-.31 (.10)	-.20 (.04)	-.20 (.04)	.37 (.14)	.04 (.00)	-.05 (.00)	-.13 (.02)	.10 (.01)	-.04 (.00)	-.28 (.08)	.17 (.03)
	N4	-.07 (.00)	.22 (.05)	<b>-.53 (.28) *</b>	-.09 (.01)	-.07 (.00)	.02 (.00)	-.09 (.01)	.19 (.04)	-.12 (.02)	-.02 (.00)	-.26 (.07)
	SW	-.42 (.18)	-.11 (.01)	-.27 (.07)	.32 (.10)	-.02 (.00)	.00 (.00)	-.08 (.01)	.14 (.02)	-.12 (.02)	-.34 (.12)	-.10 (.01)
	CRN	.05 (.00)	.19 (.04)	-.10 (.01)	-.19 (.04)	.24 (.06)	-.06 (.00)	-.20 (.04)	.21 (.04)	.11 (.01)	-.06 (.00)	.23 (.05)
	ERN	.02 (.00)	-.24 (.06)	.14 (.02)	.24 (.06)	<b>.50 (.00) *</b>	-.23 (.05)	-.23 (.05)	-.17 (.03)	-.30 (.09)	-.37 (.14)	-.05 (.00)
	Pe	-.18 (.03)	.05 (.00)	.06 (.00)	-.12 (.02)	-.21 (.04)	<u>.47 (.22)</u>	<u>.45 (.20)</u>	.38 (.14)	-.31 (.10)	-.08 (.01)	-.06 (.00)
	Error Correction	-.10 (.01)	-.04 (.00)	<u>-.46 (.21)</u>	.04 (.00)	<u>-.47 (.22)</u>	.18 (.03)	.20 (.04)	.10 (.01)	.17 (.03)	<b>.54 (.29) *</b>	<u>-.48 (.23)</u>
	Post-error slowing	.19 (.04)	-.21 (.04)	<b>.60 (.36) *</b>	.16 (.03)	<u>.47 (.22)</u>	<b>-.52 (.27) *</b>	<b>-.53 (.28) *</b>	-.37 (.14)	-.30 (.09)	-.22 (.05)	<u>.46 (.21)</u>
Incompatible	N2	.01 (.00)	.22 (.05)	<b>-.49 (.00) *</b>	-.22 (.05)	-.31 (.10)	<u>.45 (.20)</u>	<u>.46 (.21)</u>	.32 (.10)	-.36 (.13)	.06 (.00)	-.19 (.04)
	P3b	-.36 (.13)	-.09 (.01)	-.40 (.16)	.29 (.08)	-.07 (.00)	.07 (.00)	.02 (.00)	.15 (.02)	-.12 (.02)	-.22 (.05)	.00 (.00)
	N4	-.14 (.02)	.19 (.04)	<b>-.55 (.00) *</b>	-.03 (.00)	-.01 (.00)	.02 (.00)	-.07 (.00)	.15 (.02)	.00 (.00)	-.03 (.00)	-.22 (.05)
	SW	<u>-.47 (.22)</u>	-.16 (.03)	-.30 (.09)	.38 (.14)	-.05 (.00)	.09 (.01)	.04 (.00)	.16 (.03)	-.07 (.00)	-.26 (.07)	-.07 (.00)
	CRN	-.28 (.08)	.22 (.05)	-.11 (.01)	-.18 (.03)	-.07 (.00)	.03 (.00)	-.11 (.01)	.25 (.06)	<u>-.47 (.22)</u>	-.23 (.05)	.17 (.03)
	ERN	-.09 (.01)	-.26 (.07)	.02 (.00)	.16 (.03)	.22 (.05)	.02 (.00)	-.04 (.00)	.12 (.02)	-.32 (.10)	-.31 (.10)	-.04 (.00)
	Pe	.24 (.06)	.31 (.10)	-.05 (.00)	-.21 (.04)	-.08 (.01)	-.18 (.03)	-.21 (.04)	-.09 (.01)	.32 (.10)	.21 (.04)	-.24 (.06)
	Error Correction	-.07 (.00)	.24 (.06)	-.22 (.05)	-.15 (.02)	<b>-.57 (.32) *</b>	.24 (.06)	.14 (.02)	.33 (.11)	<u>.46 (.21)</u>	.43 (.18)	-.26 (.07)
	Post-error slowing	<b>-.72 (.52)</b>	-.21 (.04)	-.15 (.02)	<b>.53 (.28) *</b>	.13 (.02)	-.20 (.04)	-.25 (.06)	-.08 (.01)	-.35 (.12)	-.11 (.01)	.03 (.00)
HADS Anxiety	-.12 (.02)	<b>.62 (.38) *</b>	-.28 (.08)	-.23 (.05)	-.10 (.01)	.03 (.00)	-.04 (.00)	.15 (.02)	.08 (.01)	<b>.66 (.44) *</b>	-.03 (.00)	
HADS Depression	-.09 (.01)	<b>.51 (.26) *</b>	<b>-.53 (.28) *</b>	<b>-.62 (.38) *</b>	-.36 (.13)	-.13 (.02)	.05 (.00)	-.36 (.13)	-.01 (.00)	.40 (.16)	-.19 (.04)	

Note. OA controlling for Anxiety and Depression ( $n = 15$ ); correlations with HADS Anxiety and Depression OA ( $n = 19$ ); shaded areas = measures with age-related differences in actual test scores; \*  $p < .05$ ; underlined values =  $-.45 \geq r \geq +.45$ , values in parentheses.

Table 7.6. Pearson product-moment correlations between the measures of awareness for Level 3 – Consistent Consciousness (rows) and Level 4 – Self-Awareness (columns), and between affect (rows) and Level 4 – Self-Awareness measures for younger adults.

		Metamemory In Adulthood Subscales										
		Achievement	Anxiety	Capacity	Change	Locus	Strategy	Internal	External	Task	DEX	MARS
Compatible	N2	-.06 (.00)	-.40 (.16)	-.10 (.01)	-.38 (.14)	-.39 (.15)	-.07 (.00)	.11 (.01)	-.17 (.03)	<u>-.48 (.23)</u>	<u>.48 (.23)</u>	.28 (.08)
	P3b	-.09 (.01)	-.19 (.04)	<u>.55 (.30)</u>	-.22 (.05)	<u>-.49 (.24)</u>	.07 (.00)	.17 (.03)	-.05 (.00)	-.02 (.00)	.19 (.04)	<b>.62 (.38) *</b>
	N4	-.29 (.08)	-.01 (.00)	-.05 (.00)	-.40 (.16)	-.11 (.01)	-.29 (.08)	<u>-.47 (.22)</u>	.03 (.00)	<u>-.59 (.35)</u>	.03 (.00)	-.10 (.01)
	SW	-.01 (.00)	.31 (.10)	-.05 (.00)	<b>-.70 (.49) *</b>	-.21 (.04)	-.21 (.04)	-.25 (.06)	-.05 (.00)	-.15 (.02)	-.11 (.01)	-.04 (.00)
	CRN	.16 (.03)	-.16 (.03)	<u>.59 (.35)</u>	-.16 (.03)	-.10 (.01)	-.21 (.04)	.06 (.00)	-.30 (.09)	.29 (.08)	-.05 (.00)	<u>.58 (.34)</u>
	ERN	.42 (.18)	-.18 (.03)	.20 (.04)	.28 (.08)	<u>.51 (.26)</u>	.21 (.04)	<u>.48 (.23)</u>	-.13 (.02)	<b>.74 (.55) *</b>	-.12 (.02)	.35 (.12)
	Pe	-.40 (.16)	-.02 (.00)	-.31 (.10)	-.18 (.03)	<u>-.48 (.23)</u>	.31 (.10)	-.18 (.03)	<u>.50 (.25)</u>	-.12 (.02)	.26 (.07)	-.30 (.09)
	Error Correction	<u>-.47 (.22)</u>	.02 (.00)	<u>.47 (.22)</u>	.23 (.05)	.11 (.01)	-.14 (.02)	-.44 (.19)	.18 (.03)	.08 (.01)	-.21 (.04)	.19 (.04)
	Post-error slowing	.05 (.00)	-.20 (.04)	.14 (.02)	.40 (.16)	.22 (.05)	<u>.58 (.34)</u>	<u>.52 (.27)</u>	.27 (.07)	<b>.67 (.45) *</b>	.09 (.01)	.32 (.10)
Incompatible	N2	-.07 (.00)	<b>-.61 (.37) *</b>	-.29 (.08)	-.22 (.05)	<b>-.67 (.45) *</b>	<u>.48 (.23)</u>	<u>.45 (.20)</u>	.21 (.04)	-.42 (.18)	<b>.79 (.62) *</b>	.14 (.02)
	P3b	.14 (.02)	-.04 (.00)	<b>.66 (.44) *</b>	<u>-.46 (.21)</u>	<u>-.46 (.21)</u>	-.18 (.03)	.10 (.01)	-.29 (.08)	.02 (.00)	.05 (.00)	<b>.61 (.37) *</b>
	N4	-.31 (.10)	.16 (.03)	.05 (.00)	<b>-.64 (.41) *</b>	-.24 (.06)	-.25 (.06)	<u>-.46 (.21)</u>	.07 (.00)	-.42 (.18)	-.05 (.00)	.03 (.00)
	SW	.05 (.00)	.43 (.18)	-.03 (.00)	<b>-.73 (.53) *</b>	-.14 (.02)	-.27 (.07)	-.20 (.04)	-.16 (.03)	.01 (.00)	-.22 (.05)	.06 (.00)
	CRN	-.04 (.00)	<u>-.50 (.25)</u>	.32 (.10)	<u>.48 (.23)</u>	-.24 (.06)	<u>.50 (.25)</u>	.35 (.12)	.31 (.10)	.28 (.08)	.25 (.06)	.24 (.06)
	ERN	.01 (.00)	<u>-.50 (.25)</u>	-.25 (.06)	.42 (.18)	-.13 (.02)	<u>.55 (.30)</u>	<u>.49 (.24)</u>	.27 (.07)	.32 (.10)	.40 (.16)	.09 (.01)
	Pe	-.15 (.02)	.19 (.04)	-.38 (.14)	.01 (.00)	.05 (.00)	.22 (.05)	-.13 (.02)	.36 (.13)	.30 (.09)	.03 (.00)	-.44 (.19)
	Error Correction	-.44 (.19)	.11 (.01)	.27 (.07)	.27 (.07)	.25 (.06)	-.19 (.04)	<u>-.49 (.24)</u>	.16 (.03)	.22 (.05)	-.20 (.04)	-.01 (.00)
	Post-error slowing	-.17 (.03)	.15 (.02)	-.11 (.01)	.41 (.17)	.10 (.01)	.09 (.01)	-.19 (.04)	.25 (.06)	-.08 (.01)	.03 (.00)	<u>-.50 (.25)</u>
HADS Anxiety	-.10 (.01)	<b>.52 (.27) *</b>	<b>-.82 (.67) *</b>	<b>-.75 (.56) *</b>	-.04 (.00)	.31 (.10)	.24 (.06)	.24 (.06)	-.10 (.01)	<u>.49 (.24)</u>	-.40 (.16)	
HADS Depression	-.04 (.00)	<b>.62 (.38) *</b>	-.17 (.03)	<u>-.47 (.22)</u>	-.16 (.03)	.31 (.10)	.36 (.13)	-.09 (.01)	-.16 (.03)	<b>.70 (.49) *</b>	-.26 (.07)	

Note. YA controlling for Anxiety and Depression ( $n = 9$ ); correlations with HADS Anxiety and Depression OA ( $n = 15$ ); shaded areas = measures with age-related differences in actual test scores; \*  $p < .05$ ; underlined values =  $-.45 \geq r \geq +.45$ ,  $R^2$  values in parentheses.

#### 7.4. Discussion.

The aim of this study was to explore whether there were relationships between the measures of awareness operationalising the different levels of the HoP model (Stuss et al., 2001), and to investigate if the pattern of correlations differed between younger and older participants. The study found evidence of relationships between measures at each level comparison within each group, in support of the inter-connectivity proposed by Stuss and colleagues. Despite different patterns of correlations within each group in each level comparison, there were few differences between the groups, which indicated that the magnitude of the associations between measures of awareness related in one group, but not another, were similar. The implication of these findings were that there were few age-related differences in the inter-connectivity of the levels of awareness, and where there were age differences, this was because associations were significant in younger adults, not older adults. The relationship between Levels 2 and 3 of the HoP model are considered first.

Significant associations were found between measures at Level 2 – Sensorimotor Awareness and Level 3 – Consistent Consciousness in both groups. Target P3 amplitude elicited in the novelty auditory oddball was related to P3b amplitudes in both conditions of the 4-CRT, and between the initial attentional capture of novel stimuli and conflict monitoring, as indexed by the N2, in the older adults. In the younger adults, significant relationships were found between novelty P3 amplitude and P3b amplitude in both conditions. Similar to the younger adults, moderate correlations, albeit not significant, were found in the older adults between novelty P3 amplitude and P3b amplitudes in both conditions. These findings suggest that the processing of sensory information underlying Level 2 may also be relevant to the organising of sensory information underlying behaviour at Level 3, therefore, the processing involved in these levels may overlap to some degree. Although these P3 relationships have indicated that attentional processing is similar in a simple sensory paradigm and a more complex executive task, the associations do not inform on the inter-connectivity of the HoP model. More specifically, in the current study, the target P3 and P3b are similar components, in terms of function, time frame for peak detection, and neural generators. One of the inherent challenges in comparing stimulus-locked components associated with attentional processing in two

separate paradigms is the overlap in the cognitive processes. However, one of the issues with the definitions of the different types of awareness in the HoP model is the distinction between sensory processing at Level 2 and the organisation of sensory information at Level 3, which have been operationalised using the P3 component. Although, this is a limitation to the investigation of the inter-connectivity of the HoP model, it is important to acknowledge that this issue of component overlap is not relevant in the testing of the individual levels of awareness. Assessing attentional processing is fundamental to an investigation of performance monitoring, and particularly as the definition of Consistent Consciousness states that this type of awareness involves the organisation of sensory information.

Significant associations were also found in each group between measures at Level 3 – Consistent Consciousness and Level 4 – Self-Awareness. Better error detection in the compatible condition was significantly associated with increased perceptions of control over memory functioning in the older adults. There were also significant relationships between longer post-error slowing in the compatible condition and the perception of greater memory capabilities, and greater post-error slowing in the incompatible condition and the belief of greater stability in memory functioning over time. Moderate associations were found between longer post-error slowing in the compatible condition and with perceptions of more control over memory and fewer reported problems with memory demanding situations. Taken together, these findings suggest that older adults' self-reported awareness of memory functioning and abilities were related to the degree of the detection of mistakes in a rapidly presented four choice reaction time task, as well as the degree to which they slowed down after making such errors. These findings are consistent with the proposed inter-connectivity in the HoP model.

In the younger adults, better error detection and longer post-error slowing in the compatible condition were linked to better knowledge of memory processes. Additionally, increased allocation of attentional processing of stimuli (both compatible and incompatible) was associated with fewer reported problems with memory demanding situations. Attentional processing of compatible stimuli in the younger adults was also significantly related to the perception of increased memory capabilities. Moderate and strong correlations, however, not

significant, were again identified between the measures of awareness. Similarly to the older adults, a strong positive correlation between error detection in the compatible condition and perception of control over memory functioning was found. Despite some subtle differences between the age groups, these findings also fit well with the HoP model in that individuals who reported fewer problems, and individuals who felt knowledgeable about the functioning of their memory were those better able to detect they had made errors, who adjusted their speed post errors to compensate, and who were better able to allocate attentional resources to stimuli.

Whilst better error detection in the incompatible condition was strongly related to strategy use in the younger adults, the use of strategies and, in particular, internal mnemonic strategies were also strongly associated with longer post-error slowing. Moreover, error detection in both conditions in the younger adults was moderately associated with the use of internal mnemonic strategies to support memory functioning. Higher rates of error correction in the compatible condition in the younger adults were moderately related to perceptions of increased memory capabilities. In contrast, increased strategy use and, in particular, internal strategies were moderately related to error processing in the compatible condition in older adults, whilst in the younger adults, error processing in the compatible condition was strongly associated with the use of external memory aids. These associations suggest that better performance monitoring abilities were linked with more positive beliefs about memory abilities and the utilisation of strategies to support memory functioning.

The relationship between affect and Self-Awareness was further investigated, and higher levels of anxiety and depression, as measured by the HADS, were significantly associated with greater feelings of stress and anxiety over memory in both groups. Moreover, those who reported more anxiety generally, whether older or younger, also reported a greater number of cognitive and behavioural problems, as measured by the DEX. Additionally, in the older adults, individuals reporting more depression generally also reported reduced memory capabilities and believed in greater change in memory functioning over time. By contrast, in younger adults, greater general anxiety rather than depression was related to greater

perceptions that memory capabilities were worse, and that memory functioning had changed more over time.

These findings are consistent with the discriminant validity analyses reported by Dixon et al. (1988), who found that the MIA Anxiety subscale was related to state and trait anxiety, however, the relationship with depression was in contrast to Dixon et al. (1988) who found that depression did not correlate with the Anxiety subscale. Dixon et al. (1988) employed a different range of mood scales which may account for the different findings in this study. Nevertheless, both groups' scores on the HADS subscales were in the normal range (0-7; Zigmond & Snaith, 1983), which suggests that increased anxiety and depression, indicated by even subtle differences within the normal range, influenced metamemory perceptions. These findings suggest that there is an emotional component to Self-Awareness, providing further support for previous studies linking metamemory beliefs with affect (e.g. Abson & Rabbitt, 1990; 1991; McDougall, 1995; Verheagen et al., 2000).

The inter-connectivity of the levels in the HoP model (Stuss et al., 2001) described how the processes underlying the different types of awareness could interact, and the potential for deficits at one level to be transmitted to the next level. The current study also found some evidence of relationships between measures at different levels that had age-related reductions. Older adults had lower target P3 amplitudes (Level 2 – Sensorimotor Awareness, see Chapter 4), and lower P3b amplitudes in both conditions of the 4-CRT (Level 3 – Consistent Consciousness, see Chapter 5) compared to younger adults. These measures were also significantly associated, indicating that the older adults with lower target P3 amplitudes in the Level 2 measures also had lower P3b amplitudes in the Level 3 measures. Significantly lower Pe amplitude in the compatible condition (Level 3 – Consistent Consciousness, see Chapter 5) was found in the older adults, yet this group reported increased use of strategies to support memory functioning (Level 4 – Self-Awareness, see Chapter 6). Of note, is the relationship between these measures; attenuated Pe amplitude was associated with the use of fewer strategies. These findings offer some support for the proposition that impairments at one level may be connected to deficits at a higher level. However, support was also found for the proposition that impairments at lower levels can be circumvented by processes at higher

levels, as there were no behavioural performance differences between younger and older adults in the novelty auditory oddball or the 4-CRT; measures indexing Level 2 and 3 performance.

It is possible, however, that relationships between ERP measures may be more related to a global reduction in the power of ERP components than to a relationship between levels of awareness of functioning. In other words, the relationship between the P3 elicited by target stimuli in the novelty auditory oddball (Level 2) and the P3b elicited by the Level 3 4-CRT measure in older adults may simply reflect generalised loss of power, rather than a causal relationship between these measures of awareness. However, a generalised reduction in power would be expected to involve all ERP components studied, and this was not the case: not all ERP components were correlated in either group.

The current study has provided two sources of evidence in the assessment of the HoP model (Stuss et al., 2001). The first is the within-level (intra-connectivity) age group differences detailed in Chapters 4-6. The second is the correlational analysis of inter-connectivity detailed in the present study. As noted in the Introduction, Stuss et al. (2001) described how the inter-connectivity of the HoP model was supported by the functioning of numerous modules within each level. The current data are based on an extrapolation of this theory rather than a test of Stuss et al.'s (2001) specific modules, the operationalisations of which have not been provided by Stuss or his colleagues. However, based on the presence of correlations between measures operationalising the HoP levels, it may be concluded that the current findings are compatible with the inter-connectivity view specified within the model (Stuss et al., 2001). The presence of null findings also suggests the possibility that the measures selected were not fully representative of Stuss et al.' (2001) levels of awareness, or that only some modules within a level facilitate inter-connectivity with other modules. Moreover, caution must be taken in interpreting the correlations reported herein due to the small sample sizes. Clearly, replication with larger samples is warranted.

However, despite the relatively small samples reported, this studies offers novel findings. Only one previous study (O'Keefe et al., 2007) has utilised a multidimensional assessment of

different types of awareness in individuals with traumatic brain injury. This study investigated metacognitive awareness, associated with knowledge about the self, and on-line emergent and anticipatory awareness; the awareness that a problem exists, and the understanding that the impairment was causing a problem. O’Keefe et al. (2007) correlated composite scores representing the different types of awareness and found that on-line emergent and anticipatory awareness were highly related, and were operationalised by error monitoring on a sustained attention task, and by discrepancy scores between predicted and actual performance on two subtests of the Wechsler memory scale. However, the relationship between these types of awareness may have been due to the measures tapping into different aspects of performance monitoring; better error monitoring was associated with more accurate assessment of abilities. In support of the separation of metacognitive knowledge and on-line awareness described by Toglia and Kirk (2000, see Section 1.3.4.1.2, Chapter 1), O’Keefe et al. (2007) found that metacognitive knowledge, as measured by an awareness interview and discrepancy between predicted neuropsychological test performance and actual performance, and patient-proxy discrepancies on three questionnaires regarding functioning, was not related either to emergent or to anticipatory awareness. This suggests a separation between knowledge and awareness of the self and on-line awareness of performance. The former relating to self-awareness and knowledge about personal abilities and level of functioning, the latter referring to immediate awareness of the environment and behaviour and experience of activity. Evidence for self-awareness can be found in the self-report of functioning in this thesis and from prediction/performance discrepancies in O’Keefe et al. (2007), whilst error monitoring is an example of on-line awareness of performance (see Chapter 5 and O’Keefe et al., 2007).

There are similarities in the operationalisations employed to assess the HoP model (Stuss et al., 2001), the concepts of metacognitive knowledge and on-line awareness (Toglia & Kirk, 2000), and the operationalisation of emergent anticipatory awareness (O’Keefe et al., 2007). The current study operationalised Self-Awareness with the assessment of metamemory perceptions and self-reports of cognitive abilities, which is similar to the self knowledge component of metacognitive knowledge described by Toglia and Kirk (2000). Furthermore, Sensorimotor Awareness and Consistent Consciousness were assessed by ERP components which measure brain functioning underlying the processing of sensory information and

cognitive functions in 'real' time, and correspond to situational, on-line awareness (Toglia & Kirk, 2000). Performance monitoring was used to operationalise Consistent Consciousness, and is comparable to the self-monitoring aspect of on-line awareness (Toglia & Kirk, 2000), whilst the error monitoring paradigm employed by O'Keefe et al. (2007) is an alternative measure of on-line performance monitoring. Importantly, and in contrast to the null finding of a relationship between metacognitive knowledge and on-line awareness in O'Keefe et al. (2007), the current study found that metamemory perceptions and self-report of abilities were related to neural and behavioural aspects of performance monitoring. Despite the difference in results of relationships between metacognition and on-line monitoring abilities in the present study and in O'Keefe et al. (2007), the corresponding concepts in the different models suggest the existence of two types of awareness (cf. Toglia & Kirk, 2000); metacognitive self-awareness and on-line performance monitoring. The contribution of the HoP model (Stuss et al., 2001) is in the separation of on-line awareness into the component aspects of the initial processing of sensory and bodily information (Sensorimotor Awareness), and the ability to organise sensory input which underlies effective and adaptive behaviour (Consistent Consciousness).

The current study found some indirect evidence of the hierarchical structure of the HoP model, as correlations were found in both groups between Levels 2-4 of the model. Furthermore, evidence was found of deficits in the older adults at one level, being associated with deficits at a higher level. Reduced error processing in the older adults was also associated with the use of fewer strategies to support memory functioning, supporting the proposition that deficits at one level can be transmitted to higher levels. The operationalisations of the levels of awareness did not facilitate testing of the concept that higher levels could exert compensatory influence over lower levels. However, it is possible that the neuronal firing involved in less positive metamemory beliefs may be associated with the firing underlying reduced amplitude and longer latency ERP components. However, this is purely speculative as the neurophysiology underlying metacognition is not known. This conceptualisation of higher levels exerting compensatory influence on reduced functioning at lower levels is another example of descriptions being included into the HoP model to explain every possible outcome. This poses significant challenges to empirical investigation, and means that the model cannot be fully

tested. Future research could include functional imaging data and measures of white matter integrity, as it is not known from the current data whether there are any structural differences in the older adults which may have contributed to the differences found in the ERP components. Following the assumption that brain function underlies each type of awareness, provision for the measurement of how the regions involved in each type of awareness communicate would be helpful. Without such data, it is not possible to assess the concept of inter-connectivity, nor is it possible fully to test the HoP model. For example, a relationship was found between the N1 and the N2, the former ERP component is generated in the superior temporal plane, whereas the N2 is generated in the ACC. This relationship may be a product of the statistical analysis undertaken in this study, or there may be an association between the neural generators and/or the cognitions of attentional capture and conflict monitoring. Further studies could clarify such propositions. Nonetheless, the current study provides early and exciting indirect evidence for relationships between each type of awareness.

#### 7.4.1. Summary.

The value of the current study was that significant relationships were identified and provided statistical evidence for the inter-connectivity of the HoP model. The study found that reductions in attentional processing were similar across two different paradigms. The independent and significant impact of affect on perceptions of memory functioning and abilities was replicated in the current study. Most significantly, the study found, irrespective of age group, that neural error detection signals were associated with perceptions of control over memory abilities, and error processing with the use of internal mnemonic strategies and external memory aids, suggesting a link between brain functioning and Self-Awareness. Although the HoP model (Stuss et al., 2001) was developed to account for deficits in awareness in patients with brain lesions, the studies presented in this thesis have indicated that the model is also applicable to an investigation of different types of awareness in a healthy ageing population.

## Chapter 8. Conclusion.

The aim of this thesis was to investigate awareness in a healthy ageing population. Despite the historical development of awareness as a concept amenable to empirical investigation, and the incidence of deficits in awareness in diseases of ageing, the degree of change in awareness with normal ageing is not well established. Deficits in awareness are also found in neurological conditions (e.g. acquired and traumatic brain injury), lending validity to the close association between awareness and brain functioning.

Experimental psychology has proposed that awareness may be separated into levels or component parts, such as error monitoring. Further, contemporary neurological models of awareness (e.g. Stuss et al., 2001) have described specific processes underlying different forms of awareness. Any changes in awareness in older adulthood occur against a background of more general brain and cognitive change, and it may not be assumed that such changes influence all forms of awareness equally. The HoP model (Stuss et al., 2001) provided a theoretical framework for the investigation of different levels of awareness in healthy ageing. Cognitive neuroscience techniques were employed alongside more traditional measures of awareness to operationalise the conceptualisations of awareness described by Stuss et al. (2001).

This thesis has contributed to, and extended knowledge of awareness in ageing in four important ways. Firstly, this thesis has provided evidence of age-related differences in the measures chosen to represent the levels of awareness described by Stuss et al. (2001). Secondly, by utilising a multi-method approach to assessing awareness it was possible to demonstrate subtle effects of ageing (e.g. attenuation of selected ERP components), in the absence of any effect on other measures (e.g. neuropsychological tests). Thirdly, the thesis was unique in providing empirical support for inter-connectivity proposed in the HoP model, although this finding requires replication with larger numbers of participants. Fourthly, it was intended to demonstrate the efficacy of combining theoretical (HoP model) and neuroscientific (ERP) approaches in order to investigate a psychological concept considered by many to be too nebulous for scientific study. It was not the purpose of this thesis to confirm the definition

of awareness as described by Stuss and colleagues (2001), but to explore the feasibility of this model as a basis for interpreting multiple sources of data proposed to test awareness function in normal healthy ageing. As stated in the first chapter, the aim of this thesis was not to redefine awareness, but to operationalise conceptualisations of awareness to investigate different types of awareness in ageing. Despite the lack of empirical evidence currently available to support the theoretical basis for the inter-connectivity of the HoP model, the model provided specific definitions of awareness that were amenable to empirical investigation in healthy ageing. Although a multitude of awareness models has been developed, as discussed in Chapters 1 and 2, there were difficulties with all of the models.

The HoP model provided four descriptions of awareness, of which three were operationalised and assessed in the current thesis. It was assumed that the Arousal level of awareness was functional as all participants were awake when tested. This thesis investigated Sensorimotor Awareness through an investigation of auditory attentional processing; Consistent Consciousness was operationalised by performance monitoring, of which there are multiple aspects; and Self-Awareness was measured by self-report of current functioning and metamemory perceptions. The evidence provided within this thesis for each type of awareness can be found in Chapters 4-6.

Both younger and older adults similarly processed novel stimuli indicating that their awareness of their environment was functioning (see Chapter 4). In Chapter 5, the ERN component associated with the internal detection of errors in performance was found in both younger and older adults (albeit attenuated in the older adults), alongside equivalent behavioural performance of a complex executive task, and similar postdiction reports of performance indicating that both groups were able to monitor and were aware of their performance. As shown in Chapter 6, both younger and older adults reported some problems with their current functioning, indicating awareness of their personal abilities. The conclusion of this thesis is that awareness is a multidimensional construct, that includes (but not exclusively) on-line awareness which relates to immediate awareness of the environment and performance of tasks, and self-awareness which involves metacognitions and assessment of functions and abilities.

## 8.1. Age-related differences in Sensorimotor Awareness, Consistent Consciousness and Self-Awareness.

The HoP model (Stuss et al., 2001) is composed of four levels of awareness. The first level, Arousal, was assumed to be functional, due to the waking state of each participant.

Sensorimotor Awareness was operationalised by ERP components representing automatic and controlled attentional processes (Chapter 4), as attention is considered to be the process that directs information into consciousness (Baars, 1988). Behavioural and electrophysiological correlates of performance monitoring were employed to assess Consistent Consciousness (Chapter 5), whilst Self-Awareness was operationalised by self-report measures of awareness (see Chapter 6). As discussed in Chapters 4-6, within each level of the HoP model, subtle age-related differences were found in the measures of awareness. The main findings and implications from each study will be discussed in turn.

### 8.1.1. Sensorimotor Awareness (Level 2: HoP Model).

Sensorimotor Awareness in ageing was assessed by investigating two ERP components representing the initial capture of attention (N1), and the degree and timing of the allocation of attention to stimulus processing (P3), elicited by frequent standard stimuli, targets and novel, unexpected stimuli. Habituation to novel stimuli was also investigated. The study found that older adults had reduced N1 amplitudes, indicative of attenuated initial attentional capture, in particular for target stimuli. Previous studies using a standard oddball paradigm found that older adults had *higher* amplitudes to standard stimuli compared to younger adults, interpreted as increased attentional capture by irrelevant stimuli and a tendency to treat all stimuli as 'new' (Amenedo & Diaz, 1998; Anderer et al., 1996; Friedman & Simpson, 1994). However, studies using an auditory novelty oddball paradigm (Fabiani & Friedman, 1995; Friedman et al., 1993), found both younger and older adults processed frequent stimuli similarly. None of the previously cited studies reported the N1 to target or novel stimuli, which limited further comparison. In this respect, the current study extended knowledge of initial attentional capture

by investigating ERP components associated with target and novel stimuli in older and younger adults.

Target P3 amplitude at parietal regions, representing the amount of attention allocated to processing stimuli, was attenuated in older adults supporting earlier studies (Amenedo & Diaz, 1998; Chao & Knight, 1997b; Fabiani & Friedman, 1995; Friedman et al., 1993; Iragui et al., 1993; Polich, 1997; Weisz & Czigler, 2006). An increase in the frontal orientation of target P3 activity was also found in the older adults, consistent with previous studies (Amenedo & Diaz, 1998; Fabiani & Friedman, 1995; Friedman et al., 1993; Iragui et al., 1993). This frontal aspect of the target P3 in older adults has been suggested to represent an impairment in the ability to construct and sustain mental representations of stimuli (Fabiani & Friedman, 1995). Frontal-dominant topography of activity is also similar to that found for novel stimuli in both younger and older adults (Fabiani & Friedman, 1995; Friedman et al., 1993). Therefore, and consistent with Fabiani and Friedman (1995), even target stimuli may have been treated as 'novel' by the older adults, and/or target stimuli required greater levels of attention in older adults.

The latency of the novelty P3 was significantly delayed in the older adults, indicating a longer time to process novel stimuli, and possibly longer disengagement from processing such stimuli (Weisz and Czigler, 2006). Previous studies have not reported the P3 to standard stimuli, and the current study found that older adults had significantly increased P3 amplitude and shorter latency to standard stimuli compared to younger adults; suggesting a greater amount of attention is more quickly allocated to processing frequent, but irrelevant stimuli. Alongside the increase in frontal novelty P3 amplitude with novel stimuli repetition in older adults, this suggests that there is reduced ability to inhibit the processing of irrelevant stimuli. In other words, each stimulus may be treated more as 'new' than 'familiar' (cf. Amenedo & Diaz, 1998), but it is not confirmed if this is due to a failure of memory or an increased allocation of attention, or both.

The current study supported previous findings of a lack of habituation to novelty with increasing age (Friedman et al., 1997; Friedman & Simpson, 1994; Weisz & Czigler, 2006),

also suggesting that ageing alters neurocognitive processes associated with dynamic learning. Cycowicz and Friedman (1999) proposed that the novelty P3 was composed of two aspects: a frontal aspect representing initial orienting to novelty, and a parietal aspect associated with the categorisation of stimuli. The apparent differentiation of activity at frontal and parietal regions in the younger adults is compatible with this suggestion, and indicated that younger adults rapidly habituated to novel stimuli. Consistent with the literature, this suggests efficient learning in this group. The opposite *increase* in frontal activity with increasing experience of novel stimuli in older adults does not confirm the absence of efficient learning, but rather suggests that alternative pathways and/or increased use of existing pathways may underlie memory formation with age (Cabeza, 2002). Of particular importance was the finding that such phenomena are not necessarily explained by an increase in anxiety, nor were they associated with differences in behaviour.

In summary, with regard to age-related differences in Sensorimotor Awareness, the study found evidence that initial attentional capture was reduced in older adults, with a degree of bias towards irrelevant frequent stimuli, and an altered topographical distribution for controlled attention to processing target stimuli. Longer latency, controlled top-down processes may compensate for earlier (e.g. N1) deficits in sensory perception (e.g. Alain et al., 2004), and the increase in the frontal orientation of target and novelty P3 with repetition found in the older adults, in the absence of behavioural differences in target detection, provided support for this proposition.

#### 8.1.2. Consistent Consciousness (Level 3: HoP Model).

Consistent Consciousness is supported by the ability to organise sensory information underlying behaviour and executive functions. This study investigated performance monitoring in healthy ageing, as a means to explore the integrity of the third level of awareness in older adults. Performance monitoring included the assessment of pre-response conflict, allocation of attention, response-conflict priming, error detection, and error processing. The incompatible condition of the ERP paradigm manipulated task complexity and

response conflict. Behaviour was investigated in terms of error rate and reaction time, and the remedial actions of error correction and post-error slowing. The inclusion of postdiction report of performance accuracy provided a metacognitive measure of the individual's awareness of their performance. The study was unique in applying the Woody filter to investigate the potentially confounding effect of latency jitter on ERP component amplitudes.

The study found equivalent pre-response conflict monitoring across groups, however, the allocation of attention and response-priming were both attenuated and delayed in the older adults. The later SW component, which may reflect preparation for response switching, was also delayed in the older adults. In both conventional and Woody filter analyses of response-locked components, there were age-related reductions in the ERN and the Pe, indicating an impoverished error detection system, supporting previous studies (Band and Kok, 2000; Falkenstein et al., 2000; Falkenstein et al., 2001; Mathalon et al., 2003; Mathewson et al., 2005). The magnitude of the difference between the CRN and the ERN is considered to indicate the integrity of the error detection system (Falkenstein, 2004). Despite the amplitude reductions in these components when examined individually in the older adults, a significant difference remained between CRN-ERN amplitude indicating that the error-monitoring system was attenuated but functional. Of particular note were the lack of consistent amplitude reductions and latency increases in stimulus and response-locked components in the older adults. Therefore, the age-related differences found in the ERP components are unlikely to be explained by a global reduction in EEG power or a generalised slowing of processing.

Behaviourally, both groups managed the increase in task complexity equivalently, and had comparable rates of error correction and post-error slowing. However, the older adults consistently performed significantly more slowly than younger adults, and responded to fewer trials. It would, perhaps, be justified to reach a negative conclusion about older adults' ability based on this finding. However, a positive interpretation may also be offered, as slower responding may simply be a *different* response strategy in the older adults; a strategy that may contribute to the significant improvement in their performance over time, it is possible that older adults were taking a more general overview of the task, and thus had a strategy, albeit a different one to some of the younger adults. Moreover, although there were age-related

differences in the electrophysiological correlates of performance monitoring, there were few behavioural differences between younger and older adults. This suggests that any underlying 'deficit' or 'strategy-difference' was not sufficient to impair the behavioural performance of the task, and provides further evidence that altered ERP components in older adults may reflect adaptation to normal changes in brain structure and function, not necessarily deficit.

In summary, subtle age-related differences were found in electrophysiological aspects of performance monitoring, which may indicate altered Consistent Consciousness in older adults. However, effective remedial actions and response strategies were found, suggesting that performance monitoring was functional in the older adults, and that compensatory mechanisms may supersede challenges that occur earlier in the monitoring process. This presents a number of fascinating possibilities. Importantly, this is consistent with the possibility, as presented by Stuss and colleagues (2001), that other modules within an awareness 'level' may permit both continued functioning of that type of awareness, and continued inter-connectivity with other levels of awareness. In this respect the HoP model (Stuss et al., 2001) must be viewed as dynamic, as Stuss and colleagues appear to have intended. It would be of interest to investigate the development of awareness in childhood within the HoP model framework, in order further to understand changes in the processes underlying different types of awareness during ageing. For example, investigating whether children form each level sequentially, achieving Level 4 only after all other levels have developed. Indeed, Stuss and Alexander (2004) considered congenital and acquired brain injury in children, and suggested that frontal lobe damage particularly influenced development of Self-Awareness and theory of mind. The implications for ageing are that awareness may begin to 'deconstruct' at the higher levels first, as these levels rely on the functional integrity of the frontal lobes, but that the capacity of other modules within each level may permit adaptation to such changes in the brain.

### 8.1.3. Self-Awareness (Level 4: HoP Model).

Self-Awareness involves accurate monitoring of the self and personal abilities, and relies upon the functional integrity of the frontal lobes and inter-connectivity with the limbic system (Stuss et al., 2001). This study used multiple self-report measures, which focused on memory knowledge and capabilities, memory functioning in demanding situations, and cognitive and behavioural changes. The relationship between affect and measures of Self-Awareness was also investigated.

There were no age differences in any of the awareness measures specifically focusing on current functioning and abilities (MIA Capacity, DEX and MARS), which suggested that awareness of abilities did not alter as a function of age. Both groups reported some problems with cognitive or behavioural functioning, suggesting that both younger and older adults were aware of limitations in their abilities. However, age-related differences were found in the Change, Locus and Strategy subscales of the MIA, supporting previous studies indicating that older adults perceive more memory decline over time, less internalised control over their memory and report utilising more strategies to support memory performance (Dixon & Hultsch, 1983; Hertzog et al., 1987; Hultsch et al., 1987); the latter finding being consistent with the interpretation of Level 3 performance-monitoring results (see above, Section 8.1.2), that older adults had a different response strategy. The study additionally found that the age-related difference in perceived control over memory was explained by increased depression in the older adults. Furthermore, the current study found that older adults relied upon external memory aids to a greater extent than younger adults, providing empirical support for the numerical trend indicated by Dixon and Hultsch (1983).

The study extended knowledge regarding the influence of affect on metamemory perceptions in ageing. Anxiety and depression, as measured by the HADS, were related to increased anxiety and stress over memory, reduced memory capabilities, and more perceived change over time in memory functioning. These findings indicated that there was an emotional component to awareness of memory functioning, providing support for Abson and Rabbitt (1990; 1991) and McDougall (1995). One interpretation of these data is that awareness

function in ageing is influenced by the pre-conceptions that a particular society holds about ageing. For example, if it is assumed that ageing is associated with poorer memory function, irrespective of dementia, then ageing may be associated with the attribution of more significance to occasional memory lapses. It is conceivable that this could simultaneously lead to greater utilisation of memory strategies, and increased anxiety. 'Reduced' awareness may, therefore, result from a reaction to a belief as well as from normal brain changes with ageing. It would be of considerable interest to investigate changes in awareness with normal ageing in relation to personality characteristics (e.g. optimism), and in other societies where ageing is perhaps viewed more positively.

In summary, despite age-related differences in perceptions of change in memory functioning over time and the use of strategies to support memory performance, there were no differences in the self-report measures of Self-Awareness between younger and older adults. These findings suggest that increasing age has little effect on self-reports of Self-Awareness using traditional questionnaires. The study also suggested the importance of accounting for the role of emotion on self-report measures.

## 8.2. Multi-method Assessment of the Hierarchies of Processing Model.

As awareness is a multidimensional construct, the use of multiple measures of awareness is advantageous, as indicated by O'Keefe et al. (2007). This approach was particularly beneficial to investigating awareness in ageing, as consistent age-related differences in each measure of awareness within the levels of the HoP model were not found (see Chapters 4-6). Stuss et al. (2001) described each level of awareness as modular, which provided the inter-connectivity between the levels; this modular structure also explains how deficits at one level can be circumvented (as detailed in Chapters 2 and 7). However, this description also posed a challenge for testing the model, as it explained both findings and lack of findings of relationships between the levels in the HoP model. Therefore, it may only be assumed that lower level functioning supports higher level processes. The final empirical study (Chapter 7)

statistically explored the inter-connectivity between Levels 2-4 of the HoP model using correlation analyses.

The study found relationships in both groups between the measures of awareness at different levels, providing indirect support for the inter-connectivity of the HoP model. More specifically, increased attentional processing of auditory stimuli, operationalised to measure the processing of sensory information (Level 2 - Sensorimotor Awareness), was related to the similar processing of sensory stimuli in the more executive 4-CRT paradigm (Level 3 - Consistent Consciousness). This finding was consistent across groups, suggesting that sensory information was processed similarly in younger and older adults, irrespective of whether it was simplistic (Level 2: Sensorimotor Awareness) or more complex (Level 3: Consistent Consciousness). Relationships between electrophysiological and behavioural correlates of performance monitoring (Level 3 - Consistent Consciousness) and measures of Self-Awareness (Level 4 - Self-Awareness) were also found in both groups. Improved error detection, irrespective of group, was related to perceptions of increased control over memory functioning, whilst error detection, error processing and post-error slowing were related to the use of strategies to support memory performance.

This study was unique in assessing the HoP model (Stuss et al., 2001) in a healthy ageing population, but it was considered critical to do so in order to interpret altered awareness in diseases of ageing. Some evidence was found that changes in older adults at one level are related to changes at higher levels. For example, attenuated Pe amplitude was associated with the use of fewer strategies in the older adults. However, there were no consistent relationships across levels between those measures showing age-related differences within levels. This suggests support for the proposition that any altered awareness at lower levels can be circumvented by processes at higher levels. Furthermore, there were no behavioural performance differences between younger and older adults in the novelty auditory oddball, the 4-CRT, or in any of the cognitive measures (Levels 2 and 3). Moreover, this is the first study, to the author's knowledge, to identify that error detection (indexed by ERN amplitude) is related to perceived control over memory functioning. It also indicated that aspects of performance monitoring in general, such as error processing, may have influenced the use of

strategies to support memory functioning. Given the relatively small number of participants who on whom data were available across all the measures, however, it would be better to view these results as preliminary. Nonetheless, the current study provided evidence for relationships between measures operationalising different types of awareness, and indirect support for the inter-connectivity of the HoP model (Stuss et al., 2001) which is certainly worth replicating in a properly powered study.

### 8.3. Limitations of the Study.

The limitations of the thesis can be separated into issues relating to the lack of assessment of all Levels of the HoP model, operationalisations of the types of awareness, and methodological issues relating to power and representativeness of the older adult sample. These issues are discussed in turn.

#### 8.3.1. Lack of Formal Assessment of Level 1 of the HoP Model.

The lack of formal assessment of the Arousal level of awareness (Level 1) in the HoP model (Stuss et al., 2001) may be considered a limitation particularly in the inter-correlational analyses (Chapter 7). However, this type of awareness relates to the dimension from coma to waking state, and was assumed to be functional as the participants were wide awake at time of testing. Nevertheless, evidence discussed in Chapter 1 indicated that sleep disturbances are related to cognitive deficits, and this suggests the potential for problems in this type of awareness (e.g. daytime sleepiness) to influence processing at higher levels. The inclusion of a rigorous sleep questionnaire, for example the Functional Outcomes of Sleep Questionnaire (Weaver et al., 1997) and polysomnography (the gold standard of sleep quality assessment) would have provided multiple measures of quality of sleep with implications for daytime sleepiness. Such a study, if undertaken, would indicate the potential impact of sleep quality on higher levels of processing and functioning during the daytime, and provide data perhaps of interest to those working with older patients with altered awareness, for example, those with AD. Of note, it has already been reported that snoring, which limits sleep quality, is twice as

frequent in patients with dementia (AD  $n = 46$ ; Multi-Infarct Dementia  $n = 37$ ) compared to healthy age-similar controls ( $n = 124$ ) (Erkinjuntti, Partinen, Sulkava, Palomaki & Tilvis, 1987).

### 8.3.2. Operationalisation of Awareness Levels in the HoP Model.

The accurate monitoring of personal abilities and the self are described as the processes underlying Self-Awareness (Stuss et al., 2001), and were operationalised with self-report measures in this thesis. The present investigation of Self-Awareness found that both younger and older adults reported some problems with current functioning, and found evidence of age-related differences in perceptions of memory abilities and functioning. However, self-report measures are influenced by a range of psychological factors, including, as shown in Chapters 6 and 7, subtle variations in affect. The use of prediction and postdiction of performance on a range of neuropsychological tests would have provided a complementary addition to the assessment of Self-Awareness. Prediction and postdiction measures provide on-line evaluation and monitoring of performance and abilities, whilst the inclusion of a wider range of neuropsychological tests would have produced a greater assessment of current functioning. Moreover, the lack of data on working memory capabilities and frontal lobe functioning restricted exploration of potential associations with the multiple measures of the different types of awareness included herein. It must be recognised, however, that adding multiple additional measures may well have overburdened participants.

### 8.3.3. Power of the Study.

Although the current sample size was comparable to previous electrophysiological studies of ageing (see Chapters 4 and 5), the correlation analyses reported in Chapters 6 and 7 were underpowered, and in that each sample required 37+ participants to reveal strong correlations ( $r = .40+$ ) with an alpha level of .05, power .80. Further data collection was restricted in the current thesis by the amount of testing time required of each participant (~ 4 hours). The

generalisability of the findings from the older adult sample is also questionable because of the sample size, but moreover, the sample was recruited from the Older Adult Volunteer Panel managed by Dr. Romola Bucks at the School of Psychology. These older adults may represent a select group within the older adult population. Nevertheless, this is the first study to attempt to investigate the HoP model (Stuss et al., 2001) in this manner, so sample sizes could not be accurately predicted a priori. Importantly, it is hoped that these pilot data will permit future studies to be powered adequately to investigate relationships between levels of awareness, as described by Stuss et al. (2001), in normal ageing.

#### 8.4. General Conclusions.

The HoP model (Stuss et al., 2001) provided the theoretical framework for this thesis. Although the model was developed to account for deficits in awareness in patients with brain lesions, and has not, to the author's knowledge, been empirically tested, the studies presented in this thesis have indicated that the model is applicable to an investigation of different types of awareness in a healthy ageing population. Moreover, the conceptualisations employed to assess the HoP model may be considered to be useful and practical measures of awareness in younger and older adults.

The studies presented herein, have shown that ageing is associated with altered attentional capture, attenuated allocation of attention to stimuli in both a simple auditory oddball paradigm and in a more complex choice response task. Older adults have slower processing of novel stimuli, in addition to topographical evidence of reduced frontal lobe activity, and altered learning of novel stimuli. Slower attentional processing and response priming were additionally found in the 4-CRT. Both error detection and processing were reduced in the older adults, and this was related to their perceptions of control over memory functioning and the reported use of internal and external strategies to support current memory functioning. Although older adults perceived more change in their memory functioning over time, they did not report more difficulties with memory, or cognitive and behavioural functioning, compared to younger adults. The value of the theoretical framework provided by Stuss et al. (2001) may

lie, therefore, in the descriptions of the processes underlying the different types of awareness. These conceptualisations facilitated empirical assessment, and the current thesis employed multiple measures to operationalise the processes involved in each type of awareness. A multi-method approach to investigating awareness in ageing was found to be beneficial, as subtle and inconsistent age-related differences were found in each type of awareness. The studies indicated evidence for different types of awareness, and that some aspects of awareness are influenced by the ageing process. Some support was also found for the proposition that processing associated with functioning at lower levels was related to processing associated with higher levels of awareness.

Despite age-related differences in the amplitude and latency of ERP components representing attention and executive functions, there were few neuropsychological and behavioural differences between the groups. Significantly slower reaction times were only found in the older adults in the 4-CRT, a finding compatible with Falkenstein and his colleagues (Kolev et al., 2006; Falkenstein et al., 2006; Yordanova et al., 2004a), who also found that behavioural slowing in older adults only occurred in a 4-choice task, and not a simple choice task. Of particular note were the findings of the present study that older adults missed more trials in the 4-CRT, but that their performance improved with time and task experience, which contrasted with the younger adults, whose performance declined with time. It is suggested that the older adults up-regulated activity in attention and cognitive control circuits to compensate for alterations in the nature of information processing; that *slower facilitated surer*. Stuss et al. (2001) described the ability of higher levels to exert compensatory effects over lower level deficits in awareness. Investigation of the potential for cognitive strategies to supersede deficits in ERP components of performance monitoring would make for an interesting addition to the present study. Such evidence might indicate an influence of top-down cognitive processes in modulating dynamic changes in the arrangement of modular function within each level of awareness.

It remains unclear whether the alterations in awareness processes found in older adults have any significant influences on their everyday functioning. The equivalent management of the increase in task complexity in the 4-CRT, combined with similar cognitive and behavioural

performance across groups, suggest that altered functioning, as indexed by the ERP components, did not have a significant negative impact on performance. However, behaviour in computer-based ERP tasks may not be representative of everyday functioning (Hogan et al., 2006). The results, therefore, may not be suggestive of deficit but may be consistent with the conclusion that alteration in function is a means of adaptation to underlying brain and cognitive changes occurring during later life.

#### 8.5. Implications for Diseases of Ageing.

A range of neuropsychological and neurological models of awareness (see Sections 1.3.4.1 and 1.3.4.2, Chapter 1) has been developed to account for deficits in awareness found in patient populations, and has provided a multitude of definitions of different types of awareness. As such, clinical assessment of awareness has focused on deficits in awareness of specific functions rather than as part of a dynamic and potentially compensating system as promoted by the HoP model (Stuss et al., 2001). Several methods have been employed, including questionnaires, discrepancies between patient and carer ratings of functioning of various cognitive and behavioural domains and discrepancies between prediction and actual performance of neuropsychological tests and other experimental tasks (see Clare 2004b, for a review). Each of these types of assessments is dependent on assessment by the self (introspection) and/or others, and has the potential for influence by psychological factors such as affect, self-esteem and carer burden. Other factors may also impact on self-assessments, for example, current stress levels and fatigue. ERPs may provide a more objective measure that is complementary to the traditional subjective approach.

Another limitation of the clinical literature is that few studies have included healthy older adult populations as controls, restricting conclusions about the effects of disease pathology on awareness over and above any normal age-related changes. In other words, if the effects of normal ageing on awareness are not known, then it is not possible fully to understand deficits in awareness in diseases of ageing. Clinical awareness questionnaires were included in Chapter 6, and the study found that healthy younger and older adults reported some problems

with cognitive and behavioural functioning, and therefore had awareness of their limitations. Moreover, these results indicated that the awareness questionnaires were also appropriate for use with healthy populations, and that the inclusion of healthy older adults in clinical questionnaire studies of awareness would be feasible.

The ERP studies of Sensorimotor Awareness and Consistent Consciousness provided a more novel approach to the assessment of awareness, and are proposed to be complementary to the original questionnaire studies. The EEG technique is not reliant on self and/or informant reports, efficient memory recall, or, to a degree, cognitive capacity of the individual. Age-related differences in brain activity were found without associated performance deficits, suggesting that ERP components may also be less subject to the potential influence of psychological factors, such as affect and social desirability. Moreover, it was found that the measurement of electrical brain activity was acceptable to older adults, and, may be a valuable, non-invasive technique applicable for use in older adult patient populations.

ERP methodology may be particularly informative with regard to predicting decline in longitudinal studies of ageing. In this respect, it is interesting that age-related differences in the ERN and Pe were driven by differences between younger adults and the oldest older adults aged 70+ years (Chapter 5; and Appendix 6). If these components are confirmed as particularly sensitive to maturational change they may be candidate variables to track progression from mild cognitive impairment to AD, and from mild to moderate brain dysfunction in older adults with dementia. However, additional benefit of the ERP approach may derive from its combination with structural imaging data and neuropsychological assessments, rather than in isolation.

Older adulthood has been shown to be a time of neural and cognitive change (Sections 2.4 and 2.5, Chapter 2), and this thesis has shown that there are subtle changes in brain function underlying processes of awareness with ageing. Therefore, it cannot be assumed that any changes in awareness with diseases of ageing are occurring against a static background, rather, there are dynamic changes in organisation of neural networks underlying awareness. Normal ageing may be associated with compensatory mechanisms (Cabeza, 2002), and diseases of

ageing may become symptomatic only when the functions of these mechanisms are significantly disrupted by neuropathology.

The processes of plasticity may provide a neural explanation for such compensation, and the continued function of awareness in later life (cf. Arendt, 2001). In other words, there may be a neural explanation for why compensation is possible in normal ageing, but fails in diseases of ageing. Scheff and Price (2003) found that synaptic density was significantly reduced in layers III and V of the frontal and temporal lobes in individuals with AD. Synaptogenesis, facilitating the development or strengthening of alternative 'awareness' neural pathways, may be a compensatory mechanism that is deficient in AD (cf. Lassmann, 1996), as a reduction in synaptic density is not a typical feature of normal aging (Lassmann, 1996; Scheff, Price & Sparks, 2001). Mesulam (1999) suggested that failure of the mechanisms involved in plasticity (i.e. synaptogenesis) underlie AD and the relationship between the clinical and neuropathological characteristics of AD. Arendt (2001; 2004) further hypothesised that the effects of plasticity lie along a continuum, with beneficial cortical reorganisation at one end and neurodegeneration at the other.

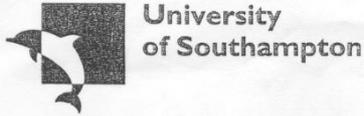
## 8.6. Awareness.

A range of models of awareness was described in Chapter 1 and, despite the different approaches and definitions of awareness, there is much commonality. In Chapter 7, the conceptualisation of a separation of awareness into two broad types was discussed: metacognitive self-awareness which relates to knowledge held about the self, and on-line situational awareness, which arises from experience of activity. This separation can be found in several of the models in Chapter 1. Awareness arising from knowledge held about the self is similar to: systemic awareness (Cavanaugh, 1989; Section 1.3.3, Chapter 1); the personal knowledge base (PKB: Agnew & Morris, 1998; Section 1.3.4.1.1, Chapter 1); metacognitive knowledge (Toglia and Kirk, 2000; Section 1.3.4.1.2, Chapter 1); Self-Awareness, at the highest level of the HoP model (Stuss et al., 2001; Section 1.3.4.2, Chapter 1); and, insight into the self in Vector 3 (Prigatano & Johnson, 2003). Awareness involving self-knowledge

also features in objective self-awareness theory (Duval & Wicklund, 1972; Section 1.3.2, Chapter 1), which states that objective self-awareness arises from self-focused attention. The conceptualisation of on-line awareness arising from experience of activity and performance monitoring is found within the metacognitive literature, in which this type of awareness is analogous to: memory monitoring (Hertzog & Dixon, 1994; Section 1.3.1, Chapter 1); subjective self-awareness (Duval & Wicklund, 1972; Section 1.3.2, Chapter 1); on-line awareness (Cavanaugh, 1989; Section 1.3.3, Chapter 1); situational awareness (Toglia & Kirk, 2000; Section 1.3.4.1.2, Chapter 1); Consistent Consciousness in the HoP model (Stuss et al., 2001). This concept also features in the Agnew and Morris (1998; Section 1.3.4.1.1, Chapter 1) model, as performance monitoring is the process which underlies mnemonic and executive anosognosia, the detection of a mismatch concerning functioning, and of errors. The commonalities in these concepts and definitions may be interpreted to indicate support for the separation of awareness into awareness about the self and on-line experiential awareness. However, such a conclusion is restricted by the lack of empirical investigation, nevertheless, the current thesis and the study by O'Keefe et al. (2007) offer some support for this proposition.

Defining the multi-dimensional nature of awareness has posed a significant challenge for research. Whilst there has been consideration for the concept of awareness as a process dependent on brain function (Prigatano & Johnson, 2003; Stuss et al., 2001), there has been little consideration of the complexity of awareness and its relationship with the brain, as this complexity is difficult to assess empirically. An alternative view is that awareness can be presumed as a factor that underlies the ability to perform everyday tasks, but that is not interesting in itself. In this view, the question of what constitutes awareness only becomes pertinent when problems with awareness develop, as is the case found in individuals with dementia. This thesis may not claim to have made a substantial contribution to age-old debates about awareness, but it has, perhaps, contributed to the view that awareness may be assessed by testing brain function, and that awareness is a multi-dimensional construct. The former is supported by evidence of alteration in brain ERP components with normal ageing, and the latter by evidence of selectivity in that alteration.

Appendix 1. Sponsorship Letter from the University of Southampton.



Legal Services

University of Southampton    Tel +44 (0)23 8059 4684  
Highfield    Fax +44 (0)23 8059 5781  
Southampton    Email legalservices@soton.ac.uk  
SO17 1BJ United Kingdom

Tel: +44 (0)23 80598848/9

Ref: RSO 4463

REC (if available)

Cassandra Richardson  
School of Psychology  
Building 44  
University of Southampton  
Southampton  
SO17 1BJ

26 May 2006

Dear Cassandra

**Project Title: Changes in awareness of cognitive deficits in older adults and individuals with Alzheimer's disease**

I am writing to confirm that the University of Southampton is prepared to act as sponsor for this study under the terms of the Department of Health Research Governance Framework for Health and Social Care (2001).

The University of Southampton fulfils the role of research sponsor in ensuring management, monitoring and reporting arrangements for research.

I understand that you will be acting as the Principal Investigator responsible for the daily management for this study, and that you will be providing regular reports on the progress of the study to the School on this basis.

I would like to take this opportunity to remind you of your responsibilities under the terms of the Research Governance Framework for researchers, principal investigators and research sponsors. These are included with this letter for your reference. In this regard if your project involves NHS patients or resources please send us a copy of your NHS REC and Trust approval letters when available.

Please do not hesitate to contact me should you require any additional information or support. May I also take this opportunity to wish you every success with your research.

Yours sincerely

A handwritten signature in black ink, appearing to read "MD", written over a printed signature line.

**Dr Martina Dorward**  
**Research Governance Manager**

cc. File  
Ruth McFadyen  
Supervisor/s: (if applicable)

Appendix 2. Participant Invitation Letter.



School of Psychology

University of Southampton Tel +44 (0)23 8059 5108  
Highfield Southampton Fax +44 (0)23 8059 4597  
SO17 1BJ Email cr2@soton.ac.uk

Dear (insert name),

Date:

My name is Cassie Richardson and I am a postgraduate research student at the University of Southampton. You are being invited to participate in a study investigating memory and awareness over the lifespan using event-related potentials (ERPs). This is a non-invasive technique which uses leads placed on the scalp to measure changes in electrical activity from the brain during task performance.

The study is designed in two parts. Both sessions will take between one and two hours. At the first visit I will ask you to complete a range of questions telling me about your mental and thinking skills as well as your mood. At the second visit, I will ask you to carry out some simple tasks whilst I measure your brain's electrical activity during those tasks. You can withdraw from the study at any time and all of the information that we collect about you will be kept completely confidential.

Please find enclosed an information sheet providing further details of the study. If you have questions please contact me Cassie Richardson on 023 8059 5108 (office number) or via email [cr2@soton.ac.uk](mailto:cr2@soton.ac.uk). If you are interested in participating in the study, please could you return the reply slip below in the enclosed FREEPOST envelope. I will telephone you to answer any additional questions you may have and to arrange the first appointment if you decide to go ahead.

Yours sincerely,

Cassie Richardson

---

Yes, I am interested in participating in the memory and awareness ERP study.

Name: \_\_\_\_\_

Address: \_\_\_\_\_

Telephone number: \_\_\_\_\_ Best time to contact me: \_\_\_\_\_



## **An investigation of brain activity associated with memory and awareness.**

### **Information Sheet**

You are being invited to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part.

Thank you for reading this information sheet.

### **What is the purpose of the study?**

We are investigating memory and awareness over the lifespan using event-related potentials (ERPs). The electroencephalogram (EEG) measures the electrical activity of the brain using leads placed on the scalp. The study will involve two appointments approximately 1 week apart and each visit will take 1 to 2 hours.

### **Why have I been chosen?**

We are writing to a number of participants of the correct age, chosen at random, from the volunteer panel.

### **Do I have to take part?**

It is up to you to decide whether or not to take part. If you do decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part you are still free to withdraw at any time and without giving a reason.

### **What will happen to me if I take part?**

The study will involve two appointments approximately 1 week apart. The first visit will involve completing a number of questionnaires, one of the questionnaires will be audio taped. A home visit can be arranged for the first appointment. With your

consent, we would also like to ask someone with whom you are close to complete some questionnaires about your abilities. The second visit will take place in the ERP room at the University of Southampton. In order to measure the electrical activity of your brain, a cap with holes in it will be placed on your head and leads will be placed on your scalp with a watery gel. The leads transmit the brain's electrical activity to the computer where it is recorded.

There is no risk involved; it is not possible for the leads to send electrical activity back to the brain. The leads can be removed in less than a minute if you decide you want to stop. Once the leads have been placed, you will be asked to complete some attention tasks on the computer. A short questionnaire about how you are feeling will be given at the beginning and at the end of the computer tasks. You will also be asked to complete four practical tasks; three everyday and one novel task. The second visit will also be videotaped.

I will endeavour to remove as much of the gel as possible at the end of the second visit, and you may wish to wash your hair when you get home. If you would prefer, there is a hairdresser on campus and we can arrange for you to have a wash and blow dry.

**What do I have to do?**

There are no special requirements of you.

**What are the possible disadvantages and risks of taking part?**

There are no significant disadvantages or risks involved in taking part in this study. Many people find the tasks fun if a little tiring.

The gel is salt based. It is harmless. However, to be certain, we will conduct a skin test to check that you do not react to it. This involves putting a tiny amount of gel on your hand to look for redness or itching.

**What are the possible benefits of taking part?**

There are no direct benefits to you from taking part in the study.

**What if something goes wrong?**

It is very unlikely that any part of this study will cause you harm. The study is entirely non invasive. However, if any aspect of the way you have been approached or treated in the course of the study causes you concern, please write to the project supervisor Dr. Romola Bucks at the School of Psychology, University of Southampton, Highfield, Southampton SO17 1BJ.

**Will my taking part in this study be kept confidential?**

All information collected about you during the course of the research will be kept strictly confidential. Personal information will not be released to or viewed by anyone other than researchers involved in this project. All of the data collected will be coded so that it is anonymous and will be stored securely. Results of this study will not include your name or any other identifying characteristics.

### **What will happen to the results of the research study?**

The results from the study will provide us with data that we intend to present within the School of Psychology, University of Southampton and an article will be submitted for publication. You will not be identified in any presentation of the data. A copy of the study findings can be provided by Cassie Richardson, on request.

### **Who is organising and funding the research?**

The Economic and Social Research Council, Gerald Kerkut Charitable Trust and The Alzheimer's Research Trust are funding the research and it is managed by the University of Southampton.

### **Who has reviewed the study?**

The study has been reviewed and approved by the University of Southampton School of Psychology Ethics Committee. If you have questions about your rights as a participant in this research, or if you feel that you have been placed at risk, you may contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, Southampton, SO17 1BJ.  
Phone: (023) 8059 3995.

### **Contact for Further Information:**

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Email: [cr2@soton.ac.uk](mailto:cr2@soton.ac.uk)

Appendix 4. Script for the Procedure.

**TELEPHONE RECRUITMENT**

**Hi, my name is Cassie Richardson and I'm a postgraduate student at the University of Southampton. Thank you for returning your reply slip expressing interest in taking part in this research. As I explained in the letter, I am studying awareness and memory and the associated brain activity.**

**The study is designed in two parts. Both sessions will take over an hour but less than two hours. You can withdraw from the study at any time and all of the information that we collect about you will be kept completely confidential. The information that we gather is purely for research purposes, and is in no way diagnostic.**

**At the first visit I will ask you to complete a range of questions telling me about your mental and thinking skills as well as your mood.**

**At the second visit, I will ask you to carry out some simple tasks whilst I measure your brain's electrical activity during those tasks. We measure brain activity using event related potentials or ERPs. These are the normal electrical signals that pass between the cells in your brain when you are doing mental tasks. We can measure this at the scalp by placing leads on your scalp. These leads pick up the signals that are produced by your brain, a bit like a receiver can pick up radio signals. However, they do not send signals into the brain. The system is totally non invasive and completely harmless. We use a salty gel to get a good connection with the skin. This is similar to the one used with ultrasound. The gel is harmless but tends to dry as white powder. This washes out easily. Are you able to get to the University? I can provide a map and directions and there is free parking near the University. I can also pay some travel expenses if you're not coming by car.**

**Can I just ask if you live with someone? (Older adults)**

**Is there someone who would be happy to be with you when you take part? (Participants with AD)**

**This is because at the first visit, I will want to ask that person some questions about your general mood and wellbeing. I hope that will be OK with you.**

**Each time we meet, I will explain what we are going to do and give you an opportunity to decide if you want to continue.**

**Are you still interested in taking part?**

**OK, do you have any questions? Answer any questions that have been asked.**

**Is it OK if I take about 10 minutes of your time to run through some screening questionnaires with you? By agreeing to answer these questions doesn't mean that you've consented to the study, it just means that you're happy for me to ask you some**

questions over the telephone. You are free to stop at any time and the information collected will be kept completely confidential.

If not a good time for telephone call: When is a good time for me to call you to run through the screening questionnaires?

### **OLDER ADULT SCREENING**

I'd like to go through a brief questionnaire about your memory and thinking skills. This is to give you a feel for the types of questions I'll be asking in the study. Some of the questions you may find simple, it's just how the questionnaire is written and I have to ask everybody the same questions.

### **TELE**

**What is your name?**

**How old are you?**

**When were you born?**

**What is today's date?**

**What month is it?**

**What year is it?**

**What day of the week is it?**

**What season is it?**

**Please repeat these words for me. KEY, TOOTHBRUSH, LAMP.**

Allow 3 trials.

**Please remember the words, as I'll ask you them again.**

**Could you count backwards from 20 by 3's?**

*\*If the person has problems, use the prompts "what is 20 take away 3?" and "and then if you take away 3 more?"*

**Who is the Current Prime Minister? (Tony Blair)**

**Who was the Prime Minister before them? (John Major)**

**A few minutes ago I asked you to remember 3 words. Could you tell me now what they were?**

*\*If the person failed recall question:*

*Which of these words did I tell you before: **Key?** (Y/N), **Ring?** (Y/N), **Chair?** (Y/N), **Picture?** (Y/N), **Toothbrush?** (Y/N), **Door?** (Y/N), **Pen?** (Y/N), **Table?** (Y/N), **Lamp?** (Y/N)*

Allow 3 trials, 1 for each word.

**Now, I am going to say two things that are similar to each other in one or more ways. I would like you to tell me the greatest similarity between them.**

**Orange & Banana.**

**Table & Chair.**

**That's great, thanks.**

**\*If the person scores less than 15 on the TELE:**

*How do you feel about the questions that I've just asked you? Do you still want to participate in the study? I ask this because, the study is long and quite challenging and I don't want to cause you any stress. Both visits involve longer and more complex questionnaires than the one I've just asked you.*

**\*If the person has any queries about their memory or performance:**

*If you have any concerns about your memory, your GP is the best person to speak to. Alternatively you can speak to my supervisor Dr. Romola Bucks about any concerns you may have.*

**\*If the person wishes to withdraw:**

*Thank you for taking the time to find out more about the study. This does not mean you cannot participate in other studies in the future. We may contact you in a little while to tell you about other studies we are conducting. If you like the sound of any of them, we would love to hear from you.*

**I'd also like to ask you some questions about your health, if that's OK?**

#### **PARTICIPANT DEMOGRAPHIC FORM**

**What is your ethnic group?**

**Are you right or left handed?**

**What is your occupation (or previous occupation)?**

**How old were you when you started school?**

**How old were you when you left school:**

**Did you have any further education?**

**Do you wear reading glasses?**

**Do you wear a hearing aid? Does it whistle at all?**

**Are you currently on any medication?**

**How long have you been taking your medication?**

**Have any of your tablets or any doses changed in the last 3 months?**

**Do you have any medical conditions such as epilepsy?**

**Do you have any skin conditions or sensitive skin?**

**Have you ever been treated for depression or any other mental health problem?**

**Have you ever had a head injury?**

**Do you have any mobility problems? E.g. a walking aid or wheelchair.**

**Do you have any experience of using computers?**

**\*If the person has epilepsy:**

*I'm really sorry but you won't be able to take part in the study. This is because the ERP study includes flashing stimulus and we have strict criteria about who can take part in the study. It is unethical of us to put you at any risk whatsoever. I'd like to thank you for your time and for your interest in the study.*

**\*If the person has a diagnosed skin condition:**

*I'm really sorry but you won't be able to take part in the study. This is because we use a gel for the EEG recording that a small number of people are allergic to and we have strict criteria about who can take part in the study. It is unethical of us to put you at any risk whatsoever. I'd like to thank you for your time and for your interest in the study.*

**\*If the person has sensitive skin:**

*A small number of people are allergic to the gel that we use for the EEG recording. If you're happy to take part in the study I will do a skin test at the first visit to see if we can use it on you. The ingredients of the gel are water, aluminium oxide, 1,2 propanediol, sodium polyacrylate, methylparaben, propylparaben, FD & C Blue 1, FD & C Red 40 and FD & C Yellow 5.*

**\*If the person has any of the conditions on the checklist:**

*I'm really sorry but you won't be able to take part in the study. This is because the EEG is very sensitive and we have strict criteria about including people with [give health condition]. I'd like to thank you for your time and your interest in the study.*

**\*If the person has uncorrected hearing problems or a whistling hearing aid:**

*I'm really sorry but you won't be able to take part in the study. This is because one of the tasks in the ERP study is an auditory one, in which you need to carefully listen to a series of tones. I'd like to thank you for your time and for your interest in the study.*

**\*If the person has uncorrected vision problems:**

*I'm really sorry but you won't be able to take part in the study. This is because there are a number of tasks in the ERP study that require you concentrating on visual images and responding to them quickly. I'd like to thank you for your time and for your interest in the study.*

**\*If the person has started any CNS medication in the last three months:**

*I'm really sorry but you won't be able to take part in the study at the present time. This is because we have strict criteria about who can take part in the study. Because you have started taking medication in the last three months, we need to wait until you have been taking it for three months. If you are still happy to take part, is it OK if I give you a call to rearrange a visit once you have been taking the medication for three months? I'm really sorry to have inconvenienced you.*

**\*If person queries the three month period:**

*This is a standard procedure and it is so that your medication is stable.*

**\*If the person has stopped taking CNS medication in the last three months:**

*I'm really sorry but you won't be able to take part in the study at the present time. This is because it is part of our study criteria that people have a wash-out period of three months after stopping taking medication. This is so that the medication is completely out of your system before you take part. I'm really sorry to have caused you any inconvenience. If you are still happy to take part, is it OK if I give you a call to rearrange a visit once you have not been taking the medication for three months? I'm really sorry to have inconvenienced you.*

**That's great, thanks for your time. Can we arrange the dates for both visits now? It makes life easier that way. Are you able to get to the University for both visits? Arrange venue for first appointment, if person cannot travel to the University for both visits. I'll send you a letter confirming the time, date and venue for the first visit.**

## **VISIT 1**

### **INTRODUCTION**

**Hi, my name is Cassie Richardson and I'm a postgraduate student at the University of Southampton. Thank you for agreeing to take part in this research. I am studying awareness and memory and the associated brain activity.**

**As I explained [on the telephone], the study is designed in two parts. This is because there are a number of questionnaires that I would like to go through with you, in addition to the brain activity study. Both sessions will take approximately one to two hours. You can withdraw from the study at any time and all of the information that we collect about you will be kept completely confidential. The information that we gather is purely for research purposes, and is in no way diagnostic.**

**\*If the person asks any questions about their memory:**

***If you have any concerns about your memory, your GP is the best person to speak to.***

**I am going to ask you to complete some questionnaires and mental tasks today. There are eleven questionnaires to go through and they vary in length. The tasks measure thinking and memory skills, and involve words, pictures and numbers. I will explain each one to you before it begins.**

**If you want to stop at any time please let me know, and we can take a short break in the middle of the session, if you would like. You are not obliged to continue if you don't want to. Any information you give me is strictly confidential, unless of course you are going to harm yourself or someone else. As this is illegal, I would have to tell somebody. Your name will not be linked to any of your results. This means that I won't be able to give you any information about how you did.**

**Do you have any questions about the research so far? Answer any questions that have been asked.**

**All of this is in the information sheet I sent you. I have another copy here. Please take a few moments to reread it.**

**Any questions? OK?**

### **CONSENT**

**Before we begin with the study and if you are still happy to go on, I will need you to sign this consent form for me. It says that you agree to take part in the research, that you are**

**aware of your rights to confidentiality, that you can withdraw at any stage, and that I have explained what is being asked of you.**

Sign consent form.

**This is your copy to keep.** Hand participant the consent form.

\*If the person wishes to withdraw:

*Thank you for taking the time to find out more about the study. Deciding not to participate today does not mean you cannot participate in this or any other study in the future. We may contact you in a little while to tell you about other studies we are conducting. If you like the sound of any of them, we would love to hear from you. Please remember, just let us know if you change your mind about being contacted in the future.*

To informant:

**Please could you also sign an assent form? The form says that you understand that your relative has consented (or not consented depending on participant's response) to you answering some questions about their abilities, that you have assented to their participation in the study and that you're aware that they and you can withdraw from the study at any time, without giving a reason.**

Relative to sign assent form

**This is your copy to keep.** Hand the relative the assent form.

**Here are the questionnaires that I would like you complete for me. Please can you read the instructions at the top of each one. If you have any queries, I'll answer them as soon as I have run through the questionnaires with your relative. Is that OK?** Give the relative the DEX questionnaire, and MARS. **There are two questionnaires that I would like to go through with you and I'll do that at the end of the visit, if that's OK?**

### MMSE

**I'm going to ask you some questions that assess your memory and thinking skills. Some of the questions you may find simple, please don't be insulted it's how the questionnaire is written and I have to ask everybody the same questions. Some of the questions may be a bit more challenging, please have a go at answering every question. If you're not sure about any of the questions, then just have a guess.**

**What is today's date?**

\*If the person cannot give the correct date straight away then move straight on to the next question. You can come back to an orientation question if you think the person knew the answer but was so nervous they could not get it the first time. Accept answers that are given later. Say: **If it comes back to you later, do let me know.** For any of the following questions, if the person has any problems recalling the information, give them a bit of thinking time before moving onto the next question.

**What is the year?**  
**What is the month?**  
**What day of the week is it?**  
**What season is it?**  
**What is the name of this place?**  
**What floor are we on?**  
**What city are we in?**  
**What county are we in?**  
**What country are we in?**

**I'm going to say some words. Listen carefully, and when I have said all of the words, please repeat them to me. BALL, FLAG, TREE.**

\*If the person has problems recalling the words:

*I'll say the words and then you repeat them, OK? Repeat BALL, FLAG, TREE. If appropriate apologise for poor pronunciation. Repeat the words until the person can correctly repeat them, or stop after 3 trials. Use positive encouragement whenever appropriate.*

**I'd like you to do some mental arithmetic for me. Starting with 100, take away 7. Take away 7 again, and again, keep going. Stop at 65.**

\*If the person initially refuses:

*Are you happy to have a go? We can stop whenever you want. If person is happy to try use the prompt below. If person refuses again, move onto next question.*

\*\*If the person has problems, then take the last answer they have given and say "*and take seven away from...*" and repeat until 65 or if the person is having difficulties move onto the next question.

**I'd now like you to spell the word WORLD backwards.**

\*If the person asks for clarification of the word: *World, as in the world we live in.*

\*\*If the person spells WORLD forwards, say: **now can you spell it backwards?** Write down a) WORLD, b) whatever backwards spelling they gave, e.g. DLROW.

Score both serial 7s and WORLD backwards. USE THE SERIAL 7s SCORE.

**Can you say the words that I asked you to repeat earlier?**

\*If the person queries that you didn't say earlier that they needed to remember the words: *You're right, I didn't tell you that you needed to remember the words!*

**What is this called?** Show wristwatch.

**And what is this called?** Show pencil.

**Please repeat this sentence?** Pause and make eye contact. **NO IFS, ANDS OR BUTS.**

**Listen carefully and then do as I say, I can only say this once.** Pause and make eye contact.  
**Take the paper in your right hand, fold it in half, and put it on your knee.**

**Read this sentence and do what it says?** Show CLOSE YOUR EYES.

\*If the person looks unsure about task:

*Read the sentence out loud for me?*

\*\*If they read it but fail to perform task:

*Do what it says?* Move on if person is still having problems with task.

**Please write a sentence for me?**

\*If the person requests clarification:

*Write anything you like as long as it makes sense.* If you can't read the writing, ask the person to read out the sentence for you.

**Copy this diagram for me?**

\*If the person looks unsure or refuses:

*It doesn't have to be perfect, just get all the corners in.* If person refuses then move onto next questionnaire.

**That's great thanks. Can I just ask when you started and finished school?**

Note the years of education. See below for MMSE cut-off scores.

Age	9-12yrs	13+
60-64	25	26
65-69	25	27
70-74	24	25
75-79	24	25
80-84	21	25
85+	22	24

\*If the person scores less than the MMSE cut-off for their age and level of education:

*How do you feel about the questions that I've just asked you? Do you still want to participate in the study? Given the difficulties you've had with the simple versions of the questionnaires, I don't think this is an appropriate study for you to take part in. The study is long and quite challenging and I don't want to cause you any stress by asking you to complete complex and difficult tasks and questionnaires.*

\*If the person has any queries about their memory or performance:

*If you have any concerns about your memory, your GP is the best person to speak to.*

*Alternatively you can speak to my supervisor Dr. Romola Bucks about any concerns you may have.*

\*If the person wishes to withdraw:

*Thank you for taking the time to find out more about the study. This does not mean you cannot participate in other studies in the future. We may contact you in a little while to tell you about other studies we are conducting. If you like the sound of any of them, we would love to hear from you.*

### **MoCA**

**I'm going to ask you some more questions that assess your memory and thinking skills. Some of the questions you may find simple, please don't be insulted it's how the questionnaire is written and I have to ask everybody the same questions. Some of the questions may be a bit more challenging, please have a go at answering every question. If you're not sure about any of the questions, then just have a guess.**

**Please draw a line, going from a number to a letter in ascending order. Begin here point to 1 and draw a line from 1 then to A and then to 2 and so on. End here point to E.**

Scoring: allocate 1 point if the participant successfully draws the following pattern: 1-A-2-B-3-C-4-D-5-E, without drawing any lines that cross. Any error that is not immediately self-corrected earns a score of 0.

Point to the cube. **Copy this drawing as accurately as you can, in the space below.**

Scoring: 1 point is allocated for a correctly executed drawing.

- Drawing must be three-dimensional
- All lines are drawn
- No line is added
- Lines are relatively parallel and their length is similar (rectangular prisms are accepted)

A point is not assigned if any of the above-criteria are not met.

Indicate in the right third space and give the following instructions:

**Draw a clock. Put in all the numbers and set the time to 10 after 11.**

Scoring: One point is allocated for each of the following criteria:

- Contour (1 pt.): the clock face must be a circle with only minor distortion acceptable (e.g. slight imperfection on closing the circle)
- Numbers (1 pt.): all clock numbers must be present with no additional numbers; numbers must be in the correct order and placed in the approximate quadrants on the clock face; Roman numerals are acceptable; numbers can be placed outside the circle contour
- Hands (1 pt.): there must be two hands jointly indicating the correct time; the hour hand must be clearly shorter than the minute hand; hands must be centred within the clock face with their junction close to the clock centre

A point is not assigned for any given element if any of the above criteria are not met.

Beginning on the left, point to each figure and say: **Tell me the name of this animal.**

Scoring: One point is given for the following responses: (1) lion, (2) rhinoceros or rhino, (3) camel or dromedary.

The examiner reads a list of 5 words at a rate of one per second, giving the following instructions:

**This is a memory test. I am going to read a list of words that you will have to remember later on. Listen carefully. When I am through, tell me as many words as you can remember. It doesn't matter in what order you say them.** Mark a check in the allocated space for each word the participant produces on the first trial. When the participant indicates that (s)he has finished (has recalled of the words), or can recall no more words, read the list a second with the following instructions: **I am going to read the same list for a second time. Try to remember and tell me as many of the words as you can, including the words you said the first time.** Put a check in the allocated space for each word the subject recalls after the second trial.

At the end of the second trial, inform the subject the (s)he will be asked to recall these words again by saying, **I will ask you to recall those words again at the end of the test.**

Scoring: No points are given for Trials One and Two.

**I am going to say some numbers and when I am through, repeat them to me exactly as I said them.** Read the five number sequence at a rate of one digit per second.

**Now I am going to say some more numbers, but when I am through you must repeat them to me in the backwards order.** Read the three number sequence at a rate of one digit per second.

Scoring: Allocate one point for each sequence correctly repeated (the correct response for the backward trial is 2-4-7).

The examiner reads the list of letters at a rate of one per second, after giving the following instruction:

**I am going to read a sequence of letters. Every time I say the letter A, tap your hand once. If I say a different letter, do not tap your hand.**

Scoring: Give one point if there is zero to one errors (an error is a tap on a wrong letter or a failure to tap on letter A).

The examiner gives the following instruction: **Now, I will ask you to count by subtracting seven from 45, and then, keep subtracting seven from your answer until I tell you to stop.** Give this instruction twice if necessary.

Scoring: This item is scored out of 3 points. Give no (0) points for no correct subtractions, 1 point for one correct subtraction, 2 points for two-to-three correct subtractions, and 3 points if the participants successfully makes four or five correct subtractions. Count each correct subtraction of 7 beginning at 45. Each subtraction is evaluated independently; that is, if the participant responds with an incorrect number but continues to correctly subtract 7 from it,

give a point for each correct subtraction. For example, a participant may respond “37 – 30 – 23 – 16 – 9” where the “37” is incorrect, but all subsequent numbers are subtracted correctly. This is one error and the item would be given a score of 3.

The examiner gives the following instructions: **I am going to read you a sentence. Repeat it after me, exactly as I say it** [pause]: *I only know that John is the one to help today.* Following the response, say: **Now I am going to read you another sentence. Repeat it after me, exactly as I say it** [pause]: *The cat always hid under the couch when dogs were in the room.*

Scoring: Allocate 1 point for each sentence correctly repeated. Repetition must be exact. Be alert for errors that are omissions (e.g. omitting “only”, “always”) and substitutions/additions (e.g. “John is the one who helped today”; substituting “hides” for “hid”, altering plurals, etc.).

The examiner gives the following instruction: **Tell me as many words as you can think of that begin with a certain letter of the alphabet that I will tell you in a moment. You can say any kind of word you want, except for proper nouns (like Bob or Boston), numbers, or words that begin with the same sound but have a different suffix, for example, love, lover, loving. I will tell you to stop after one minute. Are you ready?** [Pause] **Now, tell me as many words as you can think of that begin with the letter F.** [Time for 60 seconds]. **Stop.**

Scoring: Allocate one point if the participant generates 11 words or more 60 seconds. Record the participant’s response in the bottom or side margins.

The examiner asks the participant to explain what each pair of words has in common, starting with the example: **Tell me how an orange and a banana are alike.** If the participant answers in a concrete manner, then say only one additional time: **Tell me another way in which those items are alike.** If the participant does not give the appropriate response (*fruit*), say: **Yes, and they are also both fruit.** Do not give any additional instructions or clarifications.

After the practice trial, say: **Now, tell me how a train and a bicycle are alike.** Following the response, administer the second trial, saying: **Now tell me how a ruler and a watch are alike.** Do not give any additional instructions or prompts.

Scoring: Only the last two item pairs are scored. Give 1 point to each item pair correctly answered. The following responses are acceptable:

Train-bicycle = means of transportation, means of travelling, you take trips in both

Ruler-watch = measuring instruments, used to measure.

The following responses are NOT acceptable: train-bicycle = they have wheels, ruler-watch = they have numbers.

The examiner gives the following instruction: **I read some words to you earlier, which I asked you to remember. Tell me as many of those words as you can remember.** Make a check mark (tick) for each word correctly recalled spontaneously without any cues, in the allocated space.

Scoring: Allocate 1 point for each word recalled freely without any cues.

Take orientation responses from the answers given previously in the MMSE to avoid repeating the same questions.

Total Score: Sum all subscores listed on the right hand side. Add one point for an individual who has 12 years or fewer of formal education, for a possible maximum of 30 points. A final total score of 26 and above is considered normal.

\*If the person scores less than the cut-off:

*How do you feel about the questions that I've just asked you? Do you still want to participate in the study? Given the difficulties you've had with the simple versions of the questionnaires, I don't think this is an appropriate study for you to take part in. The study is long and quite challenging and I don't want to cause you any stress by asking you to complete complex and difficult tasks and questionnaires.*

\*If the person has any queries about their memory or performance:

*If you have any concerns about your memory, your GP is the best person to speak to. Alternatively you can speak to my supervisor Dr. Romola Bucks about any concerns you may have.*

\*If the person wishes to withdraw:

*Thank you for taking the time to find out more about the study. This does not mean you cannot participate in other studies in the future. We may contact you in a little while to tell you about other studies we are conducting. If you like the sound of any of them, we would love to hear from you.*

### **PARTICIPANT DEMOGRAPHIC FORM** (Younger adults)

**I'm going to ask you some questions about yourself and your health.**

**What is your date of birth?**

**What is your ethnic group?**

**Are you right or left handed?**

**What is your occupation (or previous occupation)?**

**How old were you when you started school?**

**How old were you when you left school:**

**Did you have any further education?**

**Do you wear reading glasses?**

**Do you wear a hearing aid? Does it whistle at all?**

**Are you currently on any medication?**

**How long have you been taking your medication?**

**Have any of your tablets or any doses changed in the last 3 months?**

**Do you have any medical conditions such as epilepsy?**

**Do you have any skin conditions or sensitive skin?**

**Have you ever been treated for depression or any other mental health problem?**

**Have you ever had a head injury?**

**Do you have any mobility problems? E.g. a walking aid or wheelchair.  
Do you have any experience of using computers?**

\*If the person has epilepsy:

*I'm really sorry but you won't be able to take part in the study. This is because the ERP study includes flashing stimulus and we have strict criteria about who can take part in the study. It is unethical of us to put you at any risk whatsoever. I'd like to thank you for your time and for your interest in the study.*

\*If the person has a diagnosed skin condition:

*I'm really sorry but you won't be able to take part in the study. This is because we use a gel for the EEG recording that a small number of people are allergic to and we have strict criteria about who can take part in the study. It is unethical of us to put you at any risk whatsoever. I'd like to thank you for your time and for your interest in the study.*

\*If the person has sensitive skin:

*A small number of people are allergic to the gel that we use for the EEG recording. If you're happy to take part in the study I will do a skin test at the first visit to see if we can use it on you. The ingredients of the gel are water, aluminium oxide, 1,2 propanediol, sodium polyacrylate, methylparaben, propylparaben, FD & C Blue 1, FD & C Red 40 and FD & C Yellow 5.*

\*If the person has any of the conditions on the checklist:

*I'm really sorry but you won't be able to take part in the study. This is because the EEG is very sensitive and we have strict criteria about including people with [give health condition]. I'd like to thank you for your time and your interest in the study.*

\*If the person has uncorrected hearing problems or a whistling hearing aid:

*I'm really sorry but you won't be able to take part in the study. This is because one of the tasks in the ERP study is an auditory one, in which you need to carefully listen to a series of tones. I'd like to thank you for your time and for your interest in the study.*

\*If the person has uncorrected vision problems:

*I'm really sorry but you won't be able to take part in the study. This is because there are a number of tasks in the ERP study that require you concentrating on visual images and responding to them quickly. I'd like to thank you for your time and for your interest in the study.*

\*If the person has started any CNS medication in the last three months:

*I'm really sorry but you won't be able to take part in the study at the present time. This is because we have strict criteria about who can take part in the study. Because you have started taking medication in the last three months, we need to wait until you have been taking it for three months. If you are still happy to take part, is it OK if I give you a call to rearrange a visit*

*once you have been taking the medication for three months? I'm really sorry to have inconvenienced you.*

*\*If person queries the three month period:*

*This is a standard procedure and it is so that your medication is stable.*

*\*If the person has stopped taking CNS medication in the last three months:*

*I'm really sorry but you won't be able to take part in the study at the present time. This is because it is part of our study criteria that people have a wash-out period of three months after stopping taking medication. This is so that the medication is completely out of your system before you take part. I'm really sorry to have caused you any inconvenience. If you are still happy to take part, is it OK if I give you a call to rearrange a visit once you have not been taking the medication for three months? I'm really sorry to have inconvenienced you.*

### **The Autobiographical Memory Interview**

**Are you happy for me to tape record the next questionnaire? I would like to tape it because it's all about your life and rather than take notes, I'd like to listen to you. It is an interview in which I am going to ask you questions about your earlier life. The questions will concern your school days, early adult life, and more recent times. If you are ready we will begin.**

**What is your full name?**

**Your date of birth?**

**How old are you?**

**Where were you born?**

**1.1 Where did you live before you went to school?**

**1.2 Name three friends or neighbours from before you went to school.**

**A1 Tell me about an incident before you went to school? You may use the prompts "Your first memory?" or "Involving a brother or sister?"**

**2.1 What was the name of the first school you attended? If the person attended several schools between the ages of 5 and 11, the details of only the first one are required.**

**2.2 Where was this school? Town or city is sufficient.**

**2.3 How old were you when you started at this school?**

**2.4 Where did you live when you started at this school? This may or may not be the same as the answer given for question 1.1. Repeated answers should be checked as far as possible for correctness.**

**2.5 Name three teachers or friends from this school.** It is not acceptable for the subject to repeat the names of friends previously given. You may use the prompts “**The headteacher?**” or “**Your form teacher?**” or “**A friend?**”

**A2 Tell me about an incident which occurred while you were at primary school?** You may use the prompts “**Involving a teacher?**”, or “**Involving a friend?**” If it becomes evident that this memory is from a later period (e.g. secondary school), the memory is unacceptable.

**3.1 What was the name of the secondary school you went to?** If the person attended several secondary schools ask which one was attended when the subject was 13 years old. If the person only ever attended one school, it can be scored twice if correct, but separate incidents must be given for primary and secondary schools, with a total of six names of friends and teachers.

**3.2 Where was this secondary school?** Town or city is sufficient.

**3.3 How many examinations did you pass when you finished secondary school?** Specific subjects and grades are not required. Examples of acceptable responses: “Two A levels” or “five O levels” or “Didn’t get any”.

**3.4 What was your address when you started at this secondary school?** This may or may not be the same as the answer given for 1.1 and 2.4.

**3.5 Name three teachers or friends from this secondary school.** You may use the prompts “**The headteacher?**” or “**Your form teacher?**” or “**A friend?**” It is not acceptable for the person to repeat the names of friends previously given.

**A3 Tell me about an incident which occurred while you were at secondary school?** You may use the prompts “**Involving a teacher?**” or “**Involving a friend?**” If it becomes evident that this memory is from a different period (e.g. 5 – 11 years), the memory is unacceptable.

**4.1 Did you obtain any qualifications after leaving secondary school?**

**4.2 IF YES: Tell me the name of the course and the name of the institution?**

**IF NO: Tell me what your first job was and the name of the firm or organisation that you worked for?**

**4.3 What was your address when you obtained the qualification(s) or started your first job?** A repeated address should be checked for accuracy.

**4.4 Name three friends or colleagues from this period in your life.** You may use the prompts “**The Principal?**” or “**The boss?**”, “**The tutor/lecturer/teacher?**” or “**Your foreman?**”, “**Any class-mates?**” or “**Any work-mates?**” It is not acceptable for the person to repeat the names of friends given previously.

**A4 Tell me about an incident from college or from your first job?** You may use the prompts “**Your first day at work or college?**” or “**An incident with a friend?**” If the person has never been employed or had a formal training, he/she should be asked for an incident from the first few years after leaving school. If it becomes evident that this memory is from a different period (e.g. while the person was in their forties), the memory is unacceptable.

**5.1 Were you married in your late teens, twenties, or thirties?**

**IF YES: When did you get married and where the marriage ceremony was held?** Town or city is sufficient.

**IF NO: What was the name of someone else’s wedding that you attended during your twenties and where did this marriage ceremony take place?** Town or city is sufficient.

**5.2 What was your address before this wedding?**

**5.3 What was your address immediately after this wedding?** If the person repeats the address given in 5.2, the interviewer should check that the address had not changed.

**5.4 What was the name of the best-man from this wedding?** If there was no best-man, the name of a guest is sufficient.

**5.5 What was the name of a bridesmaid from this wedding?** If there was no bridesmaid, the name of a guest is sufficient.

**5.6 What was the maiden name of the bride from this wedding?** This may, of course, be the person’s maiden name.

**A5 Tell me about an incident from this wedding or from any wedding which you attended in your twenties?** You may use the prompts “**An incident involving a guest at the wedding?**” or “**An incident at the reception?**” As in other Autobiographical incident questions it is particularly important to ask the person to give details of a specific incident rather than a general description of the wedding. In the rare instances in which a person may have never attended a wedding (e.g. very young amnesiac patients), a score may have to be obtained by averaging the person’s scores for A4 and A6. However, this should be avoided if at all possible.

**6.1 What is the name of your first child?** Where the person does not have any children, he or she should be asked about a niece or nephew, or, failing that, the child of a close friend.

**6.2 What is the birth date of this child?** The year is sufficient. In the case of a nephew, niece, or child of a close friend, the age of the child is acceptable.

**6.3 Where this child was born?** Town or city is sufficient.

**6.4 What is the name of your second child?** Where the person does not have a second child, he or she should be asked about a niece or nephew, or, failing that, the child of a close friend.

**6.5 What is the birth date of this child?** The year is sufficient. In the case of a nephew, niece, or child of a close friend, the age of the child is acceptable.

**6.6 Where this child was born?** Town or city is sufficient.

**A6 Tell me about a first encounter with someone while in your twenties?** The first encounter with the spouse is acceptable. You may use the prompts “**Meeting someone in an interview?**” or “**Meeting someone on holiday or at work?**”

**7.1 Where we are?** Hospital, research institute or own home.

**7.2 What is the location of this place?** Town or city is sufficient.

**7.3 What was the date when you moved to this location?** Month required, if arrived within the last 12 months. Otherwise, year only is sufficient.

**7.4 What is your current address?**

**7.5 Name three current neighbours or colleagues.**

**A7 Tell me about an incident from a previous hospital visit?** You may use the prompts “**Involving the other patients?**” or “**To do with the doctors or nurses?**”

**8.1 What is the name of the last hospital which you attended or visited?** Failing this, ask the person for the name of the last doctor, dentist or optician he or she visited within the last five years.

**8.2 Where is this hospital, or practitioner?** Town or city is sufficient.

**8.3 What was the date of this hospital visit?** Month is within the last 12 months. Otherwise, year is sufficient.

**8.4 What was your address when you attended or visited this hospital, or practitioner?**

**8.5 Name three friends, colleagues or acquaintances connected with this hospital.** If this is inappropriate, ask for the names of three people who have visited the person in the last year.

**A8 Tell me about an incident involving a relative or visitor in the last year?**

A visit to the person’s home is acceptable. You may use the prompts “**A visit by or to a relative?**” or “**Involving some news about a relative?**” Any visit by a relative or friend to the person’s home is acceptable, and again the person is encouraged to produce a memory of a specific incident, rather than a general description. The prompts allow the person to produce a memory about a visit *to* a relative, or to report *news* about a relative. What is required is recall about an incident involving the chosen person during the last year.

**9.1 Where did you spend last Christmas?** Participants should be questioned about another major religious festival should this be appropriate to their culture.

**9.2 Name one person with whom you spent last Christmas.**

**10.1 What was the name of a place that you visited on a holiday or went on a journey last year?** If necessary, a place visited in the last five years is acceptable. The name of a town or city is acceptable.

**10.2 When did this holiday or journey take place?** If it took place in the last year, the month is required. If it took place in the last five years, the year or number of years ago is sufficient.

**10.3 Name one person who accompanied you on this holiday or journey.** Any person who has accompanied the participant on a holiday or journey is acceptable.

**A10 Tell me about an incident which took place while on any holiday or journey within the last year?** Or five years if necessary. You may use the prompts “**At the place you visited?**” or “**Involving someone you met?**”

**Excellent, thank you.**

#### **Rosenberg Self-Esteem Scale**

**This is a short questionnaire about how you see yourself. Please read the instructions at the top.** Give the person the RS-ES to complete.

Answer any queries the person may have. If the person looks fatigued, offer to take a break. If the person requests help with completion, facilitate by reading through the questions, and if necessary using the prompts: “*so what do you think?*” or “*there’s no right or wrong answers, we’re interested in what you think*”.

**Excellent, thanks.**

#### **HAD Scale**

**We’ll now move onto a questionnaire that’s about how you’ve been feeling in the last week. Please read the instructions at the top.** Give the person the HAD Scale to complete.

Answer any queries the person may have. If the person looks fatigued, offer to take a break. If the person requests help with completion, facilitate by reading through the questions, and if necessary using the prompts: “*so what do you think?*” or “*there’s no right or wrong answers, we’re interested in what you think*”.

**Thanks, that’s great.**

#### **NART**

**I’m going to show you some words, and I’d like you to say them out loud for me. Some of the words are quite unusual, but have a go even if you’re not sure how to say them. In**

**fact many people don't know all the words. Don't worry if you don't, it's OK to have a guess.**

Answer any queries the person may have. If the person looks fatigued, offer to take a break.

### **Metamemory In Adulthood**

**The next questionnaire is a long one and takes quite awhile to complete, if you're happy for me to do so, is it OK if I leave it with you to complete before our next visit?** If person agrees to complete MIA in own time move onto the NART.

If the person is happy to complete the MIA during the interview: **I'd now like you to fill out a questionnaire about how you use your memory and how you feel about it. Please read the instructions on the first page.** Give the person the MIA to complete.

Answer any queries the person may have. If the person looks fatigued, offer to take a break. If the person requests help with completion, facilitate by reading through the questions, and if necessary using the prompts: *"so what do you think?"* or *"there's no right or wrong answers, we're interested in what you think"*.

**Excellent, thanks. OK we'll move onto something a bit different.**

### **Pyramids and Palm Trees (Three Pictures)**

**Here are three pictures. You have to decide which one of these two at the bottom goes with the one at the top. Is it this one or this one?** Point to each picture as appropriate.

On the first practice triad, if the person responds correctly, the examiner should say:

**That's right, they go together because a waistcoat and a bow tie are both worn by men.**

If the person responds incorrectly, the examiner should say:

**No. The bow tie goes with the waistcoat because they are both worn by men.**

For the second practice triad, the examiner should say:

**Now try this one. Which of these two pictures goes with the one at the top?**

If the person responds correctly, the examiner should say:

**That's right. They go together because they pour from a bottle into a glass.**

If the person responds incorrectly, the examiner says:

**No, it's this one. You normally use a glass with a bottle.**

Then proceed to the third practice item, saying again:

**Now try this one. Which of these two pictures goes with the one at the top?**

If the person responds correctly, the examiner says:

**That's right. They go together because both a clown and a lion can be found at a circus.**

If the person responds incorrectly, the examiner says:

**No, it's this one. They go together because you find both a clown and a lion at a circus, but not a giraffe.**

With some participants, these instructions may need to be supplemented with further help and instruction by example on the practice triads to try and make sure that the participant has the idea of what is involved. With some participants it may help to present the test as a game of guessing the connection between the items that the test compilers had in mind.

On the test trials, no feedback is allowed. Participants will sometimes want to say that the two choice items go together; these responses are not allowed. The experimenter should reiterate that: **You have to choose which of these two goes with the one at the top.** If the person is unsure, he or she should be strongly encouraged to guess. When a person completely refuses to respond to a triad, it should be given a chance score – 0.5. All 52 test triads should be attempted.

**That's great, thanks.**

### **The DEX Questionnaire**

**This questionnaire is about some of the thinking difficulties people sometimes experience. Please read the instructions given at the top.** Give the person the DEX to complete.

Answer any queries the person may have. If the person looks fatigued, offer to take a break. If the person requests help with completion facilitate by reading through the questions and if necessary using the prompts: “*so what do you think?*” or “*there's no right or wrong answers, we're interested in what you think*”.

**Thanks.**

### **Ability Scale 1 (MARS)**

May need to explain need for this scale to younger and older adult controls:

*This questionnaire has been designed for people with memory impairments, so that's why there are the smiley faces!*

**I'm going to give you some examples of everyday situations where you might need to use your memory. I want you to think about your own memory, as it is now, and tell me how you think you would manage in that situation. I want you to choose the answer which best describes how you would do. The answers are on the card here. These are the situations:**

- 1. You meet someone and are told their name. Later on you meet them again, and you need to remember their name.**
- 2. You have made an appointment. You need to remember to go along.**

**3. You have promised to do something later in the day. You need to remember to do it at the right time.**

**4. You have got a set of items to sort out, some of which you have seen before and some of which are new to you. You need to pick out the ones you have seen before.**

**5. You hear a news item on the radio.**

**(a) One of your family comes in at the end and asks you what was said.**

**(b) Later on – say half an hour later – someone else asks you what you heard.**

**6. You meet up with a group of people. Some of them you've met before, others you haven't. You need to recognise which ones you've met before.**

**7. You go to a new building and you are learning to find your way around. Someone shows you a short route which you will need to remember.**

**(a) You need to retrace the route immediately.**

**(b) You need to retrace the route again later on – say half an hour later.**

**8. You have been given a message to deliver to someone. You need to remember to give that person the message when you see them.**

**(a) You see them right away.**

**(b) You see them later on.**

**9. You are being asked to give some information about yourself, such as your age, address, date of birth and so on, and to answer a few basic general knowledge questions.**

**10. Someone asks you for today's date.**

**That's great, thanks a lot.**

### **Raven Standard Progressive Matrices Sets A to D**

It is not necessary to stick rigidly to any particular wording. The key requirements are, first, to make sure that those taking the tests understand what they are to do and the method of thought required to solve the problems. Second, to ensure that the tests are administered in the same way to all who are to be tested, and that the procedure adopted corresponds to that used when collecting any reference data with which the results will be compared. Beyond these requirements, a wide variety of options is possible.

Open the booklet at the first problem, A1.

**Look at this.** Point to the upper figure. **You see, it is a pattern with a piece cut out of it. Each of these pieces below...**point to each piece in turn...**is the right shape to fill the space, but only one of them is the right pattern. Number 1 is the right shape, but is not the right pattern. Number 2 is not a pattern at all. Number 3 is quite wrong. Number 6 is nearly right, but is wrong here.** Point to the white piece in Number 6. **Only one is right. You point to the piece which is correct to complete the pattern.**

If the person taking the test does not point to the right piece, continue your explanation until the nature of the problem is clearly grasped. Turn to Problem 2.

**Now point to the piece which goes in here.** If the person taking the test fails to do so correctly, re-demonstrate Problem 1 and request again an answer for Problem A2.

If the problem is solved correctly, turn to Problem 3 and proceed as before.

At Problem 4, before the person taking the test has time to point to one of the pieces...**Look carefully at these pieces.** Move your fingers over the pieces. **Only one of these pieces is right to complete the pattern. Be careful. Look at each of the six pieces first.** Point to each of the six pieces. **Now you point to the right one to go in here.** Point to the space.

When the person taking the test has pointed to one of the pieces, whether it is right or not...**Is that the right one to go in here?** If the person taking the test says “yes”, accept the choice with approval, whether right or wrong. If he or she wishes to change the choice...**All right. Well, point to the one that is right.** Whether the answer is right or wrong again...**Is that the right one?** If the person taking the test is satisfied, whether the choice is right or wrong, accept that choice, but if there still seems to be doubt...**Well, which do you really think is the right one?** Make note of the number of the final choice in the correct part of the answer sheet (Easy-Score answer sheet – put a single short line through the number).

Demonstrate Problem A5 in the same way as Problem 4.

At any stage between A1 and A5, Problem A1 can be used to illustrate what has to be done, with the request that the person taking the test try again.

If these five problems are solved, turn to A6.

**Look at the pattern carefully. Now which of these pieces...**point to each in turn...**goes in here?** Point to the space that needs to be filled. **Be careful, only one is right. Which one is it? Be sure you find the right one before you point to it.** Record the answer finally given.

Present each problem, giving the same instructions as long as they serve a useful purpose.

\*If the person taking the test is concerned about minor defects in the drawings, assure them that they do not need to worry.

\*\*If the person taking the test seems to get stuck on a particular item, suggest that they move on and see if they can do the later problems and then come back to the problem that is causing difficulties.

\*\*\*If, in order to make progress, it seems necessary to do so, ask the person taking the test to guess “*as guesses are sometimes correct*”.

At the end of Set A, demonstrate the first problem of Set B, again pointing in turn to each of the 3 figures on the pattern and the space to be filled.

**You see how it goes. That. That. That. What will this one be? Point to the right one of these to go here. Be careful. Look at each one in turn. Only one is right. Which one is it?**

In Problems B1 to B5, after the person taking the test has pointed to one of the pieces, whether it is right or wrong...**Is that the right one to complete the pattern?** Point to the pattern and the space to be filled. As before, if the answer is “yes”, accept (and record) the choice with approval. If the person taking the test wishes to change the choice, proceed as in Set A, and accept the one finally adhered to as right.

For the sixth problem the person taking the test should not be asked if the answer chosen is right. Simply...**Look carefully at the pattern.** Point to each of the figures in turn and the space to be filled. **Be careful. Only one of these pieces completes the pattern properly.** Point to each in turn. **Which one is it?** Record the final choice on the answer sheet either by writing the number on the piece chosen next to the problem number, or, in the case of the Easy-Score answer sheet, putting a short single line through the number of the selected piece. If a mistake has been made, or the person taking the test wants to change his or her answer, put a cross through the incorrect answer, and then write (Easy-Score answer sheet – put a single line through) the number of the final choice. Do not try to rub out the incorrect answer.

The same guidance can be given with each remaining problem of Set B as long as it serves a useful purpose. Continue with Sets C and D.

**OK, that’s all of the questionnaires that I’d like to go through with you today. Are there any questions you would like me to answer, or anything you would like me to go through?** Answer any queries the person may have.

TO INFORMANT: **Is it OK if I go through the questionnaires with you?** Check the DEX and MARS have been completed.

### Cornell Scale

**I’d like to ask you some questions about your relative.**

**Has your relative been anxious in the last week?** If necessary give prompts: **anxious expression, ruminated or worried?**

**Has your relative been sad in the last week?** If necessary give prompts: **sad expression, sad voice, tearfulness?**

**Has your relative had a lack of reactivity to pleasant events in the last week?**

**Has your relative been irritable in the last week?** If necessary give prompts: **easily annoyed, short tempered?**

**Has your relative been agitated in the last week?** If necessary give prompts: **restlessness, hand wringing, hair pulling?**

**Has your relative had slow movements, slow speech or slow reactions in the last week?**

**Has your relative had multiple physical complaints in the last week?**

**Has your relative shown a loss of interest in usual activities in the last week?**

**Has your relative had loss of appetite in the last week?**

**Has your relative lost weight in the last week?**

**Has your relative had a lack of energy in the last week?**

**Has your relative had mood variations during the day in the last week?**

**Has your relative had difficulty falling asleep in the last week?**

**Has your relative had multiple awakenings when sleeping in the last week?**

**Has your relative woken up earlier than usual in the last week?**

**Has your relative said that they feel like life is not worth living in the last week?**

**Has your relative been self-deprecating in the last week?** If necessary give prompts: **self-blame, poor self-esteem, feelings of failure?**

**Has your relative been pessimistic in the last week?**

**Has your relative been delusional in the last week?** If necessary give prompts: **delusions of poverty, illness, loss?**

#### **RAID**

**Has your relative worried about their health in the last two weeks?**

**Has your relative worried about their cognitive performance in the last two weeks?** If necessary give prompts: **failing memory, getting lost when they go out?**

**Has your relative worried over finances, family problems or the physical health of relatives in the last two weeks?**

**Has your relative been worried with false beliefs and/or perceptions in the last two weeks?**

**Has your relative worried over trifles in the last two weeks?**

**Has your relative been frightened and anxious in the last two weeks?** If necessary give prompts: **Keyed up and on edge?**

**Has your relative been sensitive to noise in the last two weeks?** If necessary give prompts: **Exaggerated startle response?**

**Has your relative had sleep disturbances in the last two weeks?** If necessary give prompts: **Trouble falling or staying asleep?**

**Has your relative been irritable in the last two weeks?** If necessary give prompts: **More easily annoyed or usual, short tempered and angry outbursts?**

**Has your relative trembled in the last two weeks?**

**Has your relative complained of headache, and/or other body aches and pains in the last two weeks?**

**Has your relative been restless in the last two weeks?** If necessary give prompts: **Fidgeting, cannot sit still, pacing, wringing hands, picking clothes?**

**Has your relative been fatigued and tired in the last two weeks?**

**Has your relative complained of racing heart or thumping heart in the last two weeks?**

**Has your relative complained of a dry mouth or a sinking feeling in the stomach in the last two weeks?**

**Has your relative hyperventilated or had shortness of breath (even when not exerting) in the last two weeks?**

**Has your relative complained of dizziness or light-headedness in the last two weeks? If necessary give prompts: Complains as if going to faint?**

**Has your relative complained of sweating, flushes or chills, tingling or numbness of fingers and toes in the last two weeks?**

**That's great, thank you for your help.**

**Can I just confirm our next visit?** Check time and date and give person the second appointment letter if applicable. **Are you OK getting to the University?** Give person map and directions if necessary. **There is free parking for 2 hours in the roads near campus, or if you prefer I can organise a visitor's permit for the University car park. I can also pay travel expenses to the University if you are not coming by car.**

Show the person the red arrow and the green arrow: **What colour is this? And this? Thank you. They are examples of the arrows that you will see in the ERP session.**

**Is it OK if I do a skin test with the gel we use in the ERP session? This is just to make sure that we can use it on you. I will put a small amount on your skin. If you feel any itching or redness, let me know and we'll stop straight away.**

*\*If the participant refuses:*

*Unfortunately the use of the Nuprep is necessary for the EEG recording.*

*\*If the participant would like further clarification:*

*In order to be able to pick up a good clear electrical signal, the skin must be free from any skin oils. The gel helps do this.*

*\*If the person decides to withdraw:*

*Thank you for your time and for taking interest in the study. If you change your mind about participation, you can contact me on the number on the information sheet.*

*\*If the participant has any redness or itching. Stop immediately:*

*Unfortunately you won't be able to take part in the study, as the use of the gel is necessary for the EEG recording. In order to pick up the electrical signals, the skin must be free from any skin oils. I'm very sorry if I have caused you any inconvenience, if you are still interested, I can send you a summary of the findings from the study.*

**Is it also OK if I take your head measurements? I'd like to do this at this visit, so that I can prepare the cap before your next session. I need to measure the circumference of your head, from ear to ear and from your eyebrows to the back of your head. This is so that I use the right size cap and so that the leads (which go through these holes) are over the right areas of your head.**

Measure the circumference of head, ear to ear and from nasion toinion.

**That's great. Thank you for your time and I look forward to seeing you again.**

### **SECOND VISIT: ERP VISIT 1**

With attending relative, make sure they are sat near the mirror in the ERP room, so that they cannot intervene in either the ERP study or the later everyday tasks. Document their presence in the lab book.

**Please can you take a seat here.**

It may be necessary to remind participants of the first visit and what was done:

**Last time we met, you were kind enough to answer lots of questions about your mood, thinking skills and your memories of your life. Today we are going to be doing some very different tasks using the computer.**

**We'll start with an auditory task during which you'll listen to a series of tones and then we have a number of arrow tasks which you will be asked to respond to. The tasks take about 30 minutes to complete. Don't worry about how you're doing on the tasks, as they're designed to be a little bit difficult. If the tasks were too easy then everyone would do really well and not tell us anything interesting! After the computer tasks, I have a few everyday tasks that I would like you to complete.**

**Before we start, I want to check that you are still happy to continue in the study and that you are still happy for me to videotape the session. Please remember, you can withdraw from the study at any time, without giving a reason.**

*\*If the person decides to withdraw:*

*Thank you for taking the time to come to the University and for taking interest in the study. If you change your mind about participation, you can contact me on the number on the information sheet.*

**Are there any questions you would like me to answer? Or anything you would like me to go through before we start?**

**Are you comfortable? Would you like a drink? Please can you turn off your mobile.**

**Did you have any redness or itching from the skin test that I did when I saw you last?**

*\*If yes:*

*Unfortunately you won't be able to take part in the study, as the use of the gel is necessary for the EEG recording. I'm very sorry if I have caused you any inconvenience, if you are still interested, I can send you a summary of the findings from the study.*

*\*If no:*

**I'd like to repeat the skin test, just to make absolutely sure that I can use it on you.**

\*If the participant refuses:

*Unfortunately the use of the Nuprep is necessary for the EEG recording. It is unethical of us to put you at any risk whatsoever and I want to be absolutely sure that I can use the gel on you.*

\*If the participant would like further clarification:

*In order to be able to pick up a good clear electrical signal, the skin must be free from any skin oils. The gel helps do this.*

\*If the person decides to withdraw:

*Thank you for your time and for taking interest in the study. If you change your mind about participation, you can contact me on the number on the information sheet.*

\*If the participant has any redness or itching. Stop immediately:

*Unfortunately you won't be able to take part in the study, as the use of the gel is necessary for the EEG recording. I'm very sorry if I have caused you any inconvenience, if you are still interested, I can send you a summary of the findings from the study.*

### **Hearing Assessment**

Make sure the audiometer has been tested prior to use with participants.

**Do you have hearing better in one ear than the other?** Make note of response.

**I am going to test your hearing by measuring the quietest sounds that you can hear. As soon as you hear a sound, press the button (or raise you finger). Whatever the sound and no matter how faint the sound, press the button (or raise your finger) as soon as you think you can hear it.**

**Are you OK with the instructions? Please remember that we can stop whenever you would like to. If you have tinnitus, please try to ignore it as much as possible. If you have any difficulty telling between the sound and the tinnitus, please let me know.**

Before putting the earphones on the person's head: **Please don't hold or move the headphones once I've put them on you, if you want to stop, please tell me and we'll stop straight away.** Place the earphones on the person: **Are they on comfortably?** Adjust if necessary.

Start with the better-hearing ear (if applicable) and at the frequency of 1000 Hz. The duration of the presented tone should be varied between 1 and 3 seconds. The timing must not be predictable.

To ensure the person is familiar with the task, present a tone of 1000 Hz that is clearly audible (at 40 dB). If there is no response at this level, increase the level of the tone in 20 dB steps until a response occurs. If the tone is still inaudible at 80 dB, increase the level of the tone in 5 dB steps until a response occurs, taking care to monitor the person for discomfort.

If the responses are consistent with the tone presentation, the person is familiarised with the task. If not, repeat. If after this repeat, their responses are unsatisfactory, re-instruct the person.

Method for finding threshold.

Following a satisfactory positive response, reduce the level of the tone in 10 dB steps until no further response occurs.

Increase the level of the tone in 5 dB steps until a response occurs.

Continue to decrease the level by 10 dB and increase by 5 dB until the person responds at the same level on two responses on the ascent are repeated.

Proceed to the next frequency, starting at a clearly audible level and use a 10 dB down, 5 dB up sequence until the threshold criterion is satisfied. Repeat familiarisation if necessary.

The test order is 1000 Hz, 1500 Hz, 2000 Hz and 500 Hz.

\*If the person has a hearing level of 25 dB or greater, photocopy the audiogram and inform the person:

*One of the Doctors advising our studies has said that anyone who has a hearing level of 25 dB or more should take this audiogram to their GP. I am not medically trained and this test is not diagnostic, so all I can recommend is that you go and see your GP with the audiogram and ask for a hearing test.*

**Please fill out this quick questionnaire about how you're feeling right now.**

Hand the person the clipboard with the PANAS on it.

**Thanks a lot.**

**This is the cap I would like to place on your head, the leads are attached where the holes are.**

**Do you want to use the bathroom at all? The cap will be on your head for about an hour. But we can take it off any time you want. I need to draw a dot on your head so that I can place the cap correctly, is that OK?**

Measure Cz and draw a dot.

\*If the person wears glasses:

*Please can you remove your glasses whilst I put the cap on. Is it OK if your glasses stay off whilst I sort out the leads?*

If the person wants their glasses back on, let them and request they be removed whenever necessary.

**I'm going to pull the cap down and fit it over your ears. I also need to use the chest strap, is that OK? Please could you lift up your arms for me? Is the strap comfortable? Adjust if necessary. I'm just going to attach the cap to the chest strap. How does that feel?**

\*If the person says the cap is too tight:

*The cap needs to be tight to your scalp in order for the electrical signals to reach the leads, if the cap were looser then we wouldn't be able to record any electrical activity.*

Take a break if necessary and talk through any concerns the person may have.

**\*\*If the person decides to withdraw:**

*Thank you for taking the time to come to the University and for taking interest in the study. If you change your mind about participating, you can contact me on the number on the information sheet.*

**It's OK if you want to move your neck and shoulders so that you don't get stiff. You can try gently rolling your shoulders, and stretching and shaking your arms out.**

**I'm going to clean either side of your eyes and underneath your right eye, so that I can place some leads in those positions. This is so that we can record eye movements. I need to use some sticky tape to keep the leads in place, is that OK?**

Place VEOGL, VEOGR and HEOGI. Plug in the leads.

**\*If the person doesn't like the eye leads:**

*We need to measure eye movements because eye movement affects the EEG recording. We can't use the EEG recording if it is affected by eye movement.*

Take a break if necessary and talk through any concerns the person may have.

**\*\*If the person decides to withdraw:**

*Thank you for taking the time to come to the University and for taking interest in the study. If you change your mind about participating, you can contact me on the number on the information sheet.*

**Just remember, if you would like me to stop, we can at any time, it only takes a minute to remove the cap and the leads.**

Open the montage on the PC. (Acquire, Open, look in SynAmps2, CassieSyn2.ast, Acquisition, Impedance).

**I'm going to clean where each of the holes are and then place the conductive gel into the holes with a syringe. Show syringe. Please let me know if you feel uncomfortable.**

Clean holes and place gel in holes ensuring impedances are kept below 10Ω.

**\*If the person doesn't like the process:**

*I need to make sure that the leads can pick up the electrical signals, and this is done via the gel.*

Take a break if necessary and talk through any concerns the person may have.

**\*\*If the person decides to withdraw:**

*Thank you for taking the time to come to the University and for taking interest in the study. If you change your mind about participating, you can contact me on the number on the information sheet.*

Close montage and open recording file. (Green arrow on Acquire toolbar). For each participant a new folder is required and each task is saved within that folder.

**How does that feel? Can you blink for a few times for me, please? And can you move your eyes from side to side, like you're watching a tennis match. Great, thanks. You need to keep your head as still as possible during the actual computer tasks. It's OK if you want to move your neck and shoulders between each task so that you don't get stiff. Try gently rolling your shoulders and stretching your arms out. If you like, I'll remind you every now and then when it's a good time, and generally when I'm speaking to you it's a good time to have a stretch. I just need you to keep your head still when you're actually doing the tasks. Thanks.**

**I'm going to turn on the video recorder, is that alright?**

Open Save box and name file as participant initials and participant number and Eyes Closed (.cnt).

Go over to the other PC and open experiment Session 1 in Presentation, go to Logfiles and set to append counter with participant number, return to Experiment and enter participant code when prompted. When 'ready' appears on screen, read instructions.

**I would like to record for just a few minutes with you doing nothing. We need to record two minutes with your eyes closed and two minutes with your eyes open. This provides us with baseline data against which we can compare your electrical signals whilst you are doing the actual tasks. Right, please close your eyes? You can think anything you like. I'll tell you when I'd like you to open them. OK? Go.**

Start EEG recording. Press 'enter' followed by the space bar for the port code in the .cnt file. Stop after two minutes.

**You can open your eyes now. That's great, thanks.** Type in next task Eyes Open.

**I'd like to record two minutes with your eyes open now. Again, you can think anything you like. I'll tell you when the two minutes is up. Is that alright? Go.**

Start EEG recording. Press 'enter' followed by the space bar for the port code in the .cnt file. Stop after two minutes.

**That's great.** Stop the EEG recording and type in next task Auditory Oddball.

### **Auditory Oddball**

**You are going to hear a series of high and low tones. When you hear the high tones, press the right mouse button. Please look at the image on the screen whilst the task is running.**

**The task lasts for about 5 minutes. OK? There is a practise trial so that you can hear what the high and low tones sound like and it starts with low tones. Remember press the right mouse button when you hear a high tone.**

Answer any queries the person may have.

Start EEG recording.

**Are you ready?** Start Auditory Oddball. The practise trial ends with the message 'End of practise trials'. **Would you like me to adjust the volume?** Adjust the volume of the person would like it adjusted.

Press the left mouse button to start the auditory oddball task.

At the end of the task, stop the EEG recording and type in next task 4CR.

If the person queries the novel tones in the Auditory Oddball:

**I'm sorry about that, we are looking at the effects of unexpected noises and if we told you that there would be other noises then they wouldn't be a surprise!**

#### **4CR**

**Cup the mouse in your hands and use your thumbs to respond.**

**Green arrows are going to appear on the screen. Press the left button when the arrow points left and press the right button when the arrow points right. You will also see red arrows. When you see a red arrow, do the opposite to the green arrows. If you see a red left-pointing arrow, press the right button with your right thumb. If you see a right-pointing red arrow, press the left button with your left thumb. So just to make sure, which button would you press for a green arrow pointing this way (point left)? And which button would you press for a red arrow pointing that way (point right)? The arrows appear quite quickly, so please try to respond as fast as you can, but as accurately as you can. Everybody makes mistakes on this task. Sometimes you will automatically try to correct your errors and this is OK. This task will last about 2½ minutes.**

Start EEG recording.

**Are you ready?** Start 4CR.

Once the first run is finished: **That's great. Using a percentage, how accurate do you think you were?**

**You get more goes at that task, so I'd like you to do that all over again. Is that OK? Remember as quick as you can.**

At the end of the second run: **That's great. Using a percentage, how accurate do you think you were?**

**I'd like you to do that all over again. Is that OK? Remember as quick as you can.**

Once the third run is finished: **That's great. Using a percentage, how accurate do you think you were?**

**You're halfway through the arrow tasks, so I'd like you to do that all over again, is that alright? Remember as quick as you can.**

At the end of the fourth run: **That's great. Using a percentage, how accurate do you think you were?**

**I'd like you to do that all over again, if that's alright? Remember quick as you can.**  
Once the fifth run is finished: **That's great. Using a percentage, how accurate do you think you were?**

**This is the last time that I'll ask you to do this task and once it's done we can get the cap off. So I'd like you to do that all over again. Remember as quick as you can.**

At the end of the sixth run: **That's great. Using a percentage, how accurate do you think you were?**

Stop the EEG recording, note percentage. Check impedances (Acquisition, Impedance).

If the person queries their performance or the repetitions:

**The task is designed to be quite challenging and most people find it a bit difficult and a little tiring. Unfortunately, because the EEG recording is so sensitive, we do lose a certain proportion of the recording because of movement and things like that. We have to make sure that we collect enough data to cover any losses, if we don't do that then we run the risk of not being able to use any of that persons data.**

### **END OF EEG RECORDING**

**Excellent, thank you. I'll just take the cap and leads off your head. Would you like a drink? And would you like to go to the bathroom?**

If person would like a drink, fetch the drink and then remove the cap and the leads and as much of the conductive gel as possible.

**Please fill out this quick questionnaire about how you're feeling right now.**

Hand the person the clipboard with the PANAS on it.

**That's great. Thanks a lot.**

**OK, I've just got a few tasks that I'd like you to do for me and then that's the end of the study. Again I'd like to videotape this part of the visit, is that alright?**

### **Everyday Tasks**

Bring the props over to the table for each task in turn and ask the person to:

**Please can you make a cheese and lettuce sandwich? Can you use everything that I've placed in front of you?**

If, after 2 minutes, the person is clearly unable to progress, assist as follows:

**I'll give you some help**, then take 2 slices of bread out the bag, and say:

**Try and complete the task now.**

If after a further 2 minutes has passed the person still cannot proceed to the next stage give a further prompt by demonstrating what needs to be done, e.g. spreading margarine on the bread. If necessary continue prompting in this way until the sandwich is made. Tick/check each stage that the person completes independently. Stages given below:

- Open bag and get out 2 slices of bread
- Spread margarine on both slices of bread
- Unwrap cheese slice and place on bread
- Take leaf/leaves of lettuce from the head of lettuce
- Place second slice of bread on top

**I'd like you to pretend that this is a letter to yourself. Please prepare it for posting, and address the envelope to yourself.**

If, after 2 minutes, the person is clearly unable to progress, assist as follows:

**I'll give you some help**, then fold the letter, and say:

**Try and complete the task now.**

If after a further 2 minutes has passed the person still cannot proceed to the next stage give a further prompt by demonstrating what needs to be done, e.g. placing the letter in the envelope. If necessary continue prompting in this way until the letter is ready for posting. Tick/check each stage that the person completes independently. Stages given below:

- Fold a letter
- Put letter in envelope
- Seal the envelope
- Address the envelope
- Put the stamp in the correct position on envelope

**Please wrap this gift for me. Use everything that I've placed in front of you.**

If, after 2 minutes, the person is clearly unable to progress, assist as follows:

**I'll give you some help**, then cut an appropriate size of paper, and say:

**Try and complete the task now.**

If after a further 2 minutes has passed the person still cannot proceed to the next stage give a further prompt by demonstrating what needs to be done, e.g. correctly positioning the paper around the gift. If necessary continue prompting in this way until the gift is wrapped. Tick/check each stage that the person completes independently. Stages given below:

- Cut appropriate size of paper
- Position the paper around the gift
- Cut piece(s) of sticky tape (teeth or scissors acceptable)
- Secure paper with sticky tape
- Tie bow around the gift

### **Action Program Test**

Place the test items in front of the person in the following order (from person's left to right): Container screw top, container, wire hook and the base with the tube and cork on the left hand side and the beaker, filled two-thirds with water, and lid on the right hand side.

**If you look in the bottom of this tube you will see a small cork. Your task is to get the cork out of the tube. You can use any of these things** (indicate equipment) **to help you. However you must not lift this up** (indicate main assembly), **nor this** (indicate beaker) **nor this** (indicate the tall tube) **and you cannot touch this** (indicate lid) **with your fingers. Now go ahead and try and get the cork out of the tube.**

If, after 2 minutes, the person is clearly unable to progress, assist as follows:  
**I'll give you some help**, then remove the lid with the wire hook, and say:  
**Try and complete the task now.**

If after a further 2 minutes has passed the person still cannot proceed to the next stage give a further prompt by demonstrating what needs to be done, e.g. attaching the screw top to the container. If necessary continue prompting in this way until the cork has been retrieved. Tick/check each stage that the person completes independently. Stages given below:

- Removes lid from beaker using wire hook
- Attaches screw top to container
- Fills container with water
- Pours one containerful of water into tube containing cork
- Pours second containerful of water into tube containing cork

**OK, that's the end of the session and the end of your participation in the study. I'd like to take this opportunity to thank you for your participation. Are there any questions you would like me to answer or anything you would like me to go through with you? I won't be able to provide a summary of the findings until I have collected all the data and analysed it, but I am happy to email you a simplified image of your EEG recording.**

## Appendix 5. Investigation of Habituation of the ERN.

### Aim of the study.

Two studies (Hajcak et al., 2003; Herrmann et al., 2004) found that the amplitude of the ERN decreased with increased error rate. The error rates for each condition did not differ between groups. Related to the findings of Hajcak et al (2003) and Herrmann et al (2004), it is also possible that variation in error-distribution profile (across the session) between participants might influence the averaged-ERN. In other words, the ERN elicited by errors predominantly made early in the session may be due to unfamiliarity with the task and may differ in morphology and power to the ERN elicited by errors predominantly made later in session, which may be influenced by greater task-familiarity, or by attentional lapse (fatigue). In order to explore this potentially confounding issue, a subset of younger and older participants who had at least five errors in the first and fourth blocks of the 4-CRT (i.e. > 5 errors per 100 trial block); other participants tended to make the majority of errors in either the first few trials (block) or unevenly distributed across blocks. Data were explored to investigate possible differences between ERN amplitudes elicited within the first and last blocks of the 4-CRT. The aim of this supplementary analysis was to investigate the hypothesis that increasing task familiarity might modulate the amplitude of the ERN. This was explored for compatible stimuli (green arrow stimuli) and incompatible stimuli (red arrow stimuli).

### Method.

#### Participants.

Five younger (YA: compatible condition; *M* age 19.2 years, *SD* 0.8; 1 male and 4 female; incompatible condition; *M* age 20.0 years, *SD* 3.5; 5 female) and five older (OA: compatible condition; *M* age 71.8 years, *SD* 8.0; 2 male and 3 female; incompatible condition; *M* age 70.0 years, *SD* 8.2; 1 male and 4 female) adults provided data for the study. Three OA and 2 YA provided for both conditions.

#### Measures.

See Chapter 5 for ERP paradigm, acquisition and processing.

## Results.

Four paired-sample T-tests were conducted: two within the YA group (1. Compatible, 2. Incompatible conditions), and two similar comparisons in the OA group. A significant ERN amplitude increase between the first and fourth block was found only in the OA group for the compatible condition;  $t(4) = 3.61, p = .023$ . There were no other significant amplitude differences. It is possible that the OAs may have been more susceptible to testing fatigue, resulting in greater uncertainty in the later trials. However, this may have been expected to affect both Compatible and Incompatible conditions equally (or perhaps the Incompatible condition to a greater degree). In general, it is concluded that error-distribution profile is unlikely to confound the results of the present study of ageing-effects on the ERN. This was necessary in order to provide data for the primary hypotheses, as detailed in Chapter 5.

Table 1. Peak ERN amplitudes in younger and older adults.

<i>M</i> ( <i>SD</i> )	Condition	<u>Younger</u>		<u>Older</u>	
		<i>n</i> = 5		<i>n</i> = 5	
		First block	Last block	First block	Last block
ERN ( $\mu\text{V}$ )	Compatible	-14.2 (5.6)	-13.5 (5.7)	-3.1 (3.1)	-7.2 (.2.3) *
	Incompatible	-11.9 (8.3)	-10.3 (7.4)	-4.6 (6.3)	-5.6 (3.4)

*Note.* \*  $p < .05$ , planned comparison using *t*-test.

## Appendix 6. Investigation of the Effect of Older Age on Response-locked Components.

### Aim of the study.

The age range of the older adult sample was 60-84 years. As indicated in Chapter 2, there can be considerable neural and cognitive variability between older adults aged at the lower and higher end of this age range. Therefore, it was of interest to investigate potential ERN and Pe differences between the young-old and old-old.

### Method.

#### Participants.

Eighteen younger ( $M$  age 21.6 years,  $SD$  3.7; 3 male and 15 female), eleven young-old ( $M$  age 64.1 years,  $SD$  3.0; 3 male and 8 female), and ten old-old ( $M$  age 73.8 years,  $SD$  4.7; 4 male and 6 female) adults provided data for the study; the division in the older adult group was based on a median split of age.

#### Measures.

See Chapter 5 for ERP paradigm, acquisition and processing.

#### Statistical Analysis.

The amplitudes of the response-locked ERP components were analysed using mixed ANOVA models: condition (x2: compatible, incompatible), component (x2: CRN, ERN) and group (x3: younger, young-old, old-old), and condition (x2: compatible, incompatible) and group (x3: younger, young-old, old-old) for the Pe.

#### Results.

There was a main effect of group in both ANOVA models,  $F(2, 36) = 3.55, p = .039$  (CRN-ERN) and  $F(2, 36) = 4.09, p = .025$  (Pe), indicating a significant decrease in amplitude in all response-locked components between the age groups. In each case, the lowest amplitudes were obtained from the old-old group, the highest amplitudes were obtained from the young adult group, and interim values were obtained from the young-old group. As indicated in

Table 1, post-hoc comparisons revealed statistically significant differences between younger-adults and old-old adults for the incompatible ERN amplitude and compatible Pe amplitude. This finding suggests the importance in future studies of including multiple age groups to examine ERP components in older adults, but does not detract from the findings of the present study (Chapter 5), which reveal reduced ERN power in older compared to younger adults.

Table 1. Peak amplitudes of response-locked components for younger, young-old and old-old adults.

		<u>Younger</u>	<u>Young-Old</u>	<u>Old-Old</u>
		<i>n</i> = 18	<i>n</i> = 11	<i>n</i> = 10
<i>M (SD)</i>		Amplitude ( $\mu$ V)		
CRN	Compatible	5.7 (3.5)	4.7 (2.9)	2.7 (2.0)
	Incompatible	5.7 (3.1)	5.0 (.2.7)	2.1 (1.6)
ERN	Compatible	13.8 (7.1)	10.9 (6.4)	10.1 (7.5)
	Incompatible	15.4 (7.1)	10.3 (5.4)	8.4 (7.7) †
Pe	Compatible	7.8 (8.0)	2.9 (6.3)	0.4 (5.8) †
	Incompatible	5.1 (6.9)	2.0 (5.8)	0.6 (2.9)

*Note.* †  $p < .05$ ; younger vs. old-old.

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