The influence of facial motion on the neural response during emotion perception in typical and atypical development

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Thesis submitted for the degree of Doctor of Philosophy

UCL

2012

DECLARATION

'I, Anna Lois Matheson, confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.'

Signed.....

ABSTRACT

The ability to interpret emotional expressions is the key to understanding our social environment. In our everyday lives we are exposed to a huge variety of facial expressions which are constantly updated in response to environmental cues. The neural networks underpinning our cognitive ability to perceive dynamic emotional expressions are poorly understood.

This thesis aims to address the effects of motion on our perception of emotional expression from a developmental perspective. The overall aim was to compare the neural correlates of emotion perception of static and dynamic images for the six basic facial expressions in typical and atypical development. Three populations were studied: 1) typically developed adults; 2) atypically developed adults, i.e. young adults who have undergone a surgical resection for paediatric temporal lobe epilepsy; and 3) typically developing infants (4-12-month-olds).

Initially, morphed dynamic images for the six basic facial expressions were created, to be used in subsequent studies. These were validated, alongside static photographs, with ratings for accuracy, confidence and intensity.

The first and second ERP studies, involving typically developed adults and atypically developed adults respectively, explored the amplitude and latency of the P1 and N170 event-related potential (ERP) components in response to observing static and dynamic images of facial expressions. The final study, involving typically developing infants, explored the amplitude and latency of the P1 and N290 (the N170 precursor).

The impact of motion on the development of emotion perception is discussed in relation to the findings presented in this thesis.

ACKNOWLEDGEMENTS

I would like to express my gratitude to my supervisors Dr Michelle de Haan and Dr Torsten Baldeweg for their continual support and commitment to my PhD. They allowed me the freedom to explore and develop my own ideas whilst providing me with insight and guidance throughout my PhD. Michelle's support with ERP analysis and thought-provoking discussions about the direction of my research were invaluable to the evolution of this thesis.

I would like to thank everyone in the DCNU at the Institute of Child Health, UCL past and present who have made my PhD years so enjoyable; providing me with advice, support and friendship over the years. In particular, to Rebecca Webb for her continued support during the challenges of recording EEGs in young infants, learning together, and eventually making experiments run smoothly.

I am indebted to my family and partner, whose unwavering love and encouragement steered me through troubled waters, especially during the final stages of thesis preparation.

Finally, I want to extend my gratitude to all the individuals who participated in my research, both young and old. I am grateful for their enthusiasm and interest in my research, without which this thesis would not have been possible.

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Abbreviations

ACC – anterior cingulate cortex

ANOVA – analysis of variance

DNET – dysembryoplastic neuroepithelial tumour

EEG – electroencephalography

EGI – Electrical Geodesic Inc.

EPN – early posterior negativity

ERP – event-related potential

FFA – fusiform face area

fMRI – functional magnetic resonance imaging

GSN – Geodesic sensor net

HS – hippocampal sclerosis

LPP – late positive potential

LMTLE – left mesial temporal lobe epilepsy

LTL – left temporal lobectomy

MTLE – mesial temporal lobe epilepsy

MTS – mesial temporal sclerosis

Nc – negative component

NIRS – near infrared spectroscopy

OFC – orbitofrontal cortex

PET – positron emission topography

RMTLE – right mesial temporal lobe epilepsy

RTL – right temporal lobectomy

STS – superior temporal sulcus

TL – temporal lobectomy

TLE – temporal lobe epilepsy

ToM – theory of mind

VEP – visual evoked potential

1. General Introduction

1.1 Introduction

Facial expressions of emotion can be considered, alongside other visual and auditory signals, as both an emotional response to a stimulus and a basis for social communication. With this in mind, it is no surprise that humans are extremely good at perceiving and interpreting messages from facial cues. An important component of the normal social environment is the dynamic properties of the social stimuli around us. Temporal information from a moving face provides us with a constantly updated version of the emotional content and social intent. The currently dominant model of the neural bases of adult face perception recognises this fact, assigning distinct brain pathways for processing static, unchanging aspects of faces versus dynamic, changeable aspects (Haxby, Hoffman, & Gobbini, 2000). Even so, the bulk of the existing literature on perception of emotional signals in the face has focused on static images of faces. Understanding the role of motion in perceiving expressions has broad implications for research and practical work related to emotion. For example, is the additional information provided by dynamic compared to static facial expressions a benefit for the quick and accurate recognising of emotion, or does it provide a processing burden which slows down this process? If dynamic cues provide a benefit, this suggests that studies showing deficits in emotion recognition in patients with brain injury using static images might be over-estimating deficits in terms of their impact under more naturalistic conditions.

This chapter provides a general overview of research on the neural processes that underpin the perception of facial expressions of emotion, with emphasis on how facial movement affects our perception of emotional expressions. I will argue that the use of static stimuli alone in researching emotional expression cannot capture the full picture of the ever-changing emotional word around us and that dynamic stimuli provide additional temporal information used to understand emotional expression. This chapter also provides the background for two of the converging approaches used to investigate this topic. One approach was to investigate how injury during childhood to brain regions involved in face processing affects emotion processing skills in young adulthood. This was undertaken by studying young adults who had undergone temporal lobectomy as children due to intractable epilepsy. The second approach was to investigate the emergence of brain systems involved in processing facial emotion in typically developing infants in the first post-natal year of life. This chapter will focus on general issues related to these topics, with more in-depth literature reviews relating to specific issues to be found in the subsequent experimental chapters. This chapter ends with a summary of the research questions to be addressed by the work in this thesis.

1.2 The Neurocognitive Bases of Adult Face Perception

Faces are potentially one of the most important stimuli in our environment and play a crucial role in social interactions. The ease with which we are able to ascertain both identity and emotional expression from a face suggests an underlying

specialised brain system at work. The face has to be identified as belonging to a particular individual taking into account viewing angle, facial expressions, changes in appearance, gender and age, and we can usually do this with ease and rapid speed. Adults' fine-tuned ability to recognise faces is thought to rely on several stages of processing, holistic (processing faces as a Gestalt), featural (sensitivity to differences in individual features between faces), and second-order relational processing (sensitivity to differences in spacing of facial features between faces) (Maurer et al, 2002; Mondloch, Le Grand & Maurer, 2002).

An extensive neural network of areas has been implicated in face processing in humans, with a right hemisphere specialisation, including the face-selective regions in the lateral fusiform gyrus (Kanwisher, McDermott, & Chun, 1997) and inferior occipital gyrus (Hoffman & Haxby, 2000) and also the superior temporal sulcus and anterior pole (Haxby, Hoffman, & Gobbini, 2002; Ishai, Ungerleider, Martin, & Haxby, 2000; Sergent, Ohta, & MacDonald, 1992) as well as several areas related to the limbic system such as the amygdala, orbitofrontal cortex and retrosplenial or posterior cingulate regions.

The currently dominant framework for understanding the neural bases of face processing proposed by Haxby Hoffman & Gobbini (2000), emphasises a distinction between the representation of invariant and changeable aspects of faces. In this model, the initial perception by the visual system, including the inferior occipital gyrus, feeds into two pathways, see Figure 1.1 below. Firstly, there is the perception of identity of an individual relying on the invariant aspects of the face, taking place in

the lateral fusiform gyrus. Secondly, there is the perception of changeable aspects of the face such as eye gaze, lip movement and facial expression which mediate social interactions in regions such as the superior temporal sulcus (STS). These two pathways correspond to the core cortical system which leads to the extended subcortical-cortical neural system which mediates further face processing, in regions such as the intraparietal sulcus, auditory cortex, amygdala, insula and anterior temporal regions. It is important to note that there is also a subcortical pathway that initially by-passes the core-cortical system that can provide a quicker, but less detailed, perception of stimuli. This pathway can influence cortical processing, probably through reciprocal connections with structures such as the amygdala.

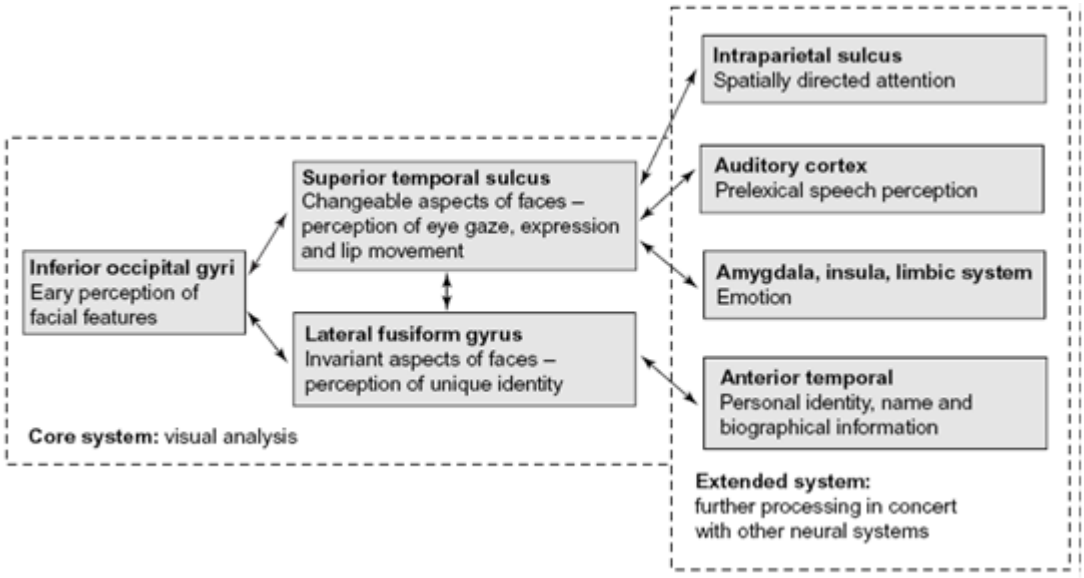


Figure 1.1 A model of the distributed human neural system for face perception as proposed by Haxby, Hoffman & Gobbini (2000).

There is a debate as to how best to characterise the function of the core cortical network. On one hand, there are those that maintain that there are brain structures that are uniquely specialised to process faces (domain-specific). Strong proponents

of this view include Kanwisher and colleagues, who argue that the fusiform gyrus can be viewed as a specialised module for face perception and termed this area the Fusiform Face Area (FFA) (Kanwisher et al., 1997). Evidence to substantiate this comes from developmental studies illustrating that infants show a preference for face-like stimuli over others from young infancy (Morton & Johnson, 1991), although this evidence is indirect since the neural bases of this preference has not been firmly established. Also, faces are processed as a whole (configural or holistic processing) whereas objects are processed as a set of features (Farah, Wilson, Drain, & Tanaka, 1998), illustrated by the inversion effect where the recognition of faces is affected by inversion but objects are not (Yin, 1969).

On the other hand, there is also evidence arguing against the view of cortical 'face' specialisation, which has demonstrated that supposed face-cortical regions can also be activated by other object categories so long as the viewer is expert in discriminating amongst category members. In this view, face processing may simply be a case of within-category discrimination of stimuli that subjects have expertise in processing (Gauthier & Tarr, 1997; Rossion, Curran, & Gauthier, 2002; Tarr & Gauthier, 2000). A third possible interpretation has been proposed stating that the representations of objects and faces are widely overlapping in the ventral temporal cortex, with specific regions primarily involved in processing a particular class of stimuli and patterns of activation in secondary associated regions (Haxby et al., 2001; Ishai, Ungerleider, Martin, Schouten, & Haxby, 1999; Ishai et al., 2000). Thus the FFA would not solely represent faces but be part of an extended system for the recognition of all objects.

1.3 Facial Expressions of Emotion

Facial expressions have been studied for years, as far back as the 1870s Darwin postulated on emotion in both humans and other animals (Darwin, 1872). In the 1970s Ekman (1972) and Izard (1977) led the research in recognition and categorisation of facial expressions.

It is generally accepted that there are a limited number of emotions, including the basic emotions (anger, disgust, fear, happiness, sadness and surprise) that activate discrete category representations. Support for this theory come from the finding that emotions are pan-cultural and that each facial expression has similar facial musculature across cultures (Ekman, 1992a; Ekman, 1994).

1.3.1 Emotional versus neutral faces

Experimental research in humans and non-human primates has suggested right hemisphere specialisation for emotion processing (Blonder, Bowers, & Heilman, 1991; Etcoff, 1984). Brain regions thought to be involved in facial affect processing include the STS, right temporal lobe, the basal ganglia, the right mesial occipital and right inferior parietal regions, the right somatosensory cortex and the amygdala. Face-selective neurons in the monkey cortex have shown enhanced neural responses to a face with various emotional expressions compared with neutral faces (Sugase, Yamane, Ueno & Kawano, 1999). This response arose approximately 50ms after the initial activation discriminating between faces and other visual objects indicating that it might represent activity in regions of the limbic system and would be consistent with direct projections between the visual cortex and the amygdala. In

fact there is evidence that there are connections from the amygdala to the visual cortical regions which play an important role in modulating sensory responses to emotional stimuli (Amaral, Behniea, & Kelly, 2003; LeDoux, 1996). There is support for this idea from a positron emission topography (PET) study (Morris et al., 1998) that showed a significant correlation between the enhancement of fusiform responses to fearful faces and the amount of amygdala activation by fearful versus happy faces. Further support comes from a functional magnetic resonance imaging (fMRI) study with patients with medial temporal lobe epilepsy; showing that patients with hippocampal sclerosis, but not those with amygdala sclerosis, showed enhanced activation of the fusiform for fearful versus neutral faces. In fact, the greater the degree of amygdala sclerosis, the smaller the differential response to fearful versus neutral faces in fusiform area of the ipsilateral hemisphere (Vuilleumier, Richardson, Armony, Driver, & Dolan, 2004). Patients also showed a weaker response to fearful faces in the STS, retrosplenial cortex, somatosensory parietal cortex, anterior cingulate gyrus and the hypothalamus, suggesting that amygdala connections contribute to a distributed network for face and emotion processing.

Brain imaging studies have shown that several regions in the human visual cortex exhibit a greater response to emotional versus neutral faces including face-selective areas in the fusiform gyrus, as well as the more obvious limbic regions such as the amygdala and orbitofrontal cortex (Ishai, Pessoa, Bikle, & Ungerleider, 2004; Vuilleumier et al., 2004). Emotional effects on the fusiform gyrus have been most commonly reported for fearful faces (Breiter et al., 1996; Morris et al., 1998). The effects on the fusiform gyrus of four different emotions (disgust, fear, happiness and

sadness) were reported in an fMRI study looking at different emotional intensities (mild and intense). The activity of the fusiform gyrus was enhanced by increasing the intensity of all four emotions but especially for fear (Surguladze et al., 2003). However, a second fMRI study found no differences in the activity of the fusiform gyrus for high and low intensities of emotion (Winston, Vuilleumier, & Dolan, 2003). An increase in activity has been observed in the occipital and temporal cortices when viewing scenes with aversive content relative to neutral scenes (Lane et al., 1998) but much more rarely for pleasant pictures, demonstrating that the response from the visual regions might be more sensitive to negative or aversive stimuli.

Most of the brain structures involved in the processing of emotional expression participate in perceptual processing, understanding the geometrical configuration of facial features, and also recognition of the emotional meaning for a given stimulus. To process fear, there would need to be perception of fear, the lexical label 'fear' and the motor representations required to produce the expression of fear (Adolphs, 2002). The perceptual analysis of facial expressions is a two-step process, first the determination of configural aspects of the facial form and later emotional meaning and context primarily reliant on dynamic aspects of facial features. A large number of brain structures are involved in the recognition of facial expressions of emotion, namely the occipito-temporal cortices, the orbitofrontal cortex, the amygdala, the basal ganglia and the right parietal cortices. They participate in various areas of perception and understanding emotion and it is therefore hard to assign a specific function to a known area.

Initially, at the point of perception of an emotionally salient stimulus, there is a feed-forward processing of information from the primary visual cortices along the occipital and temporal neocortices. There is then some debate as to when the initial categorisation of emotional stimuli would take place. It might occur anywhere between 100ms and 200ms post-stimulus onset. Some researchers tend to believe that at approximately 100-120ms specific brain regions would categorise the stimuli as emotionally salient based on the structural properties of the stimulus. At this point the early perceptual processing takes place, allowing the basic facial features and identity to be elucidated. However some researchers believe that specialised neural systems are first activated by faces around 150-200ms post-stimulus onset, as indexed by the face-selective N170 component measured over the scalp at occipito-temporal electrodes (Bentin, Allison, Puce, Perez, & McCarthy, 1996). This N170 response is thought to reflect early perceptual encoding and categorisation of face stimuli (Carmel & Bentin, 2002; Eimer, 2000a).

Consistent with the view that this is just the initial encoding of faces, many studies have found that the N170 component is not affected by the valence of expressions (Eimer & Holmes, 2002; Hermann et al., 2002; Krolak-Salmon, Fischer, Vighetto, & Mauguiere, 2001). However, some recent studies have disputed this idea and reported some emotional modulation for the amplitude of the N170 (Ashley, Vuilleumier, & Swick, 2004; Batty & Taylor, 2003; Campanella, Quinet, Bruyer, Crommelinck, & Guerit, 2002; Eger, Jedynak, Iwaki, & Skrandies, 2003; Miyoshi, Katayama, & Morotomi, 2004; Pizzagalli et al., 2002). For example, the amplitude of the N170 was found to be larger for fearful faces compared with neutral or happy expressions (Ashley et al., 2004; Batty & Taylor, 2003). A delay in the peak latency of

the N170 was also observed for fearful facial expressions (Batty & Taylor, 2003). These effects are not always selective for any specific emotional expression, potentially suggesting a role for attention or non-specific configural cues rather than emotional significance. There is also evidence that the N170 component responds primarily to the eyes within a face and suggests that the N170 is sensitive to the expression changes in the eye region (Schyns, Jentzsch, Johnson, Schweinberger, & Gosselin, 2003).

Many studies have reported that the emotional component of a face was processed at relatively later stages in the recognition procedure, later than the N170 time-window. For example, differences were observed between emotional and neutral faces in the mid-latency or post-perceptual components such as the P300 component or the Late Positive Potentials (LPP) recorded over centro-parietal scalp regions and arising beyond 250ms and most pronounced between 400-700ms (Campanella et al., 2002; Carretie & Iglesias, 1995; Krolak-Salmon et al., 2001). Some studies have shown that the LPP can arise earlier than this time-window, between 160-250ms (Ashley et al., 2004; Eimer & Holmes, 2002). This earlier deflection may reflect the detection of emotional stimuli in working memory and be elicited by the medial prefrontal cortex. It could be related to the P3a component in as much as its time course topography is similar to the P3a. The second phase, beyond 250ms, may reflect the storage of the stimuli in working memory. The LPP can also be referred to as the P3b (Schupp et al, 2006) but it is still unclear as to whether the LPP truly reflects the same underlying processes of the P3 components.

An Early Posterior Negativity (EPN) was observed at 220-280ms with bilateral occipito-temporal topography and was found to be greater when observing

threatening faces compared with neutral faces (Eimer, Holmes, & McGlone, 2003; Sato, Kochiyama, Yoshikawa, & Matsumura, 2001) and positive relative to neutral faces (Schacht & Sommer, 2009). This component has been linked to modulatory feedback from the amygdala to regions in the sensory cortices.

Additionally, a posterior negative potential is evoked by a disgusted expression at approximately 300ms over occipital regions (Ashley et al., 2004; Krolak-Salmon et al., 2001), and in another study a significant ERP activation differentiating disgust from fear and anger was observed between 350-400ms over temporal electrode sites (Sprengelmeyer & Jentzsch, 2006). An emotion-specific N230 was observed at posterior sites to angry, fearful, happy, sad, and surprised compared to neutral faces (Balconi & Pozzili, 2003). Differences between a fearful face and other emotional expressions were observed as late as 550ms (Krolak-Salmon et al., 2001; Sato et al., 2001). Some of these late responses to emotional faces can be sustained over prolonged periods of time, sometimes up to 1s (Ashley et al., 2004; Carretie & Iglesias, 1995; Krolak-Salmon et al., 2001) but these effects have not shown to be specific to particular emotions. These effects may reflect more complex cognitive processes beyond the basic perceptual processing. This tends to be observed only when direct attention is given to the emotional content of the stimuli and not when gender or identity decisions are being made.

Other researchers have demonstrated emotional effects on the amplitude of the early visual P1 component over the posterior cortex, with evidence of an enhanced P1 component in response to negative, relative to neutral or positive facial expressions (typically peaking at around 130ms) (Batty & Taylor, 2003; Eger et al.,

2003; Pizzagalli, Regard, & Lehmann, 1999; Pourtois, Dan, Grandjean, Sander, & Vuilleumier, 2005; Streit et al., 2003). Emotional effects on the P1 were also reported using an implicit processing of non-face pictures, with an enhanced P1 for unpleasant compared with pleasant stimuli (Delplanque, Lavoie, Hot, Silvert, & Sequeira, 2004). This is evidence for very rapid differentiation between positive and negative stimuli, and the negativity bias in attention allocation. This effect can be attributed to an enhanced encoding of sensory processes in visual areas as a result of feedback from regions such as the amygdala modulating the emotionally salient stimulus (Vuilleumier & Pourtois, 2007). Additional activity has been observed around 120ms post-stimulus onset in the fronto-central regions (Eimer & Holmes, 2002, 2007). A modulation of these early visual components has been found, in particular, for fearful faces. This indicates that some aspects of emotional processing can occur independently of the activity associated with the N170 (Itier & Taylor, 2004; Pizzagalli et al., 2002) and the affective meaning of the stimuli can be elucidated well before the perceptual analysis differentiating faces from other objects perhaps within the fronto-temporal limbic regions. The modulation of the P1 component by fearful faces might reflect an enhanced allocation of attention to threat-related stimuli and very rapid top-down effects on the visual cortex during initial stages of processing. This could represent enhanced sensory encoding in visual regions as a result of direct feedback from areas such as the amygdala following the rapid perceptual detection of a motivationally significant stimulus.

The P1, EPN and LPP have all been observed for emotionally salient non-face stimuli demonstrating that they may respond to the emotional context of the stimuli as opposed to the presence of an emotional face. The P1 has shown a smaller

amplitude and delayed onset in high-functioning children with autism compared with age-matched controls (around 10 years of age) (Batty, Meaux, Wittemeyer, Roge, & Taylor, 2011). At around 170ms, the amygdala and orbitofrontal cortices would then modulate perceptual processes via feedback fine-tuning the categorisation and trigger-associated-knowledge via connections to associated cortex and the hippocampus. Additionally, they would execute a motor response via connections to the motor cortex, hypothalamus and brainstem nuclei. The conceptual knowledge of emotional stimuli is achieved by 300ms.

There is a general consensus that emotions can be split into six basic emotions and research into individual emotional processing suggests that distinct brain regions contribute to the perception of specific emotions, as can be seen from functional imaging research and studies looking at brain injury and psychiatric disorder, as well as fear-conditioning in non-human primates and rodents (Kalin, Shelton, Davidson, & Kelley, 2001; LeDoux, 1996). For example, people with Huntington's disease and those with obsessive-compulsive disorder show severe deficits in recognising disgust; conversely people with lesions restricted to the amygdala are particularly impaired at recognising fear. This double dissociation indicates that distinct and non-overlapping neural substrates may be associated with the recognition of distinct basic emotions. A general agreement has been reached on the brain regions involved in the processing of fear and disgust, and a picture is being pieced together for happiness as this emotion is frequently used as a positive comparison for negative emotions such as fear. However, limited information is known about the others: anger, sadness and surprise. A brief description will be given to illustrate the current

behavioural, neuroimaging and electrophysiological findings on the neural basis of these six basic emotional expressions.

1.3.1.1 Specific emotional response - Fear

It has been established that the amygdala plays an important role in the perception and recognition of fear. It has been proposed that there are two inputs to the amygdala, one 'fast' direct route via the thalamus which allows an individual to assess the fearful stimulus initially and react with 'fight-or-flight' and the other 'slow' pathway via the sensory cortex allowing for a secondary more-measured assessment of the stimulus (Le Doux, 1996). Patient studies have illustrated that bilateral amygdalar lesions produce deficits in fear recognition (Adolphs, Tranel, Damasio, & Damasio, 1994, 1995; Anderson & Phelps, 2002). The amygdala may respond to fearful faces very early on in information processing and may modulate cortical face processing via direct feedback projections to visual areas (Amaral et al., 2003). A PET study found amygdalar activation and other areas responsive to increasing emotional intensity such as the anterior insula, pulvinar and anterior cingulate (Morris et al., 1996). Breiter and colleagues (1996) found bilateral amygdala activation on presentation of fearful versus neutral stimuli.

1.3.1.2 Specific emotional response - Disgust

There is a selective impairment of disgust in patients with Huntington's disease (Sprengelmeyer et al., 1996), with patients having neural degeneration in the basal

ganglia and caudate nucleus. The processing of disgust incorporates a neural network involving the insular cortex and the orbitofrontal cortex which is able to integrate visual, auditory and olfactory information. Activation of the right insula was observed when viewing stimuli presenting disgust (Phillips et al., 1997), and activation became greater with increased intensity. Other areas responsive to disgust were the medial frontal cortex, right putamen and thalamus. The anterior insula is connected to the ventro-posterior-medial thalamic nucleus which has been identified as the gustatory cortex in non-human primates. Activation of the basal ganglia and the anterior insula were found by Phillips and colleagues (1997) using fMRI. The converging evidence from neuroimaging studies of healthy subjects and neuropsychological studies of clinical groups thus supports the view that the perception of disgust relies on a neural network incorporating the basal ganglia and the insula cortex.

1.3.1.3 Specific emotional response – Sadness and Anger

These emotions are linked to the concept of empathy; psychopathic patients show reduced autonomic responses to sad and angry emotional expressions, reduced aversive conditioning and reduced startle reflexes. An amygdalar lesion results in impaired recognition of sadness (Fine & Blair, 2000), and damage to the orbitofrontal cortex results in 'acquired sociopathy' with associated difficulties in recognising anger (Blair & Cipolotti, 2000). An fMRI study showed that angry faces elicited an enhanced activity of the posterior part of the right gyrus cinguli and the

medial temporal gyrus of the left hemisphere (Sprengelmeyer, Rausch, Eysel, & Przuntek, 1998). A PET study showed that viewing sad expressions resulted in activation in the left amygdala and the right medial and inferior temporal gyrus, with angry expressions showing significant activation in the right orbitofrontal cortex and bilateral activation in the anterior cingulate cortex (Blair, Morris, Frith, Perrett, & Dolan, 1999).

1.3.1.4 Specific emotional response - Happiness

Smiling is an innate ability with a young infant producing their first smile only hours after birth. Happiness is the most well recognised expression (Ekman & Oster, 1979) with the mean accuracy of recognition reaching 100% in some studies. Patients with amygdala damage and Huntington's disease perform normally when processing happy faces. No amygdala activity was seen when contrasting fear with happiness (Morris et al., 1996), however in contrast the left anterior amygdala responded preferentially to happiness versus neutral faces (Breiter et al., 1996). Activation in the bilateral amygdala and orbitofrontal regions have been observed in both implicit and explicit processing of happy expressions (Gorno-Tempini et al., 2001). The amygdala activity suggests a possible generalised response to emotionally-valenced stimuli rather than a specific response to happy expressions.

1.3.1.5 Specific emotional response - Surprise

Emotion research has generally not focussed specifically on the perception of surprise. The expression of surprise follows the basic pattern of fear, with wide eyes and open mouth and research findings have reflected this. Bilateral amygdala damage has been shown to impair judgement of the intensity of fear and surprise (Adolphs et al., 1994, 1995; Calder et al., 1996). Surprise can be a transitory emotion resulting in fear, and this may explain why the amygdala is involved in the perception of surprise; alternatively it could be that perceptually the two expressions are confused due to similar facial features such as open mouth and wide eyes.

1.3.2 Dynamic properties of faces and facial expressions

Traditionally researchers have used static stimuli to investigate the processing of faces and facial affect, however these images do not necessarily reflect the true form of facial affect as it occurs in real life communication. It is only recently that dynamic stimuli have been used to explore face processing (O'Toole, Roark, & Abdi, 2002) illustrating that faces are very much dynamic objects, with movement varying along both spatial and temporal dimensions. It is clear that judgments about posed facial expressions can be made from static images (Ekman, 1992a), however, additional temporal information provided by the moving face could influence emotion recognition; incorporating specialised regions of the brain involved in the processing of biological motion. Under the conditions of a static picture of an emotional expression, the emotion is usually presented at or very near the peak of emotional display. The perceiver will not then be capable of determining whether, for example,

the elicitor's fear is emerging (indicating an impending threat) or dissipating (indicating the removal of threat). In actual 'fight-or-flight' situations this information could be critical.

Movement is crucial for the interpretation and processing of facial expressions of emotion. It provides an indication about the rapid changes in the emotional state of another individual, and can improve the three-dimensional perception of faces (Knight & Johnston, 1997), and behavioural studies have illustrated that humans are sensitive to temporal cues in facial displays (Edwards, 1998). It has been increasingly recognised that the human brain has neural networks specialised for the analysis of meaningful motion conveyed by body action patterns, or biological motion (Grossman & Blake, 2002). There have been many studies looking at the perception of biological motion; event-related potential studies and source analysis have indicated an early processing network of the right posterior extrastriate cortex (Thierry et al., 2006) with input from the posterior fusiform gyrus and the inferior temporal areas (Wheaton, Thompson, Syngeniotis, Abbott, & Puce, 2004), with activation of the amygdala and the inferofrontal cortex. The social perception of biological motion has been associated with the STS (Allison, Puce, & McCarthy, 2000); eye gaze direction and mouth movements (Puce, Allison, Bentin, Gore, & McCarthy, 1998), and hand and body action sequences (Grossman & Blake, 2002) engage the mid and posterior regions of the STS. Interestingly, the perception of biological motion is spared in Williams syndrome compared with other aspects of motion perception and visuomotor integration (Jordan, Reiss, Hoffman, & Landau, 2002). This line of research has shown the movement of face parts, such as the eyes

and lips, elicit activity in the STS and other anatomically related areas but as such the role of dynamics in emotion perception has been largely untested.

If there are specialised neural mechanisms set up to receive information about biological motion it would seem logical that the dynamic perception of expression is likely to recruit specialised processing resources in response to the facial motion, which is integral to the mental representation of faces. There are over one hundred muscles in the face, and generating facial expressions of emotion requires precise sequenced movements of these facial muscles (Ekman & Friesen, 1982), to produce facial action patterns encoding emotions. Studies have shown that the presentation of dynamic facial expressions improves recognition of emotion (Bassili, 1979; Harwood, Hall, & Shinkfield, 1999; Kamachi et al., 2001; Wehrle, Kaiser, Schmidt, & Scherer, 2000). Others indicate that motion can improve recognition of identity (Bruce & Valentine, 1988; Hill & Johnston, 2001; Lander, Christie, & Bruce, 1999); and age (Berry, 1990) compared with static images. Behavioural studies on dynamic facial expressions show better recognition accuracy during ratings tasks of dynamic compared to static facial expressions (Ambadar, Schooler, & Cohn, 2005; Harwood et al., 1999).

Dynamic face stimuli have been shown to elicit higher activation in various brain regions, such as visual cortices, amygdala and the ventral premotor cortex, compared with static face stimuli. Movement also enhances perceptual, emotional and motor reactions relative to static facial expressions (Yoshikawa & Sato, 2006). In a PET study, Kilts and colleagues (2003) contrasted static and dynamic emotional

expressions displaying happiness and anger. They presented thirteen healthy participants with dynamic videos of the expressions where the emotion waxed and waned during presentation, and also static pictures of the facial expressions at the perceived apex of the expression. The participants had to make judgements of emotion intensity throughout the experiment. The researchers found an increased activation in the visual area V5, the STS, cerebellum and periamygdaloid area for dynamic versus static angry faces, and the visual area V5, extrastriate cortex, brain stem and medial temporal cortical activations for dynamic versus static happy faces.

LaBar and colleagues (2003) presented twelve healthy adults with static photographs of fear and anger, and morphed dynamic images (from neutral to 100% emotional intensity). They reported an increased activation in the amygdala, fusiform gyrus, the ventromedial prefrontal cortex and the STS for dynamic versus static images, especially for fear. Trautmann and colleagues (2009) compared static with dynamic expressions of disgust and happiness and found an enhanced activation in the amygdala, fusiform gyrus, superior temporal gyrus, inferior frontal gyrus and the occipital and orbitofrontal cortex for dynamic versus static images. Humphreys and colleagues (1993) reported a double dissociation in two prosopagnosic patients in performance on facial identity and affect tasks. One patient, who sustained ventral occipito-temporal damage, had problems with both facial identity and expression judgments using static images, however their performance improved significantly when categorising facial expressions using moving point-light displays. The other patient, who sustained bilateral parietal lobe damage, produced a good performance on facial identity tasks but was impaired at both static and dynamic facial affect tasks.

Most studies contrasting static and dynamic images of facial expressions have used morphed dynamic images, only a few studies have used natural moving video images, these include Kilts and colleagues (2003); Gepner and colleagues (2001), who recorded videos of one actress displaying natural dynamic facial expressions of joy, surprise, sadness and disgust; and Trautmann and colleagues (2009). Although these video stimuli may prove more naturalistic it is difficult to control for differences in the level of expression portrayed and the temporal aspects of the expression which are more controlled with morphed expression.

The above studies indicate that processing of dynamic compared with static images of facial expressions appear to more reliably recruit the neural networks associated with the recognition of facial affect, such as the amygdala, fusiform gyrus, inferior occipital, middle and superior temporal regions (including the STS) and the lateral inferior frontal cortex (mirror neuron system); with dynamic faces resulting in a more pronounced and distributed activity compared with static faces. There is a larger and more widespread network of brain areas involved in emotion perception for dynamic compared with static stimuli. This might be explained by the higher complexity of the stimuli; the increased ecological validity; or the higher arousal rates. Dynamic stimuli additionally provide rapidly changing temporal cues of information, which are suggested to improve the three-dimensional perception of faces (Knight & Johnston, 1997). For example, a face changing from a neutral to a fearful face would display a change of facial muscles in a certain temporal sequence. Movement enhances the perception of facial expressions (Ambadar et al, 2005) and reflects social interactions in a more natural way (LaBar et al., 2003; Sato,

Kochiyama, Yoshikawa, Naito, & Matsumura, 2004). Dynamic stimuli have also been associated with better recognition of facial expressions compared with static stimuli in atypical populations such as autism (Gepner, Deruelle, & Grynfeltt, 2001); mentally retarded children (Harwood et al., 1999); and individuals with agnosia (Humphreys, Donnelly, & Riddoch, 1993).

1.4 Developmental perspectives of face processing and the perception of facial expressions

The emergence of human and non-human primate ability to recognise and extract social information from faces (of their own species) is due to a brain system that matures through development with input from the environment and leads to an extended neural network of structures in the brain finely tuned to recognising and interpreting faces. In other words, an immature processing system is present at birth, maturing through infancy and childhood with experience to produce the highly specialised face processing system seen in adulthood.

Faces are one of the most frequently encountered stimuli in infants' environments and in this sense it is not surprising that they will tend to be processed preferentially compared with other categories of visual stimuli. Newborns show a preference for face-like stimuli over non-face-like patterns, and movement has been shown to be an important component of this face preference (Goren, Sarty, & Wu, 1975; Morton & Johnson, 1991). Infants begin to show evidence of face prototype formation from

3 months of age (de Haan, Johnson, Maurer, & Perrett, 2001); before this age face recognition seems to be exemplar-based with each face being coded separately. This prototype formation gives infants the benefit of being able to use their experience to encode new faces. A preference to the gender of their primary caregiver is observed at this age (Quinn, Yahr, Kuhn, Slater, & Pascalils, 2002). In addition to this, infants show a preference for faces of their own racial group, due to a biased exposure at a young age to their own racial group. This ability to recognise faces from an early age has been a focus for research.

One idea is that humans are genetically wired to recognise faces, and this is an innate ability which is online from birth. In support of this view, there are regions of the brain selectively activated by faces (Kanwisher et al., 1997), and newborns preferentially orientate to faces (Johnson, Dziurawiec, Ellis, & Morton, 1991). Others argue that the excessive exposure to faces over our lifetime results in a high level of expertise with faces, but this would be no different from having expertise in another visual category (Diamond & Carey, 1986). A third theory called interactive specialisation has the view that face processing depends on both genetic and environmental factors and it is the interaction of the two that leads to cortical specialisation for face processing. This is highlighted by the Conspec/Conlern theory (Morton & Johnson, 1991) where two distinct brain systems underlie the development of face processing. 'Conspec' is a subcortical system operating from birth that orientates newborns' visual attention towards faces, and 'Conlern', a cortical system sensitive to the effects of experience, emerging around two months of age leading to a more mature face-processing ability. One hypothesis suggests that subcortical brain regions not only detect the presence of faces, orienting the

infant to the face, but also directly influences the activity in cortical areas such as the lateral occipital, fusiform and orbitofrontal cortices. Thus, subcortical regions could partly determine which cortical regions become incorporated into the social brain network during development. In addition, these cortical regions such as the fusiform gyrus, would also receive foveal cortical visual input, producing converging information from different sources, ensuring certain developing cortical circuits became specialised for face stimuli.

1.4.1 Neural bases of development of face processing

There is a general consensus that the neural circuitry for adult face processing differs from that in early infancy and childhood, but how it differs and whether there are additional neural substrates involved in infancy is still debated. There may be a fully formed system in place from birth that gradually matures towards adolescence or alternatively there may be a primitive early system that becomes specialised later and is only fully integrated in adulthood. However, this hypothesis is weakened by the fact that the N170 waveform shows a big difference in infants and adults, and does not become adult-like until adolescence. In a PET study, a distributed network of cortical areas, including the fusiform gyrus and areas not activated in adults, were activated in two-month-old infants in response to unfamiliar faces (Tzourio-Mayoyer et al., 2002). This suggests that the fusiform gyrus and STS are functional in infants during development and exhibit some degree of specificity to faces.

For technical and ethical reasons, it is difficult to use imaging methods to study the development of face processing in healthy human infants, for these reasons ERPs are

the preferred technique to investigate neural correlates. The possible precursor to the adult N170, the N290, is observed maximal over the midline and paramidline posterior cortex, gradually decreasing from approximately 350ms to 290ms between 3 and 12 months. At three months there was a larger N290 amplitude and shorter latency for human faces compared with monkey faces (Halit, de Haan, & Johnson, 2003). Between three and six months of age the N290 is unaffected by inversion (de Haan, Pascalis, & Johnson, 2002). By twelve months, the N290 demonstrates an adult-like modulation of amplitude by inversion, with inversion increasing the amplitude for human but not monkey faces (Halit et al., 2003). Thus the N290 appears to become more sensitive to upright, human faces with age (Halit et al., 2003). Although the N290 has become more adult-like by 12 months of age there are still some differences between the N290 and N170. The N290 peaks later and has a more medial distribution and has a smaller peak amplitude to human faces than the adult N170. The peak latency of the N290 at twelve months is only 15-20ms longer than the peak latency of the N170 in 4-5 year olds (Taylor, McCarthy, Saliba, & Degiovanni, 1999). No effect of inversion on the latency of the N290 is seen, however there are visible inversion effects on the N170 latency. Hemispheric differences, when present, show a larger amplitude over the right compared with left hemisphere for faces (Itier & Taylor, 2002) but not for scrambled faces or objects. The N290 is thought to be related to stages of encoding of the physical information in faces as opposed to the recognition of identity as it is unaffected by the familiarity of individual faces (Bentin & Deouell, 2000; Eimer, 2000b).

The P400 is a positive deflection which follows the N290, elicited by the presentation of a face maximal over posterior lateral cortex (Halit, Csibra, Volein, & Johnson, 2004) with a shorter latency for faces compared with objects (de Haan & Nelson, 1999). Both the N290 and P400 are thought to be precursors to the adult N170 and may reflect functionally equivalent processes, with both becoming more finely tuned to human faces with age. They may become integrated giving rise in older children and adults to the N170. This integration of the N290 and P400 to give the N170 may be due to the maturation of the different neural generators that give rise to the components. The difference in timing of maturation of regions such as the fusiform gyrus, the lateral occipito-temporal cortex and the posterior inferior temporal gyrus, all possible generators of the adult N170, may lead to the changes seen in the N290 and P400 through development as they move towards the adult-like N170. Another possibility is that as the brain grows, the spatial distribution of these generators may change producing a different location or orientation to the scalp.

The N170 was found to have a longer latency in older children than infants (Itier & Taylor, 2004; Taylor et al., 1999) with maturation occurring in mid-adolescence. By four years of age the N170 is clearly seen at the posterior parietal sites, but with latencies extending to 300ms (Taylor, Itier, Allison, & Edmonds, 2001), and can serve as a neurophysiological correlate of face perception in developmental studies from 4 years to adulthood. All this evidence suggests that there is a gradual emergence of specificity to face processing over the first year of life and beyond into adolescence.

The early visual component, P1, is very large and easily measureable in children, and offers an index of an earlier stage of visual processing than the N290. From four

years of age the P1 latency is shorter for faces than objects (flowers) (Taylor et al., 2001) and shorter for upright compared with inverted faces (Itier & Taylor, 2002; Taylor et al., 2001), and shows a steady decrease in latency across childhood (4-15 years old) (Batty & Taylor, 2006; Taylor, Batty, & Itier, 2004). However, the extent to which the P1 reflects the processing of specific face stimuli rather than general visual attentional effects remains open to debate.

1.4.2 Development and emotion perception

There is evidence that the social context of the face can affect face processing in infancy. Although infants do not possess all the mechanisms for identification of an emotion such as the verbal label for the expression or the conceptual knowledge about the emotion the expression conveys, infants are still able to respond to emotional signals and show they have some understanding of the social message. For example, infants tend to regulate their own behaviour based on social signals and they can respond to expressions with vocalisations or expressions of their own (Soken & Pick, 1999). Newborns are able to imitate facial expressions of emotion (Field, Woodson, Greenberg, & Cohen, 1982) and this might reflect the initiation of a pathway for the understanding of social meaning. In adults, research has shown that the perception of an emotion triggers an emotional response in the perceiver that can be measured (Dimberg, 1982). This would then produce an emotional state in the perceiver adding to their understanding of the other person's emotional state.

Infants only a few months old seem to be able to discriminate between different expressions of emotion (Schwartz, Izard & Ansul, 1985; Younge-Browne, Rosenfeld & Horowitz, 1977). In one study, three-month-old infants were habituated to a smiling or a frowning face and then tested on both (Barrera & Maurer, 1981). Infants looked longer at the novel expression than the one they had been habituated to. By 5-7 months the infants' visual system is sufficiently developed to support discrimination of most facial expressions (Bornstein & Arterberry, 2003), and by 6-7 months infants can discriminate between most emotional pairings in habituation paradigms, including recognising that different examples of the same expression belong to the same category (Nelson & Dolgin, 1985; Nelson, Morse, & Leavitt, 1979). There may be some evidence for an early 'positivity bias' (Vaish, Grossmann, & Woodward, 2008) as some earlier studies found longer looking times for happy faces than for angry and neutral faces in 5-month-olds (LaBarbera, Izard, Vietze, & Parisi, 1976; Wilcox & Clayton, 1968). However, by seven months, infants preferentially attend to a fearful face over a happy face (Kotsoni, de Haan, & Johnson, 2001; Nelson & Dolgin, 1985) demonstrating a 'negativity bias' later in development. By seven months, infants can discriminate happy from surprised, angry, sad and fearful expressions (Kestenbaum & Nelson, 1990; Ludemann & Nelson, 1988).

Infants as young as 9-12 months are guided by their parent's emotional behaviour, and by two years of age infants seek out the face as an emotional cue. For example, infants will crawl over a visual cliff if their mother's face displays a happy expression, whereas they avoid the cliff if their mother poses a fearful expression by 12-months-of-age (Sorce et al, 1985). Five-month-old infants show an increased eye blink startle

response when viewing angry facial expressions (Balaban, 1995). This heightened startle response is known to be mediated by the amygdala (Angrilli et al., 1996) and thus demonstrates that the amygdala and its associated network are already functional and responsive in the early stages of postnatal development. This is substantiated by research with macaque monkeys showing the cortico-amygdalar connections are established soon after birth (Amaral & Bennett, 2000). Infants are particularly sensitive to information from the eye region and can distinguish between direct and averted eye gaze from birth (Farroni, Csibra, Simion, & Johnson, 2002). Faces with direct gaze elicit a different electrophysiological response than averted gaze in four-month-olds (Johnson & Farroni, 2003) and produce an enhanced processing of faces. Infants will follow another's eye gaze but it is not until about one year that they can attend to the object of another's attention (Carpenter, Nagell, & Tomasello, 1998).

Only a few studies have investigated whether the amplitude and latency of the P1, the early visual component, and the infant face-sensitive ERP component N290 are affected by emotional expression. Given the conflicting findings of response of the early latency components to emotional stimuli in adult populations, it is unclear as to whether there would be a differential response for these components in infants. Hoehl and Striano (2008) conducted a study on the perception of fear and anger depending on eye gaze and found that the N290 was significantly larger in amplitude for fearful faces compared with angry faces regardless of eye gaze direction in 7-month-old infants. They found this effect more strongly over central occipital channels (O1 & O2) and not over lateral channel sites (P7 & P8). This fits in with the

theory that the N290 in general shows a more medial distribution compared with the lateral distribution of the adult N170. However, few studies have investigated the specific response of the N290 to different emotional facial expressions.

The P1 has been shown to be affected by emotion, peaking later for fearful faces compared with neutral and positive faces in children 4-15 years old, and this was most pronounced in children between 4-7 years old (Batty & Taylor, 2006). Some recent studies have shown that the early positive component P1 differs in latency between fearful and neutral/happy facial expressions in 1-7-year-old children (Batty & Taylor, 2006). This demonstrates that the discrimination of facial expressions may occur very early in visual processing stages in young children as it does in adults. However, some studies did not see a difference in the amplitude or latency of the P1 in 7-month-old infants when presented with fearful, happy and neutral faces (Leppänen, Moulson, Vogel-Farley, & Nelson, 2007).

They may be a negativity bias later in development, as seen in social referencing studies in older infants (Vaish et al., 2008). Fearful faces elicit a larger P400 compared with neutral or happy faces over the medial occipital scalp in 7-month-old infants (Leppänen et al., 2007). In this age group, the negative component (Nc) is larger in response to fearful compared with happy faces over fronto-central scalp regions (Leppänen et al., 2007; Nelson & de Haan, 1996). The Nc is related to the orienting of attentional resources in response to salient stimuli and has been localised to the anterior cingulate region, an area involved in the regulation of attention (Reynolds & Richards, 2005). The amygdala is thought to play a part in establishing a social brain network early in development by enhancing processing of

emotionally salient stimuli and may be functional from birth, involved in mediating the fine-tuning of cortical regions specialised for processing of emotional stimuli (Johnson, 2005; Leppänen & Nelson, 2009). The amygdala may influence cortical face processing via direct feedback projections to ventral visual areas (Amaral et al., 2003). The amygdala also projects cholinergic neurons in the nucleus basalis that releases acetylcholine onto cortical sensory neurons increasing their activity (Bentley, Vuilleumier, Thiel, Driver, & Dolan, 2003). The findings of differential ERPs to fearful faces around seven months of age raises the possibility that connectivity from subcortical brain structures such as the amygdala to cortical regions comes online early in development. There are reciprocal projections between the amygdala and visual areas of the temporal and orbitofrontal cortex, which have been observed soon after birth in anatomical tracing studies in non-human primates (Machado & Bachevalier, 2003). This implies that brain structures involved in emotion perception may be partly functional by the time infants' exhibit discrimination between different emotions. A second potential reason for the differential response to fearful faces could reflect the novelty of the open eyes and increased size of the white sclera around the dark pupil, that expressions of fear are relatively novel in normal social environments. This novelty could lead to more processing resources required to perceive and recognise fearful faces. However, this explanation is not supported by the finding that 4-month-olds show no preference between expressions, and they should find the fearful face even more novel than 7–month-olds (Ludemann & Nelson, 1988). Alongside these findings there are behavioural results that show that infants tend to look longer at fearful faces compared with happy faces, showing a preferential allocation of attention to fearful over happy faces (de Haan, Belsky,

Reid, Volein, & Johnson, 2004; Kotsoni et al., 2001; Leppänen et al., 2007; Nelson & Dolgin, 1985), however, this bias was not seen in adults, even though fearful faces elicited a larger N170 in the same study (Leppänen et al., 2007). A recent near infrared spectroscopy (NIRS) study investigated responses to happy and angry facial expressions in 6-7-month-olds over the T5/6 positions, which are sensitive to STS activity (Nakato et al., 2011). The response to happy expressions increased slowly and persisted even after the stimulus disappeared, whereas the response to angry expressions peaked quickly and disappeared quickly after stimulus offset. In addition, the response to happy expressions was prominent over the left (T5) and angry expressions on the right (T6) compared a baseline condition of pictures of vegetables.

1.4.3 Dynamic displays of emotion in infants

1.4.3.1 Development of the visual system and motion processing

Motion processing is one of the most important features of the visual system. It contributes to the perception of facial expressions, depth perception, recognising biological motion, and social communication which can be interpreted from others' actions. The primary visual cortex achieves adult-like organisation very early in life and sensitivity and binocularity skills reach adult competence within the first two years of life (Hainline & Riddell, 1995). Immaturity of cone photoreceptors is a significant factor limiting an infant's acuity, and optic nerve myelination is incomplete in the first few months resulting in long transmission latencies. Despite

this, very young infants will orientate towards a moving target (Volkmann & Dobson, 1976), however this does not imply that infants can necessarily extract motion information.

Temporal resolution, the ability to register events that are separate in time, is a prerequisite for the recognition of motion. Flicker detection is the simplest way of measuring this, and by four weeks of age infants can detect flicker at 40 Hz (about 75% of adult performance) and it reaches adult sensitivity by twelve weeks (Regal, 1981). Braddick and colleagues (2003) observed a visual evoked potential (VEP) response specific to orientation-selective neurons in the visual cortex of humans. The age of onset depended on the temporal frequency with eight orientation changes per second seen at six weeks of age and three changes per second observed at three weeks of age (Atkinson et al, 1990). This is seen in single neurons in areas visual areas V1 and V2 of the infant monkey cortex. The 4-week-old monkeys' V1 cells respond to modulated gratings up to 80% of the maximum temporal frequency of adult cells, but the temporal properties in V2 showed a much slower maturational course (Zheng et al, 2007).

Spatial integration of motion information occurs in the visual area V5, a region of the visual cortex. Its maturation during infancy can be assessed by global motion coherence, the ability to respond to extended random-dot patterns in which a percentage of the dots move in the same direction and the remaining dots move randomly. There is evidence of global integration by three months and coherence thresholds improve between three and five months (Wattam-Bell, 1994). By three months the infants demonstrate VEPs and reveal sensitivity to global motion coherence (Braddick, Wattam-Bell, Birtles, Atkinson, von Hofsten & Nystrom, 2007).

This shows that directional information is integrated for higher level perceptual processes in V5, and that connectivity of the V1 and extrastriate regions exists early on in development, even if it is not fully mature.

There is a longstanding view of the existence of two parallel hierarchical pathways in vision, a dorsal (occipito-parietal) pathway concerned with spatial properties of vision (the "where?" pathway), and the ventral (occipito-temporal) pathway concerned with identification of the visual objects (the "what?" pathway). This thinking was influenced by a study by Ungerleider and Mishkin (1982) in non-human primates that found lesions to the two distinct pathways produced specific deficits in processing visual stimuli, which was later observed in humans (Goodale & Milner, 1992). More recent studies have emphasised the functional integration of the two pathways in normal object recognition to enhance cue-variant and viewpoint-invariant recognition by use of three-dimensional information (Farivar, Blanke, & Chaudhuri, 2009). There is evidence that the dorsal stream may mature later in infancy than the ventral stream, and temporal properties seem to be sensitive indicators of neurodevelopmental disorders. Sensitivity to pattern properties (e.g. orientation) is apparent earlier in cortical development than to directional motion (Braddick, Atkinson, & Wattam-Bell, 2003), possibly due to immature temporal organisation of the inputs to visual cortical areas. In contrast, subcortical visual processing routes are functional in early infancy, as shown by control of optokinetic nystagmus by newborns (Banton & Bertenthal, 1997).

1.4.3.2 Biological motion and emotion processing

Humans detect and interpret biological motion very early on in development. This can be shown as a preference to attend to biological motion over other forms of motion such as drifting dots (Bertenthal, Banton, & Bradbury, 1993). Three- and five-month-old infants can discriminate point-light displays (Proffitt & Bertenthal, 1990) and the ability to perceive biological motion could be in place as early as twelve weeks. The movement conveys information to the infant about the underlying schematic representations. Between four and eight months, infants are able to discriminate between facial motion sequences and between different actors, suggesting that infants can not only discriminate complex and subtle biological motion cues but can also detect invariants in such displays (Spencer et al., 2006). It has been shown that 8-month-old infants process biological motion in the parietal regions of the brain compared with scrambled motion approximately 100ms after stimulus onset (Hirai & Hiraki, 2005); with this also seen when comparing upright and inverted biological motion point-light displays of walking and kicking (Reid, Hoehl, & Striano, 2006). At nine months infants can differentiate between distorted and normal human body schema when in animation (Heron & Slaughter, 2004), compared with eighteen months of age when infants can distinguish between static images of normal and distorted human body configurations. A larger positive amplitude was observed over parietal regions between 300ms and 700ms when eight-month-old infants viewed biomechanically impossible point-light displays compared with possible biomechnical motion (Reid et al, 2008).

From studies that have used moving images of facial expressions, it is clear that infants can distinguish happy from angry and sad expressions (Walker, 1982). Walker used a preferential looking procedure where two dynamic videos of facial

expressions were shown side by side whilst a single vocal expression specific to one of the facial expressions was presented. Walker observed that the infants preferentially attended to the facial expression corresponding to the vocal display they were hearing. The infants discriminated between happy and sad at five months; and happy from neutral and angry at seven months. By seven months, infants can also discriminate between discrete dynamic expressions if both are positive or negative affect, they could distinguish between happy, interested, angry and sad expressions (Soken & Pick, 1999). In a recent near infra-red spectroscopy study (NIRS), Minagawa-Kawai and colleagues (2009) measured changes in prefrontal cortex activity to explore the neural substrates underlying social and emotional attachment in 9-13-month-old infants. They revealed that activity in an infant's anterior orbitofrontal cortex increased with viewing videos of their mother's smiling face compared with their mother's neutral face, or a stranger's face. The mothers showed a similar response when viewing their own infant's smiling face, however they also showed a markedly reduced response to the unfamiliar infant's smiling face compared with the infant's response to the smiling stranger, demonstrating that the orbitofrontal cortex becomes more selective to happy expressions in adults. Dynamic stimuli have been shown to facilitate the recognition of emotional facial expressions in children with autism (Gepner et al., 2001). This is in contrast to studies which have found lower performance in autistic children when presented with static pictures of facial expressions (Gepner et al, 1994; Tantum et al, 1989).

In summary, the development of emotion perception is protracted throughout infancy. In early development, infants respond to facial social stimuli, and from a few

months can discriminate between positive and negative facial expressions. By 5-7 months infants can discriminate between all six basic emotions, and by one year they are guided by their parent's emotional behaviour. The N170 precursor, the N290, is enhanced for fearful faces compared with angry faces by seven months, and this is maximal over medial scalp locations. There has not been an effect of emotion observed on the P1 in infants, but studies in children between one and fifteen have shown a later peak for fearful faces compared to neutral and happy faces. Motion processing also matures throughout infancy, by 4-8 months infants can discriminate between complex facial motion cues. Infants can discriminate between happy and sad dynamic expressions by 5 months, and happy and anger dynamic expressions by seven months. In addition, at seven months they can distinguish between discrete dynamic expressions of positive and negative affect. The orbitofrontal cortex has been shown to be active when 9-13-month-old infants view happy compared with neutral faces.

1.5 Atypical development and temporal lobe epilepsy

One cause of emotion perception deficits is temporal lobe epilepsy (TLE), with mesial temporal sclerosis (MTS) being the most common neuropathological finding resulting in neuronal loss and gliosis of the hippocampus, entorhinal cortex and the amygdala complex (Bruton, 1988). MTS has been associated with early childhood febrile convulsions (resulting in damage to the amygdala) and subsequently with drug-resistant seizures during adolescence. Volumetric measurements reveal a

volume reduction in the amygdala that varies between 10 and 30% (Cendes et al, 1993) and isolated amygdala damage is observed in 10% of patients with TLE (Bartlett et al, 2002). The outcome for medically intractable TLE is curative temporal lobectomy, where the anteromedial temporal lobe is resected. However one consequence of this type of surgery is the resulting partial unilateral damage to the amygdala and surrounding structures after resection (Spencer et al, 1984). Importantly, neither right nor left temporal lobectomy, when confined to the anterior temporal lobe, appears to compromise basic visual perception (Huxlin & Merigan, 1998; Mendola & Corkin, 1999).

There are numerous studies investigating the effects of temporal lobe epilepsy, and temporal lobectomy, on the recognition of emotion from facial expressions. General deficits have been observed for negative emotions, particularly fear. Those patients with bilateral lesions affecting the amygdala have the most severe deficits, followed by the unilateral right-sided cases, and least so for the left-sided cases. This pattern is observed in cases before and after lobectomy. The age of onset appears to have a direct correlation with recognition deficits, with age of epilepsy onset below five years of age producing the greatest deficits. Below is a brief summary of the main findings from the studies looking at the effects of temporal lobe epilepsy and lobectomy on emotion recognition.

In two studies by Meletti and colleagues (2003, 2009) subjects were presented with pictures of facial affect and instructed to match expressions with one of the five basic emotions, anger, disgust, fear, happiness or sadness. They found that emotion

recognition was most severe for mesial temporal lobe epilepsy (MTLE) patients with bilateral damage, followed by the right mesial temporal lobe epilepsy (RMTLE) patients, and then the left mesial temporal lobe epilepsy (LMTLE) patients. There was impairment for all negative emotions for the RMTLE group, especially for fear, and to a lesser extent anger, disgust and sadness. The LMTLE group performed as well as the control groups. This is consistent with the idea that the right mesiotemporal regions are involved in the recognition of negative emotions associated with withdrawal and avoidance. For the RMTLE group, this deficit was particularly severe in subjects with seizure onset before five years of age. The comparison groups were patients with temporal lesions outside the mesial regions and those with extratemporal lesions in the earlier study (2003), and lateral temporal lobe epilepsy patients and healthy controls in the latter study (2009). There was an indication that the emotion deficits were not selective in the right side, but were also present in one in five left-sided cases.

Benuzzi and colleagues (2004) found that the RMTLE group was impaired in naming the facial affect they observed, when compared with healthy controls. They also found that the RMTLE group with early seizure onset could not identify fear of varying intensity when blended with other emotions. Controls and LMTLE cases demonstrated bilateral activation in the mesial temporal lobe structures including the amygdala in response to faces, whereas the RMTLE group was restricted to left hemisphere activation.

In an fMRI study by Batut and colleagues (2006), healthy controls and the LMTLE group who performed similarly in the recognition of fear activated the occipital and frontal network including the amygdala when perceiving fearful faces. In contrast, the RMTLE group who failed to recognise fear, had no amygdala activation, with a reduced area of activation in the left occipital and frontal cortices. When exposed to unpleasant scenes, the RMTLE group exhibited a restricted pattern of activation, without the involvement of the fusiform areas, which in contrast was activated in the LMTLE and control groups. The perception of pleasant scenes and happy faces did not produce differential behavioural responses between groups, however, the neural responses of the RMTLE & LMTLE groups to controls differed. The activated network was extended to the inferior and lateral right temporal cortex for the LMTLE group and the left temporal cortex was activated for the RMTLE group.

Golouboff and colleagues (2008) assessed recognition of children's facial expressions (anger, disgust, fear, happiness, and sadness) in children and adolescents (8-16 years old) with TLE and two control groups (extra-temporal frontocentral epilepsy and healthy controls). The LMTLE group was impaired in recognising fear and neutrality, the RMTLE group was impaired at recognising disgust, and performed worse in a face memory task. They observed more frequent fear recognition deficits in the LMTLE group than the RMTLE group but this result could be due to the earlier age of onset in the LMTLE group. Not all children with TLE were impaired in recognising emotional expressions, consistent with the evidence that mesiotemporal damage does not always result in deficits. However, all of the children who had experienced febrile convulsions were impaired at recognising fear, supporting the idea that early

seizures disrupt the mesiotemporal structures causing deficits in emotion recognition.

A recent study by Bonelli and colleagues (2009) used a memory encoding fMRI paradigm, including fearful, happy and neutral faces. They found that there was left lateralised amygdala activation for healthy controls, and bilateral amygdala activation for the RMTLE group when participants viewed fearful compared with neutral faces. In contrast, there was a significantly reduced bilateral activation for the LMTLE group. No difference in activity was observed when comparing happy with neutral faces.

Graham and colleagues (2006) used a sequential ordering task using morphed faces to assess the role of the amygdala and other temporal lobe regions in the perception of featural displacements accompanying changes in facial emotion and identity. One of the patient groups the task was administered to was a group of unilateral medial temporal lobectomy patients (six right-sided, seven left-sided). Four morph progressions were used, three emotion morphs (neutral to anger, neutral to fear and fear to anger) and an identity morph. This temporal lobectomy group did not differ from a control group in terms of sorting time or overall performance.

These studies showed that there may be deficits in emotion perception before temporal lobectomy, however impaired emotion recognition is observed after

surgery as well. One consequence of a temporal lobectomy is the resulting unilateral damage to the amygdala and surrounding structures after resection.

McClelland and colleagues (2006) assessed a population of patients with MTS that underwent a uniform right temporal lobectomy with healthy controls. Subjects were instructed to match the facial affect (anger, disgust, fear or happiness). They found that the early onset group (TLE onset before the age of six) had a significantly lower mean accuracy for fearful faces compared to the control group.

Another study focussing on the performance of emotion recognition after temporal lobectomy (TL) was carried out by Anderson and colleagues (2000). TL patients and healthy controls were asked to rate the intensity of pictures displaying the six basic facial expressions. The emotion recognition was impaired in the RTL group compared with the LTL group and controls. They provided evidence that right temporal lobe damage can result in impaired recognition of fearful faces with 25% of the RTL patients showing severely impaired ratings of fearful faces compared with controls, perhaps relating to a diminished sensitivity to fear as opposed to a total inability to recognise fear (Adolphs et al., 1994). None of the LTL group showed impairment on rating fearful faces. The RTL group also demonstrated impairments in evaluating the other negative expressions related to withdrawal, disgust and sadness, but not anger.

Adolphs and colleagues (2001) also compared temporal lobectomy patients to brain-damaged control participants whose lesions did not involve the anterior lobe, ventromedial frontal lobe or right parietal lobe. As in the study by Anderson and

colleagues, participants had to rate the intensity of the six basic expressions. Participants with RTL had significantly lower overall performance than the brain-damaged controls, with the recognition of fear being significantly the worst. There was an overall negative correlation between the extent of amygdala damage and the recognition of emotion, however they noted that some participants with complete unilateral amygdala damage performed normally and some with no visible amygdala damage had an impaired performance in emotion recognition.

A study by Hlobil and colleagues (2008) compared the deficits of emotion perception in groups of patients before and after temporal lobectomy (36 prior to surgery who had unilateral mesial temporal sclerosis and 40 after surgery, seizure-free), and healthy controls. Fourty-five patients had RMTLE with hippocampal sclerosis, and 31 had LMTLE with hippocampal sclerosis. Pictures of facial expressions were presented, four of the same emotion and one target emotion (happiness, fear or anger). The results showed that fear recognition was impaired in pre-surgery patients with RTLE with early seizure onset. A deterioration in facial emotion recognition capabilities was not observed in these patients after surgery. The results also indicate that the TLE patients improve their ability to recognise happiness after surgery. They concluded that right mesial temporal damage with seizure onset before six years of age is the most important predictor of impaired fear recognition in patients with medically intractable TLE.

The influence of dynamic facial images in emotion processing in patients with TL is unclear, as there have been few studies using moving stimuli. The limitations of these studies are described below.

Cristinzio and colleagues (2010) used computer-generated animated faces displaying dynamic expressions of anger, fear and happiness as low (50%) and high (100%) intensity, and eye movements (straight and averted). TLE patients and healthy controls rated the intensity of the expression (similar to the task in Anderson et al., 2000 and Adolphs et al., 2001). They tested the interaction between facial expressions and eye gaze. Both patient groups performed significantly worse than the control group at rating the correct emotion category for fear and anger, but not happiness.

In another study by Graham and colleagues (2007) computer-generated morphs of neutral-to-anger, neutral-to-fear and fear-to anger were presented to a patient with bilateral amygdala damage, patients with unilateral temporal lobectomy damage (six RTL & seven LTL), and matched controls. In a two-alternative forced-choice identification task, the patient with bilateral amygdala damage was found to be less sensitive to small changes in emotional intensity for both fear and anger, whereas the unilateral TL patients showed intact performance on all three morph progressions. This disparity to other studies finding differences between TLE groups, may be due to task differences, such as the use of morphed expressions, or alternatively the heterogeneity of the groups.

Schacher and colleagues (2006) used dynamic stimuli to assess amygdala activation in TLE patients and two control groups (healthy controls and those with extratemporal lesions). Subjects were presented with dynamic high intensity fearful faces taken from thriller or horror films alongside dynamic clips of landscapes whilst in a scanner. After the clips subjects were asked to rate their emotional involvement on a scale from 0 to 10. There was bilateral amygdala activation in the control and patients groups without TLE when viewing fearful faces. In contrast, the patients with TLE typically showed lateralised amygdala activation contralateral to the side of seizure onset. This showed in all cases that motion with an emotional content induced amygdala activation but was clearly lateralised in patients with TLE. These three studies do not, however, directly compare static and dynamic images of emotional expression, and did not use the full range of emotional expressions.

Young and colleagues (1996) used both static and dynamic facial expressions to assess emotion processing in a patient with a partial bilateral amygdalotomy (D.R.) compared with healthy controls. Dynamic images were video clips of actors displaying the six basic emotions, with static counterparts. The participants performed identification and matching tasks. D.R. was impaired in emotion recognition for both static and dynamic expressions for all tasks. The lack of improvement in emotion recognition in dynamic stimuli for D.R. may be due to extent of the amygdala damage which is not overcome by the additional emotional cues that may be present in the dynamic stimuli. Alternatively, there may be limitations to the dynamic stimuli such as the length of the clip (6 seconds) and the inconsistency of emotion expressed between actors (four actors). The accuracy of

emotion recognition for static and dynamic stimuli for the control group was not recorded so it is unclear as to whether there was an overall advantage for the dynamic stimuli in this study.

The main findings from the studies described indicate that patients with mesial temporal sclerosis have deficits in recognising specific facial expressions of emotion, especially fear. This is most evident amongst those with right sided lesions both before and after surgery. This group of patients seem to have selective deficits in emotion perception which do not extend to facial identity deficits or other visuoperceptual abilities, illustrating that the amygdala and other temporal structures are not necessary for general visual perception. Where presented, the bilateral cases performed even worse than the right-sided cases. Activation of the amygdala tended to be observed in the contralateral side from the lesion in right-sided cases, but had a more bilateral pattern in left-sided cases. There was a correlation between early onset of seizures and emotion deficits throughout the studies, especially for fear. It is therefore highly likely that there is a critical period for establishing the neural network underlying the ability to recognise facial expressions of emotion. It may be the case that early damage to part of the network involved in emotion perception may result in deficits whereas later structural or functional damage may not result in a deficit. There seems to be a critical period for the development of emotion recognition between five and eight years, leading to an increasing distinction between the emotions surprise and fear, surprise and happiness, and disgust and fear (Gosselin et al, 1995; Gosselin & Simard, 1999). This raises the question of plasticity, and whether seizure activity early in life prevents

functional reorganisation and affects the development of emotion recognition abilities. This can be seen from studies showing evidence that early onset seizures and the presence of right-sided MTS is the main interaction leading to severe impairment in recognising facial expressions of emotion (Benuzzi et al., 2004; Meletti et al., 2003), and supported by a recent study which showed that emotion recognition deficits were already present during infancy in patients with early onset seizures (Golouboff et al., 2008). This would be contrary to other observations where early damage is often compensated for by plastic processes. This would mean that temporal resection on patients who have early onset epilepsy (approximately < 6 years) would not alter their emotion recognition deficits later on in life.

There is a general consensus that the right-sided MTS cases have more deficits than the left-sided cases and healthy controls, fitting in with the hypothesis that there is dominance of the right hemisphere in emotion perception. However, this finding is not consistent across studies, the heterogeneity of the population is such that group differences are not always observed, with pronounced deficits only visible at the individual level. There is a great deal of variation in stimuli and tasks between studies, and comparisons made between different control groups. Caution must be taken in making a general conclusion about the hemispheric bias in emotion recognition based on lesion studies as often the sample size is small and there is variation in clinical features between individuals, such as age of epilepsy onset, duration of epilepsy, location of lesion, and etiology. The influence of motion on emotion processing in this population is still unclear, as previous studies have not

shown an advantage for dynamic stimuli. However, there has not been a direct comparison of static and dynamic facial expressions in TLE and control populations.

Disruption to the areas of the brain responsible for emotion perception during development leads to deficits in perception which last into adulthood. The main regions involved in emotion perception are found in the temporal lobe, and include structures such as the amygdala, fusiform gyrus and superior temporal sulcus, as discussed above. Both lesion and neuroimaging studies have demonstrated that the amygdala plays a large role in mediating and processing emotional stimuli (Adolphs et al., 1999; LeDoux, 2000) and has a neuromodulatory effect on cortical regions such as the fusiform gyrus, superior temporal sulcus and frontal cortex during sensory processing (Iidaka et al., 2001; Morris et al., 1998). There is evidence suggesting that bilateral amygdala damage results in generalised facial emotion recognition deficits, with fear being the most severely affected (Adolphs et al., 1995). Unilateral damage generally results in more subtle impairments (Morris et al., 1998). The right amygdala is preferentially activated in response to subliminal presentation of emotions, whereas the left amygdala is activated when the stimuli are presented for longer (Morris, Ohman, & Dolan, 1999). The temporal pole and rhinal cortex have reciprocal connections with the amygdala and have been shown to modulate emotional behaviour in animals (Aggleton & Young, 2000). The amygdala has been shown to respond to low frequency components of faces and facial expressions through magnocellular routes, whereas ventral and lateral temporal cortical areas have been linked to processing high frequency components via parvocellular channels (Vuilleumier, Armony, Driver, & Dolan, 2003). This then suggests that there

are two pathways for the perception of facial expressions, one rapid, amygdala-mediated pathway and a second, more voluntary, cortically-mediated pathway.

There have been inconsistencies between studies of patients with bilateral amygdala damage, with different patients showing varying degrees of facial emotion decoding abilities (Adolphs et al., 1999; Adolphs & Tranel, 2004). The amygdala's involvement in processing other emotions than fear has also presented mixed results (Blair et al., 1999; LaBar et al., 2003). One explanation of this could be that patients with amygdala damage fail to spontaneously attend to the eye region of the face, critical for the perception of fear. This was observed by Adolphs and colleagues (2005) on a patient S.M. who had deficits in processing fearful faces. However, when S.M. was instructed to attend to the eye region of the face her perception of fearful faces appeared normal.

The amygdala's ability to influence regions such as the fusiform gyrus during emotion processing is illustrated by a study conducted by Vuilleumier and colleagues (2004). They demonstrated that damage to the amygdala, from mesial temporal sclerosis, influenced the activity of the fusiform and occipital cortical regions and disrupted the enhanced neural response to emotional stimuli especially fearful faces. In fact the greater the degree of amygdala sclerosis, the smaller the differential response to fearful versus neutral faces in the visual areas of the ipsilateral hemisphere. Morris and colleagues (1998) postulated that there was a neuromodulatory feedback response from the amygdala to extrastriate cortical regions. In contrast to the above findings, they found a specific interaction between

the amygdala and the fusiform gyrus in response to fearful faces, where reduced activation of the amygdala was paralleled by enhanced activation of the fusiform gyrus.

1.6 General Summary

Face stimuli activate regions in the occipito-temporal cortex including the fusiform gyrus and the STS, with a right hemispheric bias. The fusiform gyrus is thought to be involved with the invariant and the STS the changeable aspects of the face. Facial expressions of emotion activate these temporal lobe structures, and in addition, areas such as the orbitofrontal cortex and amygdala, and individual emotions tend to enhance distinct regions in a distributed network. There are direct reciprocal connections between the amygdala and the visual cortices, with the amygdala influencing the activity of fusiform gyrus to facial expressions. There is a debate as to whether the early latency ERP components in adults, the P1 & N170, are modulated by emotion. Effects observed include an enhanced but delayed N170 for fearful compared with neutral and happy facial expressions, and an enhanced P1 in response to negative (unpleasant) compared with positive (pleasant) expressions. Motion improves the recognition of facial expression, with an enhanced activation observed in regions usually responsive to emotional stimuli, and a more distributed network than static stimuli.

The development of emotion processing takes place throughout infancy, by 5-7 months infants can discriminate between all the basic emotions. The N170 precursor, the N290, is enhanced for fearful faces compared with angry faces by

seven months, and is maximal over medial regions. This effect has not been observed for the P1, however studies in older children have shown a later peak for fearful faces compared to neutral and happy faces. There is a protracted development of motion processing, dynamic happy and sad expressions can be discriminated by five months, and happy and anger dynamic expressions can be discriminated by seven months. By this time, infants can distinguish between discrete dynamic expressions of positive and negative affect. In atypical development, patients with temporal epilepsy have deficits in recognising specific facial expressions, especially fear. This is a particular problem amongst RMTLE patients compared with LMTLE patients and controls, both before and after surgery, evidence that there might be a right-sided bias for emotion perception. There is inconsistency in findings across studies, which may be due to the heterogeneity of the population, sample size or variation in task properties, so caution should be taken in interpreting the results. The influence of motion on emotion processing in patients with TLE is still unclear and there has not been an obvious advantage for moving stimuli. However, previous research has not directly compared static and dynamic facial stimuli in the TLE population.

The following will be a brief outline of the thesis with questions to be addressed.

Question 1: Does motion influence the early latency processing of facial expressions in adults?

This question will be addressed in Chapters 3 & 4. In Chapter 3, the accuracy and confidence of recognition and intensity of the stimuli were obtained for a set of static facial expressions and their dynamic counterparts, covering the full range of basic emotions. These data were then used to identify a set of stimuli employed in Chapter 4, where ERPs were recorded to static and dynamic versions of the six basic emotions (anger, disgust, fear, happiness, sadness, surprise), as well as non-social static and dynamic stimuli. The main predictors were that motion would enhance the response of the early-latency components, the P1 and N170. This motion effect may be the greatest for those emotions more difficult to recognise in the static form.

Question 2: Does paediatric injury to the mesial temporal lobe affect behavioural recognition and/or early-latency neural processing of static or dynamic facial expressions?

This question will be addressed in Chapters 5 & 6. In Chapter 5, ERPs were recorded to static and dynamic stimuli, as in Chapter 4, in a population of right- and left-sided temporal lobectomy patients, alongside a healthy control group. The main predictors were that both the P1 and N170 would be diminished and/or slower for the right-sided group compared with the other two groups, particularly for fear. Any deficits observed for static stimuli may be reduced for dynamic stimuli due to the recruitment of a more distributed neural network. In Chapter 6, recognition of facial and vocal emotion was assessed in the patient groups. The main predictor was that the right-sided group would perform worse at emotion recognition tasks, particularly for fear. The basis for the predictions made in both studies was previous findings

that right-sided lesions produce greater emotion recognition deficits, which may be due to reduced activity in neural networks.

Question 3: Does motion influence infants' processing of facial expressions?

This question will be addressed in Chapter 7. ERPs were recorded for static and dynamic stimuli, for fear and happiness only, in 4-12-month-old infants. The main predictors were that static stimuli would produce a larger/faster P1/N290 response than dynamic stimuli, on the basis that the ventral visual stream matures faster than the dorsal visual stream. In addition, fear would produce a larger response in younger infants due to the sensitivity of the eye regions during development, enhancing attention towards fearful expressions.

2. General Methods

The main aim of the research presented in the thesis was to investigate the effects of motion on the neural correlates of emotional development. A comparison was performed between static and dynamic facial expressions across three different populations; typically and atypically developed adults, and typically developing infants. The main method used to address this question was electrophysiological techniques, specifically extracting event-related potentials (ERPs) from the electroencephalogram (EEG). This technique is repeated throughout the thesis (Chapters 4, 5 and 7), and thus an introduction providing a general description of the derivation and recording of ERPs, and an overview of data acquisition and analysis will be described in this chapter. In addition, a description of the creation of the dynamic stimuli in Chapter 3 will be provided and a brief summary of the behavioural assessments discussed in Chapter 6.

2.1 ERP Techniques

2.1.1 Generation and derivation of EEG and ERPs

The voltage variations observed across time when two electrodes are attached to the surface of the scalp and connected to a differential amplifier is the electroencephalogram or EEG. In order to isolate the brain's response to a specific cognitive function, voltage changes are time-locked to the presentation of a stimulus. These electrophysiological changes are called event-related potentials or

ERPs (Rugg and Coles, 1995), and can be extracted from the ongoing EEG by means of filtering and signal averaging.

The ERPs evoked represent net electrical fields associated with the electrical activity of a multitude of neuronal populations. For the voltage to be recorded at the scalp, the activity of these neurons must be synchronous and their individual electrical fields must summate to produce a dipolar field that can be detected at the scalp. It seems likely that these waveforms are a result of postsynaptic (dendritic) potentials as opposed to axonal action potentials (Allison et al, 1986). Surface electrodes cannot detect action potentials, discrete voltage spikes that travel along the axon from the cell body to the axon terminal, due to the timing of the action potential and the physical arrangement of axons. Postsynaptic potentials arise when neurotransmitters bind to the postsynaptic membrane causing ion channels to open or close, leading to a change in potential across the cell membrane which can last hundreds of milliseconds. Postsynaptic potentials are largely confined to the dendrites and cell bodies and under certain circumstances postsynaptic potentials summate making it possible to record them at the scalp. When an excitatory neurotransmitter is released at an apical dendrite of a cortical pyramidal cell, current flows into the cell, resulting in a net negativity on the outside of the cell. In turn, current will flow out of the cell body of the basal dendrites resulting in a net positivity in this region. A dipole is created by the opposite charges, and in certain circumstances the dipoles from many thousands or millions of neurons will summate allowing a resultant voltage to be measured at the scalp. The pyramidal neurons of the cerebral cortex follow the same orientation and are aligned perpendicular to the

scalp, helping with summation and making them conducive to the propagation of electrical potentials towards the scalp. However, other deeper set structures, such as the thalalmus, have an arrangement of neurones that does not produce synchronous activity sufficient to be detected at the scalp, and therefore activity in these areas of the brain goes unrecorded. However, there is a possibility the activity of these subcortical structures could be detected indirectly via their influence on cortical structures (Vuilleumier et al., 2004), For example the amygdala are reciprocally connected and send feedback projections to widespread cortical visual areas (Leppänen et al., 2009), discussed in more detail in Chapter 5.

The dipolar field produced by the summated neuronal fields generates a current which flows through the brain to the surface via volume conduction, spreading out laterally across the brain to avoid resistance. The voltage recorded at a given electrode site will depend on the position and orientation of the generator dipole and the resistance from the brain, skull and other components of the head (Luck, 2005). The spatial resolution of these scalp-recorded voltage potentials is not as precise as that possible in functional imaging techniques. The voltage recorded from a single electrode at the scalp may be due to numerous possible configurations of the neural generators, from both cortical and deeper subcortical brain structures. In contrast, the temporal resolution is very good, measureable within milliseconds post-stimulus onset, but typically dependent on sampling rate. ERPs make it possible to determine which stage or stages of processing are influenced by the specific experimental manipulations. They can also provide an online measure of stimulus processing even when there is no behavioural response, for example comparing the

processing of attended versus ignored stimuli. The main advantages of using ERPs as opposed to other measures of functional brain activation are that it provides a non-invasive, relatively easy to record technique, which is less sensitive to artefacts created by movement. All these factors are particularly beneficial for studying infant behaviour and the excellent temporal resolution means the technique is well suited to studying early perceptual processes such as the rapid processing of faces and facial expressions within 100ms.

The analysis of the electrophysiological data in this thesis will focus on specific ERP components relevant to the hypotheses. An ERP component can be described as a peak or trough in the curve of the EEG that relates to a specific brain process (Luck, 2005). However, there is an alternative theory to this, with evidence suggesting that some ERP components might be generated by stimulus-induced changes in ongoing brain dynamics (Penny et al, 2002). Makeig and colleagues (Makeig et al., 2002) demonstrated that the visual N1 ERP could have arisen from stimulus-induced 'partial phase resetting' of multiple ongoing EEG rhythms.

The description of a component will take into account both polarity (P or N for positive and negative deflections respectively) and order of appearance (1, 2, 3). For example the P1 wave is a positive deflection occurring near the beginning of the EEG trace. For specific components of significance, names are given indicating the typical post-stimulus latency of the peak, for example the N170 which has a negative deflection with a peak amplitude at 170ms post-stimulus. The following will provide a brief introduction of these components.

One well-studied component involved in adult face recognition is the N170 waveform. A greater negative potential, with a peak at approximately 170ms, is observed at the lateral occipital electrode sites for face stimuli compared with non-face stimuli, especially over the right hemisphere (Bentin et al., 1996). It is involved in the perceptual processing of the structural information from faces and its scalp distribution suggests that it is generated in the occipitotemporal/fusiform area (Itier & Taylor, 2002, 2004). Inverted faces delay the onset of the N170 and/or the amplitude is larger compared with upright faces (Eimer, 2000a).

The infant N290 component is a negative deflection, maximal over the midline and paramidline posterior cortex, gradually decreasing latency from approximately 350 to 290ms between three and twelve months (Halit et al., 2003). It is implicated as a possible precursor to the adult face-specific N170, and becomes more sensitive to upright human faces with age (de Haan et al., 2002). It peaks later, with a smaller peak amplitude, and has a more medial distribution than the adult N170.

The P1 wave is a sensory response to visual stimuli, present in both adult and infant populations. Like the N170, it is also largest at the lateral occipital electrode sites. It has an onset of 60-90ms and tends to peak positively between 100-130ms but its amplitude and latency is dependent on stimulus contrast. The origins of the P1 wave may be the dorsal extrastriate cortex and the fusiform gyrus, however there are many visual areas active during the first 100ms and many may contribute to the P1 wave (Di, Martinez, Sereno, Pitzalis, & Hillyard, 2002).

2.1.2 Recording ERPs

The software package Net Station 4.4.2 (Electrical Geodesics Inc., 2006) was used to acquire EEG and facilitate all ERP derivation and analysis.

2.1.2.1 Sensor Net

The scalp recordings taken in the following experiments were performed using a Geodesic Sensor Net (GSN). This sensor net comprises an array of 128 electrodes with a reference electrode and isolated common electrode, each enclosed in a sponge in a geodesic tension structure consisting of elastic threads joining each sensor to another in an isohedra array (Tucker, 1993). When the net is placed on the subject's head the sensors make electrical contact with the scalp. It works on the principle that the tension is distributed evenly across the head and balanced by compression directed from all points on the scalp towards the centre of the head. This geodesic tessellation of the head surface optimises the sampling of the electrical field. Each sensor contains a sintered Ag/AgCl carbon pellet connected by an insulated lead wire to the Hypertronics-compatible gold-plated pin. Each pellet is surrounded by a sponge that becomes wet with electrolyte during the operation of the net (Geodesic Sensor Net Technical Manual, 2007).

The traditional system for sensor placement has been the 10-20 system (the International Federation of Clinical Neurophysiology, Jasper, 1958) where sensors are placed at 10 percent and 20 percent points along lines of latitude and longitude. This convention however does not provide an even distribution of sensors across the two-dimensional surface of the head. An even distributed of sensors points is

needed for a complete sampling of the potential field across the head surface. The major advantages of the high-density array of the GSN are: that the greater spatial sampling making it possible to identify components that might have eluded capture with smaller arrays, where the interelectrode distances are greater; increased opportunity for source localisation; and the ability to use the average reference removing the need for a biased estimate voltage across the scalp based on the reference electrode. It has a high-operating impedance level, allowing high-input impedance and removing the need for scalp abrasion and conductance cream other systems use and critically takes a shorter amount of time to apply the net compared with other systems. Disadvantages include the fact that the electrodes are not fixed rigidly to the scalp and so movement artefacts are common and it can be difficult to place electrodes properly on unusual head shapes. The time required to reduce impedances at each electrode site is directly related to the number of electrodes present. Generic problems associated with all nets include electrode gel leakage causing an electrical bridge with an adjacent electrode, distorting the scalp distribution of the electrical potentials. Also in developmental studies, infants tend to reach for the net causing both movement artefacts and displacement of the electrodes.

2.1.2.2 The application of the Geodesic sensor net in adults

The net was applied in accordance with the instructions in the EGI manual (Electrical Geodesics Inc., 2007). In preparation, the subject was asked to wash their hair the

day before and refrain from using any products on the hair. The subject was asked to remove any items in their hair, earrings and glasses, and other objects that may obstruct the application of the net. If the subject had long hair they were requested to move their hair away from their face.

A circumference measurement of the subject's head was taken at the glabella (brow ridge) and occipital protuberance (ridge at the back of the skull). A net was selected based on this measurement. For adults the sizes were: small (53-55cm), medium (55-58cm) and large (58cm and above). For infants the sizes were: 7-16 weeks (38.5-41cm), 4-7 months (41.5-43cm), 7-10 months (43.5-45cm), 10-12 months (45-46.5cm), 1-2 years (47-48cm) and 2-3 years (48.5-50cm). The vertex was located and marked with a grease pencil as follows. The vertex is situated at the mid-point between the inion and the nasion and centred between the preauricular points. A measuring tape was used to find the mid-point for both measurements and the centre was marked on the subject's head with a grease pencil.

The sensor net was soaked in the electrolyte solution for one minute. This electrolyte solution consisted of one litre of distilled water (approximately 37°C), 8.5 grams (1.5 teaspoons) of granular/powdered potassium chloride (KCl), and 0.5cc. of Johnson's Baby Shampoo (1/10 teaspoon), mixed thoroughly. Caution was taken not to allow the Hypertronics connector to come into contact with the electrolyte. A towel was placed over the subject's shoulders to absorb excess electrolyte draining from the sensor net. The sensor net was removed from the electrolyte and the sensor sponges were touched against a towel a few times to remove excess

electrolyte. Care was taken not to press the sponges too firmly into the towel to prevent depletion of electrolyte.

The net was placed on the subject's head with the vertex sensor lying over the vertex mark on the head. The chinstrap was moved down under the subject's chin and secured using the cord lock. The orbital sensors were adjusted using the cord locks located on either side of the subject's cheeks until the infraorbital sensors fell directly below the subject's pupils (over the infraorbital foramen) and the extraorbital sensors fell just to the side of the outside corner of the eye (over the frontal process of the zygomatic bone). The sensors over the nasion and mastoids (which should be placed behind each ear) were checked for correct location, to aid placement and symmetry of the net. Each sensor was adjusted so it was sitting perpendicular to the scalp. The infant nets do not contain inferior orbital sensors but are made so that optional, separate outrider orbital sensors may be used. The orbital sensors plug into the special Geodesic net Hypertronics plug that is part of every infant net, and the net was then connected to the Hypertronics connector. The technique for applying the infant net was the same. However, in cases where the infant was particularly fussy the infant was moved in front of the screen ready for the experiment before final adjustments to the sensor net were made, so the experiment could commence as soon as possible, with the aim to reduce fussiness and obtain good quality data.

After every session the net was rinsed and disinfected. The sensors were immersed in warm water, with care taken not to get the net connector wet. The net was

agitated in the water for approximately thirty seconds; this rinsing process was repeated four times. The excess moisture was then removed and the net was transferred to the disinfectant solution. The disinfectant solution consisted of one tablespoon of Control III to 2 quarts of water (which could be reused up to ten days). The net is soaked in this solution for ten minutes and then removed and rinsed three times (as above). Excess moisture was then gently removed from the sponge tips with a clean towel and then hung up to dry, making sure that the connector did not get wet.

2.1.2.3 Recording the EEG

Before commencing the recording session, the impedance on each electrode was measured, and this was set at 50kΩ. The impedance is the opposition to the flow of current due to impediment and reducing this impedance increased the likelihood of a clean signal. High impedance can create both decreased common-mode rejection (discussed below) and increased skin potentials. The electrical potential between the surface and the deep layers of the skin changes in accordance with the skin impedance; for example sweat on a participant's skin increases the skin impedance, changing the voltage at the surface. These changes in voltage are called skin potentials and can be a source of low frequency noise in ERP recordings. Those electrodes with impedances exceeding 50kΩ were re-moistened with solution or

readjusted so the contact was more favourable, and hair was displaced from under the sponge if obstructing clean contact of the electrode to the scalp.

Throughout the recording session, the online EEG was monitored to ensure satisfactory recording from each electrode channel, and if necessary at break-points during the recording session, the electrodes were adjusted to reduce noise on individual channels. Each amplification channel has a different resting signal so to compensate for this during the amplification process the actual amplification factor ('Gains') and resting offset from zero ('Zeros') of each amplifier channel was measured and calibrated.

2.1.3 Amplification and signal filtering

Once the EEG is recorded from the electrodes it was amplified and then converted from a continuous, analog voltage to a discrete, digital form that the computer can store. In all studies the amplifier used was the EGI NetAmps amplifier with a bandpass filter of 0.1-100Hz. The signal that is recorded from the scalp has low amplitude changes so it needs amplification from a differential amplifier to increase the signal for analysis. The differential amplifier amplifies the difference between active-ground voltage and reference-ground voltage, subtracting away any electrical noise that is present in the ground and is vital for producing a clean EEG recording. This ability to subtract away environmental noise is called the common-mode rejection. The amplifier filters and measures the EEG signal from the net and samples

them at milliseconds intervals, converting the EEG into a voltage with discrete time points called samples. The sampling rate is the number of samples taken per second; in this case it was 250Hz (every 4 milliseconds). To determine this sampling rate the Nyquist theorem was used which states that the information in the analog signal can be converted to a digital signal if the sampling rate is at least twice as great as the highest frequency in the signal. At lower sampling rates information will be lost and additionally it will induce artifactual low frequencies in the digital data, called aliasing (Luck, 2005). There is a risk of aliasing when digitalising the raw EEG because the EEG may contain noise at high frequencies, so to counteract this, the amplifier contains low-pass filters that attenuate high frequencies and only pass low frequencies. So the sampling rate chosen will depend on the cut-off frequency chosen for the low-pass filters. To choose a suitable cut-off frequency for the filter it should be taken into account that filtering distorts ERP waveforms and the higher the frequency the less distortion. However, also note that a high filter frequency leads to a high digitisation rate which will produce huge data files. So a compromise of 30 and 100Hz low-pass cut-off frequency and a sampling rate of between 100 and 300Hz would be reasonable. In the experiments described in this thesis the filters were set at 0.01-100 and the sampling rate was 250Hz. The digitalised EEG samples are then transferred to the data-acquisition computer for collection and storage to disk (Geodesic Sensor Net Technical Manual, 2007).

2.1.4 ERP derivation and analysis

After data acquisition, steps were taken to derive the ERPs from the raw EEG as follows. Initially, the data was filtered, which optimised the signal by attenuating unwanted frequencies due to non-biological artefacts. Brain activity typically focuses on frequencies below 30-40Hz, so a digital 30Hz low pass filter was used to attenuate frequencies above 30Hz and remove electrical noise (of frequencies around 50Hz). The EEG was recorded as a continuous stream, and after filtering, the data was segmented into separate epochs for each stimulus (each emotional stimulus and target). Each epoch consisted of a pre-stimulus onset duration of 200ms and a post-stimulus onset duration of 1000ms, for all studies.

The next stage of ERP derivation was to identify and remove artefacts from the data, increasing signal-to-noise ratio. The term artefact refers to unwanted noise in the EEG signal, resulting from many sources such as external electrical equipment, muscle activity, or eye blinks. There are two major sources of electrical noise in the ERP lab that would contribute to artefacts, the AC line current and the video monitors. The AC line current comprises of sinusoidal oscillations at 50Hz which can induce oscillations at 50Hz in the EEG recordings. Video monitors operate at a refresh rate of between 50 and 120Hz and can result in spiky rather than sinusoidal noise; amplifier filters can attenuate this noise.

Eye blink and eye movement add a high-amplitude artefact due to changes in the voltage potential across the eyeball, affecting all channels but especially those located near the eyes. The eye blink artefact originates because there is an electrical gradient across each eye, with positive at the front and negative at the back. When the eyelid moves across the eye it propagates the conduction of electrical potentials of the eye to the surrounding areas. The eye blink response consists of a monophasic

deflection of 50-100μV with a typical duration of 200-400ms. When fixed on a point, the dipole produced across each eye creates a constant voltage gradient across the scalp which the high-pass filter of the amplifier would normally eliminate. However, when there is eye movement the voltage gradient across the scalp changes, becoming more positive at sites the eyes have moved towards. These eye movements also cause the visual input to shift across the retina creating a visual ERP response which depends on the nature of the stimuli visible when the eyes move. To minimise the amount of data eliminated due to artefacts, each participant was asked explicitly to remain as still as possible during the experiment, to relax their muscles, and minimise eye movement by fixating on the visual stimuli**.** In the case of the infants, the caregiver was instructed to remain as still as possible to avoid the transmission of movement artefact through the infant.

Additional noise was apparent in select channels due to poor scalp contact due to electrode placing or the evaporation of electrolyte from the sponge. These artefacts are typically very large compared to the ERP signals and may greatly decrease the signal-to-noise ratio of the ERP waveform. For this reason all channels were automatically scanned for amplitude differences of the order ±50μV for adults and differences of ±100μV for infants, performing a moving average of 80ms, and any channels with amplitude differences outside these values were marked as bad. A whole epoch was excluded if there were more than 10% of bad channels in the epoch (more than 12 channels out of 128). If a specific channel was bad for more than 20% of the epochs, this channel was replaced for the whole recording. Participants with less than 12 useable epochs for each of the stimulus types were excluded from further analysis (details are noted in the relevant chapters). Artefact

detection was performed for all 128 channels, not just those of interest. Although this increased the likelihood more epochs would be rejected; it improved the signal-to-noise ratio when re-referencing to the average reference, as this is calculated by subtracting the mean of all the electrodes from each channel. After the automatic artefact detection was complete, each epoch was manually checked for noise in the EEG signal that had gone undetected in the automatic detection, including ocular artefacts. Epochs where eye blinks or eye movements were identified, or where any other artefacts were observed that had not been detected automatically, were considered noisy data and excluded from further analysis. In addition, in Chapter 7, the video recordings of the infant participants were viewed alongside the EEG recordings. Trials were discarded when the infant did not attend to the visual stimulus from stimulus onset and for one second afterwards. An artefact detection tool used a moving-average algorithm based on specified thresholds was implemented to replace the signal from noisy channels with those from neighbouring channels by interpolation. Electrodes in close proximity to one another on the scalp share similar voltage values because of electrical volume conduction. The voltage at a specified location can be interpolated from voltage values at proximal locations using spherical splines as an interpolation method, providing they are evenly distributed across the scalp.

The remaining epochs were baseline-corrected 200ms pre-stimulus onset, which established a new zero-voltage value within each epoch. For each channel, the average of all samples within the baseline interval was subtracted from every sample in the segment establishing a new zero-voltage value. This increased the signal-to-noise ratio by reducing the noise fluctuations in the average waveform.

The voltage changes comprising the ERP in question are very small in comparison to the EEG waveform in which they are embedded. The most frequently used approach to produce a greater signal-to-noise ratio is to repeat the evoking stimulus multiple times. The voltage changes associated with the stimulus will thus be enhanced and those extraneous independent changes will be averaged out. The number of useable trials per condition for each experiment is noted in the relevant chapters. Signal processing techniques must be used to extract this ERP signal from the background EEG, and average ERPs were created by averaging across individual trials with useable data. By recording a multitude of EEG epochs, all containing ERPs time-locked to the same event, the background EEG will vary across epochs and tend to average to zero and the resulting waveform will represent the activity that is time-locked to the event. As more and more trials are averaged together the noise remaining becomes smaller and smaller, with the size of the noise in an average of N trials being equal to $(1/\sqrt{N}) \times R$ where R is the amount of noise, thus the signal-to-noise ratio will increase as a function of the square root of the number of trials.

The average reference is calculated by subtracting the mean of all the electrodes from each channel. The data are referenced online to one of the electrodes on the scalp (the vertex) and then later re-referenced to the average. This is thought to be a good technique because it is then unbiased to any particular electrode site and is a good approximation of the true voltages across the head which must average to zero. An average reference can be used when the inter-electrode distance is less than 2-3cm. At this point the eye channels (channels 126 & 127) were excluded from further analysis.

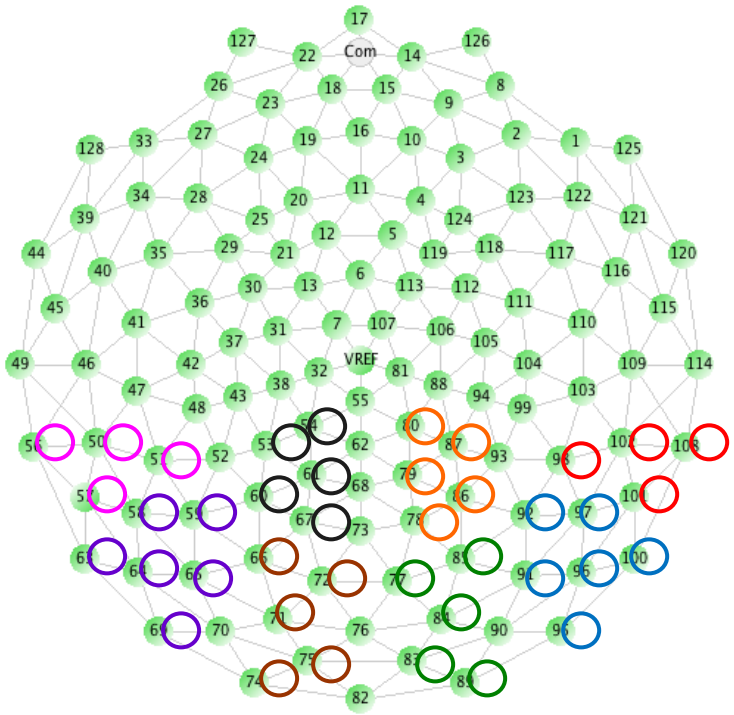
Most studies using ERP analysis tend to look at group effects, those differences observed by a group of subjects as opposed to an individual. In this thesis, a grand-average waveform was obtained by averaging all the individual-participant waveforms for a particular condition, emphasizing patterns common across subjects and reducing individual differences. This grand-average waveform provides a good indication of the average voltage at a given time-point across participants. When selecting a time-window for a specific ERP component, after estimating a time-window from the grand-average waveform, individual peak voltages were examined to ensure these lay within the selected time-window. It is these individual peak values (an average across trials for each condition for each participant), and not grand-average values, that were used in subsequent statistical analysis.

2.1.5 ERP Peak Analysis

Data was extracted for statistical analysis, to determine whether specific components were statistically different between experimental conditions. The first step was to determine a time-window within which the ERP component of choice lay. This was achieved by examining the grand-average and average waveforms for individual participants to identify the time-frame in which the peak amplitude for a specific component was present. The component was identified at particular channels consistent with the topography of the specific component generator activity, and effects observed in previously published data regarding distribution and timing. The time-window was of sufficient duration to capture the target component but not so broad as to include peaks of other components which may give false peak

amplitude and latency values. The time-windows for the specific components were kept constant for all participants in all epochs across all experimental conditions. The time-windows for the components of interest, the P1, N170 & N290, are defined in the relevant chapters (Chapters 4, 5, and 7).

Channel groupings or montages were then defined based on visual inspection of the grand-average waveform and previously reported information on the distribution of the specific component. Channel groupings were defined for eight topographical regions, split across the right and left hemispheres, and lateral-medial and dorsal-ventral topography as shown in Figure 2.1 below. The selection of these topographical regions allowed for exploration of the main hypotheses, and resulting electrophysiological patterns could be compared with previous research findings. These groupings were used for all three components, P1, N170, and the N290 across all populations. Preservation of the same channel groupings across the different studies allowed a direct comparison of the electrophysiological response between populations.



Right Lateral Dorsal, Right Lateral Ventral, Right Medial Dorsal, Right Medial Ventral,
Left Lateral Dorsal, Left Lateral Ventral, Left Medial Dorsal, Left Medial Ventral

Figure 2.1 The locations of the eight topographical regions on the 128-electrode Geodesic net; the front of the head is at the top of the figure.

The peak amplitude (the most extreme point in microvolts), and the peak latency (the latency in milliseconds at the most extreme point) were the measures extracted for each component within a specified time-window for each electrode in a pre-defined channel grouping. The peak amplitude was measured using the adaptive mean, which was calculated using an algorithm that identified a peak in the waveform within the time-window and then defined a new time-window around this peak. The mean voltage value was then calculated from the new time-window and

the measure was reported as the average of the channels in the channel grouping selected.

2.2 Behavioural assessments

In Chapter 3, recognition of facial expressions of emotion was assessed by presenting static and dynamic stimuli of the six basic emotions to adult participants. The basic details of the stimuli will be described below, including the creation of the morphed dynamic stimuli. A description of participants and procedure can be found in Chapter 3, section 3.2

Static Stimuli: Each static stimulus consisted of a colour photograph of an individual displaying one of the six basic emotions, anger, disgust, fear (with open mouth or closed mouth), happiness, sadness, and surprise; displaying the head and hair of the individual only, with the neck and clothes masked by a grey sheet, the face being central in the photograph. The background of each photograph was white and the mean luminance was kept constant throughout. These photographs were a subset of the NimStim Set of Facial Expressions (Tottenham et al., 2009). The NimStim Set contains 646 colour photographs of forty-three professional actors posing different facial expressions including the seven expressions above, in full frontal orientation. The actors of different ethnicities were asked to pose the facial expression and then facial muscles were adjusted until the desired affect was achieved. This stimulus set has been validated by 81 healthy adults using a labelling task (including a label 'none of the above').

Four female and four male models were chosen from the NimStim Set to be used in this study. The models were selected on the basis that the expressions they posed were recognised with a higher accuracy by the seventy healthy adults than those posed by the other models, consistently across all expressions. The ethnic origins of the eight models were European-American (1 female, 2 male); Latino-American (1 female); and African-American (2 female, 2 male). Table 2.1 below shows the percentage accuracy for each model, 1-4 are female models and 5-8 are male models from Tottenham et al, 2009.

Dynamic stimuli: Consistent with previous research (LaBar et al., 2003; Mayes et al, 2009), the dynamic stimuli were created using morphing software (Morpheus v1.85 Pro). Each dynamic morph was produced by morphing two static images from the same model. For example, dynamic fear was produced by morphing the static neutral image into the static fear image for a particular model (100% neutral to 100% fear). Artefacts were removed from the static stimuli which would have affected the smooth morphing of the images (Adobe Photoshop 6.0). Changes were not made to any elements of the photo which might have altered any aspect of the facial expression represented. Over 300 markers were applied and matched across images to identify corresponding spatial locations on the face. Markers were placed on facial areas considered important for conveying changes in facial expression, such as the inner and outer canthi of the eyes, the orbicularis oculi muscles, corrugators supercilii, and upper and lower lips (Bassili, 1979; Ekman & Friesen, 1978), as can be seen in Fig 2.2 on the next page. Care was taken to apply these markers in exactly the same positions in both photographs (neutral and extreme emotion) to maximise

the smoothness of the transition between expressions. The dynamic stimuli were presented with a frame rate of 60 frames/45ms for 750ms.

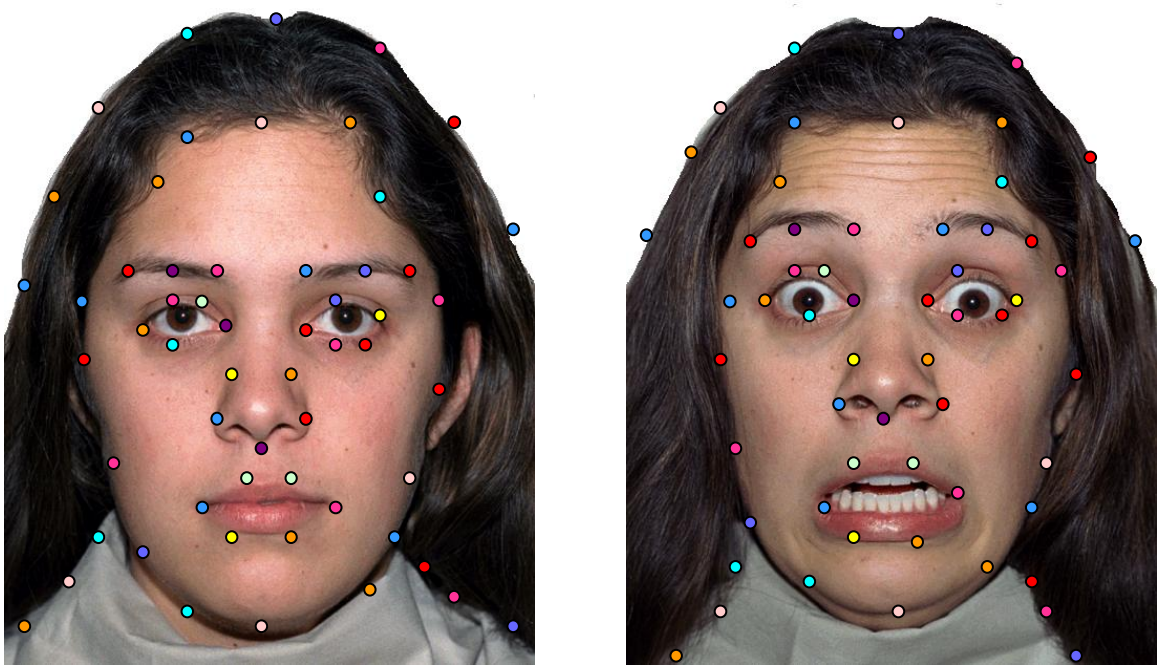


Figure 2.2 Creating the dynamic stimuli. An illustration of the facial regions highlighted to create a morph (100% neutral to 100% fear)

It was important for the future ERP experiments that the expressions were not only realistic but also that there was a level of control over the spatial and temporal parameters of the facial expressions. Morphed dynamic images were used with this in mind as opposed to video clips of actors making the expressions in 'real-time' (Sato & Yoshikawa, 2004; Trautmann et al., 2009). The NimStim Set has been used in previous research investigating facial expression recognition (Adolph & Alpers, 2010), and the underlying neural systems involved in emotional face processing (Leppänen et al., 2007); and recognition accuracy has been compared between static and dynamic counterparts (Johnston et al, 2008).

In Chapter 6, the following battery of tests was conducted on patients with temporal lobectomy (right- and left-sided), and control participants: The Matrix Reasoning subtest from the WAIS-III; the Florida Affect Battery (FAB); the Empathy Quotient (EQ); and the Social Functioning Scale (SFS). The FAB was used to assess recognition of facial expressions and emotional prosody; the EQ was used to assess more complex social awareness, specifically empathy; and the SFS was used to assess the impact of temporal lobectomy on social functioning. The EQ and SFS were self-reported and the FAB was administered by the examiner. The battery was conducted on all participants directly after the ERP experiment. These will be described in more detail in Chapter 6.

2.3 General statistical techniques

Parametric statistical analyses were performed on all ERP data throughout the thesis (Chapters 4, 5, and 7). This allowed the exploration of interactions between variables in a factorial design that would not have been possible with non-parametric alternatives, and follows the majority of ERP research using ANOVA methods. Parametric tests also have the advantage of greater power than non-parametric tests; increasing confidence when failing to reject the null hypothesis. Parametric tests can, however, only be performed when the data assumes normal distribution. With this in mind, the data were initially examined for skewness and kurtosis, and outliers were identified. Alongside values for skewness and kurtosis, the normality of distribution was assessed by the Kolmogorov-Smirnov statistic, and supported by

histograms and probability plots. There are many ways of dealing with outliers in the data, such as removing the value from the data set, transforming the value, or replacing the point with a specified value. In this case, outliers were identified as those data points that lay 2 standard deviations outside the variable mean (identification supported by boxplots); and replaced with variable mean, so the impact of the outlier was reduced. The treatment of the outliers in this way allowed the data from a particular participant to be included in the analysis, without the data from that individual disproportionately influencing the statistics. Running the analyses prior to removal of the outliers confirmed that this procedure did not affect the results obtained. The distribution of the data and the percentage of outliers identified for each analysis are noted in the relevant chapters. The data were re-examined after elimination of outliers to confirm that the distribution was within normal ranges.

Repeated measures ANOVA was performed for all ERP analyses, and significant interactions were followed up using analysis of simple main effects. Greenhouse-Geisser was applied as the adjustment for sphericity. T-tests with Bonferonni correction were performed in post-hoc analysis to compare differences between groups. Post-hoc comparisons had the benefit of guarding against the possibility of an increased Type I error that would occur with a large number of exploratory comparisons, by setting more stringent criteria for significance. Pearson's correlations were used in Chapter 6 and regression analysis was conducted in Chapter 7 to examine relations between continuous variables. Planned comparisons were also performed to investigate specific a priori hypotheses. All statistical tests conducted were two-tailed and a 5% significance level was adopted throughout.

3. The influence of facial motion on emotion recognition

3.1 Introduction

In the natural environment faces are moving, multimodal stimuli. However, it is only recently that dynamic stimuli have been used to explore face processing (O'Toole et al., 2002) emphasising that faces are very much dynamic objects, with movement varying along both spatial and temporal dimensions. It is clear that judgments about posed facial expressions can be made from static images (Ekman & Friesen, 1982) however it is possible to say that the additional temporal information provided by the moving face could influence emotion recognition. Under the conditions of a static picture of an emotional expression, the emotion is usually presented at or very near the peak of emotional display. The perceiver will not then be capable of determining whether, for example, the elicitor's fear is emerging (indicating an impending threat) or dissipating (indicating the removal of threat). In social situations, facial expressions of emotion are highly dynamic, with subtle changes in facial musculature depicting complex social signals.

Studies directly comparing recognition of static and dynamic facial emotions have often shown that the presentation of dynamic facial expressions improves recognition of emotion (Ambadar et al., 2005; Bassili, 1979; Pike, Kemp, Towell, & Phillips, 1997; Wehrle et al., 2000). Dynamic stimuli have also been shown to enhance recognition of facial expressions in atypical populations with deficits in facial emotion recognition (Harwood et al., 1999; Humphreys et al., 1993). Weyers and colleagues (2006) found that dynamic avatars led to better recognition rates in

patients with schizophrenia. Ambadar and colleagues (2005) showed that subtle dynamic expressions (ending at the first display of the emotion) were recognised more accurately and with more confidence than either static (apex of the emotion) or multi-static (from start to apex of the emotion) for all basic emotions. This implies that the motion advantage is not due exclusively to extra static information. Pike and colleagues (1997) found consistent results to Ambadar et al (2005), but interestingly, participants took longer to make a correct decision about dynamic stimuli compared to both multi-static and single static stimuli. They demonstrated that this was not the result of a speed/accuracy trade-off, as participants took just as long to produce an incorrect response as a correct one, suggesting that the higher accuracy rates were not simply due to participants taking longer to make an emotion decision. Kamachi and colleagues (2001) found that the speed of the dynamic presentation influenced recognition, with happiness and surprise being recognised more accurately from faster sequences, anger from medium-speed sequences, and sadness from slower sequences. This was reflected in the intensity ratings; with shorter displays of happiness and surprise, and longer displays of sadness being rated as more intense.

However, not all studies have reported an overall dynamic advantage. For example, Kamachi and colleagues (2001) did not find this dynamic advantage in recognition, instead finding accuracy overall was higher for static expressions. Johnston and colleagues (2008) found that dynamic images did not improve recognition accuracy in patients with schizophrenia. Motion seems to influence perception of certain emotions more than others. Harwood and colleagues (1999) found that motion improved the recognition of sadness and anger only and not disgust, fear and

surprise. Fujimura and Suzuki (2010) found that the benefits of motion depended on the emotion displayed, with happy, fearful and excited expressions showing a benefit but not calm, sleepy, sad, angry or surprised expressions. Bould and Morris (2008) also found that the influence of motion depended on the nature of the expression but they reported that the benefits of motion were greater for subtle expressions and reduced for more intense expressions. In contrast, Wehrle and colleagues (2000) did not find an effect of intensity on the recognition of either static or dynamic stimuli. Thus, while dynamic facial expressions are often recognised better and rated as more intense than static versions, there is evidence that these effects may be reduced for more intense displays of emotion.

Most studies contrasting static and dynamic images of facial expressions have used morphed dynamic images, only a few studies have used natural moving video images (Gepner et al., 2001; Kilts et al., 2003; Trautmann et al., 2009). Although these video stimuli may prove more naturalistic it is difficult to control for differences in the level of expression portrayed and the temporal aspects of the expression which are more controlled with morphed expression. This is particularly important when recording early neural responses to emotion perception using the ERP methods discussed in this thesis.

There is no consistent stimulus set used throughout facial emotion research, with disparity between various properties of the different stimulus sets such as: black and white versus colour photographs; variety of emotions expressed; gender, age and ethnicity of the actors displaying the emotions; technique for creating each expression; and intensity of expression. One of the aims of the study was to produce morphed dynamic stimuli from an existing static stimulus set that depicted all six

basic emotions (anger, disgust, fear, happiness, sadness, and surprise) and was a realistic representation of the emotions, i.e. colour photographs, with high accuracy ratings for both male and female actors from different ethnic backgrounds. The dynamic stimuli, alongside their static counterparts, were then validated by obtaining ratings for recognition accuracy, confidence in identification, and intensity of each stimulus, as described in this chapter.

The specific questions and hypotheses to be addressed are as follows:

1) The dynamic emotional stimuli will be recognised more accurately and with more confidence than static stimuli: This is based on a) findings that have shown that dynamic stimuli are recognised with more accuracy than their static counterparts (Ambadar et al, 2005; Bassili, 1978; Pike et al, 1997; Wehrle, et al, 2000); and b) correctly identified dynamic stimuli are recognised with more confidence than static stimuli (Ambadar et al, 2005; Pike et al, 1997).
2) Motion will particularly improve the recognition of those emotions difficult to recognise in the static form: This is based on a) studies that have shown that fear is the most difficult emotion to identify in the static form (Adolphs & Alpers, 2010; Montagne, Kessels, de Haan, & Perrett, 2007); b) fear has been recognised with greater accuracy for dynamic emotions (Wehrle et al, 2000); c) fear and surprise are often confused for one another, as they share similar features (Adolphs, 2002; Eisenbarth, Alpers, Segre, Calogero, & Angrilli, 2008); and d) happiness is the most easily recognised emotion, with observable ceiling effects for static stimuli, such that motion will not

significantly improve recognition (Adolph & Alpers, 2010; Kamachi et al., 2001).

3) Dynamic stimuli will be considered more intense than their static counterparts: This is based on studies which have shown that dynamic facial expressions are rated as more intense than static stimuli (Biele & Grabowski, 2006).
4) The motion advantage will be increased for less intense emotions: This is based on studies which have shown that motion is more beneficial in improving emotion recognition for less intense/more subtle expressions (Bould & Morris, 2008).

3.2 Methods

3.2.1 Participants

Thirty-six volunteers (18 female), aged 21-59 years (mean age 30.3 years, S.D. 10.19), self-reported as right-handed, participated in the experiment. All participants had normal or corrected-to-normal visual acuity, with no recorded medical problems associated with vision. All self-reported as having no extraneous neurological or psychological disorder. The study was approved by ICH/GOSH Research Ethics Committee (REC reference 07/Q0508/35), and performed according to the standards of the Declaration of Helsinki (1964). Each individual provided informed written consent prior to participation. Individuals were not paid for taking part in the study and the data from all thirty-six individuals were used in further analyses.

3.2.2 Materials

Static photographs and counterpart morphed dynamic stimuli of the six basic emotional expressions (anger, disgust, fear (both closed and open mouth), happiness, sadness, and surprise), displayed by four male and four female actors, were presented to participants. The general description of both stimuli, including the creation of the dynamic stimuli is discussed in Chapter 2, section 2.2.

3.2.3 Experimental Task

Each participant sat facing a computer screen on which the stimuli appeared in a pseudo-randomised order, with the constraint that no more than two identical emotions were presented consecutively. The visual angle subtended was 18.42° in height and 14.94° in width. The images appeared on the screen for 750ms and then the screen went blank. A total of 110 stimuli were presented; eight stimuli per emotion for each motion condition (except for fear with closed mouth that was not available from the original stimulus set for one female model). All stimuli were presented once only. The participants were instructed to passively view each stimulus in turn and, after each stimulus, they were asked to 1) identify the facial expression from the following options: 'anger', 'disgust', 'fear', 'happiness', 'sadness', 'surprise', and 'other'. If they selected 'other' they were then asked to specify the emotion they believed the expression represented; 2) rate how confident they were in their choice (on a Likert-type scale, where 1 = not confident at all and 5

= very confident); 3) rate the intensity of the facial expression (on a Likert-type scale, where 1 = not at all intense and 5 = very intense). Participants could take as long as they needed to make the three ratings for each stimulus and were then instructed to press the spacebar to move onto the next stimulus.

3.2.4 Statistical Analyses

The mean percentage responses for each facial expression type, including errors; the mean confidence ratings for correctly judged emotions; and the mean ratings of intensity were reported for each of the seven emotions overall and for the two motion conditions separately.

Reliability of ratings of static stimuli was examined using binomial distributions. The recognition, confidence and intensity scores were examined using separate 2x7x2 analysis of variance (ANOVA) with within-subjects factors of Motion (static, dynamic) and Emotion (anger, disgust, fear with closed mouth, fear with open mouth, happiness, sadness, surprise). Interactions were followed up using analysis of simple main effects and Greenhouse-Geisser was used as the adjustment for sphericity. A 5% significance level was adopted throughout, and this was adjusted for multiple comparisons using Bonferroni correction.

3.3 Results

3.3.1 Reliability of Ratings for Static Stimuli

In order to examine the reliability of the ratings obtained in this thesis with those obtained by Tottenham and colleagues (2009), binomial distributions were computed for each stimulus. The two sets of percentage correct responses are reported alongside the p values for each stimulus are given in Table 3.1 below. As can be seen there is generally a good consistency between the scores, with significant differences for four models for disgust, three models for fear with closed mouth, and two models for fear with open mouth, happiness and sadness.

There were significantly higher ratings for the static stimuli in this study than those in the original study for Disgust 02 ($p = 0.02$; difference = 12.11%); Disgust 03 ($p = 0.01$; difference of 16.37%; and Disgust 08 ($p = 0.02$, difference of 12.11%); Fear with open mouth 03 ($p = 0.04$, difference of 14.60%); and Fear with open mouth 06 ($p = 0.03$, difference of 16.90%). Happiness 03 ($p = 0.02$; difference of 10.64%); and Happiness 08 ($p = 0.03$; difference of 8.70%). There was a significantly higher ratings in the original study than those in this study for Disgust 05 ($p = 0.04$; difference of 11.58%); Fear with closed mouth 05 ($p < 0.0005$; difference of 38.36%); Fear with closed mouth 07 ($p = 0.01$; difference of 21.04%); Fear with closed mouth 08 ($p = 0.01$; difference of 18.44%). Sadness 01 ($p = 0.04$; difference of 6.20%); and Sadness 08 ($p < 0.0005$; difference of 24.63%).

Table 3.1 Binomial distributions comparing the mean percentage correct responses for the original study (Tottenham et al, 2009) and the current study for each stimulus

Stimulus	Original Mean Percentage Correct	Current Mean Percentage Correct	Binomial p value
Anger 01	87.23	80.56	0.18
Anger 02	100.00	97.22	0.30
Anger 03	87.23	86.11	0.51
Anger 04	93.62	94.44	0.63
Anger 05	95.74	91.67	0.17
Anger 06	95.74	100.00	0.23
Anger 07	87.23	80.56	0.18
Anger 08	76.60	69.44	0.19
Disgust 01	91.49	97.22	0.15
Disgust 02	85.11	97.22	0.02*
Disgust 03	80.85	97.22	0.01*
Disgust 04	76.60	83.33	0.25
Disgust 05	89.36	77.78	0.04*
Disgust 06	93.62	100.00	0.11
Disgust 07	85.11	94.44	0.08
Disgust 08	85.11	97.22	0.02*
Fear Closed 01	-	-	-
Fear Closed 02	50.00	55.56	0.62
Fear Closed 03	57.45	44.44	0.09
Fear Closed 04	72.34	61.11	0.10
Fear Closed 05	74.47	36.11	0.00*
Fear Closed 06	19.15	25.00	0.23
Fear Closed 07	76.60	55.56	0.01*
Fear Closed 08	85.11	66.67	0.01*
Fear Open 01	69.57	83.33	0.05
Fear Open 02	78.26	77.78	0.55
Fear Open 03	65.96	80.56	0.04*
Fear Open 04	87.23	91.67	0.29
Fear Open 05	65.96	58.33	0.18
Fear Open 06	55.32	72.22	0.03*
Fear Open 07	91.49	94.44	0.44
Fear Open 08	91.49	83.33	0.06
Happiness 01	93.62	100.00	0.11
Happiness 02	93.62	100.00	0.11
Happiness 03	89.36	100.00	0.02*
Happiness 04	95.74	100.00	0.23
Happiness 05	100.00	100.00	0.70
Happiness 06	93.62	100.00	0.11
Happiness 07	95.65	100.00	0.23
Happiness 08	91.30	100.00	0.03*
Sadness 01	97.87	91.67	0.04*

Sadness 02	82.22	86.11	0.35
Sadness 03	100.00	97.22	0.30
Sadness 04	73.91	75.00	0.53
Sadness 05	78.72	80.56	0.51
Sadness 06	95.74	97.22	0.58
Sadness 07	93.62	100.00	0.11
Sadness 08	91.30	66.67	0.00*
Surprise 01	76.60	72.22	0.31
Surprise 02	72.34	83.33	0.09
Surprise 03	96.74	100.00	0.33
Surprise 04	89.13	88.89	0.57
Surprise 05	89.36	86.11	0.36
Surprise 06	95.74	97.22	0.58
Surprise 07	71.74	77.78	0.29
Surprise 08	89.36	91.67	0.43

*$p < 0.05$

3.3.2 Effects of motion on the accuracy of emotion recognition

Figure 3.1 shows the percentages for correct recognition and error types for each emotion in static and dynamic form, and Tables 3.2 & 3.3 show the percentage correct for each emotion overall and for motion conditions separately.

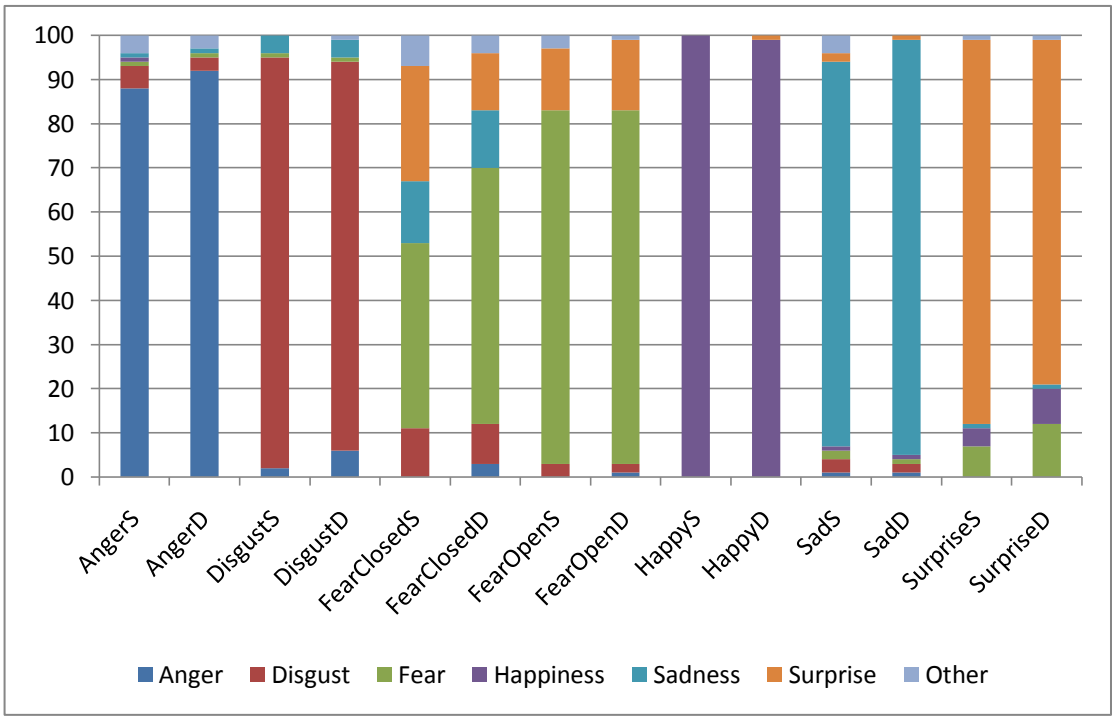


Figure 3.1 Percentage of responses for each of the seven facial expressions, including the error types

Anger was recognised with 87.85% & 92.01% accuracy for static & dynamic conditions respectively. It was incorrectly labelled as disgust, fear, sadness, and 'other' for both motion conditions, and happiness in the static form only. The emotions chosen in the 'other' category for static anger included suspicious, distrusting, neutral, confused, and perplexed; and for dynamic anger included puzzled, neutral and perplexed

Disgust was recognised with 93.06% & 87.85% accuracy for both static & dynamic conditions respectively. It was incorrectly labelled as anger, fear, and sadness, and 'other' in the dynamic form only (quizzical).

Fear with closed mouth was poorly recognised, with 41.67% & 57.94% accuracy for static & dynamic conditions respectively. It was incorrectly labelled as disgust, sadness, surprise, and 'other' for both motion conditions, and anger in the dynamic form only. The emotions chosen in the 'other' category for static fear with closed mouth included disbelief, shock, confused, apprehensive, neutral, careless, and threatening; and for dynamic fear with closed mouth included doubtful, confused, helpless, neutral, and threatening.

Fear with open mouth was recognised with 80.21% & 79.51% accuracy for static and dynamic conditions respectively. It was incorrectly labelled as disgust, surprise, and 'other' for both motion conditions, and anger in the dynamic form only. The emotion chosen in the 'other' category for static fear with open mouth was embarrassed; and for dynamic fear with open mouth was shock.

Happiness was recognised very well, with 100.00% and 98.96% accuracy for static & dynamic conditions respectively. The dynamic form was incorrectly labelled as surprise.

Sadness was recognised well, with 86.81% & 94.44% for static & dynamic conditions respectively. Interestingly, although it was correctly identified in the majority of cases, those that incorrectly identified sadness chose from the full range of other emotion options. It was incorrectly labelled as anger, disgust, fear, happiness, and surprise for both motion conditions, and 'other' in the static form only. The emotions

chosen in the 'other' category for static sadness were distrust, neutral, passive, unsure, confused, doubtful, careless, quizzical, indifference, and puzzled.

Surprise was recognised with 87.15% & 78.47% for static & dynamic conditions respectively. It was incorrectly labelled as fear, happiness, sadness, and 'other' for both motion conditions. The emotion chosen in the 'other' category for both static and dynamic surprise was shock.

The analysis of recognition accuracy revealed a significant main effect of Emotion, $F(6, 204) = 53.41$, $p < 0.0005$, demonstrating that some emotions were more accurately recognised than others. Generally, happiness was recognised best and fear was most difficult to recognise (mean percentage correct values shown in Table 3.2 below). Further analysis showed that all emotions were recognised more accurately than fear with closed mouth, $p < 0.0005$. Disgust $p < 0.001$, sadness $p < 0.0005$ and happiness $p < 0.0005$, were recognised more accurately than fear with open mouth. Happiness was also recognised more accurately than anger $p < 0.05$, fear with open mouth, $p < 0.0005$, and surprise, $p < 0.005$. Table 3.2 illustrates the percentage correct score and S.D. for each emotion.

Table 3.2 Mean percentage accuracy and S.D for the seven facial expressions

Emotion	Mean Percentage Correct	(S.D.)
Anger	89.93	(12.44)
Disgust	90.45	(9.62)
Fear with closed mouth	49.81	(21.56)
Fear with open mouth	79.86	(19.31)
Happiness	99.48	(2.30)
Sadness	90.63	(10.30)
Surprise	82.81	(13.97)

There was a trend towards a significant main effect of Motion, $F(1, 34)= 3.93$, $p = 0.056$ because static images were recognised better than dynamic images overall. However this main effect was modified by a significant interaction between Emotion and Motion, $F(6, 204) = 12.20$, $p < 0.0005$. This occurred because disgust and surprise were better recognised in static than dynamic images ($p < 0.05$ and $p < 0.001$ respectively), while fear with closed mouth, and sadness were better recognised in dynamic than static images (both $p < 0.0005$). Motion did not influence recognition of anger, fear with open mouth, or happiness. Table 3.3 lists the percentage correct score and S.D. for static and dynamic images for each emotion.

Table 3.3 Mean percentage accuracy and S.D. for the seven facial expressions for both motion conditions

Emotion for motion conditions	Mean Percentage Correct	(S.D.)
Anger Static	87.85	(14.17)
Anger Dynamic	92.01	(12.73)
Disgust Static	93.06	(10.96)
Disgust Dynamic	87.85	(13.19)
Fear Closed Static	41.67	(24.72)
Fear Closed Dynamic	57.94	(24.61)
Fear Open Static	80.21	(20.13)
Fear Open Dynamic	79.51	(21.16)
Happiness Static	100.00	(0.00)
Happiness Dynamic	98.96	(4.61)
Sadness Static	86.81	(14.00)
Sadness Dynamic	94.44	(8.15)
Surprise Static	87.15	(13.53)
Surprise Dynamic	78.47	(17.58)

3.3.3 Effects of motion on the confidence of recognising emotions

This analysis showed a significant main effect of Emotion, $F(6, 204) = 59.25$, $p < 0.0005$, which occurred because happiness was given a higher confidence rating than all the other emotions (all $p < 0.0005$), and fear with closed mouth was given a lower confidence rating than all the other emotions (all $p < 0.0005$). Table 3.4 illustrates the mean confidence rating (values out of five) and S.D. for each emotion.

Table 3.4 Mean confidence ratings and S.D. for the seven facial expressions

Emotion	Mean Confidence Rating	(S.D.)
Anger	3.73	(0.63)
Disgust	3.76	(0.71)
Fear with closed mouth	3.01	(0.62)
Fear with open mouth	3.74	(0.60)
Happiness	4.57	(0.53)
Sadness	3.74	(0.58)
Surprise	3.86	(0.68)

This analysis also showed a significant main effect of Motion, $F(1, 34) = 22.11$, $p < 0.0005$, which occurred because dynamic images were given higher confidence ratings than static images. There was a significant interaction between Emotion and Motion, $F(1, 34) = 7.32$, $p < 0.0005$. This occurred because dynamic images were given higher confidence ratings than static images for anger, $t(1, 35) = 6.64$, $p < 0.0005$, fear with closed mouth, $t(1, 35) = 3.27$, $p < 0.005$, fear with open mouth, $t(1, 35) = 3.19$, $p < 0.005$, and sadness, $t(1, 35) = 3.60$, $p < 0.001$, whereas motion did not

influence confidence ratings for disgust, happiness or surprise. Table 3.5 lists the mean confidence values (out of five) and S.D. for static and dynamic images for each emotion.

Table 3.5 Mean confidence ratings and S.D. for the seven facial expressions for both motion conditions

Emotion for motion conditions	Mean Confidence Rating	(S.D.)
Anger Static	3.57	(0.63)
Anger Dynamic	3.89	(0.67)
Disgust Static	3.79	(0.76)
Disgust Dynamic	3.73	(0.71)
Fear Closed Static	2.91	(0.66)
Fear Closed Dynamic	3.12	(0.65)
Fear Open Static	3.64	(0.65)
Fear Open Dynamic	3.84	(0.62)
Happiness Static	4.53	(0.55)
Happiness Dynamic	4.60	(0.52)
Sadness Static	3.62	(0.63)
Sadness Dynamic	3.86	(0.60)
Surprise Static	3.90	(0.69)
Surprise Dynamic	3.83	(0.71)

3.3.4 Effects of motion on perceived intensity of emotions

This analysis showed a significant main effect of Emotion, $F(6, 204) = 23.71$, $p < 0.0005$, demonstrating that different emotions were rated as more or less intense than others. Further analysis showed that fear with closed mouth was considered less intense than anger ($p < 0.0005$), disgust ($p < 0.0005$), fear with open mouth ($p < 0.0005$), and surprise ($p < 0.005$). Sadness was considered less intense than all other

emotions except fear with closed mouth (all $p < 0.0005$). Disgust and fear with open mouth were considered more intense than surprise, ($p < 0.01$ and $p < 0.005$ respectively). Table 3.6 illustrates the mean intensity rating (values out of five) and S.D. for each emotion.

Table 3.6 Mean intensity ratings and S.D. for the seven facial expressions

Emotion	Mean Intensity Rating	(S.D.)
Anger	3.68	(0.48)
Disgust	3.93	(0.50)
Fear with closed mouth	3.32	(0.47)
Fear with open mouth	3.92	(0.50)
Happiness	3.65	(0.69)
Sadness	3.19	(0.47)
Surprise	3.66	(0.55)

This analysis also showed a significant main effect of Motion, $F(1, 34) = 23.71$, $p < 0.0005$, as dynamic images were rated as more intense than static images. There was no interaction between Emotion and Motion. Table 3.7 lists the mean intensity values (out of five) and S.D. for static and dynamic images for each emotion.

Table 3.7 Mean intensity ratings and S.D. for the seven facial expressions for both motion conditions

Emotion for motion conditions	Mean Intensity Rating	(S.D.)
Anger Static	3.60	(0.53)
Anger Dynamic	3.76	(0.48)
Disgust Static	3.94	(0.52)
Disgust Dynamic	3.93	(0.53)
Fear Closed Static	3.23	(0.48)
Fear Closed Dynamic	3.41	(0.54)
Fear Open Static	3.84	(0.56)
Fear Open Dynamic	3.99	(0.48)
Happiness Static	3.56	(0.69)
Happiness Dynamic	3.74	(0.72)
Sadness Static	3.13	(0.51)
Sadness Dynamic	3.24	(0.47)
Surprise Static	3.62	(0.54)
Surprise Dynamic	3.70	(0.60)

3.4 Discussion

This study examined adults' recognition accuracy, confidence in recognition and rating of stimulus intensity in a set of six basic emotions presented in static or dynamic forms. The main predictions were: 1) that dynamic stimuli would be recognised more accurately and with more confidence than their static counterparts; 2) that motion would be particularly beneficial in recognition of those emotions poorly recognised in the static form; 3) that dynamic stimuli would be considered more intense than static images; and 4) that the motion advantage would be increased for emotions perceived to be less intense. The main findings were: 1)

there was not an overall advantage for dynamic stimuli: disgust and surprise were recognised better in static form, fear with closed mouth and sadness were recognised better in dynamic form, and fear with open mouth, anger and happiness were not influenced by motion properties; 2) dynamic stimuli were given a higher confidence rating than static stimuli, particularly anger, sadness, and both forms of fear; 3) happiness was given the highest confidence rating, and fear (with closed mouth) the lowest; 4) dynamic stimuli were perceived as more intense than static stimuli; 5) disgust and fear with open mouth were perceived as the most intense, and fear with closed mouth and sadness the least intense. Each of these findings will be discussed below.

3.4.1 Is there a motion advantage for recognising facial expressions?

The finding that static stimuli were generally recognised with more accuracy than dynamic stimuli is in contrast to the prediction, based on previous findings which describe an overall motion advantage in the recognition of facial expressions of emotion counterparts (Ambadar et al., 2005; Bassili, 1978; Pike et al., 1997; Wehrle, et al., 2000). However, an advantage for motion has not been a consistent finding, with other results suggesting that emotions in both motion conditions are recognised equally well (Fiorentini & Viviani, 2011; Gepner et al., 2001) or, as in this study, that static versions were better recognised (Kamachi et al., 2001). When comparing individual emotion categories it is evident that motion seems to influence certain emotional conditions more than others (Harwood et al., 1999). Fujimura & Suzuki (2010) found motion improved recognition for happy, fearful and excited

expressions, but not calm, sleepy, sad, angry or surprised. The general finding that motion benefits recognition of some but not all emotional expressions is also true of the current study; with fear with closed mouth and sadness being recognised better in dynamic form, and disgust and surprise being recognised better in static form. In contrast, recognition of fear with open mouth, anger, and happiness were not affected by the motion properties of the stimuli. The two emotions that show improved recognition in the dynamic form may have more subtle facial cues, which is not enough to distinguish them from other emotions with similar musculature in static versions. For example, recognition improves in the dynamic form for sadness, which in the static form is misjudged as many different emotions when incorrectly labelled. Neural circuitry may be finely-tuned to respond to specific displacements in facial features, such as the eye region, mouth and eyebrows. These differences may become more evident in the less intense/easily confused emotions. In contrast, emotions that are well-defined in the static form, such as surprise, show no obvious benefit for motion. This could be tested using graded morphs blending two emotions together with incremental changes to assess recognition.

Happiness is consistently recognised with the highest degree of accuracy, and rarely confused with other emotions (Adolph & Alpers, 2010; Adolphs, Jansari, & Tranel, 2001; Montagne et al., 2007) and for both motion conditions (Kamachi et al., 2001). This study replicated these previous findings, as happiness was recognised with 99% accuracy for the dynamic condition, and 100% for the static condition. Happiness is the only positive emotion assessed in most emotion recognition studies, and this positive-negative distinction could act to increase recognition rates of happiness,

with an observable ceiling effect. Fujimura & Suzuki (2010) attempted to overcome this bias by introducing three pleasant positive stimuli (happy, calm and excited) contrasted with three negative stimuli (angry, fearful, and sad). They found that accuracy for happiness decreased in the presence of the other pleasant stimuli and an advantage for motion was observed. This limitation in the current study and the majority of emotion research needs to be addressed in the future. The inclusion of other pleasant stimuli alongside happiness may result in a reduction in the ceiling effect and allow the influence of motion to be assessed effectively.

In this study, fear was recognised with the lowest accuracy overall and for the two motion conditions, particularly when displayed with a closed mouth. This replicates previous research which shows that fear is recognised less accurately and more slowly (Kohler et al., 2004; Montagne et al., 2007). This may be because it is often misidentified as surprise, with recognition rates decreasing significantly where both fear and surprise labels are used (Adolphs, 2002) as they share similar features such as wide eyes and open mouth (Eisenbarth et al, 2008). It is also true that fearful faces are rarely encountered in social situations and this may reduce expertise in identifying fearful faces. In this study, fear in both motion conditions, was misidentified as surprise in the majority of cases (between 15 and 25%). The recognition accuracy of happiness and fear in this study was reflected in the confidence ratings, with the highest rating given to happiness and the lowest to fear (with closed mouth) for stimuli correctly identified. Wehrle and colleagues (2000) found that fear was recognised better in the dynamic than static condition, and this was certainly true for fear (with closed mouth) in this study. In fact as predicted,

motion improved the recognition accuracy significantly for this emotion which was poorly recognised as a static image. Interestingly, the confidence rating for fear was significantly higher for dynamic stimuli compared with static. The overall higher confidence ratings for dynamic stimuli compared with static replicates previous findings (Ambadar et al, 2005; Pike et al, 1997). Sadness was recognised well for both motion conditions in this study (87% and 94% respectively). To note, those that incorrectly identified sadness chose a wide variety of alternative emotions including the other five basic emotions. This may be due to the subtlety of the expression even at its apex, and as this study shows, the additional information produced by motion assists this recognition.

3.4.2 Does motion make emotions seem more intense?

As predicted, dynamic stimuli were considered more intense than static stimuli, thus replicating the findings of Biele & Grabowski (2006), who showed that overall dynamic stimuli were rated as more intense. In this study, fear with closed mouth and sadness were considered the least intense emotions, and disgust and fear with mouth open the most intense. Interestingly, the recognition of fear with closed mouth and sadness was improved significantly by motion, with no improvement for disgust and fear with open mouth. This replicates the findings of Bould & Morris (2008) who also found that the benefits of motion were greater for subtle/less intense expressions and less pronounced for more intense expressions. The stimulus set in the current study contains high intensity emotional expressions (as discussed in Adolph & Alpers, 2010). Previous research has demonstrated that there is a direct

relationship between intensity and recognition accuracy, with higher intensity emotions resulting in an increased recognition accuracy (Adolph & Alpers, 2010). Hess, Blairy and Kleck (1997) found this relationship for the static emotions anger, disgust, and sadness, but not happiness due to ceiling effects for this emotion.

3.4.3 Methodological issues

It was important to test the reliability of the static stimuli by comparing the recognition accuracy of this study with the original study by Tottenham and colleagues (2009). There was generally a good consistency between the two scores, with 77% of comparisons statistically similar. Looking at the significantly different comparisons in more detail showed that happiness had 100% accuracy of recognition for every stimulus in this study, and performance was generally better than the original study, significantly so in two cases. Fear with closed mouth was poorly recognised across both studies, generally worse in this study, significantly so in three cases. Fear with open mouth and disgust were generally better recognised in this study, and significantly so in four cases. The other emotions showed variability within stimuli between studies but there were no obvious patterns of higher ratings for one study compared with another within emotion categories. The variability observed may be due to differences in a) sample size: a larger population in the original study compared to this study (n=81 and n=36 respectively); b) stimuli: dynamic stimuli presented alongside may have influenced recognition accuracy of static stimuli in this study; neutral and calm faces, and open and closed versions of all emotions were included in the original study; d) task demands: participants rated

the full stimuli set twice in the original study, possibly increasing familiarity with the stimuli, whereas participants were only exposed to the stimuli once in the current study.

Fear with closed mouth was poorly recognised across both static and dynamic conditions in this study (41.7% & 57.9% respectively). The purpose of including this expression was to control for mouth movements in the following ERP study with infants (Chapter 7), where the neural response to fear and happiness is examined. It has been postulated that neural responses observed to facial expressions may be dependent on differences in basic facial movement, such as mouth and eye movement, as opposed to the emotional content of the face per se. Based on the poor recognition of fear with closed mouth, further studies in this thesis will only include fear expressed with the mouth open in both static and dynamic form. Once fear with closed mouth was excluded, two female and two male models were selected based on high mean recognition scores across emotion and motion conditions. These four models will be used in the adult electrophysiological studies in Chapters 4 & 5, and the two female models will be used in the infant electrophysiological study in Chapter 7.

3.4.4 Conclusions & limitations

In summary, the effect of motion on the accuracy of emotion recognition varied across the six basic emotions. The lack of overall motion advantage in emotion

recognition in this study is possibly due to the high intensity of the stimuli, resulting in a high degree of recognition for the static images, producing less of a recognition advantage for the dynamic images. Other studies using intense expressions have also been unable to show a robust effect of motion (Fiorentini & Viviani, 2011; Gepner et al., 2001; Harwood et al., 1999), possibly due to this effect. Motion has a positive influence on certain emotions that were rated as less intense, such as fear with closed mouth and sadness. In future research, it will be important to include less intense expressions, reducing recognition accuracy, to assess motion effects. These divergent results could also be due to the different population being used in the studies, e.g. different age groups, and clinical groups (Ambadar et al., 2005; Gepner et al., 2001). The variety of emotions employed may influence the recognition accuracy, for example the omission of surprise improves fear recognition, and hence a reduction in the motion advantage (Gepner et al., 2001). Another element that varies across studies is the timing of the images, e.g. speed of dynamic images and duration, and the use of morphed versus natural moving images. Specific motion speeds have been shown to be optimal for recognition of different emotional expressions (Sato & Yoshikawa, 2004).

It is important to consider all the methodological differences and limitations when comparing across studies and making a conclusion regarding motion effects. The use of morphed images, in this study and others, reflects the desire to regulate the timing and content of the emotional expression. The purpose of using dynamic images in emotion research is to create emotional expressions that closely represent natural expressions encountered in the social environment. The ultimate aim is to replicate the neural response evoked when viewing emotional faces in a social

context. Although morphing techniques allow control over temporal aspects of the expressions, such as rate of change, apex period and offset time; future research must look to improve on dynamic stimuli to make them more realistic. The limitations to current morphing methods result in facial features changing at the same rate, with linear movement across frames, which may not be considered realistic. Improvements could be achieved by using three-dimensional morphing techniques or by regulating natural moving videos so elements of the movement can be measured. Additionally, subtle emotional judgments could be explored with the use of morphs transitioning from one emotional expression to another, e.g. happiness to sadness.

The implications of these findings for the following studies in this thesis are three-fold; firstly the degree of recognition across the six emotions for both motion conditions was high. The dynamic stimuli were overall comparable in recognition to static stimuli, with some emotions being recognised better in dynamic form, and some in static form. Thus, there is confidence that the dynamic stimuli accurately depict the emotional expression, and would potentially activate brain structures involved in emotion processing such as the amygdala, fusiform gyrus and STS. Secondly, the static stimuli are considered intense compared to other emotional stimulus sets (Adolph & Alpers, 2010) and the dynamic stimuli were perceived as more intense than the static stimuli in this study. Intensity has been shown to influence activity in certain brain regions involved in emotion processing, such as the amygdala (Vuilleumier & Pourtois, 2007), that are possibly more sensitive to the intensity than to the valence of the presented stimulus (Anderson et al, 2003). The engagement of these emotion-related brain structures may in turn modulate and

enhance cortical information processing in regions such as the fusiform gyrus and STS (Vuilleumier et al., 2004). Thirdly, there is a clear distinction between emotions in both recognition accuracy and perceived intensity, and this will be reflected in differential activity in the distributed network associated with emotion processing. These factors together will influence the activity of the underlying neural structures involved in emotion recognition and contribute to the electrophysiological responses observed in the following ERP studies.

4. The effect of motion on the early processing of emotional faces

4.1 Introduction

Electrophysiological research has contributed significantly to our understanding of the temporal sequence of face processing and emotion perception. However, most studies have used static images of faces (Krolak-Salmon et al., 2001). As outlined in Chapter 1, it is important to extend our understanding of how faces and facial expressions are processed in our naturally dynamic social environment using more ecologically valid stimuli. Not only are dynamic faces more realistic, but they can also facilitate recognition of emotional expressions (Ambadar et al., 2005; Bassili, 1979; Harwood et al., 1999; Sato & Yoshikawa, 2004; Wehrle et al., 2000;), particularly emotions that are more difficult to recognise or are displayed at lower intensity (Ambadar et al., 2005). Moreover, neuroimaging studies show that, while dynamic facial expressions activate the same basic brain network as static ones, they do so more strongly (Sato et al., 2004; Trautmann et al., 2009). A question that remains outstanding is the timing of the benefit provided by motion to emotion processing—at what point of information processing does it occur? The aim of the experiment reported in this chapter is to investigate this question by recording event-related potentials (ERPs) in response to static and moving images of the six basic facial emotions.

4.1.1 ERPs and Static Facial Emotion

Event-related potentials provide a tool for addressing questions about the timing of perceptual and cognitive processing as they have millisecond resolution. It is established that the presentation of face stimuli to adults elicits a pair of deflections in the ERP waveform known as the P1 and N170. The P1 is a positive deflection peaking around 100 ms after stimulus onset over occipital regions. It is elicited by faces, but also other visual stimuli, and reflects the initial early, rapid processing of both simple and complex visual stimuli in extrastriate cortex. There is some evidence that the P1 is modulated by emotional expression, with an increased P1 amplitude for fearful compared with neutral faces (Kolassa, Musial, Kolassa, & Miltner, 2006; Pourtois et al., 2005), indicating an initial global and automatic encoding of emotional expression. This enhancement of the P1 for fear might reflect the attention-getting properties of fearful faces, since P1 amplitude is increased to attended locations (Mangun, Hopfinger, Kussmaul, Fletcher, & Heinze, 1997). The N170 ERP component follows the P1, and is a negative deflection at occipito-temporal sites that peaks approximately 170ms after stimulus onset (Bentin et al., 1996) and is maximal over right lateral ventral temporal-occipital electrodes. The N170 demonstrates substantial specificity for faces, typically being larger and earlier for upright faces compared with other visual object categories and is thought to reflect structural encoding of faces (Bentin et al., 1996). The source generators of the N170, as indicated by intracranial recordings and ERP source analyses, are in the fusiform gyrus (Itier & Taylor, 2002), lateral temporo-occipital cortex (Allison, Puce, Spencer, & McCarthy, 1999; McCarthy, Puce, Belger, & Allison, 1999; Puce, Allison, &

McCarthy, 1999), and posterior STS (Itier & Taylor, 2004); regions that in functional magnetic resonance imaging (fMRI) studies show enhanced activation to dynamic compared to static emotional faces (Kilts et al., 2003; Trautmann et al., 2009).

There is debate, however, as to whether the N170 is modulated by emotional expression. Some authors report that the N170 is not modulated by emotional expression (Eimer et al., 2003; Eimer & Holmes, 2002; Holmes et al., 2003), and argue that processing of the emotional content is reflected in other components such as an enhanced fronto-central positivity around 120ms, and a broadly distributed positivity beyond 250ms post-stimulus (Eimer & Holmes, 2002, 2007). However, other studies have highlighted that emotional expression can affect the N170, with fearful faces evoking a larger N170 amplitude compared with neutral (Blau, Maurer, Tottenham, & McCandliss, 2007; Leppänen et al., 2007), happy (Lepännen et al., 2007) or surprised faces (Batty & Taylor, 2003). Also, happy, surprised and neutral faces have been shown to evoke an earlier N170 than fear, disgust and sadness (Batty & Taylor, 2003). In another study, the N170 was enhanced for emotional stimuli compared to non-emotional stimuli, however the effect of both fearful and happiness were the same (Williams et al, 2006). It is possible that these inconsistent results are in part due to the non-optimal nature of the static images used and that more consistent findings will emerge with dynamic images. An emotion-specific P1/N170 response was predicted for the current study based on the outcome of a previous study implementing a similar ERP protocol to the one used in this thesis (Batty & Taylor, 2003), with participants responding to target stimuli with a mouse-click to ensure they were attending to the stimuli, in an otherwise passive task. This prediction was also based on the findings from fMRI

research, illustrating a differential pattern of activity during the perception of specific emotional states, which may be reflected in the P1/N170 response. This prediction can be extended further to incorporate the effect of motion on the P1/N170 response to specific emotions, and this will be discussed below.

4.1.2 P1, N170 and Dynamic Emotional Faces

While some studies indicate that the P1 and N170 are influenced by movement of facial features (Puce et al., 1998), there is only a limited number of electrophysiological studies involving dynamic facial expressions of emotion. Mayes and colleagues (2009) used steady-state visual evoked potentials to examine emotion and gender processing for both static and dynamic faces. Participants' brain activity was recorded under four conditions: (a) passive viewing of scrambled faces; (b) passive viewing of static and dynamic male and female fearful and surprised faces; (c) active gender categorisation task for static and dynamic images; and (d) active emotion categorisation task (fearful versus surprised) for static and dynamic images. Comparison of the two passive viewing conditions revealed a shorter latency response in the time-window of the N170 for viewing static or dynamic faces compared to viewing static scrambled faces. Comparison of the two active conditions revealed no early latency (before 400ms) differences for emotion compared to gender categorisation. The authors argued that early-latency processing involves extracting of invariant facial information that occurs similarly for static and dynamic images. However, this conclusion is limited because (a) the study included only a limited range of emotions (fear, surprised); and (b) the static and

dynamic conditions were kept separate in the analysis and not directly compared. The authors did observe longer latency differences (post-200ms) indicating a differential time course of processing static and dynamic images and with a facilitated processing of dynamic images. In another study by Recio, Sommer & Schacht (2011), participants performed expression categorisations for static and dynamic facial displays of angry, happy, or neutral emotional expressions. They found longer latency (post-200ms) enhancement of ERP responses to dynamic displays but no modulation of the P1 or N170 by facial motion. Again, this study suffered from several limitations: (a) including only a limited range of emotions; (b) creating moving images from three static images of varying intensity which may have constrained the perception of movement; and (c) obtaining low categorisation rates for happiness, an unusual finding (happiness is usually best recognised and often at ceiling), which questions the representativeness of the facial images.

Neither of these studies examined the topography of P1/N170 responses and how this might differ for static and dynamic facial images. Generators of the N170 include the fusiform gyrus, lying along the ventral visual pathway involved in pattern processing, as well as the STS, lying along the dorsal visual pathway involved in motion processing, including biological motion. It is possible that static and dynamic emotional faces differentially activate these generators of the N170 thereby resulting in different scalp topographies, with the N170 elicited by static images more prominent over lateral-ventral electrodes and the N170 elicited by dynamic images more prominent over medial-dorsal electrodes.

In summary, there is reason to believe that static and dynamic emotional expressions might be processed differently as early as the P1 and N170 stages of processing, as the N170 is influenced by facial movement and the P1 is sensitive to motion (typically being larger for static than dynamic patterned stimuli; e.g. Armstrong, Neville, Hillyard & Mitchell 2002). In spite of this, the only two ERP studies examining the role of facial movement in facial emotion processing found no early latency (pre-200ms) effects. However, both suffered from methodological limitations that may have contributed to the null results.

4.1.3 Summary and Predictions

Behavioural and fMRI evidence suggest facilitated processing of facial emotion in dynamic faces with stronger activation of relevant brain networks but the time course of these effects is not clear. Only two ERP studies have attempted to investigate this issue, both finding evidence of differential processing of static compared to dynamic facial emotions after 200ms of stimulus exposure, but not before this in the time-window of the P1 and N170 components associated with encoding faces. Both studies suffer from methodological limitations including not directly comparing static and dynamic conditions (Mayes et al., 2009); implementing only a limited range of emotions (Mayes et al., 2009; Recio et al., 2010); using stimuli that may have been atypical and not optimal for eliciting effects (Recio et al., 2010); not examining topographical effects in detail; and not including a non-face control condition (Mayes et al., 2009; Recio et al., 2010). Thus, this study will address these limitations by examining high-density ERPs to the full range of basic emotions using a

validated set of stimuli, as well as a non-face control, and directly comparing static and dynamic versions. The specific questions and hypotheses to be addressed are as follows:

1. Movement will enhance the P1/N170 to emotional faces: The prediction of larger amplitude and shorter latency P1/N170 to dynamic compared to static images is based on fMRI studies showing stronger activation for dynamic compared to static emotional faces in regions thought to generate P1/N170 (LaBar et al., 2003; Kilts et al., 2003; Sato et al., 2004; Trautmann et al., 2009). To the extent that this effect reflects stronger activation of the social brain by dynamic compared with static facial emotion, this pattern will be diminished or absent, or perhaps even opposite for non-face control stimuli, as patterned static images elicit a larger P1 than dynamic ones (e.g. Armstrong, Neville, Hillyard & Mitchell 2002).
2. The effect of movement on the P1/N170 will vary by emotion: This prediction is based on behavioural studies indicating that the benefits of motion may be greatest for emotions that are more difficult to recognise (e.g., fear) or lower in intensity (e.g., sad) and neuroimaging studies indicating stronger effects of motion on processing of certain emotions such as fear (LaBar et al., 2003). Thus, the effects of motion on P1/N170 may be strongest for fear (and other less well recognised or less intense emotions) and weakest for happy (and other better recognised emotions or higher intensity emotions).
3. The topography of the P1/N170 will be influenced by motion: If dynamic stimuli are more optimal for activating the dorsal-STS pathway, and static stimuli the ventral-fusiform pathway, corresponding differences are expected

in ERP topography. To the extent that any differences reflect activation of the social brain network, this pattern will be diminished or different for the non-face control.

4.2 Methods

4.2.1 Participants

Twenty-five healthy volunteers (14 female), aged 17-40 years (mean age 25.7, S.D. 5.44) participated in the study. All participants had normal or corrected–to-normal visual acuity, with no recorded medical problems associated with vision. All self-reported as having no extraneous neurological or psychological disorder. The study was approved by ICH/GOSH Research Ethics Committee (REC reference 07/Q0508/35), and performed according to the standards of the Declaration of Helsinki (1964). Each individual provided informed written consent prior to participation in the study. Participants were recruited through advertisement at University College London. Each participant received payment for their participation (£10), and none of these volunteers participated in the previous behavioural study (Chapter 3).

Data from twenty participants were used in the final statistical analyses, (10 female), aged 17-39 years (mean age 25.5, S.D. 5.02). Exclusion of five participants was due to lack of sufficient data because of artefacts in the EEG (3); and procedural error (2). Handedness was assessed by means of the Edinburgh Handedness Inventory

(Oldfield, 1971), and laterality quotients were recorded, mean = 84.8, S.D. = 5.74. There were no recorded left-handed participants.

4.2.2 Materials

Static and dynamic images of the six basic expressions (anger, disgust, fear, happiness, sadness, and surprise) posed by two female models (one European-American and one African-American) and two male models (one European-American and one African-American) (as discussed in Chapter 3, section 3.4.3).

Additionally, a colour photograph of an open flower was presented, with a moving counterpart depicting an opening flower, acting as targets. Figure 4.1 below illustrates the first and last frame of the morph of the flower from closed to open. The morphed dynamic flower stimulus was created in the same method used to create the morphed dynamic face stimuli (as described in Chapter 2, section 2.2). The resulting morph had a frame rate of 60 frames/45ms. The open flower was used as the static stimulus.

Figure 4.1 Closed and open flower, first and last frame of the dynamic stimuli. The open flower was used as the static stimulus.

4.2.3 Experimental Task

After the application of the sensor net, (refer to Chapter 2, section 2.1.2.2 for details of application), participants sat facing a flat computer screen, surrounded by a black screen in a dimly-lit room, minimising peripheral visual distractions. Each stimulus was presented in the centre of the computer screen at a visual angle of 11° x 8° when viewed from a distance of 60cm. The stimuli were presented to the participants in two separate blocks of static and dynamic stimuli, all displayed on a white background. Stimuli were presented for 750ms and separated by a random inter-stimulus interval of 1400-1700ms in which a black cross on a white background was presented in the centre of the screen. The participants were instructed to sit as still as possible and observe the presented stimuli, and to fixate on the inter-stimulus cross as it appeared. They were instructed to respond to the target flower stimulus

with a right-hand button press and refrain from responding to all face stimuli. This task was implemented to ensure the participants attended to the stimuli. The mean correct hits for the static and dynamic stimuli were 99.2% and 98.9%.

Each of the two experimental blocks consisted of 48 trials of the seven different stimuli (six emotions and target, 672 trials in total across both blocks). The blocks of static and dynamic stimuli were counterbalanced across subjects, with each block lasting approximately 11 minutes in total, with a break in between blocks for participants to rest. The stimuli were presented with equal probability in a pseudo-randomised order, with the constraint that no more than two consecutive stimuli would be the same emotion. The target flower was presented with 14% probability. The mean luminance was equal across stimuli. The script was prepared and implemented using the software programme Presentation (version 10.3, Neurobehavioural Systems) and the stimuli were presented on a computer running Windows 2000.

4.2.4 Statistical Analyses

The channel groupings are those discussed in Chapter 2, section 2.1.5. The P1 peak was defined as the most positive peak in the time-window ranging from 83ms-163ms post-stimulus onset. The N170 peak was defined as the most negative peak in the time-window ranging from 119-215ms post-stimulus onset.

Only the ERP responses to non-target (faces) trials were included in analysis; except where specifically stated (i.e. when comparing the emotional to the non-emotional conditions).

There were 48 trials per condition presented to each participant (2 motion x 7 emotion conditions). For the final 20 participants, the mean percentage of original trials retained after ERP derivation (across participants) was 91.1% (S.D. = 5.58, range 38-47 trials), 92.0% (S.D. = 4.89, range 39-47 trials), 90.9% (S.D. = 5.54, range 38-47 trials), and 90.5 % (S.D. = 5.29, range 40-47 trials), 91.9% (S.D. = 4.11, range 41-47 trials), 93.0% (S.D. = 4.14, range 40-47 trials), and 92.5% (S.D. = 4.21, range 40-47 trials) for the Static conditions Anger, Disgust, Fear, Happiness, Sadness, Surprise, and Non-Emotional (Target) respectively. Also, 91.6% (S.D. = 5.06, range 38-47 trials), 92.2% (S.D. = 3.96, range 40-47 trials), 92.1% (S.D. = 3.93, range 40-47 trials), 90.9% (S.D. = 5.66, range 38-47 trials), 91.0% (S.D. = 4.42, range 40-47 trials), 91.4% (S.D. = 4.77, range 40-47 trials), and 91.9% (S.D. = 4.14, range 41-47 trials) for the Dynamic conditions Anger, Disgust, Fear, Happiness, Sadness, Surprise, and Non-Emotional (Target) respectively.

4.3 Results

4.3.1 P1

The P1 was bilaterally distributed and largest and quickest over medial, ventral electrodes.

4.3.1.1 P1 Amplitude

4.3.1.1.1 Does Motion Enhance Processing?

There was a significant main effect of Motion, $F(1, 19) = 42.08$, $p < 0.0005$, with a significantly larger amplitude in the static condition compared with dynamic condition (difference of 0.51μV).

4.3.1.1.2 Does the Motion Effect vary by Emotion?

There was no significant main effect of Emotion, but there was a borderline significant two-way interaction of Motion and Emotion, $F(5, 95) = 2.47$, $p = 0.054$. Inspection of the means suggested that the direction of the motion effect was the same across emotions but was particularly large for disgust, see Table 4.1 below. Also refer to Figure 4.5 at the end of the results section.

Table 4.1 Mean peak P1 amplitude & S.D. values for the motion conditions for emotions

Category	Mean (μV)	(S.D.)
Anger Static	3.63	(0.96)
Anger Dynamic	3.05	(0.79)
Disgust Static	3.70	(0.98)
Disgust Dynamic	2.96	(0.87)
Fear Static	3.57	(1.01)
Fear Dynamic	3.11	(0.86)
Happiness Static	3.57	(0.95)
Happiness Dynamic	3.05	(0.87)
Sadness Static	3.59	(1.03)
Sadness Dynamic	3.05	(0.82)
Surprise Static	3.44	(0.98)
Surprise Dynamic	3.25	(1.00)

Prior studies have reported enhanced early-latency ERPs to static fearful faces compared to other emotions over right lateral ventral regions. A targeted analysis was performed over this electrode grouping. There was a significantly larger amplitude P1 for fear compared with other emotions, for the dynamic condition $t(1, 19) = 2.57$, $p < 0.05$ (difference of 0.81μV); but not the static condition, $t(1, 19) = 0.36$, $p = 0.723$ (difference of 0.06μV). Mean peak amplitude and S.D. values are shown in Table 4.2 below.

Table 4.2 Mean peak P1 amplitude & S.D. values for emotion conditions over right lateral ventral regions

Category	Mean (μV)	(S.D.)
Fear Static	4.23	(1.54)
Other Emotions Static	4.29	(1.80)
Fear Dynamic	4.28	(1.57)
Other Emotions Dynamic	3.47	(1.47)

4.3.1.1.3 Does the P1 Topography differ for Moving versus Static Faces?

A significant two-way interaction was observed between Motion and Dorsal-Ventral Topography, $F(1, 19) = 15.49$, $p < 0.0005$. The static condition produced a significantly larger amplitude than the dynamic condition over both dorsal, $t(1, 19) = 6.46$, $p < 0.0005$ (difference of 0.37μV), and ventral regions, $t(1, 19) = 6.04$, $p < 0.0005$ (difference of 0.64μV), but with a larger difference between motion conditions over ventral regions. Mean peak amplitude and S.D. values are shown in Table 4.3 below.

Table 4.3 Mean peak P1 amplitude & S.D. values for the motion conditions over dorsal-ventral regions

Category	Mean (μV)	(S.D.)
Dorsal Static	2.77	(0.68)
Dorsal Dynamic	2.40	(0.64)
Ventral Static	4.39	(1.29)
Ventral Dynamic	3.75	(1.08)

There was a trend towards a significant two-way interaction of Motion and Hemisphere, $F(1, 19) = 3.78$, $p = 0.067$. Inspection of the means suggested that this was driven by a larger amplitude for the static compared with the dynamic condition over both hemispheres, with a more pronounced difference over the right hemisphere. Mean peak amplitude and S.D. values are shown in Table 4.4 below.

Table 4.4 Mean peak P1 amplitude & S.D. values for the motion conditions over the two hemispheres

Category	Mean (μV)	(S.D.)
Right Static	3.76	(1.18)
Right Dynamic	3.17	(1.01)
Left Static	3.40	(0.89)
Left Dynamic	2.98	(0.81)

4.3.1.1.4 Are P1 Amplitude Effects Specific to Social Stimuli?

To determine whether the main effect of Motion on the P1 was specific to social stimuli, an ANOVA was computed with Stimulus (Emotional, Non-Emotional) and

Motion (Static, Dynamic) as the within-subjects variables over all electrodes. There was an interaction of Stimulus and Motion, $F(1, 19) = 22.90$, $p < 0.0005$. The P1 amplitude for the static emotional stimuli was larger than the dynamic emotional stimuli, $t(1, 19) = 6.49$, $p < 0.0005$, but the P1 amplitude for non-emotional stimuli showed the opposite pattern, with larger amplitude for the dynamic than the static stimuli, $t(1, 19) = 2.20$, $p = 0.039$, as shown in Table 4.5 below.

Table 4.5 Mean peak P1 amplitude & S.D. values for the motion conditions for emotional and non-emotional stimuli

Category	Mean (μV)	(S.D.)
Emotional Static	3.58	(0.93)
Emotional Dynamic	3.08	(0.80)
Non-Emotional Static	2.93	(1.03)
Non-Emotional Dynamic	3.19	(1.28)

4.3.1.1.5 Summary of P1 Amplitude

In summary, the P1 amplitude was larger for the static compared to dynamic emotional faces, but showed the opposite pattern for the non-face comparison condition. There was weak evidence that the effect of motion varied by emotion, with a trend for the static-dynamic amplitude difference to be particularly pronounced for disgust. Analysis targeted at right lateral ventral sites showed an enhanced P1 to fear compared to other emotions in the dynamic but not the static condition. There was evidence that the topography of the P1 was influenced by motion, with amplitudes for static stimuli showing a stronger bias towards right, ventral sites compared to left, dorsal sites and amplitudes for dynamic stimuli showing a similar, but less pronounced, bias.

4.3.1.2 P1 Latency

4.3.1.2.1 Does Motion Enhance Processing?

There was no significant main effect of Motion.

4.3.1.2.2 Does the Motion Effect vary by Emotion?

There was no main effect of Emotion or interaction with Motion.

4.3.1.2.3 Does the P1 Topography differ for Moving versus Static Faces?

There was a significant two-way interaction of Motion and Lateral-Medial Topography, $F(1, 19) = 5.28$, $p < 0.05$. The static condition produced a significantly shorter latency than the dynamic condition over lateral sites only, $t(1, 19) = 3.31$, $p < 0.01$ (difference of 1.38ms). Mean peak latency and S.D. values are shown in Table 4.6 below.

Table 4.6 Mean peak P1 amplitude & S.D. values for the motion conditions over lateral & medial sites

Category	Mean (ms)	(S.D.)
Lateral Static	113.39	(6.86)
Lateral Dynamic	114.77	(7.39)
Medial Static	116.77	(5.69)
Medial Dynamic	116.52	(7.01)

There was also a significant three-way interaction of Motion, Lateral-Medial and Dorsal-Ventral Topography, $F(1, 19) = 5.11$, $p < 0.05$. Further analysis revealed that for the static condition, there was a significant two-way interaction of Lateral-Medial and Dorsal-Ventral Topography, $F(1, 19) = 17.61$, $p < 0.0005$. The ventral P1 was shorter in latency compared with the dorsal P1 over medial regions only, $t(1, 19) = 4.24$, $p < 0.0005$ (difference of 11.03ms). For the dynamic condition, there was a significant two-way interaction between Lateral-Medial and Dorsal-Ventral topography, $F(1, 19) = 7.37$, $p < 0.05$. Ventral regions produced a shorter latency compared with dorsal regions over the medial sites only, $t(1, 19) = 3.027$, $p < 0.05$ (difference of 8.37ms). The difference between dorsal-ventral topography decreased in the order: static medial, dynamic medial, dynamic lateral, and static lateral. Mean peak latency and S.D. values are shown in Table 4.7 below.

Overall, the static condition produced shorter latencies over the lateral sites; for medial regions, the dynamic condition was quicker than the static over dorsal electrodes, with the reverse pattern for ventral electrodes, as illustrated in Figure 4.2 below.

Table 4.7 Mean peak P1 latency & S.D. values for dorsal-ventral topography for the motion conditions over lateral-medial regions

Category	Mean (ms)	(S.D.)
Static Lateral Dorsal	113.53	(8.27)
Static Lateral Ventral	113.24	(6.15)
Static Medial Dorsal	122.28	(9.73)
Static Medial Ventral	111.25	(6.17)
Dynamic Lateral Dorsal	115.11	(8.91)
Dynamic Lateral Ventral	114.43	(6.86)
Dynamic Medial Dorsal	120.70	(11.11)
Dynamic Medial Ventral	112.33	(7.17)

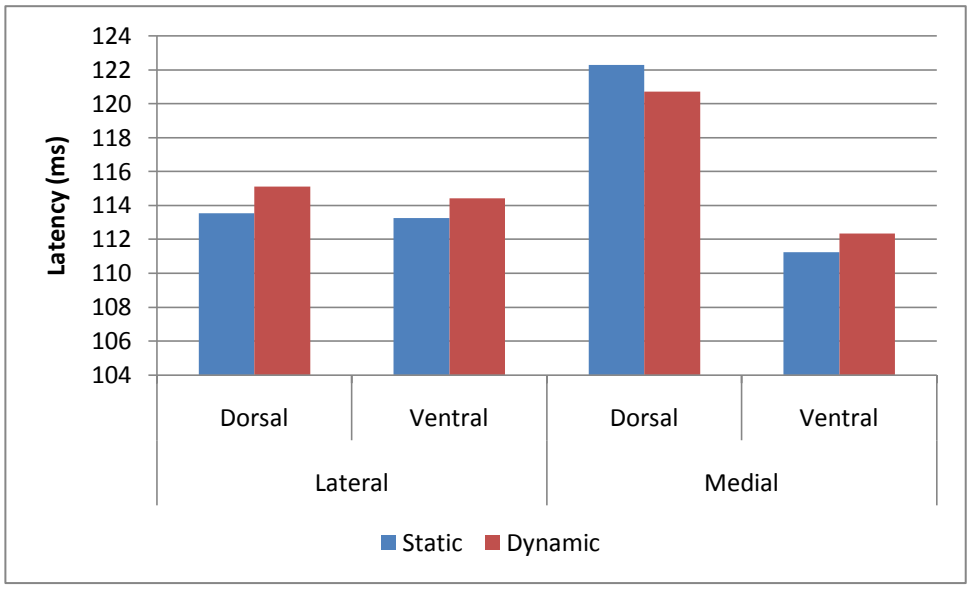


Figure 4.2 Mean peak P1 latency values for dorsal-ventral topography for the motion conditions over the lateral-medial regions

4.3.1.2.4 Are P1 Latency Effects Specific to Social Stimuli?

To determine whether the effect of motion on the P1 was specific to social stimuli, an ANOVA was computed with Stimulus (Emotional, Non-Emotional) and Motion (Static, Dynamic) and Topography (Dorsal, Ventral) as the variables over medial

electrodes. There was no significant Stimulus by Motion interaction, $F(1, 19) = 0.03$, $p = 0.857$.

4.3.1.2.5 Summary of the P1 Latency

In summary, the P1 latency did not show an overall difference by motion condition or for particular emotions. There were topographical differences between the speed of the P1 for static and dynamic conditions. Static faces were processed more quickly over lateral sites. Dynamic faces were processed more quickly over dorso-medial regions, while static faces were processed more quickly over ventro-medial regions. However, this pattern of latency differences did not differ statistically from that observed for the non-face control condition.

4.3.1.2.6 P1 Amplitude and Latency Summary

The P1 amplitude and latency were influenced by motion: the P1 amplitude was larger for static than dynamic stimuli over all sites and latencies were quicker for the static faces over lateral sites and ventral-medial sites and quicker for dynamic faces over dorsal-medial sites. Comparison with non-emotional stimuli showed that the pattern amplitude effects holds only for emotional stimuli but the latency findings are similar for non-emotional stimuli. Emotion did not have an overall influence on P1 amplitude or latency, though there was some evidence that the difference between static and dynamic amplitude was largest for disgust and there was

evidence that the P1 amplitude for dynamic, but not static, fear was larger than for other emotions over right ventro-lateral sites.

4.3.2 N170

The N170 was bilaterally distributed and largest over lateral, ventral electrodes; and quickest over medial electrodes.

4.3.2.1 N170 Amplitude

4.3.2.1.1 Does Motion Enhance Processing?

There was a significant main effect of Motion, $F(1, 19) = 34.93$, $p < 0.0005$, with a significantly larger amplitude in the static condition compared with the dynamic condition (difference of 0.68μV).

4.3.2.1.2 Does the Motion Effect vary by Emotion?

There was no significant main effect of Emotion, but there was a significant three-way interaction of Motion, Emotion and Lateral-Medial Topography, $F(5, 95) = 3.20$, $p < 0.05$. Further analysis revealed that there was a significant two-way interaction of Motion and Lateral-Medial Topography for the emotion happiness, $F(1, 19) = 18.10$, $p < 0.0005$; with the static condition producing a significantly larger amplitude compared with the dynamic condition over lateral sites only, $t(1, 19) = 6.38$, $p < 0.0005$ (difference of 0.64μV). There was also a trend towards a significant two-way

interaction of Motion and Lateral-Medial Topography for anger, $F(1, 19) = 4.06$, $p = 0.058$, with the static condition producing a significantly larger amplitude than the dynamic condition over both lateral, $t(1, 19) = 6.16$, $p < 0.0005$ (difference of 0.97μV), and medial sites, $t(1, 19) = 3.45$, $p < 0.005$ (difference of 0.65μV). There was also a trend towards a significant two-way interaction of Motion and Lateral-Medial Topography for fear, $F(1, 19) = 3.82$, $p = 0.066$, with the static condition producing a significantly larger amplitude than the dynamic condition over both lateral, $t(1, 19) = 4.55$, $p < 0.0005$ (difference of 0.93μV), and medial sites, $t(1, 19) = 3.38$, $p < 0.005$ (difference of 0.64μV). There was no interaction for disgust, sadness, or surprise. The difference in motion conditions was larger over lateral sites for all emotions and the largest amplitude was observed for static fear over lateral electrodes. Mean peak amplitude and S.D. values are shown in Table 4.8 below. Also refer to Figure 4.5 at the end of the results section.

Table 4.8 Mean peak N170 amplitude & S.D. values for the motion conditions over lateral & medial sites for anger, fear & happiness

Category	Mean (μV)	(S.D.)
Anger Lateral Static	-3.44	(1.37)
Anger Lateral Dynamic	-2.47	(1.08)
Anger Medial Static	-2.76	(1.50)
Anger Medial Dynamic	-2.11	(1.32)
Fear Lateral Static	-3.69	(1.50)
Fear Lateral Dynamic	-2.76	(1.42)
Fear Medial Static	-2.86	(1.31)
Fear Medial Dynamic	-2.22	(1.13)
Happiness Lateral Static	-3.45	(1.35)
Happiness Lateral Dynamic	-2.81	(1.12)
Happiness Medial Static	-2.43	(1.08)
Happiness Medial Dynamic	-2.38	(1.23)

Prior studies have reported enhanced early-latency ERPs to fearful static faces compared with other emotional stimuli over right lateral ventral regions. A targeted analysis was performed over this electrode grouping. There was a significantly larger amplitude for the fear compared with happiness for the static condition, $t(1, 19) = 2.11$, $p < 0.05$ (difference of 0.38μV), but not the dynamic condition, $t(1, 19) = 0.13$, $p = 0.90$ (difference of 0.02μV). Mean peak amplitude and S.D. values are shown in Table 4.9 below.

Table 4.9 Mean peak N170 amplitude & S.D. values for emotion conditions over right lateral ventral regions

Category	Mean (μV)	(S.D.)
Fear Static	-4.73	(2.18)
Happiness Static	-4.35	(2.09)
Fear Dynamic	-3.51	(2.13)
Happiness Dynamic	-3.53	(1.722)

4.3.2.1.3 Does the N170 Topography differ for Moving versus Static Faces?

A significant two-way interaction of Motion and Lateral-Medial Topography was found, $F(1, 19) = 5.98$, $p < 0.05$. The static condition produced a significantly larger amplitude than the dynamic condition over both the lateral sites, $t(1, 19) = 7.08$, $p < 0.0005$ (difference of 0.79μV), and medial sites, $t(1, 19) = 4.17$, $p < 0.001$ (difference of 0.56μV), with a larger difference between motion conditions over lateral sites. The largest amplitude was observed for the lateral static condition. Mean peak amplitude and S.D. values are shown in Table 4.10 below.

Table 4.10 Mean peak N170 amplitude & S.D. values for the motion conditions over lateral & medial sites

Category	Mean (μV)	(S.D.)
Lateral Static	-3.49	(1.34)
Lateral Dynamic	-2.70	(1.19)
Medial Static	-2.75	(1.29)
Medial Dynamic	-2.19	(1.18)

A significant three-way interaction of Motion, Lateral-Medial Topography and Hemisphere was observed, $F(1, 19) = 6.13$, $p < 0.05$. Further analysis revealed that for the static condition, there was a significant two-way interaction between Hemisphere and Lateral-Medial Topography, $F(1, 19) = 5.12$, $p < 0.05$. Lateral sites produced a significantly larger amplitude compared with medial over the right hemisphere only, $t(1, 19) = 3.09$, $p < 0.005$ (difference of 1.08μV). For the dynamic condition, there was a trend towards a significant two-way interaction of Hemisphere and Lateral-Medial Topography, $F(1, 19) = 3.48$, $p < 0.05$. Lateral sites produced a significantly larger amplitude compared with medial sites in the right hemisphere only, $t(1, 19) = 2.51$, $p < 0.05$ (difference of 0.79μV). The difference between lateral-medial topography decreased in the order: right static, right dynamic, left static, and left dynamic conditions. Mean peak amplitude and S.D values are shown in Table 4.11 below.

Overall the amplitude for static stimuli was greatest over right lateral sites, then left lateral, then right medial, then left medial sites. This pattern was similar for dynamic stimuli, but less pronounced, with the distinction between static and dynamic stimuli decreasing in the same order, as illustrated in Figure 4.3 below.

Table 4.11 Mean peak N170 amplitude & S.D. values for lateral-medial topography for the motion conditions over the two hemispheres

Category	Mean (μV)	(S.D.)
Static Right Lateral	-3.91	1.76
Static Right Medial	-2.83	1.13
Static Left Lateral	-3.07	1.43
Static Left Medial	-2.68	1.56
Dynamic Right Lateral	-3.05	1.62
Dynamic Right Medial	-2.26	1.15
Dynamic Left Lateral	-2.44	1.15
Dynamic Left Medial	-2.13	1.35

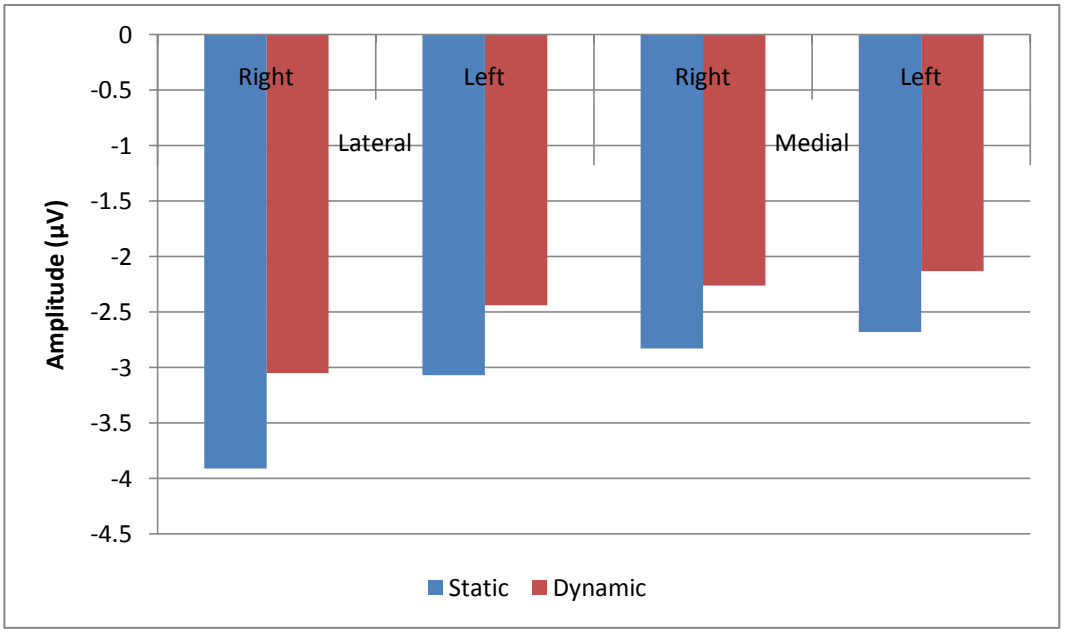


Figure 4.3 Mean peak N170 amplitude values for lateral-medial topography for the motion conditions over the two hemispheres

A significant two-way interaction of Motion and Dorsal-Ventral Topography was found, $F(1, 19) = 25.95$, $p < 0.0005$. The static condition produced a significantly larger amplitude than the dynamic condition over both the dorsal regions, $t(1, 19) = 5.22$, $p < 0.0005$ (difference of 0.41μV), and ventral regions, $t(1, 19) = 5.90$, $p < 0.0005$ (difference of 0.94μV), with a larger difference between motion conditions

over ventral regions. The largest amplitude was observed for the ventral static condition. Mean peak amplitude and S.D. values are shown in Table 4.12 below.

Table 4.12 Mean peak N170 amplitude & S.D. values for the motion conditions over dorsal & ventral regions

Category	Mean (μV)	(S.D.)
Dorsal Static	-2.28	(0.76)
Dorsal Dynamic	-1.87	(0.69)
Ventral Static	-3.96	(1.65)
Ventral Dynamic	-3.02	(1.46)

A significant three-way interaction of Motion, Lateral-Medial and Dorsal-Ventral Topography was observed, $F(1, 19) = 24.10$, $p < 0.0005$. Further analysis revealed that there was a significant two-way interaction of Motion and Dorsal-Ventral topography over both lateral, $F(1, 19) = 6.63$, $p < 0.05$, and medial sites, $F(1, 19) = 41.52$, $p < 0.0005$. Over lateral sites, the static condition produced a significantly larger amplitude than the dynamic condition in both dorsal, $t(1, 19) = 7.35$, $p < 0.0005$ (difference of 0.64μV) and ventral regions, $t(1, 19) = 6.11$, $p < 0.0005$ (difference of 0.94μV). Over medial sites, the static condition produced a significantly larger amplitude in the ventral regions only, $t(1, 19) = 5.33$, $p < 0.0005$ (difference of 0.95μV). Mean peak amplitude and S.D. values are shown in Table 4.13 below.

Overall the amplitude for static stimuli was greatest over lateral ventral sites, then medial ventral sites, then lateral dorsal sites, then medial dorsal sites. This pattern was similar for dynamic stimuli, but less pronounced, with the distinction between

static and dynamic stimuli decreasing in the same order, as illustrated in Figure 4.4 below.

Table 4.13 Mean peak N170 amplitude & S.D. values for the motion conditions over topographical regions

Category	Mean (μV)	(S.D.)
Lateral Dorsal Static	-2.97	(1.09)
Lateral Dorsal Dynamic	-2.33	(1.02)
Lateral Ventral Static	-4.01	(1.68)
Lateral Ventral Dynamic	-3.07	(1.44)
Medial Dorsal Static	-1.58	(0.85)
Medial Dorsal Dynamic	-1.41	(0.80)
Medial Ventral Static	-3.92	(1.87)
Medial Ventral Dynamic	-2.97	(1.68)

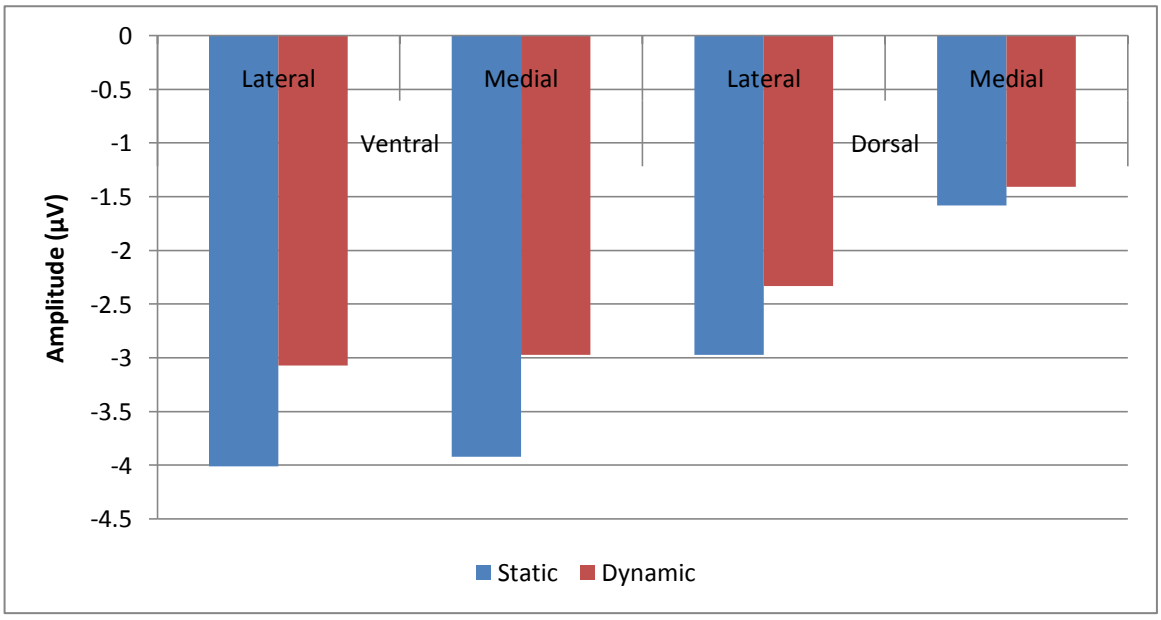


Figure 4.4 Mean peak N170 amplitude values for the motion conditions over topographical regions

4.3.2.1.4 Are N170 Amplitude Effects Specific to Social Stimuli?

To determine whether the effect of motion on the N170 was specific to social stimuli, an ANOVA was computed with Stimulus (Emotional, Non-Emotional) and Motion (Static, Dynamic) as the variables over all electrodes. This interaction did not reach significance, $F(1, 19) = 0.11$, $p = 0.744$.

4.3.2.1.5 Summary of N170 Amplitude

In summary, the N170 amplitude was larger for the static condition compared with the dynamic condition. There is some evidence that the effect of motion on the topography of the N170 varied by emotion, with the static-dynamic difference over lateral sites being particularly pronounced for anger, fear and happiness. Analysis targeting motion effects over right lateral ventral sites showed an enhanced N170 to fear compared with happiness in the static but not dynamic condition. There is also evidence that the topography of the N170 was influenced by motion, with amplitudes for static stimuli showing a stronger bias towards right, lateral, ventral sites compared to left, medial, dorsal sites. Amplitudes for dynamic stimuli showed a similar, but less pronounced bias, with the distinction between motion conditions being greatest over right lateral ventral sites.

4.3.2.2 N170 Latency

4.3.2.2.1 Does Motion Enhance Processing?

There was no significant main effect of Motion.

4.3.2.2.2 Does the Motion Effect vary by Emotion?

There was no main effect of Emotion, however there was a trend towards a significant three-way interaction of Motion, Emotion, and Dorsal-Ventral topography, $F(5, 95) = 2.39$, $p = 0.059$. Inspection of the means suggested that this was driven by a shorter latency for dynamic than the static condition over ventral but not dorsal regions for the emotion disgust. Mean peak latency and S.D. values for disgust are shown in Table 4.14 below.

Table 4.14 Mean peak N170 latency & S.D. values for the motion conditions over dorsal-ventral regions for disgust

Category	Mean (ms)	(S.D.)
Disgust Dorsal Static	162.50	(11.85)
Disgust Dorsal Dynamic	160.78	(9.57)
Disgust Ventral Static	163.11	(13.33)
Disgust Ventral Dynamic	157.97	(10.75)

A targeted analysis was performed over right lateral ventral electrodes to look for differences in N170 response between fear and other emotional conditions. There was a significantly shorter latency for fear compared with other emotions for the static condition, $t(1, 19) = 2.34$, $p < 0.05$ (difference of 3.08ms), but not the dynamic condition, $t(1, 19) = 1.03$, $p = 0.32$ (difference of 1.45ms). Mean peak amplitude and S.D. values are shown in Table 4.15 below.

Table 4.15 Mean peak N170 latency & S.D. values for emotion conditions over right lateral ventral regions

Category	Mean (ms)	(S.D.)
Fear Static	161.77	(10.04)
Other Emotions Static	164.85	(12.56)
Fear Dynamic	162.97	(13.49)
Other Emotions Dynamic	161.52	(10.45)

4.3.2.2.3 Does the N170 Topography differ for Moving versus Static Faces?

There were no interactions of Motion with any of the topographical variables.

4.3.2.2.4 Are N170 Latency Effects Specific to Social Stimuli?

To determine whether the effect of motion on the P1 was specific to social stimuli, an ANOVA was computed with Stimulus (Emotional, Non-Emotional) and Motion (Static, Dynamic) as the variables. This interaction did not reach significance, $F(1, 19) = 0.49$, $p = 0.495$.

4.3.2.2.5 Summary of the N170 Latency

In summary, the N170 latency did not show an overall difference by motion condition. There was weak evidence that the effect of motion varied by emotion, with a trend for the static-dynamic latency difference to be particularly pronounced for disgust. Analysis targeted at right lateral ventral sites showed shorter latency for

fear compared to other emotions in the static but not dynamic condition. There were no topographical differences between the speed of the N170 for static and dynamic conditions. There were no latency differences between the face and non-emotional condition.

4.3.1.2.6 N170 Amplitude and Latency Summary

The N170 amplitude was larger in amplitude for static compared with dynamic stimuli over all sites, particularly right lateral ventral sites. There was a trend for the latency to be quicker for dynamic stimuli over ventral regions for disgust. Comparison with non-emotional stimuli showed that the amplitude and latency were similar for non-emotional stimuli. Emotion did not have an overall influence on N170 amplitude or latency, though there was some evidence that the difference between static and dynamic amplitude was largest for anger, fear and happiness. There was evidence that the N170 amplitude for static, but not dynamic fear was larger than happiness; and N170 latency for static, but not dynamic fear, was quicker than other emotions, over right ventro-lateral sites.

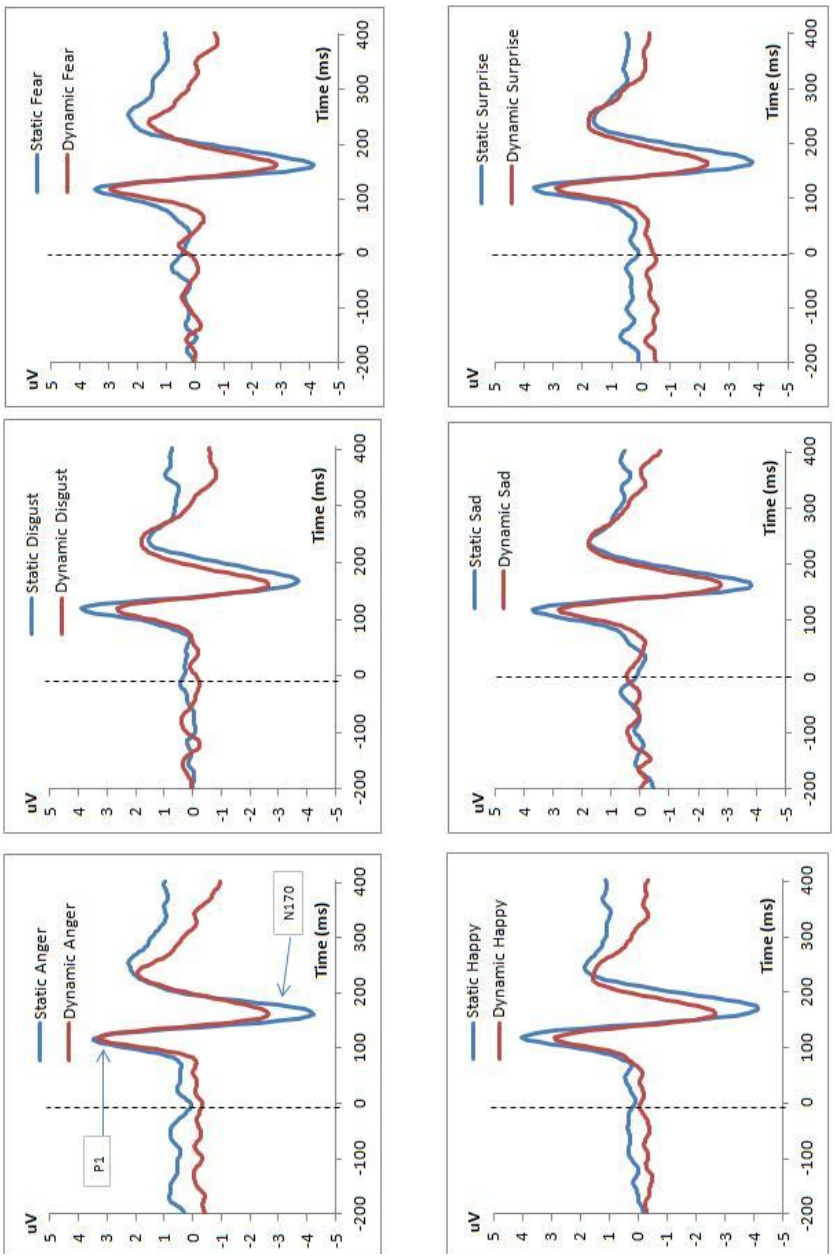


Figure 4.5 ERP waveforms illustrating the P1 and N170 for static and dynamic conditions for each of the six emotions over lateral sites.

4.4 Discussion

The study reported in this chapter aimed to investigate the influence of motion on the early-latency brain processing of facial expressions of emotion. The main predictions were that: 1) Motion will enhance the P1 and N170 to emotional faces; 2) the effect of motion on the P1 and N170 will vary by emotion, in particular being more pronounced for emotions that are more difficult to recognise (fear) or less intense (sad); 3) the topography of the two components will be influenced by motion; and 4) if the motion effects are specific to social stimuli, they will be diminished, absent, or even opposite for non-face stimuli.

The main findings will be listed below and then following this summary, each result will be discussed in turn.

In summary: There was an enhanced P1/N170 for static stimuli compared with dynamic stimuli, and this was modulated by both emotion and topography. There was evidence of activation of a 'social brain' for the P1 amplitude only.

More specifically:

1) Static stimuli produced an enhanced P1 and N170 compared with dynamic stimuli; with latencies quicker for the static faces over lateral sites and for dynamic faces over dorsal, medial sites.
2) There was no main effect of Emotion for either P1 or N170 amplitude or latency, however, there was some evidence that the influence of motion differed across emotions. There was an enhanced P1 for fear compared to

other emotions for dynamic stimuli only, an enhanced N170 for static fear compared to happiness, and an earlier N170 for static fear compared to the other emotions. The effect of motion on other emotions was less compelling. For disgust, there was a trend towards a more pronounced difference in P1 amplitude between motion conditions and a quicker latency for the N170 in the dynamic condition. For other emotions, the static-dynamic difference in N170 amplitude over lateral sites was particularly pronounced for anger, fear and happiness compared to the other emotions. There were no static-dynamic differences for sadness.

3) The P1 and N170 amplitudes for static stimuli showed a stronger bias towards right ventral (P1) and right lateral-ventral (N170) sites, with dynamic stimuli showing a similar, but less pronounced, bias. P1 latencies were quicker for static than dynamic stimuli over lateral sites, and latencies for dynamic stimuli were quicker than static over medial-dorsal sites, with the reverse pattern for ventral regions.
4) Comparison with non-emotional stimuli showed evidence consistent with specific activation of the 'social brain' for the P1 amplitude (where the direction of the effect of motion was opposite to that for the emotional faces). For the P1 latency and the N170 amplitude and latency, direct comparison of the emotional and non-emotional conditions did not produce a significant interaction, thus suggesting that effects were more general effects of motion.

4.4.1 The P1 and N170 response to static vs dynamic facial expressions

The findings show that there is a differential response of the early-latency components, the P1 and N170, to static and dynamic emotional stimuli. In contrast to the original prediction, the static emotional stimuli produced an enhanced P1 and N170 compared to emotional dynamic stimuli. The latency of the P1 also differed for static and dynamic faces, though the direction of the difference interacted with topography. It is possible that this differential response reflects activation of a common brain network, but to different degrees. This interpretation would be consistent with neuroimaging studies suggesting that the same basic network is involved in processing static and dynamic facial expressions, but that it is activated to different extents. However, the finding of an enhanced response observed for static faces indicates augmented processing for the static images compared to the dynamic images in the same neural network which is opposite to the pattern found in prior fMRI studies. Previous fMRI research has shown that dynamic emotional stimuli tend to increase activity in brain regions associated with face and emotion processing compared with static stimuli (Kilts et al., 2003; LaBar et al., 2003; Sato et al., 2004 Trautmann et al., 2009). Electrophysiological studies have found that dynamic images produce an enhanced response compared to static images in longer-latency components such as the EPN and LPP (Recio et al, 2010). It is possible that fMRI studies are reflecting this longer-latency activity.

The finding of a larger P1 for static compared with moving emotional images is consistent with prior studies of static versus moving patterned non-face stimuli

which find a similar direction of effect. However, in this study the non-face control condition produced the opposite pattern, with a larger amplitude for the dynamic compared to static condition. This difference in the face and non-face conditions can be seen as evidence that the response to faces observed in this study reflects activation of the 'social brain'. However, the non-face condition was also the target condition and it is possible that attentional differences contributed to the pattern of findings, a point that will be discussed in more detail in the General Discussion in Chapter 8.

A different reason for the differential response could be that the processing of static and dynamic depictions of facial emotion rely upon dissociable brain systems. Evidence comes from imaging studies showing differential activation patterns for dynamic stimuli compared with static, in regions associated with face and emotion processing, recruiting a wider neural network for dynamic emotional stimuli (Kilts et al., 2003; LaBar et al., 2003; Sato et al., 2004 Trautmann et al., 2009). In addition, studies in clinical populations have demonstrated differences in the recognition of moving and still images of facial expressions. In a study by Adolphs and colleagues (2003), an individual with damage to inferior temporal and subcortical limbic regions displayed difficulty recognising all static facial expressions except happiness but could recognise all dynamic facial expressions except disgust. Humphreys and colleagues (1993) reported a double dissociation in two prosopagnosic patients in performance on facial identity and affect tasks. One patient, who sustained ventral occipitotemporal damage, had problems with both facial identity and expression judgments using static images. However, their performance improved significantly when categorising facial expressions using moving point-light displays. The other

patient, who sustained bilateral parietal lobe damage, performed well on facial identity tasks but was impaired at both static and dynamic facial affect tasks. These findings suggest that static and dynamic facial emotions may be processed along different neural pathways extending the neurocognitive model of face processing by Haxby and colleagues (2000). Motion in general is thought to be processed differently from static aspects of visual stimuli. The ventral stream, encompassing occipital and inferior temporal regions involved in discriminating patterns, objects and colours; and the dorsal stream extending from the occipital to parietal regions involved in the processing spatial and motion aspects of the visual scene. One fMRI study indicated that the STS may be the central locus for neural activity responding to facial motion, integrating visual form and motion information from the two visual streams (Puce et al, 2003). It is difficult to draw a firm conclusion based on the present results as to whether a common system is activated to different extents, or dissociable systems underlie the effects of motion on emotion processing. If the same systems are active for both stimuli, with differential responses, this could be seen as evidence against the model of face perception postulated by Haxby and colleagues (2000), stating that dissociable systems are responsible for invariant and changeable aspects of face perception, as discussed further in the General Discussion in Chapter 8.

So why is there an enhanced P1 and N170 response for static emotional stimuli compared to dynamic emotional stimuli in this study? One explanation might be that the enhanced response observed for the static emotional stimuli is due to a greater allocation of attention based on higher intensity and saliency of the emotional content in the static stimuli compared with dynamic stimuli at early latencies.

Although the perceived intensity of the dynamic stimuli may be greater than static stimuli overall, as discussed in Chapter 3, this perceived intensity is based on viewing the entire transition of the emotional expression from neutral to the apex of the emotional expression. The maximal emotional expression is not fully developed until the end of the stimulus, at 750ms. So the maximal intensity of the expression will not be apparent at 100-200ms post-stimulus onset, the time window for the P1 and N170, the transition from neutral to maximal expression be only be approximately 20% complete. The amplitude of the N170 has been shown to be modulated by intensity of static emotional expressions, with an increasing negativity as the intensity increases from 50-150% (Sprengelmeyer & Jentzsch, 2006). In this study, the intensity effect was isolated to two symmetrically positioned generators within the temporo-occipital regions of the right and left hemispheres, and was not linked to the kind of expression presented (e.g. anger, disgust, fear). Schupp and colleagues (2003) found that emotionally high-salient pictures produced a stronger posterior negativity than emotionally low-salient pictures irrespective of valence, with this effect starting around 200ms. Sabatinelli and colleagues (2005) found increased activity in the inferior temporo-occipital lobe to emotionally high-salient compared to emotionally low-salient pictures. A greater response amplitude was observed to high compared with low arousal IAPS images (Cuthbert, Schupp, Bradley, Birbaumer, & Lang, 2000), and increased gamma band activity modulation for high-arousal (angry and fear) as compared to low-arousal (happy and neutral) faces (Balconi & Lucchiari, 2008). So it is possible that the N170 may be involved in coding the saliency of emotional stimuli. In addition, the N170 amplitude has been shown to be enhanced when faces are attended, suggesting that spatial attention can modulate

the structural encoding of faces (Holmes et al., 2003). These effects of intensity were not observed for the P1 (Leppänen et al., 2007; Sprengelmeyer & Jentzsch, 2006), however, a bias in selective attention towards emotional stimuli has been observed for the P1 (Holmes, Bradley, Kragh, & Mogg, 2009; Pourtois, Grandjean, Sander, & Vuilleumier, 2004; Santesso et al., 2008), with an enhanced P1 for negative relative to neutral or positive facial expressions (Pizzagalli et al., 1999; Pourtois et al., 2005). This enhanced response to intense and salient emotional stimuli is thought to reflect an increased allocation of attention and enhanced sensory encoding in visual brain areas as a result of feedback from emotion evaluation centres, such as the amygdala, following the rapid perceptual detection of a motivationally significant stimulus (Vuilleumier & Pourtois, 2007). This may account for the differences observed in the P1 and N170 time-window. It might be that the response to dynamic stimuli is only enhanced when the static conditions are sub-optimal, as observed in emotion recognition, with the dynamic advantage disappearing with intense, optimal static stimuli (Knight & Johnston, 1997; Lander et al., 1999). A possible argument could be that since the P1 is modulated alongside the N170 for static stimuli, this effect may be explained by differences in low level stimulus attributes of the two motion conditions. However, these motion effects were not observed for the non-face stimuli demonstrating that this is not the case and that the motion effects observed are specific to social stimuli.

4.4.2 Emotion effects and the influence of motion

There was no main effect of Emotion on either the P1 or N170, and the main evidence of modulation of the two components by emotion was found when directly comparing fear with other emotions combined, and happiness, separately for each motion condition. This resulted in an enhanced P1 response to fear compared with other emotions combined for dynamic stimuli only; and an enhanced N170 to fear compared with happiness, and an earlier N170 to fear compared with other emotions combined, both for static stimuli only. These effects were all observed over right, ventro-lateral electrodes. There is a proposed 'fast-route' fear recognition system, which is thought to rapidly respond to threatening environmental stimuli, possibly mediated by the amygdala which has a modulatory influence on face-sensitive areas in the occipito-temporal cortices (Krolak-Salmon, Henaff, Vighetto, Bertrand, & Mauguiere, 2004). This is part of the initial processing of visual stimuli which codes emotional salience, preceding information processing within the 'slow route' of emotion specific face recognition systems, thought to start around 300-350ms (Krolak-Salmon et al, 2003). It is possible that emotional discrimination may be based on relatively simple facial features, such as changes in the face including stretched mouth, wide open eyes, and furrowed and raised eyebrows (Kohler et al, 2004). For example, the amygdala is thought to be sensitive to the amount of white sclera exposed around the pupil (Whalen et al., 1998). It is thus possible that this enhancement of the P1 and N170 to fearful stimuli reflects amygdala modulation of early cortical processing.

There is debate as to whether early-latency components, such as the P1 and N170, are modulated by emotion. Consistent with the view that these early stages of processing are exclusively involved in the initial encoding of faces, independent of emotional content, many studies have found that these early components are not affected by emotional expressions (Eimer & Holmes, 2002, 2007; Krolak-Salmon et al, 2001). However, other studies have shown that these components are modulated by emotion. Some have shown an augmented P1 response to fearful faces relative to other emotions such as happy and also neutral faces (Pizzagalli et al., 1999; Pourtois et al., 2005; Streit et al., 1999). Studies have also reported modulation of the N170 to emotional expression (Ashley et al., 2004; Batty & Taylor, 2003; Campanella et al, 2002; Eger et al, 2003; Miyoshi et al, 2004; Pizzagalli et al, 2002), particularly an enhanced response for fearful faces compared with happy or neutral expressions (Batty & Taylor, 2003; Ashley et al., 2004). All of the above studies, however, only used static facial stimuli. The current study replicated previous findings that fear elicited a larger N170 than happiness in static faces, but in contrast to previous research, did not find a longer latency for fearful compared with happy faces (Batty & Taylor, 2003); instead finding a shorter latency to fearful compared with all other emotions combined. This study also found that fearful faces elicited a larger P1 compared to other emotions combined only for the dynamic stimuli, and did not replicate previous findings with static stimuli.

In addition, there was weak evidence that motion effects differ by emotion, with a more pronounced difference between motion conditions observed in the P1 amplitude for disgust; and the N170 amplitude for anger, fear and happiness; and a quicker N170 latency for dynamic stimuli for disgust. These findings suggest that

specific emotions are processed differently in static and dynamic form. Interestingly, the study by Adolphs and colleagues (2003) found that an individual with extensive bilateral damage to the amygdala, temporal cortical regions, orbitofrontal cortex and insula, amongst other regions, could not recognise any of the six basic emotions, except happiness, from static images of facial expressions. In contrast, the same individual could recognise all but disgust when expressed dynamically by the experimenter. This dissociation is confirmed by imaging studies comparing static and dynamic facial expressions that have shown different brain regions are preferentially active for static or dynamic images for one emotion but not another. For example, in one fMRI study, an enhanced response was observed for dynamic disgust (compared to static disgust) but not dynamic happiness (compared to static happiness) in the right middle temporal gyrus and the bilateral amygdala (Trautmann et al., 2009).

Although the behavioural findings from Chapter 3 indicated that sadness was perceived as low intensity, and that motion might facilitate recognition of sadness, there was no influence of motion on ERPs for sadness. This might indicate that any behavioural facilitation in its recognition is related to perceptuo-cognitive mechanisms occurring at a longer latency, and not due to effects on early perceptual processing.

4.4.3 Topography differences for static and dynamic stimuli

The general trend was for the P1 amplitude for static stimuli to show a stronger bias towards right ventral sites, with dynamic stimuli showing a similar but less pronounced bias. Results for the static stimuli are consistent with previous research

demonstrating a maximal P1 amplitude for static stimuli over right occipito-temporal regions (Batty & Taylor, 2003). There were differences in P1 latency for static and dynamic stimuli; with quicker P1 latencies for static compared with dynamic stimuli over lateral sites; for medial regions, dynamic stimuli were quicker than static over dorsal sites, with the reverse pattern for ventral regions. The contrasting pattern over medial dorsal and medial ventral regions suggests that the speed of processing may differ, and/or the underlying neural network may be different. The evidence of dissociable dorsal-ventral pathways for moving versus still objects fits in with this observation, as the dynamic stimuli produce a quicker response over dorsal regions, and static over ventral.

The general trend was for the N170 amplitude for static stimuli to show a stronger bias towards right lateral ventral sites, with dynamic stimuli showing a similar, but less pronounced bias. Results for the static stimuli are consistent with previous research demonstrating a maximal N170 over right lateral ventral regions for faces, reflecting neural generators in the temporal cortex for both invariant and changeable aspects of the face (Bentin et al., 1996; Itier & Taylor, 2004; Puce et al., 1998; Shibata et al., 2002). The effects of motion on topography suggest that either the same neural generators are modulated differentially or that different neural generators are activated. The fact that the dynamic response for the P1 and N170 amplitude was similar to the static stimuli distribution, but attenuated, suggests that the same neural generators may be responsible for the response observed. The observable latency differences in medial regions for the P1 may be due to processing different processing speeds along the dorsal and ventral visual streams.

4.4.4 Specificity of motion effects to social stimuli

The motion effect observed on the P1 amplitude appears to be specific to the emotional stimuli, as the opposite effect was observed for non-face stimuli, i.e. a larger amplitude for dynamic stimuli for non-face stimuli. It is not possible to conclude that this effect is exclusive to emotional stimuli alone and might be found for other social stimuli. A significant differential response was not observed for the N170 for non-face stimuli, however inspection of the means suggested that the dynamic stimuli were producing a greater response but not enough to drive an interaction. The effect observed on the P1 amplitude for non-face stimuli may reflect an increased attentional bias towards the dynamic stimuli when the emotional content is absent. Whether this effect is observed in all social stimuli or just emotional faces is unresolved. Indeed, it is not possible to conclude whether facial emotion is necessary to produce this response, or if non-emotional face stimuli would evoke the same response. Further research is necessary to ascertain the elements needed to produce the effects observed, i.e. neutral faces, eyes alone, point-light displays of the whole body in motion.

4.4.5 Conclusion, limitations, and future directions

One of the key findings is that static and dynamic emotional faces are already distinguishable from each other at P1 (100ms), indicating that they generate different neural activation patterns at very early stages of emotional face processing. This may be due to differential modulation of analogous neural generators and

networks or dissociable brain networks. The findings are suggestive of differential neural activity, however, this cannot be precisely localised as electrophysiological research is limited in spatial resolution by the nature of the summation of different neural populations. One way of elucidating whether the activity pattern observed is due to one or more networks would be to couple ERP findings with fMRI, allowing both temporal *and* spatial and properties of the activity to be accounted for. It would then be possible to ascertain whether the regional activity reported in fMRI studies is correlated with the early-latency ERP responses described in this chapter. Despite this limitation, the current data supports previous research on the topography of the P1 and N170, and response to facial stimuli, and is consistent with previous research using static images of facial expression. The early-latency differential response to static and dynamic emotional expressions suggests that the initial encoding of emotional faces/social stimuli can be further divided on the basis of invariant features and temporal aspects due to motion. There may be a greater integration of these two aspects of social stimuli, leading to an extension of the neurocognitive model of face processing postulated by Haxby and colleagues (2000). This may have implications on interpreting previous research that has based conclusions solely on the use of static emotional stimuli (Allison et al, 1999; Bentin et al, 1996; Blau et al, 2007; Narumoto et al, 2001). Previous research assessing emotion-processing deficits in clinical populations such as autism, schizophrenia, and individuals with brain lesions affecting emotion-processing domains, may have underestimated these individuals' ability to process emotions. Motion may facilitate perception of emotion in many cases where perception of static emotional images is difficult. There is already evidence that dynamic facial expressions and gait aids identification of

others based on studies in individuals with brain lesions (Adolphs et al., 2003), and prosopagnosia (Humphreys et al., 1993). There has been a great deal of research in the last few years in emotion recognition deficits in individuals with temporal lobe epilepsy, before and after temporal lobe resection. Amygdala damage is common in these individuals and deficits in fear recognition are typical. Activation of the amygdala is thought to be more pronounced for fear compared to other emotions (Adolphs, 2001), with reduction in amygdala function producing reduced activity in cortical regions associated with emotional face processing. This is interesting in light of the findings in this study that show that the P1 is enhanced for dynamic fear only, whereas the N170 is enhanced and quicker for static fear only. It may be that fear is processed differently in static and dynamic forms and this may be evident in individuals with amygdala lesions. Previous research investigating the effects of temporal lobe epilepsy on emotion perception has focused on the use of static images of facial expressions. There is little known about the impact of temporal lobe epilepsy and lobectomy on neural activity during emotion processing. In addition, little is known about how temporal lobe lesions might impact on processing moving emotional facial expressions. These questions will be addressed in the next chapter.

5. Early-latency brain processing of static and dynamic emotional expressions in patients following temporal lobectomy for paediatric-onset unilateral temporal lobe epilepsy

5.1 Introduction

Temporal lobe epilepsy (TLE) is characterised by lesions and gliosis involving medial temporal structures (Wieser, 2004; Williamson et al, 1993), and damage to the amygdala and surrounding structures is often a consequence of anteromedial temporal lobe resection as treatment for medically intractable TLE (Tellez-Zenteno, Dhar, Hernandez-Ronquillo, & Wiebe, 2007). The importance of structures in the anteromedial temporal lobes for emotion recognition has been demonstrated by findings from both lesion and functioning imaging studies (Adolphs, Damasio, Tranel, & Damasio, 1996; Adolphs et al., 2003). There is also a large volume of research reporting that the amygdala is important for the visual recognition of emotions, particularly fear, from facial expressions (Adolphs et al., 1994; Adolphs et al., 1995; Breiter et al., 1996) and this is corroborated with evidence from animal studies (Aggleton, Keith, Rawlins, Hunt, & Sahgal, 1992; Amaral et al., 2003). Consequently, research has focused on patients with TLE, pre- and post-surgery, to elucidate the influence of the temporal lobe and surrounding structures in the processing of emotional and social stimuli (Adolphs et al., 2001; Benuzzi et al., 2004b; Meletti et al., 2003; Schacher et al., 2006). Focus on patients with unilateral damage has provided a better understanding of the potential hemispheric asymmetry of amygdala function in emotion processing.

5.1.1 Emotion Recognition from Static Images Following Temporal Lobectomy

Three studies have specifically investigated emotion recognition in participants after temporal lobectomy (TL), and these have provided conflicting findings. In a study by Anderson, Spencer, Fulbright, and Phelps (2000), 23 temporal lobectomy patients (12 RTL & 11 LTL) were compared with 23 healthy controls on their intensity ratings of the facial expressions of the six basic emotions (based on Adolphs et al, 1994). They reported impaired recognition of facial expressions of emotions associated with withdrawal (e.g. disgust, fear, and sadness) in the RTL patients only. Adolphs, Tranel & Damasio (2001) used a similar experimental task to Anderson and colleagues (2000); 26 post-operative TL patients (11 RTL & 15 LTL) were compared with fifty brain-damaged controls (with no damage to the anterior temporal lobe, ventro-medial frontal lobe, or right parietal cortices). They also found that the RTL patients demonstrated an impaired recognition for negative emotions, particularly fear. Both these studies reported that an earlier age of seizure onset is correlated with a higher degree of emotion recognition deficits. Differences in emotion recognition between RTL & LTL groups in both of these two studies cannot easily be accounted for by confounds such as differences in education, general intelligence or visuoperceptual measures as these were comparable for the right and left groups. This right-sided bias for emotion processing is also illustrated in studies where patients with right mesial temporal lobe epilepsy have an impairment in recognising emotions from facial expressions even before lobectomy (Benuzzi et al, 2004; McClelland et al, 2006; Meletti et al, 2003, 2009) (discussed in more detail in Chapter 1). In contrast to

these findings, Adolphs and colleagues (1995) found no emotion deficits in either right or left post-operative patients. They compared six patients with unilateral damage to the amygdala and other cortical and subcortical regions in the anterior temporal lobe caused by temporal lobectomy or herpes simplex encephalitis (three right & three left) with ten brain damaged patients (with no damage to the amygdala) and 7 healthy control participants. The discrepant findings might be due to the heterogeneity of the populations in terms of factors such as the extent of temporal lobe damage and age of onset and duration of epilepsy before surgery; the differences in sample size; and the inconsistency of comparison groups. Crucially, the intensity rating task used for all three studies may be insensitive to subtle deficits in emotion processing, as the tasks used required identification of the six basic emotions from static pictures of intense emotional expressions. It may be that individuals can distinguish between basic emotional states relatively easily, however, find difficulty in identifying more complex social emotional states, e.g. guilt, embarrassment, particularly in the context of social interactions.

5.1.2 Emotion Recognition from Dynamic Images Following Temporal Lobectomy

There is a lack of research investigating how facial motion may influence the perception of facial expressions of emotion in patients with unilateral temporal lobectomy. This is important because studies using only static images may over-estimate the degree of deficits. This is because patients, like healthy individuals, may benefit from cues provided by dynamic images and thus may be more likely to show a normal level of performance when perceiving dynamic facial expressions. The

inclusion of dynamic stimuli to investigate deficits would allow a better understanding of emotion processing in the dynamic social world. One study conducted by Cristinzio, N'Diaye, Seeck, Vuilleumier & Sander (2010) presented 19 temporal lobectomy patients (8 RTL & 11 LTL) and 10 healthy controls with computer-generated animated faces displaying dynamic expressions (anger, fear & happiness) at low (50%) and high (100%) intensity, preceded by dynamic eye movements (straight and averted). Participants had to rate the intensity of the expression (similar to above studies), and the interaction of emotional expression and eye gaze was explored. There was a trend for a deficit in the recognition of anger and fear for both RTL & LTL patient groups.

In another study by Graham, Devinsky & LaBar (2006) computer-generated morphs of neutral-to-anger, neutral-to-fear, and fear-to-anger were presented to a patient with bilateral amygdala damage, 13 patients with unilateral temporal lobectomy (6 RTL & 7 LTL), and 15 age-matched controls. In a two-alternative forced-choice identification task, the patient with bilateral amygdala damage was found to be less sensitive to small changes in emotional intensity for both fear and anger, whereas the unilateral TL patients showed intact performance on all three morph progressions. Thus, the results of the two studies using dynamic stimuli suggest that patients may be less likely to show clear deficits. However, neither study directly compared static and dynamic facial expressions and both only included 2-3 emotional states. Dynamic stimuli may provide more structural and temporal information than static images, and perception of moving emotional images may recruit a more distributed neural network (Kilts et al., 2003; Trautmann et al., 2009) overcoming the disruption to neural structures in the temporal lobe.

5.1.3 The Role of Subcortical-Cortical Interactions

It is evident that the amygdala influences the activity of cortical structures involved in the processing of emotional information. The fusiform cortex receives prominent feedback projections from the amygdala (Amaral et al., 2003) and this could act to enhance the fusiform response to emotional faces. A study by Vuilleumier, Richardson, Armony, Driver & Dolan (2004) found increased fusiform cortex and posterior STS activation in healthy individuals and those with hippocampal damage in response to fearful compared with neutral faces. In contrast, this enhanced activation was not evident in individuals with amygdala lesions caused by medial temporal lobe sclerosis, even though visual areas were structurally intact. They found that the degree to which activation was reduced in the posterior fusiform cortex by fearful expressions was related to the amount of sclerotic amygdala tissue within the ipsilateral hemisphere. Since the fusiform cortex is one of the generators of the face-sensitive N170 ERP component, it is possible that the amygdala damage in patients who underwent temporal lobectomy for treatment of mesial temporal lobe epilepsy would result in a reduction of the typical enhancement of the N170 to fearful expressions.

5.1.4 Summary and Predictions

This study aims to address the lack of research investigating the influence of facial motion on the processing of facial expressions of the six basic emotions in unilateral

temporal lobectomy, with a direct comparison of static and dynamic facial expression. Using the ERP paradigm and comparison data from Chapter 4, the impact of unilateral temporal lobectomy on the early latency ERP components P1 & N170 will be explored. Findings from studies exploring the deficits in emotion processing in temporal lobectomy have been conflicting, as discussed above. This study aims to address the effects of motion on the early stages of emotion processing in this population, and whether this differs from a healthy population. In the next chapter, an assessment using behavioural tests emotion recognition will allow an evaluation of behavioural deficits in light of the ERP findings in this chapter.

The specific questions and hypotheses to be addressed are as follows:

1. <u>The P1/N170 response will be diminished and possibly slower in the RTL group compared with the Control and LTL group, particularly for static negative emotions:</u> This prediction is based on evidence of a right-sided bias for emotion processing, with damage to the right amygdala producing more severe deficits in emotion processing particularly for negative emotions (Adolphs et al, 2001; Anderson et al, 2000; Meletti et al, 2003, 2009). To the extent any differences reflect activation of the amygdalo-cortical network processing social information, this pattern will not be present for the non-emotional control stimuli.
2. <u>Group deficits observed may be reduced for dynamic stimuli, with the most pronounced effect observed for negative emotions:</u> Any reduction in the P1/N170 response will be less pronounced for dynamic stimuli, particularly negative emotions, resulting in more similar waveforms across groups. This prediction is based on a) studies that have shown that dynamic stimuli

increase recognition of facial expressions of emotion, and recruit a more distributed network than static emotional stimuli (Kilts et al., 2003; LaBar et al., 2003; Sato et al., 2004); b) dynamic images convey greater temporal and structural facial object properties (Harwood et al., 1999; Sato et al., 2004), which may aid recognition of facial expressions. To the extent any differences reflect activation of the amygdalo-cortical network processing social information, this pattern will be diminished or different for the non-emotional control stimuli.

3. The P1/N170 response will be diminished for fear, particularly for the RTL group: The response to fear will be reduced for the RTL group based on (a) functional imaging studies showing that the amygdala is activated disproportionately for facial expressions of fear (Breiter et al, 1996; Morris et al, 1996); and (b) patient studies showing that amygdala damage is related to a reduction in activation to fear in the fusiform gyrus, one of the generators of the N170 (Vuilleumier et al., 2004). More specifically the enhanced P1 to dynamic fear and enhanced N170 to static fear observed for typical adults (Chapter 4) may be reduced or absent in the patient groups.
4. Motion effects on the topography may differ between groups: Feedback projections from the amygdala to the ventral stream visual pathway (Amaral et al., 2003) may be disrupted in the patient group, reducing the differential P1/N170 response between dorsal and ventral regions on the scalp. To the extent any differences reflect activation of the amygdalo-cortical network processing social information, this pattern will be diminished or different for the non-emotional control stimuli.

5.2 Methods

5.2.1 Participants

Fifteen right-handed mesial temporal lobe epilepsy (MTLE) patients (9 female), aged 17-31 years (mean age 23.7, S.D. 3.81) participated in the study. This patient group was recruited from a cohort in a previous study conducted by Skirrow, C., Cross, H., Cormack, F., Vargha-Khadem, F. and Baldeweg, T. at the Institute of Child Health, UCL entitled 'Cognitive Outcome After Temporal Lobe Surgery in Childhood: A Long-Term Follow-Up Study' funded by Epilepsy Research UK and Volkswagen Stiftung.

Patients were recruited on the basis of having undergone surgical resection for intractable temporal lobe epilepsy as children at Great Ormond Street Hospital for Children. Patients had either unilateral hippocampal sclerosis (HS) or dysembryoplastic neuroepithelial tumour (DNET) before surgery (predominantly the anterior lobe). Participants had single focal neurosurgical lesions confined to one side of the anterior temporal lobe, and none were taking antiepileptic medications at the time of testing. Table 5.1 below summarises the general clinical features of individuals within the two patient groups. All participants (controls and patients) had normal or corrected–to-normal visual acuity, with no recorded medical problems associated with vision.

Table 5.1 Clinical features of the two patient groups

Subject	Gender	Age at testing (years)	Pathology	Age at epilepsy onset (months)	Age at surgery (months)	Vol. of resection (cm)
RTLE (M & S.D.)				62.83 ±55.05	175.56 ±30.15	15.89 ±9.59
1	F	29	RHS	10	202	18.4
2	M	26	RHS	9.5	190	15.2
3	F	26	RHS	18	134	12.4
4	M	25	RHS	48	170	28.1
5	F	18	RHS	52	131	8.9
6	F	26	RDNET	108	222	30.4
7	M	19	RDNET	36	163	20.7
8	M	23	RDNET	120	193	7.9
9	F	20	RDNET	164	175	1.0
LTLE (M & S.D.)				24.83 ±21.19	164.00 ±59.09	14.32 ±3.72
10	M	26	LHS	10	199	17.3
11	F	25	LHS	60	144	18.3
12	M	31	LHS	9	206	15.8
13	F	27	LHS	12	203	13.0
14	F	19	LDNET	16	53	13.5
15	F	24	LDNET	42	179	8.0

Key: F – female, M – male, RHS – right-sided hippocampal sclerosis, RDNET – right-sided dysembryoplastic neuroepithelial tumour, LHS – left-sided hippocampal sclerosis, LDNET – left-sided dysembryoplastic neuroepithelial tumour

There was access to thirty-four patients from the previous study (Skirrow et al.) who were within the inclusion criteria. Of those not included in this study, eleven were not willing to participate and five could not be contacted. Three patients took part in the study, but were excluded from the final analyses due to lack of sufficient data because of artefacts in the EEG (two LHS cases and one RDNET case). The patients were provided with compensation for travel and accommodation costs necessary for

participation in the study. Table 5.2 shows the comparison between the included and excluded groups.

Table 5.2 A comparison between included and excluded individuals

	Gender	Clinical Group	Age at epilepsy onset (Mean & S.D.)	Age at surgery (Mean & S.D)	Vol. of resection (mm)
Patients Included	6 Male 9 Female	5 RHS 4 RDNET 4 LHS 2 LDNET	46.83 ±47.45	172.87 ±41.77	14.79 ±7.64
Patients Excluded	10 Male 9 Female	4 RHS 3 RDNET 6 LHS 6 LDNET	55.16 ±48.91	155.26 ±31.38	16.34 ±6.58

Key: F – female, M – male, RHS – right-sided hippocampal sclerosis, RDNET – right-sided dysembryoplastic neuroepithelial tumour, LHS – left-sided hippocampal sclerosis, LDNET – left-sided dysembryoplastic neuroepithelial tumour

The TL patients were compared to fifteen healthy control participants (9 female), aged 17-31 years (mean age 23.3, S.D. 3.48). This was a subset of the participants from Chapter 4, matched with the patient group on age and gender. All self-reported as having no extraneous neurological or psychological disorder. Participants were recruited through advertisement at University College London, and each participant received payment for their participation (£10).

The two groups were matched for gender and age. An independent samples t-test revealed no significant difference between the ages of the participants in the two groups, $t(1, 27) = 0.85$, $p = 0.403$. Handedness was assessed by means of the Edinburgh Handedness Inventory (Oldfield, 1971), and laterality quotients were

recorded for the Control group, mean = 85.4, S.D. = 6.51; the RTL group, mean = 82.6, S.D. = 4.59; and the LTL group, mean = 84.5, S.D. = 6.16. There were no recorded left-handed participants.

The study was approved by ICH/GOSH Research Ethics Committee (REC reference 07/Q0508/35), and performed according to the standards of the Declaration of Helsinki (1964). Each individual provided informed written consent prior to participation in the study.

5.2.2 Materials

As in Chapter 4 (section 4.2.2) static and dynamic images of the six basic expressions displayed by four models (2 female, 2 male) were used, with an additional colour photograph of a flower and dynamic image of a flower opening.

5.2.3 Experimental Task

As in Chapter 4 (section 4.2.3).

5.2.4 Statistical Analyses

As in Chapter 2, also included Group as a between-subjects factor.

The channel groupings are those discussed in Chapter 2, section 2.1.5. The P1 peak was defined as the most positive peak in the time-window ranging from 83ms-163ms post-stimulus onset. The N170 peak was defined as the most negative peak in the time-window ranging from 119-215ms post-stimulus onset.

There were 48 trials per condition presented to each participant (2 motion x 7 emotion conditions). For the final 9 RTL patients, the mean percentage of original trials retained after ERP derivation (across participants) was 91.3% (S.D. = 5.22, range 40-47 trials), 89.7% (S.D. = 5.89, range 39-47 trials), 91.9% (S.D. = 4.65, range 41-47 trials), 91.6% (S.D. = 4.85, range 41-47 trials), 92.1% (S.D. = 4.70, range 41-47 trials), 91.3% (S.D. = 4.66, range 41-47 trials), and 91.6% (S.D. = 5.48, range 40-47 trials) for the Static conditions Anger, Disgust, Fear, Happiness, Sadness, Surprise, and Non-Emotional (Target) respectively. Also, 92.1% (S.D. = 4.37, range 41-47 trials), 91.1% (S.D. = 5.04, range 40-47 trials), 91.8% (S.D. = 4.71, range 41-47 trials), 91.7% (S.D. = 5.15, range 40-47 trials), 90.9% (S.D. = 5.04, range 40-47 trials), 91.6% (S.D. = 5.15, range 41-47 trials), and 91.3% (S.D. = 4.87, range 41-47 trials) for the Dynamic conditions Anger, Disgust, Fear, Happiness, Sadness, Surprise, and Non-Emotional (Target) respectively.

For the final 6 LTL patients, the mean percentage of original trials retained after ERP derivation (across participants) was 92.8% (S.D. = 4.12, range 41-46 trials), 87.7% (S.D. = 3.27, range 40-44 trials), 90.2% (S.D. = 4.58, range 40-46 trials), 91.8% (S.D. = 4.49, range 41-46 trials), 92.5% (S.D. = 4.64, range 41-47 trials), 93.2% (S.D. = 4.49, range 41-47 trials), and 90.8% (S.D. = 4.49, range 40-46 trials) for the Static conditions Anger, Disgust, Fear, Happiness, Sadness, Surprise, and Non-Emotional (Target) respectively. Also, 91.0% (S.D. = 4.98, range 41-46 trials), 90.2% (S.D. = 5.95, range 38-45 trials), 91.7% (S.D. = 3.82, range 41-45 trials), 91.5% (S.D. = 5.58, range 40-47 trials), 92.3% (S.D. = 1.97, range 43-45 trials), 90.8% (S.D. = 5.08, range 39-45 trials), and 90.5% (S.D. = 5.05, range 39-45 trials) for the Dynamic conditions Anger, Disgust, Fear, Happiness, Sadness, Surprise, and Non-Emotional (Target) respectively.

For the final 15 Control participants, the mean percentage of original trials retained after ERP derivation (across participants) was 92.3% (S.D. = 4.27, range 39-47 trials), 91.5% (S.D. = 4.88, range 39-47 trials), 90.6% (S.D. = 5.08, range 39-47 trials), 91.8% (S.D. = 4.74, range 40-47 trials), 91.1% (S.D. = 4.25, range 41-47 trials), 92.3% (S.D. = 4.42, range 40-47 trials), and 91.6% (S.D. = 4.37, range 40-47 trials) for the Static conditions Anger, Disgust, Fear, Happiness, Sadness, Surprise, and Non-Emotional (Target) respectively. Also, 91.6% (S.D. = 5.10, range 41-47 trials), 91.2% (S.D. = 4.07, range 40-47 trials), 91.3% (S.D. = 4.06, range 40-47 trials), 91.5% (S.D. = 4.91, range 39-47 trials), 92.4% (S.D. = 4.63, range 40-47 trials), 91.0% (S.D. = 4.93, range 40-47 trials), and 90.7% (S.D. = 4.06, range 41-47 trials) for the Dynamic conditions Anger, Disgust, Fear, Happiness, Sadness, Surprise, and Non-Emotional (Target) respectively.

Subsequent to the ERP task, each participant was asked to complete a set of behavioural tasks: the Matrix Reasoning subtest from the WAIS-III; the Florida Affect Battery (FAB); the Empathy Quotient (EQ); and the Social Functioning Scale (SFS). Refer to Chapter 6 for details.

5.3 Results

The results will be presented by first reporting the general effects of Motion and Emotion, then the analyses relevant to the specific predictions, and then any additional significant effects.

5.3.1 P1

The P1 was bilateral and largest over medial ventral sites, and quickest over lateral ventral sites.

5.3.1.1 P1 Amplitude

5.3.1.1.1 General effects

There was a significant main effect of Motion across groups, $F(1, 27) = 17.13$, $p < 0.0005$, partial $\eta^2 = 0.39$, with a significantly larger amplitude in the static condition compared with the dynamic condition (difference of 0.26μV). There was no main effect of Emotion.

5.3.1.1.2 Prediction 1: The P1 will be diminished in the RTL group compared with the Control and LTL groups, particularly for negative emotions

There was no main effect of Group, $F(2, 27) = 0.88$, $p = 0.43$, partial $\eta^2 = 0.06$, indicating that the P1 was of similar amplitude across the groups. Also, refer to Figure 5.6 at the end of the results section.

In order to investigate more specifically whether the RTL group showed a diminished response over the right lateral ventral regions where the P1 is typically largest, and whether this was more pronounced for negative emotions, an Emotion (Positive, Negative) by Group (Control, RTL, LTL) ANOVA was computed for just these electrodes for static images. The negative emotions included were anger, disgust, fear, and sadness; the inclusion of these negative emotions in is line with studies

finding recognition deficits for all these emotions in patients with temporal lobe epilepsy, and temporal lobectomy (Adolphs et al, 2001; Anderson et al, 2000; Meletti et al, 2003, 2009). Happiness was implemented as the positive emotion in this analysis; surprise was not included as it can be perceived as either positive (e.g., surprised by a lovely party) or negative (e.g., surprised to find a spider on your arm). However, there was no significant interaction between Emotion and Group, $F(2, 27) = 0.37$, $p = 0.69$.

5.3.1.1.3 Prediction 2: Patient group deficits observed may be reduced for dynamic stimuli, with the most pronounced effect seen for negative emotions

There was a trend towards a significant two-way interaction of Motion by Group, $F(2, 27) = 3.07$, $p = 0.06$, partial $\eta^2 = 0.19$. Inspection of the means suggests that this was driven by a larger amplitude for the static condition compared with the dynamic condition for the Control and RTL groups but not LTL group. Mean peak amplitude and SD values are shown in Table 5.3 below. Also, refer to Figure 5.6 at the end of the results section.

Table 5.3 Mean peak P1 amplitude (μV) & S.D. values for the static & dynamic conditions across groups

	Group					
	Controls (n = 15)		RTL (n = 9)		LTL (n = 6)	
	M	(S.D)	M	(S.D.)	M	(S.D.)
Static	3.30	(0.82)	3.71	(1.22)	2.95	(0.96)
Dynamic	2.91	(0.75)	3.34	(1.34)	2.89	(0.80)

There was a significant three-way interaction of Motion by Emotion by Group, F(10, 135) = 2.37, $p < 0.05$, partial $\eta^2 = 0.15$. Further analysis revealed a significant two-way interaction of Motion by Group for disgust, F(2, 27) = 4.90, $p < 0.01$, partial $\eta^2 = 0.27$; and happiness, F(2, 27) = 5.85, $p < 0.01$, partial $\eta^2 = 0.30$. Post-hoc analysis revealed no further significant group differences in the amplitude for either the static or dynamic condition for either disgust or happiness. When comparing the static and dynamic conditions within the groups for disgust, the static condition produced a significantly larger amplitude than the dynamic condition for the Control group, t(1, 14) = 7.30, $p < 0.0005$ (difference of 0.71μV) but neither patient groups. For happiness, the static condition produced a significantly larger amplitude than the dynamic condition for the Control group, t(1, 14) = 5.88, $p < 0.0005$ (difference of 0.50μV); and a trend towards significance for the RTL group, t(1, 8) = 2.11, p = 0.068 (difference of 0.38μV) but not the LTL group, as illustrated in Figure 5.1 below. In contrast, the dynamic condition produced a larger amplitude than the static condition for both emotions for the LTL group, though this did not reach significance. Mean peak amplitude and S.D. values are shown in Table 5.4 below.

Table 5.4 Mean peak P1 amplitude (μV) & S.D. values for motion conditions for the emotions disgust & happiness across groups

	Group					
	Controls (n = 15)		RTL (n = 9)		LTL (n = 6)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Disgust Static	3.46	(0.87)	3.68	(1.32)	2.91	(1.14)
Disgust Dynamic	2.75	(0.75)	3.43	(1.50)	3.03	(0.80)
Happiness Static	3.29	(0.72)	3.79	(1.38)	2.74	(0.83)
Happiness Dynamic	2.79	(0.80)	3.41	(1.49)	3.15	(1.37)

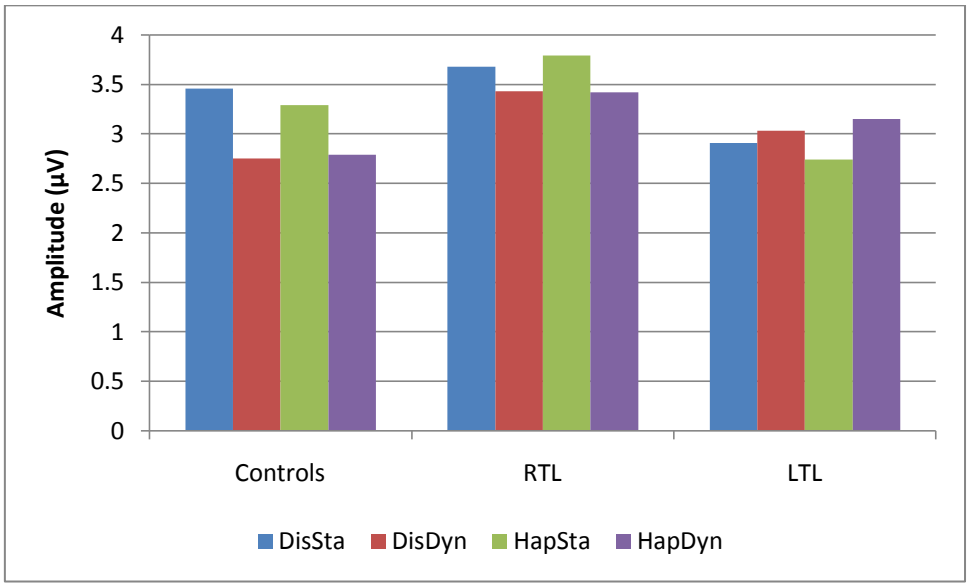


Figure 5.1 Mean peak P1 amplitude values for the motion conditions for disgust and happiness between groups

Key: DisSta = Disgust, Static; DisDyn = Disgust, Dynamic; HapSta = Happy, Static; HapDyn = Happy, Dynamic

In summary, there was no overall difference in the P1 amplitude by group. However, the influence of motion differed by group and emotion. The static condition produced a larger amplitude than the dynamic condition for the Control and RTL groups; and this was particularly apparent for the emotions disgust and happiness. By contrast, the dynamic condition tended to produce a larger amplitude than the static condition for both emotions for the LTL group.

5.3.1.1.4 Prediction 3: The P1 will be diminished for fear for the RTL group

A targeted analysis was performed over right lateral ventral sites for fear, as this is the region where the most pronounced early-latency differences between fear and other emotions have been observed in healthy adult populations. Results showed that there was no difference in fear response between the three groups.

When fear was compared with other emotions within each group, there was a significantly larger amplitude for other dynamic emotions compared with dynamic fear for the LTL group only, $t(1, 5) = 4.63$, $p < 0.01$ (difference of 0.49μV). Fear was also compared with happiness; there was a significantly larger amplitude for dynamic happiness compared with dynamic fear for the LTL group only, $t(1, 5) = 3.67$, $p < 0.05$ (difference of 0.80μV). There were no other significant differences within the groups. Mean peak amplitude and S.D. values are shown in Table 5.5 below.

Table 5.5 Mean peak P1 amplitude (μV) & S.D. values for fear, happiness, and other emotions for the two motion conditions

	Group					
	Controls (n = 15)		RTL (n = 9)		LTL (n = 6)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Fear	3.45	(1.41)	3.66	(0.60)	3.38	(1.38)
Fear Static	3.79	(1.42)	3.80	(0.66)	3.75	(1.29)
Fear Dynamic	3.12	(1.49)	3.53	(1.00)	3.01	(1.54)
Happiness	3.34	(1.47)	3.71	(0.73)	3.59	(1.49)
Happiness Static	3.76	(1.77)	3.81	(0.66)	3.37	(1.01)
Happiness Dynamic	2.92	(1.25)	3.62	(1.26)	3.81	(1.94)
Other Emotions	3.51	(1.53)	3.73	(0.52)	3.52	(1.24)
Other Emotions Static	3.87	(1.67)	4.02	(0.58)	3.55	(1.08)
Other Emotions Dynamic	3.16	(1.42)	3.45	(0.79)	3.50	(1.42)

5.3.1.1.5 Prediction 4: Motion effects on the topography may differ between groups

There was no Dorsal-Ventral Topography by Motion by Group interactions; or any other Topography by Motion by Group interactions.

5.3.1.1.6 Are these group differences specific to social stimuli?

To determine whether the effect of motion on the P1 was specific to social stimuli, an ANOVA was computed with Stimulus (Emotional, Non-Emotional) by Motion (Static, Dynamic) by Group (Control, RTL, LTL) as the variables over all electrodes. The interaction did not reach significance, $F(2, 27) = 0.69$, $p = 0.51$.

5.3.1.1.7 P1 Amplitude Summary

In summary, there was no overall difference in the P1 amplitude by group. However, the influence of motion differed by group and emotion. The static condition produced a larger amplitude than the dynamic condition for the Control and RTL groups; and this was particularly apparent for the emotions disgust and happiness. By contrast, the dynamic condition tended to produce a larger amplitude than the static condition for both emotions for the LTL group. Analysis targeted at right lateral ventral sites showed a smaller P1 to fear compared with other emotions in the dynamic but not static condition for the LTL group.

5.3.1.2 P1 Latency

5.3.1.2.1 General effects

There was a significant main effect of Motion, $F(1, 27) = 5.52$, $p < 0.05$, partial $\eta^2 = 0.17$, with the static condition producing a significantly shorter latency compared with the dynamic condition (difference of 1.08ms). There was no main effect of Emotion.

5.3.1.2.2 Prediction 1: The P1 will be slower in the RTL group compared with the Control and LTL groups, particularly for negative emotions

There was no main effect of Group, $F(2, 27) = 0.92$, $p = 0.41$, partial $\eta^2 = 0.06$, indicating that the P1 was of similar latency across the groups. In order to investigate

more specifically whether the RTL group showed a different response over the right lateral ventral regions where the P1 is typically quickest and whether this was more pronounced for negative emotions, an Emotion (Positive, Negative) by Group (Control, RTL, LTL) ANOVA was computed for just these electrodes for static images. There was no significant interaction between Emotion and Group, $F(2, 27) = 0.42$, $p = 0.66$.

5.3.1.2.3 Prediction 2: Patient group deficits observed may be reduced for dynamic stimuli, with the most pronounced effect seen for negative emotions

There was a significant three-way interaction of Motion by Emotion by Group, $F(10, 135) = 2.24$, $p < 0.05$, partial $\eta^2 = 0.14$. Further analysis revealed a significant two-way interaction of Motion by Group for anger, $F(2, 27) = 3.96$, $p < 0.05$, partial $\eta^2 = 0.23$; and disgust, $F(2, 27) = 3.37$, $p < 0.05$, partial $\eta^2 = 0.20$. Post-hoc analysis revealed that there were no significant group differences in the latency in either the static or dynamic condition for either anger or disgust. When comparing the static and dynamic conditions within the groups for anger, the static condition produced a significantly shorter latency than the dynamic condition for the LTL group only, $t(1, 5) = 3.87$, $p < 0.01$ (difference of 5.42ms). For disgust, the dynamic condition produced a significantly shorter latency than the static condition for the Control group only, $t(1, 14) = 2.16$, $p < 0.05$ (difference of 2.64ms), as illustrated in Figure 5.2 below. Mean peak latency and S.D. values are shown below in Table 5.6 below.

Table 5.6 Mean peak P1 latency (ms) & S.D. values for static & dynamic conditions for the emotions anger and disgust across groups

	Group					
	Controls (n = 15)		RTL (n = 9)		LTL (n = 6)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Anger Static	115.10	(5.32)	118.96	(6.54)	113.56	(11.45)
Anger Dynamic	115.97	(4.91)	116.95	(9.53)	118.98	(10.25)
Disgust Static	115.57	(5.43)	118.21	(8.14)	115.43	(6.90)
Disgust Dynamic	112.93	(5.80)	116.93	(6.87)	117.06	(12.67)

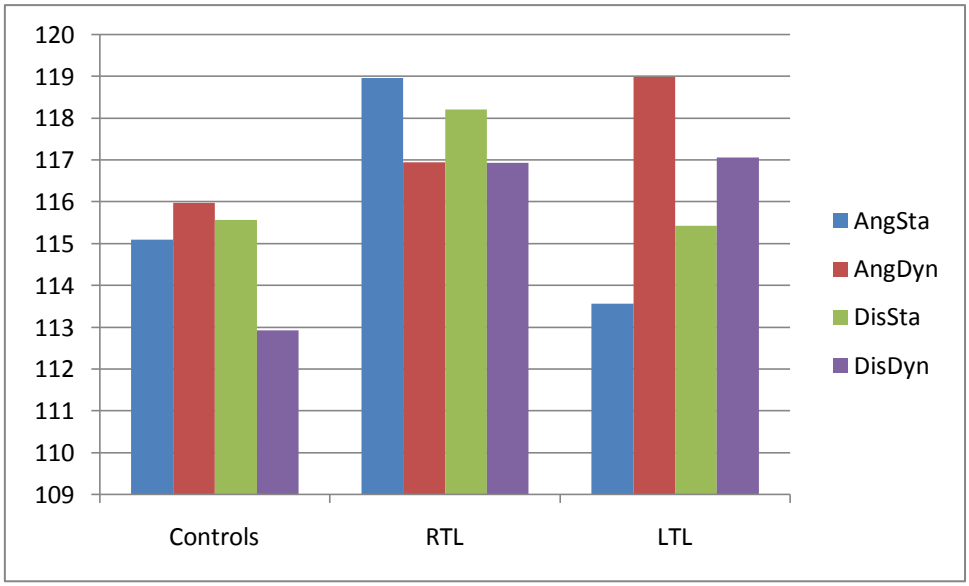


Figure 5.2 Mean peak P1 latency values for the motion conditions for anger and disgust across groups

Key: AngSta = Anger, Static; AngDyn = Anger, Dynamic; DisSta = Disgust, Static; DisDyn = Disgust, Dynamic

5.3.1.2.4 Prediction 3: The P1 will be slower for fear for the RTL group

A targeted analysis was performed over right lateral ventral sites for fear, as this is the region where the most pronounced early-latency differences between fear and

other emotions have been observed in healthy adult populations. Results showed that there was no significant difference in fear response between the three groups. Fear was compared with other emotions, and happiness, within each group. Results showed that there was no significant difference in response between emotions for the three groups.

5.3.1.2.5 Prediction 4: Motion effects on the topography may differ between groups

There was no Dorsal-Ventral by Motion by Group interactions, or any other Topography by Motion by Group interactions.

5.3.1.2.6 Are these group differences specific to social stimuli?

To determine whether the effect of motion on the P1 was specific to social stimuli, an ANOVA was computed with Stimulus (Emotional, Non-Emotional) by Motion (Static, Dynamic) by Group (Control, RTL, LTL) as the variables over all electrodes. The interaction did not reach significance, $F(2, 27) = 0.62$, $p = 0.55$.

5.3.1.2.7 P1 Latency Summary

In summary, there was no overall difference in the P1 latency by group. However, the influence of motion differed by group and emotion. The static condition produced a quicker P1 than the dynamic condition for the LTL group for anger; and the dynamic condition produced a quicker P1 than the static condition for the

Control group for disgust. Analysis targeted at right lateral ventral sites showed no difference between fear and other emotions for any of the groups.

5.3.1.2.8 P1 Amplitude and Latency Summary

The influence of motion on both the P1 amplitude and latency differed by group and emotion. The static condition produced a larger P1 amplitude than the dynamic condition for the Control and RTL groups, and this was particularly apparent for the emotions disgust and happiness. By contrast, the dynamic condition tended to produce a larger amplitude than the static condition for both emotions for the LTL group. There was a smaller P1 amplitude for fear compared with other emotions in the dynamic but not static condition for the LTL group, but no difference in the P1 latency over right lateral ventral sites for any of the groups. The static condition produced a quicker P1 than the dynamic condition for anger in the LTL group; and the dynamic condition produced a quicker P1 than the static condition for disgust in the Control group.

5.3.2 N170

The N170 was largest over right lateral ventral sites, and quickest over the left hemisphere.

5.3.2.1 N170 Amplitude

5.3.2.1.1 General effects

There was a significant main effect of Motion, $F(1, 27) = 57.52$, $p < 0.0005$, partial $\eta^2 = 0.68$, with a significantly larger amplitude in the static condition compared with the dynamic condition (difference of 0.47μV); and a significant main effect of Emotion, $F(5, 135) = 2.71$, $p < 0.05$, partial $\eta^2 = 0.09$, with surprise producing the largest amplitude and happiness the smallest, as shown in Table 5.7 below. There were significant differences in amplitude between happiness and surprise, $t(1, 29) = 2.65$, $p < 0.01$ (difference of 0.19μV); happiness and fear, $t(1, 29) = 2.56$, $p < 0.05$ (difference of 0.17μV); happiness and disgust, $t(1, 29) = 2.05$, $p < 0.05$ (difference of 0.13μV); sadness and surprise, $t(1, 29) = 2.70$, $p < 0.01$ (difference of 0.18μV); and sadness and fear, $t(1, 29) = 2.51$, $p < 0.01$ (difference of 0.16μV). However, none of these differences survived Bonferroni correction for multiple comparisons. Mean peak amplitude and S.D. values are shown in Table 5.7 below.

Table 5.7 Mean peak N170 amplitude & S.D. values for the six emotions

Emotion	Mean (μV)	(S.D.)
Surprise	-2.42	(0.80)
Fear	-2.40	(0.86)
Disgust	-2.36	(0.87)
Anger	-2.30	(0.88)
Sadness	-2.24	(0.80)
Happiness	-2.23	(0.78)

Thus, consistent with the analysis reported in Chapter 4 in normally developed adults, the N170 was larger for static than dynamic stimuli, and tended to be larger for surprise/fear and smallest for happy.

5.3.2.1.2 Prediction 1: The N170 will be diminished in the RTL group compared with the Control and LTL groups, particularly for negative emotions

There was no main effect of Group, $F(2, 27) = 0.87$, $p = 0.43$, partial $\eta^2 = 0.06$, indicating that the N170 was of similar size across the groups. In order to investigate more specifically whether RTL patients showed a diminished response over the right lateral ventral regions where the N170 is typically largest and whether this was more pronounced for negative emotions, an Emotion (Positive, Negative) by Group (Control, RTL, LTL) ANOVA was computed for just these electrodes for static images. There was no significant interaction between Emotion and Group, $F(2, 27) = 1.91$, $p = 0.17$.

5.3.2.1.3 Prediction 2: Patient group deficits observed may be reduced for dynamic stimuli, with the most pronounced effect seen for negative emotions

There was no Motion by Emotion by Group interaction in the overall ANOVA, and as there was no reduction observed in the target analysis of N170 response to static negative emotions reported above, no further analyses were conducted for this prediction.

5.3.2.1.4 Prediction 3: The N170 will be diminished for fear for the RTL group

Prior research has indicated that there are specific deficits in fear for temporal lobectomy patients, particularly the right-sided cases. A targeted analysis was performed over right lateral ventral sites for fear, as this is the region where the most pronounced effects on the N170 have been observed in healthy adult populations. The RTL group produced a significantly larger amplitude than the LTL for dynamic fear, $t(1, 13) = 2.16$, $p < 0.05$ (difference of 1.51μV), there were no other significant differences between groups for either motion conditions.

Fear was compared with other emotions within each group over right lateral ventral regions: there was a significantly larger amplitude for static fear compared with other static emotions combined for the Control group only, $t(1, 14) = 2.42$, $p < 0.05$ (difference of 0.43μV), and for dynamic fear compared with other dynamic emotions for the RTL group only, $t(1, 8) = 2.34$, $p < 0.05$ (difference of 0.63μV). Fear was also compared with happiness: there was a significantly larger amplitude for static fear compared with static happiness for both the Control group, $t(1, 14) = 2.32$, $p < 0.05$ (difference of 0.34μV), and the RTL group, $t(1, 8) = 3.06$, $p < 0.05$ (difference of 0.69μV). There was also a significantly larger amplitude for dynamic fear compared with dynamic happiness for the RTL group, $t(1, 8) = 2.45$, $p < 0.05$ (difference of 0.66μV), and a larger amplitude for dynamic happiness compared with dynamic fear for LTL group, $t(1, 5) = 2.44$, $p < 0.05$ (difference of 0.57μV). Mean peak amplitude and S.D. values are shown in Table 5.8 below.

Table 5.8 Mean peak N170 amplitude (μV) & S.D. values for fear, happiness, and other emotions for the two motion conditions

	Group					
	Controls (n = 15)		RTL (n = 9)		LTL (n = 6)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Fear Static	-4.29	(1.97)	-4.13	(1.73)	-3.57	(1.97)
Fear Dynamic	-3.02	(1.86)	-3.58	(1.45)	-2.07	(1.11)
Happiness Static	-3.95	(2.04)	-3.44	(1.25)	-2.40	(0.63)
Happiness Dynamic	-2.81	(1.04)	-2.92	(1.34)	-2.64	(1.62)
Other Emotions Static	-3.86	(1.75)	-3.73	(1.10)	-2.89	(1.15)
Other Emotions Dynamic	-2.90	(1.56)	-2.95	(1.05)	-2.65	(1.54)

5.3.2.1.5 Prediction 4: Motion effects on the topography may differ between groups

There were no Dorsal-Ventral Topography by Motion by Group interactions, however, there was a trend towards a four-way interaction of Motion by Dorsal-Ventral Topography by Hemisphere by Group, $F(2, 27) = 3.28$, $p = 0.053$, partial $\eta^2 = 0.20$. Inspection of the means suggests that this was driven by a smaller amplitude for the RTL group compared with the Control and LTL groups for static stimuli, and a smaller amplitude for the RTL & LTL groups compared with the Control group for dynamic stimuli, both in left ventral regions, as illustrated in Figure 5.3 below. Mean peak amplitude and S.D. values are shown in Table 5.9 below.

Table 5.9 Mean peak N170 amplitude (µV) & S.D. values for dorsal-ventral topography for the motion conditions in the right and left hemispheres between groups

	Controls (n = 15)		RTL (n = 9)		LTL (n = 6)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Dorsal Right Static	-2.40	(0.75)	-2.55	(0.45)	-1.94	(0.67)
Dorsal Right Dynamic	-1.86	(0.67)	-2.13	(0.72)	-1.59	(0.60)
Dorsal Left Static	-1.89	(0.72)	-1.81	(0.75)	-1.41	(0.55)
Dorsal Left Dynamic	-1.58	(0.51)	-1.61	(0.52)	-1.40	(0.70)
Ventral Right Static	-3.72	(1.49)	-3.45	(1.04)	-2.93	(1.37)
Ventral Right Dynamic	-2.79	(1.20)	-2.64	(0.87)	-2.48	(1.44)
Ventral Left Static	-3.15	(1.42)	-2.63	(1.03)	-3.28	(1.25)
Ventral Left Dynamic	-2.49	(1.30)	-1.97	(0.77)	-1.79	(0.88)

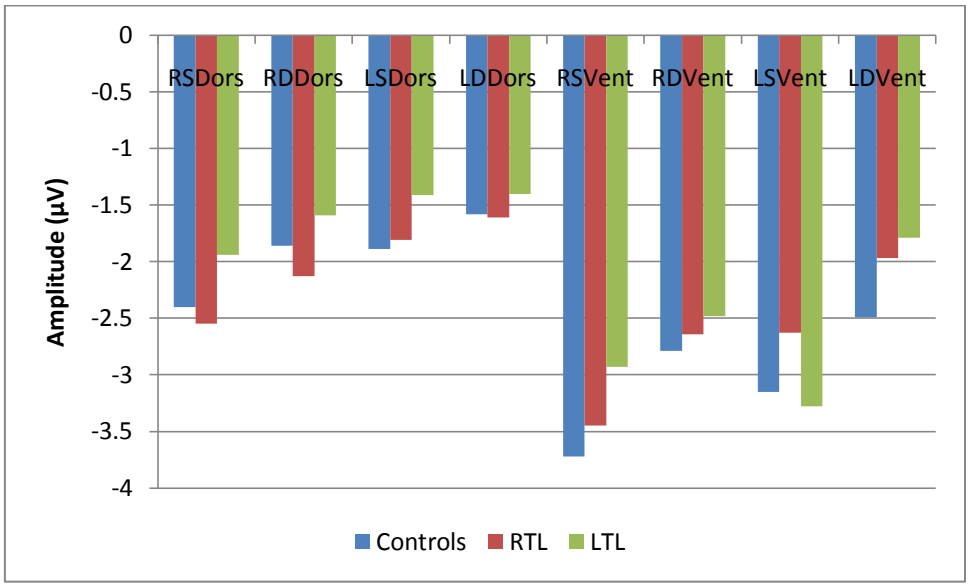


Figure 5.3 Mean peak N170 amplitude values for dorsal-ventral topography for the motion conditions in the right and left hemispheres across groups.

Key: RSDors = Right, Static, Dorsal; RDDors = Right Dynamic Dorsal; LSDors = Left, Static, Dorsal; LDDors = Left, Dynamic Dorsal; RSVent = Right, Static, Ventral; RDVent = Right, Dynamic, Ventral; LSVent = Left, Static, Ventral; LDVent = Left, Dynamic, Ventral

5.3.2.1.6 Other Effects

While there were only limited differences in Dorsal-Ventral Topography by Group, there were significant interactions involving Lateral-medial Topography.

A significant three-way interaction was observed of Motion by Lateral-Medial Topography by Group, $F(2, 27) = 3.81$, $p < 0.05$, partial $\eta^2 = 0.22$. Further analysis revealed that there was a significant two-way interaction of Motion by Group over lateral sites only, $F(2, 27) = 4.14$, $p < 0.05$, partial $\eta^2 = 0.24$. Post-hoc analysis revealed that there were, however, no significant group differences in the amplitude for either the static or dynamic condition over lateral sites. When comparing the motion conditions within groups over lateral sites, the static condition produced a significantly larger amplitude than the dynamic condition for the Control, $t(1, 14) = 7.68$, $p < 0.0005$ (difference of 0.75μV); and RTL groups only, $t(1, 8) = 2.62$, $p < 0.05$ (difference of 0.44μV). The difference between the static and dynamic condition was largest for the Control group, and smallest for the LTL group. Mean peak amplitude and S.D. values are shown in Table 5.10 below.

Table 5.10 Mean peak N170 amplitude (μV) & S.D. values for static & dynamic conditions over lateral sites across groups

	Group					
	Controls (n = 15)		RTL (n = 9)		LTL (n = 6)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Lateral Static	-3.08	(1.04)	-2.97	(0.97)	-2.23	(0.99)
Lateral Dynamic	-2.33	(0.92)	-2.53	(0.79)	-2.02	(1.18)

A significant four-way interaction of Motion by Emotion by Lateral-Medial Topography by Group was observed, $F(10, 135) = 2.92$, $p < 0.005$, partial $\eta^2 = 0.18$. Further analysis revealed that there was a significant three-way interaction of Motion by Lateral-Medial Topography by Group for emotions fear, $F(2, 27) = 3.72$, $p < 0.05$, partial $\eta^2 = 0.22$, and happiness, $F(2, 27) = 11.87$, $p < 0.0005$, partial $\eta^2 = 0.47$, and a trend towards significance for surprise, $F(2, 27) = 3.22$, $p = 0.056$, partial $\eta^2 = 0.19$. There was a significant interaction of Motion by Group in lateral sites only for fear, $F(2, 27) = 4.71$, $p < 0.01$, partial $\eta^2 = 0.26$; a significant interaction in both lateral, $F(2,27) = 5.25$, $p < 0.01$, partial $\eta^2 = 0.28$, and medial regions, $F(2,27) = 5.22$, $p < 0.01$, partial $\eta^2 = 0.28$, for happiness, however the interaction did not reach significance for surprise.

Post-hoc analysis revealed a trend towards a significantly larger amplitude for the Control group compared with the LTL group for the static condition in lateral sites for fear, $t(1, 19) = 2.05$, $p = 0.055$ (difference of 1.19μV). Also, there was a trend towards a significantly larger amplitude for the RTL compared with the LTL group for the dynamic condition in lateral sites for fear, $t(1, 13) = 2.13$, $p = 0.053$ (difference of 1.08μV). Comparing the static and dynamic conditions within groups for fear over lateral sites, the static condition produced a significantly larger amplitude than the dynamic condition for both the Control, $t(1,14) = 5.90$, $p < 0.0005$ (difference of 1.08μV), and LTL groups only, $t(1,5) = 4.08$, $p < 0.01$ (0.42μV).

For the static condition in lateral sites for happiness, there was a significantly larger amplitude for the Control compared with the LTL group, $t(1, 19) = 2.47$, $p < 0.05$ (difference of 1.16μV); and for the RTL compared with the LTL group, $t(1, 13) = 2.25$, $p < 0.05$ (difference of 1.08μV). For the dynamic condition in medial sites for

happiness, there was a significantly larger amplitude for the Control group compared with the RTL group, $t(1, 22) = 3.13$, $p < 0.005$ (difference of 0.99μV); and for the Control compared with the LTL group, $t(1, 19) = 2.11$, $p < 0.05$ (difference of 0.91μV). When comparing the static and dynamic conditions within groups for happiness over lateral sites, the static condition produced a significantly larger amplitude than the dynamic condition for both the Control, $t(1,14) = 4.87$, $p < 0.0005$ (difference of 0.74μV), and RTL groups only, $t(1,8) = 2.59$, $p < 0.05$ (difference of 0.68μV). When comparing the static and dynamic conditions within the groups for happiness over medial sites, the static condition produced a significantly larger amplitude than the dynamic condition for the RTL group only, $t(1, 8) = 3.33$, $p < 0.01$ (difference of 0.62μV).

In summary, there was a significantly smaller amplitude for the RTL and LTL groups compared with the Control group over medial sites for dynamic happiness and, in addition, a smaller amplitude for the LTL group compared with the two other groups over lateral sites for both static fear and static happiness. The difference in N170 amplitude between the static and dynamic conditions was largest for the Control group and smallest for the RTL group, both over lateral sites for fear, as illustrated in Figure 5.4 below. Mean peak amplitude and S.D. values are shown in Table 5.11 below.

Table 5.11 Mean peak N170 amplitude (μV) & S.D. values for motion conditions over lateral-medial sites for the emotions fear and happiness across groups

	Group					
	Controls (n = 15)		RTL (n = 9)		LTL (n = 6)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Fear Lateral Static	-3.39	(1.28)	-2.95	(1.29)	-2.20	(0.93)
Fear Lateral Dynamic	-2.31	(1.08)	-2.86	(0.97)	-1.78	(0.93)
Happiness Lateral Static	-3.06	(1.07)	-2.98	(1.06)	-1.90	(0.64)
Happiness Lateral Dynamic	-2.32	(0.79)	-2.30	(0.74)	-2.27	(1.47)
Happiness Medial Static	-2.12	(0.95)	-1.92	(0.86)	-1.83	(1.30)
Happiness Medial Dynamic	-2.29	(0.80)	-1.30	(0.65)	-1.38	(1.11)

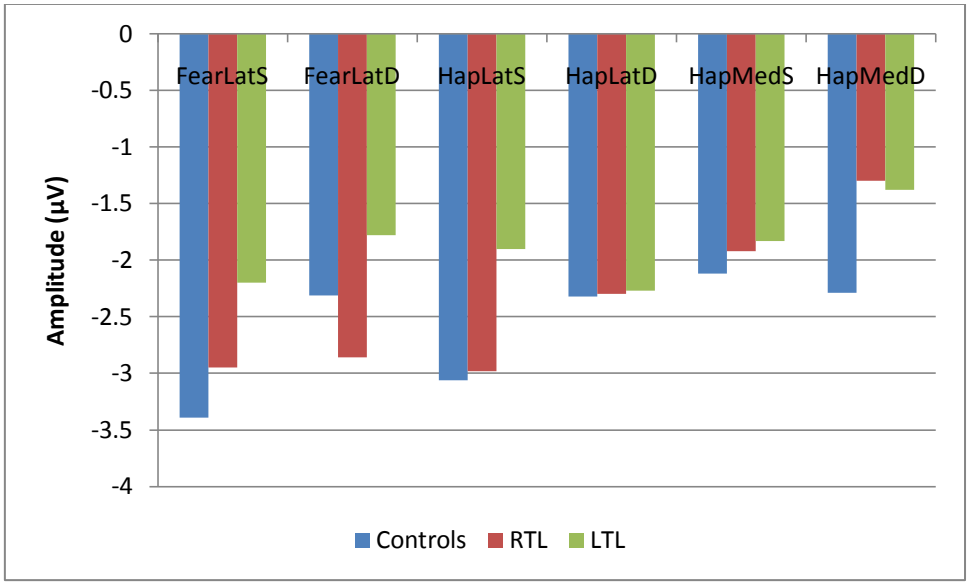


Figure 5.4 Mean peak N170 amplitude values for lateral-medial topography for the motion conditions for fear and happiness across groups

Key: FearLatS = Fear, Lateral, Static; FearLatD = Fear, Lateral, Dynamic; HapLatS = Happy, Lateral, Static; HapMedS = Happy, Medial, Static; HapMedD = Happy, Medial, Dynamic

5.3.2.1.8 Are these group differences specific to social stimuli?

To determine whether the effect of motion on the N170 over left ventral sites was specific to social stimuli, an ANOVA was computed with Stimulus (Emotional, Non-Emotional) by Motion (Static, Dynamic) by Group (Control, RTL, LTL) as the variables over left ventral electrodes. The interaction did not reach significance, $F(2, 27) = 0.10$, $p = 0.91$.

5.3.2.1.9 Summary of the N170 Amplitude

In summary, there was no overall difference in N170 amplitude between groups. However the influence of motion differed by group and emotion. The RTL group showed a larger response to dynamic fear than the LTL group. There was an enhanced N170 for static fear compared to other static emotions for the Control group, and dynamic fear compared to other emotions for the RTL group. There was also an enhanced N170 for static fear compared with static happiness for the Control and RTL groups, and dynamic fear compared to dynamic happiness for the RTL group only. By contrast, the LTL group showed an opposite pattern of effect, with a larger N170 for dynamic happiness compared to dynamic fear. There was evidence that the topography of the N170 was influenced by motion, with observable differences between groups. There was a pattern for the RTL group to produce a smaller amplitude than the Control and LTL groups for static stimuli over left ventral regions, and the RTL and LTL groups to produce a smaller amplitude than the Control group for dynamic stimuli over left ventral regions. There was a significantly smaller

amplitude for the RTL and LTL groups compared with the Control group over medial sites for dynamic happiness, and a smaller amplitude for the LTL group compared with the other two groups over lateral sites for both static fear and static happiness.

5.3.2.2 N170 Latency

5.3.2.2.1 General effects

There were no main effects of Motion or Emotion.

5.3.2.2.2 Prediction 1: The N170 will be slower in the RTL group compared with the Control and LTL groups, particularly for negative emotions

There was no main effect of Group, $F(2, 27) = 1.73$, $p = 0.197$, partial $\eta^2 = 0.11$, indicating that the N170 was of similar latency across the groups. In order to investigate more specifically whether RTL patients showed a slower response over the right lateral ventral regions where the N170 is typically quickest and whether this was more pronounced for negative emotions, an Emotion (Positive, Negative) by Group (Control, RTL, LTL) ANOVA was computed for just these electrodes for static images. There was no significant interaction between Emotion and Group, $F(2, 27) = 0.57$, $p = 0.57$.

5.3.2.2.3 Prediction 2: Patient group deficits observed may be reduced for dynamic stimuli, with the most pronounced effect seen for negative emotions

There was a trend towards a significant two-way interaction of Motion by Group, $F(2, 27) = 2.81$, $p = 0.058$, partial $\eta^2 = 0.17$. Inspection of the means suggests that this was driven by a longer latency for the RTL group compared to the Control group for the dynamic condition. Mean peak latency and S.D. values are shown in Table 5.12 below.

Table 5.12 Mean peak N170 latency (ms) & S.D. values for static & dynamic conditions across groups

	Group					
	Controls (n = 15)		RTL (n = 9)		LTL (n = 6)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Static	161.37	(7.77)	165.50	(8.08)	161.66	(13.45)
Dynamic	159.09	(5.82)	165.85	(10.49)	163.87	(12.96)

There was also a trend towards a significant three-way interaction of Motion by Emotion by Group, $F(10, 135) = 1.81$, $p = 0.067$, partial $\eta^2 = 0.12$. Inspection of the means suggests that this was driven by a longer latency for the RTL group compared to the other two groups for dynamic sadness. The latency is shorter for the dynamic condition compared to static for the LTL group. Mean peak latency and S.D. values are shown in Table 5.13 below.

Table 5.13 Mean peak N170 latency (ms) & S.D. values for static & dynamic conditions for sadness across groups

	Group					
	Controls (n = 15)		RTL (n = 9)		LTL (n = 6)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Sadness Static	160.53	(5.00)	163.67	(8.95)	165.12	(11.60)
Sadness Dynamic	158.55	(6.97)	166.12	(4.67)	158.25	(9.54)

5.3.2.2.4 Prediction 3: The N170 will be slower for fear for the RTL group

A targeted analysis was performed over right lateral ventral sites for fear. There was trend towards a significantly shorter latency for the Control compared to the RTL group for the dynamic condition, $t(1, 22) = 1.95$, $p = 0.054$ (difference of 10.51ms). There were no other significant differences between groups for either motion conditions.

Fear was compared with other emotions within each group over right lateral ventral regions; there was a significantly shorter latency for static fear compared with other static emotions combined for the Control group only, $t(1, 14) = 2.32$, $p < 0.05$ (difference of 2.26ms). There were no other significant differences observed. Fear was also compared with happiness, however, there were no significant differences found. Mean peak latency and S.D. values are shown in Table 5.14 below.

Table 5.14 Mean peak N170 latency (ms) & S.D. values for fear, happiness, and other emotions for the two motion conditions

	Group					
	Controls (n = 15)		RTL (n = 9)		LTL (n = 6)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Fear Static	160.81	(5.71)	161.95	(8.67)	151.81	(10.94)
Fear Dynamic	160.53	(8.72)	171.04	(17.77)	169.44	(21.08)
Happiness Static	159.79	(7.13)	163.40	(6.90)	157.10	(12.93)
Happiness Dynamic	160.31	(9.22)	167.85	(12.31)	165.33	(22.73)
Other Emotions Static	163.07	(6.72)	166.81	(8.00)	161.78	(13.39)
Other Emotions Dynamic	159.84	(7.27)	167.23	(10.83)	165.23	(18.43)

5.3.2.2.5 Prediction 4: Motion effects in the topography may differ between groups

There was a significant four-way interaction of Motion by Emotion by Dorsal-Ventral Topography by Group, $F(10, 135) = 2.64$, $p < 0.01$, partial $\eta^2 = 0.16$. Further analysis revealed a significant three-way interaction of Motion by Emotion by Group in the dorsal regions only, $F(10, 135) = 2.42$, $p < 0.01$, partial $\eta^2 = 0.15$. In dorsal regions, the two-way interaction of Motion by Group reached significance for disgust, $F(2, 27) = 3.63$, $p < 0.05$, partial $\eta^2 = 0.212$; and sadness, $F(2, 27) = 9.29$, $p < 0.001$, partial $\eta^2 = 0.41$; with a trend towards significance for happiness, $F(2, 27) = 3.13$, $p = 0.060$, partial $\eta^2 = 0.19$. Post-hoc analysis revealed a significantly shorter latency for the LTL group compared with the RTL group for the static condition in dorsal regions for disgust, $t(1, 13) = 2.30$, $p < 0.05$ (difference of 11.82ms).

For dynamic sadness in dorsal regions, there was a significantly shorter latency for the Control group compared with the RTL group, $t(1, 22) = 3.00$, $p < 0.01$ (difference of 10.24ms; and a significantly shorter latency for the LTL compared with the RTL

group, $t(1, 13) = 2.99$, $p < 0.01$ (difference of 11.61ms). Comparing the static and dynamic conditions within groups for sadness over dorsal regions, there was a significantly shorter latency for the static condition for the RTL group, $t(1, 8) = 2.63$, $p < 0.05$ (difference of 7.04ms); and a significantly shorter latency for the dynamic condition for the LTL group, $t(1, 5) = 6.20$, $p < 0.005$ (difference of 8.25ms).

Post-hoc analysis also revealed a significantly shorter latency for the Control group compared with the RTL group for the dynamic condition in dorsal regions for happiness, $t(1, 22) = 2.16$, $p < 0.05$ (difference of 7.72ms). Comparing the static and dynamic conditions within groups for happiness over dorsal regions, there was a significantly shorter latency for the static condition for the LTL group, $t(1, 5) = 3.29$, $p < 0.05$ (difference of 7.82ms). In summary, there was a significantly longer latency for the RTL group compared with the Control group over dorsal regions for both dynamic happiness and dynamic sadness, and a slower latency for the RTL group compared with the LTL group over dorsal regions for both static disgust and dynamic sadness, as illustrated in Figure 5.5 below. Mean peak latency and S.D. values are shown in Table 5.15 below.

Table 5.15 Mean peak N170 latency (ms) & S.D. values for motion conditions over dorsal regions for disgust, happiness & sadness across groups

	Group					
	Controls (n = 15)		RTL (n = 9)		LTL (n = 6)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Dorsal Disgust Static	162.78	(10.11)	166.74	(7.78)	154.92	(12.28)
Dorsal Disgust Dynamic	160.17	(6.08)	163.16	(12.79)	162.85	(8.67)
Dorsal Happiness Static	158.82	(7.69)	165.26	(9.35)	156.79	(9.05)
Dorsal Happiness Dynamic	158.59	(8.13)	166.31	(9.04)	164.61	(12.84)
Dorsal Sadness Static	161.99	(6.56)	161.81	(8.86)	165.49	(9.39)
Dorsal Sadness Dynamic	158.61	(8.28)	168.85	(7.78)	157.24	(6.63)

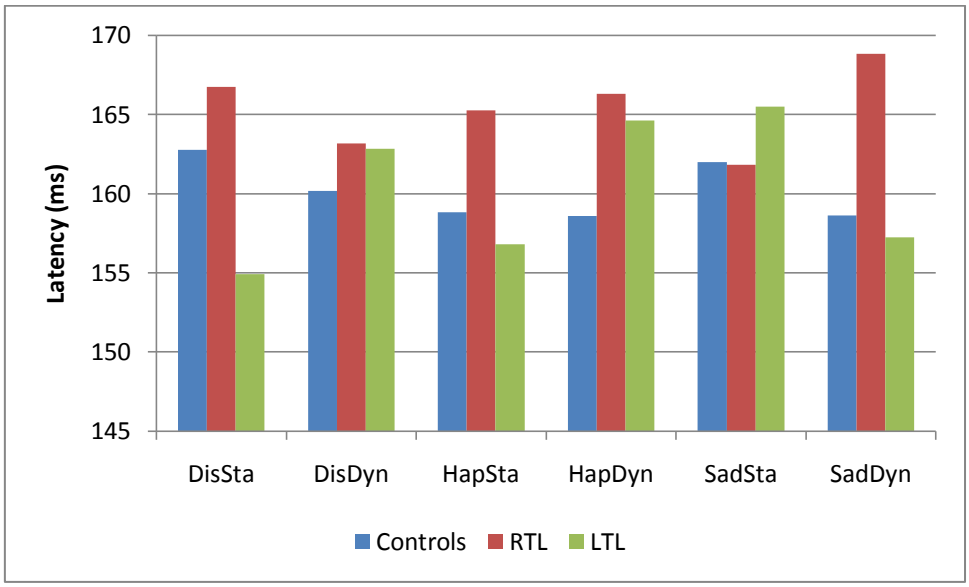


Figure 5.5 Mean peak N170 latency values for lateral-medial topography for the motion conditions for fear and happiness across groups

Key: DisSta = Disgust, Static; DisDyn = Disgust, Dynamic; HapSta = Happy, Static; HapDyn = Happy, Dynamic; SadSta = Sadness, Static; SadDyn = Sadness, Dynamic

In summary, over dorsal regions, the N170 latency for the RTL group was longer than the LTL group for static disgust, longer than the Control group for dynamic happiness, and longer than the Control and LTL groups for dynamic sadness.

5.3.2.2.6 Are these group differences specific to social stimuli?

To determine whether the effect of motion on the N170 over dorsal regions was specific to social stimuli, an ANOVA was computed with Stimulus (Emotional, Non-Emotional) by Motion (Static, Dynamic) by Group (Control, RTL, LTL) as the variables over dorsal electrodes. The interaction did not reach significance, $F(2, 27) = 1.37$, $p = 0.27$.

5.3.2.2.7 Summary of N170 Latency

In summary, there was no overall difference in N170 latency between groups. However the influence of motion differed by group and emotion. The RTL group produced a slower N170 response than the Control group for the dynamic condition, particularly for fear and sadness (where it was also slower than the LTL group). There was a quicker N170 response for static fear compared with other static emotions for the Control group only. There was evidence that the topography of the N170 was influenced by motion, with observable differences between groups. Over dorsal regions, the N170 latency for the RTL group was slower than the LTL group for static disgust, slower than the Control group for dynamic happiness, and slower than the Control and LTL groups for dynamic sadness.

5.3.2.2.8 N170 Amplitude and Latency Summary

The influence of motion on both the amplitude and latency differed by group and emotion. The RTL group showed a larger response to dynamic fear than the LTL group. There was an enhanced N170 for static fear compared to other static emotions for the Control group, and dynamic fear compared to other emotions for the RTL group. There was also an enhanced N170 for static fear compared with static happiness for the Control and RTL group, and dynamic fear compared to dynamic happiness for the RTL group. By contrast, the LTL group showed an opposite pattern of effect, with a larger N170 for dynamic happiness compared to dynamic fear. The RTL group produced a slower N170 response than the Control group for the dynamic condition, particularly for fear, and sadness (where it was also slower than the LTL group). There was a quicker N170 response for static fear compared with other static emotions for the Control group only. There was evidence that the topography of the N170 was influenced by motion, with observable differences between groups. There was a pattern for the RTL group to produce a smaller amplitude than the Control and LTL groups for static stimuli over left ventral regions, and the RTL and LTL groups to produce a smaller amplitude than the Control group for dynamic stimuli over left ventral regions. There was a significantly smaller amplitude for the RTL and LTL groups compared with the Control group over medial sites for dynamic happiness, and a smaller amplitude for the LTL group compared with the other two groups over lateral sites for both static fear and static happiness. Over dorsal regions, the N170 latency for the RTL group was slower than the LTL group for static disgust, slower than the Control group for dynamic happiness, and slower than the Control and LTL groups for dynamic sadness.

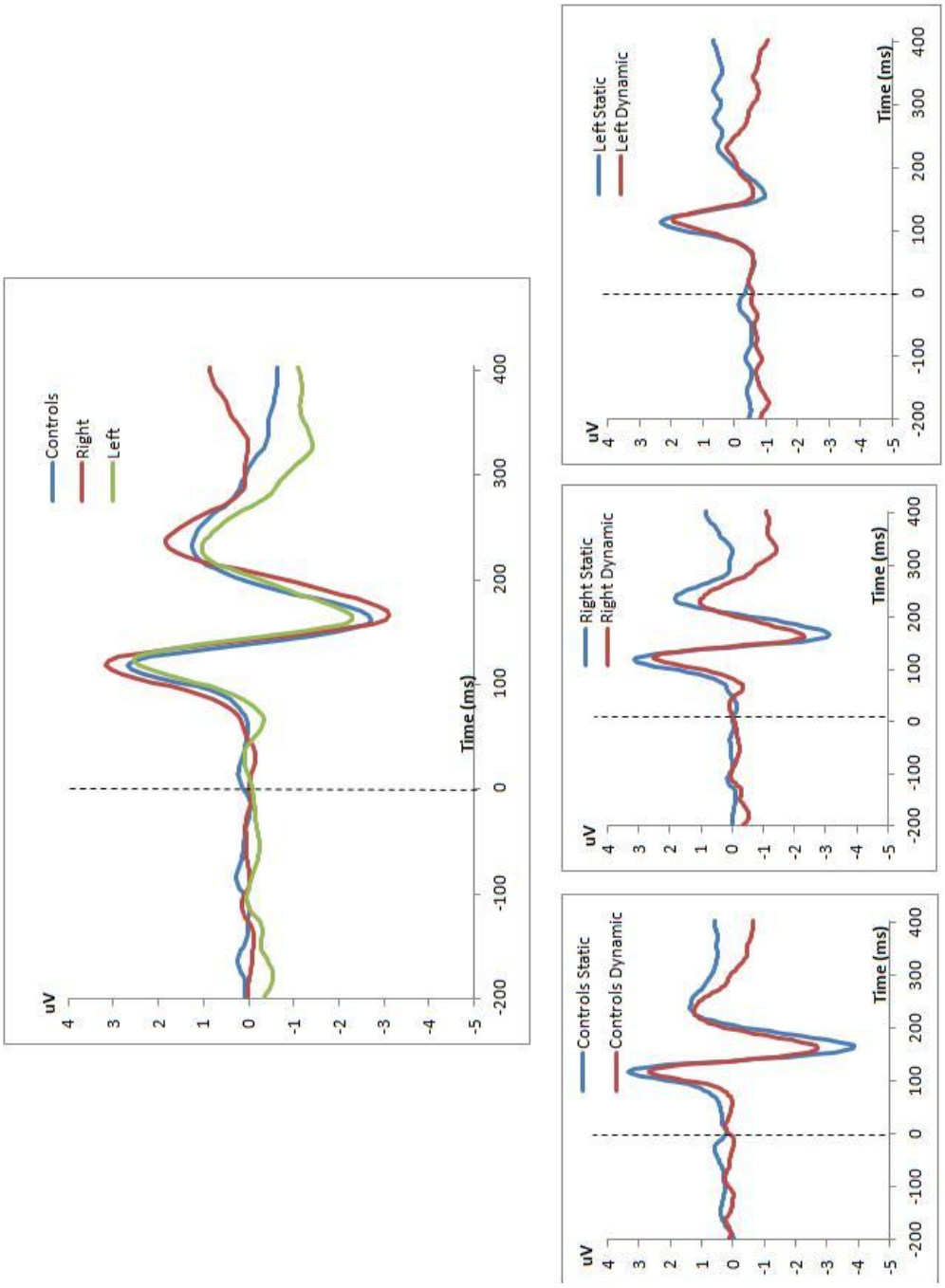


Figure 5.6 ERP waveforms illustrating the P1 and N170 in a comparison between the control, right-sided and left-sided patient groups (top figure); and static and dynamic conditions for each group over right lateral ventral sites (bottom figures).

5.4 Discussion

This study investigated the early-latency neural correlates of static and dynamic emotion processing in a population who had undergone right or left temporal lobectomy for the treatment of paediatric-onset temporal lobe epilepsy compared to age- and gender-matched healthy controls. The main predictions were that: 1) the P1 and N170 responses would be diminished and possibly slower in the RTL group compared with the Control and LTL groups, particularly for negative emotions; 2) group deficits may be reduced for dynamic stimuli, with the most pronounced effect observed for negative emotions; 3) the P1 and N170 response would be diminished for fear for the RTL group; and 4) motion effects on the topography may differ between groups.

The main findings will be listed below and then following this summary, each result will be discussed in further detail.

In summary: There was a reduced P1/N170 response for static emotions in general in the right-sided patient group, and reduced P1/N170 response for static fear in the left-sided group. This observed reduction in P1/N170 response was diminished for dynamic stimuli, and varied by topography.

More specifically:

1) The differences in N170 response between groups for static stimuli: The RTL group produced a smaller N170 response to static stimuli over left ventral regions compared with the other two groups. The RTL group produced a slower N170 response for static disgust over dorsal sites compared with the

LTL group. The LTL group produced a smaller N170 response to static fear and happiness over lateral sites compared with the other two groups.

2) The differences in N170 response between groups for dynamic stimuli: The RTL group produced a slower N170 response for dynamic stimuli, particularly fear and sadness compared with the Control group, but not the LTL group. The RTL and LTL groups produced a smaller N170 for dynamic stimuli over left ventral regions; and a slower N170 response for dynamic happiness over medial sites. The RTL group produced a slower N170 response compared with the Control group, but not the LTL group, for dynamic happiness, and a slower N170 response than the other two groups for dynamic sadness. The LTL group produced a smaller N170 response for dynamic fear compared with the RTL group. The LTL group produced a smaller response to dynamic fear compared with the RTL group.

3) Differences in the P1 response: The P1 response was smaller for dynamic fear compared with other dynamic emotions including happiness for the LTL group, and quicker for static anger compared with dynamic anger for the LTL group, but not the RTL group. The P1 response was larger for static stimuli compared with dynamic for the RTL group, particularly for disgust and happiness, but not the LTL group. In contrast the P1 response was larger for dynamic stimuli compared with static stimuli for disgust and happiness in the LTL group, but not the RTL group.

4) Comparisons of fear and other emotions: The RTL group produced a larger N170 response to static fear compared with static happiness. The RTL group produced a larger N170 response to dynamic fear compared with other

dynamic emotions including happiness. In contrast, the LTL group produced a reduced P1 for dynamic fear compared with other emotions including happiness. There were no differences between fear and other emotions for the P1.

5) Differences in motion effects on topography between groups: Over left ventral regions, the RTL group produced a smaller N170 amplitude than the other two groups for static stimuli, but not dynamic; and the RTL and LTL groups produced a smaller N170 amplitude compared with the Control group for dynamic stimuli, but not static. Over medial sites, the RTL and LTL group produced a smaller N170 amplitude than the Control group for dynamic happiness. Over lateral sites, the LTL group produced a smaller N170 amplitude than the other two groups for static fear and happiness. Over dorsal regions, the RTL group produced a slower N170 compared with the LTL group for static disgust; a slower N170 response compared with the Control group for dynamic happiness; and a slower N170 response than both other groups for dynamic sadness. There were no effects of motion on the P1 topography.

5.4.1 Group effects on the P1 and N170 response to static emotional expressions

There were group differences in both the P1 and N170 response to static stimuli, which were more pronounced for certain emotions. There was a smaller amplitude N170 for the LTL group compared with the Control and RTL groups for fear and

happiness, and a slower N170 latency for the RTL group compared with the LTL group for disgust. The RTL group also produced a larger amplitude for fear compared to happiness. The prediction was that the RTL group would have a diminished and/or slower response than the other two groups. This prediction was based on evidence that there is a right-sided bias for emotion processing in the normal population (DeKosky, Heilman, Bowers, & Valenstein, 1980; Natale, Gur, & Gur, 1983), and that damage to the right amygdala has been shown to produce more severe emotion recognition deficits than damage to the left amygdala in temporal lobe epilepsy patients before and after surgery (Adolphs et al, 2001; Anderson et al, 2000; Meletti et al, 2003, 2009) with negative emotions, and more specifically fear, being more impaired (Benuzzi et al, 2004; Meletti et al, 2003, 2009). The reciprocal connections between the amygdala and cortical structures such as the fusiform gyrus and STS, sources of the P1 and N170, would lead to the prediction that the RTL group would have a diminished/slower P1 and N170 to emotional faces, particularly negative emotions. The finding of a slower N170 for the RTL group for disgust over dorsal regions, and a smaller N170 response to static stimuli over left ventral regions is consistent with this prediction. However, the RTL group produced a larger response to static fear than static happiness. In contrast, the LTL group produced a smaller N170 response to static fear and happiness over lateral sites compared with the other two groups. This is contrary to the prediction that the responses in the LTL would be unaffected. These results suggest that the left amygdala may be influential on cortical processing of emotion and not particularly sensitive to the valence of the emotion. It is possible that the atypical findings in the LTL group reflect reduced modulation by the amygdala of the cortical pathway. One study examining

subcortical pathway activation in adolescents found that, in contrast to adults who show a right-lateralised pathway, adolescents show a left-lateralised pathway (Killgore & Yurgelun-Todd, 2010). It may be that a lack of enhancement of the early-latency ERPs to fear in the LTL group is a reflection of paediatric damage to the left subcortical pathway. These findings will now be discussed in the light of the responses to dynamic emotional stimuli.

5.4.2 Group effects on the P1 and N170 response to dynamic emotional expressions

It was proposed that the deficits observed for the static stimuli would be absent or reduced for the dynamic stimuli. This was based on evidence that dynamic stimuli recruit a more distributed network than static emotional stimuli (Kilts et al, 2003; LaBar et al, 2003; Sato et al, 2004), leading to the possibility that disruption to the temporal lobe structures may not extend to networks associated with processing dynamic stimuli. For example, reciprocal projections between the STS and the amygdala may be less affected than projections between the fusiform gyrus and amygdala. In addition, there is the possibility that dynamic images will convey greater temporal and structural facial object properties (Harwood et al., 1999; Sato et al., 2004), which may aid recognition of facial expressions by recruiting additional neural circuitry. When comparing the deficits found for static stimuli and dynamic stimuli, the only three findings consistent across motion conditions are: 1) a diminished N170 response over left ventral regions for the RTL group; 2) an enhanced N170 response to fear compared with other emotions for the RTL group; and 3) a diminished N170 response to fear compared with other emotions for the

LTL group. All other deficits observed for static stimuli are absent for dynamic, a pattern consistent with the idea that the neural processing of facial emotion may be more intact for dynamic compared to static facial emotions.

However, other findings are inconsistent with this interpretation. In particular, the RTL group demonstrated a slower N170 response to all dynamic stimuli, and more specifically to happiness and sadness compared with the other two groups, whereas this latency difference was not observed for static stimuli. One possible reason for the slower response for the dynamic stimuli in the RTL group might be that the right amygdala is more influential in processing dynamic stimuli. This might be due to motion itself or factors such as attention or salience of the stimuli. If it were attentional aspects, it might be assumed that the P1, which is known to be modulated by attention, would also be slower in the RTL group, and this is not the case. It may be that the salience of the stimuli influences the activity of the amygdala. One view of the enhanced amygdala activation for fearful faces and other negative/threat compared with positive/non-threatening emotions is that the amygdala responds preferentially to the eye region of the face, and this is the feature most prominent in fearful expressions (Smith et al, 2005). Eye gaze direction and pupil size have been shown to modulate responses to fearful faces (Adams et al, 2003; Demos et al, 2008), even when presented very briefly or masked (Whalen et al, 2004). Bilateral amygdala damage results in an inability to fixate the eye region of the face spontaneously and produces a consequent impairment in utilising high spatial frequency information from the eyes in order to recognise fear (Adolphs et al., 2005). This suggests that the amygdala might not be specialised for processing

fear or threat, or even emotional content per se, but instead might be tuned to respond to the salience of a stimulus (Whalen et al, 2007). There was a similar N170 latency pattern for the non-emotional stimuli. This may lend support to the idea that the dynamic stimuli were particularly salient and the right amygdala is most influenced by the saliency of the stimuli, however the overall N170 response was larger for static stimuli which counteracts this argument (as do the findings in Chapter 4).

Another finding that is inconsistent with the idea that the patients process dynamic images in a similar manner to the healthy controls compared with static ones is the reduced amplitude N170 for the LTL group. This suggests that damage to the left amygdala also leads to processing deficits of dynamic stimuli. This could also be due, not only to damage to the amygdala but also disruption in the reciprocal connections from the amygdala to the visual cortices. The right amygdala is thought to be preferentially activated in response to subliminally presented expressions, whereas the left amygdala is activated when stimuli are presented for longer (Morris et al., 1998; Morris et al., 1999; Whalen et al., 1998). These differences in function may lead to differential modulation of cortical regions, reflected in effects observed at different time points, and not necessarily in early-latency components. This is illustrated by the observation that the P1 response to fear is smaller compared with other emotions for the LTL group but not the RTL group. This contrasts with the finding in Chapter 4 that the P1 response in the adult group was larger for dynamic fear compared with other emotions.

The prediction that dynamic stimuli would reduce the deficits was based on studies that have shown that dynamic emotional stimuli recruit a more distributed neural network than static emotional stimuli (Kilts et al., 2003; LaBar et al., 2003; Sato et al., 2004), and convey greater temporal and structural facial objects properties (Harwood et al., 1999; Sato et al., 2004) which may aid the recognition of facial expressions. As discussed in previous chapters, functional imaging studies have shown that a more distributed neural network is active for dynamic emotional stimuli. Another possible reason for the differential responses observed might be that a greater number of neural structures are processing moving stimuli; with projections to the fusiform gyrus, STS and other cortical areas aiding processing of dynamic emotional stimuli. In addition, indirect modulation of cortical regions by the amygdala may be enhanced, particularly the cholinergic system in the basal forebrain which receives prominent inputs from the amygdala and projects to widespread cortical regions including early sensory visual areas (Holland & Gallagher, 1999). These indirect projections may still exist whilst direct connections from the amygdala to the fusiform gyrus and other visual cortical areas may be extinct. One reason for the slower response in the RTL group for dynamic stimuli could be that projections either from the dorsal visual pathway to the amygdala, projections from the amygdala to the STS, or both, could be disrupted to a greater degree than the pathway for processing static stimuli (ventral visual stream and fusiform gyrus). The two systems are thought to be integrated, however disruption to either pathway has resulted in clear dissociable deficits in moving and static stimuli respectively. This disruption may lead to a delayed response in cortical regions. This leads onto the fourth prediction that motion effects on topography may differ between groups.

5.4.3 Motion effects on topography

The findings show that there were no differences in motion effects between groups on the P1 topography. However, the N170 topography differed for the static and dynamic stimuli. There was a smaller N170 amplitude for the RTL group compared with the other two groups over left ventral regions for static stimuli, and a smaller amplitude for the LTL group compared with the other two groups over lateral sites for static fear and static happiness. There was a smaller amplitude for the RTL and LTL groups compared with the Control group for dynamic stimuli over left ventral regions, and medial regions for dynamic happiness. The N170 was slower for the RTL group for static disgust over dorsal regions. The finding that the N170 response is diminished over ventral and lateral regions for both patient groups is consistent with the view that the N170 is maximal over this region in healthy populations, with reduced sensory processing in the generators leading to a diminished response over this region. Two findings are inconsistent with possible predictions. Firstly, the reduced response in the RTL group over the left ventral regions for both static and dynamic stimuli. It might be expected that a reduced response would be seen in the ipsilateral hemisphere to the lesion. Secondly, the reduced response in the RTL group to static stimuli over dorsal regions might be predicted over ventral regions, reflecting the ventral pathway for static images, with a reduced response to dynamic stimuli observed in this region instead. In future research, some of these issues may be disentangled if complementary structural and/or functional neuroimaging data could be used alongside the ERP measures.

5.4.4 Conclusions, limitations, and future directions

There are differences in the processing of emotional stimuli by individuals who have undergone right or left temporal lobectomy compared with healthy individuals. This is observed not only between the control group and the patient groups, but between the two patient groups themselves. Generally, the RTL group produced a slower response to emotional stimuli and the LTL group produced a diminished response. There was no strong evidence that these effects were specific to social stimuli when the emotional stimuli were compared to the flower control. However, the finding of differences within the category of emotion (e.g., happy versus fear, etc.) might indicate more emotion-specific patterns. It is not possible to elucidate the exact neural mechanisms that are producing these effects. In addition, this study only addressed the effects of temporal lobe lesions on the early-latency components. It is suggested that the amygdala may be involved in both the initial rapid encoding of faces and emotional stimuli and also modulate later complex stimulus processing (Adolphs, 2008). It is also not possible to draw any definite conclusions on the processing of social stimuli in individuals with amygdala lesions. The sample size was small in this study (15 temporal lobectomy patients: 9 RTL & 6 LTL), limiting the conclusions that can be drawn from the results described in this study. It is not possible to reflect the population as a whole with such a small group of individuals, particularly due to the heterogeneity of the population in factors such as lesion location and size, amygdala volumes, age of onset etc. It is worthy to note that some previous comparable studies have also tended to have a small sample size, e.g.

Benuzzi and colleagues (2004) who recruited 8 RTLE & 5LTLE patients; and Anderson and colleagues (2000) who recruited 12 RTL & 11 LTL patients; reflecting the difficulty in recruiting this particular population. Clearly, it will be important to obtain a larger sample size in future studies to ascertain the significance of the findings in the current study, and to allow replication of results. It was not possible to address all these variables in this study, with the aim to focus on the effects of motion in the early processing of emotion in the two patient groups. Future research in this population should address these limitations and explore longer-latency ERPs to elucidate whether disruption to the temporal lobe, particularly the amygdala, influences processing at later time windows. There is a great deal of research demonstrating the deficits in facial expression recognition of static stimuli in this population. Future research needs to couple these findings with electrophysiological data to understand the neural correlates of these deficits. The next chapter aims to address this issue by assessing the same patient groups on emotion recognition tasks using static stimuli as a comparison to previous research. In addition, the impact of the temporal lobe epilepsy and lobectomy on social functioning will be examined.

6. Emotion recognition and social functioning following paediatric temporal lobectomy

6.1 Introduction

The previous chapter addressed how disruption to temporal lobe structures can affect the early-latency processing of static and dynamic emotional stimuli. This chapter aims to investigate relations between the electrophysiological measures and behavioural measures of emotion recognition deficits including both facial and vocal emotions. As discussed in detail in Chapter 1 and 5, there is evidence that temporal lobectomy (TL) can result in facial emotion recognition deficits (Adolphs et al, 2001; Anderson et al, 2000). These deficits are particularly pronounced for emotions associated with withdrawal and more frequently observed in right-sided TL patients than left-sided TL patients, and control groups (including healthy individuals and individuals with brain damage excluding the anterior temporal lobe). Although these patients have difficulty recognising facial emotion, they perform normally in facial identity tasks and have intact visuoperceptual skills. Facial emotion recognition deficits in this population are not, however, found consistently, with one study finding both RTL and LTL groups performing as well as controls (Adolphs et al, 1995). Deficits in recognising emotion from auditory cues might also be expected in cases of TL due to damage encroaching on the amygdala. The amygdala is thought to be involved in emotional processing regardless of the sensory modality, especially when the stimuli are perceived as threatening (Ethofer et al., 2006). A few studies have investigated the recognition of emotional prosody in this population, alongside facial emotion recognition (Adolphs et al., 2001; Bonora et al., 2011; Fowler et al., 2006).

Bonora and colleagues (2011) found that the accuracy of emotional prosody recognition was significantly lower in a group with mesial temporal lobe epilepsy (MTLE) compared with healthy controls. The overall accuracy of recognition for the emotional prosody task was lower than for the facial emotion task in both patient and control groups, indicating that the recognition of emotions through voices may be less accurate. In contrast, two other studies found no impairment in emotional prosody recognition in MTLE patients as a whole, only finding deficits at a single-subject level; one study in patients before surgery (Fowler et al., 2006), and one after lobectomy (Adolphs et al., 2001). The heterogeneity of this population may be a factor in the contrasting findings between studies, in terms of clinical features such as age of epilepsy onset, age at surgery, number of seizures before surgery etc. This is an important point to consider when interpreting the findings.

It is possible that disruption of the amygdala may lead to deficits in more complex emotional processing, such as empathy and 'theory of mind' (Blair et al, 2003; Shaw et al, 2004). The amygdala may play an important role in the neural systems supporting normal development of social reasoning, and paediatric temporal lobe epilepsy and lobectomy may serve to prevent these skills from developing normally. Studies have shown that TLE patients have reduced social functioning and quality of life (Dodrill, 1986).

There is evidence of a reduction in mesial temporal lobe activity ipsilateral to the lesion in right-sided TLE patients with emotion recognition deficits (Benuzzi et al., 2004). Early onset right amygdalar damage has been associated with lack of bilateral activation of cortical and subcortical regions in response to fearful faces, with this reduction correlated with emotion recognition deficits. In contrast, recognition

deficits were not reported in left-sided and healthy control groups, all with bilateral temporal lobe activation. This suggests that disruption to neural structures resulting in reduced activity leads to observable behavioural level deficits, which are limited to individuals with right-sided lesions. It is possible that this reduction in neural activity, as reflected in the early-latency ERPs, will be correlated with deficits in emotion recognition.

The aims of this study were: 1) to assess the emotion recognition of the RTL and LTL groups; 2) to explore the impact temporal lobectomy may have on general social functioning; and 3) to compare deficits in emotion recognition with the neural correlates.

The specific questions and hypotheses to be addressed are as follows:

1. There will be deficits in emotion recognition for the RTL group: A diminished recognition of negative emotions, particularly for fear, for the RTL group (Adolphs et al, 2001; Anderson et al, 2000), possibly across auditory as well as visual modalities (Royet et al, 2000).
2. There will be a reduction in social functioning for both patient groups, particularly the RTL group: This will be due to cognitive impairments, such as emotion processing and memory deficits caused by anterior temporal lesions (Blair et al, 2003; Rausch, 2002).
3. Deficits in emotion recognition may be correlated with a diminished/slower electrophysiological response: Disruption to neural substrates may result in both a change in electrophysiological response and the observed emotion recognition deficits. An fMRI study observed a reduction in mesial temporal

lobe activity ipsilateral to the lesion in an RTLE group with emotion recognition deficits (Benuzzi et al., 2004).

6.2 Methods

6.2.1 Participants

As for Chapter 5.

6.2.2 Materials and Experimental Tasks

The assessments were administered in the same order for each participant: Matrix Reasoning (subtest of the WAIS-III), Florida Affect Battery (FAB), Empathy Quotient (EQ) and Social Functioning Scale (SFS). Refer to the Appendices D & E for the EQ and SFS questionnaires respectively. All were administered after a break following the ERP experiment. All assessments were reported by the examiner except the EQ and SFS which were completed independently by the participant during the assessment period. The participant sat at a table in a brightly-lit room opposite the examiner for all assessments.

All materials, administration and scoring will be addressed for each assessment in turn.

6.2.2.1 Matrix Reasoning

6.2.2.1.1 Materials

The Matrix Reasoning is a subtest of the Wechsler Adult Intelligence Scale-III (WAIS-III), and measures perceptual organisation and nonverbal reasoning. It assesses visual information processing and abstract reasoning, without a bias on language skills. The subtest is composed of four types of tasks: classification, pattern completion, analogy, and serial reasoning. Materials consisted of a stimulus booklet, manual and record sheet. The stimulus booklet contained three sample items and 26 test items on separate pages. For each item, there was an example of one of the four task types presented as colour diagrams in an incomplete matrix, and five response options below. Subtest items were ordered according to increasing difficulty.

6.2.1.1.2 Administration

The examiner displayed each item in turn to the examinee, in the order described below. For each item, the examinee was instructed to verbally state or point to one of the five options that they believed completed the matrix. The examinees were allowed to take as long as they needed for each item. The assessment took approximately 30 minutes to administer.

Initially, the three sample items were administered, to help the examinee understand the instructions of the task. If the examinee responded incorrectly to any of the sample items the examiner explained the correct way to solve the problem. The three sample items were administered in the same order for all examinees and

were not part of the assessment. Regardless of the performance on the sample items, test items 4 and 5 were administered. If the examinee identified the correct response to these test items, then they were given full credit for test items 1-3. However, if the examinee responded incorrectly to either test item 4 or 5, test item 1-3 were administered in reverse sequence until the examinee responded correctly on two consecutive items. If the examinee responded correctly to test item 4, this was counted in the reverse sequence. When this criterion was met, full credit was given for any preceding items that were not administered. The rest of the subtest was then completed unless the examinee responded incorrectly to four consecutive test items, or four scores out of five consecutive test items, when the subtest was discontinued.

6.2.2.1.3 Scoring

One point was given for every correct response (maximum score of 26). This raw score was then transformed to a scaled score based on age-appropriate comparison norms (mean = 10, SD = 3). The scaled scores for the Matrix Reasoning subtest ranged from 0-17.

6.2.2.2 Florida Affect Battery

6.2.2.2.1 Materials

The battery assessed facial expressions and emotional prosody under a variety of task demands. It was designed to investigate the perceptual changes in these social

signals in neurological and psychiatric disorders. There are ten subtests (5 facial, 3 prosodic, and 2 cross-modal) assessing different aspects of emotion perception. All the tasks assessed facial identity and emotion discrimination comprised of black and white photographs of four female actors, with only their faces displayed and their hair covered with a surgical cap. There were 20 trials for each facial subtest (1-5), and for subtests 3-5 each of the five different emotional expressions were presented four times in a pseudo-randomised order across the task. There were two initial practice trials for subtest 1 & 2, and five practice trials for subtest 3-5, to familiarise examinees with the task. The stimuli for all tasks were presented in bound booklets, and assessment of tasks was directed by the examiner.

Subtest 1: Facial Identity Discrimination. In each trial, examinees were shown a pair of unfamiliar faces and had to determine whether the two stimuli displayed were the same person or different people. Each actor displayed a neutral facial expression. Half the trials displayed two pictures of the same person and half displayed two different people.

Subtest 2: Facial Affect Discrimination. In each trial, examinees were shown a pair of unfamiliar faces and had to determine whether the emotional expressions displayed by the two different actors were the same or different. Half the trials displayed the same emotional expression and half displayed two different emotions.

Subtest 3: Facial Affect Naming. In each trial, examinees had to verbally label the emotional expression displayed by an actor. Angry, fearful, happy, sad and neutral emotional expressions were assessed.

Subtest 4: Facial Affect Selection. In each trial, examinees were shown five photographs of different actors, in a vertical array, each expressing a different

emotional expression. The examinees were instructed to identify the photograph of the actor that corresponded to the emotion named by the examiner (i.e. “point to the happy face”). Angry, fearful, happy, sad and neutral emotional expressions were assessed.

Subtest 5: Facial Affect Matching. In each trial, examinees were shown two stimulus slides. On the left slide, a single photograph of an actor displaying a target emotional face and, on the right slide, five photographs of different actors expressing a variety of emotional expressions. The examinees were instructed to match the target expression with its counterpart on the right of the slide. Angry, fearful, happy, sad and neutral emotional expressions were assessed.

All the tasks assessing prosody discrimination comprised of an audio recording of one female actor producing sentences with semantic content and prosody varying across tasks. Subtests 6, 7, and 8a, consisted of a set of semantically neutral sentences, spoken in either an emotional or non-emotional tone of voice. Subtest 8b involved sentences whose semantic content either complemented or conflicted with the emotional tone of voice. All stimuli were presented in the same pseudo-randomised order. The stimuli for all tasks were presented to examinees via computer speakers situated in the assessment room. There were no practice trials given initially for the prosodic tasks.

Subtest 6: Non-Emotional Prosody Discrimination. In each trial, examinees were presented with a pair of sentences, spoken by one actor, in either a declarative (e.g. “the lamp is on the table”) or interrogative tone of voice (e.g. “the lamp is on the table?”). There were 16 trials in total, half the trials consisted of two sentences

conveying the same propositional prosody (i.e. both declarative or both interrogative), and half consisted of sentences differing in propositional prosody (i.e. one is declarative and one is interrogative). The examinees were instructed to state whether the sentence pairs were the same or different.

Subtest 7: Emotional Prosody Discrimination. In each trial, examinees were presented with a pair of semantically neutral sentences, spoken in the same or different emotional tone of voice. Examinees had to determine whether the sentences contained the same emotional prosody or different. There were 20 trials in total, half the trials were the spoken in the same emotional tone and half were spoken in different emotional tones.

Subtest 8a: Name the Emotional Prosody. In each trial, examinees were presented with a semantically neutral sentence spoken in an emotional tone of voice. They had to verbally label the emotional prosody: angry, fearful, happy, sad and neutral emotional tones of voice were assessed. There were 20 trials in total, with four repetitions of each of the five emotional tones of voice presented randomly throughout the task.

Subtest 8b: Conflicting Emotional Prosody. In each trial, examinees were presented with sentences where the emotional tone of voice and semantic content were either congruent or incongruent with each other. The examinee had to determine the emotional tone used in each sentence, ignoring the semantic content. A selection of trials consisted of a sentence where the emotional prosody and semantic content conveyed the same emotional meaning (congruent) (i.e. “all the puppies are dead” spoken in a sad tone of voice), and a selection where the emotional prosody and semantic content conveyed a different emotional meaning in

two separate ways (incongruent and inconsistent). In incongruent sentences the messages were completely incompatible (i.e. “all the puppies are dead” spoken in a happy tone of voice), and in inconsistent sentences the messages were not completely incompatible (i.e. “all the puppies are dead” spoke in a neutral tone of voice). Angry, happy, sad and neutral emotional tones of voice were assessed. There were 36 trials in total, with nine repetitions of each of the four emotional tones of voice presented randomly throughout the task.

The final two subtests contained both facial and vocal emotion, assessing cross-modal integration. There were no initial practice tasks given for either cross-modal subtest. There were 20 trials for each subtest, with four repetitions of each of the five emotions presented pseudo-randomly throughout each task. There were no practice trials given initially for either cross-modal task.

Subtest 9: Match Emotional Prosody to an Emotional Face. In each trial, examinees were shown three photographs of the same female actor displaying three different emotional expressions. Simultaneously, the examinees were presented with a sentence spoken in an emotional tone of voice. The examinees were instructed to point to the emotional face that matched the emotional tone of voice presented to them. There were 20 trials: angry, fearful, happy, sad and neutral emotional tones of voice were assessed.

Subtest 10: Match Emotional Face to the Emotional Prosody. In each trial, examinees were shown one photograph of an actor displaying an emotional expression. Simultaneously, the examinees were presented with three sentences, one after the other, each spoken with a different emotional tone of voice. The

examinees were instructed to verbally state which of the three sentences matched the emotional expression in the photograph. This task had a larger memory load than the other tasks, and consequently each set of three sentences was presented to the examinee twice, with 20 trials in total.

6.2.2.2.2 Administration

The examiner presented the stimuli in a fixed order to all examinees, in the order described above. For each trial, the examinee was instructed to verbally state or point to one of the label options they believed was correct. The examinee was allowed to take as long as they needed to respond.

6.2.2.2.3 Scoring

The overall percentage of correct responses for each subtest was recorded, and z-scores were computed based on the normative data. The percentage correct for fear in subtests 3, 4, 5, 8a, 9, & 10 was also recorded.

The overall percentage of correct responses were recorded for each subtest, and z-scores were computed based on the normative data collected in the study by Blonder, Bowers, & Heilman (1991). The FAB data violated the assumptions of normality, as many of the tasks had ceiling effects, and were therefore negatively skewed. These data were therefore analysed with both parametric and non-parametric tests. Since both tests produced a similar pattern of findings, parametric test results were reported for these data.

6.2.2.3 Empathy Quotient

6.2.2.3.1 Materials

A self-report questionnaire was used with adults of normal intelligence, measuring individual differences in empathy. It was designed to be short, easy to use and easy to score. It contained 40 empathy items and 20 filler/control items, randomly ordered. The filler items were included to give the participant a break from the continual focus on empathy. For each empathy item, the response to the empathic behaviour could be 'strongly agree', 'slightly agree', 'slightly disagree', or 'strongly disagree'. Half of the empathy items were worded to produce a correct response of 'agree', and half to produce a correct response of 'disagree', to reduce response bias. An example empathy question would be: 'I can easily tell if someone else wants to enter a conversation', (see Appendix D for the EQ questionnaire).

6.2.2.3.2 Administration

Each participant was instructed to complete the questionnaire independently and instructed to avoid thinking about their answers for too long. They were instructed to circle each answer clearly and were given four example questions initially, to practice the type of questions featured within the questionnaire. The questions were presented in a fixed order to all examinees. The questionnaire took approximately ten minutes to complete.

6.2.2.3.3 Scoring

For each empathy item a correct answer strongly agreeing or disagreeing would score two points, a correct answer slightly agreeing or disagreeing would score one point. An incorrect response would result in zero points being awarded for the item, regardless of whether the response was mild or strong. This gave the questionnaire a maximum score of 80 and a minimum of zero. The filler items were not scored.

6.2.2.4 Social Functioning Questionnaire

6.2.2.4.1 Materials

A self-report questionnaire was used which enabled the assessment of social functioning. It initially focussed on the needs and impairments of individuals with schizophrenia, but extended to other groups where social functioning is to be assessed. It was designed to be easy to administer, without extensive training and interview time other functioning scales require. Only the first part of the questionnaire was administered, that which is completed by the individual to be assessed. The second part, completed by a relative or someone in everyday contact with the individual was excluded for the purposes of this study. The scale is divided into seven sections: *withdrawal & social engagement* (time spent alone, initiation of conversations, social avoidance); *interpersonal communication* (quality of communication); *independence-performance* (performance of skills necessary for independent living); *recreation* (engagement in hobbies and leisure interests); *pro-social* (engagement in social activities); *independence-competence* (ability to perform

skills necessary for independent living); and *employment & occupation* (engagement in productive employment, actively seeking work, or structured programme of daily activities), see Appendix E for the SFS questionnaire.

6.2.2.1.2 Administration

Each participant was instructed to complete the questionnaire independently and encouraged to ask the examiner to clarify any questions that were not understood or unclear. The questions were presented in a fixed order to all examinees. The questionnaire took approximately 10 minutes to complete.

6.2.2.1.3 Scoring

Each section was scored according to the scoring scale provided and a total score was produced for each section. These raw scores were then translated into standardised scores resulting in a social functioning profile for each individual (mean = 100, SD = 15). The full score was obtained by taking the mean of the seven standardised subscale scores.

There were no missing data for any of the behavioural tests for any of the groups and outliers were removed as described in Chapter 2.

6.3 Results

6.3.1 Matrix Reasoning

All scores were within the normal range, with the Control group scoring highest (score = 13.93, SD = 3.17), then the LTL group (score = 11.83, SD = 2.48), and the RTL group scoring lowest (score = 10.00, SD = 2.78). The Control group scored significantly higher than the RTL group, $t(1, 22) = 3.07$, $p < 0.01$ (difference of 3.93). There were no other significant differences between groups. As there was a significant difference in Matrix Reasoning between the groups, which is indicative of a possible non-verbal IQ difference, Matrix Reasoning will be included as a covariate where indicated in the analyses to follow.

6.3.2 Florida Affect Battery

The mean z-scores and S.D. for accuracy of recognition for each Florida Affect Battery (FAB) subtest are shown below in Table 6.1.

Table 6.1 Mean z-scores & S.D. for the Florida Affect Battery

	Group					
	Controls (n = 15)		RTL (n = 9)		LTL (n = 6)	
Subscales	z-score	(S.D.)	z-score	(S.D.)	z-score	(S.D.)
1	0.42	(0.66)	0.31	(0.57)	0.59	(0.00)
2	-0.18	(0.74)	-0.56	(1.19)	-1.01	(1.06)
3	0.16	(0.90)	-1.36	(2.08)	-0.23	(0.88)
4	0.06	(0.85)	-1.91	(2.45)	-0.10	(0.93)
5	0.18	(0.54)	-1.08	(1.98)	-1.03	(2.74)
6	0.05	(1.01)	-5.34	(7.09)	-1.65	(4.81)
7	-2.00	(1.56)	-2.24	(2.91)	-1.17	(1.52)
8a	-0.86	(1.38)	-2.40	(1.93)	-0.99	(1.34)
8bi	0.45	(0.80)	0.58	(1.16)	0.25	(1.10)
8bii	0.68	(1.03)	-1.85	(3.09)	0.03	(0.91)
9	-1.43	(2.25)	-4.17	(2.78)	-3.22	(3.45)
10	-2.27	(3.14)	-5.90	(5.39)	-7.38	(3.51)

Key: Subtest 1 = Facial Identity Discrimination; 2 = Facial Affect Discrimination; 3 = Facial Affect Naming; 4 = Facial Affect Selection; 5 = Facial Affect Matching; 6 = Non-Emotional Prosody Discrimination; 7 = Emotional Prosody Discrimination; 8a = Name the Emotional Prosody; 8bi = Conflicting Emotional Prosody (Congruent); 8bii = Conflicting Emotional Prosody (Incongruent); 9 = Match Emotional Prosody to an Emotional Face; 10 = Match Emotional Face to the Emotional Prosody

6.3.2.1 Are patients impaired on facial emotion recognition?

Analysis of variance (ANOVA) with between-subjects factor of group was performed separately for subtests 2-5 (Facial Affect Discrimination, Facial Affect Naming, Facial Affect Selection, and Facial Affect Matching respectively).

Two subtests reached significance: Subtest 3 Facial Affect Naming, $F(2, 27) = 3.60$, $p < 0.05$, partial $\eta^2 = 0.21$; and Subtest 4 Facial Affect Selection, $F(2, 27) = 5.10$, $p < 0.01$, partial $\eta^2 = 0.27$. There was a significantly lower score for the RTL group compared with the Control group for Subtest 3 Facial Affect Naming, $t(1, 22) = 2.51$,

$p < 0.05$; and Subtest 4 Facial Affect Selection, $t(1, 22) = 2.88$, $p < 0.01$ whereas, the LTL group did not perform significantly worse than the Control group. When the ANOVAs were computed including Matrix Reasoning as a covariate, neither of the subtests remained significant. Subtest 3, $F(2, 26) = 1.02$, $p = 0.37$, partial $\eta^2 = 0.07$; subtest 4, $F(2, 26) = 2.18$, $p = 0.13$, partial $\eta^2 = 0.14$.

6.3.2.2 Is the recognition of fear particularly affected in facial and vocal emotion recognition?

A comparison of accuracy of recognition of fear was conducted between the three groups for Subtests 3, 4, 5, 8a, 9 & 10 (Facial Affect Naming, Facial Affect Selection, Facial Affect Matching, Name the Emotional Prosody, Match Emotional Prosody to an Emotional Face, and Match Emotional Face to the Emotional Prosody respectively), (the selection of fear being possible in these subtests only). ANOVAs were computed separately for each measure with a between-subjects factor of group. Two tests reached significance: Subtest 3 Facial Affect Naming, $F(2, 27) = 4.40$, $p < 0.05$, partial $\eta^2 = 0.25$; and Subtest 5 Facial Affect Matching, $F(2, 27) = 3.43$, $p < 0.05$, partial $\eta^2 = 0.20$. The LTL group performed significantly worse in recognition accuracy for fear than the Control group in Subtest 3, $t(1, 19) = 3.57$, $p < 0.005$ (98.33% & 79.17% respectively); and Subtest 5, $t(1, 19) = 2.51$, $p < 0.05$ (98.33% & 87.50% respectively). The RTL group did not differ significantly from the Control or LTL groups for fear recognition. When controlling for Matrix Reasoning, the results for Subtests 3 and 5 remained significant. Subtest 3, $F(2, 26) = 3.76$, $p < 0.05$, partial $\eta^2 = 0.22$; subtest 5, $F(2, 26) = 3.33$, $p < 0.05$, partial $\eta^2 = 0.20$.

6.3.2.2 Are patients worse at facial emotion than identity discrimination?

A two-way ANOVA conducted on Subtests 1 & 2 (Facial Identity Discrimination & Facial Affect Discrimination) with Task (Identity, Emotion) as the within-subjects factor; and Group as between-subjects factor (Control, RTL, LTL). The Task by Group interaction did not reach significance, $F(2, 27) = 2.08$, $p = 0.14$, partial $\eta^2 = 0.13$.

6.3.2.3 Are patients impaired on emotional prosody recognition?

An ANOVA with between-subjects factor of group was performed separately for subtests 7, 8a, 8bi & 8bii (Emotional Prosody Discrimination, Name the Emotional Prosody, and Conflicting Emotional Prosody (Congruent & Incongruent) respectively). Only Subtest 8bii (Conflicting Emotional Prosody Incongruent) reached significance, $F(2, 27) = 5.12$, $p < 0.01$, partial eta $\eta^2 = 0.28$. There was a significantly lower score for the RTL group compared with the Control group for Subtest 8bii, $t(1, 22) = 2.94$, $p < 0.01$. When the ANOVA were computed including Matrix Reasoning as a covariate, Subtest 8bii did not remain significant, $F(2, 26) = 1.85$, $p = 0.18$, partial $\eta^2 = 0.13$.

6.3.2.4 Are patients impaired on matching emotion in faces and voices?

A two-way ANOVA was conducted on Subtests 9 & 10 (Match Emotional Prosody to an Emotional Face & Match Emotional Face to the Emotional Prosody) with Direction (voice to face, face to voice) as the within-subjects factor; and Group (Control, RTL, LTL) as the between-subjects factor. The Direction by Group interaction did not reach significance, $F(2, 26) = 0.96$, $p = 0.40$, partial $\eta^2 = 0.07$.

6.3.3 The Empathy Quotient

The Control group scored 51.40 (SD = 8.46), RTL group scored 38.89 (SD = 11.69), and LTL scored 48.67 (SD = 10.33). A one-way between-groups ANOVA on EQ scores showed a significant effect of Group, $F(2, 27) = 4.62$, $p < 0.05$, partial $\eta^2 = 0.255$, with post-hoc analysis showing this was due to a higher score for the Control group compared to the RTL group, $t(1, 22) = 3.04$, $p < 0.01$. When the ANOVA was carried out with Matrix Reasoning as a covariate, this just failed to reach the conventional level of significance $F(2, 26) = 3.14$, $p = 0.06$, partial $\eta^2 = 0.19$. The percentage of participants scoring ≤ 30 points, the cut-off for poor social functioning (Baron-Cohen & Wheelwright, 2004), was 0% of the Control group; 44.44% of the RTL group; and 16.67% of the LTL group.

6.3.4 Social Functioning Scale

Analysis of variance (ANOVA) was performed separately for each SFS subscale with between-subjects factor of group. Three scales reached significance: the SFS full score, $F(2, 27) = 4.02$, $p < 0.05$, partial $\eta^2 = 0.25$; Interpersonal Communication subscale, $F(2, 27) = 7.69$, $p < 0.005$, partial $\eta^2 = 0.26$; and Independence-Competence subscale, $F(2, 27) = 6.02$, $p < 0.01$, partial $\eta^2 = 0.25$.

For each of the three scales there was a significantly higher score for the Control group compared with the RTL and the LTL groups [Full SFS scale Control v. RTL group, $t(1, 22) = 2.59$, $p < 0.05$; and Control v. LTL group, $t(1, 19) = 2.45$, $p < 0.05$;

Interpersonal Communication, Control v. RTL group $t(1, 22) = 4.26$, $p < 0.0005$; Control v. LTL group, $t(1, 19) = 3.45$, $p < 0.005$; Independence-Competence subscale, Control v. RTL group, $t(1, 22) = 3.41$, $p < 0.005$; and Control v. LTL group, $t(1, 19) = 3.24$, $p < 0.005$]. Refer to Table 6.2 below for the mean and S.D. values for the SFS.

When the ANOVAs were computed including Matrix Reasoning as a covariate, only the result for Interpersonal Communication remained significant, $F(2, 26) = 5.83$, $p < 0.01$.

Table 6.2 Mean standardised scores for the Social Functioning Scale

	Group					
	Controls (n = 15)		RTL (n = 9)		LTL (n = 6)	
Subscales	M	(S.D.)	M	(S.D.)	M	(S.D.)
Full SFS Score	123.09	(4.17)	116.41	(8.52)	117.71	(5.47)
Withdrawal	115.10	(9.77)	112.56	(16.56)	101.67	(6.90)
Inter Comm	145.00	(0.00)	125.22	(18.27)	128.17	(19.70)
Indep_Performance	118.77	(6.93)	112.83	(10.37)	109.42	(10.26)
Recreation	114.47	(12.42)	119.89	(15.85)	125.25	(15.61)
Prosocial	125.53	(10.34)	115.78	(16.82)	127.00	(4.56)
Indep_Competence	122.13	(3.36)	112.06	(10.73)	112.17	(11.09)
Occupation	120.60	(4.40)	116.56	(8.71)	120.33	(5.31)

Key: Withdrawal = Withdrawal and social engagement; Inter Comm = Interpersonal communication; Indep_Performance = Independence-performance; Indep_Competence = Independence-competence; Occupation = Occupation & employment.

6.3.4 Summary

The RTL group performed worse than the Control group in two facial emotion recognition tasks (Facial Affect Naming & Facial Affect Selection, and one emotional

prosody task (Conflicting Emotional Prosody Incongruent) in the FAB. The LTL group performed worse than the Control group in fear recognition within two facial emotion tasks (Facial Affect Naming & Facial Affect Matching). Within both patient groups, facial emotion and identity discrimination; vocal emotion and identity discrimination; emotional face and voice discrimination; emotional face and voice naming; and matching emotional voice to face and emotional face to voice were performed equally well. The RTL group performed better at face than voice identity discrimination, whilst the LTL group performed equally on both tasks. The RTL group performed significantly worse than the Control group on the EQ, with the 44% of the RTL group scoring ≤ 30 points, compared with 0% for the Control group, and 17% for the LTL group. The RTL & LTL groups scored significantly less than the Control group on the Full SFS scale, and two subscales: Interpersonal Communication & Independence-Competence.

6.3.5 Neural Correlates

Facial emotion recognition tasks in this chapter were compared with N170 responses from Chapter 5, in order to compare the deficits in emotion recognition of the RTL and LTL patients with diminished/slower neural responses reflected in the ERPs. To this end, the facial recognition tasks revealing deficits in the RTL group (Subtest 3 Facial Affect Naming, and Subtest 4 Facial Affect Selection) were compared with the observed diminished N170 response in the RTL group over left ventral electrodes for static stimuli; and the observed slower N170 response in the RTL group over dorsal

regions. Secondly, the facial recognition tasks revealing deficits in fear recognition for the LTL group (Subtest 3 Facial Affect Naming, and Subtest 5 Facial Affect Matching) were compared with the observed diminished N170 response over lateral sites. This limited analysis focused on comparing possible deficits observed in emotion recognition with specific reduced neural response reflected in the N170 component. A Pearson's correlation was performed between the N170 amplitude response for static stimuli over left ventral regions and the z-score for the facial emotion recognition tasks, however there was no significant correlation between these variables for any of the three groups. (Controls: $r = 0.29$, $p = 0.30$; RTL: -0.59, $p = 0.10$; LTL: -0.00, $p = 1.00$). This was then repeated for dynamic stimuli over left ventral regions, again there was no significant correlation between variables for any of the groups (Controls: 0.16, $p = 0.56$; RTL: -0.16, $p = 0.60$; LTL: 0.04, $p = 0.93$). A correlation was performed between the N170 latency response for static stimuli over dorsal regions and the z-score for the facial emotion recognition tasks, however there was no significant correlation between variable for any of the groups (Controls: 0.86, $p = 0.51$; RTL: 0.32, $p = 0.41$; LTL: -0.67, $p = 0.15$). This was then repeated for dynamic stimuli over dorsal regions, again there was no significant correlation between variables for any of these groups (Controls: 0.24, $p = 0.39$; RTL: -0.04, $p = 0.91$; LTL: 0.82, $p = 0.44$). A correlation was performed between the N170 amplitude response for static fear stimuli over lateral sites and the score for fear facial recognition tasks, however there was no significant correlation between these variables for any of the three groups (Controls: 0.28, $p = 0.32$; RTL: 0.22, $p = 0.56$; LTL: -0.27, $p = 0.60$). This was then repeated for dynamic fear over lateral sites, again there was no significant correlation between variables for any of the groups

(Controls: 0.29, p = 0.30; RTL: 0.42, p = 0.26; LTL: 0.02, p = 0.97). The correlations were still non-significant when collapsing across the groups.

6.4 Discussion

This study aimed to examine the behavioural recognition of facial and vocal emotion as well as social functioning in the same group of paediatric temporal lobectomy patients tested in Chapter 5, and to relate these findings to the ERP measures obtained in Chapter 5. The main predictions that were addressed in this study were that: 1) there would be deficits in emotion recognition for the RTL group; 2) there would be a reduction in social functioning for both patient groups, particularly the RTL group; 3) deficits in emotion recognition may be correlated with a diminished/slower electrophysiological response.

The main findings will be listed below and then following this summary, each result will be discussed in further detail.

1) The RTL group performed worse than the Control group in two facial emotion recognition tasks (Facial Affect Naming & Facial Affect Selection) and one emotional prosody task (Conflicting Emotional Prosody – Incongruent) in the FAB. The LTL group performed worse than the Control group in fear recognition within two facial emotion tasks (Facial Affect Naming & Facial Affect Matching). Within both patient groups, facial emotion and identity discrimination; vocal emotion and identity discrimination; emotional face and voice discrimination; emotional face and voice naming; and matching

emotional voice to face and emotional face to voice were performed equally well. The RTL group performed better at face than voice identity discrimination, whilst the LTL group performed equally on both tasks.

2) The RTL group performed worse than the Control group on the Empathy Quotient (EQ), with 44% of the RTL group scoring ≤ 30 points, compared with 0% for the Control group, and 17% for the LTL group. The RTL and LTL groups scored significantly less than the Control group on the Full Social Functioning Scale (SFS), and two SFS subscales: Interpersonal Communication and Independence-Competence.
3) The emotion recognition tasks and fear recognition were not correlated with the electrophysiological response.

6.4.1 Emotion recognition deficits

The groups were assessed for impairments in facial and vocal emotions using tasks from the Florida Affect Battery (FAB). Findings showed that the RTL group was impaired at recognising facial emotion in two tasks (Facial Affect Naming & Facial Affect Selection) and emotional prosody in one task (Conflicting Emotional Prosody – Incongruent). The Facial Affect Naming task assessed an individual's ability to verbally label an emotional expression displayed by an actor (angry, fearful, happy, sad and neutral). The Facial Affect Selection task assessed an individual's ability to identify a photograph (out of five different emotional expressions) that depicted the emotional expression (angry, fearful, happy, sad, neutral) named by the examiner.

These findings are consistent with previous research showing deficits in RTL groups for facial affect naming and identification (Benuzzi et al., 2004; Meletti et al., 2003, 2009). The Conflicting Emotional Prosody task assessed an individual's ability to identify the emotional tone (angry, happy, sad, neutral), ignoring the semantic content. The RTL group was impaired in identifying those emotional tones that were incongruent to the semantic content. There were no other impairments observed in emotional prosody for any of the groups, and the RTL group performed worse on voice compared with face identity discrimination. These findings suggest that it may be task difficulty as opposed to specific deficits in emotional prosody recognition per se, with auditory tasks generally being more difficult than visual tasks. Examining the effect of modality showed that facial stimuli were recognised better than vocal stimuli overall (z-scores of 0.44 and -2.31 respectively). The difficulty in auditory tasks has been observed previously, for both patient groups and healthy controls (Adolphs, 2002; Bonora et al., 2011). The two tasks matching emotional face and voice both have a high memory load, which would argue against the task difficulty being a factor. However, the cognitive loads on the separate task types may be different, one recruiting specific memory functioning and the other recruiting other cognitive processes, which is emphasised in the RTL group. Another view might be that the RTL group is indeed impaired at recognising emotional prosody but this deficit is only observed when the task is more complicated.

The LTL group was impaired at recognising fear within two facial emotion tasks (Facial Affect Naming & Facial Affect Matching). The Facial Affect Naming task was discussed above. The Facial Affect Matching task assessed an individual's ability to match two emotional expressions (angry, fearful, happy, sad, and neutral) posed by

different actors. The prediction was that the RTL group would be impaired compared with the other two groups, especially for fear, based on previous research showing specific deficits in fear recognition in RTL patients but not LTL patients (Adolphs et al, 2001; Anderson et al, 2000). This was not the case, with the LTL group showing a greater impairment on fear recognition compared with the Control group, which was not observed in the RTL group. This could be explained in the light of the electrophysiological findings: the N170 amplitude was reduced for the LTL group for fear stimuli. The LTL group may be impaired at processing facial fear stimuli and this is reflected in a deficit in recognising fear. However, when examining the relationship between the electrophysiological response for static fear and fear recognition in this group, there was no significant correlation. The disparity in the findings in this study that the LTL group was impaired in fear recognition, and previous research finding impairments in fear recognition for RTL patients only might be due to a number of factors. It is difficult to make conclusions based on populations that are so heterogeneous in clinical features such as age of seizure onset and extent of lesion etc. It does suggest, however, that amygdalar activity, which is thought to be influential in processing fearful stimuli, may be more severely reduced in the LTL group in this study. It is also possible that hemispheric specialisation may be less right-sided dominant resulting in greater deficits. This impairment in fear recognition was restricted to facial stimuli, with no impairments observed for fear in the auditory modality. This suggests a modality-specific deficit, however a conclusion cannot be drawn as the tasks were limited and task difficulty varied between modalities, as discussed above. Neither group were impaired on the facial or vocal identity tasks, demonstrating that the impairments observed in other tasks were specific to

emotional recognition and not face recognition or visuoperceptual deficits. These findings are consistent with previous research in this population (Benuzzi et al., 2004; Meletti et al., 2003, 2009).

6.4.2 Social judgments following paediatric temporal lobectomy

The RTL group performed significantly worse than the Control group on the EQ, with 44% of the RTL group scoring ≤ 30 points. This value was revealed as a useful cut-off for assessing empathising deficits in populations with Asperger Syndrome/high-functioning autism (Baron-Cohen & Wheelwright, 2004). In the LTL group, 17% also scored ≤ 30 points, demonstrating that there were deficits in empathising in this group as well, and that both populations were heterogeneous in empathising abilities. These findings suggest that the role of the anterior temporal lobe, especially the amygdala, extends to more complex social judgments as well as basic emotion recognition. Many of the empathy items also tap into what could be thought of as 'theory of mind' (ToM). Individuals with amygdala damage caused by early onset epilepsy have been shown to be impaired in higher-order ToM reasoning, such as detecting tactless or ironic comments or interpreting non-literal utterances (Shaw et al, 2004). This was evident in both right- and left-sided cases and encompassed both the beliefs and emotional states of others. Individuals with bilateral amygdala lesions were found to be impaired in judging the trustworthiness or approachability of other people from their faces (Adolphs et al, 1998), and amygdala activation in healthy individuals seems to correlate with untrustworthiness judgments (Winston et al, 2002). Complex emotional judgments are recognised

disproportionately by the eye region in the face (Baron-Cohen, Jolliffe, Mortimore, & Robertson, 1997; Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001). When making judgments about emotional states from images of the eye region, healthy individuals show activation of the amygdala but this was not found in autistic individuals (Baron-Cohen et al., 1999). This implies that normal functioning of social behaviour may be attributable in part to functional neuronal circuits involving the amygdala (Baron-Cohen et al, 2000). A study by Adolphs and colleagues (2002) showed that amygdalar damage (both unilateral and bilateral) impaired recognition of complex mental states and social emotions (such as guilt, arrogance, admiration). Future work might usefully compare an individual's own self-assessed EQ score with that based on the ratings by a partner or parent of that same individual.

6.4.3 Social functioning following paediatric temporal lobectomy

The SFS was initially designed to assess social functioning in individuals with schizophrenia, with the aim to improve quality of life through identification of an individual's needs and impairments. It addressed core components of daily functioning including social interaction, ability to perform skills necessary for independent living and engagement in social activities. It is intended to measure a continuous characteristic, whilst still being able to discriminate between different aspects of social functioning. The findings show that the RTL and LTL groups scored significantly less than the Control group on the Full Social Functioning Scale (SFS), and two SFS subscales: Interpersonal Communication and Independence-Competence. The Interpersonal Communication subscale explored the quality and

quantity of social interactions; and the Independence-Competence explored the ability of an individual to perform the skills necessary for independent living. The Independence-Competence is in contrast to the Independence-Performance subscale which explored how often these skills are performed. In summary, both RTL and LTL groups have a reduction in general social functioning compared to the control population and are particularly impaired in the engagement of social interactions and performing skills necessary for independent living. Disruption to the amygdala, hippocampus and surrounding temporal lobe structures may impair normal social functioning due to deficits in emotion processing, and a decreased memory function (Rausch, 2002). There is great variability in memory changes observed in patients after temporal lobectomy, and may depend on factors such as age of seizure onset and the extent of preoperative atrophy of the hippocampus (Martin et al, 2001).

6.4.4 Neural correlates

There was no significant correlation between the deficits in facial emotion recognition and the diminished/slower early-latency neural responses observed for the RTL and LTL groups. This suggests that the underlying neural activity responsible for recognition of emotional stimuli may not take place at early latencies during the initial perceptual processing of emotional stimuli but may occur at later stages when in-depth processing of affective information takes place. This lack of correlation could also be due to discrepancies between the task demands. The ERP task was a

passive task, so reducing allocation of attentional resources which would be present during the emotion recognition tasks.

6.4.5 Conclusions, limitations, and future directions

There were differences observed between groups in emotion recognition, with the RTL group demonstrating impaired recognition for facial and vocal emotions, and the LTL group demonstrating impaired fear recognition in facial stimuli. Neither group was impaired on face or voice identity suggesting that their face recognition and general visuoperceptual abilities were intact. Emotion recognition deficits were not observed consistently throughout tasks, raising the question of whether this was due to a differing cognitive load between tasks, or whether the recognition deficits were specific to the task. Generally both patient groups performed well on the tasks, and future research should include more challenging recognition tasks to elucidate the specific deficits. In general, the influence of IQ on the results suggests that the cognitive load could be a factor in recognition abilities. IQ has been proposed as a possible source for inconsistent findings of impaired facial expression evaluation in patients with amygdala damage (Adolphs et al., 1994). Anderson and colleagues (2000) did not find IQ was predictive of impairment in evaluating facial expressions in temporal lobectomy patients; and Adolphs and colleagues (2001) found a moderate correlation with VIQ but no difference in mean VIQ between the right- and left-sided groups. Meletti and colleagues (2009) found an effect of education on emotion recognition in patients with temporal lobe epilepsy but not the healthy controls. As is the case in the current study, the absence of a correlation between

education and facial emotion recognition in healthy controls could be consistent with the notion that emotion processing is a separate domain to that of other aspects of cognition and general intelligence. However, the influence of IQ on the patient group could reflect a link between general cognitive abilities and emotion recognition. There were limitations with the battery used – future research should aim to include colour photographs; pictures of male actors as well as female actors; moving images; and should include more socially complex stimuli such as guilt, embarrassment etc. In addition, less intense stimuli could be used, or morphed blends with varying intensities, which might serve to highlight more subtle deficits in emotion recognition. It is also difficult to directly compare the findings with previous research as a different battery of tests was administered. However, the Facial Affect Naming is similar to tasks used in previous studies and this was discussed above (Benuzzi et al, 2004; Meletti et al, 2003, 2009). The results of the EQ and SFS indicate that emotion perception extends outside recognising basic emotional states and deficits may be more pronounced in a socially-relevant context where more complex emotional signals are more frequently experienced. Future research should investigate whether deficits are observed in more complex social scenarios, or tasks such as 'Theory of Mind' (Shaw et al, 2004). It is difficult to generalise about the reduced electrophysiological response to static and dynamic emotional stimuli or deficits in emotion recognition in the population as a whole due to the heterogeneity in terms of clinical features. The influence of factors such as age of epilepsy onset, seizure frequency, age of surgery, location and size of the lesion etc, on cognitive functioning is still to be elucidated. It is not possible to conclude whether the cognitive deficits or reduced social functioning was present before surgery, however,

the disruption to the temporal lobe has implications for social functioning. Future research needs to explore functioning before and after surgery in more detail, to elucidate the effects of surgery on emotional processing. An investigation of individual differences should be performed to compare specific deficits with electrophysiological findings. In addition, a comparison of early-latency neural responses to emotional stimuli should be compared between different modalities. A critical age for neural development appears to be around five years of age; individuals before this age have more severe emotion recognition deficits (Meletti et al, 2003, 2009). This raises the question of neural plasticity and whether seizure activity early in life prevents functional reorganisation, influencing the development of emotion recognition abilities. Future research should explore this further by investigating the neural response to emotional stimuli in both early onset and later onset epilepsy patients. The population in this study, except two individuals, had seizure onset before five years of age, so it was not possible to address this question in this thesis. The differential response to static and dynamic emotional stimuli in this population suggests that the maturation of neural networks processing moving and static emotional stimuli is different in early development.

6.4.6 General Conclusion

The deficits observed for the RTL group were generalised to facial emotions, but not specifically fear. In particular, this group showed deficits in naming and affect selection and were not impaired for all tasks, and indeed showed better recognition for vocal emotions. This highlights that neural structures such as the amygdala could

be more sensitive to specific emotional modalities. In contrast, the LTL group did not show deficits in overall emotion recognition for either modality but was impaired in fear recognition. This suggests that either there is a higher degree of disruption to the amygdalo-cortical pathways in the LTL group compared with the RTL and Control group, or it implies that the left hemisphere may be more influential in emotion processing during development. Another possibility is that the behaviour observed in this study could be due to different social experiences individuals were exposed to during development due to their epilepsy and subsequent surgery, such as different schooling and educational experiences, as opposed to the pathology per se. It is also possible that epilepsy and the subsequent surgery had an impact on the social experiences individuals were exposed to during development, such as different schooling and educational experiences, and this may play a part in the deficits observed, alongside/as opposed to the pathology itself. It is not known when emotion processing abilities begin to develop or when they finally mature, or whether motion plays a role in the development of emotion perception skills. The social world is constantly changing and an infant will be exposed to dynamic social stimuli in their environment and this may be reflected in differences in processing static and dynamic images. The next chapter aims to address the developmental trajectory of emotion processing in young infants by investigating the early-latency responses to both static and dynamic emotional stimuli.

7. The effect of motion on the early-latency processing of emotional faces in infancy

7.1 Introduction

There has been progress in understanding the neural systems underlying emotion processing in adults, as discussed in Chapter 4. However, there has not been a great deal of research investigating the developmental trajectory of these neural networks in infancy. Studies have shown that cortical areas involved in face processing, such as the fusiform gyrus and STS, are functional in infants during development and exhibit some degree of specificity to faces (Tzourio-Mayoyer et al., 2002). Brain activation to faces may be more widespread in developmental populations: an fMRI study in 10-12-year-old children demonstrated that cortical activation is more distributed, only becoming more focussed in adulthood (Passarotti et al., 2003). The age at which these brain regions are fully matured and exhibit adult-like responses to faces and emotional expressions is still unknown. Infants demonstrate an early interest in face stimuli and can discriminate between positive and negative expressions from three months (Barrera & Maurer, 1981). By 5-7 months, an infant's visual system is sufficiently developed to support the discrimination of most basic emotions (Leppänen & Nelson, 2006). There is evidence that the amygdala network may be functional in 5-month-old infants. In a study by Balaban (1995), infants demonstrated an augmented eye blink startle response to loud noise when viewing angry facial expressions. This response is thought to be mediated by the amygdala (Funayama et al, 2001; Pissiota et al, 2003), and these findings suggest that the

amygdala may be responsive to emotional expressions early in development. It has also been speculated that the amygdala is involved in infants' enhanced attention and orienting to fearful faces that emerges by around seven months of age (Nelson, Morse, & Leavitt, 1979; Leppänen et al., 2007). This view is supported by studies in non-human primates demonstrating that cortico-amygdala projections are established soon after birth (Amaral & Bennett, 2000; Nelson et al, 2002).

7.1.1 ERP Studies of Emotion Processing

In infants, the early-latency infant N290 is thought to be a precursor to the adult N170 described in Chapter 1. Source localisation studies indicate that its generators overlap with those identified for the adult N170 that are part of the 'Core Cortical System' (Haxby et al., 2000), including the fusiform gyrus and STS (Johnson et al., 2005). The N290 shows a gradual specialisation for upright human faces over the first 12 months of life (de Haan & Nelson, 1999; de Haan et al., 2001; Halit et al., 2003). Its latency decreases from approximately 350-290ms between 3 and 12 months (Halit et al, 2003); and by 12 months the N290 latency is only 15-20ms longer than the observed latency of the N170 in 4-5-year-olds (Taylor et al, 1999, 2001). Although the N290 shows similarity to the adult N170 by 12 months, there are still differences in temporal and spatial characteristics, with the N290 having a more medial distribution, and a smaller peak amplitude to human faces than the adult N170. Throughout childhood the N170 is sensitive to face inversion and shows a rapid decrease in latency with age (Taylor et al, 1999, 2001, 2004), reaching adult levels only in later teenage years. The P1 is also sensitive to configural changes in

faces during childhood, with inversion effects observed and shows a marked decrease in latency across 4-15 years (Batty & Taylor, 2002, 2006; Taylor et al, 2004). The distribution of the P1 and N170 becomes more adult-like across this age range however, the evolution of the infant N290 to the adult N170 between 1-4 years is still unknown. This general latency decrease with age suggests an underlying neural system that is becoming more finely-tuned over time, with an increased processing speed.

As reviewed in the General Introduction (Chapter 1), some, but not all (e.g., Eimer et al, 2003; Eimer & Holmes, 2007) studies in adults have reported modulation of the P1 and N170 by different emotions, generally finding enhanced amplitudes and longer latencies for negative emotions (Batty & Taylor, 2003; Pourtois et al, 2005; Streit et al, 2003). A study of 4-15-year-old children suggested that emotion effects are observable first in the P1 at younger ages but in the N170 at older ages: 4-7-year-olds showed a longer latency P1 to fearful than to surprise and happiness, and a shorter latency to happiness than to fear, disgust and sadness (Batty & Taylor, 2006). In contrast, 14-15-year-olds showed no P1 effects but did show larger N170 amplitude for faces with negative emotions (anger, disgust, and fear) compared to positive emotions. The absolute latencies of the P1 and N170 were observed to be considerably shorter than found in other developmental studies using only neutral faces (Taylor et al 1999, 2001, 2004). These findings may suggest that a degree of emotional discrimination occurs very early in visual processing, even before the face-specific responses, but few studies have looked at emotion effects on the P1 in younger populations. There are few reports of the effects of emotional face processing on the early-latency components in infants and children. Studies with 7-

month-old infants have found a larger N290 amplitude to fearful compared to angry faces (Hoehl and Striano, 2008) but no difference in N290 or P1 among fearful, happy and neutral faces (Leppänen et al., 2007). This latter result is somewhat surprising, as the enhancement of the N170 response to fear in adults has been attributed to influence of the amygdala on the fusiform gyrus (Amaral & Price, 1984; Sugase et al, 1999; Vuilleumier et al, 2003, 2004), and the amygdala is believed to be functional early in infancy. It might be that this mechanism develops after 7 months of age, or that it is more easily activated in infants using more realistic moving faces. In summary, while there is evidence of sensitivity of different ERP components to emotional faces, including the P1 and N170 in children; and findings in adults that earlier ERP components, around 100-150ms are modulated by emotion (Batty & Taylor, 2003; Pizzagalli et al., 2002; Pourtois et al., 2004; Streit et al., 1999), there has not been a comprehensive investigation into the modulation of the P1 and the N290 in an infant population.

7.1.2 The Role of the Eye Region

Studies have shown that infants have a very early preference for eyes (Maurer, 1985), and sensitivity to gaze direction (Hood et al, 1998). The N290 amplitude is larger for faces with direct compared with averted gaze in static images (Farroni et al, 2002), whilst the amplitude of the N170 is unaffected (Taylor et al., 2001). Taylor and colleagues (2001) found that the N170 was larger for eye stimuli compared with upright, inverted, and phase-scrambled faces, and pictures of flowers between 4-15 years of age. This response diminished with increasing age, contrary to the response

to the other stimuli which either remained the same or increased (inverted faces). Eye detection may develop more quickly than face detection in infancy. This raises the question of whether the N290 is modulated by eye cues, and may be particularly sensitive to changes in the eye region in emotional expressions, e.g. wide eyes with visible sclera in fearful faces. A bias to negative emotions is also observed in development, with fearful faces eliciting a larger P400 and Nc response compared with happy faces in 7-month-old infants (Leppänen et al., 2007). Responses to eye gaze and negative emotions may be controlled by the amygdala's influence on cortical structures present early in development, enhancing processing of emotionally salient stimuli.

7.1.3 The Role of Motion

Even though infants can discriminate between static photographs of facial expressions, the question is whether this generalises to perceiving dynamic expressions such as infants would encounter in their normal social interactions. Infants can detect facial motion very early on in development (Spencer et al, 2006), and attend to biological motion over other forms of motion by twelve weeks (Bertenthal et al., 1993). Haviland & Lelwica (1987) found that 10-week-old infants discriminated between angry, happy, and sad expressions when displayed naturally by their mothers. Walker (1982) demonstrated that infants can discriminate between happy and sad at 5 months, and happy from neutral and angry at seven months. In this study by Walker, infants viewed dynamic videos of the facial expressions displayed alongside vocal emotion by a woman actor. A critical age of

development appears to be seven months, by which time infants can discriminate between different types of dynamic emotional expression within positive and negative affect categories (happy/interested, and angry/sad expressions) (Soken & Pick, 1999). In this study, infants viewed a video of moving facial expressions and heard a single vocal expression concordant with one of the facial expressions. A recent infra-red spectroscopy study indicated that there was increased activity in 9-13-month-old infants' anterior orbitofrontal cortex when they viewed their mother's smiling face compared with their mother's neutral face or a stranger's face (Minagawa-Kawai et al, 2009). The limitations of these studies are that none directly compared static and dynamic expressions, instead exposing the infants to dynamic stimuli only. In addition, the effects observed were restricted to specific age groups, namely ten weeks, and five, seven & 9-13-month-olds, limiting the conclusions that can be made on the developmental trajectory of dynamic emotion perception. In addition, the presence of vocal emotion in some of the studies may have enhanced discrimination. It is also difficult to draw any conclusions when there is no consistency between task demands or displayed expressions.

An interesting possibility is that infants could be more skilled at processing emotion from static faces than dynamic faces because of different rates of maturation of the visual pathways involved in processing form and motion. Research suggests that the dorsal visual stream undergoes a longer developmental time course than the ventral visual stream (Hickey, 1977), with both visual streams developing well beyond infancy. Studies directly comparing the two streams have used stimuli designed to selectively stimulate the two pathways, typical colour for the form pathway and

perceiving structure from movement for the motion pathway. Such studies show that motion coherence is slightly delayed in typical development (Gunn et al, 2002), with colour-contrast thresholds reaching adult levels at approximately 13 years, although detection of motion-identified forms do not reach maturity until 16 years of age (Hollants-Gilhuijs, Ruijter, & Spekreijse, 1998). Mitchell & Neville (2004) found an enhanced P1 to colour (blue and green high spatial frequency grating) compared to motion (low spatial frequency greyscale grating moving across the screen) stimuli, which decreased with age. P1 latencies for motion became shorter with age, being shorter for motion than colour stimuli in adult participants but not 6-7 or 8-9-year-olds.

7.1.4 Summary and Predictions

In summary, infants can clearly distinguish between different facial expressions early on in development and by seven months are able to differentiate between most emotions. Perception of motion matures through infancy and discrimination of biological and non-biological motion takes place by twelve weeks. It is not clear when infants can first discriminate between different dynamic expressions, but by ten weeks they can distinguish between positive and negative dynamic images, and by seven months between different types of positive and negative affect. These studies have used preferential allocation of attention (looking times) to elucidate whether discrimination occurs. To date, however, there has been no research investigating the difference in neural response to static and dynamic images of facial expressions in infants. Specifically, does dynamic emotional expression modulate

early-latency processing reflected in the P1 and N290? Motion tends to enhance neural activity in adults as demonstrated by a greater activation in structures normally associated with emotion processing, and an extended network, although the ERP results in Chapter 4 suggest that this enhancement might occur in later-latency processing as the P1 and N170 were larger for static expressions.

The specific questions and hypotheses to be addressed are as follows:

1. Static stimuli will produce an enhanced P1/N290 compared with dynamic stimuli: This prediction is based on a) previous research finding that the ventral visual stream develops earlier than the dorsal visual stream (Gunn et al, 2002; Hickey, 1997; Mitchell & Neville, 2004); b) previous findings in this thesis (Chapter 4) reporting this pattern of effects in adults.
2. Fearful faces may show an enhanced P1/N290 compared to happy faces: This is based on a) the response to eyes developing early (Farroni et al, 2002); b) an early maturation of the amygdalar network, responsible for infants' enhanced attention to fearful faces (Balaban, 1995; Funayama et al, 2001; Leppänen et al, 2007; Nelson et al, 1979; Pissiota et al, 2003); c) studies finding an enhanced N290 in 7-month-old infants to fearful faces compared to other emotions (Hoehl & Striano, 2008).
3. The topography of the P1/N290 will be influenced by motion: If dynamic stimuli are more optimal for activating the dorsal-STS pathway, and static stimuli the ventral-fusiform pathway, corresponding differences are expected in ERP topography.
4. The characteristics of the P1/N290 will change with age: Basic characteristics change between 4-12 months of age a) the N290 amplitude will increase and

latency decrease (Halit et al, 2003; Taylor et al, 1999); b) there will be a trend for the P1 to increase in amplitude over this age range (Halit et al, 2003); c) a change in medial to lateral distribution may be observed with age, although this may only be a trend, based on infant studies showing a medial distribution and adult studies demonstrating a lateral distribution (Batty & Taylor, 2003; Bentin et al, 1996; de Haan et al, 2002; Halit et al, 2003; Leppänen et al, 2007).

7.2 Methods

7.2.1 Participants

Sixty healthy infants, all reported to be developing normally by their caregiver, participated in the experiment, 29 female, aged 15.9 – 57.3 weeks (mean age 32.47, S.D 12.88). All were born full-term and were within the normal range for birth-weight. Data from 29 infants were used in the final statistical analyses, 14 female, aged 17.4 – 57.3 weeks (mean age 36.55, S.D. 12.36) evenly distributed across the age range. The 52% attrition rate was due to lack of sufficient data from individual infants because of artefacts in the EEG from eye and body movement (n=12); inattentiveness and fussiness (n=16) and procedural error (n=3) (see below for attrition rate discussion).

The study was approved by ICH/GOSH Research Ethics Committee (R&D reference 02NC01), and performed according to the standards of the Declaration of Helsinki (1964). Parental informed written consent was given for the infants prior to

participation in the study. Parents were paid £5 as a contribution towards travel costs, and infants were given an age-appropriate toy, for taking part in the study.

Recruitment of infants was a time-consuming process, there was no available database of healthy infants and so initial contact had to be made to recruit participants. Caregivers/parents and infants were recruited from a variety of environments: parent and baby film screenings in local cinemas; parent, baby and toddler groups; clinics at health centres and surgeries; infant playgroups at local libraries; and also through advertisement at University College London, and a local free newspaper (The Camden New Journal which is distributed in Camden, Islington & Westminster in London). The majority of parents and infants were recruited from the parent and baby film screenings in cinemas around London.

7.2.1.1 Attrition rate of study

Every attempt was made to reduce the attrition rate of the experiment. Mechanisms for increasing the quantity and quality of data for statistical analysis included:

- Placing the net accurately on the infant at the beginning and maintaining the correct placement for the duration of the experiment. This was achieved by using toys and blowing bubbles to distract the infant during placement of the sensor net. Infants tended to pull the net, so distraction for their hands was provided in the form of a toy to hold (so long as movement was kept to a minimum) or by asking their caregiver to hold their hands.

- Minimising the delay, after application of the net, in sitting the infant on the caregiver's lap in front of the monitor with the experiment running reduced fussiness and increased the number of useable trials.
- Minimising movement which caused artefacts and crucially making sure the infant attended to the presented visual stimuli for the length of the experiment (or as long as was feasibly possible). Advising the parent not to bounce the infant on their knee as this caused movement artefacts. If necessary, giving the infant a bottle, pacifier or rusk to hold to reduce movement and attention to the net. However, this had the potential to add artefacts to the data; there was a fine-line between keeping the data as clean as possible and adding exogenous factors which may have increased artefacts but ultimately allowed the collection of a greater number of trials and hence more useable data.
- Breaks between trials for the infant to feed or have parental attention increased the length of time the infant attended to the stimuli.
- Two researchers were present at each experimental session to help coordination of each stage of the session, facilitate the smooth running of the session, and to help reduce the time taken to bring the infant back to attending to the visual stimuli.
- The experimental session took place at a convenient time for the infant and parent, optimising the time of day when the infant would be most attentive. For example, choosing a time between feeding and sleeping sessions and when the infant was not unwell.

- The whole experimental session was recorded using a camera situated above the presentation screen, to allow the researcher to monitor the infant's visual status and looking time. This allowed the researcher to pause the recording and re-orientate the infant to the visual stimuli.

7.2.2 Materials

Static images and the corresponding dynamic morphed images of two female models (European-American) were selected to display the emotions fear and happiness (as discussed in Chapter 3). Emotions were displayed by only one model per infant, with models counterbalanced across infants, resulting in four different stimuli being presented to each infant.

7.2.3 Experimental Task

After a sensor net had been applied (refer to Chapter 2 for details of application), each infant sat on the caregiver's lap facing a computer screen surrounded by a black screen and in a dimly-lit room, minimising peripheral visual distractions. From this position the infant sat 60cm from the screen with a visual angle of 11° x 8°, however this distance was not always maintained throughout the entire experiment due to fussiness of the infant. The infant and caregiver were visible to the experimenter throughout the experiment via a digital video camera positioned on top of the screen. The camera recorded the infant's eye movements and allowed instant feedback to the experimenter as to whether the infant was attending to the stimuli.

Constant monitoring of the infant also allowed the experimenter to observe that the net was still in the correct position and that the infant was not handling the net or was in discomfort. The stimuli were presented on the monitor as soon as the infant was in place on the caregiver's lap and facing the monitor, to grab the infant's attention as soon as possible before they became distracted. Each stimulus was presented for 750 ms and, between images, a distractor was presented in the form of a concentric circle in black on a white background in the centre of the screen, with a random inter-stimulus interval between 1000-1400ms. Each stimulus was presented pseudo-randomly with the constraint that 1) no more than two consecutive stimuli would be the same and 2) that each stimulus in the set was shown before any were repeated.

The stimuli were presented continuously with no limit on exposure and only discontinued when the infant became too fussy or inattentive. This continued exposure was to optimise the number of good quality trials obtained from each infant as the attrition rate of data is very high in developmental studies involving young infants. To re-orientate attention of the infant or to counteract fussiness a distractor toy such as a rattle was used behind the monitor at pauses between trials**.** The caregiver was instructed to keep eye contact on the screen ahead and not to distract their child by pointing or vocalising. This was especially important in the case of the 10-12-month-olds who demonstrated social referencing.

7.2.4 Statistical Analyses

The channel groupings are those discussed in Chapter 2, section 2.1.5. The P1 peak was defined as the most positive peak in the time-window ranging from 135ms-251ms post-stimulus onset. The N290 peak was defined as the most negative peak in the time-window ranging from 219-359ms post-stimulus onset.

The number of trials originally presented to each infant varied considerably with each individual, dependent on attentiveness etc. For the final 29 infants, the mean percentage of original trials retained after ERP derivation (across infants) was 28.3% (S.D. = 7.92, range 12-38 trials), 28.7% (S.D. = 7.88, range 12-38 trials), 28.0% (S.D. = 7.97, range 12-37 trials), and 28.5% (S.D. = 7.92, range 12-39 trials) for the conditions Fear Static, Happiness Static, Fear Dynamic, and Happiness Dynamic respectively.

7.3 Results

To ascertain the effects of motion on the infant P1 and face-specific N290 component, the peak amplitude and latency values were submitted to a (2x2x2x2x2x3) repeated-measures ANOVA with within-subject factors of motion (2 levels: static, dynamic); emotion (2 levels: fear, happiness); hemisphere (2 levels: right, left); lateral-medial topography (2 levels: lateral, medial); and dorsal-ventral topography (2 levels: dorsal, ventral); and a between-subjects factor of age (3 levels: 4-7.2 months, 7.3-10.6 months, 10.7-13 months).

Outliers were identified and replaced with the variable means for each of the three age groups (4-7.2-month-olds; 7.3-10.6-month-olds; & 10.7-13-month-olds): 1.25%,

1.25% & 1.39% were replaced for the P1 amplitude; and 2.50%, 2.50% & 2.43% of values were replaced for the P1 latency; 3.44%, 3.44% & 3.47% of values were replaced for the N290 amplitude; and 1.25%, 1.25% & 1.04% of values were replaced for the N290 latency; as described in Chapter 2.

Regression analysis was performed to further examine the relationship between age and the peak N290 amplitude. The same approach as Carver and colleagues (2003) was used, in which the difference between two conditions was used as the dependent variable, Condition 1 minus Condition 2. For the N290, a larger response to Condition 1 resulted in a negative score. Multivariate regression was performed to determine whether the response from an individual condition or the interaction of the two conditions was correlated with age. Age-related effects were then compared to the repeated-measures ANOVA results, to reliably determine at which ages these differences were observed.

The static and dynamic conditions were compared for age effects in right lateral-medial and dorsal-ventral regions. This was based on the prediction that there would be different effects over these topographical areas. Research has shown that there is a bias to emotion-processing in the right hemisphere, which is apparent in infancy. Lateral-medial differences were explored, with a more pronounced effect over medial sites expected in early infancy; with the transition to lateral regions in later development towards adulthood. Research has also shown that static and dynamic stimuli are processed differently in the dorsal (motion) and ventral (colour and form) visual streams. The difference in the N290 peak response was defined as the static condition minus the dynamic condition.

7.3.1 P1

The P1 was bilaterally distributed and largest over left medial ventral electrodes, and quickest over medial dorsal electrodes.

7.3.1.1 P1 Amplitude

7.3.1.1.1 Do static stimuli produce an enhanced P1 compared to dynamic stimuli?

There was no significant main effect of Motion, however, there was a significant two-way interaction of Motion by Age $F(2, 26) = 3.25$, $p < 0.05$, partial $\eta^2 = 0.20$. Further analysis revealed that the static condition produced a significantly larger amplitude than the dynamic condition for the older age group only, $t(1, 8) = 2.81$, $p < 0.05$ (difference of 3.88μV). Mean peak amplitude and S.D. values are shown in Table 7.1 below. Also, refer to Figure 7.5 at the end of the results section.

Table 7.1 Mean peak amplitude (μV) and S.D. values for static and dynamic conditions across the three age groups

	Age Group					
	Younger Age Group 4-7.2 months (n = 10)		Middle Age Group 7.3-10.6 months (n = 10)		Older Age Group 10.7-13 months (n = 9)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Static	12.59	(4.61)	11.49	(4.49)	15.62	(3.98)
Dynamic	13.03	(3.40)	12.03	(4.37)	11.74	(2.33)

Regression analysis performed at right medial ventral sites, showed that there was a significant interaction between age and the difference in response to the static and dynamic conditions, $F(1, 27) = 9.41$, $p < 0.005$, $R^2 = 0.26$. The interaction between the

two conditions was significant, $\beta = 0.51$, $t = 3.07$, $p < 0.005$, and this was driven by a significant negative correlation between amplitude and age for the dynamic condition, $\beta = -0.47$, $t = -2.76$, $p < 0.01$, with a slight non-significant positive correlation for the static condition. As can be observed in Figure 7.1 below, younger infants tend to have a larger response to dynamic stimuli, and older infants to static stimuli.

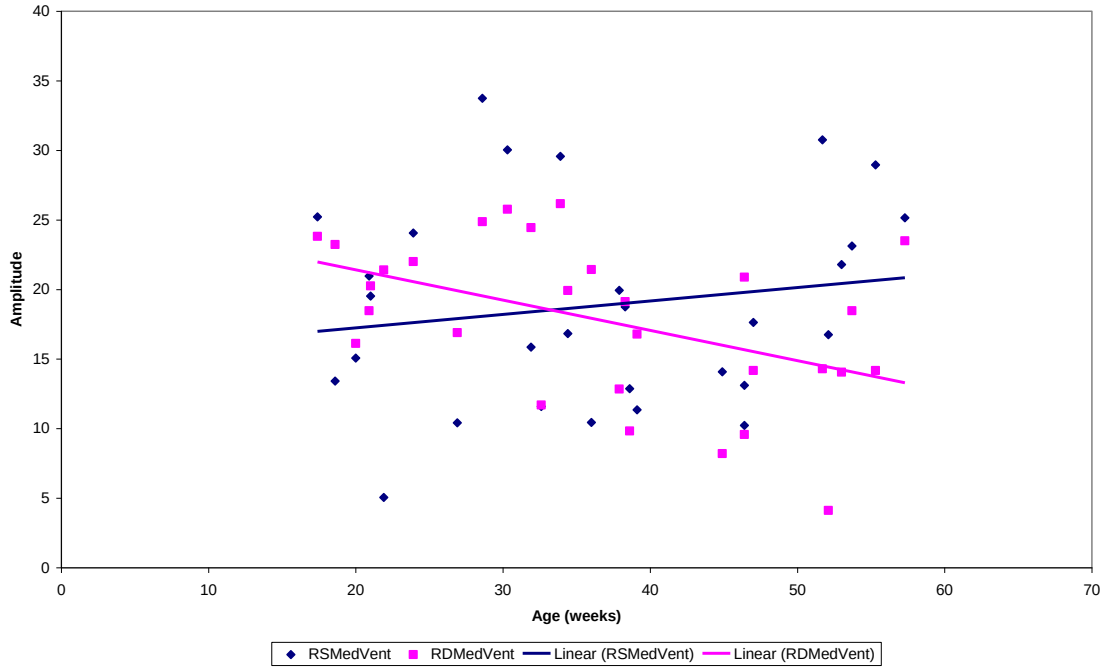


Figure 7.1 The relationship between infant age and P1 peak amplitude for the static and dynamic conditions over right medial ventral regions (amplitude in μV)

In summary, there was no significant main effect of Motion, however, older infants showed a larger response to static than dynamic stimuli, and the regression analysis indicated that the response to dynamic stimuli decreased with age and the response to static stimuli increased with age.

7.3.1.1.2 Do fearful faces produce an enhanced P1 compared to happy faces?

There was no significant main effect of Emotion. There was a significant two-way interaction of Emotion by Lateral-Medial topography, $F(1, 26) = 10.10$, $p < 0.005$, partial $\eta^2 = 0.28$; however post-hoc analysis revealed no significant difference between fear and happiness. There was also a significant three-way interaction of Emotion by Hemisphere by Lateral-Medial Topography, $F(1, 26) = 6.29$, $p < 0.05$, partial $\eta^2 = 0.20$. Further analysis revealed a significant two-way interaction of Emotion and Lateral-Medial Topography over the right hemisphere only $F(1, 26) = 14.36$, $p < 0.001$. However, post-hoc analysis revealed no significant difference between fear and happiness over right lateral or right medial sites.

There was a significant four-way interaction of Emotion by Hemisphere by Lateral-Medial Topography by Age, $F(2, 26) = 5.63$, $p < 0.01$, partial $\eta^2 = 0.30$. Further analysis revealed a significant three-way interaction Emotion by Hemisphere by Age over lateral sites only, $F(2, 26) = 4.17$, $p < 0.05$. There was a further significant two-way interaction of Emotion by Age over lateral sites in the left hemisphere only, $F(2, 26) = 5.80$, $p < 0.01$. Post-hoc analysis revealed that this significant interaction was driven by a trend towards a significantly larger amplitude for fear compared with happiness over left lateral sites for the younger age group, $t(1, 9) = 2.23$, $p = 0.053$ (difference of 2.89μV), and a trend towards a significantly larger amplitude for happiness compared with fear over left lateral sites for the older age group, $t(1, 8) = 2.12$, $p = 0.057$ (difference of 2.87μV). Mean peak amplitude and S.D. values are shown in Table 7.2 below. Also see Figure 7.5 at the end of the results section for the ERP waveforms.

Table 7.2 Mean peak amplitude (μV) and S.D. values for fear and happiness over left lateral electrodes for the three age groups

	Age Group					
	Younger Age Group 4-7.2 months (n = 10)		Middle Age Group 7.3-10.6 months (n = 10)		Older Age Group 10.7-13 months (n = 9)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Lateral Left Fear	14.42	(7.67)	13.31	(5.92)	13.16	(5.71)
Lateral Left Happiness	11.53	(7.94)	13.60	(7.16)	16.03	(6.27)

In summary, there was no significant main effect of Emotion, however, there was a larger response for fear compared with happiness over left lateral sites for the younger age group and a larger response for happiness compared with fear over left lateral sites for the older age group.

7.3.1.1.3 Does the topography differ for moving versus static faces?

There was a significant two-way interaction of Motion by Lateral-Medial Topography, $F(1, 26) = 20.51$, $p < 0.005$, partial $\eta^2 = 0.44$. The static condition produced a significantly larger amplitude than the dynamic condition over lateral sites only, $t(1, 28) = 2.37$, $p < 0.05$ (difference of 2.18μV). Mean peak amplitude and S.D. values are shown in Table 7.3 below.

Table 7.3 Mean peak amplitude and S.D. values for static and dynamic over lateral and medial sites

Category	Mean (μV)	(S.D.)
Lateral Static	10.67	(4.70)
Lateral Dynamic	8.49	(3.96)
Medial Static	15.64	(5.36)
Medial Dynamic	16.07	(4.75)

There was a significant three-way interaction of Motion by Lateral-Medial by Dorsal-Ventral Topography, $F(1, 26) = 5.84$, $p < 0.05$, partial $\eta^2 = 0.18$. Further analysis revealed a significant two-way interaction of Motion by Lateral-Medial Topography over dorsal regions only, $F(1, 28) = 31.50$, $p < 0.0005$. Post-hoc analysis revealed that for dorsal regions, there was a significantly larger amplitude for the static condition compared with the dynamic condition over lateral dorsal sites only, $t(1, 28) = 3.20$, $p < 0.005$ (difference of 2.72μV). Mean peak amplitude and S.D. values are shown in Table 7.4 below.

Table 7.4 Mean peak amplitude and S.D. values for static and dynamic conditions over dorsal-lateral and dorsal-medial sites

Category	Mean (μV)	(S.D.)
Dorsal Lateral Static	7.20	(3.91)
Dorsal Lateral Dynamic	4.48	(4.14)
Dorsal Medial Static	9.39	(5.65)
Dorsal Medial Dynamic	10.53	(5.02)

In summary, there was a larger response for the static condition compared with dynamic over lateral and lateral-dorsal sites.

7.3.1.1.3 Do the characteristics of the P1 amplitude change with age?

There was no significant main effect of Age and no significant interactions of Age and Topography.

7.3.1.1.4 Overall summary of the P1 amplitude

1) Older infants showed a larger response to static than dynamic stimuli, and the regression analysis indicated that the response to dynamic stimuli decreased with age and the response to static stimuli increased with age.

2) There was a larger response for fear compared with happiness over left lateral sites for the younger age group and a larger response for happiness compared with fear over left lateral sites for the older age group.

3) There was a larger response for the static condition compared with dynamic over lateral and lateral-dorsal sites.

4) There was no significant main effect of Age, and no significant interactions of Age and Topography.

7.3.1.2 P1 Latency

7.3.1.2.1 Do static stimuli produce a quicker P1 compared to dynamic stimuli?

There was no significant main effect of Motion, and no significant interactions of Motion and Age. Regression analyses showed that over right medial ventral sites, there was a significant relationship between age and the difference in response to the static and dynamic conditions, $F(1, 27) = 10.78$, $p < 0.005$, $R^2 = 0.29$. The interaction between the two conditions was significant, $\beta = 0.53$, $t = 3.28$, $p < 0.005$, and this was driven by a significant negative correlation between latency and age for the dynamic condition, $\beta = -0.38$, $t = -2.11$, $p < 0.05$, with no change for the static condition. As can be observed in Figure 7.2 below, younger infants tend to have a quicker response to static stimuli and older infants a quicker response to dynamic stimuli.

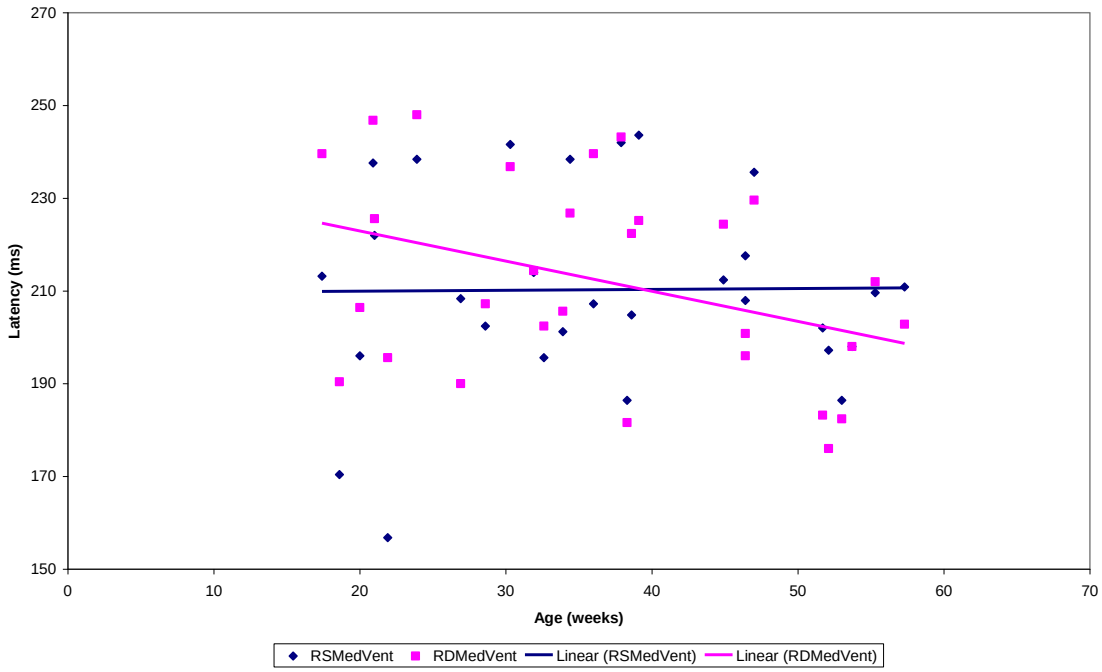


Figure 7.2 The relationship between infant age and P1 peak latency for the static and dynamic conditions over right medial ventral regions

7.3.1.2.2 Do fearful faces produce a quicker P1 compared to happy faces?

There was no significant main effect of Emotion, however, there was a trend towards a significant three-way interaction of Emotion by Hemisphere by Dorsal-Ventral Topography, $F(1, 26) = 3.69$, $p = 0.066$, partial $\eta^2 = 0.12$. Further analysis revealed a significant two-way interaction of Emotion by Hemisphere over the ventral regions only, $F(1, 28) = 4.19$, $p < 0.05$. There was a significantly shorter latency for fear compared with happiness over right ventral regions only, $t(1, 28) = 2.22$, $p < 0.05$ (difference of 7.81ms). Mean peak latency and S.D. values are shown in Table 7.5 below.

Table 7.5 Mean peak latency and S.D. values for fear and happiness over right and left ventral sites

Category	Mean (ms)	(S.D.)
Ventral Right Fear	208.64	(22.79)
Ventral Right Happiness	216.47	(16.62)
Ventral Left Fear	211.27	(21.15)
Ventral Left Happiness	212.48	(20.16)

There was a significant three-way interaction of Emotion by Dorsal-Ventral Topography by Age, $F(2, 26) = 4.90$, $p < 0.05$, partial $\eta^2 = 0.27$. There was a further significant two-way interaction of Emotion by Age over dorsal regions only, $F(2, 26) = 3.37$, $p < 0.05$. Post-hoc analysis revealed that there was a shorter latency for fear compared with happiness for the middle age group over dorsal regions, $t(1, 9) = 3.63$, $p < 0.005$ (difference of 8.02ms). Mean peak latency and S.D. values are shown in Table 7.6 below.

Table 7.6 Mean peak latency (ms) and S.D. values for fear and happiness over dorsal sites for the three age groups

	Age Group					
	Younger Age Group 4-7.2 months (n = 10)		Middle Age Group 7.3-10.6 months (n = 10)		Older Age Group 10.7-13 months (n = 9)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Dorsal Fear	197.16	(8.70)	188.60	(10.80)	196.92	(14.16)
Dorsal Happiness	201.88	(12.73)	196.62	(8.39)	191.41	(8.41)

A significant three-way interaction was observed of Emotion by Hemisphere by Age, $F(2, 26) = 4.87$, $p < 0.05$, partial $\eta^2 = 0.27$. Further analysis revealed a trend towards a significant two-way interaction of Hemisphere and Group for happiness only, $F(2, 26) = 3.04$, $p = 0.065$. Inspection of the means suggests that the interaction is driven by a shorter latency for the older infants compared with the younger infants over the right hemisphere for happiness.

In summary, there was no significant main effect of Emotion, however, there was a shorter latency for fear compared with happiness over right ventral regions. There was also a shorter latency for fear compared with happiness for the middle age group over dorsal regions. There is a pattern for the older infants to produce a shorter latency than the older infants over the right hemisphere for happiness.

7.3.1.2.3 Does the P1 topography differ for moving versus static faces?

There was a significant two-way interaction of Motion by Hemisphere, $F(1, 26) = 8.16$, $p < 0.01$, partial $\eta^2 = 0.24$. There was a significantly shorter latency for the

dynamic condition in the right hemisphere only, $t(1, 28) = 2.18$, $p < 0.05$ (difference of 4.21ms). Mean peak latency and S.D. values are shown in Table 7.7 below.

Table 7.7 Mean peak latency and S.D. values for static and dynamic conditions over right and left hemispheres

Category	Mean (ms)	(S.D.)
Right Static	204.07	(12.60)
Right Dynamic	199.86	(15.32)
Left Static	204.96	(13.97)
Left Dynamic	206.49	(14.35)

There was a significant three-way interaction of Motion by Hemisphere by Lateral-Medial Topography, $F(1, 26) = 4.29$, $p < 0.05$, partial $\eta^2 = 0.14$. Further analysis revealed a significant two-way interaction of Motion and Hemisphere over lateral sites only, $F(1, 28) = 7.65$, $p < 0.01$. There was a significantly shorter latency for the dynamic condition compared with the static condition over the right lateral sites only, $t(1, 28) = 2.44$, $p < 0.05$ (difference of 8.04ms). Mean peak latency and S.D. values are shown in Table 7.8.

Table 7.8 Mean peak latency and S.D. values for static and dynamic conditions over right and left lateral sites

Category	Mean (ms)	(S.D.)
Lateral Right Static	209.14	(17.67)
Lateral Right Dynamic	201.10	(24.84)
Lateral Left Static	214.43	(16.86)
Lateral Left Dynamic	216.79	(13.65)

There was a significant three-way interaction of Motion by Hemisphere by Age, $F(2, 26) = 4.26$, $p < 0.05$, partial $\eta^2 = 0.25$. Further analysis revealed that there was a significant two-way interaction of Motion by Age over the right hemisphere only, $F(2, 26) = 6.87$, $p < 0.005$. Post-hoc analysis revealed that the dynamic condition produced a significantly shorter latency than the static condition for the middle age group, $t(1, 9) = 4.42$, $p < 0.005$ (difference of 7.80ms), and the older age group, $t(1, 8) = 2.57$, $p < 0.005$ (difference of 9.36ms) over the right hemisphere. Mean latency and S.D. values are shown in Table 7.9 below.

Table 7.9 Mean peak latency (ms) and S.D. values for static and dynamic conditions over the right hemisphere for the three age groups

	Age Group					
	Younger Age Group 4-7.2 months (n = 10)		Middle Age Group 7.3-10.6 months (n = 10)		Older Age Group 10.7-13 months (n = 9)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Right Static	207.23	(16.40)	205.60	(7.55)	198.84	(11.96)
Right Dynamic	211.25	(17.48)	197.80	(10.19)	189.48	(8.52)

There was a significant four-way interaction of Motion by Emotion by Lateral-Medial Topography by Group, $F(2, 26) = 5.30$, $p < 0.05$, partial $\eta^2 = 0.29$. Further analysis revealed that there was a significant three-way interaction of Motion by Lateral-Medial topography by Age for fear only, $F(2, 26) = 4.85$, $p < 0.05$. There was a further trend towards significance for a two-way interaction of Motion by Age for fear over medial sites only, $F(2, 26) = 3.30$, $p = 0.053$. Post-hoc analysis revealed a significantly shorter latency for static fear compared with dynamic fear for the younger age group, $t(1, 9) = 2.84$, $p < 0.05$ (difference of 9.91ms), and the middle age group, t(1,

9) = 2.65, $p < 0.05$ (difference of 5.45ms) over medial sites. Mean peak latency and S.D. values are shown in Table 7.10 below.

Table 7.10 Mean peak latency (ms) and S.D. values for static and dynamic conditions for fear over the medial sites

	Age Group					
	Younger Age Group 4-7.2 months (n = 10)		Middle Age Group 7.3-10.6 months (n = 10)		Older Age Group 10.7-13 months (n = 9)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Fear Medial Static	193.62	(16.73)	190.47	(18.09)	198.05	(21.48)
Fear Medial Dynamic	203.56	(17.84)	195.92	(17.43)	192.91	(19.48)

In summary, there was a shorter latency for the dynamic condition compared with the static over right and right lateral sites. There was a significantly shorter latency for the dynamic condition compared with static over the right hemisphere for the middle age group and the older age group. There was a shorter latency for static fear compared with dynamic fear over medial sites for the younger and middle age groups.

7.3.1.2.4 Do the characteristics of the P1 change with age?

There was no significant main effect of Age, or significant interactions of Age and Topography.

7.3.1.2.5 Overall summary of the P1 latency

1) Younger infants tend to have a quicker response to static stimuli and older infants a quicker response to dynamic stimuli.

2) There was a shorter latency for fear compared with happiness over right ventral regions. There was also a shorter latency for fear compared with happiness for the middle age group over dorsal regions.

3) There was a shorter latency for the dynamic condition compared with the static over right and right lateral sites. There was a significantly shorter latency for the dynamic condition compared with static for the middle age group and the older age group over the right hemisphere. There was a shorter latency for static fear compared with dynamic fear over medial sites for the younger and middle age groups.

4) There was no significant main effect of Age, or interactions of Age and Topography.

7.3.2 N290

The N290 was larger over right lateral dorsal electrodes, and quickest over medial electrodes.

7.3.2.1 N290 Amplitude

7.3.2.1.1 Do static stimuli produce an enhanced N290 compared to dynamic stimuli?

There was no significant main effect of Motion. There were no significant interactions between Motion and Age. To explore whether the N290 response to static and dynamic stimuli differed across age, a regression analysis was conducted (as described above). Previous research has shown that the N290 response in infants

of this age is maximal over right medial ventral electrodes. Over this region, there was a significant relationship between age and the difference in response to the static and dynamic conditions, $F(1, 27) = 8.75$, $p < 0.01$, $R^2 = 0.25$. The interaction between the two conditions was significant, $\beta = 0.50$, $t = 2.96$, $p < 0.01$, and this was driven by a significant positive correlation between amplitude and age for the dynamic condition, $\beta = -0.41$, $t = -2.36$, $p < 0.05$, with a non-significant negative correlation for the static condition. As can be observed in Figure 7.3 below, younger infants tend to have a larger response to static stimuli, and older infants to dynamic stimuli.

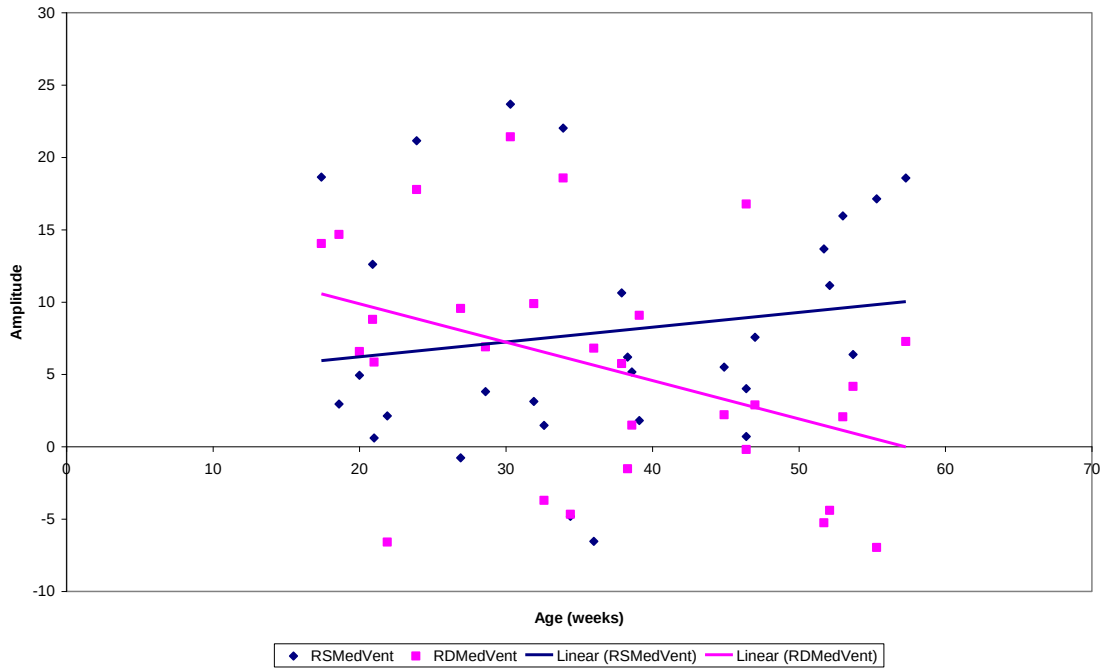


Figure 7.3 The relationship between infant age and N290 peak amplitude for the static and dynamic conditions over right medial ventral regions (amplitude in μV)

7.3.2.1.2 Do fearful faces produce an enhanced N290 compared to happy faces?

There was no significant main effect of Emotion, however, there was a significant two-way interaction of Emotion by Lateral-Medial Topography, $F(1, 26) = 6.18$, $p < 0.05$, partial $\eta^2 = 0.19$. There was, however, no significant difference between fear and happiness over either lateral or medial sites. There was also a significant three-way interaction of Emotion by Hemisphere by Lateral-Medial Topography, $F(1, 26) =$, $p < 0.005$, partial $\eta^2 = 0.27$. Further analysis revealed that over medial sites only, there was a significant two-way interaction of emotion and hemisphere, $F(1, 28) = 4.39$, $p < 0.05$, partial $\eta^2 = 0.14$. Fear produced a more negative amplitude than happiness over right medial sites only, $t(1, 28) = 2.37$, $p < 0.05$ (difference of 2.02μV). Mean peak amplitude and S.D. values are shown in Table 7.11 below.

Table 7.11 Mean peak N290 amplitude & S.D. values for fear and happiness over medial sites

Category	Mean (μV)	(S.D.)
Medial Right Fear	1.30	(4.89)
Medial Right Happiness	3.32	(4.76)
Medial Fear Left	8.69	(4.76)
Medial Happiness Left	8.52	(7.08)

7.3.2.1.3 Does the N290 topography differ for moving versus static faces?

There was a significant two-way interaction of Motion by Lateral-Medial Topography, $F(1, 26) = 10.04$, $p < 0.005$, partial $\eta^2 = 0.28$. The dynamic condition produced a more negative amplitude than the static condition over lateral sites only,

t(1, 28) = 2.79, p < 0.01 (difference of 2.90μV). Mean peak amplitude and S.D. values are shown in Table 7.12 below.

Table 7.12 Mean peak N290 amplitude & S.D. values for the motion conditions over lateral-medial sites

Category	Mean (μV)	(S.D.)
Lateral Static	2.31	(4.10)
Lateral Dynamic	-0.59	(5.46)
Medial Static	5.43	(0.98)
Medial Dynamic	5.48	(1.03)

There was a significant three-way interaction of Motion by Lateral-Medial by Dorsal-Ventral Topography, $F(1, 26) = 8.31$, $p < 0.01$, partial $\eta^2 = 0.24$. Further analysis revealed that over dorsal regions only, there was a significant two-way interaction of Motion by Lateral-Medial Topography, $F(1, 28) = 15.96$, $p < 0.0005$, partial $\eta^2 = 0.36$. The dynamic condition produced a significantly more negative amplitude than the static condition over dorsal lateral sites, $t(1, 28) = 3.87$, $p < 0.001$ (difference of 3.72μV), but not dorsal medial sites. Mean peak amplitude and S.D. values are shown in Table 7.13 below.

Table 7.13 Mean peak N290 amplitude & S.D. values for the motion conditions over dorsal-lateral and dorsal-medial sites

Category	Mean (μV)	(S.D.)
Dorsal Lateral Static	0.29	(3.58)
Dorsal Lateral Dynamic	-3.43	(5.59)
Dorsal Medial Static	-0.35	(4.60)
Dorsal Medial Dynamic	0.97	(5.73)

In summary, there was a larger response for the dynamic condition compared with static over lateral and dorsal lateral sites.

7.3.2.1.4 Do the N290 characteristics change with age?

There was no significant main effect of Age. Regression analysis was performed: the relationship between age and the difference in response to the lateral and medial conditions in right ventral regions was non-significant. However, there was a significant interaction of lateral-medial topography over right dorsal regions, $F(1, 27) = 3.73$, $p < 0.05$, $R^2 = 0.12$. The interaction between the two conditions was significant, $\beta = -0.35$, $t = -1.93$, $p < 0.05$, with a non-significant positive correlation over the lateral sites, and a non-significant negative correlation over the medial sites, see Figure 7.4 below. The response was similar over lateral and medial sites in young infants, but for older infants the response decrease in medial sites and increased in lateral sites.

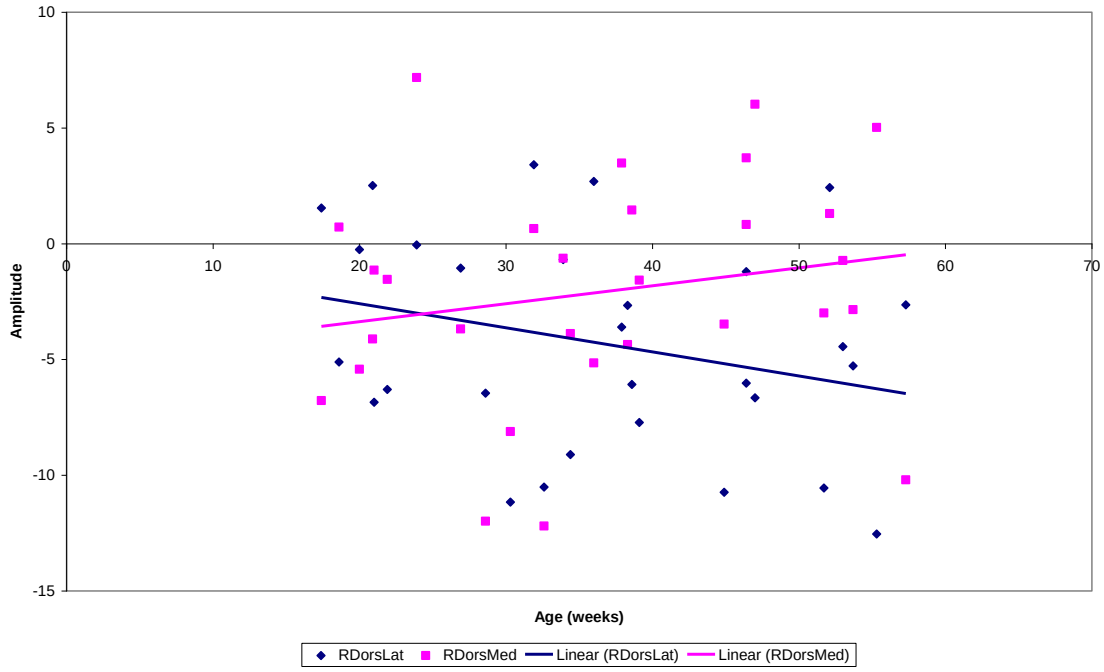


Figure 7.4 The relationship between infant age and N290 peak amplitude for lateral-medial topography over right dorsal regions (amplitude in µV)

In summary, there was no significant main effect of Age. The response was similar over lateral and medial sites in young infants, but for older infants the response decreased in medial sites and increased in lateral sites.

7.3.2.1.5 Overall summary of the N290 amplitude

1) Younger infants tended to have a larger response to static stimuli and older infants to dynamic.

2) Fear produced a larger amplitude than happiness over right medial sites.

3) Dynamic stimuli produced a larger amplitude than static stimuli over lateral, and dorsal lateral sites.

4) There was no significant main effect of Age. The response was similar over lateral and medial sites in young infants, but for older infants the response decreased in medial sites and increased in lateral sites.

7.3.2.1.6 Peak-to-peak analysis

In order to investigate whether the differences in amplitude observed for the N290 reflected processes occurring in the N290 time-window or were effects carried over from the amplitude differences already present at the P1 (e.g., a smaller P1 resulting in a larger N290), a peak-to-peak ANOVA analysis was conducted (the difference between the peak P1 amplitude and the peak N290 amplitude). If the effects were specific to N290, they should still be significant in the peak-to-peak analysis; however if they were carried over from P1 then the interactions would drop to non-significance. Analyses showed that interactions that were significant for the N290 amplitude were non-significant for the peak-to-peak analysis, indicating the N290 findings were influenced by prior differences at the P1.

7.3.2.1 N290 Latency

7.3.2.1.1 Do static stimuli produce a quicker N290 compared to dynamic stimuli?

There was no main effect of Motion. There were no significant interactions between Motion and Age. To explore whether the N290 response to motion might be different across the age groups, a regression analysis was conducted (as described

above). Regression analysis confirmed that there was no difference in latency between static and dynamic stimuli across ages.

7.3.2.1.2 Do fearful faces produce a quicker N290 compared to happy faces?

There was no main effect of Emotion, however, there was a significant three-way interaction of Motion by Emotion by Dorsal-Ventral Topography, $F(1, 26) = 13.82$, $p < 0.001$, partial $\eta^2 = 0.35$. Further analysis for the static condition revealed a significant two-way interaction of Emotion by Dorsal-Ventral Topography, $F(1, 26) = 7.96$, $p < 0.01$, partial $\eta^2 = 0.22$. Static fear produced a significantly shorter latency than static happiness over ventral regions only, $t(1, 28) = 2.17$, $p < 0.05$. For the dynamic condition, there was a significant two-way interaction of Emotion by Dorsal-Ventral Topography, $F(1, 26) = 4.28$, $p < 0.05$, partial $\eta^2 = 0.13$. However, there were no further significant differences between fear and happiness for either dynamic dorsal or dynamic ventral. Mean peak latency and S.D. values are shown in Table 7.14 below.

Table 7.14 Mean peak N290 latency & S.D. values for the emotion conditions over dorsal-ventral regions

Category	Mean (ms)	(S.D.)
Static Dorsal Fear	296.69	(22.26)
Static Dorsal Happiness	292.99	(21.74)
Static Ventral Fear	291.57	(30.21)
Static Ventral Happiness	299.37	(27.92)

There was a significant two-way interaction of Emotion by Age, $F(2, 26) = 3.54$, $p < 0.05$, partial $\eta^2 = 0.21$. Further analysis revealed that there was a significantly shorter latency for happiness compared with fear for the oldest age group only, $t(1, 8) = 2.47$, $p < 0.05$ (difference of 5.54ms). Mean peak latency and S.D. values are shown in Table 7.15 below.

Table 7.15 Mean peak N290 latency (ms) and S.D. values for fear and happiness for the three age groups

	Age Group					
	Younger Age Group 4-7.2 months (n = 10)		Middle Age Group 7.3-10.6 months (n = 10)		Older Age Group 10.7-13 months (n = 9)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Fear	290.41	(24.26)	296.28	(17.46)	294.48	(18.95)
Happiness	295.31	(20.17)	300.93	(15.78)	288.94	(16.05)

There was a trend towards a significant three-way interaction of Emotion by Lateral-Medial Topography by Age $F(2, 26) = 3.02$, $p = 0.066$, partial $\eta^2 = 0.19$. Further analysis revealed a significant two-way interaction of Emotion by Age over medial sites only, $F(2, 26) = 3.72$, $p < 0.05$. Post-hoc analysis revealed that there was a significantly shorter latency for happiness compared with fear in medial sites for the older age group only, $t(1, 8) = 2.89$, $p < 0.05$ (difference of 9.22ms). Mean peak latency and S.D. values are shown in Table 7.16 below.

Table 7.16 Mean peak N290 latency (ms) and S.D. values for fear and happiness over medial sites for the three age groups

	Age Group					
	Younger Age Group 4-7.2 months (n = 10)		Middle Age Group 7.3-10.6 months (n = 10)		Older Age Group 10.7-13 months (n = 9)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Medial Fear	283.94	(28.06)	297.89	(20.05)	294.46	(21.74)
Medial Happiness	291.07	(21.86)	296.91	(15.39)	285.24	(20.58)

In summary, there was no significant main effect of Emotion, however static fear produced a shorter latency than static happiness over ventral regions. The latencies were similar for fear and happiness in younger infants, and quicker for happiness compared with fear in the older infants, and this was particularly pronounced in medial sites.

7.3.2.1.3 Does the topography differ for moving versus static faces?

There were no significant motion effects on topography.

7.3.2.1.4 Do the characteristics of the N290 change with age?

There was no significant main effect of Age. There was a significant three-way interaction of Hemisphere by Lateral-Medial Topography by Age, $F(2, 26) = 3.78$, $p < 0.05$, partial $\eta^2 = 0.22$. Further analysis revealed a significant two-way interaction of Lateral-Medial Topography by Age over the left hemisphere only, $F(2, 26) = 5.00$, $p < 0.05$. Post-hoc analysis revealed a significantly shorter latency for medial compared

with lateral sites over the left hemisphere for the younger age group only, $t(1, 9) = 4.97$, $p < 0.001$ (difference of 21.26ms). Mean peak latency and S.D. values are shown in Table 7.17 below.

Table 7.17 Mean peak N290 latency (ms) and S.D. values for lateral and medial sites over the left hemisphere

	Age Group					
	Younger Age Group 4-7.2 months (n = 10)		Middle Age Group 7.3-10.6 months (n = 10)		Older Age Group 10.7-13 months (n = 9)	
	M	(S.D.)	M	(S.D.)	M	(S.D.)
Left Lateral	302.32	(28.11)	299.93	(16.64)	298.24	(19.26)
Left Medial	281.06	(25.29)	297.39	(16.89)	287.60	(20.26)

7.3.2.1.5 Overall summary of the N290 Latency

1) There was no significant main effect of Motion and no effects of age on motion.

2) Static fear produced a shorter latency than static happiness over ventral regions. The latencies were similar for fear and happiness in younger infants, and quicker for happiness compared with fear in the older infants and this was particularly pronounced in medial sites.

3) There were no motion effects on topography.

4) There was a shorter latency for medial compared with lateral sites over the left hemisphere for the younger age group only.

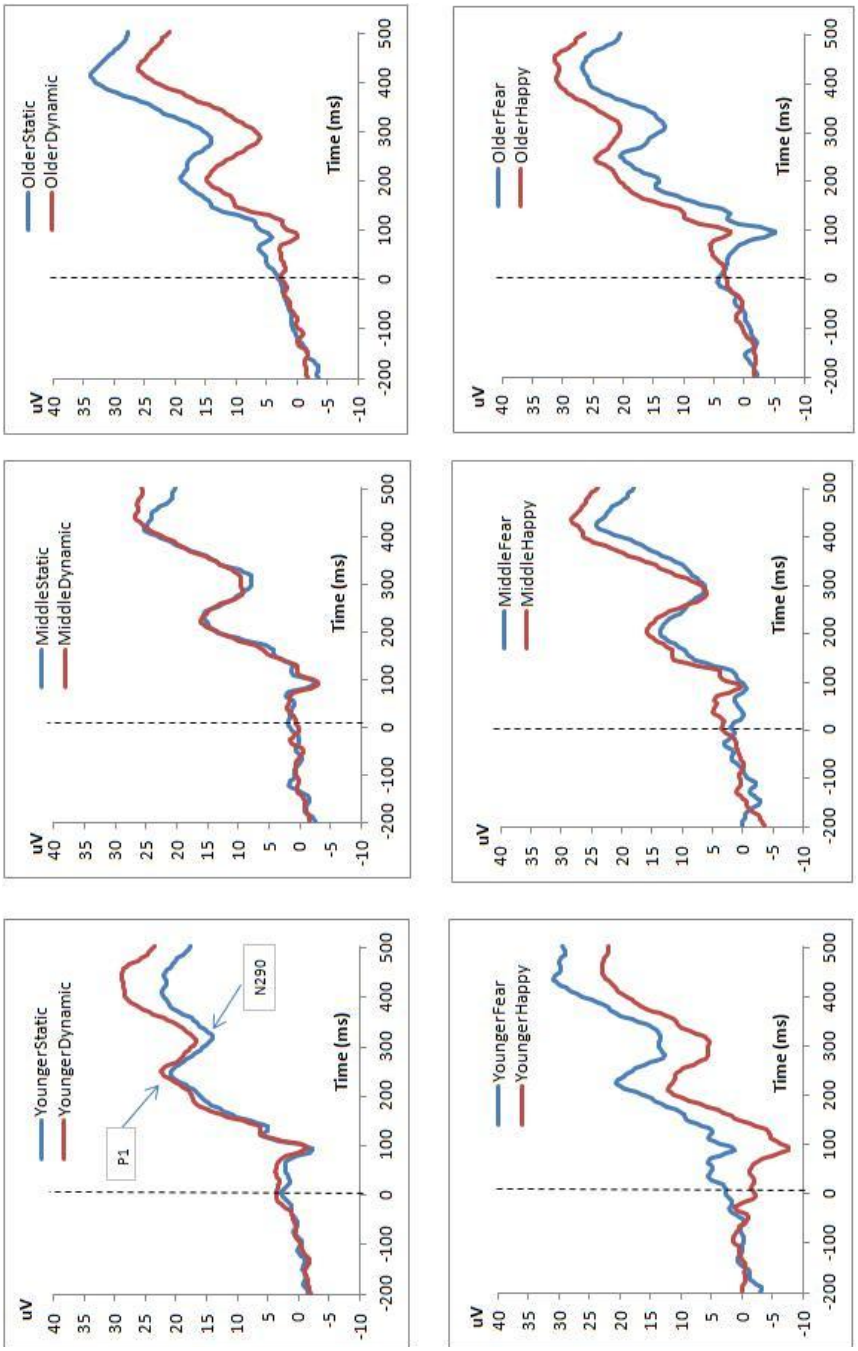


Figure 7.5 ERP waveforms illustrating the P1 and N290 for static and dynamic conditions over right medial ventral sites (top three figures); and for fear and happiness over left lateral sites (bottom three figures) for younger, middle and older aged infants.

7.4 Discussion

The main predictions that were addressed in this study were that: 1) static stimuli would produce an enhanced P1 and N290 compared with dynamic stimuli; 2) fearful faces may show an enhanced P1 and N290 compared to happy faces; 3) the topography of the P1 and N290 would be influenced by motion; 4) the characteristics of the P1 and N290 will change with age.

The main findings will be listed below, and following this summary, each result will be discussed in turn (with predictions 1 and 3 discussed first).

In summary: There was a differential P1 response for static and dynamic stimuli, which was modulated by age, emotion and topography. Motion did not affect the N290 response.

More specifically:

1) The P1 response was larger and slower for dynamic stimuli in younger infants and larger and slower for static stimuli in older infants. The N290 response was larger for static stimuli in younger infants and larger for dynamic stimuli in older infants; with no effect of age on the P1 latency of static and dynamic stimuli.
2) The P1 response was larger for fear over lateral sites in younger infants and larger for happiness in the older infants. The P1 response was quicker for fear over right ventral regions and quicker for the middle age infant group over dorsal regions. The N290 response to fear was larger over right medial sites. The N290 response was quicker for static fear over ventral regions. The N290

latencies were similar for fear and happiness in younger infants and quicker for happiness in older infants, particularly in medial sites.

3) The P1 response was larger for static stimuli over lateral and lateral-dorsal sites. The P1 response was quicker for dynamic stimuli over right and right-lateral sites; quicker for dynamic stimuli for the middle age group and older infants over the right hemisphere; and quicker for static fear compared with dynamic fear in younger and middle age group infants over medial sites. The N290 response was larger for dynamic stimuli over lateral and lateral-dorsal sites, with no motion effects on the topography of the N290 latency.
4) The N290 response was similar over lateral and medial sites in younger infants, but for older infants the response decreased in medial sites and increased in lateral sites. The N290 response was quicker for medial sites compared with lateral over the left hemisphere for the younger infants.

7.4.1 The P1 and N290 response to static and dynamic facial expressions

There was no main effect of Motion for the P1 or N290, however, when examining the response to motion across the age range, findings suggest that the effect of motion varied with age for both components. On closer inspection, the amplitude effects were opposite for the P1 and N290, with a larger P1 and smaller N290 for dynamic stimuli in younger infants and a larger P1 and N290 for static stimuli in older infants. This contrasting effect was observed particularly over lateral and lateral-dorsal sites, with static stimuli producing a larger P1 response and dynamic stimuli producing a larger N290 response. A peak-to-peak analysis resulted in non-

significance for those effects observed in the N290. As the N290 effects disappeared when taking into account the P1 findings, this suggests that the P1 deflection was influencing the N290 peak amplitude and motion does not directly affect the amplitude of the N290. This suggests that motion affects the initial perceptual stages of processing emotional stimuli, reflected in the P1, but not specific face processing stages, reflected in the N290. This is in comparison to the findings in adults (Chapter 4), where both stages of processing were modulated by motion, the P1 and N170. This indicates that the underlying neural mechanisms may be different for infants and adults at this early stage in visual perception. Dynamic stimuli produced a larger, slower P1 response in young infants compared with static stimuli, with the reverse being true in older infants. Older infants are showing a similar pattern of response to the adults in terms of amplitude of the P1. This suggests that neural structures responsible for the early perceptual processing of moving and static emotional face stimuli may mature throughout infancy and may start to show signs of adult-like responses towards the end of the first year of life. This is in line with the observations that the responses of the P1 and N290 become more adult-like in this first year, with an increased specialisation towards upright human faces (de Haan et al, 2003; Halit et al, 2003).

Further motion effects were observed in specific regions of the scalp, with a larger P1 response for static stimuli over lateral and lateral-dorsal sites, and a quicker P1 response for dynamic stimuli over right and right-lateral sites. In addition, the P1 response was quicker for dynamic stimuli for the middle and older age groups over the right hemisphere, and quicker for static fear compared with dynamic fear in the younger and middle age groups over medial sites. Generally, the P1 response is

slower for dynamic stimuli, particularly in the younger infants. The findings that young infants produce a larger, slower P1 response compared with older infants and adults suggest that, at this age, the underlying neural structures and networks may be immature. There may be additional feedback loops between cortical and subcortical structures, such as the amygdala and the dorsal visual cortices, involved in processing dynamic emotional stimuli which may result in the enhanced but delayed response. It is thought that the ventral visual stream develops earlier than the dorsal stream (Gunn et al, 2002; Hickey, 1997; Mitchell & Neville, 2004). The maturation of this system in the first year of life may lead to the faster response observed in older infants for dynamic stimuli, as the dorsal visual areas become finely-tuned to moving face stimuli. It is also possible that the dynamic stimuli result in a larger allocation of attention compared with static stimuli in this age group, which would be reflected in the P1, and might explain why these effects were not observed on the N290. In Chapter 4, there was a clear differential N170 response between static and dynamic stimuli however, the results in infants indicate that the N290 is not affected by motion. As discussed above in terms of the P1, this may be due to differences in the underlying neural networks. Although the general perceptual processing may be affected by motion, reflected in the P1, the specific activity required for face processing, reflected in the N290, may not be sensitive to motion at this stage in development. It is possible that as the N290 becomes more specialised in face processing motion effects will be observed. The P400, a positive deflection occurring after the N290, becomes more finely-tuned to faces throughout development. It is possible that the two components reflect processes that become integrated with each other to eventually produce the mature N170 (de Haan et al.,

2002). These distinct or overlapping processing stages reflected in the two components may become integrated after the first year of life but, before this point in development, the processing stages producing the P400 may be influenced by motion, whereas the N290 is unaffected.

7.4.2 The P1 response to fearful and happy expressions

The prediction was that fearful faces may show an enhanced response compared to happy faces. Findings showed that there was no main effect of Emotion however, the P1 was larger for fear over lateral sites in younger infants and larger for happiness in the older infants. The P1 response was also quicker for fear over right ventral regions in general and quicker for fear in the middle age group infants over dorsal regions. This pattern of results suggests that the response to fear diminished with age and the response to happiness increased; this can be observed in the mean values over lateral sites in Table 7.2. The P1 amplitude was similar for both fear and happiness for the middle age group (7.3-10.6 months). This parallels findings by Leppänen and colleagues (2007), who found no difference in response between fear and happiness in seven-month-olds. It is difficult to compare the results directly due to differences between experimental conditions, electrode sites and exact age group studied however, it indicates that the processing of fear and happiness may be similar at this age in development. The enhanced P1 response to fearful faces in young infants may be due to increased allocation of attention towards aversive stimuli. There is evidence that infants may be particularly responsive to the eye region of the face, with studies showing that infants have a very early preference for

eyes (Maurer, 1985) and sensitivity to gaze direction (Hood et al, 1998). It is thought that eye detection develops more quickly than face detection in infancy and infants may be sensitive to changes in the eye region such as the visible sclera observed in fearful faces. Although most studies investigating eye cues in infants have focussed on the modulation of the N290 to eye cues (Farroni et al, 2002), it is possible that the P1 is also affected due to increased attention. Infants tend to look longer at novel expressions and infants are generally exposed to fearful faces less frequently then happy faces during development. Attention to emotional expression and changes in the eye regions of the face in infants may be controlled by amygdala circuitry. It is thought that connections between the amygdala and cortical structures may be functional early in development, enhancing processing of emotionally salient stimuli and influencing the specialisation of cortical structures to face and emotional stimuli (Balaban et al, 1995; Funayama et al, 2001; Nelson et al, 1979; Pissiota et al, 2003). The transition to an enhanced response to happy compared with fearful faces towards the end of the first year of life suggests that attentional resources may be preferentially recruited for positive affect, possibly alongside a reduction of the feedback loop from the amygdala to cortical regions in response to fear. With increasing age there may be less bias towards the eye region of the face and a more holistic approach to face and emotion processing. The correlation between neural response and attention to facial details could be assessed in future using eye tracking whilst recording ERPs in developmental populations. It could be that the enhanced response to positive affect later in infancy may be related to the increased awareness of social interactions and may provide a basic perceptual basis for social referencing and social behaviour. Motion does not seem to influence the

discrimination between fear and happiness in this age group for the P1, with the only Motion by Emotion interaction occurring over medial regions resulting in a quicker P1 response for static fear compared with dynamic fear in younger and middle age group infants. The results as a whole suggest that a degree of emotional discrimination can occur very early in visual processing in development, even before face-specific responses. However, whether the differential response between fear and happiness is due to emotional expression per se or simply differences in facial features such as the eye region remains to be resolved.

7.4.3 Age effects on the characteristics of the P1

The predictions were that there would be a trend for the P1 to increase in amplitude over this age range; and there may be a change in scalp distribution from medial to lateral sites with age. The first prediction is based on a study by Halit and colleagues (2003) which illustrated that the P1 amplitude increases in amplitude between three and twelve months of age. No differences in P1 latency were observed over this time period however, they were investigating the specificity of the N290 and P400 to faces in this age group and did not look specifically at the P1 component. In addition, the P1 has been shown to become more adult-like between the ages of 4-15 years of age, with a decrease in latency (Batty & Taylor, 2002, 2006; Taylor et al, 2004). There are no other studies looking specifically at the changes in the early-latency components over this age range to compare with the study in this thesis. The prediction that there may be a change in topography of the P1 over this age range is based on face-sensitive responses being more medially distributed in infants and

more laterally distributed in adults (Batty & Taylor, 2003; Bentin et al, 1996; de Haan et al, 2002; Halit et al, 2003; Leppänen et al, 2007). This was a tentative prediction as little is known about changes in topography over the first year. The results show that there was no significant main effect of Age on the P1 response for either amplitude or latency, or interactions of age with topography.

7.4.5 Conclusions, limitations, and future directions

One key finding is that motion effects on emotional face processing change across the first year of life. An enhanced P1 response was observed for dynamic stimuli in older infants (between 10-13 months of age) and an enhanced response for static stimuli in younger infants (between 4-7 months of age). There does not seem to be an effect of motion on processing between 7-10 months of age. The N290 appears unaffected by motion, indicating that the integration of structures involved in face processing may not be fully complete. This study raises the question of why motion processing changes through the first year of life. Possible ways of exploring this might be to track eye movements to assess whether there is a bias in attention towards the eye region for the full duration of the stimulus; and whether this bias may differ with age as expected with a reduced response to fear during the first year. It would be important in future to investigate the effects of motion on the P400, as the possible integration with the N290 to the adult N170 may result in motion influencing the processing that underlies this component. The possibility that motion may modulate attention in infancy could be explored by investigating the effects of motion on the Nc component which is thought to reflect an infant's

allocation of attention. It is not possible to conclude whether the effects observed are exclusive to emotional face-stimuli. Further research would need to be conducted with non-emotional face and non-face stimuli, to assess whether the effects on the early-latency components are specific to social stimuli or whether the effects observed in this study are due to motion in general.

The next chapter will bring together the findings from the previous studies with the aim of addressing the main question of how facial motion influences the neural response during emotion perception in typical and atypical development.

8. General Discussion

8.1 Introduction

The aim of this thesis was to investigate the influence of facial motion on our perception of facial expressions of emotion. This question was tackled using diverse approaches that included behavioural (Chapters 3 & 6); and electrophysiological measures in typical adults (Chapter 4), young adults who had undergone paediatric temporal lobectomy for epilepsy (Chapter 5), and typically developing infants (Chapter 7). The initial focus was to examine the early-latency neural responses in a normally developed adult population. Subsequently, the developmental trajectory of these responses was examined using two converging methods: (a) assessing how disruption to neural systems early in development may affect the facial motion processing in adulthood; and (b) looking specifically at the normative developmental pathway in the first postnatal year of life. This work is original as there are no published studies comparing early-latency ERP components to dynamic and static facial expressions in both adults and infants or in patients who have undergone temporal lobectomy for epilepsy.

The following section will address the main findings and common themes that have emerged throughout the thesis, positioning them in the context of the original predictions. Limitations will then be discussed alongside suggestions for future research.

8.2 Summary of the main findings in this thesis

An overall summary:

Facial expression recognition was modulated by motion, with emotions perceived as less intense being recognised with higher accuracy in dynamic form, and those perceived as more intense being recognised better in static form. Dynamic facial stimuli were given higher overall confidence ratings and perceived as more intense than static stimuli. In typically developed adults, an enhanced P1/N170 response was observed for static compared with dynamic stimuli, modulated by both emotion and topography; with activation of the social brain for the P1 amplitude. Right-sided temporal lobectomy (RTL) patients displayed a reduced P1/N170 response to static emotions in general; and left-sided temporal lobectomy (LTL) patients for static fear. This observed reduction in activity was diminished for dynamic stimuli, and varied by topography. These findings were mirrored in facial recognition tasks, with the RTL group displaying deficits in general facial emotion recognition; and the LTL group displaying deficits in facial fear recognition. These deficits were also extended to higher social emotion processing and social functioning. In typically developing infants, a differential P1 response was observed between static and dynamic stimuli, modulated by age, emotion and topography. Motion did not, however, modulate the N290 response in infants.

More specifically:

1) Motion influences the recognition of specific emotions in adults. The pattern observed in this study is consistent with the view that motion facilitates recognition for less intense (low arousal) emotions. Motion also leads adults

to feel more confident in their recognition and to rate emotions as more intense.

2) Typical adults produced an enhanced P1 and N170 response to static emotional expressions compared with dynamic. For the P1 amplitude, the pattern of response was opposite for non-emotional stimuli, providing evidence of activation of a 'social brain'.

3) The speed of the P1/N170 depended on the brain area over which activity was recorded: generally, the latency was quicker for static stimuli over lateral sites and quicker for dynamic over medial-dorsal sites. In the group with paediatric temporal lobe surgery, the right-sided temporal lobectomy (RTL) group produced a diminished response for static stimuli, and the left-sided temporal lobectomy (LTL) group produced a diminished response for static fear specifically. These deficits were reduced for dynamic stimuli indicating that the neural processing of facial emotion may be more intact for dynamic compared with static stimuli. This may reflect disproportionate disruption to amygdalo-cortical pathways involved with perception of static facial expressions. The reduction in response to fear in the LTL group may reflect a bias in emotion processing in the left subcortical pathway during development.

4) The RTL group demonstrated deficits in facial emotion recognition generally and the LTL group in facial fear recognition. The RTL group showed deficits in complex social judgments and both groups showed a reduced social functioning, particularly for social interactions. This reflects the importance of

temporal lobe structures in emotion perception. Indications suggest that the deficits observed are influenced by a reduced non-verbal IQ.

5) In a typically developing population, young infants produced an enhanced and slower P1 response to dynamic stimuli, with the opposite true for older infants. This indicates that in early infancy the early perceptual processes are influenced by motion, with a more adult-like pattern of a larger P1 to static images emerging by twelve months. When taking into account the effect of motion at P1, there was no further effects on the N290, indicating that the neural system underlying the N290 may not be fully mature, and under-developed in terms of motion processing, and possibly also in terms of specialisation to face stimuli.

8.3 Models of visual processing in adults

There was evidence of a differential response in the early-latency processing of static and dynamic emotional stimuli in the typically developed adults in Chapter 4. In general, the static stimuli produced an enhanced response for both the P1 and N170 components. However, this effect was modulated by both topography and the type of emotional expression presented. Two interpretations of these findings are that the early-latency processing of static and dynamic visual stimuli is reliant on differential activity of the same neural system or, alternatively, activity of dissociable neural systems. One of the current dominant cognitive models for understanding the neural bases of face processing was proposed by Haxby, Hoffman and Gobbini (2000), which emphasises the distinction between processing the invariant and

changeable aspects of facial stimuli. They proposed that after the initial rapid perceptual processing of visual stimuli in the inferior occipital gyrus, the representation of the invariant aspects is mediated by the face-responsive fusiform gyrus, whilst the representation of the changeable aspects of facial stimuli is mediated by the face-responsive superior temporal sulcus. Aspects such as attention and facial expressions are also processed by an extended network involving other cortical and subcortical regions with reciprocal connections to cortical visual regions. Essentially cognitively distinct aspects of face perception are mediated by disparate neural representations. This implies that the differential response observed to static and dynamic stimuli could be due to activity in two dissociable systems. There is evidence from imaging studies which substantiates that different neural structures are active in response to static and dynamic stimuli, with the recruitment of a wider neural network for dynamic stimuli (Kilts et al, 2003; LaBar et al, 2003; Sato et al, 2004; Trautmann et al, 2009). Studies in clinical populations have shown that deficits in facial identity and emotion recognition for static stimuli can be absent for dynamic stimuli and vice versa (Adolphs et al, 2003; Humphreys et al, 1993). This also fits in with the idea of two parallel hierarchical pathways in vision, the dorsal (occipito-parietal) pathway concerned with spatial properties of vision and visually guided actions, and the ventral (occipito-temporal) pathway critical for visual object identification (Ungerleider & Mishkin, 1982). This theory was based on observed patterns of behaviours in monkeys following lesions to specific areas of the cortex suggesting that the visual cortex can be decomposed into two pathways. This was later revised in terms of vision for perception (ventral stream) and vision for action (dorsal stream) (Goodale & Milner, 1992). The finding in the current study, that the

speed of the P1/N170 was quicker for static stimuli over lateral sites and quicker for dynamic over medial-dorsal sites could reflect the differential activation of these two pathways by the static and dynamic images. In addition, the finding that infants' responses to static and dynamic images show a different developmental trajectory could also be seen as evidence for operation of two pathways. The findings in temporal lobectomy patients could also support the dissociable pathway view, as the processing of dynamic stimuli was relatively spared compared with processing of static stimuli. This will be discussed later in the context of the influence the amygdala has on emotional face processing.

The alternative view is that the same neural circuitry is responsible for the differential response observed, with the enhanced response to static stimuli indicating augmented processing within this neural system. A revision to the model of the visual pathways suggests that the dorsal and ventral visual pathways have a degree of functional integration in normal object recognition to enhance cue-variant and view-point variant recognition by use of three-dimensional information (Farivar, 2009). Studies indicate that the dorsal visual regions may participate in complex object recognition including the recognition of unfamiliar faces (Farivar et al., 2009). Studies have also shown that dynamic stimuli produce an enhanced response to dynamic emotional stimuli in neural structures associated with processing static emotional stimuli (Kilts et al, 2003; LaBar et al, 2003; Sato et al, 2004; Trautmann et al, 2009). The overall larger response to static compared to dynamic stimuli for both P1 and N170 could be seen as consistent with this view that a single system is activated, but to a different extent.

To summarise, there was evidence of a differential response between static and dynamic stimuli at early-latencies (by approximately 100ms) in adults, though the data cannot conclusively show whether this reflects activation of separable pathways for static and dynamic emotion processing or a common pathway activated to different degrees. Future studies using adaptation paradigms, where the participant would be adapted to one form of input and then the response to the other is evaluated, may help to better distinguish between these possibilities. Further research will be needed to explore this in more detail, and this is discussed in section 8.8 below. Regardless of whether the same system or dissociable systems are responsible, the results show that motion is already influencing facial emotion processing at this early perceptual stage.

8.4 Models for the development of face processing

There was a differential processing of static and dynamic stimuli across the infant population, with an enhanced, slower P1 in younger infants for dynamic stimuli compared with static stimuli; and the opposite effect in older infants. This suggests that the influence of motion on face processing changes throughout development. There are currently several theories suggested for the development of face processing, one theory called interactive specialisation proposes that the cortical system underlying face processing is initially not specific to faces however, developmental mechanisms generate increasingly specialised processing within the cortical regions (Johnson, 2000; Morton & Johnson, 1991). In this model, two distinct systems are proposed to underlie development: 'Conspec' is a subcortical system

operating from birth that orientates the newborn's visual attention towards faces, and 'Conlern', a cortical system sensitive to the effects of experience, emerging around two months of age leading to a more mature face-processing ability. One hypothesis suggests that subcortical brain regions not only detect the presence of faces, orienting the infant to the face, but also directly influence the activity in cortical areas such as the lateral occipital, fusiform and orbitofrontal cortices. Thus, subcortical regions could partly determine which cortical regions become incorporated into the social brain network during development. In addition, these cortical regions such as the fusiform gyrus, would also receive foveal cortical visual input, producing converging information from different sources, ensuring certain developing cortical circuits became specialised for face stimuli. This view is supported by studies showing that there is a decrease in discrimination abilities for non-human faces with age (Halit et al, 2003). This increased specialisation to human faces during development may underlie the increasing response to static stimuli towards one year of life, as observed in Chapter 7, becoming similar to the findings in typically developed adults in Chapter 4. The influence of the amygdala on the development of cortical structures, such as the fusiform gyrus and STS, is highlighted by the findings that disruption to the amygdala produces reduced activity to emotional stimuli (Chapter 5). This is discussed further in the next section.

8.5 The influence of subcortical activity on cortical processing

The mechanisms by which the amygdala enhances processing of emotional stimuli through mediation of cortical regions is becoming established (Vuilleumier et al.,

2004). The current theory proposes that the amygdala responds to coarse, low-spatial-frequency information regarding emotional stimuli, e.g. global shape and configuration of facial expressions in the initial stages of information processing (Luo et al, 2007) which subsequently enhances more detailed perceptual processing in cortical face-sensitive areas such as the fusiform gyrus and the STS (Vuilleumier et al, 2003, 2004). The amygdala might enhance cortical activity through direct feedback projections to visual-representation regions (Freese & Amaral, 2005) or through connections to basal forebrain cholinergic neurons that transiently increase cortical excitability (Bentley et al, 2003; Kapp et al, 1994). Findings point to a broad role in processing biologically-relevant stimuli (Adolphs, 2008), and in evaluating and acquiring information about associations between stimuli and emotional significance (Hooker et al, 2006; Paton et al, 2008). This may explain why individuals with damage to the temporal lobe often have deficits in recognition of fearful faces and general aversive stimuli (Anderson et al, 2000; Meletti et al, 2003). Previous studies have found that there is a right-sided bias to the deficits, with right-sided temporal lobe epilepsy/lobectomy cases producing more severe deficits than left-sided cases, particularly for fear. It has thought that this is a result of amygdala damage which is influential in processing aversive stimuli. The right-sided temporal lobectomy patients in this thesis (Chapter 5) did show a reduction in the response to static emotional stimuli, but contrary to the prediction, the left-sided group produced more of a deficit in the processing of static fear. This suggests that the left amygdala and left subcortical pathway may be more influential in emotion processing during development, with the right subcortical-cortical pathway being more active during adulthood. Previous studies have revealed a pattern of left-lateralised amygdala and

prefrontal activation in adolescents when viewing overtly presented affective stimuli (Killgore & Yurgelun-Todd, 2004). It is possible that there may be a period of cerebral organisation (Hasan et al, 2007; Shaw et al, 2008), neuronal structure (Rabinowicz et al, 2009), and volume changes in the amygdala (Yurgelun-Todd, Killgore, & Cintron, 2003). Amygdala activations may become more right lateralised with maturation towards adulthood. Shifts in the lateralisation of cortical and limbic processing may be responsible for the disparity between the right-sided bias for emotional stimuli, particularly fear, in adults, and the findings in this thesis that the left-sided cases have a reduced response and are impaired at fear recognition. The deficits in neural response observed in the two temporal lobectomy groups for static stimuli (Chapter 5) were reduced for dynamic stimuli. This reflects a disproportionate disruption to amygdalo-cortical pathways involved with the perception of static facial expressions. One possible interpretation is that there are connections between subcortical and cortical regions for the processing of motion in addition to those for processing static information which is possibly evidence for the dissociable pathway theory discussed above. These connections may serve to provide additional information regarding the emotional stimulus to enable perception to take place effectively despite disruption to pathways processing the static information.

8.6 Attentional resources

The differential response between static and dynamic stimuli observed in the adult population (Chapter 4) was modulated by emotional expression, being more obvious for specific emotions. In addition, the bias in the P1 and N170 response towards

either static or dynamic was different for specific emotions. Fear tended to produce a differential N170 response compared with other emotions solely for static images whereas the P1 response to fear was differential only for dynamic stimuli. This has a bearing on previous research that has failed to identify emotional modulation on the P1 response. This is an interesting finding in the context of the role dynamic stimuli may play on attention allocation, as indexed by the P1. There is thought to be a 'fast-route' processing of aversive stimuli, particularly for fear, to enable an immediate response to threatening environmental stimuli, as in 'fight-or-flight' (LeDoux, 1996). This is thought to be mediated by the amygdala which in turn has modulatory influences on cortical regions which may underlie the early-latency components discussed here (Vuilleumier et al., 2004). It is possible that the moving image of fear is more emotionally salient than the static form and enhances amygdala activity, producing a very rapid top-down effect on the visual encoding, reflected in the P1 response. In contrast, the N170 response was enhanced for fear only in the static form. The allocation of attention to aversive stimuli is a key role of the amygdala (Vuilleumier & Pourtois, 2007), and these findings suggest that the moving images of fear work to recruit attentional resources to the stimulus, even before the facial features are processed fully. One question that is raised is whether it is the emotional expression per se, that influences brain responses at this early latency, or whether it is changes in specific facial features from one position to another. One aspect of the face that has the most pronounced changes for fearful expressions is the eye region of the face and this is particularly true in infant populations (Farroni et al., 2002; Johnson & Farroni, 2003; Maurer, 1985). Allocation of attention may be focussed on the eye region during initial assessment of the aversive stimulus,

controlled by the amygdala's influence on cortical regions. This emphasises the amygdala's influence in processing visual stimuli very early on. However, this cannot be the full picture as the P1, and longer-latency components the EPN and LPP, have all shown enhanced responses to emotionally salient faces (Eimer, Holmes et al., 2003; Krolak-Salmon et al., 2001; Sato et al., 2001). This suggests that it is the neural structures underlying the ERP components which may be responding to the emotional context rather than the face per se. This is partially substantiated by the findings in this thesis that the motion effects on the P1 amplitude are specific to emotional stimuli; with the P1 response enhanced to static emotional stimuli and the response to non-face stimuli being the opposite. This has implications for an enhanced response to static stimuli compared with dynamic in adults, which was contrary to the prediction. The dynamic emotional expression would not have evolved to the maximal point by 100-200ms post-stimulus onset. It could be assumed that the salience of the emotional expressions would be greater for the static images at this time-point, resulting in greater activity in structures such as the amygdala which would feed-forward to cortical structures responsible for generating the early-latency components.

8.7 Emotion Recognition

It is important to note the findings from the emotion recognition study in adults (Chapter 3), alongside these ERP results. In general, static stimuli were recognised with higher accuracy than dynamic stimuli but there was variation between emotions. Interestingly, those emotions perceived as less intense were recognised

better in dynamic form than static. It was concluded that there was a dynamic advantage for emotion recognition, only when the intensity of the stimuli were perceived as low. This replicates other studies showing that dynamic stimuli only consistently improve emotion recognition when the intensity levels of the stimuli are low (Bould & Morris, 2008). This suggests that intense stimuli present the viewer with enough information to deduce the emotional state and, in this case, movement does not add benefit. One question to answer is: what is it about the intensity of emotional stimuli that influences recognition and, specifically, how does motion aid this process? One possibility is that the activity of neural structures is influenced by the spatial distribution of key facial features, such as the eye region, the mouth, and eyebrows. Intense expressions provide a larger displacement from 'the norm', and this exaggerated expression might provide key information to process and recognise the emotion expressed. More subtle facial cues might not be enough to distinguish one emotional expression from another, especially when the facial configuration is similar for different emotional states. This is illustrated nicely by the results from Chapter 3, which show that sadness, when incorrectly identified, is wrongly labelled as the whole spectrum of other emotions; and is also, crucially, perceived as less intense than other emotions. Although the accuracy rating was high for static sadness, there was still a bias for dynamic faces to be recognised better. The movement from neutral to the apex of the emotional expression in the dynamic stimuli may accentuate the displacement of facial features displayed for sadness; and the direction of the displacement would be apparent which would be more unique to a given emotion. The neural basis for this is unclear, however, it is possible that the activity of groups of neurons in structures, such as the amygdala, is

influenced by the displacement of facial features. The combination of activity within these neuronal clusters produces the perception of an emotional state, and if the displacement is not unique to one particular emotional state, this may result in a lack of recognition. Motion may increase activity in the neuronal group or activate other neuronal clusters for recognition to be achieved. In light of these conclusions, there are two limitations of the study which need to be addressed: firstly the recognition of the static stimuli was high across the seven emotional categories (two categories of fear). This limited the influence movement might have on improving recognition rates. Secondly, the intensity of the stimuli was considered to be generally high for both static and dynamic stimuli (as discussed in more detail in Chapter 3), with the dynamic advantage only visible for those stimuli considered less intense. One possible way to approach this might be to create blended morphs: one emotional expression transitioning into another, such as happy to sad, with the aim to assess subtler changes in emotional state as opposed to presenting emotional faces at the apex of maximal expression. To clarify the advantage facial movement has on emotion recognition, it will be important in future research to take account of these factors which lead to differences in recognition of static versus dynamic faces, as discussed below.

8.8 Limitations and future directions

The work in this thesis has been important in identifying that facial motion influences the processing of emotional expressions in both developing and

developed populations. It has extended our knowledge of the deficits in emotion perception observed in individuals with temporal lobe lesions and explored the changes in response to facial motion over the first year of development. The studies, however, are not without their limitations and these need to be addressed in future research.

One of the unresolved questions raised by findings in this thesis is whether the differential early-latency responses observed between static and dynamic stimuli are due to augmented neural activity or dissociable networks. To address this question it would be useful to compare findings from fMRI and ERP experiments. The advantage of this technique would be that precise temporal *and* spatial parameters for the neural activity would be obtained which is not possible with one technique alone. It would be important to control for individual differences therefore, ideally, individuals would participate in both the ERP and fMRI experiments. However, the problem with this is that exposure to the stimuli in one setting would confound the findings from the other, i.e. familiarity and recognition may increase and as a consequence neural activity may be altered. A way round this might be to have two separate groups of individuals, each participating in only one experiment. However, it would be difficult to draw conclusions based on two separate groups and it would be necessary to have a very large sample size to reduce individual differences.

An additional advantage of using fMRI alongside ERP techniques would be to explore the hypothesis that subcortical structures such as the amygdala are influencing the P1/N170 responses. It is difficult to detect activity in deeper neural structures using EEG and so tentative predictions need to be made about the role of such structures in producing the activity observed at the scalp. It would be possible to detect

amygdalar activation using MRI techniques and this, coupled with source localisation of the P1 and N170, would shed light on the role and influence of these subcortical structures on the neural activity between 100-200ms post-stimulus onset.

Another key issue which is unresolved is: whether it is motion per se or the intensity of the static/dynamic stimuli that produces the differential response observed. It is not possible to be certain that it is the movement of the stimuli that produces the response rather than intensity. This is a limitation of the paradigm used and, to take this question further, one would need to introduce various paradigms that reliably test each hypothesis. For example, static and dynamic stimuli with varying intensities could be compared; a range of examples from those perceived as low intensity to high intensity. The high intensity stimuli used in this thesis taken from the NimStim Stimulus Set could be contrasted with less intense stimuli from the same battery to control for model, luminance, etc. An ERP paradigm similar to that described in Chapter 4 could be used, with a comparison of high- and low-intensity static images, followed by a comparison of high- and low-intensity dynamic images. If the responses vary for differing intensities this might suggest that intensity is a factor in the observed differential response in this thesis. It would be important to control for other aspects of the stimuli such as mouth movements, i.e. opening of the mouth in extreme fear or surprise.

Another limitation is that an assumption cannot be made that the motion effects observed are exclusive to faces, facial expressions, or even social stimuli in general. The only evidence pointing towards specificity for social stimuli was the enhanced P1 amplitude response observed for static stimuli in the typically developed adult population in Chapter 4, which resulted in the opposite effect for the flower stimuli.

One approach to answer this question would be to compare neural responses to different categories of social stimuli; such as dynamic face and body parts, and dynamic moving bodies. In addition, to elucidate the elements of the face influenced by motion, a comparison could be made between dynamic facial expressions and dynamic images of faces with a neutral expression, e.g. morphing from one person to another to elucidate the influence motion has on facial emotion; and also, upright dynamic faces versus inverted dynamic faces. The rationale for comparing the emotional faces to a flower was for the inclusion of a comparable evolving dynamic stimulus; the opening of the flower was similar in low-level visual properties to the facial expressions evolving. A neutral face was not used as a control comparison as there is debate that neutral is really perceived as expressionless (Iidaka et al., 2005; Somerville et al., 2004), especially for children (Lobaugh et al., 2006; Thomas et al., 2001) and could therefore be perceived as another emotional condition. In addition, morphing a neutral face with one person to another may have the added confound of a changing identity. An alternative approach could be to have a facial gesture such as a yawn which would not be perceived as emotive.

There were differences in the experimental tasks between the adult and infant populations making it difficult to directly compare the two sets of findings. Adult participants were presented with six basic emotions (anger, disgust, fear, happiness, sadness, and surprise) and infants only fear and happiness. The infants were only presented with a limited number of emotional categories for two reasons: firstly to compare the response observed in this thesis with previous research that has compared positive valence with negative valence. In addition, there are constraints on the length of time infants will attend to a task, and limiting the number of

variables resulted in a larger number of trials than would have been possible if including additional emotional categories. Difficulties in distinguishing between emotions such as fear and surprise may have been reflected in differential responses between the emotions. This may be a confound as differential ERP responses observed could in fact be due to differences in ease of processing as opposed to emotion per se. In fact, even when there was a clear main effect of emotion in the P1/N170 response in Chapter 5, emotions fear and surprise resulted in similar amplitudes/latencies. The inclusion of all of the six basic emotions in the adult studies was to extend knowledge of the neural responses to some of these emotions less well explored. A majority of studies use selective emotions, such as anger, fear and happiness to investigate valence differences. However, there is little research investigating dynamic facial expressions and, for this reason, it was important to explore whether there might be a differential response to motion between the different emotions.

The comparison between experimental tasks is also limited based on the contrast in neural responses evoked by passive and active tasks (Costafreda et al, 2008; Lange et al, 2003). The ERP experiments were passive tasks, the only requirement for adult participants was a button-press in response to the target flower stimulus. This was employed to confirm that each participant was attending to the stimuli throughout the experiment due to the passive nature of the task. This could not obviously be employed in the infant study and so attention was measured by recording eye movement fixated on the stimulus. In addition, the behavioural tasks performed in the adult population to assess emotion recognition were active tasks, so direct comparison of the neural activity observed in the ERP experiments with behavioural

measures is limited. Therefore, attentional resources are not similarly allocated across active and passive conditions, which may be a confounding factor. It is possible that explicit emotion discrimination tasks do not evoke the recruitment of an identical set of neural structures to those evoked by the spontaneous appearance of emotional face stimuli in the visual field in the absence of specific task demands. The research in this thesis may only be generalised to explicit emotional face processing, as different spatio-temporal changes may be involved in implicit emotional face processing. It would be necessary in future research to assess the perception of static and dynamic implicit versus explicit emotional face processing. One approach might be to compare neural responses when engaged in an active task versus a passive task. For example, two faces posing different emotional expressions could be presented to the right and left of a central point, with participants instructed to press a button when the identity of the face was the same. This could then be compared with a task where participants are instructed to passively view the pictures.

Another limitation of this study was the use of morphed dynamic images, which produced a linear transition between neutral and maximal expression, with every component of the face changing at the same rate. Natural facial expressions in the social environment are smooth, reflex-like and ballistic (Ekman, 1977; Hess, 1989). The purpose of using morphed images instead of natural videos of emotional expression was to control the timing, speed and duration of the unfolding expression, so each expression for each actor would be revealed in the same way. This is particularly important when investigating early-latency ERP responses where a degree of control must be exercised over the timing of the stimuli presented. This

is critical to making conclusions based on responses observed in a particular time-window. It is possible that morphed faces may not be perceived as saliently as natural expressions encountered in the social environment. This may influence the neural response recorded at the scalp due to reciprocal connections from the amygdala to regions such as the fusiform gyrus; the timings of facial motion may be critical to elicit a maximal response. Although imaging studies have not directly compared the neural response to natural versus computer-generated images, both types of stimuli have been used to elicit responses to dynamic facial expressions. In future research, it will be important to compare these two types of stimuli to ascertain whether there are differences in neural response. It is possible that the more naturalistic images produce a greater response or a differential pattern of response. In addition, it would be interesting to investigate whether the response to morphed versus natural videos changes the early-latency responses, driven by subcortical emotion-sensitive regions such as the amygdala (Breiter et al, 1996).

The dynamic facial expressions are evolving over the whole time course of the stimulus exposure. This presents a confound when comparing the neural responses at early-latency time-windows for the static emotional expressions with dynamic. Since the emotional expression is not fully evolved at this time-window, this raises the question of how to directly compare the emotional content of static with dynamic. One approach to determine the time point that the emotional expression first appears would be to instruct individuals to press a button as soon as they recognise the developing emotion. The stimulus could then be presented from this time point however, this is limited, as the intensity and salience of the two conditions would differ with the apex of the emotion not occurring until the end of

the time course. In addition, recognition of the emotional expression may occur at a longer latency than perception, which may not be reflected in earlier brain activity, such as the P1 or N170. As indicated in earlier chapters, previous research has demonstrated that the longer-latency components, the EPN and LPP, show an enhanced response to emotionally salient faces (Eimer, Holmes et al., 2003; Krolak-Salmon et al., 2001; Sato et al., 2001). As discussed in Chapter 6, the reduced ERP responses did not correlate with the deficits in emotion recognition in the clinical population. It would therefore be interesting to explore whether these behavioural deficits would correlate with longer-latency neural activity, possibly reflected in the EPN or LPP. Crucially, further analysis of these time-windows would extend our knowledge of the pattern of neural activity for dynamic emotional stimuli. Only two studies have investigated ERP/evoked potential responses to dynamic stimuli (Mayes et al., 2009; Recio et al., 2011) and both studies have limitations, i.e. not directly comparing static and dynamic images and using multiple static images to create the moving images.

Varying the presentation speed of the dynamic stimuli may influence the early-latency responses observed. It may be that particular velocities optimise the early-latency responses. Kamachi and colleagues (2001) found that the speed of dynamic presentation influenced emotional recognition, with happiness and surprise being recognised more accurately from faster sequences, anger from medium-speed sequences, and sadness from slower sequences. An additional dimension is that the emotional expression would be revealed at different rates. It would then be possible to examine whether this had an impact on the degree of response at these early

latencies. Would a quicker presentation increase the response to the dynamic stimuli? Would this vary depending on the emotional expression?

One further limitation of the morphed dynamic stimuli is that the neutral face chosen for the start of the stimulus may determine when the emotional expression is first perceived. There is debate as to whether neutral faces can really be considered emotion-less (Iidaka et al., 2005; Somerville et al., 2004), and the choice of neutral face as a starting point for the emotional expression may influence the time point at which the emotional expression is perceived. For example, if the neutral face was perceived as more positive than negative in nature, the point at which a negative emotion, e.g. disgust, could be perceived first might be different from the time point at which happiness will be perceived.

In summary, future research must look towards a more precise measurement of the characteristics of facial motion, such as intensity, duration, speed, onset and offset of expression, with the aim to produce more naturalistic stimuli, with temporal aspects that can be accurately quantified.

One limitation with drawing conclusions on the influence of the amygdala on the responses observed in cortical regions is that the degree of amygdala damage was not compared across subjects. A direction for further research in this population would be to compare the location of the resection in more detail; to investigate correlations of amygdala volume with deficits in neural response and also emotion recognition. It is also not possible to comment on the influence of early versus late onset of seizures on the observed findings as the patient population was predominantly early age of onset (before 6 years of age). A larger sample size and a

more balanced split between early and late onset would allow an assessment of this factor on ERP responses. This is a long-term follow-up of the patient group, as the individuals were in their late teens to early thirties at the time of the study. It is possible that an absence of deficits at the time of assessment may be due in part to a degree of compensation for deficits they may have experienced in development.

One interesting extension to the ERP studies in typically and atypically developed adults (see Chapters 4 & 5) would be to implement an emotion recognition task of the presented static and dynamic stimuli to individuals after the ERP task, as described in Chapter 3. The findings could then be used to explore whether the differential ERP responses observed were correlated with the recognition of the emotional content of these stimuli. In addition, the recognition accuracy of the dynamic stimuli could be compared to the assessment of the static stimuli from the Florida Affect Battery administered to the clinical population in Chapter 6. One would be in a better position to interpret the deficits observed in this population and to observe whether they extend to recognition of moving emotional faces or limited to static stimuli. The pattern of ERP results tentatively indicates that the deficits may be reduced for dynamic stimuli and it would be interesting to pursue this further. As discussed above, the role of motion would need to be addressed, to elucidate whether it is moving emotional faces, moving social or biological stimuli, or movement per se that triggers a differential response and what this really means in terms of brain activity and ultimately perception.

In terms of the developmental trajectory, it would be interesting to extend the age range of the infants beyond 13 months through adolescence towards adulthood, to ascertain the changes in the P1/N170 response during that time period. At what age

would the response resemble the adult response pattern observed in this thesis? How would the maturation of the neural networks be reflected in the topographical distribution in this time period? The paradigm described in this thesis could be implemented in a wide range of age groups to explore this question further.

In conclusion, a neural model has yet to be determined to account for the differential early-latency responses observed between static and dynamic stimuli. It is difficult to conclude whether the augmented neural network theory or the dissociable neural network theory best represents the findings described. The limitations of the studies and questions raised by the findings in this thesis have been discussed with a view of how to address these pertinent questions in future research. It is apparent that aspects of the moving emotional stimuli, whether it is the movement per se, intensity of the stimuli, or attentional resources, influence the early-latency responses to the stimuli. The current findings in typically and atypically developed adults and developing infants have important implications for our understanding of emotion processing throughout development and the role motion plays in emotion perception.

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9. Appendix A Chapter 4: ANOVA Tables for the P1 and N170

Chapter 4 P1 Amplitude ANOVA Table

Effect or interaction	F	df	p
Motion	42.08	1, 19	0.00
Emotion	0.07	5, 95	0.99
Hemisphere	1.90	1, 19	0.18
Lateral-Medial	15.97	1, 19	0.00
Dorsal-Ventral	67.05	1, 19	0.00
Motion*Emotion	2.47	5, 95	0.05
Motion*Hemisphere	3.78	1, 19	0.07
Emotion*Hemisphere	3.11	5, 95	0.02
Motion*Emotion*Hemisphere	1.15	5, 95	0.34
Motion*Lateral-Medial	0.19	1, 19	0.67
Emotion*Lateral-Medial	0.59	5, 95	0.65
Motion*Emotion*Lateral-Medial	1.88	5, 95	0.13
Hemisphere*Lateral-Medial	3.48	1, 19	0.08
Motion*Hemisphere*Lateral-Medial	3.53	1, 19	0.08
Emotion*Hemisphere*Lateral-Medial	1.79	5, 95	0.14
Motion*Emotion*Hemisphere*Lateral-Medial	1.32	5, 95	0.27
Motion*Dorsal-Ventral	15.49	1, 19	0.00
Emotion*Dorsal-Ventral	0.94	5, 95	0.44
Motion*Emotion*Dorsal-Ventral	0.79	5, 95	0.52
Hemisphere*Dorsal-Ventral	0.01	1, 19	0.92
Motion*Hemisphere*Dorsal-Ventral	0.69	1, 19	o.42
Emotion*Hemisphere*Dorsal-Ventral	0.74	5, 95	0.55
Motion*Emotion*Hemisphere*Dorsal-Ventral	0.43	5, 95	0.77
Lateral-Medial	0.90	1, 19	0.35
Motion*Lateral-Medial*Dorsal-Ventral	0.40	1, 19	0.53
Emotion*Lateral-Medial*Dorsal-Ventral	1.79	5, 95	0.14
Motion*Emotion*Lateral-Medial*Dorsal-Ventral	0.63	5, 95	0.63
Hemisphere*Lateral-Medial*Dorsal-Ventral	0.82	1, 19	0.38
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.12	1, 19	0.73
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	1.01	5, 95	0.40
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	2.14	5, 95	0.08

Chapter 4 P1 Latency ANOVA Table

Main Effects & Interactions	F	df	p
Motion	1.61	1, 19	0.22
Emotion	1.15	5, 95	0.34
Hemisphere	0.02	1, 19	0.88
Lateral-Medial	1.92	1, 19	0.18
Dorsal-Ventral	10.92	1, 19	0.00
Motion*Emotion	2.24	5, 95	0.07
Motion*Hemisphere	0.14	1, 19	0.71
Emotion*Hemisphere	1.10	5, 95	0.36
Motion*Emotion*Hemisphere	0.64	5, 95	0.62
Motion*Lateral-Medial	5.28	1, 19	0.03
Emotion*Lateral-Medial	1.09	5, 95	0.36
Motion*Emotion*Lateral-Medial	1.57	5, 95	0.19
Hemisphere*Lateral-Medial	4.70	1, 19	0.04
Motion*Hemisphere*Lateral-Medial	0.02	1, 19	0.91
Emotion*Hemisphere*Lateral-Medial	0.47	5, 95	0.73
Motion*Emotion*Hemisphere*Lateral-Medial	0.86	5, 95	0.49
Motion*Dorsal-Ventral	2.28	1, 19	0.15
Emotion*Dorsal-Ventral	1.61	5, 95	0.19
Motion*Emotion*Dorsal-Ventral	1.28	5, 95	0.29
Hemisphere*Dorsal-Ventral	1.16	1, 19	0.30
Motion*Hemisphere*Dorsal-Ventral	0.82	1, 19	0.38
Emotion*Hemisphere*Dorsal-Ventral	1.03	5, 95	0.39
Motion*Emotion*Hemisphere*Dorsal-Ventral	1.39	5, 95	0.25
Lateral-Medial	12.44	1, 19	0.00
Motion*Lateral-Medial*Dorsal-Ventral	5.11	1, 19	0.04
Emotion*Lateral-Medial*Dorsal-Ventral	0.74	5, 95	0.55
Motion*Emotion*Lateral-Medial*Dorsal-Ventral	1.64	5, 95	0.17
Hemisphere*Lateral-Medial*Dorsal-Ventral	3.06	1, 19	0.10
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral	1.43	1, 19	0.25
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	1.11	5, 95	0.36
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	1.79	5, 95	0.16

Chapter 4 N170 Amplitude ANOVA Table

Main Effects & Interactions	F	df	p
Motion	34.92	1, 19	0.00
Emotion	2.12	5, 95	0.09
Hemisphere	2.50	1, 19	0.13
Lateral-Medial	6.47	1, 19	0.02
Dorsal-Ventral	50.23	1, 19	0.00
Motion*Emotion	1.77	5, 95	0.15
Motion*Hemisphere	3.11	1, 19	0.09
Emotion*Hemisphere	0.84	5, 95	0.49
Motion*Lateral-Medial	5.98	1, 19	0.02
Emotion*Lateral-Medial	1.36	5, 95	0.26
Hemisphere*Lateral-Medial	3.93	1, 19	0.06
Motion*Dorsal-Ventral	25.95	1, 19	0.00
Emotion*Dorsal-Ventral	5.12	5, 95	0.00
Hemisphere*Dorsal-Ventral	2.35	1, 19	0.14
Lateral-Medial*Dorsal-Ventral	23.19	1, 19	0.00
Motion*Emotion*Hemisphere	0.76	5, 95	0.53
Motion*Emotion*Lateral-Medial	3.20	5, 95	0.02
Emotion*Hemisphere*Lateral-Medial	1.54	5, 95	0.21
Motion*Emotion*Dorsal-Ventral	3.23	5, 95	0.02
Motion*Hemisphere*Dorsal-Ventral	0.34	1, 19	0.57
Emotion*Hemisphere*Dorsal-Ventral	0.64	5, 95	0.65
Motion*Lateral-Medial*Dorsal-Ventral	24.10	1, 19	0.00
Emotion*Lateral-Medial*Dorsal-Ventral	0.76	5, 95	0.54
Hemisphere*Lateral-Medial*Dorsal-Ventral	0.05	1, 19	0.82
Motion*Emotion*Lateral-Medial*Dorsal-Ventral	0.58	5, 95	0.67
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.12	1, 19	0.74
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.42	5, 95	0.80
Motion*Emotion*Hemisphere*Lateral-Medial	2.17	5, 95	0.10
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.39	5, 95	0.80

Chapter 4 N170 Latency ANOVA Table

Main Effects & Interactions	F	df	p
Motion	1.13	1, 19	0.30
Emotion	1.20	5, 95	0.32
Hemisphere	1.46	1, 19	0.25
Lateral-Medial	12.32	1, 19	0.00
Dorsal-Ventral	0.67	1, 19	0.43
Motion*Emotion	1.88	5, 95	0.13
Motion*Hemisphere	1.89	1, 19	0.19
Emotion*Hemisphere	0.66	5, 95	0.58
Motion*Emotion*Hemisphere	1.15	5, 95	0.34
Motion*Lateral-Medial	1.67	1, 19	0.21
Emotion*Lateral-Medial	3.59	5, 95	0.02
Motion*Emotion*Lateral-Medial	1.32	5, 95	0.28
Hemisphere*Lateral-Medial	1.46	1, 19	0.24
Motion*Hemisphere*Lateral-Medial	0.75	1, 19	0.40
Emotion*Hemisphere*Lateral-Medial	2.12	5, 95	0.11
Motion*Emotion*Hemisphere*Lateral-Medial	0.45	5, 95	0.74
Motion*Dorsal-Ventral	0.02	1, 19	0.90
Emotion*Dorsal-Ventral	0.85	5, 95	0.51
Motion*Emotion*Dorsal-Ventral	2.39	5, 95	0.60
Hemisphere*Dorsal-Ventral	0.20	1, 19	0.66
Motion*Hemisphere*Dorsal-Ventral	0.42	1, 19	0.52
Emotion*Hemisphere*Dorsal-Ventral	0.54	5, 95	0.66
Motion*Emotion*Hemisphere*Dorsal-Ventral	1.02	5, 95	0.40
Lateral-Medial	6.42	1, 19	0.20
Motion*Lateral-Medial*Dorsal-Ventral	1.19	1, 19	0.28
Emotion*Lateral-Medial*Dorsal-Ventral	1.64	5, 95	0.19
Motion*Emotion*Lateral-Medial*Dorsal-Ventral	0.61	5, 95	0.64
Hemisphere*Lateral-Medial*Dorsal-Ventral	4.10	1, 19	0.06
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.83	1, 19	0.37
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.82	5, 95	0.52
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.54	5, 95	0.71

10. Appendix B Chapter 5: ANOVA Tables for the P1 and N170

Chapter 5 P1 Amplitude ANOVA Table

Main Effects & Interactions	F	df	p
Motion	17.13	1, 27	0.00
Motion*Group	3.07	2, 27	0.06
Emotion	0.16	5, 135	0.97
Emotion*Group	0.32	10, 135	0.96
Hemisphere	1.87	1, 27	0.18
Hemisphere*Group	0.65	2, 27	0.53
Lateral-Medial	16.50	1, 27	0.00
Lateral-Medial*Group	0.13	2, 27	0.88
Dorsal-Ventral	65.11	1, 27	0.00
Dorsal-Ventral*Group	0.73	2, 27	0.49
Motion*Emotion	0.32	5, 135	0.90
Motion*Emotion*Group	2.37	10, 135	0.02
Motion*Hemisphere	2.55	1, 27	0.12
Motion*Hemisphere*Group	0.55	2, 27	0.58
Emotion*Hemisphere	1.07	5, 135	0.37
Emotion*Hemisphere*Group	1.47	10, 135	0.18
Motion*Emotion*Hemisphere	0.77	5, 135	0.55
Motion*Emotion*Hemisphere*Group	0.50	10, 135	0.85
Motion*Lateral-Medial	0.43	1, 27	0.52
Motion*Lateral-Medial*Group	1.66	2, 27	0.21
Emotion*Lateral-Medial	0.86	5, 135	0.49
Emotion*Lateral-Medial*Group	0.76	10, 135	0.64
Motion*Emotion*Lateral-Medial	0.87	5, 135	0.49
Motion*Emotion*Lateral*Medial*Group	1.02	10, 135	0.43
Hemisphere*Lateral-Medial	2.27	1, 27	0.14
Hemisphere*Lateral-Medial*Group	0.19	2, 27	0.83
Motion*Hemisphere*Lateral-Medial	0.99	1, 27	0.33
Motion*Hemisphere*Lateral-Medial*Group	0.40	2, 27	0.67
Emotion*Hemisphere*Lateral-Medial	0.77	5, 135	0.54
Emotion*Hemisphere*Lateral-Medial*Group	2.82	10, 135	0.01
Motion*Emotion*Hemisphere*Lateral-Medial	2.15	5, 135	0.08
Motion*Emotion*Hemisphere*Lateral-Medial*Group	0.70	10, 135	0.69
Motion*Dorsal-Ventral	4.42	1, 27	0.05
Motion*Dorsal-Ventral*Group	2.88	2, 27	0.07
Emotion*Dorsal-Ventral	0.72	5, 135	0.55
Emotion*Dorsal-Ventral*Group	0.68	10, 135	0.68
Motion*Emotion*Dorsal-Ventral	1.93	5, 135	0.11
Motion*Emotion*Dorsal-Ventral*Group	1.60	10, 135	0.14
Hemisphere*Dorsal-Ventral	0.00	1, 27	0.99
Hemisphere*Dorsal-Ventral*Group	0.20	2, 27	0.82
Motion*Hemisphere*Dorsal-Ventral	2.57	1, 27	0.12

Motion*Hemisphere*Dorsal-Ventral*Group	0.42	2, 27	0.66
Emotion*Hemisphere*Dorsal-Ventral	0.87	5, 135	0.50
Emotion*Hemisphere*Dorsal-Ventral*Group	1.34	10, 135	0.22
Motion*Emotion*Hemisphere*Dorsal-Ventral	0.48	5, 135	0.74
Motion*Emotion*Hemisphere*Dorsal-Ventral*Group	0.42	10, 135	0.90
Lateral-Medial	0.24	1, 27	0.63
Lateral-Medial*Group	0.79	2, 27	0.47
Motion*Lateral-Medial*Dorsal-Ventral	0.12	1, 27	0.73
Motion*Lateral-Medial*Dorsal-Ventral*Group	0.06	2, 27	0.94
Emotion*Lateral-Medial*Dorsal-Ventral	0.70	5, 135	0.60
Emotion*Lateral-Medial*Dorsal-Ventral*Group	0.83	10, 135	0.58
Motion*Emotion*Lateral-Medial*Dorsal-Ventral	1.19	5, 135	0.32
Motion*Emotion*Lateral-Medial*Dorsal-Ventral*Group	1.72	10, 135	0.11
Hemisphere*Lateral-Medial*Dorsal-Ventral	0.10	1, 27	0.76
Hemisphere*Lateral-Medial*Dorsal-Ventral*Group	0.24	2, 27	0.79
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.34	1, 27	0.57
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Group	0.37	2, 27	0.69
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.77	5, 135	0.55
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Group	1.11	10, 135	0.36
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	1.49	5, 135	0.21
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Group	0.34	10, 135	0.96

Chapter 5 P1 Latency ANOVA Table

Main Effects & Interactions	F	df	p
Motion	5.52	1, 27	0.03
Motion*Group	1.15	2, 27	0.33
Emotion	1.53	5, 135	0.20
Emotion*Group	0.62	10, 135	0.77
Hemisphere	0.93	1, 27	0.34
Hemisphere*Group	0.09	2, 27	0.92
Lateral-Medial	4.05	1, 27	0.05
Lateral-Medial*Group	0.22	2, 27	0.80
Dorsal-Ventral	19.40	1, 27	0.00
Dorsal-Ventral*Group	0.13	2, 27	0.88
Motion*Emotion	1.83	5, 135	0.12
Motion*Emotion*Group	2.24	10, 135	0.02
Motion*Hemisphere	2.52	1, 27	0.12
Motion*Hemisphere*Group	2.06	2, 27	0.15
Emotion*Hemisphere	2.59	5, 135	0.04
Emotion*Hemisphere*Group	1.19	10, 135	0.31
Motion*Emotion*Hemisphere	2.00	5, 135	0.10
Motion*Emotion*Hemisphere*Group	1.51	10, 135	0.16

Motion*Lateral-Medial	4.14	1, 27	0.05
Motion*Lateral-Medial*Group	2.38	2, 27	0.11
Emotion*Lateral-Medial	0.84	5, 135	0.51
Emotion*Lateral-Medial*Group	0.89	10, 135	0.53
Motion*Emotion*Lateral-Medial	0.70	5, 135	0.61
Motion*Emotion*Lateral*Medial*Group	1.60	10, 135	0.13
Hemisphere*Lateral-Medial	2.98	1, 27	0.10
Hemisphere*Lateral-Medial*Group	1.12	2, 27	0.34
Motion*Hemisphere*Lateral-Medial	0.33	1, 27	0.57
Motion*Hemisphere*Lateral-Medial*Group	0.59	2, 27	0.56
Emotion*Hemisphere*Lateral-Medial	0.52	5, 135	0.73
Emotion*Hemisphere*Lateral-Medial*Group	0.86	10, 135	0.56
Motion*Emotion*Hemisphere*Lateral-Medial	1.92	5, 135	0.11
Motion*Emotion*Hemisphere*Lateral-Medial*Group	1.31	10, 135	0.25
Motion*Dorsal-Ventral	0.32	1, 27	0.58
Motion*Dorsal-Ventral*Group	1.04	2, 27	0.37
Emotion*Dorsal-Ventral	0.49	5, 135	0.75
Emotion*Dorsal-Ventral*Group	1.28	10, 135	0.26
Motion*Emotion*Dorsal-Ventral	0.14	5, 135	0.96
Motion*Emotion*Dorsal-Ventral*Group	1.24	10, 135	0.29
Hemisphere*Dorsal-Ventral	0.05	1, 27	0.82
Hemisphere*Dorsal-Ventral*Group	0.06	2, 27	0.94
Motion*Hemisphere*Dorsal-Ventral	4.25	1, 27	0.05
Motion*Hemisphere*Dorsal-Ventral*Group	0.14	2, 27	0.87
Emotion*Hemisphere*Dorsal-Ventral	0.91	5, 135	0.46
Emotion*Hemisphere*Dorsal-Ventral*Group	1.75	10, 135	0.09
Motion*Emotion*Hemisphere*Dorsal-Ventral	1.14	5, 135	0.34
Motion*Emotion*Hemisphere*Dorsal-Ventral*Group	1.53	10, 135	0.15
Lateral-Medial	14.81	1, 27	0.00
Lateral-Medial*Group	0.09	2, 27	0.91
Motion*Lateral-Medial*Dorsal-Ventral	1.58	1, 27	0.22
Motion*Lateral-Medial*Dorsal-Ventral*Group	1.89	2, 27	0.17
Emotion*Lateral-Medial*Dorsal-Ventral	2.44	5, 135	0.06
Emotion*Lateral-Medial*Dorsal-Ventral*Group	1.10	10, 135	0.37
Motion*Emotion*Lateral-Medial*Dorsal-Ventral	1.34	5, 135	0.26
Motion*Emotion*Lateral-Medial*Dorsal-Ventral*Group	1.80	10, 135	0.09
Hemisphere*Lateral-Medial*Dorsal-Ventral	2.08	1, 27	0.16
Hemisphere*Lateral-Medial*Dorsal-Ventral*Group	0.54	2, 27	0.59
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.09	1, 27	0.77
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Group	0.99	2, 27	0.38
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.26	5, 135	0.89
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Group	1.06	10, 135	0.39
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	1.90	5, 135	0.12
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Group	1.19	10, 135	0.32

Chapter 5 N170 Amplitude ANOVA Table

Main Effects & Interactions	F	df	p
Motion	57.52	1, 27	0.00
Motion*Group	1.17	2, 27	0.32
Emotion	2.71	5, 135	0.04
Emotion*Group	1.48	10, 135	0.18
Hemisphere	12.83	1, 27	0.00
Hemisphere*Group	0.42	2, 27	0.66
Lateral-Medial	9.61	1, 27	0.00
Lateral-Medial*Group	0.74	2, 27	0.49
Dorsal-Ventral	38.08	1, 27	0.00
Dorsal-Ventral*Group	1.10	2, 27	0.35
Motion*Emotion	0.74	5, 135	0.58
Motion*Emotion*Group	1.52	10, 135	0.15
Motion*Hemisphere	1.75	1, 27	0.20
Motion*Hemisphere*Group	0.13	2, 27	0.88
Emotion*Hemisphere	2.46	5, 135	0.05
Emotion*Hemisphere*Group	1.37	10, 135	0.23
Motion*Emotion*Hemisphere	1.47	5, 135	0.22
Motion*Emotion*Hemisphere*Group	1.63	10, 135	0.12
Motion*Lateral-Medial	0.00	1, 27	0.96
Motion*Lateral-Medial*Group	3.81	2, 27	0.04
Emotion*Lateral-Medial	1.17	5, 135	0.33
Emotion*Lateral-Medial*Group	0.86	10, 135	0.56
Motion*Emotion*Lateral-Medial	0.90	5, 135	0.47
Motion*Emotion*Lateral*Medial*Group	2.92	10, 135	0.01
Hemisphere*Lateral-Medial	3.80	1, 27	0.06
Hemisphere*Lateral-Medial*Group	0.04	2, 27	0.96
Motion*Hemisphere*Lateral-Medial	3.26	1, 27	0.08
Motion*Hemisphere*Lateral-Medial*Group	0.29	2, 27	0.75
Emotion*Hemisphere*Lateral-Medial	0.86	5, 135	0.48
Emotion*Hemisphere*Lateral-Medial*Group	0.93	10, 135	0.49
Motion*Emotion*Hemisphere*Lateral-Medial	1.53	5, 135	0.20
Motion*Emotion*Hemisphere*Lateral-Medial*Group	1.68	10, 135	0.11
Motion*Dorsal-Ventral	44.48	1, 27	0.00
Motion*Dorsal-Ventral*Group	0.46	2, 27	0.64
Emotion*Dorsal-Ventral	1.88	5, 135	0.13
Emotion*Dorsal-Ventral*Group	2.18	10, 135	0.04
Motion*Emotion*Dorsal-Ventral	2.17	5, 135	0.09
Motion*Emotion*Dorsal-Ventral*Group	0.54	10, 135	0.80
Hemisphere*Dorsal-Ventral	5.15	1, 27	0.03
Hemisphere*Dorsal-Ventral*Group	0.50	2, 27	0.61
Motion*Hemisphere*Dorsal-Ventral	0.27	1, 27	0.61

Motion*Hemisphere*Dorsal-Ventral*Group	3.28	2, 27	0.05
Emotion*Hemisphere*Dorsal-Ventral	1.05	5, 135	0.38
Emotion*Hemisphere*Dorsal-Ventral*Group	1.30	10, 135	0.26
Motion*Emotion*Hemisphere*Dorsal-Ventral	2.05	5, 135	0.10
Motion*Emotion*Hemisphere*Dorsal-Ventral*Group	1.10	10, 135	0.37
Lateral-Medial	23.63	1, 27	0.00
Lateral-Medial*Group	0.22	2, 27	0.80
Motion*Lateral-Medial*Dorsal-Ventral	6.60	1, 27	0.02
Motion*Lateral-Medial*Dorsal-Ventral*Group	0.59	2, 27	0.56
Emotion*Lateral-Medial*Dorsal-Ventral	0.30	5, 135	0.86
Emotion*Lateral-Medial*Dorsal-Ventral*Group	0.52	10, 135	0.83
Motion*Emotion*Lateral-Medial*Dorsal-Ventral	0.44	5, 135	0.78
Motion*Emotion*Lateral-Medial*Dorsal-Ventral*Group	0.91	10, 135	0.51
Hemisphere*Lateral-Medial*Dorsal-Ventral	0.00	1, 27	0.98
Hemisphere*Lateral-Medial*Dorsal-Ventral*Group	0.41	2, 27	0.67
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.70	1, 27	0.41
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Group	3,32	2, 27	0.05
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	1.22	5, 135	0.31
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Group	1.55	10, 135	0.16
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	1.62	5, 135	0.17
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Group	1.47	10, 135	0.18

Chapter 5 N170 Latency ANOVA Table

Main Effects & Interactions	F	df	p
Motion	1.19	1, 27	0.28
Motion*Group	2.80	2, 27	0.08
Emotion	0.76	5, 135	0.55
Emotion*Group	0.84	10, 135	0.56
Hemisphere	7.80	1, 27	0.01
Hemisphere*Group	3.13	2, 27	0.06
Lateral-Medial	0.17	1, 27	0.68
Lateral-Medial*Group	7.44	2, 27	0.00
Dorsal-Ventral	0.21	1, 27	0.65
Dorsal-Ventral*Group	0.12	2, 27	0.89
Motion*Emotion	2.40	5, 135	0.05
Motion*Emotion*Group	1.81	10, 135	0.08
Motion*Hemisphere	0.48	1, 27	0.50
Motion*Hemisphere*Group	1.07	2, 27	0.36
Emotion*Hemisphere	0.30	5, 135	0.83
Emotion*Hemisphere*Group	0.63	10, 135	0.71

Motion*Emotion*Hemisphere	2.80	5, 135	0.03
Motion*Emotion*Hemisphere*Group	1.37	10, 135	0.22
Motion*Lateral-Medial	0.32	1, 27	0.58
Motion*Lateral-Medial*Group	0.71	2, 27	0.50
Emotion*Lateral-Medial	13.25	5, 135	0.00
Emotion*Lateral-Medial*Group	1.24	10, 135	0.29
Motion*Emotion*Lateral-Medial	2.29	5, 135	0.07
Motion*Emotion*Lateral*Medial*Group	1.11	10, 135	0.37
Hemisphere*Lateral-Medial	2.04	1, 27	0.17
Hemisphere*Lateral-Medial*Group	3.07	2, 27	0.06
Motion*Hemisphere*Lateral-Medial	1.08	1, 27	0.31
Motion*Hemisphere*Lateral-Medial*Group	0.84	2, 27	0.44
Emotion*Hemisphere*Lateral-Medial	1.57	5, 135	0.19
Emotion*Hemisphere*Lateral-Medial*Group	0.58	10, 135	0.79
Motion*Emotion*Hemisphere*Lateral-Medial	2.45	5, 135	0.05
Motion*Emotion*Hemisphere*Lateral-Medial*Group	0.72	10, 135	0.68
Motion*Dorsal-Ventral	4.12	1, 27	0.05
Motion*Dorsal-Ventral*Group	1.62	2, 27	0.22
Emotion*Dorsal-Ventral	2.17	5, 135	0.10
Emotion*Dorsal-Ventral*Group	0.67	10, 135	0.67
Motion*Emotion*Dorsal-Ventral	0.42	5, 135	0.79
Motion*Emotion*Dorsal-Ventral*Group	2.64	10, 135	0.01
Hemisphere*Dorsal-Ventral	3.27	1, 27	0.08
Hemisphere*Dorsal-Ventral*Group	3.50	2, 27	0.04
Motion*Hemisphere*Dorsal-Ventral	1.90	1, 27	0.18
Motion*Hemisphere*Dorsal-Ventral*Group	0.25	2, 27	0.78
Emotion*Hemisphere*Dorsal-Ventral	3.15	5, 135	0.02
Emotion*Hemisphere*Dorsal-Ventral*Group	1.48	10, 135	0.18
Motion*Emotion*Hemisphere*Dorsal-Ventral	1.70	5, 135	0.17
Motion*Emotion*Hemisphere*Dorsal-Ventral*Group	0.51	10, 135	0.82
Lateral-Medial	0.37	1, 27	0.55
Lateral-Medial*Group	3.12	2, 27	0.06
Motion*Lateral-Medial*Dorsal-Ventral	0.12	1, 27	0.74
Motion*Lateral-Medial*Dorsal-Ventral*Group	0.14	2, 27	0.87
Emotion*Lateral-Medial*Dorsal-Ventral	3.33	5, 135	0.02
Emotion*Lateral-Medial*Dorsal-Ventral*Group	1.05	10, 135	0.40
Motion*Emotion*Lateral-Medial*Dorsal-Ventral	4.15	5, 135	0.01
Motion*Emotion*Lateral-Medial*Dorsal-Ventral*Group	2.91	10, 135	0.01
Hemisphere*Lateral-Medial*Dorsal-Ventral	4.83	1, 27	0.04
Hemisphere*Lateral-Medial*Dorsal-Ventral*Group	0.30	2, 27	0.75
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral	1.23	1, 27	0.28
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Group	4.35	2, 27	0.02
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	2.57	5, 135	0.04
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Group	1.22	10, 135	0.29
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	1.66	5, 135	0.17
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Group	0.70	10, 135	0.68

Appendix C Chapter 7: ANOVA Tables for the P1 and N290

Chapter 7 P1 Amplitude ANOVA Table

Main Effects & Interactions	F	df	p
Motion	1.48	1, 26	0.23
Motion*Age	3.25	2, 26	0.06
Emotion	0.15	1, 26	0.70
Emotion*Age	1.85	2, 26	0.18
Hemisphere	35.76	1, 26	0.00
Hemisphere*Age	0.21	2, 26	0.81
Lateral-Medial	50.19	1, 26	0.00
Lateral-Medial*Age	0.13	2, 26	0.88
Dorsal-Ventral	112.59	1, 26	0.00
Dorsal-Ventral*Age	1.03	2, 26	0.37
Motion*Emotion	1.39	1, 26	0.25
Motion*Emotion*Age	0.78	2, 26	0.47
Motion*Hemisphere	1.80	1, 26	0.19
Motion*Hemisphere*Age	0.15	2, 26	0.87
Emotion*Hemisphere	1.37	1, 26	0.25
Emotion*Hemisphere*Age	0.81	2, 26	0.45
Motion*Emotion*Hemisphere	1.71	1, 26	0.20
Motion*Emotion*Hemisphere*Age	0.34	2, 26	0.71
Motion*Lateral-Medial	20.51	1, 26	0.00
Motion*Lateral-Medial*Age	2.07	2, 26	0.15
Emotion*Lateral-Medial	10.10	1, 26	0.00
Emotion*Lateral-Medial*Age	0.65	2, 26	0.53
Motion*Emotion*Lateral-Medial	2.54	1, 26	0.12
Motion*Emotion*Lateral*Medial*Age	0.23	2, 26	0.80
Hemisphere*Lateral-Medial	5.08	1, 26	0.03
Hemisphere*Lateral-Medial*Age	0.02	2, 26	0.98
Motion*Hemisphere*Lateral-Medial	0.87	1, 26	0.36
Motion*Hemisphere*Lateral-Medial*Age	0.88	2, 26	0.43
Emotion*Hemisphere*Lateral-Medial	6.29	1, 26	0.02
Emotion*Hemisphere*Lateral-Medial*Age	5.63	2, 26	0.01
Motion*Emotion*Hemisphere*Lateral-Medial	0.01	1, 26	0.92
Motion*Emotion*Hemisphere*Lateral-Medial*Age	0.25	2, 26	0.78
Motion*Dorsal-Ventral	0.08	1, 26	0.78
Motion*Dorsal-Ventral*Age	2.00	2, 26	0.16
Emotion*Dorsal-Ventral	0.34	1, 26	0.57
Emotion*Dorsal-Ventral*Age	0.48	2, 26	0.63
Motion*Emotion*Dorsal-Ventral	3.11	1, 26	0.09
Motion*Emotion*Dorsal-Ventral*Age	0.49	2, 26	0.62
Hemisphere*Dorsal-Ventral	6.52	1, 26	0.02
Hemisphere*Dorsal-Ventral*Age	1.75	2, 26	0.19

Motion*Hemisphere*Dorsal-Ventral	0.42	1, 26	0.52
Motion*Hemisphere*Dorsal-Ventral*Age	0.08	2, 26	0.92
Emotion*Hemisphere*Dorsal-Ventral	0.62	1, 26	0.44
Emotion*Hemisphere*Dorsal-Ventral*Age	0.62	2, 26	0.54
Motion*Emotion*Hemisphere*Dorsal-Ventral	0.50	1, 26	0.49
Motion*Emotion*Hemisphere*Dorsal-Ventral*Age	0.04	2, 26	0.96
Lateral-Medial	13.78	1, 26	0.00
Lateral-Medial*Age	0.45	2, 26	0.64
Motion*Lateral-Medial*Dorsal-Ventral	5.84	1, 26	0.02
Motion*Lateral-Medial*Dorsal-Ventral*Age	0.01	2, 26	0.99
Emotion*Lateral-Medial*Dorsal-Ventral	0.01	1, 26	0.94
Emotion*Lateral-Medial*Dorsal-Ventral*Age	0.81	2, 26	0.46
Motion*Emotion*Lateral-Medial*Dorsal-Ventral	0.10	1, 26	0.76
Motion*Emotion*Lateral-Medial*Dorsal-Ventral*Age	0.58	2, 26	0.57
Hemisphere*Lateral-Medial*Dorsal-Ventral	0.03	1, 26	0.87
Hemisphere*Lateral-Medial*Dorsal-Ventral*Age	0.99	2, 26	0.38
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral	1.48	1, 26	0.24
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Age	0.78	2, 26	0.47
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.07	1, 26	0.79
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Age	0.67	2, 26	0.52
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.96	1, 26	0.34
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Age	0.02	2, 26	0.98

Chapter 7 P1 Latency ANOVA Table

Main Effects & Interactions	F	df	p
Motion	0.94	1, 26	0.34
Motion*Age	2.07	2, 26	0.15
Emotion	2.68	1, 26	0.11
Emotion*Age	1.29	2, 26	0.29
Hemisphere	2.64	1, 26	0.12
Hemisphere*Age	1.93	2, 26	0.17
Lateral-Medial	20.99	1, 26	0.00
Lateral-Medial*Age	1.86	2, 26	0.18
Dorsal-Ventral	34.07	1, 26	0.00
Dorsal-Ventral*Age	1.13	2, 26	0.34
Motion*Emotion	2.67	1, 26	0.11
Motion*Emotion*Age	0.37	2, 26	0.70
Motion*Hemisphere	8.16	1, 26	0.01
Motion*Hemisphere*Age	4.26	2, 26	0.03
Emotion*Hemisphere	0.57	1, 26	0.46
Emotion*Hemisphere*Age	4.87	2, 26	0.02

Motion*Emotion*Hemisphere	0.38	1, 26	0.54
Motion*Emotion*Hemisphere*Age	0.03	2, 26	0.97
Motion*Lateral-Medial	1.36	1, 26	0.25
Motion*Lateral-Medial*Age	2.71	2, 26	0.09
Emotion*Lateral-Medial	0.19	1, 26	0.67
Emotion*Lateral-Medial*Age	2.04	2, 26	0.15
Motion*Emotion*Lateral-Medial	0.00	1, 26	0.97
Motion*Emotion*Lateral*Medial*Age	5.30	2, 26	0.01
Hemisphere*Lateral-Medial	11.27	1, 26	0.00
Hemisphere*Lateral-Medial*Age	0.10	2, 26	0.01
Motion*Hemisphere*Lateral-Medial	4.29	1, 26	0.05
Motion*Hemisphere*Lateral-Medial*Age	1.77	2, 26	0.19
Emotion*Hemisphere*Lateral-Medial	1.12	1, 26	0.30
Emotion*Hemisphere*Lateral-Medial*Age	1.47	2, 26	0.25
Motion*Emotion*Hemisphere*Lateral-Medial	0.04	1, 26	0.84
Motion*Emotion*Hemisphere*Lateral-Medial*Age	0.06	2, 26	0.95
Motion*Dorsal-Ventral	0.25	1, 26	0.62
Motion*Dorsal-Ventral*Age	0.43	2, 26	0.66
Emotion*Dorsal-Ventral	0.92	1, 26	0.35
Emotion*Dorsal-Ventral*Age	4.90	2, 26	0.02
Motion*Emotion*Dorsal-Ventral	0.50	1, 26	0.83
Motion*Emotion*Dorsal-Ventral*Age	0.52	2, 26	0.60
Hemisphere*Dorsal-Ventral	10.37	1, 26	0.00
Hemisphere*Dorsal-Ventral*Age	0.36	2, 26	0.70
Motion*Hemisphere*Dorsal-Ventral	0.06	1, 26	0.80
Motion*Hemisphere*Dorsal-Ventral*Age	1.69	2, 26	0.21
Emotion*Hemisphere*Dorsal-Ventral	3.69	1, 26	0.07
Emotion*Hemisphere*Dorsal-Ventral*Age	0.48	2, 26	0.62
Motion*Emotion*Hemisphere*Dorsal-Ventral	0.07	1, 26	0,79
Motion*Emotion*Hemisphere*Dorsal-Ventral*Age	2.34	2, 26	0.12
Lateral-Medial	10.53	1, 26	0.00
Lateral-Medial*Age	1.56	2, 26	0.23
Motion*Lateral-Medial*Dorsal-Ventral	0.51	1, 26	0.48
Motion*Lateral-Medial*Dorsal-Ventral*Age	1.05	2, 26	0.36
Emotion*Lateral-Medial*Dorsal-Ventral	0.01	1, 26	0.93
Emotion*Lateral-Medial*Dorsal-Ventral*Age	1.21	2, 26	0.32
Motion*Emotion*Lateral-Medial*Dorsal-Ventral	1.39	1, 26	0.25
Motion*Emotion*Lateral-Medial*Dorsal-Ventral*Age	0.86	2, 26	0.43
Hemisphere*Lateral-Medial*Dorsal-Ventral	1.14	1, 26	0.30
Hemisphere*Lateral-Medial*Dorsal-Ventral*Age	3.86	2, 26	0.03
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral	4.69	1, 26	0.04
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Age	1.59	2, 26	0.22
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	2.25	1, 26	0.15
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Age	2.06	2, 26	0.15
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.61	1, 26	0.44
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Age	1.30	2, 26	0.29

Chapter 7 N170 Amplitude ANOVA Table

Effect or interaction	F	df	p
Motion	2.66	1, 26	0.12
Motion*Age	1.71	2, 26	0.20
Emotion	0.00	1, 26	0.97
Emotion*Age	0.47	2, 26	0.63
Hemisphere	39.84	1, 26	0.00
Hemisphere*Age	0.16	2, 26	0.85
Lateral-Medial	30.01	1, 26	0.00
Lateral-Medial*Age	0.72	2, 26	0.50
Dorsal-Ventral	65.06	1, 26	0.00
Dorsal-Ventral*Age	1.66	2, 26	0.21
Motion*Emotion	2.74	1, 26	0.11
Motion*Emotion*Age	1.42	2, 26	0.26
Motion*Hemisphere	3.06	1, 26	0.09
Motion*Hemisphere*Age	0.40	2, 26	0.67
Emotion*Hemisphere	0.01	1, 26	0.93
Emotion*Hemisphere*Age	1.23	2, 26	0.31
Motion*Emotion*Hemisphere	0.36	1, 26	0.56
Motion*Emotion*Hemisphere*Age	0.42	2, 26	0.66
Motion*Lateral-Medial	10.04	1, 26	0.00
Motion*Lateral-Medial*Age	0.68	2, 26	0.51
Emotion*Lateral-Medial	6.18	1, 26	0.02
Emotion*Lateral-Medial*Age	0.41	2, 26	0.67
Motion*Emotion*Lateral-Medial	0.69	1, 26	0.42
Motion*Emotion*Lateral*Medial*Age	0.49	2, 26	0.62
Hemisphere*Lateral-Medial	0.09	1, 26	0.77
Hemisphere*Lateral-Medial*Age	0.03	2, 26	0.97
Motion*Hemisphere*Lateral-Medial	3.18	1, 26	0.09
Motion*Hemisphere*Lateral-Medial*Age	0.43	2, 26	0.65
Emotion*Hemisphere*Lateral-Medial	9.59	1, 26	0.01
Emotion*Hemisphere*Lateral-Medial*Age	2.39	2, 26	0.11
Motion*Emotion*Hemisphere*Lateral-Medial	0.88	1, 26	0.36
Motion*Emotion*Hemisphere*Lateral-Medial*Age	0.50	2, 26	0.61
Motion*Dorsal-Ventral	0.37	1, 26	0.56
Motion*Dorsal-Ventral*Age	2.00	2, 26	0.16
Emotion*Dorsal-Ventral	1.05	1, 26	0.32
Emotion*Dorsal-Ventral*Age	0.50	2, 26	0.61
Motion*Emotion*Dorsal-Ventral	2.89	1, 26	0.10
Motion*Emotion*Dorsal-Ventral*Age	2.61	2, 26	0.09
Hemisphere*Dorsal-Ventral	8.11	1, 26	0.01
Hemisphere*Dorsal-Ventral*Age	1.07	2, 26	0.36
Motion*Hemisphere*Dorsal-Ventral	0.67	1, 26	0.42

Motion*Hemisphere*Dorsal-Ventral*Age	1.08	2, 26	0.35
Emotion*Hemisphere*Dorsal-Ventral	1.56	1, 26	0.22
Emotion*Hemisphere*Dorsal-Ventral*Age	0.06	2, 26	0.95
Motion*Emotion*Hemisphere*Dorsal-Ventral	0.70	1, 26	0.41
Motion*Emotion*Hemisphere*Dorsal-Ventral*Age	0.51	2, 26	0.61
Lateral-Medial	16.36	1, 26	0.00
Lateral-Medial*Age	0.32	2, 26	0.73
Motion*Lateral-Medial*Dorsal-Ventral	8.31	1, 26	0.01
Motion*Lateral-Medial*Dorsal-Ventral*Age	0.69	2, 26	0.51
Emotion*Lateral-Medial*Dorsal-Ventral	0.08	1, 26	0.79
Emotion*Lateral-Medial*Dorsal-Ventral*Age	0.74	2, 26	0.49
Motion*Emotion*Lateral-Medial*Dorsal-Ventral	1.77	1, 26	0.20
Motion*Emotion*Lateral-Medial*Dorsal-Ventral*Age	1.19	2, 26	0.32
Hemisphere*Lateral-Medial*Dorsal-Ventral	0.39	1, 26	0.54
Hemisphere*Lateral-Medial*Dorsal-Ventral*Age	3.72	2, 26	0.04
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.22	1, 26	0.65
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Age	0.57	2, 26	0.57
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.27	1, 26	0.61
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Age	3.83	2, 26	0.04
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.06	1, 26	0.82
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Age	1.41	2, 26	0.26

Chapter 7 N170 Latency ANOVA Table

Main Effects & Interactions	F	df	p
Motion	0.19	1, 26	0.69
Motion*Age	0.34	2, 26	0.72
Emotion	0.56	1, 26	0.46
Emotion*Age	3.54	2, 26	0.04
Hemisphere	0.00	1, 26	0.98
Hemisphere*Age	0.30	2, 26	0.74
Lateral-Medial	4.08	1, 26	0.05
Lateral-Medial*Age	0.89	2, 26	0.43
Dorsal-Ventral	0.02	1, 26	0.89
Dorsal-Ventral*Age	0.47	2, 26	0.63
Motion*Emotion	0.04	1, 26	0.84
Motion*Emotion*Age	2.62	2, 26	0.09
Motion*Hemisphere	2.45	1, 26	0.13
Motion*Hemisphere*Age	5.49	2, 26	0.01
Emotion*Hemisphere	3.88	1, 26	0.06
Emotion*Hemisphere*Age	0.36	2, 26	0.70

Motion*Emotion*Hemisphere	3.21	1, 26	0.09
Motion*Emotion*Hemisphere*Age	1.91	2, 26	0.17
Motion*Lateral-Medial	1.64	1, 26	0.21
Motion*Lateral-Medial*Age	0.85	2, 26	0.44
Emotion*Lateral-Medial	2.90	1, 26	0.10
Emotion*Lateral-Medial*Age	3.02	2, 26	0.07
Motion*Emotion*Lateral-Medial	0.00	1, 26	0.98
Motion*Emotion*Lateral*Medial*Age	0.54	2, 26	0.59
Hemisphere*Lateral-Medial	13.46	1, 26	0.00
Hemisphere*Lateral-Medial*Age	3.78	2, 26	0.04
Motion*Hemisphere*Lateral-Medial	0.08	1, 26	0.78
Motion*Hemisphere*Lateral-Medial*Age	0.23	2, 26	0.80
Emotion*Hemisphere*Lateral-Medial	1.95	1, 26	0.17
Emotion*Hemisphere*Lateral-Medial*Age	1.78	2, 26	0.19
Motion*Emotion*Hemisphere*Lateral-Medial	0.86	1, 26	0.36
Motion*Emotion*Hemisphere*Lateral-Medial*Age	2.21	2, 26	0.13
Motion*Dorsal-Ventral	0.36	1, 26	0.56
Motion*Dorsal-Ventral*Age	2.64	2, 26	0.09
Emotion*Dorsal-Ventral	0.39	1, 26	0.54
Emotion*Dorsal-Ventral*Age	0.05	2, 26	0.96
Motion*Emotion*Dorsal-Ventral	13.82	1, 26	0.00
Motion*Emotion*Dorsal-Ventral*Age	0.04	2, 26	0.96
Hemisphere*Dorsal-Ventral	2.23	1, 26	0.15
Hemisphere*Dorsal-Ventral*Age	1.13	2, 26	0.34
Motion*Hemisphere*Dorsal-Ventral	0.06	1, 26	0.81
Motion*Hemisphere*Dorsal-Ventral*Age	0.00	2, 26	1.00
Emotion*Hemisphere*Dorsal-Ventral	0.35	1, 26	0.56
Emotion*Hemisphere*Dorsal-Ventral*Age	1.15	2, 26	0.33
Motion*Emotion*Hemisphere*Dorsal-Ventral	2.17	1, 26	0.15
Motion*Emotion*Hemisphere*Dorsal-Ventral*Age	1.95	2, 26	0.16
Lateral-Medial	0.42	1, 26	0.52
Lateral-Medial*Age	0.03	2, 26	0.97
Motion*Lateral-Medial*Dorsal-Ventral	2.50	1, 26	0.13
Motion*Lateral-Medial*Dorsal-Ventral*Age	0.94	2, 26	0.41
Emotion*Lateral-Medial*Dorsal-Ventral	1.88	1, 26	0.18
Emotion*Lateral-Medial*Dorsal-Ventral*Age	1.92	2, 26	0.17
Motion*Emotion*Lateral-Medial*Dorsal-Ventral	0.00	1, 26	0.96
Motion*Emotion*Lateral-Medial*Dorsal-Ventral*Age	0.73	2, 26	0.49
Hemisphere*Lateral-Medial*Dorsal-Ventral	0.01	1, 26	0.94
Hemisphere*Lateral-Medial*Dorsal-Ventral*Age	0.00	2, 26	1.00
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral	2.25	1, 26	0.15
Motion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Age	2.66	2, 26	0.09
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.48	1, 26	0.49
Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Age	2.10	2, 26	0.14
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral	0.01	1, 26	0.92
Motion*Emotion*Hemisphere*Lateral-Medial*Dorsal-Ventral*Age	0.94	2, 26	0.40

Appendix D Chapter 6: Empathy Quotient

The Cambridge Behaviour Scale

Please fill in this information and then read the instructions below.

ALL INFORMATION REMAINS STRICTLY CONFIDENTIAL

Name: ……. Sex: ……

Date of birth: ……..…….. Today's date: ……

How to fill out the questionnaire

Below are a list of statements. Please read each statement very carefully and rate how strongly you agree or disagree with it by circling your answer. There are no right or wrong answers, or trick questions.

IN ORDER FOR THE SCALE TO BE VALID, YOU MUST ANSWER EVERY QUESTION.

Examples

El. I would be very upset if I couldn't listen to music every day.	strongly agree	slightly agree	slightly disagree	strongly disagree
E2. I prefer to speak to my friends on the phone rather than write letters to them.	strongly agree	slightly agree	slightly disagree	strongly disagree
E3. I have no desire to travel to different parts of the world,	strongly agree	slightly agree	slightly disagree	strongly

disagree

E4. I prefer to read than to dance. strongly agree slightly agree slightly disagree strongly disagree

1. I can easily tell if someone else wants to enter a conversation, strongly agree slightly agree slightly disagree strongly disagree

2. I prefer animals to humans. strongly agree slightly agree slightly disagree strongly disagree

3. I try to keep up with the current trends and fashions, strongly agree slightly agree slightly disagree strongly disagree

4. I find it difficult to explain to others things that I understand easily, when they don't understand it first time. strongly agree slightly agree slightly disagree strongly disagree

5. I dream most nights. strongly agree slightly agree slightly disagree strongly disagree

6. I really enjoy caring for other people. strongly agree slightly agree slightly disagree strongly disagree

7. I try to solve my own problems rather than discussing them with others. strongly agree slightly agree slightly disagree strongly disagree

8. I find it hard to know what to do in a social situation. strongly agree slightly agree slightly disagree strongly disagree

9. I am at my best first thing in the morning. strongly agree slightly agree slightly disagree strongly disagree

10. People often tell me that I went too far in driving strongly slightly slightly

strongly
my point home in a discussion. agree agree disagree
disagree

11. It doesn't bother me too much if I am late meeting strongly slightly slightly
strongly
a friend. agree agree disagree
disagree

12. Friendships and relationships are just too difficult, strongly slightly slightly
strongly
so I tend not to bother with them. agree agree disagree
disagree

13. I would never break a law, no matter how minor. strongly slightly slightly
strongly
agree agree disagree
disagree

14. I often find it difficult to judge if something is strongly slightly slightly
strongly
rude or polite. agree agree disagree
disagree

15. In a conversation, I tend to focus on my own strongly slightly slightly
strongly
thoughts rather than on what my listener might be agree agree disagree
disagree
thinking.

16. I prefer practical jokes to verbal humour. strongly slightly slightly
strongly
agree agree disagree
disagree

17. I live life for today rather than the future. strongly slightly slightly
strongly
agree agree disagree
disagree

18. When I was a child, I enjoyed cutting up worms strongly slightly slightly
strongly
to see what would happen. agree agree disagree
disagree

19. I can pick up quickly if someone says one thing strongly slightly slightly
strongly
but means another. agree agree disagree
disagree

20. I tend to have very strong opinions about morality. strongly slightly slightly

	strongly agree	agree	disagree	strongly disagree
21. It is hard for me to see why some things upset people so much.	strongly agree	slightly agree	slightly disagree	strongly disagree
22. I find it easy to put myself in somebody else's shoes.	strongly agree	slightly agree	slightly disagree	strongly disagree
23. I think that good manners are the most important thing a parent can teach their child,	strongly agree	slightly agree	slightly disagree	strongly disagree
24. I like to do things on the spur of the moment.	strongly agree	slightly agree	slightly disagree	strongly disagree
25. I am good at predicting how someone will feel.	strongly agree	slightly agree	slightly disagree	strongly disagree
26. I am quick to spot when someone in a group is feeling awkward or uncomfortable,	strongly agree	slightly agree	slightly disagree	strongly disagree
27. If I say something that someone else is offended by, I think that's their problem, not mine.	strongly agree	slightly agree	slightly disagree	strongly disagree
28. If anyone asked me if I liked their haircut, I would reply truthfully, even if I didn't like it.	strongly agree	slightly agree	slightly disagree	strongly disagree
29. I can't always see why someone should have felt offended by a remark.	strongly agree	slightly agree	slightly disagree	strongly disagree
30. People often tell me that I am very unpredictable.	strongly agree	slightly agree	slightly disagree	strongly disagree

31. I enjoy being the centre of attention at any social gathering	strongly agree	slightly agree	slightly disagree	strongly disagree
32. Seeing people cry doesn't really upset me.	strongly agree	slightly agree	slightly disagree	strongly disagree
33. I enjoy having discussions about politics.	strongly agree	slightly agree	slightly disagree	strongly disagree
34. I am very blunt, which some people take to be rudeness, even though this is unintentional,	strongly agree	slightly agree	slightly disagree	strongly disagree
35. I don't tend to find social situations confusing.	strongly agree	slightly agree	slightly disagree	strongly disagree
36. Other people tell me I am good at understanding how they are feeling and what they are thinking.	strongly agree	slightly agree	slightly disagree	strongly disagree
37. When I talk to people, I tend to talk about their experiences rather than my own.	strongly agree	slightly agree	slightly disagree	strongly disagree
38. It upsets me to see an animal in pain,	strongly agree	slightly agree	slightly disagree	strongly disagree
39. I am able to make decisions without being influenced by people's feelings,	strongly agree	slightly agree	slightly disagree	strongly disagree
40. I can't relax until I have done everything I had planned to do that day.	strongly agree	slightly agree	slightly disagree	strongly disagree
41. I can easily tell if someone else is interested or	strongly	slightly	slightly	

bored with what I am saying.	strongly agree	agree	slightly agree	slightly disagree	strongly disagree
42. I get upset if I see people suffering on news programmes.	strongly agree	agree	slightly agree	slightly disagree	strongly disagree
43. Friends usually talk to me about their problems as they say that I am very understanding.	strongly agree	agree	slightly agree	slightly disagree	strongly disagree
44. I can sense if I am intruding, even if the other person doesn't tell me.	strongly agree	agree	slightly agree	slightly disagree	strongly disagree
45. I often start new hobbies but quickly become bored with them and move on to something else.	strongly agree	agree	slightly agree	slightly disagree	strongly disagree
46. People sometimes tell me that I have gone too far with teasing.	strongly agree	agree	slightly agree	slightly disagree	strongly disagree
47. I would be too nervous to go on a big rollercoaster.	strongly agree	agree	slightly agree	slightly disagree	strongly disagree
48. Other people often say that I am insensitive, though I don't always see why.	strongly agree	agree	slightly agree	slightly disagree	strongly disagree
49. If I see a stranger in a group, I think that it is up to them to make an effort to join in.	strongly agree	agree	slightly agree	slightly disagree	strongly disagree
50. I usually stay emotionally detached when watching a film	strongly agree	agree	slightly agree	slightly disagree	strongly disagree
51. I like to be very organised in day to day life and	strongly	slightly	slightly		strongly

often make lists of the chores I have to do. strongly agree slightly agree slightly disagree strongly disagree

52. I can tune into how someone else feels rapidly and intuitively, strongly agree slightly agree slightly disagree strongly disagree

53. I don't like to take risks. strongly agree slightly agree slightly disagree strongly disagree

54. I can easily work out what another person might want to talk about. strongly agree slightly agree slightly disagree strongly disagree

55. I can tell if someone is masking their true emotion. strongly agree slightly agree slightly disagree strongly disagree

56. Before making a decision I always weigh up the pros and cons. strongly agree slightly agree slightly disagree strongly disagree

57. I don't consciously work out the rules of social situations. strongly agree slightly agree slightly disagree strongly disagree

58. I am good at predicting what someone will do. strongly agree slightly agree slightly disagree strongly disagree

59. I tend to get emotionally involved with a friend's problems. strongly agree slightly agree slightly disagree strongly disagree

60. I can usually appreciate the other person's viewpoint, even if I don't agree with it. strongly agree slightly agree slightly disagree strongly disagree

Thank you for filling this questionnaire in.

11. Appendix E Chapter 6: Social Functioning Scale

Social Functioning Scale

Name:_____
Sex:_____

Date of birth:_____ Today's date:_____

Instructions

This form asks some questions about your everyday life. Please complete each question by circling the appropriate choice, filling in the blank, or ticking the appropriate box. If you have any questions about what to do, please ask.

Part 1

1. What time do you get up each day?

   (a) Average weekday (*circle one choice*)

   Before 9am 9-11am 11am-1pm After 1pm

   (b) Average weekend (*circle one choice*)

   Before 9am 9-11am 11am-1pm After 1pm

2. How many hours of the day do you spend alone? (*circle one choice*)

   Very little time Some of the time Quite a lot

   A great deal of time Practically all the time

3. How often do you start a conversation at home? (*circle one choice*)

   Almost never Rarely Sometimes Often

4. How often do you leave the house? (*circle one choice*)

   Almost never Rarely Sometimes Often

5. How do you react in the presence of strangers? (*circle one choice*)

   Avoid them Feel nervous Accept them Like them

Part 2

1. How many friends do you have at the moment? _____ *(please write the number in this space)*

2. Do you have a boy/girlfriend? (*circle one choice*)

   Yes No

3. How often are you able to carry out a sensible and rational conversation? (*circle one choice*)

   Never Rarely Sometimes Often

4. How easy or difficult do you find talking to people at the moment? (*circle one choice*)

   Very easy Quite easy Average Quite difficult Very difficult

Part 3

Place a tick in the box for each item to show how often you have carried out each of the following during the **past 3 months**

	Never	Rarely	Sometimes	Often
Buying items from shops (without help)				
Washing up, tidying up etc				
Regular washing & bathing				

Part 3, Continued

Place a tick in the box for each item to show how often you have carried out each of the following during **past 3 months**

	Never	Rarely	Sometimes	Often
Doing the food				

shopping				
Prepare & cook a meal				
Leaving the house alone				
Using public transport				
Using money				
Budgeting				
Choosing & buying clothes for self				
Take care of personal appearance				

Part 4

Place a tick in the box for each item to show how often you have carried out each of the following during the **past 3 months**

	Never	Rarely	Sometimes	Often
Playing musical instruments				
Sewing, knitting				
Gardening				
Reading				
Watching television				
Listening to records or radio				
Cooking				
DIY activities				
Fixing things (car, bike, household etc)				
Walking, rambling				
Driving/cycling (as recreation)				
Swimming				
Hobby (e.g. collecting things)				
Shopping				
Artistic activity (painting etc)				

Part 5

Place a tick in the box for each item to show how often you have carried out each of the following during the **past 3 months**

	Never	Rarely	Sometimes	Often
Cinema				
Theatre/concert				
Watching indoor sports (squash, table-tennis)				
Watching an outdoor sport (football, rugby)				
Art gallery/museum				
Exhibition				
Visiting places of interest				
Meetings, talks etc				
Evening classes				
Visiting relatives in their homes				
Being visited by relatives				
Visiting friends (including boyfriend/girlfriend)				
Parties				
Formal occasions				
Disco etc				
Night/social club				
Playing an indoor sport				
Playing an outdoor sport				
Club/society				
Pub				
Eating out				
Church activity				

Part 6

Place a tick in the box for each item to show how able you are at doing or using the following.

	Adequately	Needs help	Unable	Not known
Public transport				
Handling money				
Budgeting				
Cooking for self				
Weekly shopping				
Looking for a job				
Washing own clothes				
Personal hygiene				
Washing, tidying up				
Purchasing from shops				
Leaving the house alone				
Choosing & buying clothes				
Caring for personal appearance				

Part 7

1. Are you in regular employment? (Includes industrial therapy, rehabilitation or retraining courses) (*circle one choice*)

   Yes No

   (a) If yes
   What sort of job is it?_____
   How many hours do you work each week?_____
   How long have you had this job?_____

   (b) If no
   When were you last in paid employment?_____
   What sort of a job was it?_____
   How many hours did you work per week?_____

2. Are you registered disabled? (*circle one choice*)

   Yes No

3. Do you attend hospital as a day patient? (*circle one choice*)

   Yes No

If unemployed:

4. Do you think that you are capable of some sort of employment? (*circle one choice*)

   Definitely yes Would have difficulty Definitely no

5. How often do you attempt to find a new job? (e.g. go to the job centre, read the newspaper) (*circle one choice*)

   Almost never Rarely Sometimes Often