Emotions Are Rising: The Growing Field of Affect Neuropsychology

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Abstract
Thirty years ago, the neuropsychology of emotion started to emerge as a mainstream topic. Careful examination of individual patients showed that emotion, like memory, language, and so on, could be differentially affected by brain disorders, especially in the right hemisphere. Since then, there has been an accelerating interest in uncovering the neural architecture of emotion, and the major steps in this process of discovery over the past 3 decades are detailed in this review. In the 1990s, magnetic resonance imaging (MRI) scans provided precise delineation of lesions in the amygdala, medial prefrontal cortex, insula and somatosensory cortex as underpinning emotion disorders. At the same time, functional MRI revealed activation that was bilateral and also lateralized according to task demands. In the 2000s, converging evidence suggested at least two routes to emotional responses: subcortical, automatic and autonomic responses and slower, cortical responses mediating cognitive processing. The discovery of mirror neurons in the 1990s reinvigorated older views that simulation was the means to recognize emotions and empathize with others. More recently, psychophysiological research, revisiting older Russian paradigms, has contributed new insights into how autonomic and other physiological indices contribute to decision making (the somatic marker theory), emotional simulation, and social cognition. Finally, this review considers the extent to which these seismic changes in understanding emotional processes in clinical disorders have been reflected in neuropsychological practice. (JINS, 2017, 23, 719–731)

Keywords: Emotion, Affect, Social cognition, Arousal, Decision making, Social cognition

The neuropsychological assessment of emotion is typically overshadowed by assessment of language, executive processes, learning, and so on, as affect is difficult to assess using traditional approaches. Despite the challenges, emotion is an aspect of brain function that is a critical determinant of human behavior and so fundamental to neuropsychology. The past 3 decades has seen growing momentum in understanding its neural underpinnings and clinical implications.

In 1872, Darwin described emotions as behavioral patterns that are important for the species’ survival (Darwin, 1872). Supporting this, seminal cross-cultural research (Ekman and Friesen, 1971; Izard, 1971) revealed that people of different cultures are highly consistent in their expression of anger, fear, happiness, surprise, disgust, and sadness, suggesting these are innate and integral to communication. This set the stage for a generation of research that almost exclusively focused on the perception, expression, and experience of these six basic emotions. Increasingly, the social nature of emotions is coming into focus and other emotions (trust, envy, flirtation, etc.) are being examined (Adolphs, Tranel, & Damasio, 1998; Reynders, Broks, Dickson, Lee, & Turpin, 2005; Rosenberg, McDonald, Rosenberg, & Westbrook, 2016). These are more subject to learning and cultural variation but, none-the-less, critical when considering how emotion disorders disrupt social competence. The following overview (see Table 1 for summary) considers major milestones in research of the basic emotions in the past 30 years.

THE 1980s: THE RIGHT HEMISPHERE IN EMOTION PROCESSING

In 1975, Heilman and colleagues reported several patients with right hemisphere lesions who could not comprehend emotional prosody of speech (Heilman, Scholes, & Watson, 1975). In 1979, Ross and Mesulam described two cases with right hemisphere lesions who lost their capacity to impart affect in speech. Thus, the right hemisphere was attributed a role in affective speech processing, potentially mirroring the role of the left hemisphere in propositional speech (Ross, 1981). The 1980s onward saw sustained research extending the role of the right hemisphere in emotion to include expressivity and perception of face Borod, Koff, Lorch,
### Table 1. Major milestones in emotion research over the past 30 years

<table>
<thead>
<tr>
<th>Time</th>
<th>Method</th>
<th>Findings</th>
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<tbody>
<tr>
<td>1971</td>
<td>Cross-cultural studies of emotion</td>
<td>Reporting that basic categories of emotion are invariant across cultures, consistent with the view that they are innate and mediated by specific neural structures.</td>
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<td>1975–1980</td>
<td>Observation/testing of normal adults and adults with lateralised lesions on CT scans</td>
<td>First reporting of RH patients with disorders of emotional prosody. Evidence that RH is dominant for emotions and/or specialised for negative emotions.</td>
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<td>1980s</td>
<td>Testing children with autism</td>
<td>First suggestion that social dysfunction in autism reflects a lack of ToM.</td>
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<td>1990s</td>
<td>MRI scans of people with disorders of emotion</td>
<td>Specific structures found to be associated with emotions: amygdala, prefrontal cortex, anterior cingulate gyrus, insula, and somatosensory cortex; RH often but not always implicated.</td>
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<td></td>
<td>fMRI scans of normal adults engaged with emotional materials</td>
<td>Bilateral activation of same structures as above; differential activation of LH vs. RH dependent on task demands.</td>
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<td>Single cell recording of neurons in the macaque monkey</td>
<td>Discovery of mirror neurons in F5, that fire when an action is executed and when observing someone else performing that action.</td>
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<td></td>
<td>Skin conductance recording in adults with brain lesions</td>
<td>Evidence that patients with ventromedial frontal lesions lack an orienting response to emotional material.</td>
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<td></td>
<td>Iowa Gambling Task given to adults with frontal/amygdala lesions</td>
<td>Evidence of impaired task performance and lack of skin conductance used to propose somatic marker theory, i.e., that people use somatic cues to guide complex decision making.</td>
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<td>2000s</td>
<td>Convergence of MRI, fMRI, and animal research</td>
<td>Evidence of a mirror neuron system in human inferior frontal/parietal areas; proposal that we simulate actions and experiences of others and, therefore, understand the intentions behind them.</td>
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<tr>
<td></td>
<td>fMRI scans of normal adults experiencing actions, touch and pain and observing others doing the same</td>
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<td>2010s</td>
<td>Facial mimicry, skin conductance, etc., in adults with brain lesions</td>
<td>Increasing evidence (some from decades earlier) for the role of simulation in emotional understanding and empathy; evidence for a specific loss of response to negative materials in some clinical disorders.</td>
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<td></td>
<td>Convergence and expansion of ToM and emotion research in patients with neurological, psychiatric and developmental disorders</td>
<td>Development of a broad construct of social cognition, a specialized system for understanding others that entails both affective and cognitive aspects. Rapid expansion of interest in the field as indicated by numerous new journals emerging with specialized focus on social and affective neuroscience.</td>
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*Note. “Patients” refer to adults with brain lesions. RH = right hemisphere; LH = left hemisphere; CT = computed tomography; MRI = magnetic resonance imaging; fMRI = functional MRI; ToM = Theory of Mind.*
Nicholas, 1985, 1986) and gestures (e.g., crying and laughing) (Ross & Mesulam, 1979). During the 1980s, for the first time, it was demonstrated that people with right hemisphere damage had broader problems extracting emotional content from pictures (Cicone, Wapner, & Gardner, 1980) and words (Brownell, Michel, Powelson, & Gardner, 1983; Wapner, Hamby, & Gardner, 1981), leading to the view that the right hemisphere is dominant for emotional processing generally (Heilman, Bowers, & Valenstein, 1985). Research in healthy adults supported this, finding a right hemisphere advantage for emotion perception via tachistoscopic viewing (Heller & Levy, 1981) and dichotic listening (Bryden, Ley, & Sugarman, 1982) and for facial expressivity (Borod & Caron, 1980).

A contemporaneous, alternative view was the “valence hypothesis,” that is, that the left hemisphere is specialized for positive and the right hemisphere specialized for negative emotions (Silberman & Weingartner, 1986) or the related “approach (left) - withdrawal (right) hypothesis” (Davidson, 1984). Rapid presentation of facial expressions to one hemisphere in normal adults (Ley & Bryden, 1979; Reuter-Lorenz & Davidson, 1981) demonstrated a hemispheric bias for processing positive (left) and negative (right) emotions (Silberman & Weingartner, 1986). Patients with right hemisphere damage were reportedly indifferent or euphoric while those with left hemisphere damage commonly experienced depression (Gainotti, 1972; Sackeim et al., 1982). These responses were thought to reflect the disablement of normal positive, approach related responses following left hemisphere lesions (thus a swing to depression) and conversely the disablement of negative, withdrawal responses following right hemisphere lesions (thus the swing to euphoria) (Davidson, Pizzagalli, Nitschke, & Kalin, 2003). Research into emotion perception has also suggested a valence effect with the right hemisphere dominant for negative emotions while both hemispheres played a role in positive (Adolphs, Jansari, & Tranel, 2001).

THE 1990s: MRI IDENTIFIES SPECIFIC STRUCTURES UNDERPINNING EMOTION

Lesion-based research in the 1980s was hampered by two issues. First, most work focused on patients with middle cerebral artery stroke who were heterogeneous with respect to intra-hemispheric pathology. At the same time, computed tomography (CT) produced low-resolution images. In the 1990s, magnetic resonance imaging (MRI) provided dramatic improvements in the visualization of brain lesions and identification of specific structures associated with emotion processing.

The amygdala, interconnected with the hypothalamus, thalamus, basal forebrain, and brainstem, as well as neo-cortex, reciprocally interacts with the autonomic nervous system (Emery & Amaral, 2000) making it integral to emotional behavior. Using MRI, rare patients with focal amygdala damage were identified who had impairments in recognizing fear and other negative emotions in faces (Adolphs, Tranel, Damasio, & Damasio, 1994) and voices (Scott et al., 1997) and loss of the autonomic fear response to aversive stimuli (Bechara et al., 1995). Lesions were usually bilateral, although in some studies right amygdala lesions produced poorer performance than left (Adolphs & Tranel, 2004; Adolphs, Tranel, & Damasio, 2001).

The prefrontal lobes also represented an important candidate for mediating emotions. It was long known that frontal lobe injury led to changes to socio-emotional behavior (Benton, 1968; Stuss & Benson, 1986; Weinstein & Kahn, 1955). In the 1990s, circumscribed lesions in the orbital/medial prefrontal cortex and ventral, anterior cingulate cortex were identified as specific to emotional behavior, associated with poor emotion perception, lowered self-reported emotional experience, especially for negative emotions, and apathy, disinhibition, lability, aggression, and personality change (Barrash, Tranel, & Anderson, 2000; Grafman et al., 1996; Hornak, Rolls, & Wade, 1996). This pattern led to views that the medial prefrontal system (especially the right) has a role in regulating “high autonomic arousal” emotions such as anger and fear (Adolphs, 2002a). Lesions to the (left) anterior insula cortex along with the basal ganglia were also implicated, specifically perception of disgust in patients with focal brain lesions (Calder, Keane, Manes, Antoun, & Young, 2000) and in Huntington’s disease (Sprengelmeyer et al., 1996). Selective impairment in fear (amygdala) and disgust (insula) were regarded as evidence for a double dissociation, demonstrating that specific emotions were associated with specific structures (Adolphs, 2002b).

A major stepping stone in current emotion theories came from evidence that emotion perception could be disrupted by lesions in somatosensory and motor cortex (especially in the right hemisphere: Adolphs, Damasio, & Tranel, 2002; Adolphs, Damasio, Tranel, & Damasio, 1996). These studies suggested that processing visual or auditory affective stimuli, “as if” it were one’s own, might play a role in its identification. Dissociations were also reported suggesting that prosody and facial affect engaged overlapping but discrete cerebral systems (Adolphs et al., 2002) as did moving versus static emotional stimuli (Adolphs, Tranel, & Damasio, 2003).

THE 1990s: FUNCTIONAL IMAGING REVEALS NEURAL ACTIVATION

In parallel to MRI lesion research, functional imaging rapidly emerged as a powerful adjunct, providing opportunities to observe localized brain activity in normal adults when viewing emotional materials. Using positron emission tomography (PET) normal adults showed left amygdala activation to angry and fearful faces even when their attention was diverted from the expression (Morris et al., 1996). This was even found in a patient with unilateral brain damage and no conscious awareness of an image transmitted to his blinded right hemi-field (Morris, DeGelder, Weiskrantz, & Dolan, 2001). Functional magnetic resonance imaging (fMRI; Belliveau et al., 1991), a cheaper, less-invasive technique than PET, led to an explosion of interest in emotion. There have been literally hundreds of fMRI studies of normal adults exposed to emotional stimuli.
Meta-analyses of these generally suggest bilateral activation of specific structures in response to emotional stimuli. Amygdala activation was found when participants passively observe facial expressions. Despite earlier reports, there was little evidence of greater activation to negative expressions (Baas, Aleman, & Kahn, 2004; Costafreda, Brammer, David, & Fu, 2008; Sergerie, Chocol, & Armony, 2008). Left and right amygdala activation varied due to task. Language-based tasks activate the left amygdala, whereas masked emotions are more likely to activate the right amygdala briefly with rapid habituation (Costafreda et al., 2008; Sergerie et al., 2008).

Using fMRI meta-analysis, Lindquist, Wager, Kober, Bliss-Moreau, and Barrett (2012) also challenged traditional views that certain types of emotion (such as fear) were mediated by specific structures (such as the amygdala). Their results suggested key structures are not activated by specific emotions but, rather, play a role that is important for specific emotions. Thus, the amygdalae are engaged in orienting responses to novel and arousing events rather than fear specifically, the insula is engaged in self-awareness of body sensations rather than disgust in particular, and the ventromedial frontal lobes appears to integrate external and internal high arousal sensations rather than simply mediating anger. It has also been shown that early sensory cortices are activated by affective (vs. neutral) sensory input, suggesting a role in emotional experience beyond simply processing sensory inputs (Satpute et al., 2015).

2000s: TWO NEURAL MODELS OF EMOTION PROCESSES

The explosion of MRI, fMRI, animal, and experimental research during the 1990s provided the impetus to develop two complementary models of neural systems of emotion that emerged in the 2000s.

Explicit Versus Implicit (Autonomic) Processing

Animal research by LeDoux and colleagues suggested that the “flight or fight” response is mediated by very rapid, coarse evaluation of potential sources of danger in the environment processed mainly by subcortical routes (LeDoux, 1995). In humans, a ventral system was identified that entails the amygdalae, insula, ventral striatum, and anterior cingulate and prefrontal cortex. This system identifies emotionally significant stimuli; produces affective, autonomic responses; and provides automatic regulation (Lieberman, 2007; Phillips, Drevets, Rauch, & Lane, 2003).

In contrast, complex emotional judgements occur more slowly, engaging the dorsal lateral and medial prefrontal cortex and anterior cingulate gyrus as well as hippocampus and temporo-parietal regions (Lieberman, 2007; Phillips et al., 2003). This system mediates effortful processing of emotional stimuli and engagement of relevant cognitive processes (e.g., language). The two systems are intimately and reciprocally associated. Emotion tasks that require verbal mediation, for example, labeling, increase activation of dorsolateral prefrontal cortex (Hariri, Bookheimer, & Mazzotta, 2000; Keightly et al., 2003) and reduce activation in both amygdalae (Hariri et al., 2000).

Shared Neural Representations for Self Versus Other

In 1992, a major breakthrough occurred with the discovery of mirror neurons in the F5 area of the macaque’s frontal cortex (approximating the human Broca’s area), which activated when the subject both undertook a meaningful action and observed that same action in others (di Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992). It has since been revealed that, in humans also, overlapping areas in the inferior frontal lobe, including the premotor area, and inferior and superior parietal lobes activate when both observing and performing actions (Grèzes & Decety, 2001; Molenberghs, Cunnington, & Mattingley, 2012) or even hearing sounds associated with actions (Gazzola, Aziz-Zadeh, & Keysers, 2006).

Such “mirroring” only occurs to meaningful actions and is thought to provide a mental representation of an action and its goal, allowing the observer to anticipate and understand the intention behind it (Rizzolatti & Sinigaglia, 2010). Emotional stimuli lead to similar patterns of activation extending to the insula, amygdalae, and the superior temporal sulcus (Carr, Iacoboni, Dubeau, Mazziotta, & Lenzi, 2003; Pfeifer, Iacoboni, Mazziotta, & Dapretto, 2008), suggesting a role for action simulation in emotion understanding (Iacoboni, 2009).

Whether this co-activation reflects mirror neurons in humans remains controversial. Unlike single cell recordings in monkeys (Rizzolatti, Fadiga, Gallese, & Fogassi, 1996), most evidence in humans is indirect arising from voxel-based patterns of activations, although single-cell recordings in humans support their existence (Mukamel, Ekstrom, Kaplan, Iacoboni, & Fried, 2010). Even so, the notion that overlapping areas mediate both direct emotional experience and observed experience has gained momentum. Furthermore, simulation is not limited to observing actions. Both simple touch to the hand and observation of touch activate the somatosensory cortex (Kuehn, Mueller, Turner, & Schutz-Bosbach, 2014; Schaefer, Heinze, & Rotte, 2012). The anterior cingulate gyrus, thalamus, somatosensory cortices, and insula are activated when experiencing painful stimuli in oneself with overlapping but not identical structures involved when observing others in pain (Jackson, Rainville, & Decety, 2006).

PHYSIOLOGICAL EVIDENCE FOR MODELS OF EMOTION PROCESSES

fMRI and other techniques, such as electroencephalography (Fox et al., 2016), provide unprecedented opportunity to observe brain activation. However, as they are correlational in nature, they give only indirect evidence of the functional significance and, on occasion, this may be erroneous. For example, Feinstein et al. (2016) reported a rare patient with
bilateral lesions of the “pain matrix,” that is, the insula, anterior cingulate, and amygdala, who still experienced pain, sometimes excessively. They argued that the common interpretation that the pain matrix mediates the emotional significance of pain may be misdirected, and it may have another role, for example, regulation.

Psychophysiological measurements provide a complementary approach to understanding emotional processing. Although such measures had a long tradition in Russian neuropsychology from the 1960s, much of this literature was never published in English (e.g., see Luria, 1973). Western psychophysiological research, which has accelerated since the 2000s, has converged with theorizing from structural and functional imaging to refine understanding of emotion processes.

EVIDENCE FOR AUTONOMIC RESPONSES TO EMOTIONAL STIMULI

In normal adults, skin conductance responses (SCR, indicating the orientation reflex) occur in response to fearful stimuli even when participants are not overtly aware of them (Esteves, Dimberg, & Ohman, 1994; Ohman & Soares, 1994). Damasio, Tranel, and Damasio (1990) reported that five patients with ventromedial frontal lesions lacked this orienting response to emotionally charged pictures. Furthermore, people with traumatic brain injuries (TBIs), many of whom experience ventromedial frontal pathology, have abnormally low arousal (skin conductance level) at baseline (Fisher, Rushby, McDonald, Parks, & Pignut, 2015; McDonald, Rushby, et al., 2011) and habituate unusually rapidly to angry faces (McDonald, Rushby, et al., 2011). Their startle response is also abnormally unresponsive to negative images (Saunders, McDonald, & Richardson, 2006; Williams & Wood, 2012). A similar lack of startle response was reported in people with amygdala lesions (Angrilli et al., 1996; Buchanan, Tranel, & Adolphs, 2004; Funayama, Grillon, Davis, & Phelps, 2001) supporting the notion that the amygdalae, in concert with the ventromedial frontal lobes, mediate autonomic responses to emotional events with or without conscious awareness.

Evidence for Simulation

Psychophysiological techniques have also shed light on the role of simulation in emotion. Studies of normal adults in the 1990s demonstrated that observers experience activation of their own cheek (zygomaticus major) and brow (corrugator supercilii) muscles when observing another person expressing happy or angry emotions, respectively. Mimicry occurs spontaneously and rapidly, even without conscious awareness (Dimberg & Thunberg, 1998; Dimberg, Thunberg, & Elmehed, 2000), suggesting an automatic process. This observation re-invigorated a view initially proposed by Lipps (1907, cited in Hoffman, 1984) that facial feedback engenders a shared emotional experience (emotional contagion) that helps identify the emotion being observed. This view clearly dovetails into implications of the mirror neuron system. Furthermore, deficits in facial mimicry have been reported in people with low empathic ability (Sonnby-Borgström, Jönsson, & Svensson, 2003) as well as clinical conditions associated with low empathy, such as schizophrenia (Park, Matthews, & Gibson, 2008).

Clinical evidence based on people with lesions to ventromedial frontal cortex or as a result of TBI suggests that loss of emotion simulation extends beyond facial mimicry. While there can be loss of facial expressivity (Dethier, Blairy, Rosenberg, & McDonald, 2012) and spontaneous facial mimicry (Angrilli, Palomba, Cantagallo, Maietti, & Stegagno, 1999; McDonald, Li, et al., 2011), there is also lowered autonomic arousal to facial expressions (Blair & Cipolotti, 2000; de Sousa et al., 2011; Hopkins, Dywan, & Segalowitz, 2002) and reduced subjective appraisal of emotional material (Hornak et al., 1996; Saunders et al., 2006) and the effects of postural feedback (Dethier, Blairy, Rosenberg, & McDonald, 2013).

Thus, disruption of emotional contagion can occur at numerous points in a complex system from motor mimicry through to arousal and the subjective evaluation of the emotional experience. Of interest, when emotional contagion is impaired, this is usually limited to negative emotions (Blair & Cipolotti, 2000; McDonald, Rushby, et al., 2011). This valence specific effect suggests that disruption occurs at a different stage to mirroring which, otherwise, should affect all emotions equally. It does, however, fit with the notion that autonomic responses are mediated by the ventral system (Phillips et al., 2003) that has evolved to detect potential danger and elicit an immediate arousal response.

While both emotion perception and emotional contagion can be disrupted following brain lesions, evidence for a causal link is far from established. On the one hand, accumulating behavioral evidence suggests that loss of mimicry impedes emotion recognition. Disrupting the (left) premotor cortex using transient magnetic stimulation decreases emotion recognition accuracy and increases reaction time (Balconi & Bortolotti, 2013). Botulin toxin that impairs facial movement also reduces emotional experience and emotion recognition (Davis, Senghas, Brandt, & Ochsner, 2010). People with locked in syndrome and facial paralysis are less efficient than normal when recognizing (negative) emotions (Pistoia et al., 2010) as are normal adults prevented from mimicry (Niedenthal, Brauer, Halberstadt, & Innes-Ker, 2001).

However, there is also evidence to the contrary. While loss of contagion and poor emotion recognition do co-occur in individual patients (Blair & Cipolotti, 2000), they are not correlated in people with brain injury (McDonald, Li, et al., 2011; McDonald, Rushby, et al., 2011) nor in adults with a complete disorder of peripheral autonomic function (Heims, Critchley, Dolan, Mathias, & Cipolotti, 2004). A similar lack of relation is seen in normal adults (Blairy, Herrera, & Hess, 1999; Hess & Blairy, 2001). Thus, while mimicry may facilitate emotion perception, there is insufficient evidence that it is essential. It may have other functions, for example, to communicate empathic understanding (Bavelas, Black, Chovil, Lemery, & Mullett, 1988).
ATTRIBUTING EMOTIONS WITH A ROLE IN COGNITION

A major development in contemporary neuropsychology has been the recognition that emotion has a role in cognition. Two important theoretical developments have been the somatic marker theory (1990s) and the construct of social cognition (2000s).

Somatic Marker Theory

The influential “somatic marker” hypothesis arose in the 1990s (Damasio, Tranel, & Damasio, 1991), suggesting that emotional responses can guide understanding of complex response contingencies even before conscious awareness (Bechara, Damasio, Tranel, & Damasio, 1997). Most work used the Iowa Gambling Task (IGT) in which people learn reward contingencies given feedback. Both normal adults and those with ventromedial frontal lobe lesions have a SCR following feedback about their choices. Normal adults reportedly demonstrate these changes in anticipation of their choice, even before they can verbalize the rules. People with ventromedial damage do not (Bechara et al., 1997; Bechara, Tranel, Damasio, & Damasio, 1998; Naccache et al., 2005). Similar results were found with people with specific amygdala damage, except in these cases, there was no SCR at all, even after feedback (Bechara, Damasio, Damasio, & Lee, 1999).

As a result of this work, it was argued that somatic responses assist with decision making, especially in personal and social situations, where precise calculations of the outcome of a course of action is not possible (Bechara, 2004). Although interest in the somatic marker theory has been high, poor performance on the IGT is not limited to those with frontal lesions (Levine et al., 2005). Other cognitive abilities, such as working memory (Hinson, Jameson, & Whitney, 2002), intelligence (Toplak, Sorge, Benoit, West, & Stanovich, 2010), and reversal learning (Fellows & Farah, 2005) influence IGT performance.

Whether physiological responses occur before conscious awareness also remains controversial. Densely amnesic patients who cannot learn the IGT do not display anticipatory SCRs (despite having intact SCRs to punishment), and healthy adults have demonstrated differential anticipatory SCRs only after explicit knowledge of IGT contingencies was attained (Fernie & Tunney, 2013; Gutbrod et al., 2006). In all, it appears that somatic markers are involved in decision making on the IGT, as is conscious processing (Guillaume et al., 2009), but are not necessarily critical to successful performance.

Social Cognition

Since the 2000s, there has been a groundswell of interest in the role of emotional processes in social performance. This was heralded by the 1980s research describing how emotional and humorous materials were poorly processed following right hemisphere lesions. In 1985, Baron Cohen, Leslie, and Frith proposed that children with autism lack the ability to impute mental states, that is, lack Theory of Mind (ToM; Baron Cohen, Leslie, and Frith, 1985). More recently, these attributes have been subsumed under the construct of social cognition, that is, the ability to understand the minds of other people in relation to ourselves (Amor and Frith, 2006), including judgments about what they are thinking, feeling, and intending by their behavior.

Social cognition also includes empathy, social connectedness, and making broader social judgments, such as attitudes about social groups and morality. Not all social cognition is related to emotion. Cognitive ToM tasks simply require understanding what another person might think and believe. On the other hand, affective ToM refers to understanding what another person feels (Mitchell & Phillips, 2015). fMRI research has demonstrated activation of the anterior dorsal medial prefrontal cortex and bilateral temporo-parietal junctions when performing any mental judgment but with additional activation of the orbitofrontal cortex, temporal poles, and bilateral prefrontal cortex for affective judgements (Molenberghs, Johnson, Henry, & Mattingley, 2016). Lesion studies suggest that ventromedial (especially right) lesions are associated with impaired affective ToM judgments, such as understanding irony and faux pas (Shamay-Tsoory, Tomer, Berger, Goldsher, & Aharon-Peretz, 2005).

IMPLICATIONS FOR CONTEMPORARY CLINICAL PRACTICE

Since the late 1990s, it has been recognized that impairments in emotion processing are pivotal to understanding neuropsychiatric disorders (Borod et al., 1999), including schizofrenia (Kohler, Walker, Martin, Healey, & Moberg, 2010; Phillips, 2003), bipolar and other mood disorders (Cusi, Nazarow, Holshausen, MacQueen, & McKinnon, 2012; Green, Cahill, & Mahli, 2007), and cerebral and genetic developmental disabilities (Roelofs, Wingermühle, Egger, & Kessels, 2017), as well as people with acquired brain injury. Characterization or socio-emotional impairments has greatly aided diagnosis and understanding of these disorders. For example, psychosocial changes following severe TBI are common (Brooks, Campsie, Symington, Beattie, & McKinlay, 1986) but were poorly understood before systematic research into emotional impairments.

Recently it has been estimated that up to 39% of adults with moderate-severe TBI suffer problems of emotion perception (Babbage et al., 2011). They also self-report disproportionately low levels of empathy (Williams & Wood, 2010; Wood & Williams, 2008), blunted emotional experience (Croker & McDonald, 2005), and low self-awareness of their own emotions (i.e., alexithymia; Williams & Wood, 2010). Up to 70% of adults with TBI demonstrate apathy (Kant, Duffy, & Pivovarnik, 1998), and irritability is one of the most common behavioral complaints (Demark & Gemeinhardt, 2002) along with impulsivity and aggression (Kinsella, Packer, & Olver, 1991). Emotion disorders are not necessarily correlated to standard neuropsychological tests (Spikman, Timmerman, Milders, Veenstra, & van der Naalt, 2012), but do predict social behavioral problems in adults. 
In frontotemporal dementia, different constellations of emotion impairment characterize sub-types. Semantic dementia, arising from initial asymmetric deterioration of the temporal lobes (primarily left), causes problems, not only with language and semantic comprehension, but restricted food interests; changed eating behavior; increased apathy; impaired emotion perception, insight, and empathy; poor mental state judgments; and poor emotional memories (Landin-Romero, Tan, Hodges, & Kumfor, 2016). Patients can also present with asymmetric deterioration of the right temporal lobe, resulting in even more severe emotion perception and behavioral problems, which suggests that it is the right temporal lobe that mediates these deficits in both groups (Kumfor et al., 2016).

Behavioral variant frontotemporal dementia is similarly characterized by early behavior disturbance, disinhibition, poor emotion perception, and low empathy; although, in this case, these difficulties appear to arise from bilateral orbitofrontal and left temporal deterioration (Kamminga et al., 2015). Impaired empathy and emotional perception predict carer distress in the frontotemporal dementias (Hazelton, Irish, Hodges, Piguet, & Kumfor, 2016).

Schizophrenia is defined by changes in socioemotional processes (Penn, Corrigan, Bentall, Racenstein, & Newman, 1997), including emotional flattening, anhedonia and percutaneous delusions (Phillips, 2003), disorders of social cognition broadly (Savla, Vella, Armstrong, Penn, & Twamley, 2013), and emotion process specifically (Irani, Seligman, Kamath, Kohler, & Gur, 2012). Poor social cognitive abilities in schizophrenia more accurately predict community function than traditional cognitive measures (Fett et al., 2011).

Assessment

Given the relevance of emotional processes to interpersonal function and outcome, they should be assessed routinely. However, while some clinical measures have been available for decades (Borod, Tabert, Santschi, & Strauss, 2000), this is one area where change has been slow. Despite 84% of clinicians working in brain injury reporting that at least half of their clients had problems in social cognition (including multiple facets of emotion), less than 22% reported using any kind of formal assessment (Kelly, McDonald, & Frith, in press). Furthermore, professions differ in terms of who they believe to be responsible, with psychologists, neuropsychologists, occupational therapists, and speech pathologists each of the view that the responsibility lies with the other (Kelly, McDonald, & Frith, 2017).

Current, consensus-based recommendations about outcome measures in brain injury include self- and proxy questionnaires to assess emotional behaviors (such as aggression and irritability), psychological status (such as negative and positive mood states), and arousal (apathy, disinhibition) (Wilde et al., 2010), and only recently has emotion perception and social cognition been added (Honan et al., in press). In contrast, in the field of schizophrenia, emotion perception has been recognized as a core cognitive ability and included in the National Institute of Mental Health (USA) MATRICS cognition battery (Kern et al., 2008; Nuechterlein et al., 2008).

Although clinicians do not typically assess emotion, there are tests available with normative data. The Florida Affect Battery (Bowers, Blonder, & Heilman, 1991) and the Diagnostic Analysis of Non-Verbal Accuracy (Nowicki, 2010) assess emotion perception as does the social perception substest in the Advanced Clinical Solutions battery (Wechsler, 2009). The Awareness of Social Inference Test (TASIT) (McDonald, Flanagan, & Rollins, 2011; McDonald, Flanagan, Rollins, & Kinch, 2003) uses audiovisual vignettes of actors to assess emotion, mental judgments, and conversational inference (McDonald, 2012) in adults and also adolescents (McDonald et al., 2015), and a brief version has recently been developed (Honan, McDonald, Sufani, Hine, & Kumfor, 2016). The NEPSY-II (Korkman, Kirk, & Kemp, 2007) assesses both emotion and ToM in children from 3 to 16 years while the Social Language Development Test (Bowers, Huisng, & LoGiudice, 2010) and the Clinical Evaluation of Language Fundamentals (Wig & Secord, 2014) assess conversational and pragmatic inference from child to early adulthood.

Rehabilitation For Emotional Disorders

Research into remediation for emotional and social perception deficits started to emerge in the 2000s for people with autism spectrum disorders (Bölte, Feineis-Matthews, & Pousta, 2008), schizophrenia (Combs et al., 2007; Horan, Kern, Green, & Penn, 2008), and acquired brain injury (Bornhofen & McDonald, 2008a, 2008b). Accruing evidence, especially in schizophrenia, suggests that training of emotion perception, via direct instruction, repetitive practice, and role plays, can be effective (Cassel, McDonald, Kelly, & Togher, 2016; Kurtz & Richardson, 2012; Roelofs et al., 2017).

Training of emotion regulation such as anger management has traditionally followed cognitive-behavioral principles (Demark & Gemeinhardt, 2002; Golden & Consorte, 1982). The use of autonomic measures to monitor deficits in arousal and regulation opens the horizons to investigate other techniques, such as biofeedback, to improve heart rate variability and consequently emotional balance (Francis, Fisher, Rushby, & McDonald, 2016). There is also potential in using direct cortical stimulation to improve depression, psychomotor speed (Boggio et al., 2008; Loo et al., 2012), and inhibitory control (Jacobson, Javitt, & Lavidor, 2011).

CONCLUSION

The past 30 years has witnessed a seismic shift in understanding the neural and functional nature of human emotion. From broad sweep notions of right hemisphere dominance, new methodologies have specified brain structures and networks. Breakthroughs have arisen from a convergence of methodologies to identify damaged structures, observe...
normal neural activation, and measure psychophysiological responses at different stages in emotional processing. Emotion research has expanded neuropsychology to consider the role of emotions in cognitive processes such as decision making (Bechara, Tranel, & Damasio, 2000) and memory and learning (Rolls, 2000). The construct of social cognition has emerged that addresses how emotional processes affect social judgements more broadly.

Despite this, many questions remain. One conundrum is the contrast between apparent bilateral activation for emotion perception and social cognition as revealed by fMRI and yet specific right hemisphere involvement suggested by lesion research. This parallels language research where fMRI activation consistently implicates bilateral activation in language tasks in contrast to lesion work that consistently implicates the left hemisphere (see Price, 2012). So what exactly is the contribution of right hemisphere processes? Possibly the right hemisphere plays the larger role in emotion processing simply because the left hemisphere has re-organized to accommodate other functions such as language (De Winter et al., 2015). Alternatively, the right hemisphere may mediate arousal and attention to emotional significant events (Langner & Eickhoff, 2013).

While simulation has captured the research community’s imagination, its neural and functional nature is yet to be articulated. Observation of another’s emotional state causes resonance throughout the brain, from early sensory cortices, through to association and motor cortex, frontal, amygdala, and subcortical processing. This suggests that emotional experience develops continuously, reflecting the fluctuating and dynamic interaction of multiple sources (Raz et al., 2013). However, exactly how feed-forward and feedback interactions occur and their functional significance remains underspecified.

Ongoing research focusing on the temporal dynamics of emotion systems should provide new insights. In the meantime, emotion appears to influence almost every perceptual, mnemonic, cognitive, and executive process in the brain.

The past 15 years has seen a groundswell of interest in the emotional sequelae of various clinical disorders, useful not only diagnostically but because they add to the capacity of neuropsychological testing to predict everyday difficulties (Catran, Oddy, Wood, & Moir, 2011; Fett et al., 2011; Shimokawa et al., 2001). Emotion is also the target for a new era in remediation research. Despite this, neuropsychological practice has changed little. Assessment of emotion perception, emotional experience (other than mood disorders), and/or emotion regulation using available tests/questionnaires is not the norm. Hopefully, this will change as clinical training embraces the latest evidence of the role of emotions and a new generation of clinicians step forward to take up the mantle of assessors and remediators in the complex and vital field of emotion.

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The growing field of affect neuropsychology


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