
CRITICAL REVIEW

Emotion perception deficits following traumatic brain injury: A review of the evidence and rationale for intervention

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Abstract

While the cognitive disturbances that frequently follow severe traumatic brain injury (TBI) are relatively well understood, the ways in which these affect the psychosocial functioning of people with TBI are yet to be determined and have thus received little attention in treatment research. Growing evidence indicates that a significant proportion of individuals with TBI demonstrate an inability to recognize affective information from the face, voice, bodily movement, and posture. Because accurate interpretation of emotion in others is critical for the successful negotiation of social interactions, effective treatments are necessary. Until recently, however, there have been no rehabilitation efforts in this area. The present review examines the literature on emotion perception deficits in TBI and presents a theoretical rationale for targeted intervention. Several lines of research relevant to the remediation of emotion perception in people with TBI are considered. These include work on emotion perception remediation with other cognitively impaired populations, current neuropsychological models of emotion perception and underlying neural systems, and recent conceptualizations of remediation processes. The article concludes with a discussion of the importance of carrying out efforts to improve emotion perception within a contextualized framework in which the day-to-day relevance of training is clear to all recipients. (*JINS*, 2008, *14*, 511–525.)

Keywords: Rehabilitation, Facial expression, Social interaction, Social behavior, Affect, Recovery of function, Cortical contusion, Traumatic brain injury

INTRODUCTION

The effects of traumatic brain injury (TBI) on psychosocial functioning comprise an even greater long-term barrier to adjustment and rehabilitation than the effects on cognitive and physical functioning (Eslinger et al., 1995; Godfrey et al., 1996; Grattan & Ghahramanlou, 2002; Hoofien et al., 2001; Tate et al., 1989b; Thomsen, 1984; Yates, 2003). In the months and years following TBI, a significant proportion of individuals experience breakdowns in one or more aspects of social functioning. This includes loss of employment, disruption to intimate relationships, and reduced social networks (Elsass & Kinsella, 1987; Gosling & Oddy, 1999;

Hallett et al., 1994; Kersel et al., 2001; Oddy & Humphrey, 1980; Oddy et al., 1978; Peters et al., 1990; Tate et al., 1989b; Thomsen, 1974; Weddell et al., 1980; Ylvisaker & Feeney, 2000). In many cases, circumstances do not improve with time, but instead tend to worsen (Burleigh et al., 1998; Dombrov & Olek, 1997; Durgin, 2000; Godfrey & Shum, 2000; Gomez-Hernandez et al., 1997; Hammond et al., 2004; Morton & Wehman, 1995; Oddy et al., 1985; Olver et al., 1996; Tate et al., 1989b).

The principal causes for these social difficulties are likely to be complex, including a variety of internal factors, such as cognitive, emotional, and physical status, and external circumstances, such as reduced social opportunities and limited support (Biddle et al., 1996; Bond & Godfrey, 1997; Chapman, 1997; Chapman et al., 1995; Chapman et al., 1997; Godfrey et al., 1991; Godfrey & Shum, 2000; Marsh, 1999; McDonald, 1992, 1993, 2000; Ylvisaker, 1993). The

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major focus of this review is the role that impaired emotion perception may play in reducing social competence post TBI, and even more importantly, the extent to which this can be seen as a legitimate target for remediation. The notion that emotion perception deficits are a major problem for many people with TBI has only recently been recognized, with just two papers that reported such deficits (Jackson & Moffat, 1987; Prigatano & Pibram, 1982) before the 1990s. There is now, however, an established literature that attests to the prevalence of emotion perception deficits in this population. The emergence of this literature coincides with an increased interest in the efficacy of cognitive rehabilitation for people with brain injuries in general and renewed efforts to provide relevant, theoretically driven approaches to remediation that will lead to real functional gains. In this review, we will address the literature to date that defines the nature of emotion processing, its relevance to social competence and how it is impaired in those with TBI. Next, we will examine evidence for treatment of emotion perception disorders in other populations that may have some relevance to TBI. Finally, we will describe the specific neuropsychological and neuroanatomical underpinnings of emotion perception and relate these to potential remediation approaches.

THE SIGNIFICANCE OF EMOTION PERCEPTION

Emotion perception, that is, the ability to accurately perceive and appreciate affective information from facial expressions, emotional prosody, body posture, and contextual parameters (such as the type of social occasion, the relationship between speakers, etc.), is critical to social competence. Numerous studies of normal adults have demonstrated that those who are poor at reading social cues also experience poor social skills in general (Boice, 1983; Morrison & Bellack, 1981; Trower, 1980). Poor emotion perception has also been shown to be related to social adjustment in school-aged children (Leppanen & Hietanen, 2001).

Furthermore, disruption to emotion perception appears to play a role in a variety of clinical disorders, such as autism (Bolte & Poustka, 2003; Humphreys et al., 2007; Lindner & Rosen, 2006; Mazefsky & Oswald, 2007), schizophrenia (Edwards et al., 2002; Mandal et al., 1998; Tremeau, 2006), attention-deficit hyperactivity disorder (ADHD) in children (Kats-Gold et al., 2007; Pelc et al., 2006), intellectual disability (Rojahn & Warren, 1997; Williams et al., 2005; Wishart et al., 2007), and anxiety disorders (panic disorder, Kessler et al., 2007; obsessive-compulsive disorder, Aigner et al., 2007; social anxiety, Montagne et al., 2006). Indeed, evidence from clinical groups [e.g., TBI (Watts & Douglas, 2006), schizophrenia (Hooker & Park, 2002; Kee et al., 2003; Morrison & Bellack, 1981; Mueser et al., 1996; Sergi et al., 2006), autism (Boraston et al., 2007), and children with ADHD characteristics (Kats-Gold et al., 2007)] mirrors studies of normal adults in suggesting that those who are poor at reading affective information

demonstrate low levels of social skills and/or social functioning. In the schizophrenia field, it has been shown that emotion perception ability is not only related to level of social functioning (Penn et al., 2001) but may adversely affect social functioning independently of both cognitive deficits and positive and negative symptoms of the disorder (Kohler et al., 2000). Others within this field suggest that emotion perception may mediate between cognitive and social functioning in psychosis (Addington et al., 2006). Such findings have clear implications for treatment of emotion perception in schizophrenia (Green et al., 2005). They also highlight the potential importance of emotion perception deficits following TBI.

EMOTION PERCEPTION AND THE CASE OF TBI

The first report of impaired emotion perception in a group of people with TBI was made by Prigatano and Pibram in 1982. Since that time, accumulating evidence has indicated that a significant proportion of people with TBI demonstrate impaired perception of emotional information across a range of media. In terms of visual media, these include both relatively simple (black and white line drawings) and more complex (photographs and video scenes) stimuli, as well as input from different bodily sources, that is, facial expression and body posture (Allerdings & Alfano, 2006; Croker & McDonald, 2005; Green et al., 2004; Jackson & Moffat, 1987; McDonald et al., 2003; McDonald & Saunders, 2005; Milders et al., 2003). Deficits in judging prosodic affective cues were first reported in 1996 (McDonald & Pearce, 1996) and have since then been repeatedly found (McDonald & Saunders, 2005; Milders et al., 2003; Spell & Frank, 2000). Recently, it has been shown that people with TBI also show impairment in interpreting the emotional content of multichannel stimuli in audiovisual format (McDonald & Flanagan, 2004; McDonald et al., 2003; McDonald & Saunders, 2005). Across all modalities, perception of negative emotions (anger, disgust, sadness, and fear) seems to be worse than perception of positive emotions (happiness and surprise; Croker & McDonald, 2005; Green et al., 2004; Hopkins et al., 2002; Jackson & Moffat, 1987; McDonald et al., 2003). Additionally, there is growing evidence of impairment in the ability to make mental state inferences—which rely heavily on the interpretation of affective cues (Bibby & McDonald, 2005; McDonald & Flanagan, 2004; McDonald et al., 2003, 2004).

Deficits in emotion perception vary among individuals with TBI, with some demonstrating little or no impairment on measures used and others performing at near chance levels (Bornhofen & McDonald, in press; Croker & McDonald, 2005; McDonald & Pearce, 1996; McDonald & Saunders, 2005). Similarly, there are suggestions that individuals with TBI who have emotion perception deficits may differ with respect to modality of deficit, such that some cases may demonstrate difficulties with recognizing facial expressions but not emotional prosody, and

others, vice versa (McDonald & Saunders, 2005). These results may reflect heterogeneity with respect to pathology as well as the impact of other cognitive deficits present in this group (Allerdings & Alfano, 2006). Another potentially important factor is the availability of social opportunities for people with TBI who may no longer be able to work or whose mobility has been limited by physical impairments. In such cases, it is possible that sheer isolation from prior social networks and poor community reintegration may maintain or intensify deficits in emotion processing. This possibility has not yet been directly examined in research.

Finally, recent research has raised the possibility that emotional reactivity may be disturbed following TBI (Blair & Cipolotti, 2000; Croker & McDonald, 2005; Hornak et al., 1996; Saunders et al., 2006), including reactivity to facial expressions of others (Angrilli et al., 1999; Blair & Cipolotti, 2000; Hopkins et al., 2002). There is only very limited evidence, to date, that affective reactivity and the ability to accurately recognize emotions in others are related following TBI (Croker & McDonald, 2005; Hornak et al., 1996). However, work with normal adults (e.g., McHugo & Smith, 1996; Wild et al., 2001) has suggested that the identification of and affective responses to emotional expressions are functionally intertwined and, furthermore, that emotional reactions to faces may provide an important cue to their identification (Wild et al., 2001).

In summary, it is apparent that a significant proportion of the TBI population demonstrates specific impairments in the perception of one or more types of nonverbal affective cues used in everyday social encounters. Although the nature of these deficits is still being clarified, evidence to date points to impairments across both visual and auditory modalities, as well as across static and dynamic media, in this population. Perception of negative emotions appears to be relatively more impaired than that of positive emotions. Overall, degrees of impairment across individuals appear to vary widely, as might be expected given the heterogeneous nature of this condition. There is additional evidence that emotional responsivity to facial expressions and other emotionally significant stimuli is also impaired.

REMEDICATION OF DEFICITS IN EMOTION PERCEPTION IN OTHER CLINICAL POPULATIONS

The prevalence of problems related to emotion perception in TBI necessitates effective treatment; however, to date there is little evidence regarding the efficacy of emotion perception training in people with TBI. In contrast, work on remediating emotion perception deficits in other clinical populations with similar cognitive profiles, namely autism, intellectual disability, and schizophrenia, has produced a growing body of evidence in this area. While clear differences exist between these groups and TBI, it is also the case that they share several characteristics that are relevant to treatment such as learning deficits, slowed processing and

impaired executive functioning (Anderson, 2001; Calhoun, 2006; Green et al., 2000; Lezak, 1995; Russo et al., 2007; Welsh et al., 2005). This suggests that valuable insights might be gained from these remediation efforts. The first such insight is that treatment of this kind of deficit, at least in other populations, is feasible. In autism (Bolte et al., 2002, 2006; Solomon et al., 2004) and intellectual disability (McAlpine et al., 1992; McKenzie et al., 2000; Rydin-Orwin et al., 1999), measurable improvement in emotion perception ability has been demonstrated across pre- and posttreatment in all published studies. In some instances, more widespread gains have also been noted. For example, McAlpine and colleagues (1992), working with adults with intellectual disability, reported evidence of generalization from judgments based on photographs to judgments based on videotaped role plays and maintenance of skills for at least 9 months. Bolte and colleagues (2006), working with individuals with autism, observed brain activation changes following treatment in brain regions associated with visuospatial and facial processing. These results are remarkable given the developmental and longstanding nature of these disorders, which are likely to have limited any awareness of emotional material from an early age (Hill et al., 2004; Leonard et al., 2002; Rieffe et al., 2007).

The most extensive research in emotion perception remediation has been carried out in schizophrenia (Combs et al., 2006, 2007; Frommann et al., 2003; Penn & Combs, 2000; van der Gaag et al., 2002; Wolwer et al., 2005). Again, significant gains have been reported in all studies. Of particular note is that some of these studies incorporated strategies such as errorless learning, self-instruction, and direct positive reinforcement, which took into account the particular cognitive impairments presented by its target population (Frommann et al., 2003; van der Gaag et al., 2002; Wolwer et al., 2005). Several of these impairments, such as poor organization, and reduced learning and attention, are shared with the TBI population, which suggests that the techniques have the potential to be relevant to TBI. Indeed, many of these same techniques mentioned have been applied in the TBI population and shown to be effective for remediation of deficits in attention, memory, strategy use, problem solving, and self-regulation of behavior (Burke et al., 1991; Dou et al., 2006; Lawson & Rice, 1989; Melton & Bourgeois, 2005; Squires et al., 1997; Turkstra & Bourgeois, 2005; Webster & Scott, 1983).

Despite these generally positive findings, a potential shortcoming of many of such studies (e.g., Bolte et al., 2006; Frommann et al., 2003; Penn & Combs, 2000; Wolwer et al., 2005) is their use of photograph-based stimuli and assessment measures pertaining exclusively to facial affect. Given that emotional information derives from a wide range of sources (posture, gesture, voice, contextual factors, and facial expressions), this creates problems for generalization of learning to naturalistic social situations in which emotions present dynamically and by means of multimodal channels (or in the case of phone communication, by means of voice alone). Indeed, problems with generalization and mainte-

nance of gains after training with only photographs have been found in several studies that have measured these factors (Bolte et al., 2002; Penn & Combs, 2000), although not in others (Combs et al., 2006; McAlpine et al., 1992). A minority of the studies used other stimuli in treatment, such as voice and body language, or videos incorporating these cue modalities (e.g., McKenzie et al., 2000; Rydin-Orwin et al., 1999; Solomon et al., 2004; van der Gaag et al., 2002). Of these, only Rydin-Orwin and colleagues used multimodal stimuli and assessment measures, and none assessed durability of treatment gains or generalization to measures of broader social functioning. Clearly, an important goal of remediation is an improvement in actual function, so a model remediation program must address the multimodal nature of emotions and the transfer of treatment gains to everyday social settings.

A second possible weakness of these studies is their focus on recognition of emotional stimuli (primarily facial expressions) without attention to appropriate ways of responding to emotional communication. As noted by McAlpine and colleagues (1992), recognizing the emotional significance of stimuli does not necessarily lead to socially acceptable use of such information in social behavior. This suggests that emotion perception training should incorporate components aimed at improving self-regulation of social behavior in order for maximum benefit to be realized. Some effort in this direction has been made by Combs and colleagues (2007), who have developed a program for individuals with schizophrenia that encompasses both emotion perception training and training in using emotional information effectively within social interactions. Preliminary findings have been promising and included evidence of generalization of treatment gains to social functioning. In TBI, this area remains to be explored.

Despite the similarities among these disorders, it is important to note that individuals who have sustained a TBI in adulthood may differ in important respects from individuals with developmental disorders, who are likely to have had longstanding emotion processing deficits, in many cases, from early childhood. For adults with TBI sustained after normal development, some aspects of emotion perception may remain intact and semantic knowledge of emotions (social cognition) tends to be unaffected (Croker & McDonald, 2005). These relative strengths in ability, therefore, offer avenues that could be used to support training aimed at areas of deficit. For example, training aimed at improving facial expression, voice tone, and body language recognition may draw rich references from individuals' semantic awareness of emotions and past emotional experiences.

NEUROPSYCHOLOGICAL BASES OF EMOTION PERCEPTION DEFICITS IN TBI

Recent research on the mechanisms of emotion perception provides the opportunity for a more sophisticated conceptualization of emotion perception training and thus offers

insights into how emotion perception remediation might be designed so as to maximize effectiveness. Specifically, current work into the neural underpinnings of emotion perception, combined with recent theorizing regarding the basis of remediation can help configure remediation approaches and treatment goals to effectively address emotion perception disorders following TBI within broader rehabilitation goals.

From the outset, it needs to be emphasized that the neuropsychological profile from which emotion processing deficits appear in TBI is a complicated one, due largely to the multifaceted nature of the injury (Tate, 1987). There are few studies that have specifically examined neuropathological correlates of emotion processing disorders in TBI. On the other hand, a body of neuroimaging and lesion studies in animals and humans has accrued, which provides a general picture of the neural architecture of emotion processing. Several interacting subsystems are implicated primarily situated in the frontal and temporal regions. These systems involve the amygdala, anterior cingulate gyrus, insular, parietal and somatosensory cortices, and numerous structures within the frontal lobes. The roles of these separate structures appear to be complex and suggest interactions between interrelated structures and the temporal features of the perceptual processes as well as concomitant cognitive demands. The interested reader should refer to authoritative overviews on this topic (e.g., Adolphs, 2002b; Adolphs & Damasio, 2000; Phillips et al., 2003). To summarize the reported relationships: the amygdala has been associated with negative facial expressions and emotional prosody (Adolphs & Tranel, 2003, 2004; Adolphs et al., 1995, 2001b; Cardinal et al., 2002; Critchley et al., 2000b; Morris et al., 1996, 2001; Phillips et al., 2003; Scott et al., 1997; Stork & Pape, 2002; Yang et al., 2002); the anterior cingulate cortex with facial and prosodic expressions (Holland & Sonderman, 1974; Hornak et al., 2004; Streit et al., 1999); the anterior insula with both facial and prosodic expressions of disgust (Calder et al., 2000, 2001, 2003; Phillips et al., 2003); the right somatosensory cortex (Adolphs et al., 1996, 2000) and the temporal visual cortex with facial expressions (Critchley et al., 2000b; Haxby et al., 2002; Streit et al., 1999; Weddell, 1994); the middle temporal gyrus with bimodal emotional expressions (Pourtois et al., 2005); the right frontoparietal operculum, bilateral frontal pole, and middle right superior temporal sulcus with emotional prosody (Adolphs, 2002b; Grandjean et al., 2005; Pell, 2006); and the orbitofrontal and medial prefrontal cortices, which together comprise the ventromedial prefrontal region, with facial and prosodic expressions (Barrash et al., 2000; Blair et al., 1999; Cicerone & Tanenbaum, 1997; Damasio, 1994; George et al., 1996; Hornak et al., 1996, 2003; Morris et al., 1999; Phillips et al., 2003).

Extensive evidence has indicated that processing of emotional stimuli may be predominantly lateralized to the right hemisphere. This may especially be the case with processing of emotional prosody (Adolphs et al., 2002; Adolphs & Tranel, 1999; George et al., 1996; Grimshaw et al., 2003;

Heilman et al., 1984; Mitchell et al., 2003; Pell, 2006; Pell & Baum, 1997; Pihan et al., 2000), although some bilateral contribution has been found in a few studies (Adolphs et al., 2002; Morris et al., 1999; Sander & Scheich, 2001). The issue of lateralization appears to be more complicated for recognition of facial expressions. Some reports are indicative of right hemispheric dominance in the processing of all emotions (Adolphs & Damasio, 2000; Borod, 1993; Borod et al., 1998; Rapcsak et al., 1993), and others suggest hemispheric asymmetry in processing positive and negative emotions (Adolphs & Damasio, 2000; Adolphs et al., 2001a,b; Silberman & Weingartner, 1986). In the case of the latter, that is, the “valence hypothesis,” evidence has been seen to favor either clear hemispheric biases toward processing positive (left) and negative (right) emotions (Silberman & Weingartner, 1986) or the possibility that positive emotions are processed bilaterally, whereas negative emotions are processed chiefly by the right hemisphere (Adolphs et al., 2001a). Still other data have suggested that emotion processing performed by the left hemisphere is modality specific, whereas the right hemisphere appears to predominate in processing all emotional expressions across modalities (Kucharska-Pietura et al., 2003). Overall, although findings remain inconclusive (Critchley et al., 2000a), most evidence appears at least suggestive of right hemispheric primacy in emotion processing (Adolphs et al., 1996, 2002; Erhan et al., 1998).

The neural structures associated with emotion processing, whether lateralized or distributed bilaterally, may be particularly vulnerable to damage in TBI due to their anatomical location in the frontal and temporal lobes (Fontaine et al., 1999). Most of these structures are situated near the orbital surfaces of the brain, which are directly adjacent to common points of impact and numerous bony protuberances lining the interior of the skull. Rapid jolting of the brain within the skull cavity due to sudden impact, for example, in a motor-vehicle accident, the most common cause of severe TBI (Kahn, 1970; Kalsbeek, 1980; Tate et al., 1998), can thus lead to multifocal lesions concentrated in these sites as well as shearing of axonal connections with other systems (Adams et al., 2001; Besenski et al., 1996; Gaetz, 2004). The high likelihood of this kind of damage in the majority of severe TBI cases is consistent with the now established finding that emotion perception difficulties are a common feature of this type of injury.

Numerous neuroanatomical models of emotion perception have been proposed (for other examples, see: Adolphs, 2001, 2002a,b; Adolphs et al., 2002; George et al., 1993; Heberlein & Adolphs, 2005). Perhaps the most comprehensive of these to date is that of Phillips and colleagues (2003), whose model (summarized in Figure 1) incorporates processes for both the rapid appreciation of emotionally significant stimuli and processes for automatic and controlled responding to emotional input. Reviewing evidence from a range of human and animal research, these authors identify several neuroanatomical structures that potentially underpin key processes involved in emotion

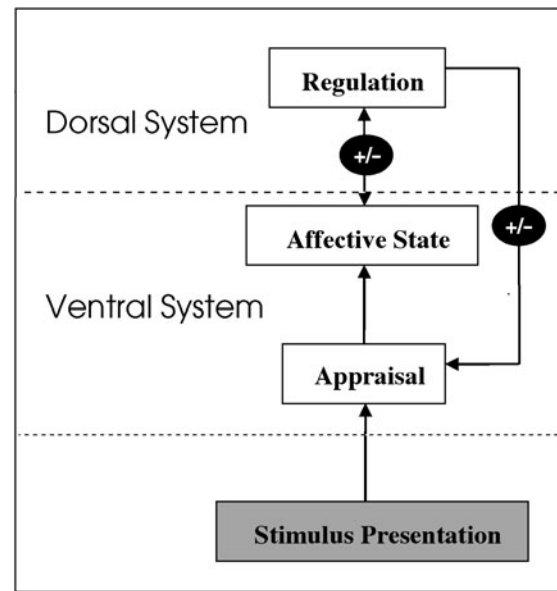


Fig. 1. Model of the neuroanatomical correlates of three key processes in emotion perception (adapted from Phillips et al., 2003). Anatomical structures within the ventral system include the amygdala, insula, and ventral regions of both the prefrontal cortex and the anterior cingulate gyrus. This system is thought to mediate both the production of emotional states and the identification of emotion-related stimuli. The dorsal system includes the dorsal regions of the prefrontal cortex, anterior cingulate gyrus, and the hippocampus. This system mediates the regulation of emotional states and behavior and can, as indicated by the negative and positive signs in the circles, modulate or inhibit the activity of the ventral system so that emotional states and behaviors are contextually appropriate.

perception. With the exception of the sensorimotor region, which Phillips and colleagues do not mention, these structures are the same as those described earlier. Two neighboring and closely linked neural systems are indicated: a ventral system, including the amygdala, insula, and ventral regions of the anterior cingulate gyrus and prefrontal cortex, and a dorsal system, including the dorsal regions of the prefrontal cortex and anterior cingulate gyrus as well as the hippocampus. According to the model, the ventral system supports the rapid orientation to, appraisal, and identification of emotionally significant information as well as the production of affective responses even before consciousness, while the dorsal system is responsible for effortful processing of emotional stimuli, including the engagement of other cognitive processing such as language and memory, as well as the regulation of emotional behavior. These systems, although neuroanatomically separate, are not functionally independent, such that damage to either is likely to disturb emotion perception generally. Thus, the processes underpinned by the ventral system initiate activity in the dorsal system, and, conversely, the dorsal system can also inhibit or modulate ventral activity to facilitate the rapid processing of critical emotional information (e.g., threat) and to ensure that behavioral responses to what is perceived remain contextually appropriate.

A strength of this model is its configuration of the interrelationship between conceptually different processes governing emotion perception. The model suggests that interventions designed to treat emotion perception deficits may address several component processes of emotion: initial orientation and appraisal, affective responsivity, and strategic, cognitively mediated processing. Furthermore, as the system is highly interactive, improvements in one aspect of emotional processing may lead to improvements in others. One example of how this interaction may occur arises from a study of patients with focal frontal lesions reported by Damasio and colleagues (1990). These patients failed to show skin conductance changes (arousal) when passively viewing emotionally charged material but demonstrated a normal response when they were required to describe the pictures. These findings suggest that the dorsal (effortful) system can stimulate the ventral to overcome an initial lack of orientation and responsivity to the emotional material. Although similar dissociations have not yet been reported in people with TBI, it is highly likely that some will experience relatively greater impairment in one process involved in emotion perception compared with another. According to this interactive model, such individuals may derive benefit from treatment targeting both systems.

Before leaving this discussion of the neuropsychology of emotion perception deficits in TBI, it is important to acknowledge that impairment of “nonsocial cognitive processes” (a term used by Adolphs and others in research on social cognition—Adolphs, 1999, 2003, 2006; Brüne et al., 2007; Decety & Jackson, 2004; Satpute & Lieberman, 2006), such as attention, learning, information processing speed, cognitive flexibility, and awareness of deficits, are likely to contribute to the failure of those with TBI to perceive emotional cues accurately and efficiently (Lezak, 1995; McDonald, 2003; McDonald et al., 2006; Prigatano, 1999; Sohlberg & Mateer, 2001; Trower, 1980). There is some evidence for an association between executive function and emotion perception in TBI (Bornhofen & McDonald, *in press*; McDonald et al., 2006; McDonald & Saunders, 2005). Other types of dysfunction, such as slowed information processing and variable attention may compromise an individual’s ability to track the continuous flow of data presented in social interactions. The impact of these factors may account for some of the variability seen in emotion perception competence among individuals with TBI noted earlier; however, no research to date has investigated whether remediating attention and/or executive deficits improves emotion perception ability in individuals with TBI. In the meantime, it appears clear that any intervention that is tailored to addressing emotion perception deficits in people with TBI must address these nonsocial deficits also either directly or in terms of modifying techniques to maximize the likelihood of success on remediation tasks.

A MODEL OF COGNITIVE REMEDIATION

Having identified major cognitive and affective processes that contribute to emotion perception, the next task is to consider whether contemporary models of remediation are relevant to emotion perception and/or whether there is sufficient evidence to consider them a credible basis for the development of treatment strategies in TBI. Before the 1990s, the prevailing view in neuroscience was that neural structures in the brain were fixed at maturity, and thus damage to neural systems after early childhood was regarded as permanent (Sohlberg & Mateer, 2001). Over the past decade, however, research into mechanisms of functional recovery after brain injury has produced evidence to suggest that the potential for neuronal reorganization and regeneration following injury is much greater than previously believed (Cornelissen et al., 2003; Hallett, 2001; Keyvani & Schallert, 2002; Mohammed et al., 2002).

Largely the product of research into motor and sensory recovery after deafferentation of limbs in animal models and stroke in humans, this evidence has indicated that the brain not only remains plastic throughout the lifespan, but it is, in fact, constantly changing as a result of experience (Merzenich & Jenkins, 1993; Merzenich et al., 1984). Functional (motor) recovery can be achieved by intensive, carefully targeted training and is associated with reorganization and expansion of relevant cortical areas (Liepert et al., 2000; Nudo & Milliken, 1996; Nudo et al., 1996a,b). The critical therapeutic factor in such remediation appears to be a combination of attention and carefully targeted, graduated practice (Taub et al., 1998; Taub & Wolf, 1997). Beyond applications to motor recovery, this kind of approach has been used with cognitive-perceptual impairments, including aphasia (Cornelissen et al., 2003; Fridriksson et al., 2006; Pulvermuller et al., 2001), cortical blindness (Pleger et al., 2003), sustained attention (Longoni et al., 2000), unilateral neglect (Pizzamiglio et al., 1998), and dyslexia in children (Temple et al., 2003). In each case, improvement in function has been associated with use-dependent expansion of cortical areas subserving those processes (Cornelissen et al., 2003; Fridriksson et al., 2006; Longoni et al., 2000; Pleger et al., 2003; Temple et al., 2003).

The neurological underpinnings of these functional gains have been conceptualized in terms of a Hebbian learning framework (Keyvani & Schallert, 2002; Mateer & Kerns, 2000; Robertson & Murre, 1999), whereby the strength and density of synaptic connections within a functional circuit change significantly through repeated, synchronous activation of pre- and postsynaptic neurons. At the neural level, “learning” occurs as synaptic connections are forged (i.e., through dendritic and axonal branching) and strengthened by experience, according to the Hebbian principle that “cells that fire together, wire together.” In practical terms, this suggests that repeated practice of a functional activity is likely to strengthen the neural correlates of that activity, whereas disuse or loss of stimulation may eventually lead to loss of

function (Fitzsimonds & Poo, 1998; Kayser & Miller, 2002; Kolb, 1999; Nelson et al., 2003; Rosenzweig, 1999).

Building on Hebb's central principle of activity-dependent synaptic activity, Robertson and Murre (1999) have argued that a neural network which has been partially damaged by a lesion should be amenable to reconnection if the process subserved by the network is repeatedly activated through precisely targeted experience enabling connectivity to be reinstated. According to Robertson and Murre's proposed model, it is critical that the target process, in this case, emotion recognition, is precisely and repeatedly activated by means of either bottom-up (i.e., stimulus-bound) or top-down (attention-enhancing) techniques (Robertson & Murre, 1999). Precise targeting is required, as evidence suggests that an impaired system may be dominated, masked or suppressed by the activity of other systems which remain intact (Kapur, 1996; Robertson & Murre, 1999; Vuilleumier et al., 1996). Care must be taken to minimize or eliminate stimulation that is inaccurately or too-broadly defined, because attention directed to such stimuli may foster maladaptive or faulty connections within the impaired system.

Robertson and Murre's model seems well matched to the task of emotion perception remediation for two related reasons. First, given the relatively localized systems in the frontotemporal and somatosensory areas that appear to underpin emotion processing, it seems plausible that a Hebbian mechanism could support the restoration of emotion perception if activation during remediation was precisely targeted. Second, the bottom-up and top-down techniques described by Robertson and Murre fit well with the model of ventral and dorsal affective processes outlined by Phillips and colleagues (2003) and may furnish the means by which both types of processes could be targeted. Specifically, bottom-up techniques which use external cues could potentially activate processes mediated by the ventral system. Examples might include repetitive practice of orientation to important emotion cues (e.g., the eyes and mouth in facial expressions) and stimulus discrimination. Top-down strategies may activate the dorsal system by reinforcing the use of sustained and selective attention and by supporting self-regulation of behavior. Examples of these strategies include cued rehearsal, mental rehearsal and self-monitoring procedures such as self-cueing with structured questions. Both approaches are eminently suited to the direct retraining of emotion perception, as the stimuli can be specific and easily exaggerated, and discriminative features easily highlighted and described. Thus, abundant opportunities for graduated practice of the process/es can be provided while minimizing maladaptive influences on gains achieved (Robertson & Murre, 1999). In summary, a program should entail both training in orienting to basic features of emotional cues and repetition (bottom-up strategies), especially in the early stages of the program, plus training in self-regulation of attention and responses to emotional stimuli (top-down strategies), especially toward the latter half of the program once basic ability to discriminate between key features has

been established. Use of both approaches is likely to maximize benefit across all components of emotion perception in a way that supports independence of the trainee and thereby increases the likelihood that improvements will be sustained.

According to Robertson and Murre's position, there may also be instances in which damage to the emotion processing system is so severe as to make restoration of function impossible. In such cases remediation targeting impairment would not likely be successful, and compensatory training aimed at minimizing the impact of deficits would be more appropriate. Such training could, for example, engage support persons to provide structured verbal cues to assist in appropriate responding within social contexts.

CONTEXTUALIZED APPROACHES

While such a theoretical account of remediation as applied to emotion perception deficits seems plausible, whether or not it is suitable for people with TBI remains questionable. Robertson and Murre's (1999) model of rehabilitation, based largely on findings from cognitive neuroscience derives primarily from research on focal injury. As such, it has been heavily criticized as a model for guiding treatment in brain injury cases whose profiles reflect more widespread, multifocal damage. Wilson (2005) argued that a broader-based approach to rehabilitation was necessary for the provision of effective support and treatment of rehabilitation recipients, many of whom have multifaceted needs. According to this position, decisions as to whether restitution- or compensation-oriented rehabilitation is more appropriate for any one individual cannot reliably or practically be done on the basis of the extent of system damage, as imaging findings may often be unavailable or inconclusive and behavioral observations before therapy unreliable for this purpose. This may be particularly true for emotion recognition deficits. According to the interactive model proposed by Phillips et al. and others, superficially similar deficits in emotion recognition may reflect damage to numerous systems. Thus, with the current state of knowledge, it would be difficult to assess the extent of system damage on the basis of behavioral observation or imaging results. Such a situation may not actually represent a huge obstacle, as a range of remediation techniques could potentially be used to target several key emotion processes (e.g., appraisal, arousal/responsivity, and regulation of emotional behavior) simultaneously, which may provide convergent activation. Notwithstanding, Wilson has argued that in the vast majority of rehabilitation settings, decisions concerning which approach to use are best made on the basis of individual response to restitutional strategies. She concluded that rehabilitation should be guided by multiple models, including models of emotion, behavior, learning, and neuroscience, as no single model or theory now available is able to address the full range of needs experienced by rehabilitation recipients and their families within the environments in which they live.

Another important factor to consider is the need for remediation to be contextualized. As emphasized by Wilson (2005) and others (Prigatano, 1999; Sohlberg & Mateer, 2001; Turkstra, 2001; Wilson et al., 2002; Ylvisaker et al., 2003), rehabilitation following TBI takes place amidst an array of social, institutional, medical, and personality factors that must be taken into consideration for a program to be successful in providing both short-term and long-term benefits for any one individual. Just as a major shortcoming of emotion remediation research in schizophrenia has been its failure to address emotion deficits in context, so too the diverse interplay of these factors has often been overlooked in TBI remediation in the past, to the detriment of the long-term outcome for individuals and their families (Turkstra, 2001; Wilson, 2002, 2005; Ylvisaker & Feeney, 1998, 2000; Ylvisaker et al., 2002). A context-sensitive approach to rehabilitation, in contrast, makes clear and explicit the relevance of a rehabilitation program to the individual's personal goals, and aims to highlight the personal significance of gains achieved throughout the course of treatment (Wilson et al., 2002; Ylvisaker & Feeney, 1998). It, therefore, serves to enhance motivation as well as facilitate transfer of newly learned skills to the treatment recipient's day to day environment. In addition, directly linking remediation goals to the individual's context may capitalize on procedural/implicit learning processes, which tend to be context-specific, and remain relatively more intact following TBI than explicit learning mechanisms (Ylvisaker & Feeney, 1998).

In the realm of emotion perception remediation, there is good reason to believe that a contextualized approach could be compatible with focused attention techniques as advocated by Robertson and Murre (1999). This is so because the inherent social nature of emotion processing, which readily supports repeated practice of emotional perception skills across a variety of social interactions (i.e., therapeutic, domestic, commercial, etc.), provides the clinician with abundant opportunities to demonstrate relevance. For example, education regarding the fundamental role of emotion perception in social life, discussion of real-life examples in which an individual may have misperceived another's intentions or mood, and role play of inappropriate *versus* appropriate responding to emotional cues may all be used to engender greater awareness of the function of emotion perception skills. Once a working understanding of the importance of these skills has been established, this understanding may be further enhanced in the course of a specifically targeted training program. Role-plays within session and practice tasks for completion outside of sessions (ideally with caregivers or primary support figures) can repeatedly highlight and reinforce the significance of emotional cues in any interaction with others (e.g., family, friends, or members of the community). All aspects of treatment, including emotion stimuli and therapy activities, can be made relevant to the individual's typical social exchanges and aimed at strengthening skills that can directly be applied to improve functioning in the everyday environment.

Finally, the inclusion of a full range of emotion stimuli within training, spanning visual, auditory and audiovisual modalities, rather than only still photographs, is likely to support holistic encoding of naturalistic emotion displays. Using static and dynamic stimuli as well as role play interaction activities could provide rich opportunities for developing skills necessary for efficient and accurate interpretation of emotion cues in a social context. Extending practice of these skills by means of homework tasks to be completed in the individual's home and community environment will further support transfer of learning to these settings. If these considerations were met, it could be anticipated that the overall benefits of treatment would include increased maintenance and generalization of newly acquired skills to the individual's day-to-day setting, particularly when supported by liaison with family and other support persons. Thus, it is likely that a contextualized approach to emotion perception training would complement an approach based on the neuroscientific model of rehabilitation described by Robertson and Murre (1999). Used in combination, they may broaden the benefits to individuals with TBI which could otherwise be gained from restitution strategies alone.

Unfortunately, little research investigating emotion perception training in people with TBI has thus far been carried out. The authors have published two small studies (Bornhofen & McDonald, 2008a, 2008b) evaluating remediation programs that encompass both the notion of directed attention and repeated activation as well as the use of contextually relevant activities. Results indicated gains in both cases, but highlighted the complex nature of emotion perception disorders in people with chronic TBI. As foreshadowed by Wilson's work, deficits in emotion perception sat amidst highly variable constellations of other cognitive deficits and circumstantial issues that both limited and defined the rehabilitation process. The fact that improvements were found is encouraging, and provides an impetus for further research in this emerging field. But it is clear, that there is a long way to go in understanding the nature of these critical deficits and the best approach to their amelioration.

CONCLUSION

In this review we have laid out the evidence to date regarding the neuropsychological mechanisms of emotion perception disorders in TBI and recent theoretical discussions concerning the potential basis for remediation. The fact that contemporary remediation theory is highly relevant to emotion perception suggests that specific treatment approaches for emotion perception disorders with the TBI population are feasible. This combined with the positive findings of treatment efficacy seen in other clinical populations and the amenability of emotion perception training to both targeted practice and a contextualized approach gives scope for optimism with regard to treatment gains in the TBI group. But there is much yet to be learned regarding the optimal approach for individuals with unique con-

stellations of deficits and how techniques can be designed to ensure maximal generalization and actual social benefit. Given the central importance of emotional communication in our daily lives, this area offers potentially unique benefits for those treated and their families and a compelling area for future research.

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