

Broad-Perspective Perceptual Disorder of the Right Hemisphere

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Traditional accounts of right-posterior brain injury describe a syndrome of low-level perceptual sequelae producing marked acute dependency and transient safety concerns. The syndrome is also held to spare cognition and to carry a generally favorable long-term prognosis. The present paper reviews publications and anecdotal data that challenge this picture. Recent theoretical expositions and empirical studies stipulate three major cognitive functions of the right posterior association cortex: processing novel input, guiding reactions to emergencies, and anticipating consequences. Appearing benign after acute recovery, the impairment of these processes produces vocational, social and marital dysfunctions that increase as a function of chronicity, ultimately becoming more broadly disabling than focal injuries in other cortical loci. The unique symptom picture and serious implications suggest that the long-term syndrome should be labeled (Broad-Perspective Perceptual Disorder) and incorporated in future clinical taxonomies, underscoring the need for extraordinary long-term assistance and specialized therapeutics. Procedures for assessment and differential diagnosis are outlined.

KEY WORDS: brain injury prognosis; right hemisphere; right parietal syndrome; posterior association cortex; hemispheric specialization; anosagnosia; aprosodia; nonverbal learning disability.

The right posterior association cortex provides specialized, high-level perceptual capabilities that are essential for effective complex cognition and adaptation. Jackson (1876) pronounced this region to be of paramount importance, but his conception was rejected (Jackson, 1893; Joannette and Goulet, 1990; Roth, 1999) in favor of the prevailing view of a “minor hemisphere” that made no cognitive contributions (Kolb and Whishaw, 2003). Recent papers describe a right hemisphere with a number of specific, important cognitive functions (Mesulam, 2000a), but these discoveries and the implications for medical care and rehabilitation treatment have not been incorporated into most clinical texts (e.g., Adams et al., 1997; Bannister, 1992; Lezak, 1995; Reitan and Wolfson, 1985a; Rowland, 1995; Sohlberg and Mateer, 2001; Zillmer and Spiers, 2001).

A brain deprived by injury of its nonverbal-perceptual guidance sees what it expected to see before injury, ignoring emergent obstacles and hazards and underestimating the urgency of those that make themselves obvious. Planning and problem-solving are crippled by naively optimistic forecasting of outcomes. Social intercourse is conducted with a blanket disregard for the feelings, wishes, needs and expectations of others. The resulting disabilities have an insidious onset because reliance on the pre-injury perceptual schemata becomes progressively less functional as situations evolve over time. Hence spouses, children, friends and employers, satisfied with early recovery, become increasingly alienated by a track record of poor decisions and bad conduct over the ensuing years. Extended follow up finds unusually high levels of divorce, unemployment, and social isolation. This pattern of long-term deficit may be sufficiently distinctive and pronounced to merit inclusion in the clinical taxonomy. The label of Broad-Perspective Perceptual Disorder (BPPD) is proposed. Three critical cognitive symptoms and their cumulative effects on adaptation will be described, explained and illustrated,

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followed by a discussion of neuropsychological assessment strategies.

TRADITIONAL DESCRIPTIONS OF “RIGHT PARIETAL SYNDROME”

Jackson (1864) introduced the concept of right-posterior cortical dominance for visuospatial perception. Lateralized circuitry for spatial coordinate mapping (Kosslyn and Koenig, 1992; Lynch, 1980) and a unique, “wide-screen” view of the world (Deouell et al., 2003; Kaplan, 1989; Milberg et al., 1996; Posner and Raichle, 1994) augment somatosensory, topographical, and spatial localization and visual search (Damasio et al., 2000; Farah, 2003; Vallar, 1999). The lateralized aptitude for complex pattern recognition (Walsh and Darby, 1999; Zaidel, 1985) supports object recognition, and configural and gestalt perception. The “right parietal syndrome” produced by focal damage (Meier et al., 1987; Mesulam, 2000a) impairs these perceptual skills (Benton and Tranel, 1993) and causes dense contralateral neglect (Heilman et al., 2003a,b; Mesulam, 1985b; Rafal, 1997a). Associated practical problems include getting lost easily (Butters et al., 1970), falling (Diller and Weinberg, 1970), crashing vehicles into obstacles (Joseph, 1997; Webster et al., 1995), and handling objects clumsily (Hecaen and Albert, 1978). The syndrome also impairs perception of social-emotional signals (Code, 1987; Heilman et al., 2003b; Ross, 2000; Segalowitz, 1983), a homologue of receptive aphasia (Ross, 1985) impacting interpretation of implicit messages and speaker’s intent (Povinelli, 2001; Sabbagh, 1999) and mental state (Adolphs, 2003; Bowers et al., 1993; Ornstein, 1997; Winner et al., 1998). Finally, patients fail to recognize their own impairments, a symptom termed anosagnosia (Adair et al., 2003).

Because the syndrome is traditionally conceptualized only in terms of basic perceptual deficits, there is no warning of the long-term compromise of higher cognition and macroscopic-level adaptive behavior resulting from the loss of perceptual guidance. Moreover, most post-acute patients present well and sound bright and expressively fluent, creating an impression of cognitive intactness that misleads even expert evaluators (Critchley, 1953; Hebb, 1949). Hence the current standard of care is to release these patients from restrictions and terminate their treatment upon recovery of basic perception (Joseph, 1997), usually achieved within six months (Cassidy et al., 1998; Nelson et al., 1994; Sisson, 1998; Stone et al., 1992; Tham et al., 2000).

CRITICAL COGNITIVE-BEHAVIORAL SYMPTOMS OF BPPD

Higher cognition relies extensively on the afferent pattern-comparator (Calvin, 1989; Kosslyn and Koenig, 1992; Neisser, 1967), which evolved for object recognition (Hofstadter, 2001) but is “borrowed” for advanced mental skills such as problem solving and induction (Carlson, 1997; Gattis, 2001; Jerison, 2001; Pinker, 1997). The analyzer is also used to search the posterior cortex’s gnostic library quickly and exhaustively to determine the potential adaptive impact of emergent stimuli and situations (Gibson, 1979; Luria, 1973), and to compare plans with anticipated outcomes (Gross, 1982) in order to narrow the choice of goals and plans to those that are most practical (Miller et al., 1960; Schlesinger, 1962). The process requires a coordinated interplay of the executive and afferent analyzers (Gross and Schutz, 1986), executed through the frontoparietal distributed network (Mesulam, 2000a). (Extended explanation of the functions of this network is beyond the scope of this paper, and is deferred to a future discussion.) The afferent data are accessed by the network through the parietal integrating area (Geschwind, 1965; Goldberg and Costa, 1981). Damage to the posterior end of the network thus disconnects the executive from the guidance system, and consequently impacts three major network functions.

The Inability to Assimilate Novelty

The right brain possesses special capabilities for processing novel stimuli (Goldberg and Costa, 1981; Pribram, 1981; Regard and Landis, 1989; Rogers, 2000). There is full access to input from the peripheral (Cook and Beech, 1990) and distal (Hellige, 1993; Mesulam, 1985a) spatial fields, the most common entry points for new stimuli, and only the right brain can redirect attention to new peripheral targets in both hemifields (Posner and Raichle, 1994). It also excels in perceiving fleeting and poorly illuminated stimuli (Gazzaniga et al., 1998). A novel stimulus triggers immediate release of norepinephrine mainly in the right hemisphere (Edelman and Tononi, 2000; Goldberg, 2001; Mesulam, 2000a), and the noradrenergic neurons resist habituation, holding the attentional gates wide open to take in a generous exploratory data base (Tucker and Williamson, 1984). The input is then explored by induction, which utilizes pattern matching (Edelman and Tononi, 2000; Gentner et al., 2001) to define the boundaries (Barlow, 1985) and properties (Gattis, 2001; Hofstadter, 2001; Thagard, 2000) of the newly-discovered phenomena. This pattern matching is a right-hemisphere

function (Gregory, 1970; Smith, 1989), as demonstrated by EEG (Molle et al., 2000) and functional MRI (Goebel et al., 1997; Shuren and Grafman, 2002), and verified by lesion studies (Bradshaw and Mattingly, 1995; Walker et al., 2001).

This “furious pattern matching” (Reason, 1990) requires great data-integrating power. The right brain’s integrative assets include more white matter and a larger posterior association cortex (Kolb and Whishaw, 2003), denser association fibers (Goldberg and Costa, 1981), greater interconnection of neuronal columns (identified in the temporal lobe) (Gazzaniga et al., 1998), and larger integrative structures (Galaburda, 1995; Hochberg and LeMay, 1975) in the perisylvian area (Cummings, 1985; Gorelick and Swiontoniowski, 1989), the posterior–inferior parietal lobe (Dimond et al., 1980; Jancke et al., 1994; Kolb and Whishaw, 2003), and particularly intraparietal area PEG (Edelberg and Galaburda, 1984). Right-sided structures that detail the properties of people and things (Farah, 2003; Ungerleider and Mishkin, 1982), including Heschl’s gyrus and the superior temporal gyrus, are also larger (Campain and Minckler, 1976; LeMay, 1976; Whitaker and Osemann, 1977). Neuronal organization is mirrored by the circulatory system: The main venous drainage on the left (the vein of Labbe) lies in the temporal language area, while the main right-sided drainage (vein of Trolard) is offset into the parietal integrating area (Witelson, 1980). Superior integration is verified by stronger intrahemispheric intercorrelations of EEG tracings (Liotti and Tucker, 1995) and the greater overlap of cortical projection zones for somatosensory (Semmes et al., 1960; Teuber et al., 1960) and auditory inputs (Milner, 1975) on the right side, improving heteromodal processing (Semmes, 1968) and recognition of broad patterns (Beeman et al., 2000; Broadbent, 1977; Christman, 1997; Ivry and Robertson, 1998; Sergent, 1982; Valenstein, 1999) such as main themes (Gardner, 1975; Hellige, 1993), complex arrays (Hellige, 1995; Young and Ratliff, 1983; Zaidel, 1985), and perceptual gestalten (Nebes, 1978; Ratey, 2002). Broad patterns are the starting point in exploring novelty (Krech and Calvin, 1953; Navon, 1977; Neisser, 1967). The lesions of BPPD maximally disrupt this global processing (Doyon and Milner, 1991; Heinze and Munte, 1993; Rafal, 1997b; Robertson et al., 1988).

The right brain’s powerful integrative processing station can perform complex multivariate data analyses (Goldberg and Costa, 1981) in parallel (Bradshaw and Nettleton, 1981; Cohen, 1973). Right-brain problem solving generates a matrix of alternative solutions, as contrasted with the left brain’s single solution of best fit (Brownell, 1988; Tucker and Williamson, 1984). This an-

swer matrix remains active while alternative solutions are explored (Richards and Chiarello, 1997), a method suitable for the open-ended possibilities inherent in a novel situation.

The learning of new skills takes place in the heteromodal association cortex (Prigatano, 1991), where the right brain starts the process by building a general conceptual framework, subsequently using it to choreograph the left brain’s acquisition of fine discriminations (Kinsbourne, 1982). Novices learning music, Morse code, geometry, or interior decorating show more arousal on the right side (Goldberg and Costa, 1981). The differential activation shifts to the left side after extensive practice (Goldberg, 2001), even for a “spatial” skill like tap dancing (Falk, 1992) or playing a musical instrument (Bever and Chiarello, 1974). In a similar vein, novel tasks are executed better in left hemispace (Gardner, 1975; Sperry, 1985). Right-sided lesions differentially impair early language acquisition (Goldberg, 2001), recall of novel terms (Mills et al., 1993; Thal et al., 1991), stories (Segalowitz, 1983), maze routes (Rausch, 1985), semantic associations (Seeger et al., 2000), symbols (Koul and Lloyd, 1998; Zaidel, 1985), faces (Damasio et al., 2000), and gestures (Goldenberg and Strauss, 2002), and identification of unusual uses for common objects (Diggs and Basili, 1987), pictorial incongruities (Milner, 1975), and unexpected (Wapner et al., 1981) or bizarre material that was inserted into stories (Gardner et al., 1983).

Many developmental years are required to learn by induction the massive catalogue of social rules and rituals (Goffman, 1967), the gnostic base of the social skills (Schore, 1994). “Right-hemisphere deficit disorder,” a developmental syndrome including all of the major features of BPPD (Rourke, 1989), is associated with massive knowledge gaps concerning approved and forbidden forms of conduct (Ratey, 2002). Although bright and verbally fluent, these children seem unable to learn lessons about age-specific expectations, rules and routines from observation or experience, repeating immature responses and other sanctioned behaviors again and again. Their actions appear unacceptably “weird” to peers, leading to rejection and isolation (Casey and Strang, 1994; Rourke, 1989; Rourke and Fisk, 1988; Weintraub and Mesulam, 1983; Worling et al., 1999). The most disabled tend to have posterior lesions (Myklebust, 1975; Voeller, 1986).

Impaired gnosis from damage to the posterior association cortex prevents updating existing perceptual schemata as well as creating new ones (Luria, 1973). For example, right-hemisphere lesions focally impair learning the topography of new settings (Cogan, 1966). Patients with right-parietal lesions take longer to learn new routes (Barrash, 1998), follow maps less effectively, get lost more

often (Critchley, 1953; Cummings, 1985; Damasio, 1985), and forget where things were put (Butters et al., 1970). They take long walks searching for the car in a parking lot. One outpatient insisted on trying to leave the clinic through a back-corridor bifold closet door. Another complained that staff had moved the elevator. A CVA patient with a master's degree needed 3 hr to shop because she could not learn the scheme of the market.

Right CVA patients show no updating of their schemata for self-guidance after receiving performance feedback (Gordon, 1987; Kihlstrom and Tobias, 1991; Mizuno, 1991; Ramachandran, 1995; Rausch, 1985). Social schemata for familiar persons become closed to new information after right-posterior damage (Damasio, 1994), while schemata for new persons are impoverished and superficial (Segalowitz, 1983; Sergent, 1995; Wapner et al., 1981). Empathy seems to depend upon the functioning of the right parietal integrating and right somatosensory areas (Adolphs, 2003; Adolphs and Damasio, 2000), and may be markedly limited after right posterior lesions (Heilman, 1999a). BPPD patients often ignore tragedies that befall their spouses and associates, for example, health or business crises or the death of a parent. They also ignore changes in social partners' feelings toward them, particularly problematical when the others have become alienated by a long-standing pattern of inconsiderate and unreliable behavior.

The most extensive revision of existing schemata required after brain injury incorporates the "harsh reality" (Prigatano, 1987) of new limitations and flaws into the self-concept (Haney, 1986; Kelly, 1955; Prigatano, 1999). Deficit awareness is a generic problem in acquired brain injury (Fisher, 1989; Weinstein and Kahn, 1955): Damage to any functional system inhibits feedback about the system's defects (Bisiach and Geminiani, 1991; Prigatano and Schachter, 1991). However, focal damage to the association cortex, for example, in the prefrontal region, interferes with deficit awareness more extensively (Damasio and Anderson, 2003; Goldberg, 2001; Stuss, 1991). The most dramatic and enduring symptoms of anosagnosia are seen for lesions of the right hemisphere (Heilman, 1991; Prigatano, 1994), especially in the posterior association cortex (Goldberg and Barr, 1991; Roth, 1949; Sohlberg and Mateer, 2001). This is the primary locus for recognition of behavioral errors (Barco et al., 1991; Bisiach and Geminiani, 1991), feelings of concern about accuracy of self-perception (Mesulam, 2000a,b), and the monitoring and revision of the self-image (Johnson et al., 2002; Maddock et al., 2001; Maguire et al., 2001). Right-sided damage impairs self-perception (Prigatano, 1987), "freezing" the self-concept at the time of onset (Damasio, 1994) and blocking recognition of changes even in personality

traits (Kihlstrom and Tobias, 1991; Luria, 1973; McGlynn and Schachter, 1989). In BPPD, the self-concept grows increasingly obsolete by because it is based on "old information growing older by the second" (Damasio, 1994). According to Crosson et al. (1989), this deficit is often permanent.

Postmorbidity imposes many novel demands on the brain-injury survivor (Haney, 1986; Prigatano, 1999), and patients with BPPD have unusual difficulty in adapting to them. The rehab clinic is a strange situation, less controlling and arbitrary than a medical facility (Howard, 1998; Plovnick, 1998) but more demanding than a commercial service business. Patients with BPPD break rules by falling asleep in sessions (Gordon, 1987), skipping assignments, being late or absent without good cause, or cheating on self-scored exercises. Patients are less successful in learning to perform familiar tasks in novel (compensatory) ways (Diller, 1962; Gardner, 1975; Joseph, 1997), and more insistent on using nonfunctional, familiar approaches (Damasio, 1994) after right-sided lesions from posterior tumor (O'dell et al., 1998), ischemic infarction (Maccocchi et al., 1988), ruptured aneurysm (Clinchot et al., 1997) and CVA (Alexander, 1994; Joseph, 1997; Scranton et al., 1970; Ween et al., 1996), even at 3–24-month follow-ups (Cherney et al., 2001; Chester and McLaren, 1989; Jehkonen et al., 2000; Katz et al., 1999; Kinsella et al., 1993; Robertson et al., 1997; Schutz, 2002; Stone et al., 1993). Moreover, in right CVA, the least successful accommodations are made in the presence of posterior association cortex lesions (Damasio, 1994; Gordon et al., 1978; Heilman, 1999b; Prigatano and Wong, 1999; Saecki et al., 1994) or nonverbal perceptual deficits (Jongbloed, 1986, reviewing 33 studies) such as somatosensory loss (Patel et al., 2000; Reding and Potes, 1988; Sterzi et al., 1993), left neglect (Cassidy et al., 2001; Denes et al., 1982; Fullerton et al., 1988; Hier et al., 1983; Katz et al., 1999; Pedersen et al., 1996; Ring et al., 1997), topographical disorientation (Kwakkel et al., 1996; Sze et al., 2000), and constructional apraxia (Suhr and Grace, 1999).

Pathological Indifference and the Breakdown of the Emergency Warning System

The right brain specializes in the perception of negatively valenced stimuli (Heilman, 1999b). Aversive stimuli such as unpleasant video images (Davidson, 1984, 1995; Gainotti, 1997), threats of punishment (Davidson, 1995; Tucker et al., 1977), and noxious smells (Ehrlichmann, 1987) and tastes, even in newborns (Davidson and Fox, 1988), elicit stronger EEG responses over the right hemisphere. Negative voice tone

(Bryden and MacRae, 1989) and facial expressions (Natlé et al., 1983) consistently evoke stronger reactions in left hemisphere.

Only right-sided lesions produce anosodiaphoria (Critchley, 1953; Mesulam, 1985a), a pathological indifference and non-responsiveness to negative stimuli (Adolphs and Damasio, 2000; Tucker, 1987) such as graphic surgical pictures (Gainotti and Caltagirone, 1989), faces displaying negative emotions (Adolphs et al., 2001; Jansari et al., 2000; Mandal et al., 1991), task failures and mention of the subject's symptoms (Cummings, 1997; Gainotti, 1972; Gordon, 1987). Indifference to the risk of missing contralesional stimuli helps to explain why left neglect is so extreme and lasts so long (Bisiach and Vallar, 1988; Egly et al., 1994; Mesulam, 2000b). Right-lesion patients with anosagnosia seem incapable of depression (Joseph, 1997; Prigatano, 1987); many in the current series were unable to muster as much distress as the best-adjusted neurology patients showed, even after incurring serious setbacks.

The right hemisphere operates a distributed network for rapid responding to danger and other urgent problems (Mesulam, 2000b). It preferentially processes environmental challenge, stress and pain (Gainotti, 1997; Wittling, 1995) and manages self-protective responses (Gazzaniga, 1998; Geschwind and Galaburda, 1985; Moscovich, 1983; Simon, 1967; Webster, 1977) such as avoidance (Hugdahl, 1995) and escape (Bannister, 1992; Davidson, 1984, 1995; Ornstein, 1995). The emergency network's activities are coordinated by the right posterior association cortex (Jackson, 1874; Kastner et al., 1998; Tucker and Williamson, 1984), which links stimuli with their historic adaptive consequences (Gibson, 1979) such as doing harm (Janis and Mann, 1977a) via visual images (Jackson, 1874; Luria, 1973). Screened for motivational urgency across a link with the limbic system at the cingulate gyrus (Heilman et al., 1993a; Mesulam, 1981), images trigger arousal (Heilman et al., 2003, b) and emotion felt in the right somatosensory area (Adolphs and Damasio, 2000; Mesulam, 2000a) and interpreted contextually (Ben-Ze'ev, 2000) in the right parietal cortex (Heilman et al., 1993b). Recognition of danger prompts the right brain to release norepinephrine, which locks awareness on the stimulus and generates compelling negative emotions such as hostility, dread and fear (Bear, 1983; Ochsner and Schachter, 2000; Tucker and Williamson, 1984). Such emotion interrupts other, ongoing activities (Allport, 1989) and seizes the whole brain's focus (Heilman and Van Den Abell, 1980; Mesulam, 1981) through the primary network for directed attention and vigilance (Mesulam and Geschwind, 1978; Tucker et al., 2000), which extends from the right parietal lobe to the

thalamus and reticular activating system (Dimond, 1979; Mesulam, 1985b; Nadeau, 1999). Emotionality compels rapid decisions (Geschwind, 1978), made in fragments of a second (Janis and Mann, 1977a). Gnostic imagery of the habitual self-protective response to a threat, sent to the prefrontal cortex (Barlow, 1985), is implemented immediately, bypassing the planning process in the name of haste (Damasio, 1994; Markman and Moreau, 2001; Mountcastle et al., 1975). Emotionality is thus the right brain's "red phone," compelling the mind to handle urgent matters without delay.

The narrowed perspective of right-lesion victims overlooks, and thus prevents addressing, urgent hazards endemic to specific situations. Some make no effort to avoid public displays of poor hygiene or immodesty in the hospital (Kirshner, 1986). A fastidious woman with a right parietal intracerebral hemorrhage knocked over her 32-ounce beverage, staring as the contents slowly emptied out onto the conference table. Though she spilled food and drink almost daily, she never took precautions to avoid doing so. A computer engineer hospitalized for a right parietal infarct eloped after removing his wrist alarm and stopped at the hospital cafeteria's ATM to withdraw a getaway bankroll. When apprehended 1 hr later, he still sat across from the ATM, unconcerned about completing his escape.

Right-sided lesions sometimes induce frank alexithymia (Cummings, 1997; Heilman, 1994; Schore, 1994), a total indifference to discomforts like starvation (Brown-Sequard, 1874), sexual deprivation (Goldstein, 1952; Tucker and Frederick, 1989), and even the pain of pressure sores (Harrington, 1995). One patient's electrolyte imbalance from the inadvertent deprivation of food and drink started a seizure disorder. Patients premorbidly proficient in dietary control of their diabetes induced diabetic crises after injury. Some patients remained in clubs with loud music and light shows despite phonophobia and photophobia.

Survivors remain indifferent to threats even when they grow progressively more serious over time. A woman with right-sided cortical atrophy after peripartum anoxia and a young man struck with right carotid dissection and occlusion in the delivery room continued living in their mothers' homes after 4 years, passive and complacent as spouses petitioned for divorce, older children became more bitter and distant and their newborns grew up as strangers. An executive with a grade three right posterior temporal astrocytoma postponed surgery for two years. A physician planned a healthy lifestyle after diabetes and cholesterol led to his right parietal CVA, but he smoked and ate with abandon until his death one week after his rehabilitation discharge.

Certain social situations demand urgent responding, and non-response to the signals of urgent need evokes extreme ill feelings (Goffman, 1967). An intact brain remains vigilant for such signals, while one with right-hemisphere damage monitors carelessly and ignores messages that are received, resulting in frequent inappropriate omissions (Joseph, 1997). Survivors of right-sided, and particularly posterior, lesions fail to respond to others' urgent requests and complaints (Molloy et al., 1990; Searle, 1979; Segalowitz, 1983; Weylman et al., 1989), acute distress (Adolphs and Damasio, 2000; Heilman, 1999a; Taylor, 1987), or appeals for help (Damasio, 2003); even the most urgent warnings of therapists are often ignored. Such unresponsiveness is usually interpreted as indifference, lack of commitment, or disrespect (Jones et al., 2001; Miller, 2001; Stemmer et al., 1994), rather than neurobehavioral malfunction (Goldberg, 2001).

The tendency to ignore or minimize urgent warnings can have a devastating impact in the workplace: Supervisory complaints and even warnings of impending termination are often ignored, especially when delivered in the polite style (Hawkins, 1977) of the white-collar employer (Chiarello, 1999). A young technical worker with a right parietal CVA dismissed so many important complaints from his supervisor that he was slated for firing. He corrected errors and kept his job only after explicit feedback was conveyed in writing. A young attorney with a right parietal contusion denied receiving ongoing criticisms for his on-the-job errors. Even a half-hour stern, formal review of the infractions aroused no suspicion that he had lost his job.

Forewarning–Foresight Blindness

Visual imagery is the medium for the cognitive function of anticipation (Bortner, 1968; Horowitz, 1972), accomplished by “constructing an image or model, running the model faster than the environment, and predicting that the environment will behave as the model does.” (Galanter and Gerstenhaber, 1956; see also Calvin, 1989). Anticipation of future events and consequences is the responsibility of the “response selector mechanism” situated in the parietal cortex (Battelli et al., 2003; Corbetta et al., 2000; Corbetta and Shulman, 2001; Schlesinger, 1962; Tulving et al., 1994). It depicts the results and effects of each response option (Gregory et al., 1982; Miller et al., 1960) to guide planning (Goldberg, 2001), preventing errors (Reason, 1990; Walter, 1953) by identifying the option with the best cost-benefit ratio (Damasio, 2003): “Achievement of the most effective and economical organization of action depends in large measure on . . .

anticipation. . . (It) is essential to adequate performance.” (Kahneman, 1973). Superior anticipation afforded by the evolutionary expansion of the parietal lobes (Bradshaw, 1997; Eccles, 1989; Kolb and Whishaw, 2003) lies at the heart of the human cognitive advantage (Bogdan, 2000; Bronowski, 1973; Deacon, 1997; Leakey and Lewin, 1977; Smith, 1989).

The most complex imagery for hypothetical constructs is generated by right hemisphere activity (Beeman et al., 2000; Bryden and Ley, 1983; Kasher et al., 1999; Pegna et al., 1997; Ratcliff, 1987), and right-sided lesions sometimes block anticipatory imagery (Crosson et al., 1989; Tulving, 1992), eliminating a view of the future from the temporal perspective. For example, a veteran department manager stopped making major decisions after her right parietal CVA because she no longer pictured the outcomes. A design engineer could no longer imagine the end-product of his modifications. A fabricator of custom machine parts turned over most of his work to his employees. In each instance, the afflicted individuals reported having been exceptionally reliant on visual imagery prior to the injury.

For the modal adult, the right brain supplies the images of negative outcomes and side-effects (Bradshaw, 1997; Hellige, 1991; Rourke, 1989). The right posterior cortex not only previews failures and disasters but also feels their emotional impact in advance, stiffening resolve to avoid them (Damasio, 1994). BPPD survivors report feeling avoidant prompts so weak that they are not heeded. When anticipation fails, hazards are not avoided (Janis and Mann, 1977a) and maladaptive courses of action are pursued (Janis and Mann, 1977b). Children with right-brain deficit disorder ignore task obstacles, accept impossible challenges, make grossly inadequate efforts (Rourke, 1989), and are stunned by the poor outcomes (Johnson and Myklebust, 1967). These children act fearless because they overlook “the dangers inherent in the situation” (Rourke, 1989). In the same manner, adults with right-brain injuries are prone to make impulsive errors (Binder et al., 1987; Joseph, 1997; Mesulam, 1985a; Pimental and Kingsbury, 1989).

Here are examples from the author's series. A post-polio victim with a right parietal CVA was careful about falling, but he rocketed his electric scooter down a familiar flight of stairs. A teen runaway with a right parietal contusion, exiled to a distant state, dove out of a second-story window at his first chance and got lost in the woods. A widow with right parietal CVA nightly traversed an unlit grand staircase covered with cats while wearing a floor-length nightgown. Only hours after being approved to drive, a woman with a temporoparietal removal shopped for a safe SUV, but purchased the one

that was scandalously prone to lethal rollovers. A man with right frontoparietal contusions and a 10-pound lifting restriction loaded 50-pound bags of rocks into his car “without thinking.” Driving restricted for his right parietal CVA, a man drove his noisy sports car up to the outpatient unit, blasted a questioning therapist with vulgarities, and challenged her to try to stop him from driving. A dentist, whose right parietal contusion occurred when his enraged girlfriend deliberately drove into his car, planned his discharge into her care.

Navigation of social relationships relies upon anticipation of the social partner’s future reactions (Goldberg, 2001; Mithen, 1996; Pinker, 1997). Patients with right-brain injury generate erroneous and illogical behavioral predictions (Beeman, 1993; Brownell et al., 1997). Children with right-brain deficit disorder fail to anticipate predictable negative reactions to their verbose, impulsive, immature communication style, which contributes to their rejection and isolation (Johnson and Myklebust, 1967; Rourke, 1989). Adults with BPPD fail to anticipate negative reactions to violations of important social rules such as reciprocity (Trivers, 1985). For example, the physician with the right parietal CVA sold and replaced his wife’s car without discussing it with her, never suspecting that she would resent his presumption. While voicing gratitude for his stalwart support during her convalescence, a young manager with a right parietal contusion returned her suitor’s ring, proposed that they end their cohabitation, and allotted no time to see him without anticipating that he would feel rejected and respond in kind. A trauma victim with a right parietotemporal contusion stalked her former lover, unwilling to admit that the bogus police report of his sexual abuse she made a year earlier had brought their relationship to a close.

Oversimplified planning in BPPD ignores the possibility of failure because of the combined effects of defective anticipation and anosagnosia (Damasio and Anderson, 2003; Dormer, 1996). Over-optimistic expectation of easy success (Bisiach and Geminiani, 1991) elicits cursory planning (Kunda, 1999) and casual implementation (Damasio, 1994). When the crudely planned and executed actions produce incorrect behaviors, the damaged perceptual system’s persistent illusion of adequate performance (Heilman, 1991; Kihlstrom and Tobias, 1991; Prigatano, 1991) blocks recognition of any need for mid-course corrections (e.g., Joannette et al., 1990; Molloy et al., 1990). For example, a neurosurgeon agreed that the massive right parietal damage from carotid dissection shown by his CT scan would be totally disabling to his patients, but he planned to resume doing surgery and driving immediately. A right temporoparietal trauma patient decided to quit her job to become a vocalist with-

out worrying about recent difficulties in carrying a tune. Though he failed at eight marketing jobs in six years, a business school graduate with a right parietal contusion continued to seek similar positions. A tennis prodigy with a right posterior temporal CVA resumed playing despite a poor prognosis, struggling for four years before admitting that his neglect, impaired aim and slow reactions had destroyed his tennis career. A receptionist with right parietal contusion did not adjust her job search strategy despite 22 consecutive unsuccessful interviews. Unable to anticipate future difficulties, right-lesion patients frequently deny any need for rehabilitation treatment (Adair et al., 2003; Calvanio et al., 1993; Diller, 1987; Gerstein, 1975; Gorelick and Swiontoniowski, 1989; Prigatano, 1999).

CUMULATIVE EFFECTS: GLOBAL MALADAPTATION

Perceptual guidance is needed most for unusually challenging tasks and roles (Mesulam, 2000a): Exploring unfamiliar environments, avoiding serious hazards, and making on-line decisions to exacting specifications. During postacute recovery, patients progress from tightly circumscribed lifestyles to full responsibilities, with declining tolerance for errors. Thus the need for perceptual guidance grows gradually, and the BPPD deficits surface only after a delay.

The deterioration of social networks typically begins in the second six months or second year post onset (Lehr, 1990). The limited social circles, abbreviated romances, and stressed marriages of chronic patients testify to the interpersonal impact of BPPD. Members of a long-term support group describe the members with BPPD as “high maintenance” due to inconsiderate behavior, demandingness, rule-breaking, and unreliability. As their relationships deteriorate, they neither acknowledge the decline nor make changes, which only amplifies partners’ alienation. Damasio (2003) notes the correlation of right parietal damage with divorce and filial estrangement.

The author (Schutz, 2002) investigated marital stability as an aspect of functional recovery in a sample including 48 discharged rehab patients with BPPD. The subjects were working at onset, referred for intensive post-acute cognitive rehabilitation, and available for follow-up interview held between 3 months and 11 years post discharge (mean interval 16.5 months, standard deviation of 21.4). They ranged in age from 18 to 67. Inclusion in the BPPD group was based on CT or MRI evidence of a lesion in the right parietal or right parietotemporal area. The etiology was traumatic brain injury in approximately two-thirds of the cases and cerebrovascular accident in the remaining

Table 1. Sample Description and Outcomes from Schutz (2002)

	BPPD sample	Non-BPPD sample
Sample		
Number of cases	48	48
Mean age (<i>SD</i>)	33.02 (12.0)	34.02 (15.4)
Mean education (<i>SD</i>)	14.78 (3.4)	13.16 (2.6)
Months chronicity at f/u (<i>SD</i>)	32.07 (18.1)	28.30 (15.4)
Etiology		
Closed head injury	63%	64%
Penetrating brain wound	6%	2%
Cerebrovascular accident	31%	33%
Findings at follow-up		
Unemployed	84%	51%
Divorced	52%	15%
24-hr supervision	30%	18%

third. A comparison group with focal lesions located in other regions was matched for severity, etiology, age, and time post discharge (See Table 1). Of the 21 patients with BPPD who were married at onset, 11 or 52% had divorced by the time of the follow-up, in contrast to 15% for the non-BPPD group. The marital estrangement did not surface until the second year, as spouses became increasingly aware of the survivor's high-maintenance features and more alienated and offended by poor conduct. In some cases, the divorce did not occur until the third to fifth year post discharge. Survivors also became estranged from adolescent or adult children during this phase.

Expert clinicians designate right-sided lesions and anosagnosia as risk factors for vocational failure (Lezak, 1987; Prigatano and Schachter, 1991; Weinstein, 1991). The rate of stable competitive employment with BPPD was 16% compared to the non-BPPD group's 49% (Schutz, 2002). An earlier study (Knapp, 1959) of younger CVA patients found none of the 10 with left hemiparesis employed while 54% of 11 with right hemiparesis held jobs. In another study, most of the unemployed penetrating wound cases had right-sided foci and constructional deficits (Kaufman et al., 1985). Finally, neuropsychological tests maximally sensitive to BPPD lesions are among the best predictors of vocational outcome (Boake et al., 1997; Dennerl et al., 1966; Dikmen and Morgan, 1980; Heaton et al., 1978; Heaton and Pendleton, 1981; Ruff et al., 1993).

Patients with impaired visual imagery fail to prioritize their actions to assure a satisfying life (Damasio, 2003). Many BPPD survivors have become isolated and lonely, but very few have even attempted to replace lost spouses, friends and associates. Only four single young ladies started new romances, and all of them were short-lived. A few former patients retraced old paths of relationship conflict, reuniting unsuccessfully with ex-spouses or

abusive parents. Career change was also rare; only six even attempted it. Four of these are presently unemployed, one is in supported employment, and one works for minimum wage in a competitive job.

The ultimate test of long-term recovery is the overall adaptation to the demands of life (Symonds, 1970). The criteria for mental health (Jahoda, 1959) are compromised for almost all of the patients in the author's series. Beyond the above-captioned loss of opportunities for love and work, reality awareness is distorted by excessive expectations, autonomy is compromised by poor decision making and safety judgment, and effective pursuit of future goals is rare (see also Damasio, 2003). In these terms, only 2 of the 52 former patients could be described as well-adjusted. Additionally, personal problems tend to go unresolved. For example, children with right-brain syndrome decline in adjustment with age, accumulating problems instead of solving them (Greiffenstein and Baker, 2002; Johnson and Myklebust, 1967). By adulthood, they have accumulated enough psychosocial difficulties to display "among the worst (prognoses) that can be imagined from a psychological perspective" (Rourke, 1989). On this basis, the survivors appear to be at risk for further declines in adjustment over time.

DIFFERENTIAL DIAGNOSIS OF BPPD

Focal lesions in grey matter of the inferior parietal lobe and parieto-occipito-temporal junction of the right hemisphere produce the symptoms of BPPD (Hecaen and Albert, 1978; Luria, 1973; Mesulam, 1985a). The 52 cases in the present series all have radiographically or neurosurgically documented lesions in this region. Similar symptoms result from focal lesions of the white matter tracts projecting to and from the posterior association cortex (Filley, 2001; Geschwind, 1965; Mesulam, 2000b; Rourke, 1989), especially those severing the tracts that directly underlie the junction (Leibovich et al., 1998; Vallar and Perani, 1986). Furthermore, lesions of unimodal perceptual zones in the occipital, temporal and superior parietal lobes and of the reticulo-cortical tracts involved in activating the system can produce similar symptoms (Goldberg, 2001). The syndrome can also occur without focal findings, for example, in some cases of cerebral anoxia without radiographic abnormalities or in milder trauma cases. In such cases, the syndrome is revealed by behavioral symptoms, such as those shown on neuropsychological tests.

Diagnosis by analysis of test behavior is hazardous. The symptoms of BPPD converge with deficits seen in both the dorsolateral- and orbital-prefrontal focal

syndromes (Goldberg, 2001; Stuss and Benson, 1986). The overlap is due in large measure to the conjoint participation of the anterior and posterior association cortices in the distributed network for planning and problem-solving (Goldberg and Costa, 1981; Mesulam, 2000a) mentioned above. Damage to either region results in planning and execution errors. Careful investigation reveals a dissociation in how the errors are made, with anterior lesions impairing executive functions such as initiation, set shifting and sequential organization while posterior lesions compromise perceptual guidance (Husain et al., 2000; Luria, 1980). For example, frontal impulsivity reflects the loss of abilities to inhibit and plan accurately, while BPPD produces casual planning and careless execution because easy success is assumed. Simple cuing to anticipate errors corrects them only in BPPD, which shows that full control of the efferent functions is preserved. The symptom patterns of frontal lobe damage and damage to the right and left posterior association cortices are presented in Table 2.

Association cortex disorders can produce any of several patterns of defective test performance (Prigatano and Klonoff, 1990; Trexler et al., 1994). Thus testing for parietal lobe syndrome must examine a heterogeneous collection of functions (Kolb and Whishaw, 2003; Luria, 1980): "A greater variety of clinical manifestations is likely to arise from disease of the parietal lobe . . . than any other." (Walsh and Darby, 1999). BPPD is associated with three patterns of impaired test performance, which may be seen individually or in combination. The first pattern consists of prominent deficits in basic visual perception and hemi-field awareness, including neglect on line bisection, figure drawing and visual search tests, and defects in visual search accuracy and perception of size, shape or angulation on such measures as the Visual Search and Attention Test and Benton's Visual Form Discrimination and Judgment of Line Orientation. This pattern is associated with defective maze performance due to problems with anticipatory gaze tracking and leftward route-finding. Rotational errors are seen on procedures such as the Symbol Digit Modalities Test. Finally, slow time scores may be

achieved on Trail-Making and pegboard tasks because of slow, inaccurate, targeting and balky target shifting.

The second pattern of impaired constructional praxis can be elicited by Kaplan's Parietal Lobe Battery of the Boston Aphasia Exam on figure drawing tasks and on WAIS Block Design (Hellige, 1993; Lezak, 1995). Qualitative features such broken contours (Kaplan, 1989), design rotations and(or a trial-and-error response style (Joy et al., 2001) often accompany poor scores.

The third pattern consists of topographic disorientation and somatosensory dysfunction, as demonstrated on tests of stereognosis and real-world route finding (Barrash, 1998; Butters et al., 1970). The third pattern, and to some extent the other two, are associated with impairment on the most sensitive measure, the Tactual Performance Test (Teuber and Weinstein, 1954). TPT requires not only tactile exploration and learning of a form board, but cross-modal integration of sensorimotor stimuli into a spatial map (Reitan and Wolfson, 1985b). Along with impaired total time and often a peak on the nondominant hand score, search excursions may go off the board, poorly targeted movements push placed blocks out of their slots, and reaches to pick up blocks make misguided return trips to place them in particular slots on the board. Blocks tend to be tried repeatedly in slots of grossly incorrect shape (e.g. hexagon into rectangle, star into triangle).

CONCLUSIONS

The pivotal contribution that broad-perspective perception makes to adjustment and adaptation becomes obvious in the long-term follow-up of cases of acquired damage. When maps to navigate the physical and social world cannot be upgraded or replaced, decisions and game plans guided by the narrowed perspective shift farther off-target as the lesion ages. Blinded to dangers and risks, survivors casually choose and implement unsafe and unwise courses of action. Unable to anticipate, recognize or understand others' reactions, they take actions

Table 2. Cognitive-Perceptual Symptoms of Focal Lesions by Region

Symptom	Right posterior AC	Left posterior AC	Frontal
Neglect/hemi-inattention	Persistent/left	Temporary/right	Minimal
Perceptual defects	Panoramic/gestalt	Foveal/detail	Minimal
Aphasia	None	Receptive	Expressive
Aprosodia	Receptive	None	Expressive
Anosagnosia	Severe/global	Limited to language	Moderate
Imperception of novelty	Severe	None	Inconsistent
Emergency responding	Disengaged	Intact	Disorganized
Planning/problem solving	Can't foresee risks	Lacks confidence	Executive deficits

that frustrate, disappoint, offend, betray and alienate the most important people in their worlds with increasing frequency until they have destroyed most of their roles and relationships. The loss of these major components of the lifestyle only serves to accelerate the obsolescence of the old navigational programs (Horn, 1992), while the individuals remain unable to imagine any new direction to take. Awareness of this syndrome and its profound long-term consequences alerts health care providers to prepare patients and families for their difficult futures, prompts cognitive rehabilitation specialists to develop compensatory procedures that specifically target BPPD symptoms, and encourages follow-up researchers to chart the late progression of the adaptive deficit closer to its end-point.

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