curiosity, intrigue, ambition, love, and hate: emotions that are likely to translate into behavioral goals that will be pursued through action plans that are sustained over long periods of time. Within the left hemisphere, subjective value can thus be incorporated into the serial generation and modification of distributed concept representations that underlie thinking, action, and language. In this conceptual scheme, some emotions have bihemispheric components. The love of an individual is sustained over years but the thought or perception of the loved person elicits an acute emotional experience. Fear is typically elicited by particular situations but for members of discriminated minorities, it translates into a way of life. Furthermore, the process of settling into constellations of attractor states in both OFCs assures that in any behavioral context, proactive and reactive emotional knowledge will be brought to bear. The hypothesis that the right hemisphere plays the cardinal role in the reactive emotional function and the left hemisphere plays a major role in proactive emotional function provides a basis for understanding the emotional function that accommodates both the foundational network anatomy detailed by Rolls and the nonsequential/sequential hemispheric organizational principle. Unfortunately, I have been unable to identify any studies bearing on this thesis.

LESION EFFECTS

Neural networks encode knowledge in the strengths of their neural connections and they support the processing of input from other networks. This knowledge is acquired through experience that presumably begins in utero and its encoding in network connectivity instantiates regularity, frequency, and age of acquisition effects. Encoded knowledge can represent structure, pattern, or sequence (Nadeau, 2001). The mechanisms underlying neural settling assure that processing in different networks and hemispheres occurs simultaneously and in a coordinated fashion that assures parallel constraint satisfaction, e.g., between propositional language and affective prosody.

Lesions of networks produce graceful degradation of network-specific knowledge and processing. Lesions of white matter between networks disconnect them but also destroy the knowledge encoded in internetwork connectivity. With these general principles in mind, the mechanisms by which lesions produce their effects can be broadly understood.

Because of the special role of the right hemisphere in emotional processing, right temporal and parietal lesions, most often due to stroke, degrade processing of perceptual input conveying affective prosody, emotional facial expression, and emotional gesture (Chapters 4 and 5). Consequently, this input cannot adequately engage the right frontal cortex via corticocortical connections to enable repetition or mimicry. It also cannot adequately engage the substrates for objective emotional semantics (presumably widely distributed) and subjective semantics (OFC and limbic structures) to enable derivation of emotional meaning. Loss of input to the right frontal cortex conveyed from right temporal and parietal cortex and from OFC blunts emotional contagion.

Lesions of the right frontal cortex, also usually due to stroke, degrade affective prosodic, facial, and somatic emotional expression (Chapters 4 and 5). It would seem that they should leave intact emotional perception, recognition, and comprehension. However, as we have discussed, networks are heavily interconnected. Thus damage to one network of a neural ensemble will inevitably impact the processing in connected networks. The computational basis for these remote effects was systematically explored by Farah and McClelland in their study of category-specific knowledge deficits (Farah and McClelland, 1991).

Damage to OFC and limbic structures, usually due to degenerative disease (e.g., frontotemporal lobar degeneration (Chapter 13), Parkinson's disease and other Parkinsonian disorders, Huntington's disease (Chapter 9), and autoimmune encephalitis (Chapter 11)), degrades neural networks encoding subjective semantic knowledge, thereby degrading both comprehension and expression of emotions, as well as the autonomic manifestations of these emotions.

Damage to the white matter as a result of traumatic brain injury (Chapter 12), multiple sclerosis, stroke, ischemic demyelination due to microvascular disease, infection (e.g., human immunodeficiency virus and progressive multifocal leukoencephalopathy), or to dysmyelinating diseases (e.g., metachromatic leukodystrophy and adrenoleukodystrophy) can disconnect the major components of the global emotional network (at once indirectly causing some dysfunction of each of the components, as noted previously), and it can directly affect the operations of individual networks, wherein neurons are connected by myelinated axons.

The mechanisms of impairment following focal lesions discussed in the forgoing can provide a logical explanation for the disorders of affective prosody and facial emotional comprehension and expression discussed, respectively, in Chapters 4 and 5. They can even account for some of the cases that seemed to defy explanation, e.g., the impairment in comprehension of affective prosody associated with OFC lesions (Hornak et al., 1996, 2003) discussed in Chapter 4.

ALEXITHYMIA

As detailed in Chapter 3, alexithymia is characterized by impaired awareness of one's feelings and reduced ability

to explicitly identify and describe them. There is limited differentiation of emotional states. The complexity of alexithymia and the variety of disorders in which it is observed defies simple mechanistic explanations. In this section, we consider alexithymia in terms of the PDP architecture we have described, a perspective that implicitly invokes cerebral systems rather than individual structures.

The defining characteristics of alexithymia suggest a reduction in the influence of subjective semantics, encoded in OFC, on objective emotional semantics, encoded broadly in convexity association cortices. This could be the result of any one of a number of factors that have been associated with increased prevalence of alexithymia (see Chapter 3 for details). Learning to cope with pervasive adverse childhood experiences could involve adaptive attenuation of OFC-convexity cortical connectivity such that behavior becomes shaped to a greater extent by objective knowledge and strategies and is not so influenced by emotions that threaten to become overwhelming. Damage to OFC as a result of penetrating trauma could attenuate projections to convexity cortices, as would diffuse damage to cerebral white matter incurred in closed head traumatic brain injury. Penetrating traumatic brain injury involving the anterior insula is associated with an increased prevalence of alexithymia. Alexithymia has been associated with a multidimensional deficit in interoceptive awareness involving cardiac, respiratory, muscular, and gustatory signals (Murphy et al., 2018), signals conveyed to OFC (and the amygdala) via the insula (Yeterian et al., 2012). Alexithymia is more prevalent after right brain stroke than left, consistent with the Gestalt processing that is particularly characteristic of the right hemisphere and the particular role of the right OFC in reactive emotionality. There is an increased prevalence of alexithymia in individuals with posttraumatic stress disorder (PTSD), although this might well reflect premorbid factors that actually predispose to the development of PTSD.

The consequence of any one of these various factors would be a failure to develop knowledge (e.g., as a result of pervasive adverse childhood experiences) or loss of knowledge (loss of neural connections due to gray matter or white matter injury). Because the major hidden unit fields (insula, OFC, convexity cortices, and their connections with each other) operate as a system, damage in any one field could affect the operations of the entire system, albeit in somewhat different ways that reflect the peculiarities of the knowledge stored in any one network. As we have noted, knowledge, at least in the cerebral cortex, degrades gracefully. The result is the preservation of the knowledge most deeply represented in neural connectivity by virtue of implicit statistical regularities, frequency, and age of acquisition effects. The behavior of patients with semantic dementia is exemplary (Nadeau, 2012, 2020). As noted earlier in this chapter, as the disease progresses, knowledge of less frequent and unrepresentative exemplars (e.g., platypuses) is lost. In tests of naming to confrontation, zebras become "donkeys." Ultimately, any animal becomes an "animal," or the patient is unable to produce any response at all. The system we have described, coupled with PDP principles, would predict that with attenuation of knowledge within or between the insula, OFC, and convexity cortices, there would be dedifferentiation of representations of emotional states—precisely what has been observed (Chapter 3). With severe dedifferentiation, for example, there need not be a loss of emotionality but all negative emotions could be reduced to a simple sensation of emotional distress (the emotional counterpart of the "animal" response).

There also exist physiologic mechanisms for attenuation of representations, most obviously attentional and intentional mechanisms (Nadeau, 2021). In addition, as noted earlier, the essential computational function of the basal ganglia appears to be dimensionality reduction. In the sensorimotor basal ganglia, a complex multidimensional array of sensory and motor input is translated into one or more optimal behavioral responses. Dopamine regulates the degree of dimensionality reduction. With insufficient dopamine, as in Parkinson's disease, there is excessive reduction, with the result that few, perhaps zero or one behavioral representation becomes available (hence the long-recognized difficulty that patients with Parkinson's disease have with doing two things simultaneously). As noted previously, in humans, the functions of the sensorimotor basal ganglia appear to have been superseded by cortical mechanisms. However, we do not know if this is true of prefrontal/limbic portions of the basal ganglia (ventral head of caudate, nucleus accumbens). If these anterior portions of the basal ganglia contribute meaningfully to human cortical function, then dopamine deficiency might plausibly be associated with alexithymia and administration of dopaminergic drugs might relieve it (see also Chapter 14). The prevalence of alexithymia in patients with Parkinson's disease is twice that in controls (Chapter 3). However, we do not know if this reflects the effects of dopamine deficiency or cortical disease (Braak et al., 2003) (i.e., we have no data comparing behavior on and off dopaminergic agents). Furthermore, dopamine levels are considerably less depleted in the prefrontal/limbic striatum than in the sensorimotor striatum (Kish et al., 1988) so dopamine deficiency in the former might not be a factor in alexithymia.

DEPRESSION

Depression is a chronic state that is experienced in different ways by different people. Its development may be