Introduction

Deciphering the natural environment, including human form and function, is best achieved by interdisciplinary study. The differing sciences inform each other and there is often two-way or bidirectional information exchange between disciplines. For example, evolution informs neuroscience and neuroscience has the power to inform evolution. This was well described by David Lewis-Williams in his interpretation of cave rock paintings in which certain artistic depictions reflected different stages of hallucination in the human mind. In his elegant overview of how ancient art helps understand the neuroscience of our minds, presented in his book *The Mind in the Cave*, he describes how the intensified trajectory of altered consciousness level of visual hallucinations (fully fledged hallucinations) in ancestral humans was recognized as being identical to migrainous fortification spectra (jagged lines), familiar to contemporary neurology [1].

The Nobel prize-winning physiologist Eric Kandel eloquently portrayed how neuroscience and art inform each other, detailed in his two exceptional books, *The Age of Insight* [2] and *Nature's Reductionism: Bridging the Two Cultures* [3]. The parent field of medicine, biology, is often instrumental in the understanding of human brain mechanisms, such as theories of how we developed superb color vision. Isbell's snake-detection theory helped inform current-day neurology and psychiatry about snakes having acted as a primary selective pressure operating on primates and expanding their visual systems. In brief, evolutionary exposure to venomous snakes, which are usually patterned and colored, induced trichromacy in the African primates, as opposed to the mere dichromacy of the South American primates, the latter having had minimal venomous snake exposure. The third component of the theory, the Madagascar lemur, which has the worst color vision of the primates, had no exposure to venous snakes at all [4]. Many arts and sciences are therefore valuable in helping to understand the evolution of the human executive mind. From my perspective, the discipline of clinical neurology, which is concerned with brain lesions and their consequences, or fractured brain circuits, can inform the discipline of archeology that deals with the analysis of fractured skulls. Hence, fractured minds and brain circuits can similarly be regarded as a two-way process in the study of neuro-archeology. As can be seen from Figure 0.1, many disciplines helped inform the assembly of our frontal lobes and our minds. There are, of course, numerous disciplines in the arts and sciences. Perhaps increased interaction among them will lead to ever-greater insights. Clinical medicine just happens to be a discipline that is among the least interdisciplinary at the present time, as we shall see in Chapter 12. The great visionary and biologist Edward Wilson coined the term *consilience* in his book of the same name, emphasizing the unity of all knowledge and disciplines, and conceived of all the arts

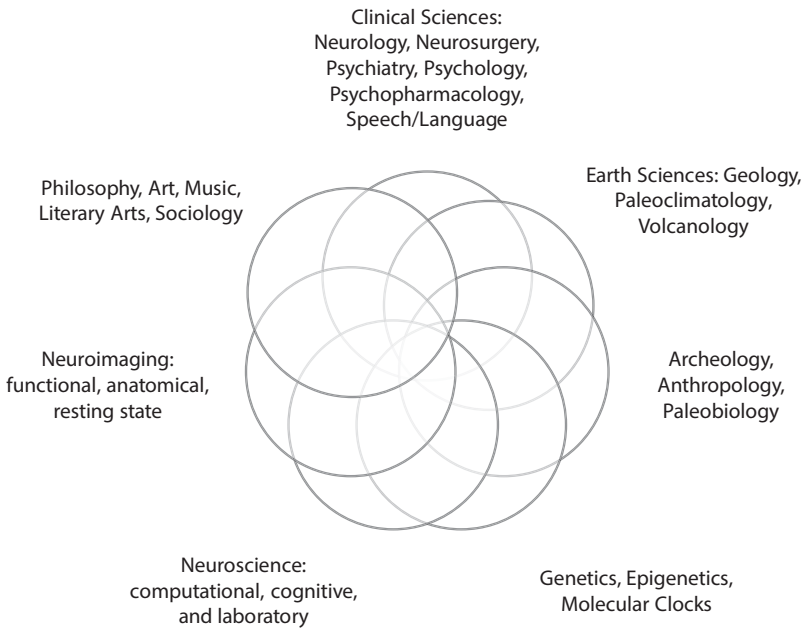

Figure 0.1 Science, art and clinical disciplines inform our brain functions.

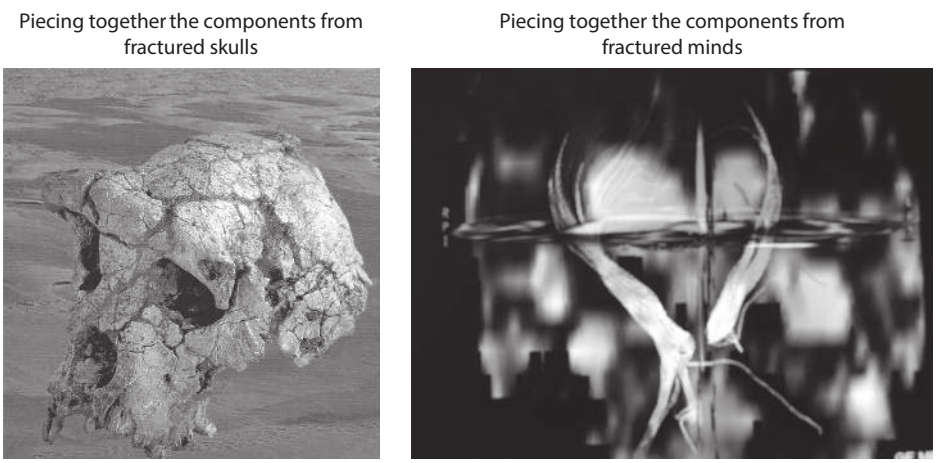

Figure 0.2 The unravelling of the human mind: a method of studying human cognitive evolution. Reprinted by permission from Springer Nature from Wood B. Hominid revelations from Chad. *Nature* 2002;418:133–135.

and sciences as being innately bound by a small number of natural laws in physics and chemistry [5].

One can either gather fractured skulls and bones or study fractured brain circuits in deciphering brain function and how it evolved (Figure 0.2). Similar to the approach of Vilayanur Ramachandran and Oliver Sacks, individual case reports and sometime case series can be very illuminating, as have the recent spate of *n*-of-1 trials that are gaining

momentum as part of the precision medicine movement. This book is woven around key case reports, case series, case control studies, and cohort studies, most of them published by the author, emanating from four different stroke and cognitive neurology registries cited in the acknowledgments. These are used throughout the book to highlight how the multitude of frontal lobe presentations and related cognitive and behavioral syndromes, after various brain lesions, illuminate the unraveling of the mind, together with an evolutionary perspective. From a terminological perspective, frontal lobes refer to the anatomical entity Brodmann areas (BA) 4, 6, 8, 9, 10, 11, 12, 13, 32, 14, 44, 45, 46, and 47. The executive mind, strictly speaking, refers the lateral prefrontal cortex or areas BA 9 and 46 and the behaviorally relevant frontal regions BA 11, 12, 13, and 14. *Frontal network systems* (FNS) refers to the frontal-type syndromes that may be due to frontal lobe lesions, but are actually more often due to lesions of the expansive networks throughout the brain that the frontal lobes are part of. For the purposes of this book, *the executive mind* is used to refer to the frontal lobes, both executive and behavioral, and their expansive networks in their entirety.

Hence, neuro-archeology has been used as an overarching term to refer to the relationship of mind functioning as it pertains to archeological finds [6]. Archeologists delve in material remains and infer past human behavior. The discipline has spawned a number of fields of study, including cognitive archeologists who endeavor to infer mental capabilities and modes of thought processes from reconstructed behaviors. Cognitive neuroscientists are concerned with the neurobiological circuitry of behaviors associated with psychological processes. Neuroscientists pursue molecular chemistry, genetics, and cellular structures largely by studying extant neural organisms. Clinical neurology capitalizes on a vast array of neurological diseases and conditions that lead to relatively stereotyped syndromes that in their own way disentangle the mind and illuminate some of its workings. In the vast majority of these clinical cases and series there lies hope. Not only does precise diagnosis elucidate the – sometimes bewildering – presentations, but through the diagnosis lies the potential for management and treatment. At times it is nature that heals and sometimes with treatment and intervention there is marginal benefit and sometimes there are dramatically positive results. Frontal lobe lesions and their brain behavior relationships is a relatively new science; it was launched by the seminal Boston Crowbar case in 1864 in the USA and the Broca's aphasia report from France, but only blossomed in the mid-1980s [7,8].

In the 1950s, the neurosurgeon Penfield performed intraoperative stimulation experiments and made the important observation that frontal lobe stimulation revealed no response at all, other than a movement response when stimulating the motor cortex [9]. Luria's important frontal lobe function observations, mostly from traumatic brain injury (TBI) patients, still profoundly influence neuropsychology today. He was the first to make the crucial distinction between behavior from frontal lobe damage and cognitive function impairment from frontal lesions. For example, he reported the case of a man seen in his office who caught the next train out of Moscow's central station without any consideration for its destination [10]. The behavioral syndromes of frontal lobe pathology later expanded and were elegantly demonstrated by Lhermitte's innovative style of frontal testing and description of various field-dependent behaviors, in differing environments. These methods were key to understanding the syndromes and how very simple techniques may be illuminating in discerning frontal lobe behavior. He described how field-dependent behavior syndromes frequently emerge following disruptions of the mirror neuron network in the brain [11,12].

People with frontal lesions or pathology seldom seek evaluation; if they do, initial cognitive screening tests commonly employed, such as the Mini-Mental State Examination (MMSE), are insensitive to frontal cognitive syndromes, while the Montreal Cognitive Assessment (MOCA) is impervious to frontal behavioral syndromes [13,14]. Yet, the presentations may include those with profane, puerile, irascible, or facetious behaviors that are difficult to quantify and have no devised metric tests.

Mesulam belabored the common and astonishing paucity of formal neuropsychological test scores, often normal, associated with frontal lobe lesions [15]. Specific behaviorally oriented tests – such as Frontal Systems Behavioral Evaluation, Frontal Behavioral Inventory, Bar-On Emotional Intelligence Test, and the Behavioral Rating Inventory of Executive Function – are more likely to elicit the salient abnormalities [16–18]. Another major contribution by Mesulam was the introduction of the concept of an FNS, rather than frontal lobe syndrome. As subcortical infarcts, multiple sclerosis, TBI, toxic metabolic encephalopathies, and other multifocal processes are more common causes of frontal syndromes as opposed to lesions of the anatomical frontal lobe themselves, this becomes a preferable designation [19–21].

Things have changed for the better. Isolated case reports or case series are unable to provide insights with regard to frequencies of cognitive syndromes and FNS, the solution being computerized registry-based analyses. In addition, the protracted period associated with autopsy-verified lesion locality has been supplanted by increasingly sophisticated neuroimaging methods. Magnetic resonance imaging (MRI) nowadays has at least a dozen different imaging sequencing modalities that can detail acute strokes, arteries, veins, fiber tracts, neurovascular activity, and spectroscopy, all with superior resolution. Most importantly, intrinsic connectivity analyses by fMRI have yielded insights into brain network function. A registry-based approach for analyses of specific higher cortical function deficit (HCFD) subtypes, in particular FNS, determined that the latter were important and pervasive cognitive syndromes, the most common and ubiquitous in neurological and psychiatric disease. However, testing of HCFD by the three major clinical brain disciplines – neurology, psychiatry, and neuropsychology – differs markedly, that is with respect to discipline culture, historically, and philosophically. At the same time, each has unique contributions and so all complement each other. Certain brain disease processes such as stroke, epilepsy, meningitis, and encephalitis, however, require emergent evaluation.

Clinical cerebrovascular decision-making is constrained by a 4.5-hour thrombolytic therapy window or, even more demanding, the so-called *golden first hour* of intervening with clot-busting agents [22]. Multiple concurrent procedures, including neuroimaging and laboratory and cardiac investigations leave only a few minutes for clinical assessments, and no place for neuropsychological testing. Despite the challenge of performing cognitive evaluations in the emergency situation, at least a cursory appraisal of FNS is pertinent as these are the most common clinical neurological impairments. Clinical monitoring of FNS is crucial for appreciating any improvement or deterioration of the patient. A typical stroke damages approximately two million neurons and 14 billion synapses each minute [23]. The degree of attention and cooperation by the person is also severely limited in such a scenario. Yet the relevance of FNS testing is that the expansive supervisory cognitive network (metacognition) may be the most sensitive indicator of cognitive status once emergent evaluation has been accomplished. Based on one of the stroke registries, a system was devised that incorporated (1) behavioral neurological assessment of the myriad

known syndromes quantified in ordinal and nominal data terms; (2) a neuropsychiatric syndrome evaluation according to pre-specified criteria (DSM-IV), configured to nominal data; and (3) a neuropsychological battery approach recorded in predominant numerical, normed evaluations. The semi-quantitative bedside test was devised, incorporating cognitive, neuropsychiatric, and behavioral syndromes, and enabling assessment within approximately 20 minutes. By incorporating the extensive testing of syndromes germane to behavioral neurology and neuropsychiatry, in addition to brief neuropsychological batteries, a reasonable appraisal of FNS was accomplished by the development of the COCONUTS evaluation (**co**mprehensive, **co**gnitive test **neu**rological **t**est in **s**troke) [24].

Other registry-based research revealed that HCFD, including FNS, were not only very common in acute and subacute stroke, but that FNS were evident regardless of lesion localization. Hence, frontal, subcortical, posterior parietal, or occipital, and even subtentorial and brainstem strokes often had associated FNS. The frequency was surprising, with approximately half of people with subtentorial stroke manifesting with FNS [25–27]. In retrospect, as the frontal lobes and their networks connect to all areas of the brain, this now seems less surprising. Further research into the neurobiological mechanisms of these processes in isolated brainstem or cerebellar stroke by SPECT brain scanning has suggested a neurotransmitter perturbation to be a likely candidate [28]. These findings were corroborated by a subsequent clinical analysis of stroke patients with minimal or no long tract signs (one-sided weakness, numbness, or vision disturbance) with FNS caused by isolated subtentorial (brainstem or cerebellum) stroke [29].

Sometimes we find simple tests that may discern and diagnose complex processes. The mirror neuron system (MNS), for example, evolved in stages during our primate history from about 60 million years ago and can be affected by cerebral lesions. We can test for the MNS by documenting syndromes such as echopraxia, utilization behavior, and environmental-dependency syndromes. These are not commonly employed tests, yet they offer an important opportunity to improve neurological evaluation and monitoring of complex FNS [30]. Such an example proved to be a most decisive one in my formative years in neurology registrar (resident) training in the large modern subtropical city of Durban, South Africa, largely serving the Zulu population. During this time and even today, tuberculosis (TB) and human immunodeficiency virus (HIV) related neurological illness was rampant, to the extent that many neurological syndromes we encountered were often considered TB-related until proven otherwise. TB neurology can be immensely protean and can be a great masquerader, much like luetic disease was in earlier European history. This was probably an important reason why the young, well-educated Zulu man, a teacher, I encountered presenting initially with a seizure captivated our attention one morning on clinical rounds. Whatever we did, he did. For example, on completing the tendon reflex examination with the traditional Queen's Square reflex hammer, he picked it up and proceeded to elicit all his own reflexes. He also imitated actions, words, and gestures faithfully, without instruction to do so and even when asked not to do so, with his mystified physicians looking on. For example, he spontaneously started teaching the resident physicians when asked to accompany them into a room that was reminiscent of a classroom in which he took on the role of a teacher – the environmental-dependency syndrome. He displayed many different field-dependent behavioral syndromes, since described more precisely by Professor Lhermitte of Paris in 1986, which he termed imitation behavior, utilization behavior being the environmental-dependency syndromes [11,12]. The cause was an unusual bifrontal stroke, attributed to TB-related

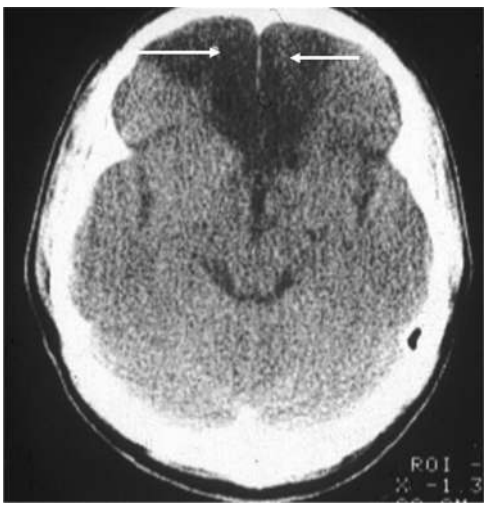

Figure 0.3 Bilateral frontal infarcts (strokes) decoupling brain circuits that hint at our evolutionary past (arrows).

vasculitis (inflammation) of his frontal brain arteries (Figure 0.3). The brain circuitry that had been lesioned, with the various field-dependent behaviors as the predominant clinical syndrome and without other neurological sequelae, had "uncoupled" his mirror neuron circuitry [31]. The MNS and its relevance to human cognition (language, praxis, theory of mind, learning) has only relatively recently been appreciated [32]. From an evolutionary perspective, the MNS is viewed as a fast-track learning mechanism, forgoing learning by trial and error, and is viewed as the neurobiological substrate of our cultural revolution, prompting Ramachandran to expound that "The MNS will do for psychology what DNA has done for biology" [33].

Studies of people with MNS uncoupling yields unique insights into brain functions. In a large clinical study some 25 years after the event of the Durban Zulu man with pervasive field-dependent behaviors, a series of 73 MNS-uncoupled patients derived from a registry of 1436 people with stroke were evaluated, and a much wider range of presentations was uncovered. The loss of personal autonomy that occurs may take many forms and is described in more detail in Chapter 11 [34]. Fortunately, the majority of people beset with sudden field-dependent behavior syndromes recover within days to weeks. During primate and subsequent human evolution, progressively increasing frontoparietal integration became the foundation of exaption (using a device or feature for another purpose) for a more elaborate MNS, one of the functions enabled being the conversion of visual data into knowledge within the general cognitive domains of attention and memory, as well as more specialized domains such as language and tool use [35].

Convergent evidence accumulating from research in archeology, evolutionary neuropsychology, genetics, and linguistics has advanced a hypothesis that working memory may be the "cognitive missing link" that enabled intra-connectivity of the various intelligence domains (social, natural history, technical), with cognitive fluidity and cross-modal connectivity culminating in increased creativity. Working memory may be viewed as a kind of "operating system of our brain" and the "engine of cognitive connectivity" and

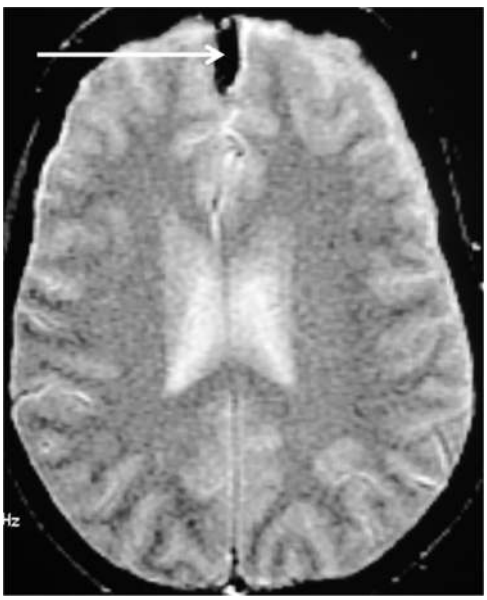

Figure 0.4 Isolated frontopolar lesion (midline dark area, indicated by arrow) due to a discreet brain hemorrhage.

executive function [36]. The working memory circuit and the mirror neuron circuitry are both extensive frontoparietal cerebral circuits and are key circuits that make us human and constitute core frontal systems today. These can both be assessed clinically relatively rapidly by simple bedside tests.

Emotional intelligence (EI) is an important subcomponent of frontal function and has rarely been addressed in neurological patients, including those suffering from TBI, stroke, dementia, and multiple sclerosis. Emerging evidence has indicated that EI is a critical "intelligence" for success, whether intrapersonal, interpersonal, or with career achievements. In analysis of stroke data, EI was found to be negatively affected by diverse brain lesions. However, areas most impactful were frontal, temporal, and subcortical, as well as subtentorial regions [29,37].

At times the heralding of a stroke is nonspecific, such as a seizure or severe headache without discernible neurological deficit. The rapid access to high-volume comprehensive stroke centers in many parts of the world today, with imaging mandatory within 30 minutes, often depicts a stroke that is unexpected or has no obvious clinical accompaniment. Such was the case with a middle-aged, well-educated woman with a very unusual stroke lesion location with a rather unusual cause. The most anterior aspect of her brain, called the frontopolar cortex (FPC), BA 10, was damaged by a ruptured dural arteriovenous malformation (abnormal blood vessel) with discrete hemorrhage measuring 2.3 cm^3. BA 10 is one of the few brain areas that is dramatically enlarged in humans in comparison to the closest extant primate, the chimpanzee. FPC functions arbitrate our most apical human cognitive qualities, such as the simultaneous consideration of diverse options and task switching, elaborated on in more detail in Chapter 11 [38,39]. This study remains, to date, the sole, isolated FPC lesion analysis reported (Figure 0.4). Cognitive testing was normal with the exclusive impairment in EI subtest scores. This medial FPC lesion was

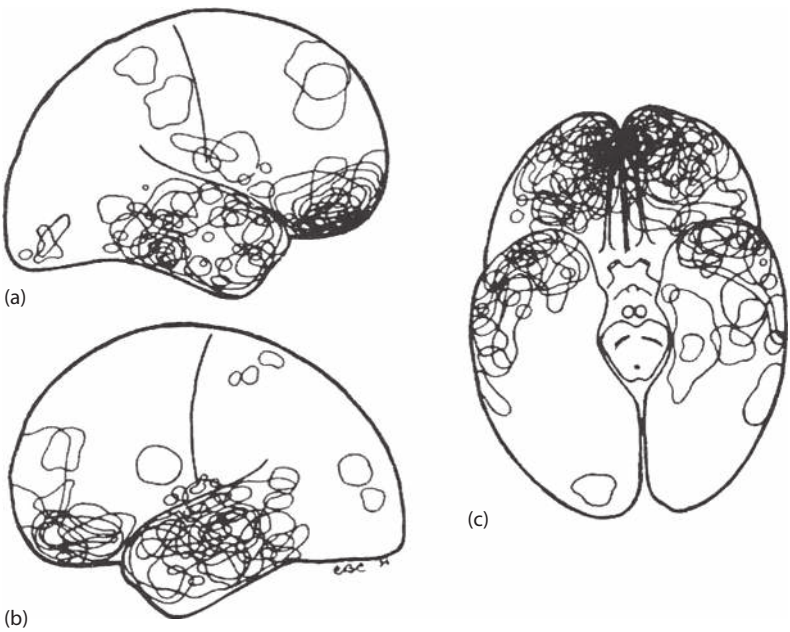

Figure 0.5 Silent neurological lesions, silent neuropsychological lesions, and often silent neuroimaging (unless using DTI, PET, IFC): TBI and the frontotemporal predilection. (a) sagittal right brain; (b) sagittal left brain; (c) horizontal from below. Distribution of contusions in 40 consecutive autopsy cases.

Sources: Courville CB. *Pathology of the Nervous System: Part 4.* Pacific, Mountain View, CA, 1937; Courville CB. *Trauma of the Central Nervous System.* Williams and Wilkins, Baltimore, MD, 1945.

consistent with being a necessary component involved in the emotional processing of internal states of a person [40].

Frontal lobe presentations can be enigmatic, bewildering, covert, and silent. Standard clinical neuroscience texts typically list two dozen or more differing presentations. The first frontotemporal dementia, Pick's disease, was described in 1892 [41]. Although it could have served as a very revealing pathology of differing frontal functions, it was largely ignored, with a possible explanation that it could be the existence of several different types of pathology, in addition to Pick bodies, that cause the so-called frontotemporal lobe dementia (FTD). Pick's disease happened to be associated with very infrequent pathology, while the generic FTD was a common dementia. This pathology–clinical mismatch deprecated FTD and related frontotemporal lobe syndrome (FTS) over the next century, and consequently they were trivialized as clinical syndromes.

TBI syndromes today are reminiscent of the Pick's/FTD debacle. TBI has a predilection for the frontotemporal lobes and associated circuitry (Figure 0.5), and was first described pathologically in 1937 [42] and has been corroborated by neurosurgical studies since. Predictably, they are relatively "blind" to standard neuropsychological testing (NPT) that samples differing brain circuits, and often to neuropsychiatric assessments, including depression. Many seminal neurological cases published in the last 100 years have repeatedly alerted clinicians to the dramatic cognitive–behavioral dissociation after frontal lobe lesions. Frontal lobe patient reports have persistently emphasized the profound behavioral impairments in the context of otherwise normal (NPT) patients in the

last few decades. Arnold Pick, in his landmark description of Pick's disease, also noted presentations principally with inhibition and abulias (poverty of thought, action, and speech), not cognitive impairment as a rule. The commonly used screening tests such, as the MMSE, MOCA, and even those specific to frontal lobe function, such as the Frontal Assessment Battery (FAB), are often within normal range and hence they may miss the entry criteria of NPT [43]. In-depth NPT testing itself is often normal or only mildly impaired. Various degrees of abulia, however, are a common and pervasive accompaniment of frontal and subcortical lesions and may hinder adequate behavioral and NPT testing.

The marked clinical cognitive–behavioral dissonance accounts for some enigmatic justifications offered by patients, such as "driving through a red light is wrong but I may do so." This clinical cognitive–behavioral sundering may also further delay diagnosis because their language skills and cognitive skills are often remarkably convincing. At the cost of possible oversimplification of intricate frontal lobe function, the wide array of frontal behavioral presentations can be understood in terms of two broad categories: abulia (A) and disinhibition (D). For example, the FTD-behavioral variant (bv) syndrome may be divided into FTD(bv)-A and FTD(bv)-D. Abulia, as an overarching deportment, includes episodic dysmemory due to inattention, impaired registration and impaired retrieval, self-neglect, emotional flatness, lack of empathy, stereotyped behavior and ritualistic behavior – humming, hand rubbing, foot tapping, grunting, lip-smacking, clock watching, counting, punding, and feasting on the same foods. Disinhibition, on the other hand, accounts for the syndromes of socially inappropriate behavior, abnormal eating behaviors (eating off other people's plates), and impulsivity. Most, if not all, may originate in the network defraying caused by mirror neuron network disruption and uncoupling.

For too long neuroimaging has been unhelpful. The pervasively vague, polysymptomatic presentations, and borderline NPT results are characteristic of mild TBI patients. Buttressed by the frequently so-called "normal" anatomical brain scans, this generally cements the notion that there is "nothing significantly wrong with the brain." Newer neuroimaging techniques are changing this quagmire. Diffusion tensor imaging (DTI), in particular, has been instrumental in being able to zero-in on the fiber tracts that bear the brunt of the damage, which amount to about 100 000 miles in the human brain. Functional imaging with metabolic positron emission tomography (PET) brain scans often reveal hypometabolic (decreased activity) areas in both frontal and anterior temporal lobes. More promising are the newer MRI-based network scans. Resting-state networks (default mode network, salience network) or intrinsic connectivity network (ICN) imaging have shown a more extensive brain connectomal disruption after TBI. This gels fittingly with the concept of hub vulnerability hypothesis discussed in Chapter 12. In brief, the human connectome (the brain's entire fiber network) has "hotspots" that are susceptible to traumatic, vascular, and metabolic injury. These critically important hubs subserve higher cognitive processes and are the most energy-consuming regions of the brain [44].

Evaluation with the FTS criteria of Rascovsky et al. [45] or the Daphne criteria [46] are important for deficit estimation and to guide further treatment responses. Thereafter, assessment for FTS with behavioral neurological testing – such as the Frontal Behavioral Inventory (FBI), Frontal Systems Behavioral Examination (FRSBE), or Behavior Rating Inventory of Executive Function (BRIEF) – may provide more insightful appreciation of the range, extent, and gravity of the manifold syndromes [16,18,47]. Behavioral

neurological tests that interrogate the inferior frontal, anterior temporal lobes and uncinate fasciculus and frontotemporal circuitry are required for a more representative evaluation of TBI. Pertinent syndromes may include partial or complete forms of the Geschwind-Gastaut and Klüver–Bücy syndromes, not captured by NPT (see Chapter 11). TBI is now seen as a progressive inflammatory, apoptotic, and vascular disease that may progress for months to years [48].

Perhaps the preponderance of the diagnostic challenges may be ascribed to the inherent difficulty pertaining to the assessment of the most intricate aspect of human behavior. Historically, the memory-centric focus associated with Alzheimer's disease likely overshadowed frontotemporal behavioral presentations, where memory is mostly spared. New insights from a pathophysiological point of view inform us that mild and moderate TBI is a chronic inflammatory response with the activation of several inflammatory and apoptotic pathways [49,50]. Recent findings point to a process that may progress and worsen over several years. This is in direct contradistinction to the classic teaching of rapid recovery over weeks to months after "concussion" [51]. Furthermore, it is also a "microvascular disease" process with vasospasm (narrowing of brain arteries) described in the initial phases with neurovascular uncoupling. An alarming 4.43 hazard ratio for developing FTD after TBI has since been reported, and data from a rat model indicate that behavioral impairments are likely due to TDP-43 short fragment accumulation [51].

Why the diagnostic and syndromic concern? With more precise diagnosis, treatment prospects become more discerning and effective. The nature and extent of TBI diagnosis is important in view of emerging treatments that may help our patients. These include computerized exercises such as BrainHQ and Cogmed, which facilitate working memory and attention, pharmacotherapy (amantadine, methylphenidate), and specific attention to an omega 3/6 ratio diet. Lindelov et al. presented important data that hypnosis in TBI improves working memory, fundamental to all other brain functions [52]. Kitagishi et al. showed that supplementation with natural compounds such as dietary fish oil (rich in polyunsaturated fatty acid) induces PTEN expression (activation of peroxisome proliferator-activated receptor). This has a key role in neuroprotection, stimulates cell proliferation, and enhances cell survival [53]. Neuroplasticity-centered treatments are a particularly exciting prospect, with the first positive trial reported during November 2017 by the Helius group on cranial nerve noninvasive neuromodulation with mild-to-moderate TBI in 122 randomized subjects [54,55].

At times subtle or covert frontal syndrome presentations are key to avoiding fulminant irreversible brain disorders. During my first months as a newly graduated neurologist, a call from the emergency room was for evaluation of a middle-aged man whose wife was concerned that on arriving at their long-established home that day, after a routine outing, he stated that their bedroom was unfamiliar to him. Examination revealed a mild pyrexia without other accompanying general medical or neurological examination abnormalities. Magnetic resonance brain scan imaging revealed classic medial temporal and inferior frontal lobe abnormalities, consistent with herpes simplex encephalitis 1 (HSV-1), cerebrospinal fluid (CSF) indicative of viral infection, and subsequent confirmation by brain biopsy. The heralding neurological presentation was a relatively sudden onset of "jamais vu" or feeling of unfamiliarity in relation to his own bedroom. This could also be regarded as a delusional misidentification syndrome for place, as will be discussed in more detail in Chapter 10. HSV-1 presentation is more often fulminant, associated with headache, fever, seizures, and altered mental status. When the presentation is covert,