CHAPTER 1

Validity Assessment in Clinical Settings

How It Differs from Forensic Settings and Why It Is Important

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VALIDITY ASSESSMENT: GROWING FROM FORENSIC TO CLINICAL SETTINGS

Historically, it was believed that nearly all individuals who underwent neuropsychological testing for clinical purposes produced valid data (Green & Merten, 2013; Mittenberg, Patton, Canyock, & Condit, 2002). This was likely because clinicians held a perspective that essentially all clinical patients were motivated to receive accurate and clinically helpful information regarding their health care questions. In contrast, it was known that individuals who underwent neuropsychological testing for forensic purposes had clear external incentives to malinger (e.g., obtain financial compensation or avoid criminal responsibility). As a result, it was thought that production of invalid data on neuropsychological testing was a phenomenon that primarily occurred in forensic evaluations (Merten et al., 2013; Mittenberg et al., 2002). Correspondingly, much of the early research on neuropsychological validity assessment was conducted in forensic settings, by forensic practitioners, and for forensic practitioners (Suchy, 2019). Given the forensic nature of this research, invalidity was largely considered synonymous with malingering, validity tests were sometimes called "malingering tests," and the most commonly used performance validity test (PVT) in North America was even named the Test of Memory Malingering (TOMM; Martin, Schroeder, & Odland, 2015; Mittenberg et al., 2002; Nies & Sweet, 1994; Slick, Tan, Strauss, & Hultsch, 2004).

Over time, research on neuropsychological validity assessment began to increase in both volume and scope (Martin et al., 2015; Suchy, 2019). As noted by Martin et al. (2015), in the mid-1990s, roughly 7% of articles published in two commonly referenced neuropsychology journals, *Archives of Clinical Neuropsychology* and *The Clinical Neuropsychologist,* addressed topics regarding neuropsychological validity assessment. By the mid-2010s, however, roughly 25% of articles investigated topics related to neuropsychological validity assessment (see Figure 1.1). With this expansion in empirical investigation, the focus of the research began to shift from detecting malingering in forensic settings to understanding the effects of performance invalidity more generally, whether in forensic or clinical settings (Suchy, 2019).

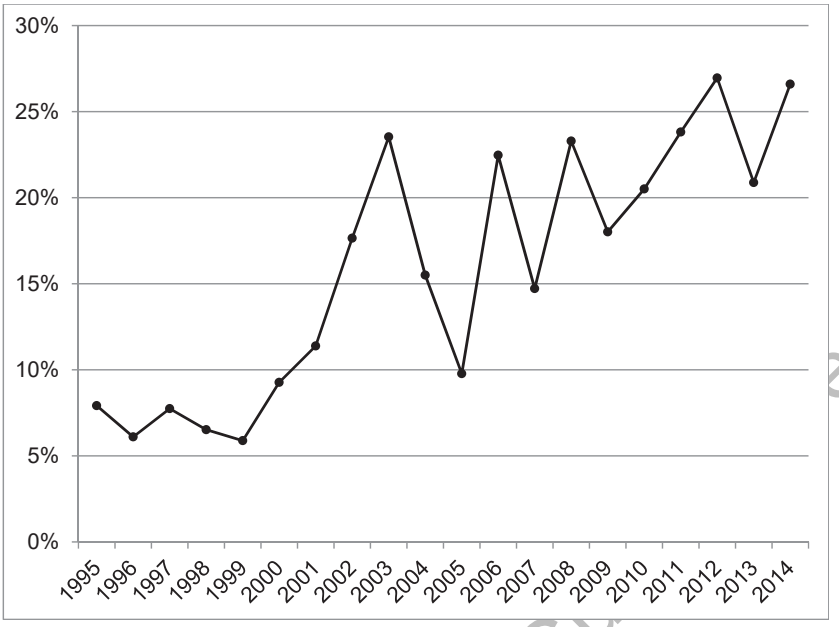

FIGURE 1.1. Average proportion of articles published on neuropsychological validity assessment by year in *The Clinical Neuropsychologist* and *Archives of Clinical Neuropsychology*. Data from Martin, Schroeder, and Odland (2015).

In a seminal study, Mittenberg et al. (2002) documented that invalidity occurred in clinical nonforensic settings, albeit less frequently than in forensic settings. Green, Rohling, Lees-Haley, and Allen (2001) found that when patients completed testing invalidly, the invalidity significantly impacted cognitive test performance, as it explained roughly 50% of the variance in cognitive test scores. This was notably more variance than that accounted for by age (4%), education (11%), Glasgow Coma Scale score (1%), degree of posttraumatic amnesia (1%), and presence of positive neuroimaging findings (<1%). The utility of formal approaches to detect invalidity also became abundantly evident. For example, Larrabee (2003) found that accurate identification of invalidity via concurrent use of multiple validity tests far exceeded the classification accuracy rates achieved by use of clinical judgment alone (see Faust, Hart, & Guilmette, 1988a; Faust, Hart, Guilmette, & Arkes, 1988b; Heaton, Smith, Lehman, & Vogt, 1978). Similarly, Meyers and Volbrecht (2003) demonstrated that use of multiple validity tests resulted in strong classification accuracy rates, even in nonforensic clinical samples.

As a result of the expanded literature base, it became clear that validity assessment should be considered a critical and core component of *all* neuropsychological evaluations, a perspective that was adopted and documented by several well-cited professional position papers. With regard to incorporating validity testing in clinical evaluations, specifically, the National Academy of Neuropsychology released a validity testing position paper in 2005, emphasizing that "adequate assessment of response validity is essential in order to maximize confidence in the results of neurocognitive and personality measures and in the diagnoses and recommendations that are based on the results" (Bush et al., 2005, p. 419). The position paper further elaborated that "assessment of response validity, as a component of a medically necessary evaluation, is medically necessary" (p. 419).

Two years later, the American Academy of Clinical Neuropsychology (AACN) released practice guidelines for neuropsychological assessment and consultation (Board of Directors, 2007). Within these practice guidelines, it was specified that "the assessment of effort and motivation is important in any clinical setting, as a patient's effort may be compromised even in the absence of any potential or active litigation, compensation, or financial incentives" (p. 221). In 2009, the AACN released a consensus statement focused on the use of neuropsychological validity testing (Heilbronner et al., 2009) and stated that "response bias may occur in routine clinical and medical referrals, when no forensic context is evident" and "when clinicians are evaluating a (nonforensic) patient who by virtue of claimed injuries is reasonably likely to become a litigant or claimant, the clinician should consider the increased risk of insufficient effort and response bias" (pp. 1105–1106).

Organizations outside of neuropsychology also began to emphasize the need for validity assessment in neuropsychological evaluations. For example, the American Medical Association's guides to the evaluation of permanent impairment (American Medical Association, 2008) stated that "it is standard practice that a neuropsychological test battery should include instruments that include . . . validity tests" (p. 351). The Institute of Medicine of the National Academies (Institute of Medicine, 2015), a nonprofit institution that provides independent objective analysis to inform public policy decisions, declared that "it is important to include an assessment of performance validity at the time cognitive testing is administered" (p. 202). It was further stated that "all cognitive evaluations should include a statement of evidence of the validity of the results" (p. 203).

By virtue of the robust literature base and ensuing organizational practice recommendations, most neuropsychologists are now aware that invalid test performance and symptom report can occur in many contexts, even within routine clinical contexts in which external incentives to underperform are absent (Martin et al., 2015; Martin & Schroeder, 2020; Sweet, Benson, Nelson, & Moberg, 2015). This is a particularly important concept to appreciate given that neuropsychologists evaluate cognitive and emotional statuses (Board of Directors, 2007), aspects of human functioning that cannot be quantified by methods such as laboratory values, neuroimaging findings, or electrophysiological procedures—tests that are relatively impervious to patient behavior (Schroeder, Martin, & Walling, 2019). Assessment of cognitive and emotional capabilities relies heavily on patient engagement, motivation, and cooperation (Bianchini, Mathias, & Greve, 2001). Thus, if a patient provides suboptimal engagement, motivation, or cooperation, or produces test data or symptom report with the intent to deceive, his or her cognitive and emotional functioning cannot be accurately captured. If a clinical patient's inaccurate results are erroneously identified as being accurate, the neuropsychologist could inadvertently harm the patient by (1) providing inaccurate and emotionally impactful diagnoses (e.g., telling a patient that he or she has significant persistent cognitive deficits or even a neurodegenerative condition), (2) reinforcing noncredible symptoms, (3) recommending unnecessary and potentially costly additional workup (e.g., magnetic resonance imaging [MRI] of the brain), (4) recommending unnecessary and potentially harmful treatment (e.g., unneeded medication prescription), and/or (5) recommending restrictions to daily living activities and/or independence (e.g., stopping driving or moving from independent living to assisted/sheltered living). As demonstrated by this historical review, neuropsychological validity assessment has clearly evolved over time, and neuropsychological validity tests are no longer viewed as simply being tools for detecting malingering, primarily in forensic settings. Rather, validity assessment is now viewed as a means to ensure accuracy of neuropsychological test data regardless of cause of invalidity and

clinical setting, a sentiment that is reinforced by the updated AACN validity assessment consensus statement (Sweet et al., 2021).

DIFFERENCES IN VALIDITY ASSESSMENT BETWEEN CLINICAL AND FORENSIC EVALUATIONS

As previously described, it is now clear that validity assessment should be incorporated within all neuropsychological evaluations, including clinical nonforensic evaluations. On the surface, addressing validity issues in nonforensic evaluations might seem to be a straightforward task given that a significant amount of literature has amassed on validity assessment in forensic settings, and the basic elements of a neuropsychological evaluation are largely the same in clinical and forensic evaluations (see Table 1.1). When attempting to apply the large forensic literature base to clinical settings, however, it becomes obvious that there are differences in assessing validity status, interpreting and documenting validity test results, and providing feedback to others when validity tests are failed. In order to understand the differences in validity assessment that are related to practice setting, it is imperative that neuropsychologists be aware of the core distinctions between clinical and forensic evaluations, a topic that we discuss next.

As there is not yet a consensus-achieved, formal definition of what constitutes a clinical (as opposed to forensic) neuropsychological evaluation, we provide the following operational definition of a *clinical neuropsychological evaluation,* which is based on previously published descriptions of services provided (i.e., Binder, 2019; Donders, 2016; Sweet, Kaufmann, Ecklund-Johnson, & Malina, 2018). We define a clinical neuropsychological evaluation as an evaluation where a neuropsychologist provides health care services to a patient who is seeking treatment for a malady. In providing this evaluation, the neuropsychologist is acting as a *treating doctor* (a term applied regardless of whether assessment or intervention is directly provided) and entering into a patient–doctor relationship.

To highlight the distinguishing features of the clinical neuropsychological evaluation, we unpackage the aforementioned definition. First, because a neuropsychologist

TABLE 1.1. Basic Elements of Neuropsychological Evaluations

	Clinical evaluations	Forensic evaluations
Accept referral	Yes	Yes
Review records	Yes	Yes
Obtain informed consent	Yes	Yes
Conduct clinical interview	Yes	Yes
Complete testing	Yes	Yes
Interpret data	Yes	Yes
Reach conclusions	Yes	Yes
Write report	Yes	Yes
Provide feedback to patient	Yes	No

provides health care services to a patient, that neuropsychologist is operating within a health care system. As such, other health care providers often serve as the referral source, referring individuals with diverse clinical conditions, including significantly impactful conditions such as dementia (Sweet et al., 2015). The neuropsychologist evaluates and/or treats these patients, typically billing medical insurance for rendered services (Donders, 2016; Sweet et al., 2018). By billing medical insurance, the neuropsychologist agrees to the stipulations set forth by insurance companies, which includes time-limit restrictions on how much testing can be completed (Lamberty, 2012). Accordingly, survey data show that reimbursement factors and evaluation context (i.e., clinical or forensic evaluation) are both cited by neuropsychologists as factors that impact length of the neuropsychological evaluation (Sweet et al., 2015).

Second, because neuropsychologists conducting clinical evaluations are acting as a treating doctor by entering into a patient–doctor relationship, it is expected that the exam is designed to provide information that will clinically benefit the patient (Binder, 2019). The neuropsychologist typically provides a clinical opinion to the patient (Binder, 2019), acts as an advocate for the patient (Donders, 2016), and attempts to minimize harm to the patient (Binder, 2019). Additionally, within clinical evaluations, it is generally understood that confidentiality is protected except under special circumstances (Donders, 2016).

Whereas operating as a treating doctor and entering into a patient–doctor relationship are key features of the clinical neuropsychological evaluation, such characteristics are incongruent with accepted definitions and principles of forensic practice. According to the American Psychological Association (2013), a forensic evaluation is one in which the psychologist applies scientific, technical, or specialized knowledge of psychology to the law to assist in addressing legal, contractual, and/or administrative matters. Stated more simply, a forensic neuropsychological evaluation is one in which neuropsychologists apply neuropsychological knowledge and facts to answer legal questions (Larrabee, 2012; Greiffenstein & Kaufmann, 2018).

The aforementioned definition of a forensic neuropsychological evaluation can be unpackaged to show how it differs from a clinical neuropsychological evaluation. Because forensic neuropsychologists apply knowledge to the law to answer legal questions, attorneys or administrative professionals often serve as the referral source (Sweet et al., 2018). Forensically referred examinees often have relatively restricted causes of their cognitive complaints, with trauma-based conditions, such as traumatic brain injury, being frequently claimed (Sweet et al., 2015). Because the goal of the evaluation is to answer legal questions, the neuropsychologist is expected to provide an impartial impression in which considerations of accuracy clearly trump those of examinee well-being (Sweet et al., 2018).

Because, within a forensic evaluation, the neuropsychologist does not practice within a typical health care system or provide health care services to the examinee, the neuropsychologist does not form a doctor–patient relationship with the examinee (Binder, 2019). Additionally, the neuropsychologist does not bill medical insurance; therefore, the neuropsychologist is not confined to the limits imposed by insurance reimbursement policies (Lamberty, 2012). Furthermore, the neuropsychologist has no obligation to help the examinee and is not an advocate for the examinee; instead, he or she is an advocate for the truth (Binder, 2019). Because it is known that the "truth" is often exaggerated or feigned within forensic settings (Larrabee, Millis, & Meyers, 2009; Mittenberg et al., 2002), the neuropsychologist might be inclined to view the examinee more skeptically than in clinical settings (Bush & Heilbronner, 2012). Finally, because healthcare laws are

not pertinent in forensic evaluations and the results of the evaluation will become part of a public record, there is not the same legal mandate to protect an examinee's confidentiality as there is within the clinical evaluation (Sweet et al., 2018).

The key differences that distinguish clinical from forensic neuropsychological evaluations (as just described and as summarized in Table 1.2) translate into important differences in validity assessment practices, which are described in Table 1.3. In addition to recognizing these individual differences, one should also recognize that clinical and forensic neuropsychologists are likely to proceed with the evaluation in different manners given the different factors/goals inherent within the evaluations. Specifically, while both types of neuropsychologists should recognize that the consequence of validity test failure is the same (i.e., do not interpret neuropsychological test data as accurate), the resultant interpretive question will likely be different. In forensic settings, the question is often "What does this say about the examinee and his or her claims?" In clinical settings, however, the operative question is likely more appropriately "How can I provide useful clinical services when I have invalid test data?" Given the many differences in validity assessment practices and interpretive questions, it should be understood that it is not always possible or appropriate to generalize research findings, practice recommendations, or guidelines from forensic literature to practice in the clinical nonforensic setting.

Because of issues in generalizing forensic literature to nonforensic cases, and because there is limited guidance in addressing methods and approaches to validity assessment in clinical settings, managing invalidity in nonforensic settings is arguably more challenging than managing invalidity in forensic settings. In this book, therefore, we aim to thoroughly examine the validity assessment literature through the lens of clinical practice in order to provide guidance and resources for utilizing and addressing validity assessment in nonforensic settings. Chapters 6–12 provide resources on how to conduct ethical, efficient, and accurate validity assessments in clinical settings. Guidance on how to move forward when PVTs are failed, including ways to conceptualize invalidity in clinical settings (Chapters 2–4), provide feedback when testing is invalid (Chapter 4), and write clinically useful reports when testing is invalid (Chapter 5) are also provided. Discussions of validity assessment in specific clinical settings and contexts are also provided in the

TABLE 1.2. Key Factors Differentiating Clinical and Forensic Neuropsychological Evaluations

Clinical evaluations	Forensic evaluations
Doctor–patient relationship	No doctor–patient relationship
Address health care questions	Address legal questions
Advocate for patient	Advocate for truth
Goal: objectivity, accuracy, and patient well-being	Goal: objectivity and accuracy
Notable limits on amount of time to conduct evaluations	Minimal limits on amount of time to conduct evaluation
Invalidity is often less common	Invalidity is often more common
Patient obtains results	Third parties obtain results
Confidentiality strongly protected	Less confidentiality

TABLE 1.3. Examples of How Key Differences between Clinical and Forensic Evaluations Impact Validity Assessment

1. Time to review records to ensure accurate report of history and symptoms is less in clinical settings.
2. Clinical patients present with highly diverse diagnostic conditions, including dementia, which can impact validity assessment.
3. Time-efficient validity assessment is more necessary in clinical settings than in forensic settings.
4. Differing base rates of invalidity impact interpretation of PVTs via influence of positive and negative predictive power.
5. Base rates of causes of invalidity in each setting can impact conclusions drawn regarding reasons for invalid test findings.
6. There is a need to balance validity considerations with treatment recommendations in clinical settings.
7. Report-writing goals and/or styles vary based on differences in time limits and expectations inherent in each setting.
8. Feedback is provided to the patient in clinical settings versus a third party in forensic settings.

last section of this book (Chapters 13–22). Finally, Chapter 23 includes information on conceptualizing legal matters and responding to queries when clinical cases turn forensic in nature.

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CHAPTER 2

Explanations of Performance Validity Test Failure in Clinical Settings

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Although performance validity test (PVT) failure in forensic evaluations is predominately believed to be the result of malingering, surveyed neuropsychologists report believing that a variety of conditions and factors can cause PVT failure during clinical evaluations (Martin, Schroeder, & Odland, 2015). In this chapter, we discuss the empirical evidence on conditions and factors that are sometimes used to explain PVT failure, first briefly describing common mechanisms underlying many of the conditions and factors. In describing these mechanisms, we also provide definitions for key terminology, including external and internal incentive, conscious and subconscious deception, and effort.

COMMON MECHANISMS UNDERLYING VARIOUS PVT FAILURE EXPLANATIONS

The term *external incentive* is often used to describe a motivation driven by external or material factors rather than internal/psychological factors. External incentives can be related to obtaining items or resources such as financial compensation, drugs, or academic accommodations. External incentives can also be related to avoiding punishment or negatively perceived responsibilities such as criminal responsibility, military duty, or work responsibilities. Conversely, the term *internal incentive* is often used to describe a motivation driven by internal/psychological factors. Internal incentives can be related to factors such as gaining nurturance, sympathy, or attention. Additionally, internal incentives can be related to obtaining relief from unpleasant psychological states such as guilt, anxiety, or internal conflicts.

Conscious deception is a behavior that is willfully or deliberately carried out with a goal to deceive. This construct has also been referred to as *other-deception* (Boone, 2007), as individuals engaging in conscious deception are believed to be doing so to deceive others as opposed to deceiving themselves. Conversely, *subconscious deception* is a deceptive behavior that is subconsciously or unintentionally carried out. This construct has also been referred to as *self-deception* (Boone, 2007), as individuals engaging

in subconscious deception are believed to be doing so to deceive themselves as opposed to deceiving others. An example of this would be when a person subconsciously converts emotional conflict into somatic symptoms to relieve psychological distress, a process that is thought to underlie certain somatoform disorders.

Incentives and deception interact. Conscious deception can be related to obtaining external incentives (resulting in malingering) or internal incentives (resulting in factitious disorder), while subconscious deception is thought to be solely motivated by desires for internal incentives (resulting in somatoform disorders). While malingering, factitious disorder, and somatoform disorders are distinctly different and arise from different combinations of deception and incentive types, it is important to note that these excessive illness behaviors are not mutually exclusive. For example, a person who has a somatoform disorder could become involved in an accident and exaggerate or malinger deficits secondary to that accident while still having symptoms from the somatoform disorder. Likewise, a person who has a factitious disorder could become involved in an accident and exaggerate or malinger deficits secondary to that accident while still carrying out behaviors linked to the factitious disorder.

While external or internal incentives and conscious or subconscious deception can sometimes drive a patient to fail PVTs, not all patients failing PVTs do so because of these mechanisms. Some patients might, instead, simply be unwilling to provide their best performance to such an extent that their underperformance results in PVT failure. When this occurs, it can be stated that the patient failed PVTs as a result of poor or suboptimal effort. However, *effort* requires defining, as the term has been (and is sometimes still) used inappropriately when applied to performance validity testing.

By definition, *effort* is a construct that indicates degree of physical or mental exertion. While some patients might fail PVTs due to a poor expenditure of mental exertion, others might expend mental exertion in an attempt to look impaired, particularly when deception and external incentives are involved. Boone (2013) cautions against conceptualizing all cases of PVT failure as a result of poor effort in forensic cases, noting that "in the context of litigated cases, lack of interest or investment in the testing is not the operative issue; rather the test taker is typically expending *considerable* effort to pretend to have symptoms that are not real" (p. 23). This same point holds true in clinical cases, particularly when external incentives are present. Additionally, as discussed in Larrabee (2012), PVTs should not be conceptualized as measures of effort given that many patients can still pass PVTs while exerting suboptimal effort due to the relatively minimal cognitive demand required to pass PVTs. Furthermore, examinees can fail PVTs for reasons other than poor effort, and examinees who provide poor effort do not always fail PVTs. In other words, poor effort is neither necessary nor sufficient for PVT failure, which implies that PVT failure should not automatically be explained in terms of poor effort, inadequate effort, variable effort, or suboptimal effort. Rather, such terms should be reserved for only those cases for which there is sufficient reason to believe that poor effort was indeed the mechanism by which PVTs were failed.

EXPLANATIONS SOMETIMES PROVIDED FOR PVT FAILURES

A variety of factors are used by clinicians to explain PVT failure (Martin et al., 2015), although not all of these are consistent with findings from the empirical literature. In this section, we discuss the empirical literature (or the lack therefore) in support of multiple explanations that are sometimes provided for PVT failures.

Genuine Cognitive Dysfunction

The basic tenet of PVT development is that the tests/indices should identify invalid test taking when present (i.e., they should be sensitive to invalidity) while also being largely unaffected by genuine cognitive dysfunction (i.e., they should be highly specific to invalidity). However, as is the case with diagnostic testing in general, there is a trade-off between sensitivity and specificity. Unless a test is perfectly accurate 100% of the time, the test can never be 100% sensitive while also being 100% specific. Despite PVTs being well-validated and robust detectors of invalidity, they are not, and will never be, 100% sensitive and specific at the same time. Because of this, and because a false-positive finding of invalidity is often considered more unacceptable than a false-negative finding, PVT cutoffs are usually set to maintain specificity rates at 90% or higher (Schroeder, Boone, & Larrabee, 2021), which allows for a false-positive rate of no more than 10%.

Given typically applied standards in PVT validation research, cutoff points have been empirically identified for most PVTs to allow sensitivity to be maximized while maintaining specificity at 90% or above (see Chapter 8, this volume). When false-positive findings occur due to genuine cognitive dysfunction, they generally occur in patients with clinical histories of substantial neurocognitive compromise such as having (1) dementia-level cognitive impairment that leads to dysfunction in carrying out instrumental activities of daily living; (2) intellectual disability; (3) frequent epileptic seizures, with seizure activity occurring in close proximity of neuropsychological testing; or (4) large and clearly identifiable cerebrovascular accidents (Drane et al., 2016; Martin, Schroeder, & Olsen, 2020a; Meyers & Volbrecht, 2003; Smith et al., 2014).

Research by Meyers and Volbrecht (2003) demonstrates the aforementioned point well. The authors examined nine PVTs in a large sample of patients divided by diagnoses, functional status, and litigation involvement. Nonlitigating patients with various severities of traumatic brain injury (TBI), ranging from those with mild injuries to those with severe injuries, did not fail PVTs at rates greater than 10%. Likewise, nonlitigating patients with chronic pain or depression that was sufficient enough to lead to hospitalization or partial hospitalization did not fail PVTs at rates greater than 10%. However, nonlitigating patients who were institutionalized due to cognitive impairment or patients who had substantial cognitive impairment due to those factors listed in the previous paragraph produced false-positive findings in excess of 10%.

Somewhat similarly, Martin, Schroeder, and Olsen (2020) examined a group of older patients who did not have external incentives to underperform on testing. They found that older patients with no cognitive impairment and older patients with mild cognitive impairment typically passed PVTs at generally recommended cutoff points. Conversely, older patients who met diagnostic criteria for major neurocognitive disorder/dementia often failed PVTs at generally recommended cutoffs, which necessitated altering the cutoffs.

Finally, the same trends can be seen when examining systematic reviews of Reliable Digit Span (RDS) and the Test of Memory Malingering (TOMM), the most commonly utilized embedded and stand-alone PVTs, respectively (Martin et al., 2015). In their systematic review of RDS, Schroeder et al. (2012) found that validly performing individuals with mild TBIs, moderate-to-severe TBIs, chronic pain, attention-deficit disorder, psychiatric disorders, and other conditions typically passed RDS at specificity rates of 90% or above. Conversely, adequate specificity was not consistently demonstrated in groups of patients with severe memory disorders/dementia, low intellectual functioning/intellectual disability, or cerebrovascular accidents. Similarly, when considering studies of the

TOMM examined in Martin et al.'s (2020b) systematic review, validly performing individuals with psychiatric conditions, mild TBIs, moderate-to-severe TBIs, chronic pain, attention-deficit/hyperactivity disorder, substance abuse, toxic exposure, and other conditions typically passed the TOMM at specificity rates of 90% or above. Even patients with mild intellectual disability generally passed Trial 2 and Retention of the TOMM with specificity values of 88% and above across reviewed studies. The only clinical condition in which TOMM specificity rates consistently fell below 90% was dementia.

In summary, empirical research indicates that most patients do not fail well-validated PVTs due to cognitive dysfunction so long as there is not *substantial* cognitive impairment. If a patient is at risk for a false-positive PVT failure due to significant cognitive dysfunction, there should generally be evidence that his or her cognitive deficits negatively impact independence in activities of daily living. This significant level of impairment should also typically be apparent to the neuropsychologist after completing the clinical interview. In such cases, the clinician can adjust PVT selection or PVT cutoffs to account for the substantial cognitive impairment that is present (e.g., see Chapter 14, this volume, for discussion of PVTs validated in dementia samples).

Somatoform Disorders

Somatoform disorders are frequently suspected to be a cause of invalidity in nonforensic settings (Martin et al., 2015; Schroeder, Martin, & Odland, 2016). In fact, neuropsychologists have ranked somatoform/conversion disorder as likely being the second most common cause of invalidity in nonforensic settings, while validity testing experts have ranked it as being the first most common cause. These beliefs do not seem unreasonable when it is understood that (1) the prevalence of individuals with somatoform symptoms is relatively high, especially within subsamples of patients who present to specialty clinics, (2) individuals with medically unexplained conditions, including those with somatoform disorders, often report having cognitive symptoms, and (3) individuals with somatoform conditions can be viewed as inherently engaging in deception, albeit self-deception (Boone, 2017; Nimnuan, Hotopf, & Wessely, 2001).

In Chapter 19 of this volume, Graver and Boone comprehensively review the literature on somatoform disorders and PVT performance. As their review indicates, there is some evidence that processing speed and motor–sensory PVTs might be failed at relatively high rates by individuals with somatoform conditions. At the same time, Graver and Boone discuss that somatization is not always the operative cause of PVT failure in invalidly performing patients with somatoform conditions. Research indicates that patients with somatoform presentations have higher rates of unemployment (29 vs. 15%) and occupational impairment (55 vs. 14%) than individuals with true medical conditions (Harris, Orav, Bates, & Barsky, 2009), which implies that individuals with somatoform disorders might be more likely to pursue disability. Thus, some individuals with somatoform conditions might be influenced by both subconsciously driven internal incentives and consciously realized external incentives. Indeed, Graver and Boone report rates of validity test failure on memory-based PVTs to be much higher in patients with somatoform presentations who also had external incentives (24 to 56%) versus those without (~10%). In summary, while individuals with somatoform disorders sometimes fail processing speed and motor–sensory based PVTs at relatively high rates, the possibility of comorbid malingering must be considered, especially when multiple additional PVTs are failed.

Depression and Anxiety

In Chapter 17 of this volume, Marshall and Schroeder comprehensively review the literature on psychiatric conditions and PVT performance. Across studies examining validly performing patients with depression or anxiety, traditional PVTs were found to maintain at least 90% specificity in every instance. Low PVT failure rates were even observed in studies that comprised individuals with significant psychopathology: Individuals in one study had *minimum* Beck Depression Inventory–II scores of 30 (i.e., Yanez, Fremouw, Tennant, Strunk, & Coker, 2006); those in another study were psychiatric inpatients with severe major depression (i.e., Guilmette, Hart, Giuliano, & Leininger, 1994); and individuals in yet another study were psychiatric inpatients, many of whom required prolonged hospital stays (>6 weeks) and/or electroconvulsive therapy (i.e., Rees, Tombaugh, & Boulay, 2001). Thus, while there might be very rare exceptions, which should be clinically obvious even before testing begins (e.g., patients who have such severe vegetative symptoms that they are unable to engage meaningfully in the evaluation at all), there is no empirical evidence to indicate that cognitive, emotional, or behavioral dysfunction caused by depression or anxiety increases risk for PVT failure. As stated by Green and Merten (2013), if examinees with depression or anxiety fail PVTs, tests that are typically failed only by individuals with severe neurocognitive conditions such as dementia, then causes other than the psychiatric condition should be suspected.

Apathy

Apathy is a significant loss of interest or motivation, often associated with psychiatric or neurological conditions (Levy & Dubois, 2006). Given the reduction of self-generated behaviors that occur with apathy, it has been argued that patients with apathy might be unable to invest sufficient interest or effort into neuropsychological testing, which could cause them to fail PVTs despite attempting to perform at their best ability (Bigler, 2015). Only a few studies have examined this hypothesis. While not a primary objective of Paul and colleagues' (2017) study, the authors found that apathy scores were not significantly different between patients with human immunodeficiency virus (HIV) who passed versus failed TOMM Trial 1. Likewise, in a study of patients with Parkinson's disease (Martínez-Horta, Pagonabarraga, de Bobadilla, García-Sánchez, & Kulisevsky, 2013), there were no significant differences on TOMM Trial 1 between individuals with apathy (mean TOMM Trial 1 = 46.6) and those without apathy (mean TOMM Trial 1 = 47.0). In a more comprehensive study of apathy and performance validity, Dandachi-FitzGerald, Duits, Leentjens, Verhey, and Ponds (2020) examined individuals with Parkinson's disease, mild cognitive impairment, or dementia. The authors found that PVT failure was associated with cognitive impairment—likely due to the inclusion of patients with dementia—but not with degree of apathy. The authors concluded that "our findings are not supportive of the notion that apathy might lead to an increased risk of false-positive classification of PVTs" (p. 318). In summary, findings from the limited literature base, overall, indicate that apathy is not an expected cause of PVT failure.

Attitude toward Testing

Examinees can sometimes approach the neuropsychological evaluation with a test-taking attitude that interferes with the collection of valid data. Specifically, a patient might be

either negatively engaged in testing (e.g., being intentionally uncooperative or obstructive) or minimally engaged in testing (e.g., responding carelessly or without strong effort). These problematic attitudes might be due to personality issues such as Cluster B personality traits, diagnosable behavioral conditions such as oppositional defiant disorder, a simple lack of desire to participate in the evaluation, or other causes.

With regard to empirical literature on this topic, possibly the greatest evidence that test-taking attitudes can sometimes cause PVT failure comes from studies that have examined PVT performance by healthy research volunteers instructed to perform their best. Multiple studies have demonstrated that at least some healthy undergraduate research participants fail one or more PVTs (An, Kaploun, Erdodi, & Abeare, 2017; DeRight & Jorgensen, 2015; Ross et al., 2016), although failure rates (4–56%) have been noted to vary substantially, likely due to differences in study design (An et al., 2017). In hypothesizing that this heterogeneity in PVT failure rates was due to PVT type and level of cutoff stringency, An et al. (2017) examined failure rates in a sample of 120 undergraduate research participants according to whether PVTs were stand-alone or embedded, and whether liberal or conservative cutoffs were applied. Participants were administered seven PVTs (four stand-alone and three embedded) over an approximate 2-hour test battery. When using liberal PVT cutoffs, they found that 10% of participants failed at least one stand-alone PVT, and 31% failed at least one embedded PVT. When utilizing a cutoff of two or more PVT failures out of seven, a validity testing standard more generalizable to clinical practice, it was found that 7% of the sample met this criterion for invalidity. When using conservative PVT cutoffs, the authors found that 3% of participants failed at least one stand-alone PVT, and 16% failed at least one embedded PVT. When using a cutoff of two or more PVT failures (using conservative cutoff values), it was found that 2.5% of the sample met this criterion for invalidity. In summary, this study indicated that 2.5–7% of healthy undergraduate research volunteers would be classified as invalidly performing when using a criterion of two or more PVT failures.

The design of the undergraduate studies would suggest that the volunteers were not motivated by an external incentive to underperform, and given that the students were cognitively healthy, they certainly did not have severe cognitive impairment that might plausibly cause PVT failure. At the same time, the healthy research controls did not have an incentive to perform to their maximum cognitive ability in most cases, other than to be compliant with research instructions. If anything, the design of these studies might encourage some participants to sacrifice performance accuracy in order to complete testing as quickly as possible given the lack of incentive to do well while being asked to devote hours of time to participate. In support of this hypothesis, DeRight and Jorgensen (2015) asked undergraduate research participants to rate the degree of effort expended after completing a brief neuropsychological battery of PVTs, and the authors found that those self-reporting a lower degree of effort were both more likely to fail PVTs and to complete the battery in significantly less time than those reporting good effort. In summary, given the absence of other plausible alternative explanations for invalid performance, it would seem that a minimal motivation to do one's best is the most parsimonious explanation as to why some undergraduate research participants fail PVTs.

Survey data also provide support that attitude toward testing can cause invalidity in clinical evaluations. In nonforensic settings, North American neuropsychologists, overall, reported that they believe attitude toward testing is the third most common cause of invalidity, and validity testing experts reported that they believe attitude toward testing is the second most common cause of invalidity (Martin et al., 2015; Schroeder et al., 2016). In an even more recent survey, neuropsychologists estimated that clinical patients with

oppositional attitudes toward testing produce invalid data 37.5% of the time (Martin & Schroeder, 2020). Thus, in summary, data from healthy research samples, along with clinical experience as documented through survey data, indicate that attitude toward testing can cause PVT failure, especially in nonforensic settings in which external incentives and desire to complete testing might be less motivating.

A Cry for Help

It has previously been suggested that some patients might exaggerate their symptoms to ensure that clinicians know that they are in distress and in need of help (Dahlstrom, Welsch, & Dahlstrom, 1972). This theory, often termed "cry for help," deserves discussion given that it has frequently been used to explain elevations on the Minnesota Multiphasic Personality Inventory—2nd Edition (MMPI-2) "F" scale (Boone, 2013; Graham, 2006). However, the legitimacy of the theory has been questioned given that research indicates patients identified as overreporting are less likely to complete treatment than individuals responding validity to the MMPI-2 (Greene, 1988).

Neuropsychological research also suggests that patients typically do not fail PVTs as a "cry for help." Moore et al. (2013) conducted a study in which they found that the Repeatable Battery for the Assessment of Neuropsychological Status Effort Index (EI) predicted which examinees with schizophrenia/schizoaffective disorder had low versus high psychosocial treatment adherence. As a group, individuals with EI scores suggestive of invalidity had poorer psychosocial treatment adherence, whereas individuals with EI scores suggestive of valid test-taking performance had stronger psychosocial treatment adherence. Other variables of interest, which included degree of cognitive dysfunction and degree of psychopathology, did not predict treatment adherence.

In another study, Goedendorp, van der Werf, Bleijenberg, Tummers, and Knoop (2013) offered cognitive-behavioral therapy to individuals with chronic fatigue syndrome. The authors found that those individuals who failed the Amsterdam Short-Term Memory Test (ASTM; a freestanding PVT), were significantly more likely to drop out of the study than those who passed the ASTM. Finally, in a third study on the topic, Jurick et al. (2020) examined whether veterans with posttraumatic stress disorder (PTSD) and mild to moderate TBI who failed at least one of three PVTs would be more likely to terminate, or show less benefit from, psychological treatment than those passing all PVTs. Unlike the previously cited studies, the authors did not find significant differences in treatment completion/adherence rates between those patients who failed versus those who passed PVTs. The authors did find, however, that individuals passing PVTs reported greater reductions in their PTSD symptoms than individuals who failed at least one PVT. It is not entirely clear why this finding was observed, but one might speculate that those patients who failed PVTs intentionally reported a greater degree of ongoing symptoms in an attempt to receive increased Veterans Affairs service-connected disability ratings.

In summary, there is no empirical research supporting the "cry for help" theory as an explanation for PVT failure. Conversely, the available research suggests that, overall, individuals who fail PVTs are less likely to complete treatment than individuals who pass PVTs.

Physical Factors

In the context of explanations for PVT failure, we refer to pain, medication effects, and daytime sleepiness/fatigue as physical factors. Of these, the impact of pain on PVT

performance has been most thoroughly examined in neuropsychological research. Overall, this research indicates that pain does not cause high PVT failure rates so long as incentives to underperform on testing are not present. For example, in a study of examinees with chronic pain, Gervais and colleagues (2001) found that 35% of examinees with fibromyalgia failed at least one of two freestanding PVTs when external incentives were present; however, only 4% failed either of the PVTs when external incentives were absent. Similarly, Greve et al. (2010) found that examinees with chronic pain rarely fail RDS when external incentives were absent. Likewise, when malingering is excluded, it has been found that patients with chronic pain pass the TOMM and Portland Digit Recognition Test with specificity rates well above 90% (Greve, Bianchini, Etherton, Ord, & Curtis, 2009; Iverson, Page, Koehler, Shojania, & Badii, 2007). This is also true in acute pain, as demonstrated by findings that research volunteers subjected to cold-pressor-induced pain pass both freestanding and embedded PVTs with specificity rates above 90% (Etherton, Bianchini, Ciota, & Greve, 2005a; Etherton, Bianchini, Greve, & Ciota, 2005b).

Another physical factor that has received some, but less, research attention is the impact of medication effects on PVT performance. This topic was first empirically studied by Loring and colleagues (2011) in an article examining the effects of lorazepam on Word Memory Test (WMT) performance. Specifically, they examined how 28 healthy participants who were administered 2 mg of oral lorazepam performed on the WMT. They found that six of 28 individuals (i.e., 21%) failed the WMT when administered lorazepam. Loring and colleagues suggested that acute administration of lorazepam might have interfered with encoding of information, possibly by altering levels of attention or alertness.

Green and Merten (2013) critiqued the Loring et al. (2011) study and disagreed with their conclusions. Specifically, they noted that two individuals in the study failed the WMT validity indices when administered the placebo (which, obviously, should not impact cognition), with one of these individuals later dropping out of the study. Furthermore, they noted that three of the individuals who failed the WMT did not produce a genuine memory impairment profile, which suggests that cognitive impairment from lorazepam was not the cause of the PVT failure. Hence, they concluded that the most reasonable explanation for the WMT failures was that the individuals were "simply not as motivated to do well" (p. 84).

Like Green and Merten (2013), Rohling (2013) opined that the WMT failures were due not to medication effects but to poor effort. Specifically, Rohling reanalyzed the original Loring et al. (2011) dataset, but in this case, he examined multiple additional indicators of invalidity, including excessive variability in performances, Medical Symptom Validity Test (MSVT) performance, and embedded PVTs derived from study data (for a critique of this analysis and study outcome, see Loring, Meador, & Goldstein, 2020). Additionally, Rohling (2013) examined indications of invalidity not only following the lorazepam and placebo conditions, but also at baseline testing to help ensure that PVT failure was the result of the experimental manipulation (i.e., lorazepam). In doing so, Rohling found that 11 of 28 individuals demonstrated evidence of invalidity. He concluded that

> the current reanalysis of their data shows that invalid test scores from 40% of (subjects) were obtained not just during the lorazepam trial, but also during baseline and placebo trials. Such a finding argues against the proposed reason for the (subjects') WMT failure and suggests that the WMT results were not "false positives" but "true positives" with respect to poor effort detection. The (subjects') low scores were more likely caused by low motivation.

Relatedly, low motivation could explain why there was very little practice effect noted on the cognitive test administered (i.e., CNS Vital Signs) across study trials; trials occurred at 1-week intervals. When individuals who were deemed to be invalidly performing were removed from analyses, however, Rohling found that practice effects were nearly five times larger than originally estimated, as might be expected given the brief intervals between testing sessions.

Additional empirical research indicates that patients taking medications that can impact cognitive functioning do not fail PVTs at high rates so long as there is not an external incentive to feign symptoms. For example, while not reported in the Schroeder and Marshall (2011) study, the vast majority of patients with psychotic disorders were taking antipsychotic medications at the time of the evaluation. Despite this, only 7% of the sample failed two or more out of seven PVTs. Relatedly, in a recent meta-analysis of PVT performance in individuals with psychotic disorders, the use of antipsychotic medications was not a significant moderator variable for PVT failure ($p = .17$; Ruiz, Raugh, Bartolomeo, & Strauss, 2020). With regard to pain medications, Dorociak, Schulze, Piper, Molokie, and Janacek (2018) examined a sample of individuals with sickle cell disease. They reported that 94% of their sample were taking at least one opioid pain medication, and 33% took an opioid pain medication (most typically acetaminophen/hydrocodone, morphine, or hydromorphone) on the day of their evaluation. Despite this, 91% of the sample passed RDS at a cutoff of 6, 96% passed TOMM Trial 1 at a cutoff of 40, and 98% passed TOMM Trial 2 at a cutoff of 45; these rates match the specificity rates that are typically observed for these PVTs when examined in other clinical samples (Martin et al., 2020b; Schroeder, Twumasi-Ankrah, Baade, & Marshall, 2012). Thus, with perhaps the exception of extreme cases involving medication misuse, research suggests that medication side effects should not cause PVT failure in the vast majority of examinees.

Research on how fatigue or daytime sleepiness impacts PVT outcome is complicated by the fact that there are relatively few studies on the topic, and the majority of these involve individuals with chronic fatigue syndrome or fibromyalgia. Both of these conditions are medically unexplained disorders, and many individuals with the conditions have internal and/or external incentives that have been shown to impact PVT outcome regardless of co-occurring fatigue (Suhr & Spickard, 2007). Given this, a careful and critical analysis of this literature is necessary to determine whether fatigue, itself, actually impacts PVT outcome. In a study utilizing patients with fibromyalgia, Kalfon, Gal, Shorer, and Ablin (2016) found that 16% of individuals failed the TOMM; however, when using multiple regression analysis, TOMM scores were found to be unrelated to fatigue, pain, or depression. Furthermore, those who failed the TOMM did not differ from those who passed the TOMM on ratings of fatigue, pain, or depression. The authors concluded that "since no difference was found between the groups in levels of pain, fatigue and depression, as mentioned above, the difference in cognitive performance between these groups cannot be attributed to these clinical factors; this finding emphasizes the singular effect of effort on cognitive performance" (p. 34).

In the previously described Dorociak et al. (2018) study, the authors examined how daytime sleepiness related to symptoms from sickle cell disease (a medically explained condition) impacted PVT results. In their sample, the mean Epworth Sleepiness Scale (ESS) score was 8.00 ($SD = 5.23$), and a third of the sample "reported significant daytime sleepiness (e.g., ESS ≥ 10)" (p. 88). Neither RDS, TOMM Trial 1, or TOMM Trial 2 significantly correlated with the ESS score, and Pearson's correlations ranged from –.04 to .02. Thus, while there is a limited amount of empirical literature on how fatigue and

daytime sleepiness directly impact PVT performance, the critically reviewed research suggests that these factors should not be routinely considered as causes of PVT failure given the minimal impact on PVT performance.

Factitious Disorder

Survey data indicate that neuropsychologists believe that factitious disorder is sometimes the cause of invalid test performance, although less often than other factors, including somatoform/conversion disorder, attitude toward testing, and malingering (Martin et al., 2015; Schroeder et al., 2016). One possible explanation for this belief is that the prevalence of factitious disorder is generally thought to be relatively low. For example, the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013) notes that approximately 1% of patients in hospital settings are estimated to have factitious disorder, although its true prevalence is unknown, likely because of the deceptive nature of the condition. Indeed, various researchers, including those within psychology and neuropsychology, note that the prevalence of factitious disorder may very well be underestimated in the empirical literature (Chafetz, Bauer, & Haley, 2020; Velsor & Rogers, 2019).

While there is exceedingly little research on how individuals with factitious disorders perform on neuropsychological validity tests, it is theorized that a number of these individuals will fail validity tests (Chafetz et al., 2020), because, similar to individuals who malinger, patients with factitious disorder engage in conscious other-deception; thus, they fabricate symptoms in an attempt to deceive others. Indeed, in their publication on research criteria for identifying factitious disorder in neuropsychological settings, Chafetz and colleagues assert that both PVTs and symptom validity tests (SVTs) "can be utilized in the same way for [factitious disorder], as these validity tests are blind to the motivations of the examinee" while being sensitive to other-deception provided by examinees (p. 467).

In what, to our knowledge, is the only study that has sought to examine how individuals with factitious disorder perform on validity testing, Rogers, Bagby, and Vincent (1994) found that a small sample of examinees with factitious disorder had elevated scores on six Structured Interview of Reported Symptoms (SIRS; a stand-alone SVT that examines for feigned psychological symptoms) subscales. After comparing results to those obtained by a group of malingering examinees (who had elevated scores on eight subscales), the authors concluded that responses to the SIRS were not consistently different between the groups, which suggests that individuals with factitious disorders might indeed fail validity tests at similar rates to individuals who are malingering.

Given that it is likely that individuals with factitious disorder will fail validity tests, how does one differentiate factitious disorder (an other-deception presentation) from malingering (another other-deception presentation)? Rogers, Jackson, and Kaminski (2005) attempted to address this question by conducting a simulation study that examined differences on the Personality Assessment Inventory between factitious simulators, malingering simulators, and controls. The authors found that controls and individuals simulating malingering scored lower on the Borderline Features scale than individuals simulating factitious presentations, which might not be surprising given that it has previously been suggested that individuals with factitious disorder might also have comorbid borderline personality traits. Furthermore, low scores on the Defensiveness Index (i.e., scores of ≤1) resulted in moderate sensitivity (.59) and relatively strong specificity (.88) when differentiating simulated factitious disorder from simulated malingering.

In addition to using psychological tests, both Chafetz and colleagues (2020) and Boone (2011) suggest that a feature that might help differentiate the two groups is that an individual with factitious disorder lives as an ill individual throughout his/her daily life, while an individual who is malingering "dons the cloak of feigning when it is most suitable for secondary gain . . . and thus does not incur the secondary losses of the person with (factitious disorder; e.g., being in a wheelchair)" (Chafetz et al., 2020, p. 468). Thus, if reliable information regarding investment in illness outside of the evaluation/treatment setting can be obtained, this information might be useful in differentiating the two conditions.

Malingering

Figure 2.1 provides definitions of malingering that should be known by neuropsychologists. As can be seen, malingering is not simply an exaggeration or feigning of symptoms; all of the definitions indicate that malingering is a conscious and deliberate exaggeration or fabrication of symptoms *that is motived by either obtaining items/resources or avoiding negatively perceived responsibilities/punishment*. As detailed in Chapter 3, patients presenting for clinical evaluations are not exempt from pursuing these external incentives, although clinical patients do not always report having this goal. This makes identification of malingering in clinical settings a challenge, and it can alter a clinician's perspective of the prevalence of malingering in nonforensic settings. Empirical research, however, consistently indicates that external incentives increase risk for PVT failure in clinical settings and, when external incentives are present, PVT failure rates can approximate those seen in forensic settings. For example, studies indicate that 20–25% of college students undergoing clinical evaluations due to pursuing stimulant medications and/or academic accommodations fail validity testing (Leppma, Long, Smith, & Lassiter, 2018; Marshall et al., 2010). Furthermore, approximately 30% of veterans, a group of examinees that often has external incentives even in nonforensic evaluations given its members' eligibility for service connection, fail validity testing in clinical settings (Denning, 2012; Denning & Shura, 2019). Additionally, 35 to just over 55% of nonforensic clinical patients pursuing disability fail validity testing in clinical settings (Buddin, Schroeder, Teichner, & Waid, 2012; Schroeder, Clark, & Martin, 2021; Denning & Shura, 2019). This research

Slick, Sherman, and Iverson (1999) definition of malingering of neurocognitive dysfunction: "Malingering of Neurocognitive Dysfunction (MND) is the volitional exaggeration or fabrication of cognitive dysfunction for the purpose of obtaining substantial material gain, or avoiding or escaping formal duty or responsibility" (p. 552).
Sherman, Slick, and Iverson (2020) definition of neurocognitive, somatic, and psychiatric malingering: "Malingering is the volitional feigning or exaggeration of neurocognitive, somatic, or psychiatric symptoms for the purpose of obtaining material gain and services or avoiding formal duty, responsibility, or undesirable outcome" (p. 739).
***Diagnostic and Statistical Manual of Mental Disorder, Fifth Edition* (2013) definition of malingering:** "The essential feature of malingering is the intentional production of false or grossly exaggerated physical or psychological symptoms, motivated by external incentives such as avoiding military duty, avoiding work, obtaining financial compensation, evading criminal prosecution, or obtaining drugs" (p. 726).

FIGURE 2.1. Definitions of malingering.

indicates that *it is the incentive, not the setting* that matters when determining the likelihood of validity test failure, and malingering should be on the differential diagnosis when PVTs are failed even in nonforensic settings.

WHEN CASES JUST DON'T FIT

Sometimes patients fail PVTs for reasons that do not seem to fit any of the previously described explanations. Such a scenario seems to occur with enough frequency in nonforensic settings that some neuropsychologists have considered additional mechanisms by which patients might distort their symptoms or fail PVTs (e.g., Armistead-Jehle, Lippa, & Grills, 2020; Delis & Wetter, 2007; Henry et al., 2018; Slick & Sherman, 2012; Suhr & Gunstad, 2002; Suhr & Spickard, 2012). Many of these hypothesized causes of invalidity appear to relate to (1) a conscious and deliberate exaggeration or feigning of symptoms that differs in some way from DSM-5 criteria for factitious disorder or malingering, (2) subconscious illness beliefs that do not distinctly meet criteria for one of the DSM-5 somatic symptom and related disorders, and/or (3) inaccurate illness beliefs or misattribution of symptoms, which then leads to an exaggeration of cognitive deficits. In the remainder of this chapter, we discuss two proposed, theoretically based diagnostic conditions, then review the available empirical research regarding various psychological variables that have been hypothesized to potentially relate to performance invalidity.

Adjustment Disorder/Adjustment Problem with Specious Symptoms

Adjustment disorder with specious symptoms (ADSS) and adjustment problem with specious symptoms (APSS) are proposed clinical diagnoses that were initially described by Slick and Sherman (2012), who note that ADSS and APSS are proposed diagnoses "for application to cases in which a person exaggerates or fabricates symptoms in order to obtain psychosocial secondary gains, rather than material–legal secondary gains. In APSS and ADSS, the feigning of symptoms is primary directed toward (1) obtaining and maintaining psychological benefits such as increased attention, affection, and support from others; (2) managing problematic interpersonal relationships (e.g., controlling others); and/or (3) escaping from aversive interpersonal situations or avoiding informal obligations such as household chores or schoolwork" (Slick & Sherman, 2012, p. 128). The difference between ADSS and APSS is with regard to the severity of the symptom presentation. APSS occurs when there is only situational feigning of symptoms, whereas ADSS occurs when there is "a severe, deeply entrenched, and pervasive condition in which a person's life revolves around the sick role as a means of obtaining psychosocial reinforcement and managing internal relationships" (pp. 128–129). Slick and Sherman further note that, conceptually, individuals with either ADSS or APSS can fail PVTs even at below-chance performance levels.

Of note, individuals with ADSS or APSS would appear to meet DSM-5 diagnostic criteria for factitious disorder. DSM-5, however, indicates that a "diagnosis of factitious disorder emphasizes the objective identification of falsification of signs and symptoms of illness, rather than an inference about intent or possible underlying motivation" (American Psychiatric Association, 2013, p. 326). Thus, advantages of Slick and Sherman's (2012) proposed diagnoses of ADSS and APSS are that they appear to identify a specific subtype of factitious behavior and describe mechanisms or motivations by which invalidity occurs.

Cogniform Disorder and Cogniform Condition

Cogniform disorder and cogniform condition are proposed clinical diagnoses initially described by Delis and Wetter (2007), who stated that these proposed diagnoses are "subtypes of the somatoform disorders to encompass cases of excessive cognitive complaints and inadequate test-taking effort in the absence of sufficient evidence to diagnose malingering" (p. 589). Cogniform disorder, specifically, is said to reflect a condition in which a patient has pervasive and excessive cognitive symptom complaints impacting multiple areas of his/her life, "thereby suggesting a conversion-like adoption of the sick role manifested primarily as cognitive dysfunction" (p. 589). Individuals believed to have a cogniform disorder are said to report significant cognitive issues that cannot be substantiated from the clinical interview or formal testing. They also report struggling with daily activities to such a degree that they might stop performing these activities (e.g., forfeiting a driver's license). Delis and Wetter theorize that cogniform disorder could manifest in different ways during neuropsychological testing, resulting in normal performance, variable performance, or consistent and markedly poor performance across cognitive tests and PVTs.

Cogniform condition is described as reflecting a less severe form of cogniform disorder (Delis & Wetter, 2007). Specifically, Delis and Wetter note that "the essential features of cogniform condition are the same as those of cogniform disorder in every respect, with the exception of the degree to which the individual exhibits cognitive dysfunction in widespread areas of his or her everyday life" (p. 597). Thus, individuals with cogniform condition do not report struggling with activities of daily living to such as degree that they stop performing those activities. For example, they might continue driving without difficulty despite scoring very poorly on tests of visuospatial ability. In summary, these proposed conditions appear to be related to subconscious illness beliefs that do not distinctly fit under a DSM-5 somatic symptom and related disorders diagnosis.

Diagnosis Threat

Suhr and Gunstad (2002) found that examinees who were reminded of their history of mild TBI and provided with information on the cognitive impact of mild TBI performed more poorly on tests of general intellect and memory than examinees with mild TBIs who tested with instructions that did not connect their history of mild TBI to cognitive functioning. To explain their findings, Suhr and Gunstad theorized that having negative expectations about an injury and its symptoms—for example, believing that a mild TBI causes memory impairment—results in an increased experience of those symptoms, a phenomenon they referred to as *diagnosis threat*.

While there is some evidence that diagnosis threat can negatively impact cognitive test performance (Pavawalla, Salazar, Cimino, Belanger, & Vanderploeg, 2013; Suhr & Gunstad, 2005), there is no research indicating that this phenomenon impacts performance to such a degree that it would account for PVT failure. Although the Suhr and Gunstad (2002) study did not include any formal PVTs, age-corrected Digit Span performance was examined, which has been validated as an embedded PVT. The diagnosis threat group did not score significantly lower on this measure than the non-diagnosis threat group, producing an average Digit Span scaled score of 11.1 (*SD* = 2.6), which would not be indicative of performance invalidity. Somewhat similarly, Carter-Allison, Potter, and Rimes (2016) found that individuals in a diagnosis threat group who had concerns about cognitive consequences of mild TBI produced a mean scaled score of

11.5 (*SD* = 3.2) on Digit Span, which, again, is well within normal limits and not suggestive of performance invalidity. In a follow-up study by Suhr and Gunstad (2005), the authors specifically examined how individuals in a diagnosis threat group performed on a stand-alone PVT, the WMT. They found that the diagnosis threat group performed no worse than the non-diagnosis threat group on the PVT, with average performances on the validity indices being near 99% correct. In summary, these empirical findings suggest that diagnosis threat does not result in suppression of cognitive performances to such a degree that it would account for PVT failure.

Cogniphobia

There is research indicating that some individuals with headaches engage in a fearful avoidance of headache triggers (Martelli, Zasler, Grayson, & Liljedahl, 1999). In instances when such individuals avoid the specific trigger of mental exertion, the term *cogniphobia* has been applied. Because avoidance of mental exertion would presumably lead to decreased cognitive effort, it has been hypothesized that cogniphobia might resulted in diminished effort during cognitive testing, potentially resulting in PVT failure (Martelli et al., 1999; Silverberg, Iverson, & Panenka, 2017; Suhr & Spickard, 2012).

Suhr and Spickard (2012) examined a group of 74 undergraduate students who reported frequent headaches. They found that six of these individuals (8% of the sample) failed the WMT at cutoffs for chronic pain samples proposed by Greve, Ord, Curtis, Bianchini, and Brennan (2008). Suhr and Spickard (2012) noted that those who failed the WMT "were not significantly different from those who passed on Fear/Avoidance, although the results were in the right direction, $p = .13$ and the effect size was medium to large, suggesting the analysis was grossly underpowered due to the small sample of the WMT failures" (p. 1135). In another study, Silverberg et al. (2017) examined outpatients with mild TBI who reported having headaches. They found a significant correlation between a Cogniphobia-Avoidance scale and a stand-alone PVT, the MSVT; however, given that the vast majority of patients included in the study were receiving or seeking injury-related compensation, the authors were unable to control for the possibility "that other factors (e.g., compensation seeking) explained both reduced effort test performance and high scores across questionnaires, including the cogniphobia scale" (p. 2144). Thus, while an intriguing theory, further research evidence is necessary before determining cogniphobia to be a credible explanation for invalidity.

Other Psychological Variables

Henry et al. (2018) conducted a study using a mixed forensic and nonforensic sample that comprised individuals with various neurological, psychiatric, and general medical conditions. They examined relationships between PVT results and the following psychological variables: self-efficacy, suggestibility, dissociation, symptom identity, illness consequences, psychological effects of illness, and cogniphobia. Multivariate logistic regression analysis identified three significant predictors of PVT performance. Two of these predictors were psychological variables, cogniphobia and symptom identity (i.e., the attribution of symptoms to a remote injury or illness), and the other predictor was a contextual factor, forensic evaluation. Of importance, the study authors noted that elevated scores occurred much more frequently in their forensic sample when compared to their nonforensic sample, the two discussed psychological variables can coexist with malingering, and the results could have been influenced by a forensic referral bias. Given

these caveats, it is unclear whether similar findings might emerge in clinical samples that do not have external incentives to underperform and whether cogniphobia and symptom identity can account for PVT failure independent of malingering.

Iverson, Terry, Karr, Panenka, and Silverberg (2018) studied *perceived injustice*, which is the belief that an individual has been treated unfairly and is suffering unnecessarily because of another's actions. The authors found that individuals who were seeking or receiving compensation for their injuries or who failed validity testing endorsed having greater perceived injustice. The authors noted that "it is possible that perceived injustice, which includes feelings of invalidation (not being 'taken seriously') motivates test-takers to 'prove' their injury-related problems through exaggeration. It also is possible that high perceived injustice scores merely reflect a general over-reporting bias" (p. 1163), which cannot be ruled out given that 87% of the sample was seeking or receiving compensation. Thus, it must be considered that perceived injustice might actually be a reason why some people malinger rather than an explanation separate from malingering.

Armstead-Jehle et al. (2020) examined the psychological constructs of self-efficacy and health locus of control related to PVT outcome in a sample of individuals with mild TBI. Similar to Henry et al. (2018), Armstead-Jehle et al. (2020) did not find that self-efficacy related to PVT outcome. The authors also found that there was no relationship between health locus of control and PVT outcome.

Finally, in studies on history of abuse, Donders and Boonstra (2007) found that 21 of 87 patients with TBI failed performance validity testing, with only eight invalidly performing individuals having financial external incentives. They noted that compensation-seeking status did not significantly add to the prediction of PVT failure by logistic regression. Conversely, both premorbid psychiatric history (i.e., prior personal abuse and prior psychiatric treatment) and having no to minimal loss of consciousness following the brain injury increased likelihood of invalidity. When discussing reasons for these findings, the authors noted that "a (patient with a) prior psychiatric history may simply be more likely to make reattribution errors, characterized by under-estimation of their premorbid problems and selective augmentation of cognitive symptoms due to the perception of dysfunction as the result of physical trauma like a car accident being more socially acceptable" (p. 324). Somewhat similarly, Williamson, Holsman, Chaytor, Miller, and Drane (2012) found that PVT failure in patients with PNES [psychogenic nonepileptic seizures] "was strongly associated with reported abuse but, contrary to expectations, was not associated with the presence of financial incentives or severity of reported psychopathology" (p. 588). The underlying mechanism causing some patients without external incentives to fail PVTs in both the Donders and Boonstra (2007) and the Williamson et al. (2012) study is not entirely clear. Nonetheless, results suggest that individuals with abuse histories might be at greater risk for failing PVTs, particularly when presenting with medically unexplained neurological and/or cognitive symptoms.

CONCLUSIONS

We have reviewed and discussed the literature on common explanations provided for PVT failure. In summary, cognitive dysfunction that is sufficiently severe to impact activities of daily living (e.g., intellectual disability or major neurocognitive disorder/dementia-level impairment) can sometimes cause PVT failure despite patients' valid performance. Less severe forms of cognitive dysfunction, however, should not result in failure of most well-validated PVTs. The empirical research also indicates that many

previously proposed explanations of PVT failure (i.e., apathy, fatigue/daytime sleepiness, pain, medication effects, depression or anxiety, a "cry for help") do not actually cause PVT failure in the vast majority of cases, although rare exceptions might potentially occur when there are extreme presentations. Conversely, the empirical research indicates that either self-deception in the form of somatization or other-deception in the form of malingering or factitious disorder can cause PVT failure. Additionally, poor effort resulting in a lackadaisical approach or lack of positive engagement also appears to result in at least some cases of invalidity. It is important to keep in mind that PVT failure due to problematic attitudes and deception (regardless of whether self- or other-deception) should not be considered false-positive errors; instead, the failures are true-positive hits, as the PVT findings indicate that the cognitive test data are not reflective of the patient's true neurocognitive ability.

While both empirical research and survey data (Martin & Schroeder, 2020a) suggest that incentives and attitudes toward testing cause PVT failure in the majority of cases, there appear to be times when these factors are not the cause. If incentives and attitudes are ruled out as likely causes of PVT failure, it is at this point that neuropsychologists might start thinking about the less frequent possibility that psychological factors described in the latter part of this chapter might contribute to the invalid testing. Considering such factors could be useful for clinical conceptualization purposes; however, we recommend that neuropsychologists be cautious in formally ascribing PVT failure to any of these psychological factors given their limited empirical research support.

Finally, in some cases, it is important to keep in mind that the cause of PVT failure will be unclear, but the result is clear: PVT failure renders cognitive test findings invalid (McWhirter, Ritchie, Stone, & Carson, 2020). In this instance, neuropsychologists should consider the analogy of motion artifact on neuroimaging scans; that is, there are a variety of reasons why a patient might move around during a scan, thus causing motion artifact, but there is one common outcome: image degradation that renders the obtained diagnostic test data difficult or even impossible to interpret. In the same way, validity test failure indicates a problem with the image that is drawn from other neuropsychological tests, and it might not always be possible to understand the issues or motivations causing the degradation. In this instance, it will likely serve the neuropsychologist well simply to describe the invalid findings just as they are: invalid.

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