

Symptom Validity Testing in Somatoform and Dissociative Disorders: A Critical Review

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Abstract When patients present with unusual, atypical, and difficult-to-understand complaints known as dissociative and somatoform disorders or medically unexplained symptoms, clinicians may administer symptom validity tests (SVTs) to determine whether or not the patient exhibits negative response bias. Such tests are especially informative in a context where incentives play a substantial role (e.g., the legal arena). If patients fail SVTs and exhibit negative response bias, how should that bias be interpreted? Some authors have argued that psychological problems (e.g., unconscious conflicts and depression) and circumstances (e.g., a cry for help) may explain such bias. In the current article, we critically review this “psychopathology = superordinate” position. We argue that (1) there is no empirical evidence to suggest that psychological problems may foster SVT failure per se and (2) that the “psychopathology = superordinate” position invites circular argumentation: to clarify the nature of the atypical symptoms, SVTs are administered and a negative response bias is found, which is explained away by the atypical symptoms. Negative response bias allows for only one conclusion: the patient’s self-report of symptoms and life history can no longer be taken at face value.

Keywords Medically unexplained symptoms · Somatoform disorders · Hysteria · Malingering · Symptom validity testing · Negative response bias

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Introduction—The Problem

Looking back at the developments in neuropsychology over the last two decades, symptom validity testing can be conceived as a success story. The use of specially constructed self-report scales and cognitive tasks allow for more precise evaluations of the credibility of claimed symptoms and of the validity of neuropsychological test profiles. Consequently, the importance of symptom validity testing has increasingly been stressed by professional bodies and guidelines (e.g., Bush et al., 2005; Heilbronner et al., 2009). Symptom validity testing has not only become standard practice in forensic neuropsychology, but it can also be crucial in a number of routine clinical contexts (Sweet & Guidotti Breting, 2013).

In the 1980s and early 1990s, symptom validity tests (SVTs) were conceived as malingering tests. In the process of conceptual clarification and refinement that followed, this idea was largely abandoned. Many experts now would agree that

- (a) SVTs may help to clarify the nature of certain symptom constellations
- (b) symptom validity assessment comprises both self-report measures that tap over-endorsement of symptoms and tasks (i.e., “effort tests”) that tap cognitive underperformance (Larrabee, 2012, referred to these cognitive SVTs as *performance validity tests*)
- (c) symptom over-endorsement and/or cognitive underperformance represent two aspects of negative response bias. In some cases, they occur together, in other cases only one of the two aspects is present (Iverson, 2006)
- (d) malingering is considered to be only one possible source of negative response bias

Below, we employ the term *malingering* to refer to “... the intentional production of false or grossly exaggerated

physical or psychological symptoms, motivated by external incentives...” (American Psychiatric Association, 1995, p. 701). Likewise, *fictitious disorders* are “...characterized by physical or psychological symptoms that are intentionally produced or feigned” (p. 483), but not primarily motivated by external incentives. In *fictitious disorders*, “...the motivation for the behavior is to assume the sick role” (p. 483). The prevailing view today is that SVTs may help the expert to differentiate between credible and noncredible symptom presentations rather than that they directly detect absence or presence of malingering (see Fig. 1). This means that positive SVTs indicate that a patient’s test profile is probably uninterpretable, but it does not inform clinicians about the cause of this failure. Also, SVT failure may occur in cooperative patients with authentic neurocognitive or mental impairment (false-positive results).

A milestone in the domain of SVT research was the publication of diagnostic criteria for malingered neurocognitive dysfunctions (MND) by Slick, Sherman, and Iverson (1999). These criteria (see Table 1) have gained broad attention in the literature, reaching beyond the traditional province of neuropsychological assessment in patients with brain injury. For example, Bianchini, Greve, and Glynn (2005) adapted these criteria for the malingered pain-related disability. Similarly, Morel (2008) and Morel and Marshman (2008) has repeatedly stressed their importance for the detection of response bias in patients with claimed post-traumatic stress disorder.

According to the Slick et al. criteria, evidence of negative response bias is a necessary, but not a sufficient condition to diagnose possible, probable, or definite malingering in a patient. Evidence of a substantial external incentive (criterion A) is a necessary condition for all degrees of diagnostic certainty (definite, probable, or possible). Criterion D stipulates that psychiatric, neurological, or developmental factors cannot fully explain the response distortion. When criterion D is not met, only *possible MND* can be diagnosed.

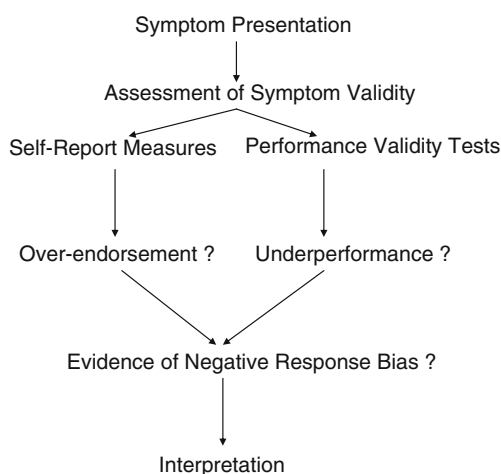


Fig. 1 Conceptual levels involved in symptom validity testing

In medicolegal contexts, the percentage of patients with the diagnosis of somatoform, dissociative, or pain disorders who exhibit negative response bias is considerable and may amount to at least one third. Conditions such as fibromyalgia, sick building syndrome, multiple chemical sensitivity, and whiplash injury overlap with these disorders (for base rate estimates, cf. survey data from Mittenberg, Patton, Canyock, and Condit (2002) and experimental studies, e.g., Schmand et al. (1998)). Much the same is true for conversion disorder which, in DSM-IV (American Psychiatric Association, 1995), is classified as a subgroup of somatoform disorder. Likewise, persistent symptomatic complaints after mild traumatic brain injury (i.e., postconcussive syndrome) can be viewed as a type of somatoform disorder (e.g., Larrabee, 2004). The widely varying complaints in these conditions have also been conceptualized as *medically unexplained symptoms* (see below).

Even in nonlitigating contexts, base rates of negative response bias in these patient groups may be nontrivial. For example, Brooks, Johnson-Greene, Lattie, and Ference (2012) reported negative response bias to be present in 37 % of their fibromyalgia patients treated in a tertiary care clinic. Although the evaluations were performed in a clinical setting, potential secondary gain was identifiable in 58 % of the sample. Williamson, Holsman, Chaytor, Miller, and Drane (2012) observed that 35 % of a sample of patients with psychogenic non-epileptic seizures displayed negative response bias. The presence of negative response bias was related to self-reported childhood abuse rather than self-reported financial incentives. Kemp et al. (2008) found that 11 % of patients with neurologically unexplained symptoms (e.g., conversion, nonorganic sensory deficits, and functional blindness) demonstrated negative response bias. The authors speculated that such a bias may be produced by “various nonspecific reasons such as fatigue, pain, general malaise, or the presence of medical symptoms” (Kemp et al., 2008; p. 324).

The current article addresses problems surrounding the Slick et al. (1999) D criterion. It critically reviews the assumption that negative response bias can be fully explained by the alleged presence of a somatoform disorder, other mental disorders or factors such as fatigue, pain, or cry for help. To preview our conclusion: our position is that clinical and forensic experts become lost in a circular argument when they maintain that nonspecific symptom constellations go along with a negative response bias *because of these symptoms*.

Controversy About SVTs: The German Example

How clinicians interpret response bias may have far-reaching consequences and this is nicely illustrated by a recent discussion among German-speaking forensic experts. In a series of articles, a group of psychiatrists questioned the appropriateness of SVTs and the competence of (neuro)psychologists to

Table 1 Summary of the diagnostic criteria for malingered neurocognitive dysfunctions (MND)

Criteria	Specifications
A. Identification of a substantial external goal	Evidence of substantial external goal as a <i>conditio sine qua non</i> for malingering
B. Evidence of negative response bias in neurocognitive testing	B1. Below-chance response pattern in a forced-choice SVT B2–B6. Other (specified) evidence of negative response bias from neuropsychological testing
C. Evidence of negative response bias from self-report	C1–C5. Evidence of negative response bias from self-report questionnaires and discrepancies between self-report and information from other sources (such as patient's history, known patterns of brain function, observable behavior, reliable third party)
D. Exclusion of alternative explanation	Response distortions cannot be fully accounted for by psychiatric, neurological, or developmental factors

contribute to forensic evaluations of patients with alleged mental disorders, notably posttraumatic stress symptoms (e.g., Dressing & Foerster, 2010). The influential *Deutsche Gesellschaft für Psychiatrie, Psychotherapie und Nervenheilkunde* (German Society of Psychiatry, Psychotherapy, and Neurological Therapy) published an official statement addressing the use of SVTs (Dressing, Foerster, Widder, Schneider, & Falkai, 2011). This document raises doubt that such tests can be conceived as objective methods that may help the clinician to detect response bias and malingering. Furthermore, it maintains that when patients suffer from mental problems rather than cognitive symptoms, they would automatically pass SVTs. However, this assumption is not supported by clinical experience or empirical research (e.g., Dandachi-FitzGerald, Ponds, Peters, & Merckelbach, 2011). It is not uncommon for patients to demonstrate negative response bias who are not explicitly reporting cognitive problems and it is well documented that failure in performance validity tests occurs in conditions which are not commonly associated with major cognitive problems. Failure in cognitive SVTs in such patient populations (e.g., fibromyalgia, soft tissue injury, chronic low back pain, or posttraumatic stress patients; e.g., Gervais et al., 2001; González, Capilla, Santamaría, & Casado, 2012; Richman et al., 2006; Merten, Thies, Schneider, & Stevens, 2009) is not limited to individuals with explicit claims of cognitive impairment. Similarly, Binder, Spector, and Youngjohn (2012) recently described three patients with apparent psychogenic speech and language abnormalities who underwent forensic neuropsychological assessment. In all three cases, malingering was determined on the basis of well-documented negative response bias and a motivational analysis of incentives.

In their official statement, Dressing et al. (2011) further argue that SVTs would “invite” patients to produce negative response bias. In particular, test performance below empirically established cutoffs could be “caused by very different factors” (p. 389), for example, somatoform and depressive disorders. The authors further believe that symptom validity tests do not allow for differentiating between malingered

neurocognitive impairment and “neuropsychological impairment developing in the context of mental disorders, such as conversion disorder” (p. 389). Germane to this is also the emphasis that the authors put on the primacy of psychiatric expertise to arrive at valid differential diagnoses. In a rebuttal, Schmidt, Lanquillon, and Ullmann (2011) pointed out that professional politics and vested interests appeared to be a major factor in the debate about SVTs. Be that as it may, the document has its merits in that it so clearly articulates the idea that psychological entities like depression or somatoform symptoms may produce negative response bias. Clearly, this idea bears relevance to the D criterion of Slick et al. (1999).

In a similar vein, some authors contributing to the German debate have argued that patients with functional or somatoform syndromes (such as fibromyalgia, chronic fatigue, and pain disorder) produce scores on SVTs erroneously suggesting malingering (Noeker & Petermann, 2011). These authors posited that when patients with somatoform disorders produce an excessive array of symptoms, they nevertheless respond “in a subjectively truthful manner” (Noeker & Petermann, 2011; p. 450). The discrepancy between their symptoms and objective medical data should not be taken as indicating biased responding. It should rather be understood as “the sensitive expression of major subjective distress and impairment” (Noeker & Petermann, 2011; p. 450) (for further discussion of the resistance against SVTs, see Green & Merten (2013)). Thus, the recent German debate illustrates that it is not at all uncommon in the extant psychiatric and psychological literature to claim with great confidence that genuine symptoms may underlie negative response bias.

Somatoform and Dissociative Disorders

A key feature of both somatoform and dissociative disorders is the presence of pseudosomatic complaints (notably pseudoneurological symptoms, such as paralysis, amnesia, and derealization in dissociative disorders) that are suggestive

of an underlying medical dysfunction, but that are presented in the absence of evidence for known abnormalities that could fully explain the symptoms. The symptoms that constitute these disorders are often defined by the very discrepancy between subjective complaints and objective signs, as is also true for factitious disorder and malingering. The common denominator that these conditions share is that they, in the words of Eisendrath (2002, p. 396), "... all represent abnormal illness-affirming behavior" (see, for a similar analysis, Kanaan & Wessely, 2010). Thus, the symptom spectrum of somatoform and dissociative disorders involves experiences that cannot be objectively verified, but are inferred from the patients' reports (such as complaints about sensory symptoms, pain, localized weakness, lump in throat, or problems with memory retrieval) or from behavior that is under motor control (as is the case with functional movement disorders, psychogenic seizures or psychogenic stupor). In neurology, the traditional working field of neuropsychologists, 10–30 % of patients may present with such medically unexplained symptoms (e.g., Reuben, Mitchell, Howlett, Crimlisk, & Grünewald, 2005).

The critical difference with malingering or factitious disorder is that somatoform and dissociative disorders are assumed to be caused by an underlying psychological or emotional factor and that symptom presentation does not occur *consciously*, or in the words of Slick et al. (1999), they are *not* "the product of an informed, rational, and volitional effort aimed at least in part towards acquiring or achieving external incentives" (p. 554), as would be the case in malingering.

The absence of a somatic cause for their constituent symptoms often makes somatoform and dissociative disorders *diagnoses by exclusion*. Meanwhile, longstanding clinical lore in internal medicine, neurology, and other branches of medicine dictates that the diagnosis of a mental disorder should not be based on exclusion (e.g., the absence of neuroradiological abnormalities). Rather, it should be based on *positive* evidence (e.g., Crimlisk & Ron, 1999; Frick, 2005). However, an uncooperative patient may evade a thorough evaluation of possible conflicts or other emotional factors. He or she may omit or distort essential diagnostic information that may be suggestive or not of a mental disorder. Thus, uncooperativeness (or negative response bias) can effectively prevent the collection of positive evidence. Consequently, in the absence of positive evidence, what can be concluded at most is the *possibility* of a disorder.

Ideally, such a tentative diagnostic conclusion must take into account the following scenarios:

- (a) There is no disease at all in this patient (as would be the case with pure malingering)
- (b) There is a somatic cause for the symptoms which has not yet been detected
- (c) There is a mental disorder of a very different kind (such as factitious disorder) fully accounting for the apparent discrepancies
- (d) Several of the above factors may co-exist in a given patient

In everyday clinical practice, however, once a patient has been seen by a psychotherapist or a psychiatrist, the odds appear to be high that somatoform or dissociative disorder is diagnosed, dismissing (and often not even considering) the other scenarios (see also Page & Wessely (2003)).

When the basic requirement of positive evidence is not fulfilled, diagnostic errors are likely to occur on a nontrivial scale. Thus, in the past, it was not uncommon for neurological patients, such as those with multiple sclerosis, to be misdiagnosed with "hysteria" and to be referred for psychotherapy (e.g., Stone et al., 2005). Similarly, there are case histories of patients referred to psychotherapy because of unexplained symptom report, with a subsequent diagnosis of a brain tumor—although this diagnostic error has become rarer with the advent of modern neuroradiology. According to Stone et al. (2005), the rate of this type of misdiagnosis (mistaking a neurological disease for a mental disorder) has dropped considerably from the 1950s onwards.

To avoid premature diagnostic conclusions, some authors have suggested using the etiologically neutral term *medically unexplained symptoms* to refer to complaints that cannot be satisfactorily explained by underlying somatic pathology. Brown (2007) defined them as "a heterogeneous group of conditions characterized by persistent physical symptoms that cannot be explained by medical illness or injury" (p. 769). This term has been criticized for its imprecision (e.g., Creed et al., 2010), but this very imprecision (or diagnostic uncertainty) appears to capture very well the nature and the ambiguity of the symptoms involved. A number of authors (e.g., Rief & Broadbent, 2007) have used the label *medically unexplained symptoms* as an equivalent for, or interchangeably with, somatoform disorders and somatization.

Digression on Hysteria and Medically Unexplained Symptoms

The problems discussed above were in the past the subject of a heated controversy in the context of what was then called hysteria. The difficulty to differentiate between malingering and hysteria were framed in the famous work of Jones and Llewellyn (1917) when they wrote that "nothing, it may be said, *resembles malingering more than hysteria; nothing hysteria more than malingering*. In both alike we are confronted with the same discrepancy—between fact and statement, between objective sign and subjective

symptom” (p. 117; italics in the original). These authors also emphasized that there was no objective test to discriminate between the two conditions. They pointed out that published lists of indicators for both hysteria and malingering were overlapping to a considerable degree. Even criteria that were conceived as the classical signs of hysteria have never lived up to expectation when they were tested empirically: these criteria turned out to yield elevated false-positive classification rates in patients with true brain pathology (Gould, Miller, Goldberg, & Benson, 1986; Stone, Smyth, Carson, Warlow, & Sharpe, 2006).

A systematic study testing the efficiency of classical hysteria signs or other categorical criteria to distinguish between hysteria and malingering has, to our knowledge, never been undertaken. These criteria are often poorly described and when evaluated, their reliability is low (Stone, Zeman, & Sharpe, 2002). The validity of some common signs for hysteria or medically unexplained symptoms must be expected to be low or even zero. Examples of these signs include impairment of motor or sensory functions, disappearance of symptoms when unobserved (dubbed, in the context of hysteria, as *give-away signs*), symptom presentation in a dramatic, theatrical way, *la belle indifférence*, or excessive seeking of medical attention. Reciprocally, these clinical signs per se are expected to have little value to prove intentional fabrication. As Faust (1995) wrote in the early years of modern malingering research:

Finally, clinical guides or signs for malingering detection may represent little more than educated guesses or clinical lore, which have not been properly validated and screened. These kinds of signs may be frankly erroneous because one usually does not find out after the fact whether positive and negative judgments about malingering are correct. (p. 259)

A good example of the conceptual chaos surrounding criteria lists is provided by Ganser syndrome, which is commonly conceptualized as a dissociative disorder. Its hallmark is the tendency of patients to give approximate answers to simple questions (Merckelbach et al., 2006). However, behavioral approximation (i.e., producing a close, but not exact answer) also figures prominently on lists of faking bad response styles (e.g., Hall & Poirier, 2001). Indeed, Rogers, Harrell, and Liff (1993) included alertness to *near misses* in their list of the six most prevalent strategies to detect malingering.

It is easy to see how attempts to empirically study the accuracy of differential diagnosis in this field will be flawed by methodological problems. Thus, in an analysis of the current state of the diagnosis *conversion disorder*, Nicholson, Stone, and Kanaan (2011) remarked what is true for all kind of medically unexplained symptoms: “...these studies are limited by the assumption that the conversion disorder patients

studied are not actually misdiagnosed feigners and, conversely, those studying ‘real’ feigners (rather than controls instructed to feign) are not conversion disorder cases” (p. 1271).

Taken together, there are good reasons to question the idea that hysteria and its modern successors (i.e., somatoform and dissociative disorders) are coherent diagnostic entities. One of the most outspoken critics of the concept of hysteria was Slater (1965), who viewed hysterical symptoms not as signs of disease, but of health. According to Slater, patients diagnosed with hysteria do not constitute “in medically significant terms anything more than a random selection” (Slater, 1965; p. 1399). This author also pointed out that the label *functional* as opposed to *organic* was misleading. Yet, these terms continue to be used both in the clinical context and in research papers (e.g., Reuben et al., 2005; see also De Renzi, 2002). To extend Slater’s arguments, one could maintain that no symptomatology is more obviously functional in nature than malingered symptoms which, by definition, are instrumental and directed towards an external gain. Slater (1965) himself went as far as stating that “the diagnosis of ‘hysteria’ is a disguise for ignorance and a fertile source of clinical error. It is in fact not only a delusion but also a snare” (p. 1399).

Since Slater’s time, the diagnostic confusion has not become less, although hysteria was abandoned and redefined as somatization disorder, conversion disorder, dissociative disorder or, even broader, medically unexplained symptoms (Merten, 2001). This modernization of the diagnostic vocabulary has not resolved the fundamental question: are these disorders anything else than an array of unusual symptoms in the absence of evident somatic pathology?

Differential Diagnosis Based on Clinical Judgment

Patients who feign health problems and patients with somatoform disorder are indistinguishable in their electrophysiological responses, as noted in a paper by Wilbourn (1995). Likewise, Dyck et al. (1998) pointed out that “we know of no algorithm of psychophysical testing that can reliably overcome the bias toward showing abnormality inherent in patients who wish (*consciously or unconsciously*) to demonstrate more disability than they have, for whatever reason” (p. 1213, italics added).

Because objective signs for somatoform or dissociative disorders are lacking, clinicians often rely on their subjective judgments about the genuineness of unusual symptoms. In particular, the clinical expert has to make inferences about whether the patient engages in conscious and intentional symptom production and, if so, what the underlying motivation for this might be (external vs. internal incentives; cf. Table 2). As described by Eisendrath (1995, 2002), these two dimensions (i.e., conscious/intentional versus unconscious/non-intentional and internal versus external

Table 2 Differential diagnosis between three forms of atypical symptom presentation

	Intentional symptom production	Unintentional symptom production
External Incentives	Malingering	
Internal Incentives	Factitious disorder	Somatoform and dissociative disorders

incentive) remain the decisive aspects of differentially diagnosing malingering and factitious disorder versus somatoform and dissociative disorders. In somatoform and dissociative disorders, the motivation for symptom presentation must be outside the individual's awareness.

Arguably, to the extent that diagnostic experts rely on clinical experience, their accuracy will depend largely on their ability to evaluate volitional processes within the patient; detect possible deception; differentiate between self-deception and other deception; correctly evaluate the patient's self-reported autobiography (that may or may not contain clues as to an underlying conflict or other emotional factors and that may or may not be presented with essential distortions); and finally, to perform a valid motivational analysis. This, of course, is an almost impossible endeavor (Boone, 2007b). Note that the diagnostic interpretation of patients' self-reports is affected by their ability to convincingly give a largely distorted history. Some patients score high on fantasy proneness and this comes with a talent to fabricate credible stories. This aspect is important because fantasy proneness is known to overlap with dissociative symptoms (Schelleman-Offermans & Merckelbach, 2010).

Another point that should be mentioned in this context is what has been called *hidden agendas* (e.g., a patient striving for insurance benefits; Van Egmond & Kummeling, 2002). These seem to be quite common among psychiatric patients. For example, in a sample of 166 Dutch psychiatric outpatients, Van Egmond, Kummeling, and Balkom (2005) observed that 42 % of them had clear expectations about additional benefits from being in therapy, other than just getting better. For example, patients expected support from therapists to obtain disability status, sick leave, or a new accommodation. Interestingly, in most cases, attending clinicians were unaware of these expectations.

The experts' ability to reliably differentiate between truth and lies, between honest communication and deception, is a decisive factor for diagnosis in this domain. Often, clinical intuition is the major source on which experts rely for making these distinctions. There is ample evidence that experts' competence to make accurate judgments about the truth or falseness of communications (e.g., Ekman & O'Sullivan, 1991; Faust, 1995; Vrij, 2008) is as much restricted as neuropsychologists' intuitive ability to distinguish between authentic and feigned cognitive test profiles (e.g., Faust, Hart, & Guilmette, 1988; Heaton, Smith, Lehman, & Vogt, 1978).

Lie detection is a productive research line in forensic psychology (for a recent meta-analysis, Hartwig & Bond, 2011). This research has identified behavioral and personality characteristics of highly effective liars (Vrij, Granhag, & Mann, 2010). An informal inspection reveals that these characteristics may also apply to successful malingerers in medicolegal contexts. However, until now, there has been little intellectual exchange between lie detection research and studies on negative response bias, although both fields could benefit from each other's methodological repertory (Merten, 2010).

There are reasons to assume that in clinical practice, malingering and factitious disorder are underdiagnosed (for the latter, cf. Eckhardt, 1989). When clinicians rely on their intuition and subjective judgment, they strive to minimize false-negative errors (misclassifying patients with serious problems as healthy malingerers; Rassin & Merckelbach, 1999). Interpreting the atypical symptoms of patients in terms of dissociation, somatoform disorder, or medically unexplained symptoms helps clinicians to avoid such false-negative errors, but the price paid for this is a failure to detect those instances of negative response bias that refer to malingering and factitious disorder. Some authors openly favor such a biased clinical decision making. For example, Reuben et al. (2005) wrote that: "*Fortunately*, it is rarely necessary for a clinician to determine whether symptoms are intentional" (p. 308, italics added). They maintained that "even if patients see no other way of reducing psychological distress than feigning illness or exaggerating pathophysiologically explained symptoms, it may be appropriate to offer medical or psychological attention" (p. 308).

With the obvious exception of novice clinicians, validity of clinical judgment and clinical experience appear to be unrelated and constitute an "illusory correlation" (Dawes, 1989). As far as the detection of malingering is concerned, one obvious reason for this is that clinicians will usually not receive feedback on the accuracy of their judgment. This lack of feedback prevents successful learning.

Symptom Validity Assessment in Patients with Mental Disorders

A new dimension in the assessment of unusual symptoms opened up when in the 1990s SVTs—mostly simple cognitive tests that tap into memory—became used on a wider

basis. Unlike clinical judgment, these tests are firmly based on empirical research. For many of them, decision rules (e.g., cutoffs, the meaning of subscales, strategies to rule out neurological patients) have been published, they are teachable, and test results can be easily replicated and communicated. In the meantime, a large database on the sensitivity and specificity of these tests in different groups has been accumulated, which allows inferences about the degree of certainty of individual diagnostic decisions (see, for examples, various chapters in Boone (2007c) and Larrabee (2007)). With the development of SVTs, determinations about feigned impairment entered the stage of evidence based assessment.

In *bona fide* patients, rates of failure on SVTs are usually low. This is true for depression (Ashendorf, Constantinou, & McCaffrey, 2004; Lee et al., 2000; Rees, Tombaugh, & Boulay, 2001), anxiety (Ashendorf et al., 2004), and depression with chronic pain (Iverson, Le Page, Koehler, Shojania, & Badii, 2007). Studies in which SVTs were administered to healthy and brain-injured children are informative with regard to the real cognitive load that is required to pass these tests (e.g., Blaskewitz, Merten, & Kathmann, 2008; Carone, 2008; MacAllister, Nakhutina, Bender, Karantzoulis, & Carlson, 2009). In general, SVT failure in these groups is rare, implying that the cognitive load of most SVTs is very limited.

From other research, we know that severe brain pathology such as dementia, organic amnesia, mental retardation, and moderate to severe aphasia may cause SVT failure (e.g., Dean, Victor, Boone, Philpott, & Hess, 2009; Henry, Merten, & Wallasch, 2008; Hurley & Deal, 2006; Merten, Bossink, & Schmand, 2007; Rudman, Oyebode, Jones, Bentham, 2011; Shandera et al., 2010). On the other hand, the presence of severe brain damage is not invariably related to SVT failure. Whether or not this occurs, depends on factors like the demand characteristics of a particular test and the choice of patients included in a study, in particular the degree and profile of impairment (e.g., Brockhaus & Merten, 2004; Green, Montijo, & Brockhaus, 2011; Shandera et al., 2010). For example, even mild dementia is not automatically associated with SVT failure (Rudman et al., 2011). Merten et al. (2007) noted that in patients with suspected Alzheimer's disease, an increased probability of failure on the Test of Memory Malingering (Tombaugh, 1996) was reliably associated with Mini-Mental State Examination (Folstein, Folstein & McHugh, 1975) scores of less than 24. Indeed, insensitivity of a test to genuine cognitive dysfunction has been proposed as a quality marker of SVTs (Hartman, 2002).

In medicolegal contexts where substantial incentives are to be expected, a sizable proportion of patients who claim a mental disorder fail SVTs. In the Mittenberg et al. (2002) survey, high rates of response bias suggestive of possible

malingering were reported for fibromyalgia, chronic fatigue, and pain and somatoform disorders, amounting to up to 38.6 % of the referrals (adjusted estimates). A number of studies and case reports confirm that a nontrivial proportion of patients with claimed mental disorders fail on SVTs (e.g., Dandachi-FitzGerald et al., 2011; Demakis, Gervais, & Rohling, 2008; Morel, 2008; Stevens, Friedel, Mehren, & Merten, 2008; Youngjohn, 1995).

The propensity of persons who claim psychological complaints to fail on SVTs can easily be modeled in experimental studies. Even with scenarios that are not specific as to the would-be symptom spectrum, a substantial number of experimental malingerers have a preference for cognitive problems (concentration difficulties, extreme forgetting, and perceptual problems) when they are instructed to feign symptomatology and so become detectable with cognitive SVTs (Dandachi-FitzGerald & Merckelbach, 2013).

It would make for an incoherent theoretical position if we were to assume that psychological complaints that produce minimal interference with everyday functioning produce failures on SVTs comparable to those of moderate or severe dementia or other conditions associated with serious cognitive impairment (see also Larrabee, 2012). With this in mind, SVT failure in these groups is most likely to reflect negative response bias produced by poor cooperation, whether this is in the presence or absence of genuine psychological complaints. There appears to be no convincing evidence to suggest that SVT failure can *be fully explained* by somatoform disorder, dissociative disorder or (other) medically unexplained symptoms, mild depression, or posttraumatic stress disorder.

The biggest methodological problem that plagues SVT research in patients with these psychological complaints is that there is no a priori guarantee that *bona fide* patients can be reliably recruited. In contrast, in acute neurological or neurotraumatological centers, *de novo* patients after cerebrovascular insult, with brain tumors, neuroinflammatory diseases or after moderate, and severe traumatic brain injury usually perform to the best of their abilities. It is only during the later stages that patients might develop open or hidden agendas that may substantially interfere with their willingness to cooperate. In rehabilitation centers, patients may have such agendas (e.g., disability claims; insurance compensation claims) that need not to be obvious to clinicians. The wish to be referred to rehabilitation may in itself serve as a motive to produce distorted symptomatology and exaggerate somatic, psychological, or cognitive complaints. For example, Locke, Smigielski, Powell, & Stevens (2008) examined 87 outpatients who sought admission to an intensive brain injury rehabilitation program. Twenty-two percent of them failed on an SVT indicating that negative response bias may also be an important issue in treatment-seeking patient populations.

Self-Report SVTs

Self-report scales are increasingly used as measures of negative response bias. They intend to detect either over-reporting of cognitive complaints (Gervais, Ben-Porath, Wygant, & Green, 2007) or, more importantly in this context, over-endorsement of psychiatric symptoms, and emotional and pain complaints. Following the example of the MMPI-2 Fake Bad Scale (Lees-Haley, English, & Glenn, 1991), a number of MMPI-2 indices have been specifically tailored to detect negative response bias in persons who are involved in civil litigation (Henry, Heilbronner, Mittenberg, & Enders, 2006; Henry, Heilbronner, Mittenberg, Enders, & Roberts, 2008). For example, Meyers, Millis, and Volkert (2002) designed an index for the detection of feigned pain disorders. In contrast to the early MMPI-2 fake-bad scales of the *F* family (the *F*, *F_b*, and *F_p* scales), these *new generation* scales do not tap implausible endorsement of “hard” psychiatric symptoms like unusual hallucinations. Rather, they look for unusual symptom constellations. Thus, they can be expected to be much more sensitive to detect feigned psychological complaints.

Unlike traditional cognitive SVTs, self-report measures are relatively subordinate in the Slick et al. (1999) criteria insofar as even gross symptom over-endorsement on these measures does not qualify for a diagnosis of definite MND. The underlying assumption is that self-report measures do not reach the sensitivity and specificity values of cognitive SVTs. However, some analog simulation studies have found that self-report instruments may outperform cognitive SVTs in the detection of negative response bias (Jelicic, Ceunen, Peters, & Merckelbach, 2011). Of course, outside the laboratory, research comparing the diagnostic efficacy of self-report indices and cognitive SVTs is hindered by the difficulty to define a gold standard.

Patients who fail on self-report SVTs also often exhibit elevations on the MMPI-2 *clinical scales* (e.g., scales 1, 2, 3, 7; Larrabee, 2004) or any other instrument that taps into symptoms (e.g., Dandachi-FitzGerald et al., 2011). This creates an interpretational problem: if the diagnosis of a mental disorder is substantially based on self-report evidence and this very evidence is biased in the same direction as known from persons who malingers, how can we possibly conclude that the diagnosis is accurate?

The point is illustrated by a study of González, Santamaría, and Fernández (2010). These authors evaluated 61 clinical patients with somatoform symptom presentation who were all on sick leave. Employing a list of clinical indicators (such as identifiable external gain, antisocial behavior, symptom exaggeration, discrepancy between self-reported history and information from previous reports, and insufficient compliance with treatment), the group was divided into two subsamples: probable malingerers and

nonmalingerers. Then, the authors administered to both subsamples the Structured Inventory of Malingered Symptomatology (SIMS; Smith, Widows & Smith, 2005), a self-report index that taps over-endorsement of atypical symptoms. The probable malingerers had considerably higher SIMS scores than the control patients.

Göbber, Petermann, Piegza, and Kobelt (2012) administered the German version of the SIMS (Cima et al., 2003) to 595 patients in a German psychosomatic rehabilitation clinic. Slightly more than 40 % of these patients obtained scores above the cutoff indicating negative response bias. In a subgroup of migrant patients, the proportion of those with SIMS scores above the cutoff was particularly high (50 %). One could argue that the patients with elevated SIMS scores in the studies of González et al. (2010) and Göbber et al. (2012) displayed negative response bias *because of* their psychopathology. That interpretation would imply that genuine psychopathology is the superordinate diagnosis and that negative response bias is a secondary accompaniment of genuine psychopathology. Nothing in the extant literature would justify this type of argument and the evidence gathered by González et al. (2010) who even argues against the “psychopathology = superordinate” doctrine because in that study negative response bias covaried with variables like external gain rather than psychopathology.

Patients who fail cognitive SVTs have a higher probability to exhibit over-endorsement of symptoms on self-report SVTs as well. Jones, Ingram, and Ben-Porath (2012) found in their sample of 501 military participants claiming minor traumatic brain injury that SVT failure was associated with overreporting on MMPI-2 scales, in particular the Response Bias Scale (RBS) and the Symptom Validity Scale (FBS-r), with effect sizes of 1.69 and 1.34, respectively.

The link between failure on cognitive SVTs and symptom over-endorsement can be easily explained away by invoking untestable notions. Consider a study by Boone and Lu (1999), who interpreted a 1-3/3-1 code type profile on the MMPI or MMPI-2 as evidence for somatization or conversion. Two thirds of their patients, who were all involved in litigation, presented at least some evidence of failure on cognitive SVTs. If one were to conclude, as the authors did, that in these cases failure on SVTs was due to unconscious symptom fabrication, one must agree that in clinical decision making self-reported symptoms have a superordinate status and failure on SVTs is a subordinate issue. There is no evidence that could warrant such a position, and in fact the reverse would be more legitimate: when patients fail on SVTs, there is every reason to take into account the possibility that negative response bias is the overriding feature. Boone (2007a) later wrote: “Unfortunately, personality inventories are similarly unhelpful in distinguishing between consciously and non-consciously created symptoms” (p. 677).

The Meaning of Below-Chance Response Patterns

Forced-choice SVTs allow for the identification of performance that is so poor that it is below the threshold of random guessing. Forced-choice SVTs have a special place in the evaluation of negative response bias. For most authors, performance below chance is a strong indication of the patient voluntarily endorsing incorrect answers (Bush et al., 2005; Frederick & Speed, 2007; Slick et al., 1999; Merten & Merckelbach, 2013), or as “tantamount to confession of malingering” (Larrabee, 2004). Thus, Iverson (2003) proposed a standard formulation for how to report such test performance and its meaning to the referral party (e.g., the court):

The patient scored below chance on a (...) forced-choice procedure, indicating that she knew the correct answer and deliberately chose the incorrect answer. This performance invalidates the entire set of neuropsychological test results. (p. 169)

In line with this, a 2008 consensus conference of the American Academy of Clinical Neuropsychology (Heilbronner et al., 2009) stated:

There is consensus regarding the meaning of significantly below-chance findings and what has been referred to as a ‘compelling inconsistency’ (Bianchini et al., 2005), and both are viewed as individually reflecting a deliberate attempt to misrepresent one’s abilities for which there are no alternative explanations. (p. 1103)

The widely voiced view that below-chance performance is indicative of intentional underperformance is sometimes contradicted. A minority of authors maintain that somatoform or other mental problems may *explain* such below-chance responding. These critics often refer to the historical argument that forced-choice testing was first developed in the context of a case of hysterical blindness (Brady & Lind, 1961; Grosz & Zimmermann, 1965). However, the argument is far from convincing because all we know about this particular case is that the hysterical blindness was inconsistent and doubtful. Thus, at some stage, the allegedly blind patient confessed that he could, in fact, see and was *not* blind (Brady & Lind, 1961). How, then, could we possibly know that this patient, treated 50 years ago, suffered from a mental disorder that *caused* response patterns below chance? The reverse possibility, namely that the patient might have malingered blindness, was discussed in detail by Grosz and Zimmerman (1965). The authors argued that there were clues in the patient’s history that pointed in the direction of intentional symptom fabrication. For example, at one point in time, the patient told a nurse that he “was beginning to see a little bit, but

asked her not to tell anyone” (p. 257). Like many authors before and after, Grosz and Zimmermann appeared to accept the “possibility that hysteria and malingering may gradually and imperceptibly supersede one another, or, for that matter, coexist in the same patient at the same time” (p. 258). Brady (1966), in a response to Grosz and Zimmermann, admitted that hysteria and malingering should be conceptualized as points on a continuum, and that, in this case, the patient might have moved over time “farther from the hysterical end of the continuum and may have been predominately malingering” (p. 322).

For reasons that remain obscure, Pankratz (1983), who introduced forced-choice testing in neuropsychological assessment, argued that below-chance responding might be *functional*. In other cases, he went on, such response patterns might be indicative of *cheating*, and clinical judgment was needed to appreciate these differences in a correct way. In another paper, Pankratz, Fausti, and Peed (1975) stated that in cases of claimed sensory loss below-chance responding meant that they perceived the stimuli, “but consciously or unconsciously chose to deny it” (p. 1975). The confidence that Pankratz (1983) placed in clinical judgment is, of course, problematic.

Reviewing a broad literature on forced choice visual discrimination in hypnotic blindness, visual conversion disorder, and blindsight patients (e.g., patients with damage to the primary visual projections to the occipital areas), Bryant and McConkey (1999) concluded that in all these conditions patients are sensitive to (implicit) visual primes and therefore perform above chance. Accordingly, these authors interpreted below-chance level performance as an indication of malingering. In sum, the special consideration that this form of negative response bias is given in the Slick et al (1999) criteria (see Table 1) is fully justified precisely because a psychopathology = superordinate doctrine is unable to explain below-chance performance.

Pseudo-Explanations and the “Cry for Help”

Alternative explanations for negative response bias, other than malingering, have been widely discussed in the literature. Uncooperativeness during neuropsychological assessment may be caused by motivational factors other than malingering. For example, a person may simply choose not to invest full effort because he or she does not understand the test or does not accept its importance. The result will, nonetheless, be the *correct* determination of insufficient effort and an invalid test profile, meaning that the clinician is simply unable to determine the precise problem of this person.

Various other variables, often referred to as “psychological factors”, have been proposed to explain why a person

may fail SVTs (cf. Iverson, 2006). Thus, Silver (2012) discussed a number of factors that can “appear as ‘symptom magnification’ or ‘poor effort’, which are incorrectly interpreted as the result of a conscious process” (p. 836). According to this author, relevant antecedents were loss aversion, stereotype threat, anger, and revenge. In our view, this is a light version of the psychopathology = superordinate doctrine. Silver also suggested that “people in litigation or who are applying for disability may perform more poorly on the effort tests because of trying too hard and increased effort” (p. 839), an idiosyncratic view with no support from the extant literature. In clinical practice, such as in acute neurology, a sizable proportion of patients are trying very hard to obtain good results on neuropsychological tests, but this does not appear to cause SVT failure, even in the presence of severe neuropsychological impairment (e.g., Goodrich-Hunsaker & Hopkins, 2009). Also, parents who seek custody for their children are usually highly motivated to pass a court ordered neuropsychological evaluation. They may be full of “loss aversion, stereotype threat, anger, and revenge”, yet they do not fail SVTs (Flaro, Green & Robertson, 2007). Similarly, in the field of sports-related concussion research, there appears to be no evidence for elevated rates of SVT failure. Athletes are usually highly motivated to return to play as soon as possible, which usually is not considered problematic for the validity of test results (Bailey, Echemendia, & Arnett, 2006). Rather, with the increased awareness of concussion in contact sports and the introduction of special concussion monitoring programs, underperforming (or sandbagging) at baseline testing appears to be the problem (Erdal, 2012).

In an analysis of ethical challenges in the context of SVT, Iverson (2006) identified “cry for help” as one of the most inappropriate psychological pseudo-explanations or euphemisms for failure in SVTs. He wrote:

Clinicians should be careful to not simply use a cry for help as a stock standard inference for the cause of the exaggeration. This explanation for the exaggeration, like any explanation (including malingering), should be based on clear and converging evidence. It could be considered biased if a clinician has a much lower threshold, and relies on much less evidence, to attribute exaggeration to a cry for help versus deliberate misrepresentation of symptoms and problems to influence the results of a forensic evaluation. (p. 82)

Indeed, there appears to be no scientific basis for the assumption that cry for help may explain failure on SVTs. As with the attempt to explain SVT failure *as a direct consequence* or *caused* by minor psychopathology, psychological pseudo-explanations should be challenged when they are brought forward, and the scientific basis for such

claims should be questioned. Often, their character is rather one of *explaining away* negative response bias.

Cogniform Disorder and Cogniform Condition

Delis and Wetter (2007) coined the labels “cogniform disorder” and “cogniform condition”. Both diagnoses were proposed for patients who report excessive cognitive complaints or exhibit poor performance on cognitive testing and who may also display negative response bias on SVTs (“evidence of insufficient test-taking effort or exaggeration”). The difference between the two labels is that persons with cogniform disorder apparently adopt the sick role, while this excessive illness behavior is lacking in cogniform condition. Delis and Wetter considered cognitive disorder to be a subtype of somatoform disorder.

One of the reasons why these labels do not seem to have a deep impact on neuropsychologists’ current diagnostic and research practice is that they are unable to solve any of the conceptual problems discussed in the current paper. Delis and Wetter (2007) seem to assume that SVT failure might be the result of the unconscious pathology inherent to cogniform disorder or cogniform condition, and in doing so these labels are yet another version of the psychopathology = superordinate doctrine. However, these authors did acknowledge that neither of their labels should be used “if there is reasonable evidence that the excessive cognitive symptoms are produced in an intentional or volitional manner” (p. 598), and they accepted below-chance performance as such evidence.

A Threat to the Scientific Database

The clinical practice of diagnosing medically unexplained symptoms, somatization, or dissociative disorders without attempts to exclude malingering (and factitious disorder) as alternative explanations may result in gross over diagnosing and elevated false-positive errors. Such errors are expected to be particularly high in litigating populations and in patients who seek other forms of external gain, like those, for example, with repeated and/or prolonged sick leaves.

As has been previously pointed out by Rosen (2004) and Rubenzer (2009) in the context of posttraumatic stress disorder, false-positive diagnoses contaminate the scientific database, particularly that of medically unexplained symptoms and related conditions. These databases must be expected to be skewed and inaccurate to an unknown degree. Thus, when studying cognitive test profiles in mental disorders, the results will strongly depend upon the proportion of patients in the sample who are litigating or who expect any kind of substantial gain from poor test results.

Published studies usually present neither a detailed account of the number of litigating participants in the samples, nor do they take sufficient care to test for the presence of negative response bias with SVTs.

More than a decade ago, some researchers warned that studies with patient samples that are potentially contaminated by litigating research participants, may not only yield uninterpretable results, but will also become unpublishable (Hartman, 2002). Although a number of clinical studies do include SVTs today, this is far from being common practice and it seems to be a mere illusion to expect that matters will change in the research domain of medically unexplained symptoms in the foreseeable future. The problem has more recently been reformulated in the context of complex regional pain syndrome by Victor, Boone, and Kulick (2010), who stated:

In the absence of adequate consideration of compensation status and effort, such conclusions are likely inaccurate, and it is our belief that the practice of continuing to publish such papers harms the field through the perpetuation of misleading information. The clinical impact of studies neglecting these factors is potentially damaging. (p. 1151)

Hidden agendas may also be implied in individuals who present at memory clinics for an assessment of possible dementia or mild cognitive impairment, especially when they are of working population age and their activities of daily living are intact. A case in point is a recent study by Rienstra et al. (2013), who examined the relationship between hippocampal volume and memory test performance in patients who were suspected to be in the early stages of dementia. A substantial correlation between volumetric measures and memory was obtained, but not for a subgroup of patients who failed on SVTs. Depending on the percentage of uncooperative patients in a given sample, such correlations may be substantially attenuated or even disappear, with distorting effects on theory building.

Conclusions

The old debate about how to best discriminate between subjective complaints not substantiated by medical findings (hysteria or functional, somatoform, conversion, and dissociative disorders, or more recently, medically unexplained symptoms), malingering, and factitious disorder has not been resolved to a satisfying degree. Relying on newer, but similar diagnostic labels, this debate is unabatedly haunting current clinical and forensic science and practice alike. Most authors would agree that the various diagnostic labels that are discussed in this context do not constitute distinct entities (although *pure* forms of malingering, somatoform disorder,

and so on may exist). They rather lie on several continua (e.g., Boone, 2007a; Turner, 1997): a continuum between self-deception and other deception, a continuum in the degree of reflection (or consciousness) about this deception, and a continuum between external and internal gains. As far as the latter continuum is concerned, in social welfare states there is almost always some form of external gain present (such as sick leave and sick pay). Furthermore, it appears to be indisputable that over time, the position of a patient on these continua may shift (e.g., Brady, 1966; Delis & Wetter, 2007; see for a recent discussion Merckelbach and Merten (2012)), and it may shift repeatedly and in various directions.

The decisive factor in the differential diagnosis continues to be subjective judgment by clinical and forensic experts. That judgment is influenced by the degree to which experts rely on knowledge of the empirical literature; logical stringency in their thinking and in their argumentation; their subjective confidence; and the degree to which they accept doubt. Confirmatory bias appears to be a relevant factor to consider. Important lessons may be learned from research on intuitive decision making. “True experts, it is said, know when they don’t know. However, nonexperts (whether or not they think they are) certainly do not know when they don’t know. Subjective confidence is therefore an unreliable indication of the validity of intuitive judgments and decisions” (Kahneman & Klein, 2009, p. 524).

What we know for sure is that in forensic contexts, a substantial portion of patients with minor psychopathology fail on SVTs. Some authors believe, almost as an article of faith, that the claimed psychopathology itself is a sufficient condition to explain such failure. However, this psychopathology = superordinate doctrine or “pathologization of malingering” as Kanaan and Wessely (2010) called it is not justified by currently available empirical research and invites, in fact, circular argumentation. Given the current knowledge base, SVT failure should be interpreted as uncooperativeness. Such an interpretation is dictated by *lex parsimoniae* or Occam’s razor: among competing scientific explanations, the one should be preferred that makes the fewest assumptions. The assumption that unconscious psychopathology causes failure in cognitive SVTs is not only more complex than that of uncooperativeness, but it resorts to additional and, more importantly, untestable conjectures (e.g., the idea that there is a causal link between genuine psychopathology and negative response bias).

Promises of functional neuroimaging techniques to reliably differentiate between conscious feigning and somatoform or dissociative disorders (e.g., Spence, Crimlisk, Cope, Ron, & Grasby, 2000) have remained unfulfilled (for a discussion, Kingery and Schretlen (2007), Ruchow, Hermlle, and Kober (2010), and Van Hooff (2008)). Interestingly, a number of authors continue to cite the Spence et al. (2000) study as strong support for the assumption that patients with somatoform or

dissociative disorders do not intentionally feign symptoms. However, a critical analysis of methodology and interpretation of this and related studies must come to the conclusion that the results are, at best, preliminary and in urgent need of replication and refinement (Nicholson et al., 2011).

The medical or psychological expert witness should be cautious not to fall into the trap of explaining away SVT failure or other signs or uncooperativeness by speculative psychological factors (such as cry for help) unless there is clear and independent evidence that such factors serve as causative antecedents. Also, (s)he should not fall into the trap of judging *in dubio pro aegroto*—in cases of doubt, conclude in favor of the patient. This principle does not apply to civil and social legislation (at least not in Western European states). When the forensic expert would follow this principle, (s) he may be suspected of leaving the ground of impartiality. Rather, doubt should be expressed in an appropriate way and be detailed to such a degree that the trier of fact is in the position to apply relevant law to the case at hand. Rosen and Phillips (2004) gave the advice that “when questioned about the actual occurrence of subjective symptoms, or the truthfulness of a patient’s report, the wise clinician would do well to be less than certain” (p. 133).

There appears to be no a priori reason why a patient with medically unexplained symptoms could *not* be cooperative during a psychological evaluation, so as to enable the expert to arrive at a valid diagnosis. After all, full cooperation is what we also expect from and observe in patients with serious brain injury. And indeed, a substantial proportion of patients with somatoform disorders behave in a cooperative way. Moreover, it has been held that patients with conversion disorder are thought to be even *more likely* to be cooperative (e.g., Resnick, West, & Payne, 2008). Thus, the presence of minor psychopathology does not *in itself* imply uncooperativeness. If a patient exhibits negative response bias, the legal consequences of poor cooperation are not a matter for the forensic expert. The expert’s role is to determine whether there is any reason to suspect that poor cooperation is directly and causally linked with psychopathology. A direct link may be accepted, for instance, in patients suffering from anhedonia or lack of initiative, as seen for example in schizophrenia (Gorissen, Sanz, & Schmand, 2005), in patients with the most severe forms of a depressive episode or a subset of patients with frontal lobe damage. To be sure, failure on SVTs may represent a false-positive finding, for example in moderate or severe dementia. Yet, we would not expect to find below-chance performance in cooperative patients with dementia, schizophrenia, or severe depression.

There is no reason to believe that malingering (i.e., the *conscious* act of inventing or exaggerating symptoms) occurs less frequently in patients with claimed or genuine somatoform disorders than in patients with other mental

disorders that are either claimed or genuine, like depression or posttraumatic stress disorder. Also, it would be wrong to think that genuine mental disorders cannot coexist with malingering in a situation where substantial external gain is at stake. Likewise, it would be wrong to assume that symptoms of somatoform disorder—or any other mental disorder for that matter—cannot be invented altogether to obtain such a gain.

With these considerations in mind, we would argue that negative response bias must be named by what it is: lack of cooperation. In the presence of an identifiable external goal, malingering will be the primary conclusion. This is, indeed, the route that is recommended by DSM-IV (American Psychiatric Association, 1995): “... the presence of some factitious or malingered symptoms, mixed with other nonintentional symptoms, is not uncommon... In such mixed cases, both Somatization Disorder and a Factitious Disorder or Malingering should be diagnosed” (p. 461).

There are authors who opine that malingering and psychopathology are exclusive categories. Boone (2007b) wrote that: “Clearly, somatoform disorder needs to be ruled out before a diagnosis of malingered cognitive dysfunction can be made” (p. 31). The problem is how this squares with the DSM-IV criterion of the exclusion of malingering for the diagnosis of somatoform disorder. If experts would follow Boone’s recommendation, neither somatoform disorder nor malingering could be determined in any case in which SVT failure points to negative response bias.

As a preliminary solution to this conceptual dilemma, we propose that for the diagnosis of somatoform disorders and other forms of medically unexplained symptoms, it may be useful to formulate criteria for different degrees of diagnostic certainty (definite, probable, or possible), as has been done for malingering (Slick et al., 1999) and for Alzheimer’s Disease (McKhann et al., 2011). Although it is beyond the scope of this review to articulate such criteria, we strongly believe that presence of negative response bias both in symptom report and cognitive SVTs should play a major role in the degree of certainty that can be ascribed to a DSM-IV diagnosis.

For a careful differential diagnosis, it appears inevitable to include modern SVTs on a larger scale in clinical contexts than is currently done. We agree with Lamberty (2008) who wrote:

In a sense, symptom validity measures, including performance related measures (e.g., PDRT, TOMM, WMT), embedded measures of symptom validity, and specialized validity indices from standard personality measures (e.g., FBS, HHI, RBS), may well represent the technology that helps to move the current set of somatoform disorders into a different realm of understanding. (p. 66)

Negative response bias in neuropsychological assessment and other evidence for poor cooperation should provide

grounds for doubt as to the genuineness of claimed psychopathology and functional impairment. In the presence of such evidence, the degree of certainty of a DSM-IV diagnosis should be scaled down. The presence of below-chance response patterns should further diminish the degree of certainty that an expert can ascribe to a DSM-IV diagnosis. Of course, uncooperativeness and malingering per se never exclude the presence of a mental disorder. However, the reverse is as much true, and that is the take home message of this paper.

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