

Validity assessment and the neurological physical examination

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Abstract.

BACKGROUND: The assessment of any patient or examinee with neurological impairment, whether acquired or congenital, provides a key set of data points in the context of developing accurate diagnostic impressions and implementing an appropriate neurorehabilitation program. As part of that assessment, the neurological physical exam is an extremely important component of the overall neurological assessment.

PURPOSE: In the aforementioned context, clinicians often are confounded by unusual, atypical or unexplainable physical exam findings that bring into question the organicity, veracity, and/or underlying cause of the observed clinical presentation. The purpose of this review is to provide readers with general directions and specific caveats regarding validity assessment in the context of the neurological physical exam.

CONCLUSIONS: It is of utmost importance for health care practitioners to be aware of assessment methodologies that may assist in determining the validity of the neurological physical exam and differentiating organic from non-organic/functional impairments. Maybe more importantly, the limitations of many commonly used strategies for assessment of non-organicity should be recognized and consider prior to labeling observed physical findings on neurological exam as non-organic or functional.

Keywords: Neurological exam, validity assessment, effort, functional neurologic disorder, malingering, symptom exaggeration

1. Introduction

The neurological physical assessment of persons with congenital or acquired neurological impairment can be complex and fraught with challenges regarding both form and interpretation. These challenges are more significant the less specifically trained the clinician is in neurological assessment and validity assessment more specifically. One key issue that is often inadequately

taught during professional training is that of validity assessment in the context of conducting the neurological exam. As neurorehabilitation professionals, it is important to understand the nuances involved in thorough neurological physical examination. There is a clear need for understanding specific validity assessment techniques that can be utilized in this context and to understand how these findings should be integrated with the rest of the neurological exam. Studies have shown that doctors are relatively poor at differentiating functional findings from consciously embellished or simulated ones (Stone, 2008). Clinicians should also learn how to interpret abnormal findings as they reveal

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themselves and understand if, in the context of the condition being examined, such findings make sense relative to the spectrum of neurological impairment associated with that condition.

Clinicians must remember that “abnormal” neurological physical exam findings may be organic, non-organic or a combination of both (Shaibani & Sabbagh, 1998). It must also be remembered that psychogenic disorders coexist with organic neurologic disorders in up to 60 percent of patients, making them diagnoses of exclusion (Enslin & Taylor, 2013). As an example of the prevalence of non-organic presentations, symptoms considered functional or medically unexplained account for about one third of all new referrals to neurology outpatient departments/clinics (Gould, Miller, Goldberg & Benson, 1988; Stone, Zeman, & Sharpe, 2002).

Validity assessment, in general and as specific to the neurological physical exam, is paramount in both clinical and clinicolegal contexts, as without such assessment practitioners cannot develop medically probable opinions on an objective, evidence-based foundation nor come to accurate conclusions about the true degree of impairment resulting from neurological injury or disease. As clinicians, we are taught to believe our patients (not just what they say but how they present on exam). This practice and inherent training bias tends to color our approach to assessment and may narrow the scope of differential diagnostic considerations, particularly when evaluating more complex cases. As clinicians, we must understand that patients and clinicians alike have biases that may enter into the mix of sign and symptom reporting, neurological physical examination findings, as well as clinician interpretation of same (Martelli, Nicholson, & Zasler, 2013). In a medicolegal context, such as an independent medical exam, sophisticated validity assessment is paramount as there are already substantive hurdles to face when doing exams in this context related to accurate impairment evaluation (Busse et al., 2014). Good clinical or medicolegal assessment requires an eye to detail and inclusion of reasonable measures to assure the validity of reported signs, as well as symptoms. A critical piece of the art of validity assessment is to assure that optimal effort is being exerted by the patient or examinee in the context of specific neurological exam assessments (Larrabee, 2012).

It is important to acknowledge the differences between performance and symptom validity as elaborated by Larrabee (2012) and relevant not only to neuropsychology but all clinician assessments. Performance validity refers to the validity of actual test

performance, whether neuropsychological or otherwise; whereas, symptom validity refers to the validity of the actual symptom report (whether physical, cognitive or otherwise). Clinicians need to become familiar with how to gauge both types of validity; however, in the context of the current discussion there will be a focus on performance validity. It is important to consider both types of validity assessment when looking at any case from a holistic standpoint, and readers are referred to other articles in this issue that address the topic of symptom validity assessment.

Various factors can produce invalid neurological exam physical findings and/or interpretations including patient/examinee, as well as clinician/examiner, biases, as well as sub-optimal patient/examinee effort and engagement. Importantly, effort testing has been explored in the context of medically unexplained symptoms, and it may surprise most clinicians that it was found to be quite low (Kemp et al., 2008). Taking a careful and detailed history is important in the context of validity assessment. Historical facts such as a prior history of functional complaints, multiple lawsuits and injuries, models for illness, affective illness, external incentives, primary gain incentives, ongoing litigation, recent stressors, illness beliefs (including those culturally based) and childhood/prior traumatic life experiences (i.e., physical, emotional or sexual abuse) must all be taken into consideration (Martelli, Nicholson, & Zasler, 2013). In the context of interpretation of findings, examining practitioners should understand the neurology and neuropathology of the neurological disease process being evaluated (if known) and the neuropathology, if any, in those conditions that may not have a diagnostic label (i.e., based on brain imaging findings, SSEPs, VEPs, among other tests).

It is important to mention that some clinician researchers have argued that symptom validity testing and effort assessment in persons with brain impairment may be flawed conceptually secondary to disrupted neurobiological mechanisms associated with drive, effort, and motivation, which may be adversely impacted by acquired brain impairment (Bigler, 2012). Criticisms of such testing have included the lack of a more rigorous and formal definition and application of the term “effort”, the absence of lesion location studies in symptom validity research, the methodological soundness of symptom validity research designs (or deficiency associated with same), problems with cut scores for such testing, the concerns regarding diagnosis threat relative to performance validity, and the lack of adequate peer reviewed literature examining the cognitive

neuroscience of symptom validity performance (Bigler, 2012, 2014).

Functional neurological physical exam abnormalities may be pseudo-neurological in origin secondary to disorders such as conversion disorder (now termed functional neurological symptom disorder [FNSD]) (American Psychiatric Association, 2013), exaggerated (implying that some level of impairment is present but is being amplified), suppressed, or feigned, the latter either in the context of malingering or factitious disorder (Reuber, Mitchell, Howlett, Crimlisk, & Grunewald, 2005). Clinicians should also be familiar with some of the more common FNSDs including pseudoparalysis, pseudosenosry syndromes, non-epileptic psychogenic seizures, pseudocoma, functional gait disorders, pseudo-neuroophthalmologic syndromes and functional aphonia (Shaibani et al., 1998), as well as general rehabilitation principles applicable to patients with functional presentations (Zasler & Martelli, 2002).

This article reviews validity assessment in the context of the neurological physical exam and includes specific recommendations on examination techniques for a variety of neurological physical impairments, as well as interpretation caveats for same.

The topics of cognitive, behavioral, and language validity assessment in the context of neurological and neurorehabilitation evaluations are addressed in other articles in this special issue of *NeuroRehabilitation* edited by Dr. Shane Bush.

2. General approaches

When conducting the neurological physical examination, clinicians need to understand the patient's history regarding both pre-onset (as relevant) and post-onset impairment relative to ascertaining apportionment of abnormal findings. Clinicians also need to understand the patient's history regarding the nature of the initial medical insult or traumatic injury including as relevant: biomechanics of injury, injury severity parameters, acute diagnostic test findings and condition prognosis, the latter based on neurologic impairment cause, degree of CNS pathology and natural history of the neurological condition. All the aforementioned need to be considered in the context of interpreting the neurological physical exam findings.

If findings do not make sense relative to the primary condition being evaluated, then other more parsimonious explanations need to be sought. Clinicians should understand that abnormal neurological physical exam

findings may be signs of not only the primary condition being evaluated but also comorbid neurological conditions. For example, the patient with a C4 complete spinal cord injury who also had a concurrent brain injury is going to have an array of upper motor neuron impairments that may be due to both injuries or either one of the injuries. It should also be understood that clinicians may encounter unusual, bizarre, and potentially rare or atypical findings on neurological physical examination that in fact are attributable to the underlying neurological disorder (e.g., kinetic tremor after mild traumatic brain injury or parietal lobe drift after cerebrovascular insult to the non-dominant parietal lobe). There is a tendency to conclude that atypical presentations and/or presentations never before observed by the clinician are more likely to be non-neurological in origin; this is particularly so with young women, homosexual men and patients with psychiatric issues, often with gender dependent bias on the part of the examiner (Campbell, 2012).

In the context of the neurological physical assessment, it is also of utmost importance for clinicians to understand what types of physical findings are common, as well as potentially uncommon but associated with the disorder they are evaluating. When a finding falls outside of the range of impairments associated with the disorder, then the clinician can further scrutinize the overall clinical presentation to decide if the finding is organic in nature or of a functional etiology. Etiologies of exam findings may include pseudo-neurological findings associated with FNSD (i.e., conversion), symptom exaggeration of real physical neurological impairment, or feigned impairment as would be seen in factitious disorder or malingering. As an example, visual impairments after concussion may include blurry vision, double vision, and problems with close reading due to convergence or accommodative insufficiency, among other problems. When a patient following MTBI complains of tunnel vision, scotomas in their visual field or triple vision, then the examiner's mindset must shift accordingly to address such unexpected reports and/or findings.

It is also paramount for examining clinicians to understand what types of neurological physical exam findings should be seen in association with other findings.

For example, a patient with a claimed hemiparesis at six months post injury/illness onset should have other neurological physical exam findings including asymmetrical deep tendon reflexes, pronator drift on upper extremity drift testing contralateral to the side of the

brain insult, spasticity in the affected limbs, a hemiparetic gait pattern and central facial weakness on the hemiparetic side. If the findings are not consistent with expectancy, then the clinician must explain the findings in a way that appropriately accounts for not just the physical findings, but also their consistency across time and exam situation, the correlation of said findings with multimodal neurodiagnostic testing (such as Goldmann visual field analysis, cerebral MRI, EEG, neuropsychological testing, among other assessments), and the nature and neuropathology of the original injury.

Clinicians should also be familiar with specific techniques that may be helpful in the context of validity assessment during the neurological physical exam such as Hoover's tests, the Bowls-Currier test, Honest Palm Sign, among others, as well as more general approaches to validity assessment using such techniques as forced choice testing. Forced choice testing (FCT) is well described in the literature as a methodology to assess the veracity of claimed impairment and involves developing a "yes/no" paradigm testing situation for the particular impairment and then tracking whether the patient or examinee responds at a rate significantly less than chance which would strongly indicate conscious distortion of exam findings. Additionally, with FCT, persons with various disorders or severity of disorders may be expected to perform considerably above chance, so use of appropriate normative comparison groups can be valuable. FCT is inadequately taught to neurorehabilitation professionals, aside from psychologists and neuropsychologists, but is an important tool to be familiar with in the context of validity assessment, neurological and otherwise (Martelli, Nicholson, & Zasler, 2013; Merten & Merckelbach, 2013).

3. Factors that may influence the validity of neurological physical exam findings

There are likely multiple factors that have the potential to influence the validity of neurological physical exam findings and general clinical presentation. Such factors can include primary and secondary gain incentives, suboptimal or poor effort, symptom minimization, symptom exaggeration, malingering, factitious disorder, cultural perspectives on neurological disability, inherent coping resources, feelings of loss of nidus of internal control, diagnosis threat, as well as nocebo effects of an inappropriate neurological diagnosis, among the more common ones seen in general neurorehabilitation clinical practice (Martelli,

Nicholson, & Zasler, 2013). It is critical, therefore, to take these potential factors into consideration when assessing a patient or examinee. The only real way to do so is to take the time to thoroughly review all relevant records, ask appropriate questions, as well as to conduct specific exams, both stand alone and embedded, for sign and symptom validity checks in the broader context of holistic patient assessment.

Patient distractibility, as well as lack of engagement, can also impact neurological exam findings, as can fatigue (i.e., in MS or TBI). Ideally, during the neurological exam, the clinician should ascertain if the patient is, and encourage the patient to be, fully attentive and engaged in the exam process. Certain medications may impact the presence and/or degree of neurological exam findings and must also be taken into consideration (i.e., anti-spasticity medications).

4. A systematic approach to neurological physical exam validity assessment

4.1. Cranial nerves

When examining the cranial nerves it is important to use objective and standardized assessment techniques as much as possible. When testing cranial nerve one, which is often ignored even though smell impairment is a good marker of executive dysfunction and risk of frontotemporal contusional injury, commercially available tests like the Alberta Smell Test (Fortin, Lefebvre, & Ptitto, 2010) or the Sensonics Smell Identification Test series should be used (particularly the 40-item version which has malingering criteria based on the provided nomogram combining age and smell test score) (Callahan & Hinkebein, 2002). Visual acuity should be assessed using a Snellen Chart at 20 feet; however, if there are concerns about the validity of the findings, have the patient step back another 20 feet (without informing them of the distance) and then check acuity in each eye at 40, 30 and again at 20 feet to assess for consistency if there are doubts regarding validity. Photosensitivity is generally considered a totally subjective complaint; however, there are now formal neuro-optometric assessment methods that can corroborate this complaint including abnormal (relative to normative data) thresholds for contrast sensitivity, scotopic sensitivity, critical flicker frequency, pattern glare and coherent motion (Kapoor, 2012).

If there are concerns regarding non-organic visual field loss such as tunnel vision on confrontation testing

(remember to test both eyes separately and together) and/or subjective patient report, then formal visual field perimetry testing (e.g., Goldmann, tangent or automated) can be requested. It is critical to remember that in the case of tunnel vision there are several organic etiologies that can be responsible for this complaint and it should not be assumed to be non-organic. Functional hemianopias are uncommon and usually occur in association with other lateralized complaints. The most frequent pattern consists of decreased vision in one eye, an ipsilateral hemianopia on testing the “affected” eye, full fields in the other eye, and a complete hemianopia toward the affected side on testing with both eyes open. The incompatibility of the monocular and binocular fields provides evidence of the functional nature of this visual loss. Additional tests that may be of value to objectify visual impairments include cerebral imaging, as well as multifocal visual evoked potentials (Bruce & Newman, 2010; McBain, Robson, Hogg, & Holder, 2007).

Bedside assessment of cranial nerves 3, 4 and 6 as related to extra-ocular muscle dysfunction is fairly straightforward (Campbell, 2012) and impairments related to these cranial nerves are generally not seen in clinical practice on a non-neurogenic/non-organic basis. If there is impairment in cranial nerve 5 function based on testing of any of the three sensory branches in the face, then the clinician should test all cranial nerve 5 sensory nerve branches in the face, as well as smell detection of noxious odors such as ammonia. Devices such as monofilaments (Von Frey hairs/filaments or Semmes and Weinstein monofilaments) can also be used to more precisely and objectively quantify sensory loss in any of the 3 branches of cranial nerve 5 and determine consistency across trials (Dellon, Mackinnon, & Brandt, 1993; Keizer, van Wijhe, Post, Uges, & Wierda, 2007).

Cranial nerve 7 is assessed via inspection for facial asymmetry and involuntary movements. Motor exam should include asking the patient to raise both eyebrows, frown, close eyes tightly, smile to show teeth and puff out one’s cheeks (Campbell, 2012). Taste testing can also be done if one has appropriate gustatory function testing materials. Smiling in response to request is mediated via different neural pathways than spontaneous smiling and may produce “disparate” clinical results. To assure full effort is made when testing eyelid closure ability, the examining clinician should check to see if the patient’s eyes are deviated downwards during the effortful part of the task. Pseudoptosis or functional ptosis may also be found on exam. The clinician should be aware that functional eyelid weakness, when unilateral, presents with a

persistently depressed eyebrow with a variable ability, or lack thereof, to elevate the frontalis muscle in association with orbicularis over-activity and photophobia; whereas, organic unilateral ptosis is usually associated with frontalis over-activity (Stone, Carson, & Sharpe, 2005).

Cranial nerve 8 assessment involves both auditory and vestibular system elements (Sanders & Gillig, 2010). Auditory assessment should look for the presence of hearing loss (conductive, sensorineural or non-organic), presence of phonosensitivity, as well as hyperacusis (Campbell, 2012). Tinnitus may be one of the most challenging subjective complaints to objectify and it is certainly not feasible to do so by traditional bedside exam as it requires audiological assessment via such methodologies as tinnitus frequency matching (Coelho & Hoffer, 2013; Henry, Dennis, & Schecter, 2005). In cases where there is a concern regarding non-organic hearing loss, the patient can be referred for more comprehensive audiological testing including the Stenger’s test for dissimulated hearing loss and other tests for functional/dissimulated hearing (Lin & Staecker, 2006; Mehta & Singh, 2000).

Assessment of cranial nerve 8 and its role in vestibular function is more challenging as bedside exam has limited sensitivity and specificity to these types of impairments. There are numerous causes for the non-specific complaint of dizziness which may be related to vertigo, postural lightheadedness, pre-syncope, vertebralbasilar insufficiency, postural instability, migraine, as well as epilepsy, as the most common causes. Getting an accurate history of the complaint of dizziness is therefore critical. As clinically indicated, the clinician should consider ordering additional tests for objectification of impairment including electronystagmography with calorics and dynamic posturography, among others depending on the diagnostic impressions and/or rule-outs. Other causes of vestibular-type symptoms should also be considered including TMJD, referred autonomic myofascial symptoms, endolymphatic hydrops, perilymphatic fistulas, and cervicogenic dizziness before a complaint is potentially labeled as non-organic (Iverson, Lange, Gaetz, Zasler, 2013; Shepard, Handelsman, & Clendaniel, 2013).

Examination of cranial nerves 9 and 10 is simplified in part due to the difficulty for a patient to consciously modify exam findings. Articulation, both palatal and guttural, should be assessed, as well as symmetry of palatal arch movement (check for asymmetric movement indicating a pathological finding like a curtain reflex) and gag response (Campbell, 2012).

Cranial nerve 11 assessment should involve testing motor strength of shoulder elevators and head rotator musculature. Cranial nerve 12 should be assessed by having the patient protract their tongue and move it side to side (inside and outside the mouth). Check for any abnormal movements such as fasciculations, myoclonus, asymmetric protraction or unilateral tongue atrophy (Campbell, 2012).

4.2. *Deep tendon reflex exam*

Abnormally increased reflexes, particularly when asymmetric, may be associated with upper motor neuron lesions (i.e., along the neural axis). Deep tendon reflexes (DTRs) can be influenced by age, metabolic factors such as thyroid dysfunction or electrolyte abnormalities, anxiety, and volitional suppression or exaggeration. Signs of hyperreflexia *may* include spreading of reflexes to other muscles not directly being tested, crossed adduction of the opposite leg when the medial aspect of the knee is tapped, as well as clonus.

DTRs may be diminished by abnormalities in muscles, sensory neurons, lower motor neurons, neuromuscular junction, acute upper motor neuron lesions, as well as by volitional suppression. If there are concerns regarding the presence of upper motor neuron findings on DTR examination, the clinician can reinforce the DTR through various different measures, as well as perform other tests that may be consistent with an efferent motor system lesion including Hoffman's or Tromner's reflex for the upper extremity or a Babinski reflex and as necessary associated reflexes (i.e., Stransky, Oppenheim, Chaddock) for the lower extremity (Campbell, 2012). DTRs are constant in neurological disease and therefore one way to assess for veracity is to test them with the involved limb in different positions and/or test them with the patient standing, laying supine or laying prone, or at different times during the exam, among other options. If the findings vary across testing paradigms then this is a clue to a potential non-neurogenic etiology.

4.3. *Sensory exam*

The sensory exam is a common area where clinicians may find evidence of non-organic findings and one of the most common areas of pseudo-neurologic presentation (Rolak, 1988). The sensory exam should include testing of pinprick, light touch and proprioception sensations which respectively tap the lateral spinothalamic, anterolateral spinothalamic and anterolateral spinothalamic tracts. Certainly, additional testing can also be

performed examining such functions as graphesthesia, tactile gnosis, two point discrimination, and pain, among other modalities.

There is debate on whether a patient's eyes should be closed during the majority of the sensory exam. Some have advocated that this is a good way to check validity and additionally suggest that rather than asking "do you feel this?", ask the patient to let the examiner know when they do feel something and if they do to then describe it. Some believe that the latter methodology is "trickery" and should not be performed; however, this author would recommend that the technique be used as a second level check when the sensory exam findings are unclear.

Organic sensory abnormalities tend to follow "distribution rules" including central patterns with lesions in the afferent pathways of the brain (whether cortical or sub-cortical), radicular syndromes causing dermatomal sensory loss, peripheral neuropathic disease causing stocking glove distal sensory impairment, and spinal cord injury producing a sensory level (complete or incomplete), with central cord syndrome producing a suspended sensory level. Peripheral nerve injuries have specific patterns of sensory loss with associated patterns of autonomic change. It is neurological dogma that organic neurological sensory loss does not vacillate although other impairments may do so including various motor functions such as speech and gait quality (Lanska, 2006).

Functional sensory loss typically affects all sensory modalities in either a hemibody or single limb distribution including vision, hearing and smell. Such sensory complaints often manifest as numbness but on exam tend to yield inconsistent exam findings and non-anatomic distributions to same, incongruent with normal sensory innervation patterns. Some of the more common patterns of functional sensory impairment include midline split of sensory loss, abrupt changes in sensation from normal to abnormal across joints, abrupt transitions in vibration sense along the same bone (with classic examples being the frontal bone or sternum) (Lanska, 2006). It should be remembered to test the front of the body, as well as the back of the body, to look for consistency of findings. True hemianesthesia does not split the genitalia due to overlapping innervation. Clinicians must realize that there are conditions such as myofascial pain syndromes that can present with patchy areas of sensory change that need to be differentiated from non-organic/functional sensory losses.

There are some caveats regarding the specificity of traditionally viewed functional sensory findings that are worth noting. It is common thinking that exact

splitting of the midline cannot occur in organic disease due to the overlap in cutaneous branches of the intercostal nerves from the contralateral side which creates a sensory demarcation of one or two centimeters off the midline. Research has shown, however, that midline splitting can occur in organic neurologic conditions specifically in association with thalamic stroke. In thalamic stroke, one can see a profound loss of several sensory modalities. Several studies have shown functional imaging changes in hemisensory loss, as well as a significant frequency of midline splitting in association with organic disease (Stone, Carson, & Sharpe, 2005). Theoretically, splitting of vibratory sense across bones that cross the midline, such as the sternum or frontal bone, should not occur; yet, research has shown that this can take place in patients with organic disease (Lanska, 2006; Stone, Zeman, & Sharpe, 2002) bringing into question our current understanding of the neural correlates of sensory system function. The bottom line on these types of sensory findings is that, in and of themselves, they are not pathognomonic for non-organicity but should be considered in the context of the entirety of the presentation. Such findings can also be seen in both functional disorders and feigned disorders such as malingering or factitious presentations.

Other techniques like the Bowls-Currier Test can be used to confirm upper extremity sensory loss involving the arms and fingers. The test basically relies on the principle of “sensory confusion” and involves asking the patient to clasp their hands together with the hands crossed right to left with their fingers interdigitated, extend the arms and internally rotate them so that the thumbs are pointing downward and the palms facing outwards while the shoulders are extend to bring the upper arms to the side of the body with the elbows flexed at about 45 degrees. The patient is then asked to close their eyes and the examiner, using pinprick or light touch, rapidly and randomly touches fingers of both hands and requests the patient to tell them, as quickly as possible, which side was touched. This test is very challenging for patients without true sensory loss to consistently evidence the same sensory impairment. Patients with non-organic sensory loss will not only have inconsistent findings but also tend to respond with greater hesitation to each stimulus (Lanska, 2006; Shaibani, & Sabbagh, 1998).

The examining clinician should also assess for discrepancies in test performance, such as scoring well below chance on proprioceptive testing at the ankle and correlating this finding with the presence of impaired versus non-impaired functional abilities such as gait quality.

If the patient has a low rate of correct responses on testing (i.e., lower than if the person was just guessing), one would expect significant proprioceptive gait impairment if in fact the findings were real. If gait impairment is not present in such a patient, then one would more likely conclude a “mismatch” suggesting a non-organic cause (Zasler & Martelli, 2002).

4.4. *Cerebellar exam*

As with other elements of the neurological exam, it is important for the examining clinician to understand the constellation of signs (as well as symptoms) associated with cerebellar disease or insult. Some of the more classic signs of cerebellar neurological dysfunction include nystagmus (in primary gaze and/or with fixation) and other oculomotor abnormalities, dysmetria, action tremor (also inappropriately referred to as intention tremor), ataxia including ataxia of gait, rebound and check phenomena, pendular knee jerk, hypotonia, dysdiadochokinesia, as well as cerebellar dysarthria (think of it as an ataxia of speech) (Campbell, 2012).

Given that gait abnormalities are one of the more common non-neurologic and sometimes functional presentations seen, it is important to understand how true ataxic gait will present on exam. Bilateral cerebellar disease results in a lurching, reeling, staggering gait quality that can be confused with astasia abasia (although in the latter, patients will very rarely fall to the floor). Okun et al. (2007) have described the “chair test” as an aid to assessment of psychogenic/non-organic gait disorders including astasia-abasia. Unilateral cerebellar disease will cause gait abnormalities and deviation ipsilateral to the side of the injury or lesion. Tandem gait assessment is probably the most sensitive test to gauge the integrity of cerebellar vermal function. It is important to remember that gait ataxia may also be proprioceptive associated with posterior column disease and less commonly parietal injury. Severe vestibular disease may also present with gait abnormalities that can be confused with ataxia. The Romberg test is a test of posterior column function related to proprioception and not cerebellar function. Balance relies on an integration of at least two of three systems (proprioceptive, visual and vestibular). If the patient sways or loses balance once the eyes are closed and visual input negated, then one of the two other sensory systems is awry (Campbell, 2012; Shepard, Handelsman, & Clendaniel, 2013).

Decomposition of limb movement including trajectory disturbances like dysmetria occur with lateral zone involvement of the cerebellum, as do

dysdiadochokinesis. Hypotonia and pendular reflexes are noted when there is unilateral cerebellar disease. Titubation can also be seen with cerebellar involvement, particularly midline, and involves a tremor of the head or body (think bobble animals that people used to put in their cars) (Campbell, 2012).

Dysarthria is generally only found in severe cases of cerebellar injury or disease and presents with uneven articulation, “scanning and measured speech”, word slurring and variability in loudness and pitch. If dysarthria is suspected to be of functional origin, then other qualities of speech should be explored and use of drug-assisted interviews can be considered (Nichols, Zasler, & Martelli, 2012).

4.5. *Motor exam*

There are a number of methods used to assess functional weakness whether due to feigning or FNSD. Functional weakness typically presents with weakness or paralysis of an entire limb, the lower extremities or the hemibody. Historical dogma was that functional hemiparesis was more common on the left but this claim has been refuted (Jones, 1908; Stone et al., 2002). There is also debate regarding the use of muscle testing for determination of sincerity of effort as noted by Robinson and Dannecker (2004). They noted considerable issues with limitations of submaximal contractions versus maximal contractions, concerns about methodology including the lack of reliable and clinically relevant methods for differentiating effort levels. They espoused for the need to take into consideration fear of injury, pain, medications, work satisfaction and other motivational factors in the context of such testing.

Assessing movement during wakefulness as well as sleep can be helpful in establishing if the affected body parts are truly paretic or plegic. Observe the patient. Watch how they undress or don clothing, inspect assistive devices and/or shoes for appropriate wear if there is a gait impairment, inspect gait quality both observed and “unobserved” (i.e., patient walking into your office or out of your office), and review surveillance data if it is available (Lanska, 2006).

Negative findings on exam that would support a non-neurogenic motor impairment involving weakness include normotonicity and symmetric DTRs; absence of muscle atrophy, vasomotor changes, and/or contractures; lack of facial weakness if presenting as hemibody involvement; and lack of sphincter disturbances in paraplegia or tetraplegia (Lanska, 2006; Reuber, Mitchell,

Howlett, Crimlisk, & Grunewald, 2005; Shaibani, & Sabbagh, 1998).

Significant limb or other body part weakness in the absence of indicators of peripheral nerve dysfunction should be associated with distal greater than proximal weakness as a rule. Differences in tested muscle strength versus functional use of the limb or body may also suggest a non-neurogenic etiology to the weakness. Give-way weakness may also be a sign of non-neurologic motor impairment although this can sometimes be seen in conditions associated with rapid muscle fatigue. Pseudowaxy flexibility and co-contraction are other signs that have been associated with functional weakness.

Movement disorders may also be encountered in the context of the physical neurological exam. Psychogenic movement disorders, including dystonia, typically present with inconsistent or incongruous movements which do not appear organic, which do not respond to “tricks” that normally modulate real movement disorders, that are seen in conjunction with other pseudo-neurological features, that have acute onset with fixed posturing or that have a cyclical presentation with periods of remission in between periods of exacerbation (Fahn & Williams, 1988). Psychogenic movement disorders must be differentiated from movement disorders associated with neurological disease, as well as psychiatric disease, the latter including medication-induced side effects such as dyskinesias with antipsychotics (Lees, 2002). The Shill-Gerber diagnostic criteria for psychogenic movement disorder were published in 2006 but did not consider neurological phenomenology when diagnosing psychogenic movement disorders (Shil & Gerber, 2006). An earlier set of diagnostic criteria for psychogenic movement disorders, which is still commonly used, was published in 1988 by Fahn and Williams, although never validated (Fahn & Williams, 1988). Polymyography coupled to accelerometry can be used to demonstrate the major electrophysiological criteria of psychogenic tremor, namely spontaneous variability of tremor frequency and frequency entrainment induced by contralateral rhythmic tasks (Apartis, 2014).

Examination of drift is a key component of the motor exam. Clinicians should be familiar with the various forms of drift including non-pronator (which would be due to feigned upper extremity weakness or proximal shoulder girdle orthopedic pathology), pronator drift due to upper motor neuron-related weakness, parietal or proprioceptive drift and cerebellar drift. All these drifts have a different clinical presentation but the starting point for the exam is the same: have the patient stand with eyes

open and place their arms in front of them supinated and flexed 90 degrees at the shoulder with elbows in full extension and wrists in neutral followed by requesting that they close their eyes. Downward drift with pronation is what one would expect to see if there was contralateral motor strip or efferent pathway impairment of function in the brain (with the assumption the lesion is above the decussation). Classically, parietal drift presents as the arm rising up and moving laterally contralateral to the side of the lesion, whereas cerebellar drift presents with only lateral movement of the arm ipsilateral to the side of the lesion.

Grip strength testing remains a somewhat debated area relative to the best assessment procedures for determining impairment, including standardization of grip strength testing protocol, as well as methods for assessing grip effort (Roberts et al., 2011). Various dynamometer tests have been utilized and researched relative to assessing for submaximal effort as related to grip strength. The use of such testing has limitations as certain neurological disorders will inherently be at risk for presenting with variability across tests beyond the typical allowable 20% including multiple sclerosis and myasthenia gravis. Historically, grip strength testing via dynamometer using a device such as a Jamar™ device (Taylor & Shechtman, 2000; Westbrook, Tredgett, Davis, & Oni, 2002) has been performed by having the patient do three repetitions at any setting and making sure that the grip strength did not vary more than 20% and/or generated a bell-shaped curve if all five positions were tested and peaks strength testing at grip position three assuming full effort. The rapid exchange grip (REG) method has also been described and found to be more difficult to falsify than grip strength testing via dynamometer. The REG may be a more valid measure of the degree of grip effort; however, one of the limitations is that there is no standardized test protocol for this specific examination technique (Taylor & Shechtman, 2000; Westbrook, Tredgett, Davis, & Oni, 2002). It should also be noted, however, that some studies have shown a negative impact of pain, as well as depression, on the outcomes of REG testing (Phillips, Biland, Costa, & Souverain, 2011). Furthermore, other studies have found that the REG strength test cannot reliably detect voluntary submaximal effort (Westbrook, Tredgett, Davis, & Oni, 2002).

A simple test to assess for effort during grip strength testing in someone without reported or evident weakness has recently been described and referred to as the “Honest palm sign”. The test involves asking the patient to place their fingers in their palms and grip as tightly as possible.

The presence of nail marks in the palm would be considered a “negative” test result, whereas the absence of such marks would be a “positive test” and indicative of incomplete effort and invalid grip strength testing (Black, 2014).

Assessment of functional gait disorders is a topic worth further discussion and there is some published information on same (Lempert, Brant, Dieterich, & Huppert, 1991). Dragging monoplegic gait has been described by some as the most common functional gait disturbance and involves the lower extremity being dragged behind the patient without the typical patterns associated with hemiplegic gait such as hip circumduction, stiff knee, or toe drag. Patients with gait disorders can present with organic impairments that look non-organic, so clinicians should not automatically jump to conclusions about a lack of neurogenic basis for the gait disorder. Features suggestive for functional gait disturbance include uneconomic postures with waste of muscle energy, sudden buckling of knee or hip flexors, pseudoataxia, “walking on ice” gait pattern or excessive slowness. Astasia abasia is a classic functional gait pattern but can be associated with organic disease. Astasia abasia gait is manifested by the inability to stand or walk normally despite possessing good motor strength and conserved voluntary coordination. Symptomatic astasia abasia has been associated with lesions affecting the thalamus, pontomesencephalic region, cingulate cortex or corpus callosum (Lempert, Brant, Dieterich, & Huppert, 1991).

Special examinations may also be used to reinforce clinical impressions regarding the cause of weakness when neurogenic etiologies are in doubt. Drug-assisted interviews with sodium amobarbital or other agents such as short-acting benzodiazepines will generally positively modulate motor impairments if they are due to a FNSD, whereas feigning patients will likely show no difference in findings (Nichols, Zasler, & Martelli, 2012). Other tests for assessing weakness validity on exam include the arm drop test, unexpected painful stimulation to the affected limb, Hoover’s tests, Sonoo abductor test and Babinski thigh-trunk sign (Martelli, Nicholson, & Zasler, 2013; Shaibani, & Sabbagh, 1998; Stone, Zeman, & Sharpe, 2002).

The arm drop test involves holding the affected arm of the patient above his head or face. If, when the arm is released, it falls away from the face, then there is volitional effort underlying the movement, as in true organic hemiplegia the arm would fall onto the person’s face or head. This test can also be used in so-called pseudocomma. Patients with functional or dissimulated weakness

will often demonstrate some initial hesitation with associated transient sustained motor function before letting the arm actually drop, whereas with non-organic weakness there would not be hesitation and the arm would fall from the point of release (Campbell, 2012; Martelli, Nichols, & Zasler, 2013; Zasler & Martelli, 2002).

It is always preferable to explain to a patient being examined what you are going to do to them before you actually do it and to avoid deception. It has been suggested though that providing an unexpected and unannounced nociceptive/painful stimuli to the limb with questionable functional weakness can demonstrate volitional motor control and/or spontaneous withdrawal confirming the diagnosis; however, some would question the ethics of such a practice. Balancing the potential harm that could arise from possibly causing a brief painful sensation to the patient against the potential value of being able to accurately diagnose and treat the patient is the ethical challenge for clinicians.

Hoover's tests takes advantage of the normal crossed extensor response noted at the hip with attempted flexion of the contralateral side (Hoover, 1908). Two separate tests were described by Dr. Hoover to assess for unilateral functional lower extremity weakness. Both tests are important to perform as a functional abnormality on one or both tests is more sensitive than a functional abnormality on either test alone. To perform the test, the clinician holds the heels of both feet while standing at the foot of the bed so that the clinician's hands are between the bed surface and the patient's heel. The patient is then asked to forcefully press each heel into the bed with all their effort. Failure of extension of the purportedly non-affected leg suggests a poor effort/withholding of effort. If a person with leg weakness is then asked to lift the paretic leg from the hip, a simultaneous but contralateral extension will occur at the hip in the unaffected limb which supports organic weakness whereas a weak downward force in the unaffected leg supports functional leg weakness. If a person with leg weakness is asked to lift the unaffected leg and this causes downward pressure in the affected limb, it supports organic weakness, whereas downward pressure in the affected limb greater than the manually tested strength supports functional weakness. One can reinforce these findings by placing one's hand on the shin and providing a downward force to the leg while it is being lifted and keeping the hand cupped under the heel on the other side. Maximal involuntary/voluntary force ratio (IVVR) has been calculated using Hoover's test and found to be markedly increased in the affected limbs in those with FNSD. The investigators concluded

that Hoover's sign in functional paralysis is a preservation or increase of a normal synkinetic phenomenon (Ziv, Djaldetti, Zoldan, Avraham, & Melamed, 1998). It should be noted that Hoover's test does not differentiate functional from feigned weakness. A Hoover's sign in the arms was also described by Dr. Hoover and later more formally studied by Ziv; however, it is not in common use (Ziv, Djaldetti, Zoldan, Avraham, & Melamed, 1998).

The Sonoo hip abductor test is similar to Hoover's tests in that it can be used to differentiate functional lower extremity weakness from organic weakness (Sonoo, 2004). For this test, the clinician holds his/her hands on the lateral aspects of the patient's legs distally. The clinician should then ask the patient to abduct both legs from the hip simultaneously with as much effort as possible while at the same time pushing inwards with an equal opposing force. In both functional and organic weakness, the affected leg remains fixed in an abducted position opposing the examiner's hand while the paretic leg moves in the direction of abduction because of a stronger opposing force being applied by the examiner. The next step is to ask the patient to abduct each leg in turn against the opposing force of the examiner with a non-abducting leg initially placed in an abducted position along the midline. The patient's attention should be directed to the abducting leg but it is the behavior of the non-abducting leg that provides for the diagnostic information in that with organic weakness the paretic leg should be overpowered by the opposing force of the examiner moving the limb into a hyperabducted position; whereas, while abducting the paretic leg, the abducting leg will be overpowered by the examiner, but the sound leg will remain fixed in its original position. In functional or feigned weakness, while abducting the sound leg, both abduction of the sound leg and the opposition of the purportedly paretic leg are strong and the paretic leg should remain fixed, not crossing the midline. When abducting the weak leg, both abduction of the weak leg and the synergistic opposition of the unaffected leg are weak and easily overpowered by the examiner so that the unaffected leg moves to a position of hyperabduction (Sonoo, 2004).

The Babinski thigh trunk test is potentially helpful in evaluating functional or feigned hemiplegia (Babinski, 1897). In this test, the patient lays recumbent and is asked to sit up while keeping the arms crossed in front of their chest. In normal individuals, there is extension at the hip into the bed or table and the legs will remain motionless. In patients with organic hemiparesis, the weak limb involuntarily flexes and elevates off the bed; whereas, in patients with functional leg weakness

neither leg, or only the normal leg, will flex and elevate off the bed.

More sophisticated technical assessment of questionable neurogenic motor weakness may involve such modalities as electromyography (EMG), magnetic brain stimulation over motor cortex, and motor evoked potentials (MEPs) to name a few. What role functional brain imaging may have in this context remains unclear.

5. Discussion

It is critical to realize that one should not jump to conclusions with regard to assuming that a given condition is non-organic or medically unexplainable when one sees the first “functional sign”. Specifically, the task of deciding whether a presentation is purely neurogenic, neurogenic with functional symptom disorder overlay, neurogenic with exaggeration, purely functional, or feigned is a process that includes both appropriate preparation relative to reviewing relevant records, both pre- and post-insult/injury, as well as doing a thorough neurological exam. The identification of cases of functional neurologic symptom disorders, as well as cases involving feigning is clearly challenging and should, ideally, involve a multi- if not inter-disciplinary, biopsychosocial approach to both assessment and treatment.

As clinicians we can never be “certain” that our conclusions are fully accurate, although we may believe that they are. For example, the most convincing evidence of certain or definite malingering is concession or admission by the party in question to “faking bad”, but this seldom occurs and in fact may not even be truthful. Another form of evidence is when a person is detected to have discrepant performance, either through surveillance or corroboratory report although neither is necessarily 100% conclusive (Greer, Chambliss & Mackler, 2005). Given the percentage of previously medically unexplained or functional conditions that are later found to be neurologically based, it is of crucial importance to be careful before labeling a patient with a functional disorder.

Misinterpreting commonly assumed “functional” signs that in fact are not necessarily non-organic is a common error made by clinicians in this context (Gould, Miller, Goldberg, & Benson, 1988). It is therefore critical to familiarize oneself with the nuances of the neurological exam and the spectrum of plausible impairment associated with any given frequently encountered neurological condition whether congenital or acquired, before one “steps out into the arena” of

diagnosing medically unexplained and non-organic presentations.

6. Conclusions

Neurorehabilitation assessment without awareness of the need to consider symptom and sign validity can produce both false positive and false negative diagnoses. It is paramount that neurorehabilitation training, regardless of the specific specialty, emphasizes the need for comprehensive, multidisciplinary assessment including inclusion of training to teach methodologies for effort, response bias and overall validity assessment (Smith et al., 2007). It is also paramount not to “jump the gun” and label observed behavior or findings as “non-organic” before doing an adequate and thorough assessment due to the potentially dire consequences of such a diagnosis in the presence of organic disease. The aim of this review article is to increase reader awareness to these important issues and provide readers with a good starting foundation for understanding the many nuances and caveats associated with validity assessment in the context of the neurological physical exam. Certainly, further research is required to refine our knowledge in this area.

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