Reflexes in Psychiatry

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In this series, Drs. Sanders and Gillig explain how aspects of the neurological examination can aid in differential diagnosis of some common (and some uncommon) disorders seen in psychiatric practice.

ABSTRACT

Psychiatric patients often do not cooperate fully with the neurologic examination. Reflexes virtually bypass patient effort and are difficult to consciously determine. This article reviews muscle stretch (deep tendon) reflexes, and pathological reflexes including the extensor plantar (Babinski) and primitive release reflexes. Topics include findings in common psychiatric and neurologic conditions and methods for eliciting these signs.

INTRODUCTION

One immediately senses that the neurologic examination is an important clinical bridge between mind and brain. It does not take much experience with real patients, though, to find that the neurologic examination can be difficult to conduct and/or to interpret. Some patients are overtly uncooperative, some seem distracted, and some seem to be applying little effort. Examining reflexes holds appeal because little or no active participation is required.

A great number of reflexes have at some time found favor for revealing lesions in association cortex, the corticospinal motor system, various peripheral sites, or for purposes other than localization (e.g., diagnosis and prognosis). This article will focus on the psychiatric applications of common reflexes and on reflexes useful in psychiatry.

There are two basic types of reflexes: those that we expect to find in a healthy adult and those that we do not. The group of normally encountered reflexes includes muscle stretch (i.e., deep tendon or myotatic) and cutaneous (i.e., superficial). The clinical relevance of these is in the vigor of response, particularly when contrasted with that of the contralateral reflexes. The group of reflexes that we do not expect to encounter in health is referred to as pathological reflexes. The pathological reflexes include tests of corticospinal tract integrity e.g., pyramidal tract) (the Babinski or plantar extensor reflex is the best known of these), and the primitive reflexes (developmental, cortical, frontal, release). We will review these categories.

We cover three muscle stretch reflexes (i.e., biceps brachii, patellar, ankle jerk), two corticospinal pathological reflexes

(i.e., the Babinski and Chaddock plantar extensor methods), and two primitive reflexes (i.e., grasp and palmomental). Some reflexes involving cranial nerves, including jaw-jerk, corneal, glabellar, snout,[1] and gag,[2] were discussed in previous articles of this series. Cutaneous reflexes, such as the cremasteric and abdominal reflexes, have no specific relevance to psychiatry or behavioral neurology, and are therefore outside the scope of this article.

ANATOMY AND PHYSIOLOGY

Muscle stretch reflexes. Most readers will recall from med school classes the monosynaptic (2 neuron) circuit underlying the muscle stretch reflex. Tapping a tendon activates the muscle spindle and the fast-conducting, myelinated 1a afferent nerve fibers. The one synapse is in the ventral horn of the spinal cord, at the level of the innervated muscle, but spinal interneurons and afferents are also stimulated. Removing any element of the two-neuron circuit, including the spinal cord at that level, the afferent or efferent neuron, or the muscle, will make the reflex impossible. Thus, the basic teaching is that areflexia (i.e., absent reflexes) indicates a lower motor neuron lesion. In most psychiatric patients, though, the basic lower circuit is intact. Muscle stretch reflexes are attenuated through influences of the brain (via descending reticulospinal, vestibulospinal, and corticospinal pathways). These attenuating influences are affected by upper motor neuron lesions and by distraction (reinforcement) techniques. Thus, the basic teaching is that hyper-reflexia indicates an upper motor neuron lesion. Diffuse and bilateral hyper-reflexia can have many causes. Unilateral hyper-reflexia has more localizing significance.

Pathological reflexes. All pathological reflexes seem to represent a lack of inhibitory brain influence, but they vary in their underlying anatomy.

The localizing (corticospinal tract) reflexes are present in early childhood, but almost always disappear during normal development. Plantar responses are extensor until about 18 months of age. Unlike the primitive reflexes, corticospinal reflexes rarely reappear in healthy aging, so when they are present in an adult, this indicates the presence of a pathological lesion.

The primitive reflexes (i.e., grasp, palmomental, and snout) are much more commonly visualized clinically in normal early childhood than normal later childhood and adulthood. The primitive reflexes reappear in association with a wide variety of neuropsychiatric diseases, and also reappear in healthy elderly persons. The traditional notion that primitive (i.e., frontal release) reflexes reflect "frontal lobe pathology" has little empirical support.[1,2] The only exception would be the grasp reflex, which has been consistently related to lesions anterior to the central sulcus (frontal lobe, supplementary motor area, and/or anterior cingulate gyrus).[3,4]

The palmomental reflex is often categorized as nociceptive, sometimes called the "wince" reflex. Brisk stimulation (to cause discomfort, not actual pain) of the thenar eminence of the palm elicits a visible twitching in the ipsilateral (sometimes bilateral or contralateral) chin. Although a minority of normal people have a visible response, with sufficient stimulus and sensitive enough electromyographic measure of the response, every healthy person has a palmomental reflex.[5] (This is also true of the snout reflex[6] and may also be true of other primitive reflexes not yet tested neurophysiologically.) Tactile stimuli are conveyed along the median nerve to the facial motor nuclei. There are two circuits: a short loop that reaches the facial nuclei and a long loop that includes thalamus and cortex.[7] The effector is the mentalis muscle of the chin. Healthy newborns virtually all have a visible palmomental reflex. By roughly eight years of age, the percentage drops to adult levels of roughly 10 percent (much variation in estimates), then rises in middle and advanced age to roughly 60 percent.[5–7]

CLINICAL APPLICATIONS

Muscle stretch reflexes. Because muscle stretch reflexes are not highly reproducible between examiners or over time,[8–10] it is better not to over-interpret isolated findings unless other findings are consistent with the pathology that the reflex changes imply. Even brief clonus and overflow can be seen in anxiety and hyperthyroidism, as well as more ominous conditions, such as tetany. Clearly and persistently asymmetric reflexes evidence one or more focal lesions. Hyporeflexia, specifically delayed return to the resting position, raises the question of hypothyroidism. Distal hypo-reflexia may represent peripheral neuropathy.

Early descriptions and studies of "dementia praecox"/schizophrenia included frequently hyperactive muscle stretch reflexes.[11–15] Asymmetric reflexes were also fairly common in some large series.[14,15] Diffusely brisk (but not frankly abnormal) reflexes continue to be observed frequently in some studies of schizophrenia,[16,17] but do not discriminate schizophrenia from mood disorders.[17] Recent studies comparing schizophrenia to healthy controls find no significant differences in rates of hyper- or hyporeflexia.[18,19] Asymmetry is the most diagnostically significant of these findings and has been found at a consistent and fairly low frequency in idiopathic schizophrenia over a

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century of studies. Depression was described in at least one older text as accompanied by hyper-reflexia,[20] but we are aware of no data. Not surprisingly, hyper-reflexia is described in chronic alcoholism.[21] We often assume that hypoactive reflexes, including absent ankle jerk reflexes, are to be expected in healthy aging, but this is not supported by data.[22,23] An epidemiological study of rural India[24] found that hypoactive muscle stretch reflexes were associated with cognitive impairment after adjusting for age, gender, and literacy.

Pathological corticospinal reflexes. The extensor plantar (i.e., Babinski and Chaddock) reflex is clearly the most significant of the pathological reflexes and has been known for over a century to represent corticospinal (i.e., pyramidal) tract pathology. Multiple eponymous variations reflect the several different ways of eliciting the extensor response.[25] The extensor response is a sign of upper motor neuron corticospinal tract lesion.

In studies using the Babinski spanning nearly a century,[26] an extensor response was elicited in 0.5 to 8 percent of patients with schizophrenia and 0 to 5 percent of healthy comparison subjects. Unequivocal Babinski and related reflexes are uncommon in schizophrenia, and probably not much more common than in healthy people. In Alzheimer-type dementia, the corticospinal pathological reflexes are uncommon until terminal stages,[27] but are not seen in normal aging either.[28] Localizing signs are expected in vascular dementia, so we might expect to find studies demonstrating more corticospinal pathological reflexes in vascular than Alzheimer-type dementia. One paper describing Japanese patients with multi-infarct dementia is all we could find.[29] The extensor plantar reflex was one of the most common exam findings. The Chaddock reflex was frequently positive despite negative or equivocal Babinski reflex.

Primitive reflexes. Developmental or primitive reflexes (again, pathological reflexes not localizable to corticospinal tract) are typically found to be fairly common in normal populations[30] and increasingly common with normal aging,[31] especially with cerebrovascular lesions.[32] Most studies find them to be more common in age-matched neuropsychiatric samples, such as schizophrenia and dementia, but fairly extensive research has found little value in clarifying etiology or diagnosis.[33–35] It is clear that a single primitive reflex means nothing in terms of diagnosis, but multiple primitive reflexes imply brain disease at any age.[28,32]

The grasp reflex may be an exception among the primitive reflexes. It is found infrequently in Alzheimer-type dementia, but like corticospinal pathological reflexes, it is virtually unheard of in normal aging.[28] The grasp reflex has more pathological significance than the other primitive reflexes.

In distinguishing vascular from idiopathic Parkinsonism, one large but unreplicated study[36] found that all pathological reflexes were more prevalent in vascular Parkinsonism. Surprisingly, primitive reflexes were more helpful than corticospinal reflexes in distinguishing the two groups; when either the suck reflex or the palmomental reflex is present, the sensitivity for the diagnosis of vascular Parkinsonism was 84 percent and the specificity was 82 percent.[36] When a person with dementia develops incontinence, pathological reflexes (corticospinal and primitive) may help answer the important question of whether the incontinence is reversible or represents a new phase of the dementia.[37]

Academic interest in the primitive reflexes seems to have out-stripped any reasonable expectation of clinical value. Although a few studies have had interesting results, there are serious problems that are likely to prevent any widespread translation of these results to clinics. Different groups find vast differences in percentages of positive reflexes, which would be expected with minor variations in technique.[38,39] Further, it is fairly clear that these signs are not always stable over time.[40–43] Still, there are some scenarios in which they can be of value.[4] For instance, several primitive reflexes in an apathetic patient with noncontributory brain imaging strengthen the case for a neurodegenerative rather than primary psychiatric disorder, and new or strikingly more severe primitive reflexes in a patient with a known frontal lobe lesion could signal an extension of that lesion.

EXAMINATION METHODS

Muscle stretch reflexes. There are no demonstrated differences among reflex hammers, nor have hammers been shown superior to fingers or knuckles. Popular among neurologists (in the United States at least) is the disc-shaped "Queen Square," the shape of which holds some advantages. Most popular in the United States is the tomahawk-shaped Taylor hammer, the size of which is more convenient.

The deep tendon reflexes are elicited by a short, sharp blow to the tendon of a slightly extended muscle. They can be difficult to elicit if the muscle and/or opposing muscles have elevated baseline tension or if the muscle is flaccid. Depending on the situation, a patient might need to be calmed or distracted, or (more often) reinforcement measures may be needed. If no response is detected, it is important to try again with reinforcement using the Jendrassik maneuver. In the Jendrassik maneuver, the physician asks the patient to hook together the fingers of his or her hands, and to pull apart as hard as possible when told "now." The signal is given immediately before tapping the tendon. In most cases when reflexes are not appreciated without reinforcement, they are

detectable after using this maneuver.[44]

Tables of reflexes and corresponding spinal levels are critical when diagnosing and treating certain conditions, but in screening the psychiatric patient, it is usually adequate to check the biceps brachii (C5-6), patellar (knee jerk, L2-4), and ankle jerk (Achilles, S1) reflexes.

To elicit the biceps reflex, the arm is generally rested on the lap with the elbow bent at about 120 to 160 degrees. Stand to the patient's side with one finger over the biceps tendon and another over the distal biceps muscle. Strike the finger overlying the tendon, feeling the response of the muscle.

To elicit the patellar reflex, the leg is generally dangling with the shin at a roughly 90-degree angle to the thigh. Stand slightly to the side, resting one hand slightly on the distal thigh. Strike the patellar tendon directly, just above its insertion in the tibia.

Traditionally, the ankle jerk is elicited by striking the achilles tendon directly. Tell the patient not to help, then push upward on the plantar surface of the foot until it is everted and dorsiflexed. Strike the tendon.

The ankle jerk may be more reliably elicited by striking the plantar surface of the foot, rather than the Achilles tendon.[45] Without manipulating the patient's position, rest the dorsal surface of the fingers against the sole of the patient's foot and strike the palmar surface of your finger over the mid-arch region.

The American Academy of Neurology has a standard grading system for reflex intensity[46] where 0=absent, 1=present but small or only with reinforcement, 2=present but in the lower half of the normal range, 3=present and in the upper half of the normal range, and 4=reflex increased, with or without clonus. It is worthwhile to record exam results using this or another standard scale.

Pathological corticospinal reflexes. The Babinski reflex is elicited by drawing a fairly dull object (e.g., a tongue blade) down the lateral sole of the foot, starting just anterior to the heel and proceeding to the lateral ball of the foot, then continuing medially to finish just posterior to the medial ball of the foot. The entire L-shaped motion takes 1 to 2 seconds. An extensor response is noted at the base of the first toe. Some patients are impervious to the stimulus, requiring a firmer repetition. More often, patients have difficulty tolerating the stimulus, withdrawing the foot and obscuring any response of the great toe. Often, one can elicit the response with a minimal fragment of the stimulus, or stimulating part of the less sensitive lateral sole. If this fails, other variants can be used. The Chaddock reflex requires applying a similar stimulus, also moving from posterior to anterior, beneath the lateral malleolus.[47] The Babinski and Chaddock methods are comparably sensitive to corticospinal lesions, and both have fair reliability.[48]

Primitive reflexes. To elicit the palmomental reflex, the physician should ask the patient to look to the side and tell the him or her to expect scratching on the palm. Draw the tip of a key or similar semi-sharp object firmly across the thenar eminence, moving distally from near the wrist. The stimulus should be mildly noxious. Repeat about six times, observing the ipsilateral mentalis closely. Repeat the same sequence with the other hand.

Persistence of the mentalis (chin) twitch is considered more important than the initial response.[5]

To elicit the grasp reflex, the physician applies firm pressure to the patient's central palm, moving toward the fingers. This elicits a brief flexion response. By now, the physician's pressure has progressed to the base of the patient's fingers and is still proceeding distally, meanwhile abducting the hand from the body. This elicits a flexion response in the fingers, which holds the physician's hand firmly and persistently. The reflex is much more significant if the patient is asked not to hold on; otherwise, some patients assume that they are expected to do so. The physician should avoid contact with the back of the hand, as this may counteract the reflex. More extreme variants of this include tenacious clinging to nearby objects, such as bedrails, and exploratory movements of the hand after the object is removed.[4]

The muscle stretch and extensor plantar reflexes, and even the primitive reflexes, when properly done and integrated with other findings, can give information about patients' brain functioning that may not be revealed by interviews, imaging, or through performance of deliberate tasks.

CONCLUSION

Reflexes are a valuable part of the neurological exam, particularly in patients with limited capacities to cooperate with the assessment. The muscle stretch and extensor plantar reflexes, and even the primitive reflexes, when properly done and integrated with other findings, can give information about patients' brain functioning that may not be revealed by interviews, imaging, or through performance of deliberate tasks.

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