Adopted: 09/05

Deep Tendon Reflexes (DTR)

Deep tendon reflexes, more properly referred to as muscle stretch reflexes, are an integral part of the neurological examination. A stretch reflex is an involuntary reaction of a muscle to being passively stretched by percussion of the tendon.

Although the reflex has a sensory and motor component, deep tendon reflex testing primarily assesses the integrity of the motor system. This reflex provides information on upper and lower motor neurons. In its simplest form, the quick stretch of the muscle-tendon unit stimulates the muscle spindle receptors which, in turn increases the firing of Ia afferents from the spindles. These Ia afferents enter the spinal cord segmentally by the dorsal roots and monosynaptically facilitate alpha motor neurons that supply the homonymous muscle causing the muscle to contract.

The table below demonstrates that DTR testing generally has a high specificity and moderate to reasonably good positive likelihood ratios, making them useful when abnormally depressed. On the other hand, they tend to have poor sensitivity, making them a poor at ruling out neurological conditions.

Stretch Reflex (DTR)	Nerve root	Test Validity for Nerve Root	Peripheral nerve
Biceps	**C5-**C6	For a radicular syndrome: Specificity 95%, LR+ 4.9 For C6 radiculopathy:* Sensitivity 53%, -LR 0.5 Specificity 96%, +LR 14.2	Musculocutaneous nerve
Brachioradialis	C5-**C6**	For C6 radiculopathy:* Sensitivity 53%, -LR 0.5 Specificity 96%, +LR 14.2	Radial nerve
Triceps	C6-T1, **C7**	+LR 28.3 (C7 or C8)	Radial nerve
Patellar	L2-**4**	For L3 or L4 radiculopathy: +LR 6.9	Femoral nerve
Achilles	L5-S2, **S1**	For S1 radiculopathy: Sensitivity 85% For L5 radiculopathy: Poor For lumbar disc herniation: Sensitivity 45%,-LR 0.6 Specificity 89%,+LR 4.3	Tibial nerve
Medial hamstring	**L5-S1**	Not reported	Sciatic nerve

* Although C5 may be the dominant root, clinical studies have found it more strongly linked with the more common **C6** root lesion (McGee 2001).

RELIABILITY

A small number of studies (Litvan 1996, Manschot 1998, Stam 1990) have evaluated the reliability of deep tendon reflex testing. Due to the disparate methodologies used in these studies, the pooled results are equivocal or contradictory. The following observations about reliability in DTR testing can be made:

- Intraobserver reliability (kappa values of .77 to .91, near perfect to perfect agreement) is higher than interobserver reliability (Kappa values of .50 to .64, moderate to substantial agreement).
- Reliability of assessment of lower extremity DTRs is higher than that of upper extremity DTRs.
- Clinical observation of asymmetrical reflexes has a significant agreement with EMG findings of reflex asymmetry (Stam 1990).

TECHNIQUE

The extremity being tested should be in a *relaxed* position. A quick, precise tap on the tendon with a reflex hammer should elicit the response. It is important to use the lightest force possible to bring out the reflex and to be consistent from side to side. There are a number of things that can be done if the practitioner is having trouble eliciting a reflex.

1. Alter the technique.

- Use the whole length of the reflex hammer; let the hammer fully swing. (Fuller 2004) Don't choke down on it; don't make small, stabbing thrusts at the tendon.
- Have the patient contract the muscle first.
- The tendon should be neither too slack nor under too much tension. Some pre-tension is helpful, for example dorsiflexing the ankle will enhance an Achilles reflex. On the other hand, if the tendon is pressed too hard or if the contact is too uncomfortable (as can occur when holding the biceps tendon), the reflex may not be obtained, even from a normal person. (Orient 2005)
- **Reflex hammer size**. Elicitation of hyporeflexic DTRs requires greater force than do normoreflexic or hyperreflexic DTRs (Marshall 2002). The lightweight, triangular-tipped, Taylor reflex hammer has a ceiling effect in the hyporeflexic range. Therefore, the heavier, long-handled Babinski reflex hammer is a better choice.

2. Ensure the <u>patient</u> is relaxed.

- Simply having the patient change position may help to bring out a reluctant reflex. (Orient 2005)

3. Try different patient positions.

- For example, the Achilles reflex is sometimes easier to get with the patient kneeling on the adjusting table or a chair rather than sitting.

4. Use a reinforcement maneuver (Jendrassik maneuver).

- *See next page.*

Reinforcement Maneuvers

When reflexes are difficult to elicit, a reinforcement technique, such as a Jendrassik maneuver, may be used to augment the response. The patient is asked to contract a "remote" muscle at the other end of the body. For example, in the case of a lower extremity reflex, the patient interlocks fingers together and pulls when cued by the practitioner. A reinforcement technique for an upper extremity reflex is to have the patient contract a lower extremity muscle (Delwaide 1981) or clench the jaw (Tarkka 1983), while the practitioner taps the tendon.

The facilitation only lasts from 1 to 6 seconds after initiation of the voluntary contraction (Hayes 1972). However, it is at *maximum* for only 300 milliseconds (Kawamura 1975). Neurophysiologic studies of facilitation (Kawamura 1975) reveal that the facilitation begins after the instruction to contract the remote muscle, but 100 milliseconds *before* muscle contraction begins. (Orient 2005)

NOTE: If a reinforcement technique is used, it must be documented in the grading of the reflex. A (J) can be written next to the reflex grade.

Tips to enhance the reflex

- Ask patients to close their eyes as this may help them relax.
- Distracting patients by asking casual questions (i.e., When is your birthday? Do you have any pets?) may help them relax.
- A slight preload of the tendon (ask the patient to "barely push" – very slight contraction of the muscle involved in the test) especially in those that do high resisted activities using the muscle that is being tested (chart as reinforced, like the Jendrassik).
- The amplitude of the reflex may also be increased by having the patient turn his/her head toward the side being tested; this maneuver will also shorten the latency time. Turning the head away from the side being examined has the opposite effect (Tarkka 1983).
- The louder the command (e.g., "pull"), the greater the facilitation (Scheirs 1982).
- Patients may respond to the instruction "pull" by tensing all the voluntary muscles, not just the ones remote from the area. This effectively suppresses the deep tendon reflexes. (Orient 2005) Ask patients to focus just on the remote muscle that they are being asked to contract.
- The effectiveness of the Jendrassik maneuver is proportional to the degree of the patient's effort. If at first unsuccessful, be sure that the patient is exerting maximal effort on the voluntary contraction of the remote muscles (Hayes 1972).
- The practitioner should strike at the same moment that s/he is giving the command because the peak facilitation occurs for only 300 milliseconds and begins *before* the actual contraction is initiated.
- *Equivocal reflexes may need to be checked over several visits to determine if they represent a true and clinically significant neurological abnormality.*

GRADING SCALE

There are a number of accepted scales for grading deep tendon reflexes. The scale used at WSCC clinics is the National Institute of Neurological Disorders and Stroke (NINDS 1991) muscle stretch reflex scale. The reflex is graded based on the amplitude of the response. *The only reflex that is always abnormal is clonus*. The "+" after the number is to distinguish from muscle testing, it is not a meant as a "plus" or "minus" in the traditional sense.

Grades 1, 2, 3 are not by definition normal or pathological—they are depressed, average or elevated (Ferezy 1992). Most neurologists, as well as WSCC clinics, equate "4+" with the presence of clonus, although some use "5+" to designate clonus.

In general, the grades are as follows:

1+ = present but depressed
2+ = normal / average
3+ = increased
4+ = clonus

ASSESSMENT STRATEGY

1. **Decide if it is necessary to elicit DTR reflexes.** Whenever a neurological condition is suspected, DTRs are performed along with muscle strength and sensory testing. Potential candidates for testing include those with spinal pain and any arm or leg symptoms, headache, cranial nerve symptoms, head trauma, or suspected metabolic diseases, such as diabetes or thyroid disease.
2. **If a reflex appears to be absent or diminished,**
   a) repeat (several times, altering technique, patient position, etc.)
   b) use reinforcement.
3. **If a reflex appears increased,** repeat using an increasingly *lighter* force, while comparing to other reflexes.
4. **If a reflex is abnormal in any way,**
   a) compare bilaterally and with other DTRs in general.
   b) correlate with other sensory and motor exam findings.
   c) repeat on subsequent visits to confirm findings and to monitor the trend over time (this can be one of the most important ways to determine the clinical significance of an altered reflex).

INTERPRETATION

The deep tendon reflex depends on the integrity of both the upper motor neuron and the lower motor neuron. As a general rule, disease/injury of the lower motor neuron (e.g., nerve roots or peripheral nerves) will cause a reduction or loss of a reflex. Disease/injury of the upper motor neuron (e.g., spinal cord, brainstem or brain) will cause an exaggeration of a reflex with possible clonus.

Absent or exaggerated reflexes, by themselves, do not verify neurological disease. Instead, they are significant only when it is associated with one of the following clinical settings:

1. The reflex amplitude is asymmetric. This may indicate an upper motor neuron disease associated with hyperreflexia on one side or a lower motor neuron disease associated with hyporeflexia on the other side. Occasionally, asymmetrical reflexes are seen in normal patients.

2. The reflex is unusually brisk compared with reflexes from a higher spinal level, which raises the possibility of spinal cord disease at some level of the spinal cord between the segments with normal/diminished reflexes and those with exaggerated ones.

3. Other neurological signs are present indicating either an upper motor or lower motor neuron disease. These signs will help to localize the pathology (see chart below).

UPPER MOTOR NEURON LESION (UMNL)	**LOWER MOTOR NEURON LESION (LMNL)**
Hyperreflexia	Hyporeflexia
Pathological reflexes	Fasciculations
Pathological reflex (Babinski sign, clonus)	Atrophy
Spastic paralysis/paresis	Flaccid paralysis/paresis
Hypertonia (clasp-knife)	Hypotonia

Sensory deficits may be present along with either upper or lower motor neuron signs. In the case of peripheral nerve disease, sensory deficits should fall within a peripheral nerve pattern. With nerve root lesions, deficits should follow dermatomes. In spinal cord and brain lesions, deficits will be more generalized.

TESTING RESULTS

The practitioner must differentiate normal from abnormal reflexes, then must assess the cause or diagnosis.

Differentiating normal from abnormal

1. Exaggerated reflex or clonus: This suggests an upper motor neuron lesion above the root at that level. Normally, in children, upper extremity reflexes are stronger than lower extremity reflexes. (Orient 2005) *Increased reflexes are often normal, especially when symmetrical; clonus should never be interpreted as normal.*

2. Depressed reflexes: Reflexes are diminished or lost in nerve root lesions, peripheral nerve lesions, metabolic diseases such as diabetes or hypothyroidism, and muscle disease. Diminished reflexes may be an early finding of radiculopathy. In one study, deep tendon reflex abnormalities in the upper extremity increased the likelihood 2.5 times that there would be a positive electrodiagnostic study and increased the likelihood of radiculopathy fourfold (Lauder 2000). They may also be depressed in the acute phase of a severe UMN due to spinal shock (but more often hyperreflexia is seen with cord damage). Hyporeflexia may sometimes also be seen with cerebellar disease. *Decreased reflexes are often normal, especially when symmetrical.*

3. Absent reflexes: The absent reflex may be associated with other findings of lower motor neuron disease (weakness, atrophy, fasciculations):

- *generalized:* indicates peripheral neuropathy
- *isolated:* indicates either a peripheral nerve entrapment or mononeuropathy, or more commonly a nerve root lesion
- *bilateral absent ankle reflexes:* most commonly indicates a peripheral neuropathy; also occurs with bilateral SI nerve root lesions or very rarely bilateral sciatic nerve lesions.

4. Reflex spread or inverted reflex: The reflex tested is present but this response goes beyond the muscle (reflex spread), or is absent in the reflex tested but creates a response in other muscles (inverted). For example, the fingers are seen to flex when the supinator reflex is tested or the hip adductors are seen to contract when testing the knee reflex. Reflex spread or an inverted reflex indicates an upper motor neuron lesion occurring above the level of innervation of the muscle to which the reflex spread.

5. Slow relaxing reflex: This is especially seen with the Achilles reflex and may be difficult to note. It is associated with hypothyroidism.

Scale	**NIND scale** (0-4) (Used at WSCC)	**British scale** (0-5)	Description	Interpretation
0	**Reflex absent**	Reflex absent	No perceptible response. No reflexes should ever be described as "absent" unless Jendrassik maneuver has been done. (Orient 2000)	LMNL, metabolic disease (e.g., thyroid, diabetes), shock phase of an UMNL, may also be normal
1+	**Reflex small, less than normal; includes a trace response or a response brought out only with reinforcement**	Reflex small, less than normal; includes a trace response or a response brought out only with reinforcement	Contraction barely but definitely perceptible	May be normal for the individual, LMNL, metabolic disease, or the shock phase of an UMNL
2+	**Reflex normal or in lower half of normal range**	Brisk, within the median range of normal	Contraction obviously perceptible	Reflex is within normal limits
3+	**Reflex in upper half of normal range**	Reflex enhanced, high normal or hyperreflexia	Vigorous contraction apparent from across the room	May be normal for the individual, or UMNL, or patient in an anxious state
4+	**Reflex enhanced, more than normal; includes clonus if present, which optionally can be noted in an added verbal description of the reflexes**	Reflex enhanced, more than normal; include intermittent clonus	Reflex "beats" multiple times at end of arc	UMNL
5+*	N/A	Sustained clonus		UMNL

* This grade is only in the British system that distinguishes intermittent clonus (4+) from sustained (5+).

UPPER EXTREMITY REFLEXES & Localization (1, 2, 3, 4):

Biceps Reflex. The elbow jerks toward flexion. This reflex is innervated by C5 & C6 nerve roots conveyed along the musculocutaneous nerve. In one study of neck and arm pain patients, subjects with an abnormal biceps reflex were 10 times more likely to have nerve root involvement than those with a normal reflex (Lauder 2000). In a prospective study, a decreased biceps stretch reflex had a 95% specificity and an LR+ 4.9 for a radicular syndrome (Wainner 2003). The primary input is mediated through the **C5** nerve root, but clinical studies have found it more strongly linked with the more common **C6** root lesion (McGee 2001).

Brachioradialis Reflex. (sometimes confusingly referred to as the *supinator* reflex) The elbow jerks toward flexion and sometimes supination; a normal reflex may also involve only an observable biceps/brachioradialis twitch. This reflex is innervated by C5-C6 nerve roots conveyed along the radial nerve. The primary input is mediated through the **C6** nerve root. Finger flexion as a response, especially if the finger flexion is spastic, suggests a transverse cord lesion at the level of the missing motor response. (Orient 2005)

Triceps Reflex. The elbow jerks toward extension. This reflex is innervated by C6-T1 nerve roots conveyed along the radial nerve. The primary input is mediated through the **C7** nerve root, but can be associated with C8 lesions. Teeth clenching (Tarkka 1983) or contraction of a lower extremity muscle (Delwaide 1981) is evidence suggesting that these specific maneuvers can enhance the triceps reflex.

LOWER EXTREMITY REFLEXES & Localization (1, 2, 3, 4):

Patellar Reflex. (quadriceps, knee jerk) The knee jerks toward extension. This reflex is innervated by L2-4 nerve roots conveyed along the femoral nerve. The primary input is mediated through the **L4** nerve root. In 1889, Jendrassik reported that the patellar reflex was absent in 1.6% of 1,000 controls of various ages. (Orient 2005)

Medial Hamstring Reflex. A palpable or observable hamstring muscle twitch and occasionally a slight jerk toward knee flexion is produced. This reflex is innervated by the sciatic nerve and the L5-S1 nerve roots with the primary input mediated through the **L5** nerve root, however, an S1 lesion is also possible.

Achilles Reflex. The ankle jerks toward plantar flexion. This reflex is innervated by L5-S2 nerve roots conveyed along the tibial nerve. The primary input is mediated through the **S1** nerve root. This reflex is not very accurate in differentiating S1 from L5 nerve root lesions (Kerr 1988, Kortelainen 1985). Sensitivity is thought to be poor for L5 nerve root lesions, but has been reported to be as high as 85% for S1 nerve roots. (Rico 1982)

Bowditch (1996) assessed the prevalence of absent ankle reflexes in 1074 adult patients. Those with probable pathologic reflex loss were excluded. The absence of one or both ankle reflexes was strongly related to increasing age. However, this finding was rare enough that it should still be considered a definite clinical sign. In an earlier prospective study (Impallomeni 1984), reflexes on hospital patients were controlled for anxiety (reflexes were examined the day after admission when patients were presumably less anxious) and Jendrassik maneuvers were used. Using this method, only 6% of all elderly patients (mean age 82 years) had missing ankle jerks, and most of these had a known explanation. For example, diabetes is a common cause of absent ankle jerk, although only 32% of all diabetics lose this reflex (Abraham 1966). Orient (2005) argues that if underlying diseases are carefully ruled out, *only 1.5% of elderly patients have loss of Achilles reflex due to age and* only 0.2% of patients under the age of 60 will have absent ankle jerks as a normal variant. (Orient 2005)

Other Causes of Altered Reflexes

Thyroid disease

The relaxation phase of the ankle jerk has been well studied. It is discernibly briefer than normal in 14% to 93% of patients with hyperthyroidism (Abraham 1966, Rives 1965) and immediately following exercise (Martin 1970). It is prolonged in 62% to 100% of patients with hypothyroidism (Abraham 1966, Reinfrank 1967, Rives 1965) as well as in some other disorders. (Orient 2005)

Diabetes

The prolonged ankle jerk of diabetic neuropathy can sometimes be elicited after repetitive tapping of the ankle jerk, every 1 or 2 seconds, for at least six taps (Roberts 1982). These phenomena are not seen in myasthenia gravis or thyroid disease (Orient 2005). Sometimes the reflex could even be extinguished. One particular presentation of diabetes creates a sensorimotor neuropathy that includes significant pain, distal sensory loss, and generalized areflexia (Patton 2001).

Some medications and illegal drugs may produce altered reflexes.

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