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Understanding degenerative cervical myelopathy in musculoskeletal practice

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ABSTRACT

Background: Degenerative cervical myelopathy (DCM) is a clinical syndrome characterized by a progressive compression of the spinal cord. DCM often looks like common symptoms of aging or bilateral carpal tunnel syndrome in its early stages, requiring careful differential diagnosis. Identifying DCM is a real challenge as no validated screening tools are available for making the DCM diagnosis. Potentially, individuals with DCM may experience misdiagnosis or substantial diagnostic delays, with an enhanced risk of irreversible neurological consequences if not promptly addressed. Despite the increasing prevalence, there is a lack of awareness about DCM among both the public and healthcare professionals. However, patients may seek physiotherapy to obtain a diagnosis or access treatment.

Methods: A comprehensive (non-systematic) review of the literature about DCM epidemiology, pathophysiology, clinical presentation, diagnostic methods, and management was conducted.

Results: A guide and essential knowledge to facilitate clinicians to understand DCM and to enhance clinical reasoning skills, performance and interpretation of the examination are provided. Interdisciplinary collaboration and optimal referral methods are also handled.

Conclusion: The aim of this article is to summarize and enhance physiotherapists' essential knowledge of the differential diagnosis and management of patients with DCM.

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Spinal cord diseases; spinal cord compression; Physiotherapy; intervertebral disc degeneration; guideline adherence; neck pain

Background

Symptoms associated with nonspecific neck pain have been observed in early manifestations of select, serious cervical spine pathologies [1]. Subsequently, triage for the potential of serious cervical spine pathologies is a priority [2–6]. Degenerative cervical myelopathy (DCM) is a clinical syndrome characterized by progressive compression, torsion and/or friction of the cervical spinal cord (Figure 1) [8–11]. Aetiology includes ligamentous ossification, and/or vertebral and disk degenerative changes that progressively compromise the spinal cord [12,13]. Epidemiological data are still unclear and considerable variability has been observed between countries; however, the incidence is reported between 4–8 cases per 100.000 people annually [11,12]. Global prevalence is estimated to be 2.3% [10]; two-thirds of cases occur in females, with a mean age of 40 years [14,15]. Individuals aged ≥65 years exhibit pathological or radiological evidence of cervical degenerative disease in over 70% of cases and symptoms of spinal cord compression occur in one-quarter of these people [12]. With an increase in global population of people aged ≥65 years, DCM is expected to increase in prevalence [12].

The natural history of DCM is poorly understood, and the progression of symptoms is highly variable and challenging to predict [16]. It has been estimated that 75% of patients with DCM commonly report non-consistent episodic changes in symptoms, with asymptomatic periods lasting months to years [17–22]. Diagnosis of DCM in early phases of the disease

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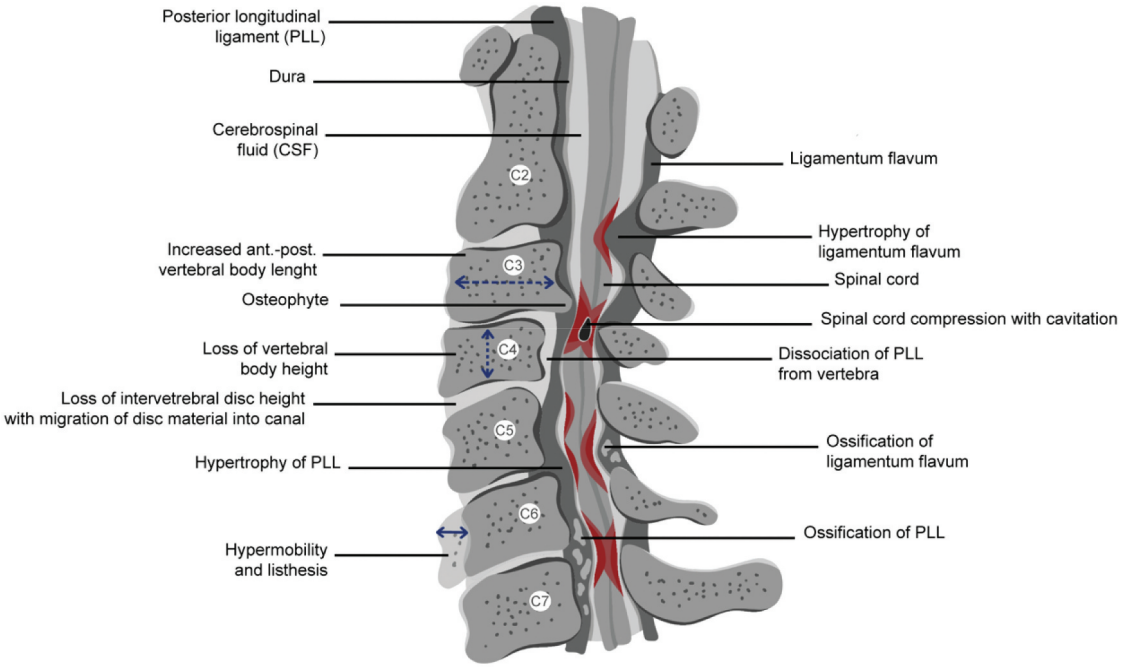

Figure 1. Sagittal section of the spinal cord indicating the presence of cervical stenosis *(adapted from Davies et al*. [7]).

spectrum is challenging as there are no validated screening tools for establishing its diagnosis [11]. Further, individuals with DCM frequently experience misdiagnosis or substantial diagnostic delays, averaging 2.2 ± 2.3 years [18]. This delay typically involves an average of 5.2 ± 3.6 consultations with primary care physicians before an accurate diagnosis is reached [18]. Missed and delayed diagnoses in DCM may stem from subtle and highly nonspecific presentations, incomplete neurological assessments by clinicians, and a lack of awareness among both the public and healthcare professionals [23,24]. Delayed diagnosis can have devastating consequences including paralysis, incontinence, and reduced life expectancy [15,25]. Surgical prognosis worsens over time and post operative recovery is dependent on the extent of the existing pathology [15,25]. DCM is therefore currently associated with lifelong disability, high levels of unemployment and amongst the poorest quality of life scores of any long-term condition [12,16,26,27]. The severity of DCM and management decision-making are determined using the Modified Japanese Orthopaedic Association (mJOA) scale or the Nurick grading system [12,28].

Because of missed diagnosis during the medical screening process, physiotherapists may be the first to recognize this condition, making it a priority for clinicians to understand its early manifesting features, with the aim of facilitating timely triage and management of DCM cases [9]. This article's aim is to summarize and enhance physiotherapists' essential knowledge of the differential diagnosis and management of patients with DCM.

Neuroanatomy and etiopathogenesis

The spinal cord consists of an H-shaped gray matter centrally, surrounded by white matter. The anterior white matter contains the ascending contralateral spinothalamic tract responsible for pain control, temperature, and fine touch, and the descending ipsilateral anterior corticospinal tract for motor function [15]. The leading cause of DCM is cervical spine stenosis, which can be congenital or secondary to intervertebral disc degeneration and spondylosis [12]. Canal stenosis and persistent compression reduce local blood flow, leading to ischemia of the cervical spinal cord [29]. Post-mortem histopathological studies have shown that demyelination in DCM predominantly occurs in the gray matter. This suggests that reduced perfusion through the anterior spinal artery and its terminal branches could play a relevant role in the etiopathogenesis. When the blood flow is reduced, the limited compensatory neovascularization leads to compromised perfusion and symptoms, further aggravated by repeated movements of the neck [8,12].

Depending on the location of the compression, symptoms can include pain in the neck, shoulders/arms, sensory deficits, motor weakness, impaired gate, and/or bladder dysfunction [11,30–32]. When white matter is involved, DCM mainly manifests as an upper motor neuron syndrome with spasticity and hyperreflexia. It has been observed in myelopathy related to mixed connective tissue disease that gray matter involvement seems to be less severe and has a more favorable prognosis. Usually, these patients do not present with disabling

neurological deficits; however, an increased risk of DCM recurrence exists [33]. Patients with DCM may also experience concurrent lower motor neuron dysfunction due to nerve root compression alongside central canal stenosis [11].

History taking

Patients with DCM may present with a wide range of symptoms and physiotherapists should comprehensively investigate the characteristic symptoms of the condition to ensure timely clinical action and prevent symptom worsening [34]. Table 1 reports the frequency and stage of commonly presenting DCM symptoms that should be investigated during history taking [35,36]. Generally, upper extremity symptoms (frequency 74%, 95%CI 70%–77%) are more commonly reported than the lower extremity (frequency 25%, 95%CI 22%–29%) [35]. Despite patients often experiencing bilateral symptoms, DCM should not be excluded when someone presents with unilateral symptoms (e.g. painful radiculopathy or myeloradiculopathy) [37]. In addition, clinicians should be aware that carpal tunnel syndrome may present bilaterally (with one-side dominance) with overlapping symptoms compared to DCM at the hand (e.g. paresthesia, hand wasting and loss of dexterity) [37].

It is worth noting that neck and/or shoulder pain and upper extremity pain are reported, respectively, in 51% (95% CI 49%–53%) and 43% (95% CI 40%–46%) of cases; whereas a minority of patients complain of back (frequency 19%, 95% CI 14%–27%) or lower extremity pain (frequency 10%, 95% CI 3%–24%) [35]. Other symptoms commonly related to DCM, such as the Lhermitte's phenomenon, are only present in 25% of the cases (95% CI 23%–29%) [35].

Severe DCM may present a variety of urinary symptoms with a frequency of 38% (95% CI 34%–43%), such as difficulty urinating, inability to completely empty the bladder, and spastic bladder (i.e. increased urinary frequency and incontinence) due to detrusor-sphincter dyssynergia and impaired feedback from the pontine micturition center [38,39]. Bowel dysfunction (e.g. defecation difficulty) and sexual dysfunctions are less frequent (respectively, 23 and 4% of the cases) [35] (Table 1).

Patients seeking physiotherapy care may not intuitively link their presenting symptoms with DCM, resulting in potential underreporting. Thus, it is important for physiotherapists to actively inquire about DCM-related symptoms. The majority of guidelines recommend using red flags to identify serious cervical pathology [40]. However, red flags for serious cervical pathology are mainly supported by expert opinions with a general lack of consensus among guidelines [41]. This leads to confusion and inconsistency in the management [40,41]. Therefore, it is essential to ascertain a clear understanding of the duration, progression, and nature of each and every symptom as this will significantly guide to determine the level of concern (index of suspicion) about the presence DCM. Astute clinicians should consider the evidence to support red flags, predict symptom progression and contextualize these findings within the individual profile of the person's health determinants, including the age of the patient, baseline function and their general health [40,42,43]. Thinking about the level of concern means to identify specific concerns about the generation of

Table 1. Frequency and stage of commonly presenting DCM symptoms that should be investigated during history taking *(adapted from Jiang et al.* [35]).

ASSOCIATED SYMPTOMS	FREQUENCY	STAGE
(1) Hand numbness	82% (95%CI 80%-85%)	Early
(2) Hand paresthesia	79% (95%CI 68%-87%)	Early
(3) Upper extremity symptoms	74% (95%CI 70%-77%)	Middle
(4) Gait impairment	72% (95%CI 70%-74%)	Early-Middle
(5) Hand clumsiness	69% (95%CI 67%-72%)	Middle
(6) Upper extremity numbness	69% (95%CI 66%-72%)	Middle
(7) Lower extremity numbness	61% (95% CI 57%-65%)	Late
(8) lower extremity paresthesia	58% (95%CI 37%-78%)	Late
(9) Upper extremity weakness	58% (95%CI 55%-60%)	Middle
(10) Upper extremity paresthesia	57% (95%CI 54%-60%)	Middle
(11) Lower extremity weakness	54% (95%CI 0.51%-57%)	Late
(12) Neck and/or shoulder pain	51% (95%CI 49%-53%)	Early
(13) Upper extremity pain	43% (95%CI 40%-46%)	Middle
(14) Radicular/radiating pain	39% (95%CI 35%-42%)	Middle
(15) Bladder dysfunction	38% (95%CI 34–42%)	Late
(16) Hand fine motor disturbance	29% (95%CI 25%-33%)	Early
(17) Bowel dysfunction	23% (95%CI 15%-33%)	Late
(18) Back pain	19% (95%CI 14%-27%)	Middle-Late
(19) Lower extremity pain	10% (95%CI 3%-24%)	Late

a DCM hypothesis, thinking about urgency of action required, and to reason about the safest approach with the best prognosis [44].

Clinical testing

Examination of DCM involves both upper and lower motor neuron assessment. Alongside the clinical history, the neurologic examination should aid clinicians in developing a working diagnosis and inform referral decision-making. The assessment of clinical signs for DCM should consider the signs of upper motor neuron imprairment, such as pathological hypertonic reflexes, upper limb motor impairment and gait function.

The assessment of reflexes, with particular attention to hypertonicity, is essential in the examination of the upper motor neuron signs. Several examination signs have been referred to within the literature, including, but not limited to, the Hoffmans sign, Tromner sign, Babinkski sign, Romberg's sign, deep tendon hyperreflexia and the inverted supinator sign [45]. However, there is no consensus with regard to the clinical signs which are most diagnostic and therefore must routinely be considered in the clinical examination for DCM. The presence of clinical examination signs are associated with greater disease severity, given the disease continuum and heterogeneity in underlying etiologies. The Tromner sign (a reverse Hoffmann sign) has demonstrated the higher diagnostic utility than any of the tests for DCM [45]. The accuracy of the Hoffmann's sign is dependant on disease severity and may be positive in only 3–7% of early diagnosed cases [46], with higher sensitivities in cases with severe cord compression (50–80% in surgical cases) [45,47]. Further, the Babinski sign, which demonstrates high specificity for DCM, has been associated with a poor mJOA score and is therefore a key clinical indicator warranting expedited referral for diagnostic imaging and surgical consultation [45,47]. Although this diagnostic decision rule has not been externally validated (i.e. in a different cohort than the one used to develop the diagnostic rule), a negative Hoffmann, Babinski, gait dysfunction, clonus and inverted supinator reflex (1 or fewer positive findings), may be helpful in ruling out DCM when interpreted in the context of a sound clinical reasoning framework [45]. Deep tendon hyperreflexia, a highly sensitive sign for DCM, demonstrates higher sensitivity in the lower extremities than in the upper extremities as a screening tool for DCM [45]. Exaggerated reflexes below the level of spinal cord compression can be explained by the reduced descending inhibition from the descending fibers of the corticospinal tract, which causes an exaggerated activation of motor neurons via the reflex arc [47]. Exaggerated responses may also be nonpathological in naturally hyperreflexic individuals but should be considered with relevance to the clinical history [19].

In DCM, early motor dysfunction may manifest as progressive weakness of the intrinsic muscles of the hands due to anterior horn cell damage [48]. Early assessment of grip strength, using a dynamometer, and hand function, such as clumsiness (e.g. using phone or buttoning clothes), are characteristics of early DCM [49,50]. Although accuracy is not reported, a positive grip and release test and the finger escape sign can be indicative of cervical cord impairment [49,50]. The characteristic nature of these signs allows the distinction between myelopathy and changes due to nerve root problems [51]. In assessing motor strength in DCM, hand, triceps, deltoids, and biceps strength testing are more sensitive tests for the upper extremities, whereas iliopsoas, quadriceps, and ankle dorsiflexion/plantarflexion strength testing are for the lower extremities [45]. Despite this, the presence of weakness in DCM is typically non-myotomal, in contrast to myotomal weakness seen in cases of cervical radiculopathy [45].

Gait changes seen in DCM cases include reduced cadence, increased step width, decreased step length, and longer stride time [52]. Although difficult to observe in early DCM, gait impairments are one of the earliest manifestations observed in 80% of cases by the Nurick grading system [19,22,48,53]. Severe DCM patients may exhibit specific gait characteristics, including spastic-like hyperextension of the knee in the stance phase without ankle dorsiflexion during the swing phase, and may rely on walking aids [54]. Clinicians could quantify walking time by performing the 30-meter walking test. This test may also reveal clumsiness and a lack of lower limb coordination, resulting in falls or unsteadiness [13,55]. Additionally, tandem gait/heel-to-toe walking is commonly used in clinical practice and is recognized as an integral part of the assessment of gait dysfunction in neurologic conditions [56–58]. However, there is no standardized or guideline-based protocol scoring system to help quantify imbalance and gait dysfunction in DCM [59]. Lower extremities' sensory proprioceptive disturbances may be assessed by the 10-step tandem gait test and the Romberg test [60,61]. However, as gait impairment and the Romberg sign are shared signs of a variety of conditions, they possess low sensitivity for DCM [45] and should be considered alongside other clinical signs and the clinical history [62].

The occurrence of symptom exacerbation or shooting pain with neck flexion, known as the Lhermitte sign, is a highly specific sign [8,11,13,30,63] and affects 27% of cases [64]. The Lhermitte's sign occurs when the demyelinated dorsal column of the spinal cord (primarily at the

cervical level) is stretched [65]. Neck movements in all directions should be evaluated to determine any symptom reproduction and discriminate, as far as possible and contextually, with cervical radiculopathy. For example, it is common that cervical extension might reproduce paresthesia in the extremities in patients suffering from radicular or myelopathic symptoms, with the involvement of one or both extremities aiding in the distinction [55].

To enhance the utility of clinical signs, it is relevant to screen for patient-reported DCM symptoms and utilize clusters of clinical signs suggesting spinal cord compression, prior to evaluating imaging [45]. Especially in early DCM stages, examination findings alone may not be sufficient to triage DCM, as upper motor neuron signs are strongly dependent on disease severity and may be, therefore, subtle or unremarkable [1,45]. In the absence of consistent and valid examination tools, the clinician must be equipped with a wide knowledge of tests to interpret in the context of a sound clinical reasoning framework [66]. Multiple clinical signs increase the specificity, but imaging and neurophysiology testings are required to confirm the diagnosis.

Figure 2 illustrates a decision tool (informed by the IFOMPT framework) to be followed when clinicians suspect DCM [40]. Clinical action is based on the stage and the severity of the presentation. The authors invite the reader to integrate Appendix 1 (which includes test descriptions and their diagnostic accuracy) to interpret findings in the context of the neurological examination, the diagnostic performance, and a sound clinical reasoning) [66].

Outcome measures and complementary assessment

Functional impairment and severity can be assessed using the mJOA scale or the Nurick grading system [12,28]. The mJOA scale evaluates various aspects of functional abilities, including upper and lower limb motor function, upper limb sensation, and sphincter function. It is scored on a scale of 0 to 18, with lower scores indicating greater disability [67]. The severity of DCM can be classified as mild (mJOA score 15–17), moderate (mJOA score 12–14), or severe (mJOA <12) (Table 2) [12,25,28]. Patients with mJOA scores of 12 or less generally have a poorer neurological recovery compared to those with a mJOA score above 12 [25]. The Nurick grading system uses a six-point scale to assess functional status, including gait impairments (Table 3) [67].

Imaging

A definitive diagnosis of DCM is confirmed by magnetic resonance imaging (MRI) [68]. MRI is considered the 'gold standard' with T2-weighted axial and sagittal views helping to characterize spinal cord compression, effacement of adjacent cerebrospinal fluid and in advanced cases myelomalacia (i.e. changes in cord signal intensity) [68]. MRI demonstrates high sensitivity and specificity (respectively, 79–95% and 82–89%, with LR + 4.39–7.92; LR- 0.06-0.27) in identifying selected abnormalities [69]. However, it is important to establish clinical-radiographic correlation due to the high prevalence of asymptomatic spinal cord compression, which alone does not directly equate to signs and symptoms [70]. In cases where symptoms and clinical signs of DCM are recognized, MRI should be performed even if electromyography suggests bilateral carpal tunnel syndrome, especially for patients with sensory disturbances in their hands [8]. Computed tomography (CT) scans may be helpful in the surgical-decision making process as it clearly visualizes bony and calcific changes, such as calcified disks, ossification of the posterior longitudinal ligament, and facet hypertrophy [8,19]. In patients where MRI is contraindicated, CT myelography provides diagnostic information on the degree of bony-related spinal cord compression. Plain radiography is not diagnostic as it lacks spinal cord visualization [71]. However, flexion-extension radiography could be considered if a spondylolisthesis with suspected instability is present [8].

Interpretation of findings and physiotherapy management

During management of DCM, the physiotherapist should assess the patient for relevant signs and symptoms that are reflective of prognosis and assess the need for referral for further evaluation or surgery. Early DCM may masquerade as other common conditions treated by physiotherapists (e.g. cervical radiculopathy, bilateral carpal tunnel syndrome) [25,72] and the variably progressive nature of DCM should prompt the physiotherapist to explore a past medical history. Physiotherapists should recognize subtle clues (readers are invited to refer to the History Taking section for more details) from the subjective examination leading to DCM hypothesis, and further confirm this hypothesis with a targeted physical examination [5,55,65,73–80]. They should systematically inquire about characteristic DCM symptoms, just as clinicians do when screening for conditions such as cauda equina syndrome. When DCM is suspected, referral for further medical opinion and diagnostic imaging are essential

Stage (Symptoms onset and progression)	Symptoms	Clinical signs (progressively cumulative)	Complementary assessment	Clinical action
Chronic: within several months/years)	Neck Pain and stiffness; sensory changes (pain, numbness, paresthesia) in one or both upper extremities; fine motor clumsiness; early gait impairment.	Finger escape sign, grip and release test, walking Romberg sign, 10-step tandem gait test, 30-meter walking test, brisk deep tendon reflexes.	mJOA (score 15-17, **mild presentation**).	Routine MRI should be considered to confirm the diagnosis. Referral required to deem extent of imaging findings. Consider a supervised trial of structured rehabilitation. Watchful waiting, safety net, and vigilance.
Sub-acute: within recent weeks/months	Clear gait impairment; followed progressively by hand clumsiness and sensory changes (numbness, paresthesia, reduced sensation) in the upper extremity; Lhermitte sign may be present.	Hyperreflexia, ataxic gate, spastic weakness in the lower extremities, upgoing clonus, Babinski, Tromner signs, Hoffmann signs, and Lhermitte sign. Lower motor neurons finding (muscle weakness/atrophy and reduced sensation) in the upper extremity.	mJOA (score 12-14, **moderate presentation**)	Referral and MRI are required. Consider surgical treatment.
Sub-acute: within weeks/months	Widespread sensory changes (numbness, paresthesia, reduced sensation) in the lower extremity; gait instability.	Spastic and unsteady gait.	mJOA (score 12-14, **moderate-severe presentation**)	Urgent referral, MRI, and surgical treatment are required.
Acute: within hours, days, or short week (*i.e. acute cord compression – very rare with DCM)*	Bladder/Bowel dysfunction.	Increased urinary frequency and incontinence; defecation difficulty; sexual dysfunctions.	mJOA (score 0-11, **severe presentation**)	Same day/emergency referral and surgical treatment are warranted.

Figure 2. Diagnostic triage and clinical actions to be followed when clinicians suspect DCM. Triage depends on local pathway, symptoms and their onset/progression, clinical signs, and complementary assessment. DCM, degenerative cervical myelopathy; ED, emergency department; MRI, Magnetic Resonance Imaging, mJOA, Modified Japanese Orthopaedic Association Scale.

Table 2. Modified Japanese Orthopaedic Association scale for cervical myelopathy evaluation (0 to 18 points) [67].

Evaluation Description	Point
Upper Extremity Motor Subscore (/5)	
Unable to move hands	0
Unable to eat with a spoon, but able to move hands	1
Unable to button a shirt but able to eat with a spoon	2
Able to button a shirt with great difficulty	3
Able to button a shirt with mild difficult OR other mild fine motor dysfunction (marked handwriting change, frequent dropping of objects, difficult clasping jewelry, etc.)	4
Normal hand coordination	5
Lower Extremity Subscore (/7)	
Complete loss of movement and sensation	0
Complete loss of movement, some sensation present	1
Inability to walk but some movement	2
Able to walk on flat ground with walking aid	3
Able to walk without walking aid, but must hold a handrail on stairs	4
Moderate to severe walking imbalance but able to perform stairs without handrail	5
Mild imbalance when standing OR walking	6
Normal walking	7
Upper Extremity Sensory Subscore (/3)	
Complete loss of hand sensation	0
Severe loss of hand sensation OR pain	1
Mild loss of hand sensation	2
Normal hand sensation	3
Urinary Function Subscore (/3)	
Inability to urinate voluntarily (requiring catheterization)	0
urinary incontinence (more than once per month)	1
Urinary urgency OR occasional stress incontinence (less than once per month)	2
Normal urinary function	3

Table 3. Nurick scale for clinical myelopathy evaluation (0 to 5 points) [67].

Grade	Description
0	Signs and symptoms of root involvement without spinal cord disease
1	Signs of spinal cord disease without difficulty in walking
2	Slight difficulty in walking that does not prevent full-time employment
3	Difficulty in walking that prevents full-time employment or daily takes without requiring assistance with walking
4	Ability to walk only with assistance
5	Chair bound or bedridden

Table 4. Information needs of people with degenerative cervical myelopathy.

Explanation of patients' MRI scan may enhance understanding of the condition, the need for and urgency of treatment, and the sense of experiential validation (surgeon-led).
Information about the condition, severity, and symptomatology.
Information about the treatment to the long-term clinical course.
Information about postoperative care and adjusting to life with a disability.
Information about the clinical course, also as safety net tool in the case symptomatology gets worse.

MRI, Magnetic Resonance Imaging.

for appropriate management [81]. Upon confirmation, patients should be informed of potential disease progression and conservative treatment options if mild symptoms are present [11,16,29].

Importantly, to ensure timely diagnosis and the best possible outcome, physiotherapists must work collaboratively with other healthcare professionals [73,81]. Given the complexity of the condition and the high risk of misdiagnosis, open communication, prompt access, and common language should be cultivated.

Education and safety netting

Educating the patient about the disease is a relevant aspect for the management of DCM progression. If DCM is suspected, it is important to inform the patient about the possible course of the disease and the signs of disease progression/deterioration [12]. Patients should be taught how to recognize signs and symptoms that are important regarding additional necessary actions that should be taken [55]. As an example, reported symptoms such as spastic paraparesis or bowel and bladder dysfunction are strong indicators for a prompt surgical evaluation [25]. When non-surgical management is identified, shared decision-making between both patient and surgeon and consistent follow-up should be maintained to monitor signs of DCM progression and safety netting information. Information about actions to be taken if the condition changes is given to the patient or the caregiver, so that they can seek help in a timely manner [12,40,76,82].

Patients often find it helpful to receive information about the condition, the treatment, the recovery, residual disability, and making adjustments in their daily life. Using patients' MRI scans as an educational tool

may improve understanding of the pathophysiology, foster the need for treatment, and enhance a sense of validation. However, information regarding the diagnosis, etiology, surgery, and recovery is often patient-specific and is in most cases surgeon-led [10]. Furthermore, involvement with support groups such as the Myelopathy.org Facebook community, acts as an informational resource and forum for patients, positively influencing their mental health and distress levels [83]. While surgical decisions are made in a shared-decision making manner led by surgeons, it is the responsibility of physiotherapists to recognize DCM signs and symptoms and direct patients toward the timely triage of suspected DCM cases. Suggestions regarding conservative therapies, safety netting information, minimizing the risk of falls, and avoiding whiplash and cervical manipulation is critical for patients with DCM, as these may lead to further spinal cord/canal compromise [12,84]. Table 4 summarize the information that a patient with DCM may need.

Management

Surgery is recommended for moderate-to-severe cases of DCM [85]. In patients with mild DCM, an initial conservative approach may be considered [8,19,25,63,85–88]. The anterior (e.g. anterior cervical discectomy and fusion), posterior (e.g. laminectomy and instrument fusion, laminoplasty) or combined surgical approach is identified based on the number of levels involved and the severity [63,88]. The primary objective of a surgical approach has traditionally been to prevent further deterioration and maintain the current neurological status [12]. Recent promising evidence showed that surgical decompression could lead also to neurological function improvement [12]. Surgery may provide a positive impact on long-term neurological function (e.g. improving gait stability), disability, and health-related quality of life [12], particularly for those patients with severe myelopathic changes [85,87]. A minority of individuals (<5%) may also achieve a full recovery [10,25]. Notably, surgery was associated with 6.5%–16.6% of episodes of adverse events or complications (e.g. permanent morbidity, prolonged hospitalization, surgical reoperation). Therefore, the potential risks and benefits should be carefully considered when deciding the management pathway [10,25].

Several non-operative, conservative management approaches for DCM that encompass bracing, analgesics, therapeutic exercise, manual therapy, bed rest, and avoidance of risky activities and environments (e.g. slippery floors or contact sports) are reported [12,25,84]. The overall principles of musculoskeletal interventions involve ROM and mobility exercises, flexibility training, stabilization exercises of deep neck flexor muscles, gentle strengthening of upper quarter muscles, activity modification, postural awareness, balance training, and the implementation of home exercise programs [86].

The long-term outcomes of non-operative treatment are poorly studied, including the risk of complications following musculoskeletal interventions [12,25,85]. A significant proportion of patients (23%–54%) who initially undergo non-operative management eventually require surgical intervention within a follow-up period between 29–74 months. Therefore, multimodal structured non-operative care may delay the progression of DCM but does not lead to long-lasting effects [12,85].

Conclusion

Although DCM is the most prevalent cause of spinal cord impairment in adults, there is a lack of clinical studies to provide a comprehensive understanding of its natural history and progression. Particularly, diagnosis of early stages DCM is a real challenge. It is important to interpret findings in the context of patient-reported symptoms, a complete neurological examination, and a sound clinical reasoning framework. The decision for the management approach should be individualized and shared with the surgical team and the patient. Based on current evidence, surgical intervention remains the primary treatment for DCM.

Recommendations/Implications for practice

Clinicians must recognize and understand the early symptoms, avoiding underestimating subtle clues. Early detection and timely intervention are essential for improving patient outcomes and drastically enhancing the prognosis. There are no current diagnostic criteria or validated interview tools for DCM screening. Thus, recognition of DCM features relies on the physiotherapist's clinical reasoning and pattern recognition skills. Although it may be challenging at first, physiotherapists working in a direct access setting should develop the knowledge and ability needed to triage for DCM appropriately.

In non-severe cases, conservative treatment may be indicated. However, the long-term effectiveness of conservative approaches remains unclear. Rehabilitation may play a crucial role in optimizing surgical outcomes, but conservative treatments should be mainly proposed for milder cases or patients that present increased risks to surgery. A multidisciplinary approach should be adopted to ensure the best possible management strategy.

Disclosure statement

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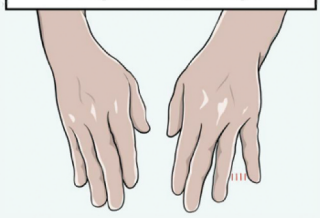
Appendix 1 Summary of potentially relevant neurological tests in Degenerative Cervical Myelopathy

Finger Escape Sign



DESCRIPTION & SIGNIFICANCE
The patient is asked to extend and adduct the fingers and maintain that position for up to a minute.
The test is positive when the 5th digit drifts into abduction.
The characteristic nature of the signs allows the distinction between myelopathy and changes due to nerve root or peripheral nerve entrapment.

INTERPRETATION
Decreased dexterity in the intrinsic muscles of the hand due to pyramidal tract damage, leading to problems in handwriting, typing, shirt buttoning, or other fine motor tasks.

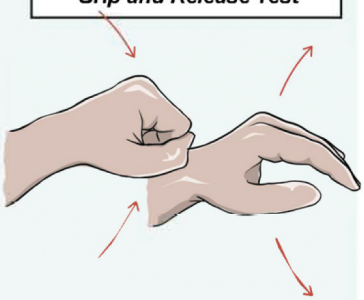
ACCURACY
N/R

Grip and Release Test

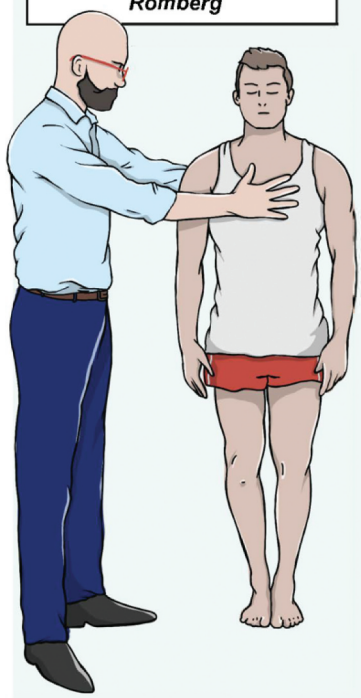
DESCRIPTION & SIGNIFICANCE
With the palms facing downward and the forearms placed on a stable armrest in pronation to minimize the influence of the forearm motions, the patient is asked to repeatedly grip and release as fast as possible.
The test is positive when the patient exhibits a reduced performance compared to healthy. It is thought to be normal when the patient performs over 20 motion cycles in a 10-second period. Quality of motion should also be observed.
The characteristic nature of the signs allows the distinction between myelopathy and changes due to nerve root or peripheral nerve entrapment.

INTERPRETATION
Decreased dexterity in the intrinsic muscles of the hand due to pyramidal tract damage, leading to problems in handwriting, typing, shirt buttoning, or other fine motor tasks.

ACCURACY
N/R



Romberg



DESCRIPTION & SIGNIFICANCE
With the patient without shoes and standing with both feet together, arms next to the body or crossed in front of the body:

1. The clinician assesses the patient's body movement relative to balance while the patient stands erect and keep eyes open;
2. The clinician observes swaying of the body and notes any balance impairment for 1 minute while the patient stands erect and keep eyes closed.

Note: swaying of the body indicates the proprioceptive balance correction for the lack of visual or vestibular compensation.

The sensitivity of the test can be increased by:
- "Sharpened Romberg test" by narrowing the patient's base of support with feet in a heel-to-toe tandem position;
- Standing with their eyes closed on a compliant surface. Using a foam rubber surface nullify the proprioceptive inputs from the foot allows to test the vestibular system rather than that of proprioception.

The test is positive when the patient loses balance with their eyes closed. Loss of balance is defined as increased body swaying with eyes closed, foot movement in the direction of the fall, or falling.

INTERPRETATION
Sensory disturbances in the lower extremities due to proprioception deficits secondary to posterior cord compression. Posterior dorsal column damage leads to dysfunction of vibration sense, deep touch, proprioception, and joint positioning.

ACCURACY
Romberg test has significant larger differences between open and closed eye and between patients with myelopathy compared to a normal population. Increasing differences in Romberg test is significantly associated with increased odds of having symptomatic myelopathy, cord compression on imaging.

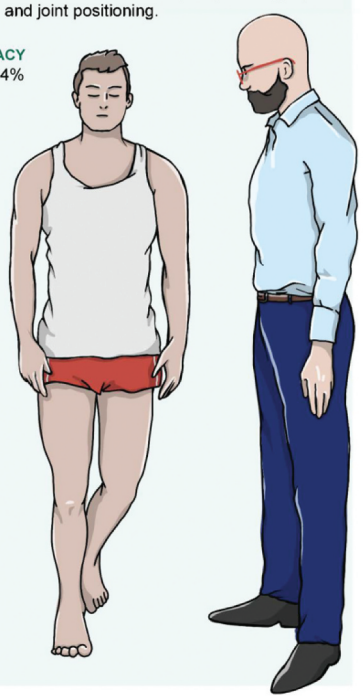
Sn 34-49%

Walking Romberg

DESCRIPTION & SIGNIFICANCE
The patient is asked to walk 5 meters with their eyes open and then 5 meters with their eyes closed.
The test is positive when the patient sways, feels of instability or is unable to complete the walk with eyes closed.

INTERPRETATION
Sensory disturbances in the lower extremities due to proprioception deficits secondary to posterior cord compression. Posterior dorsal column damage leads to dysfunction of vibration sense, deep touch, proprioception, and joint positioning.

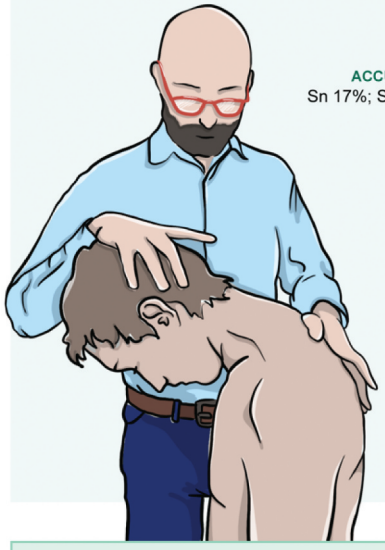
ACCURACY
Sn 34-74%



Lhermitte Sign

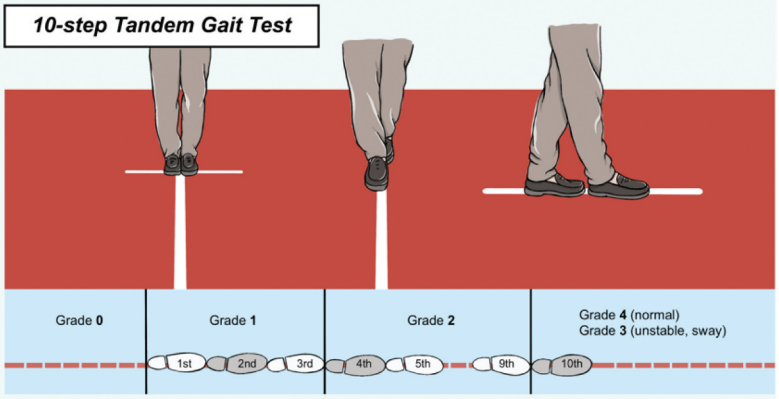
DESCRIPTION & SIGNIFICANCE
With the patient in sitting, the examiner asks for an assisted cervical flexion of neck.
The test is positive when the patient presents shooting pain down the spine, upon flexion of the neck.

INTERPRETATION
Sensory changes due to compression of the spinothalamic tract, posterior column, and spinal roots. The Lhermitte sign occurs when a demyelinated dorsal column of the spinal cord is stretched at the cervical region. It is caused by miscommunication between the affected nerves that have lost their protective myelin coating.



ACCURACY
Sn 17%; Sp 97%

10-step Tandem Gait Test



DESCRIPTION & SIGNIFICANCE
With the patients standing comfortably with their feet together, the clinician instructs the patient to carefully walk 10 steps in a straight lined-up flat floor. The patient performs a toe-to-heal touch with each step at their own velocity. The number of steps made before the first misstep is counted. The test is repeated a second time in the same line and the number of steps is counted in the same manner. Of the two tests, the one with a higher tandem gait grade is assessed.
The test is defined in five grades according the JOA sub-scores as follows: grade 0 (impossible to walk), grade 1 (≤ 3 steps), grade 2 (< 10 steps), grade 3 (10 steps, but unstable with swaying from side to side), and grade 4 (10 steps, normal).

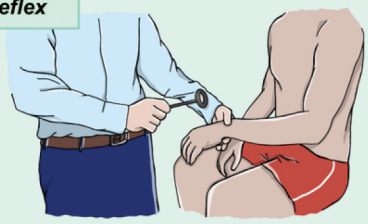
INTERPRETATION
Sensory disturbances in the lower extremities due to proprioception deficits secondary to posterior cord compression. Posterior dorsal column damage leads to dysfunction of vibration sense, deep touch, proprioception, and joint positioning.

ACCURACY
The Tandem Gait step and grade showed significant correlations with gait parameters and the degree of association was comparable to those of the Japanese Orthopaedic Association sub-score and Nurick scale. The balance parameters showed higher correlation coefficients with the tandem gait grades and the Nurick scale grades than the Japanese Orthopaedic Association sub-scores.

Hyperreflexia

Hyperreflexia is an upper motor neurone sign. Hyperreflexia results from decreased inhibitory input from a damage to the descending fibers of the corticospinal tract; this decreased inhibition causes exaggerated activation of motor neurons from connections with both sensory neurons and interneurons.

Hand Withdrawal Reflex

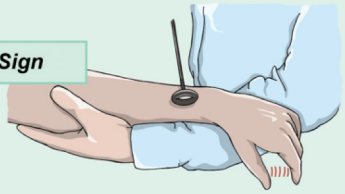


DESCRIPTION & SIGNIFICANCE
With the patient sitting or standing, the examiner grasps the patient's palm and strikes the dorsum of the patient's hand with a reflex hammer.

The test is positive when an abnormal response of flexor muscles is observed.

ACCURACY
Sn 39%; Sp 63%
+LR 1.05; -LR .97

*Inverted Supinator Sign

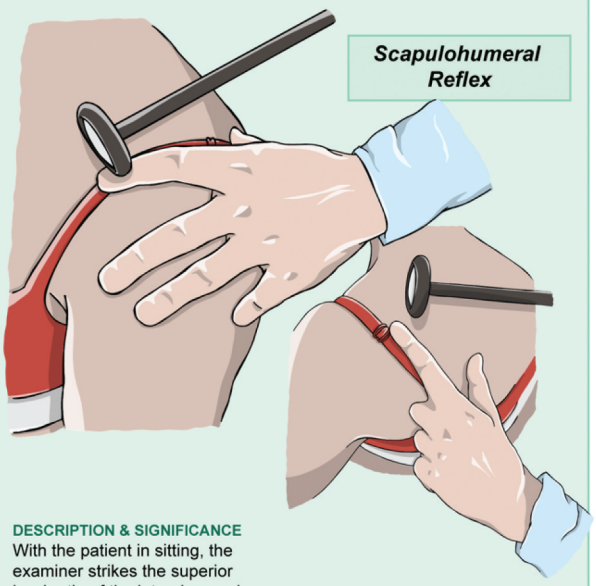


DESCRIPTION & SIGNIFICANCE
With the patient in a seated position with the forearm in pronated relaxed position.

The examiner applies a series of quick strikes with a reflex hammer near the styloid process of the radius at the attachment of the brachioradialis tendon.
The test is positive when finger flexion is observed.

ACCURACY
Sn 18-75%; Sp 78-99%
+LR 2.71-14.64; -LR .29-.83
For compression at C5-6 level

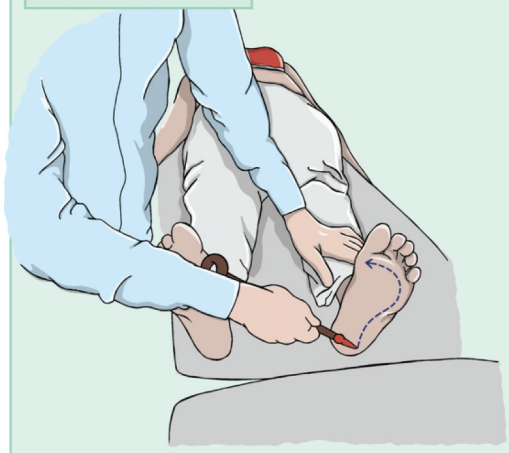
Scapulohumeral Reflex



DESCRIPTION & SIGNIFICANCE
With the patient in sitting, the examiner strikes the superior border tip of the lateral acromion process or the midpoint of the spine of the scapula with a reflex hummer.
The test is positive when the patient involuntarily shrugs or abducts the shoulder.

ACCURACY
Sn 20%

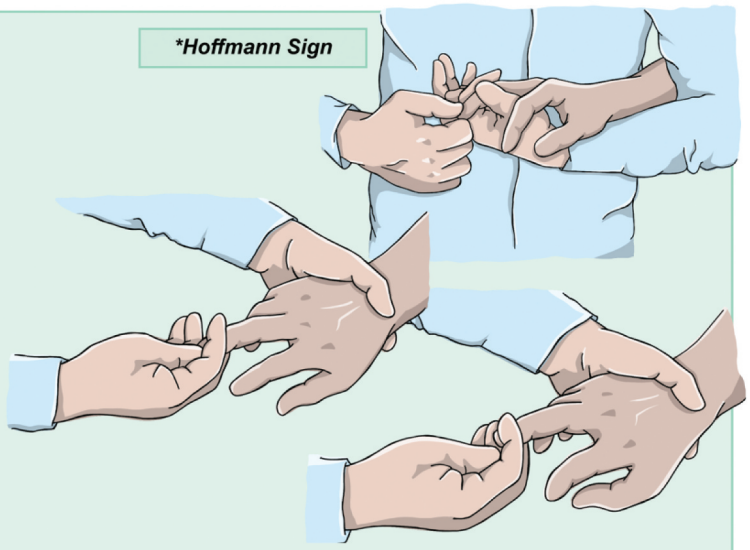
*Babinski Sign



DESCRIPTION & SIGNIFICANCE
With the patient in supine position, the examiner stimulates the plantar aspect of the foot (typically lateral to median from heel to metatarsal) with the blunt end of a reflex hammer.
The test is positive when an extension of the great toe and fanning of the fingers is observed.

ACCURACY
Sn 7-36%; Sp 93-100%
+LR 4.50; -LR .064-.093

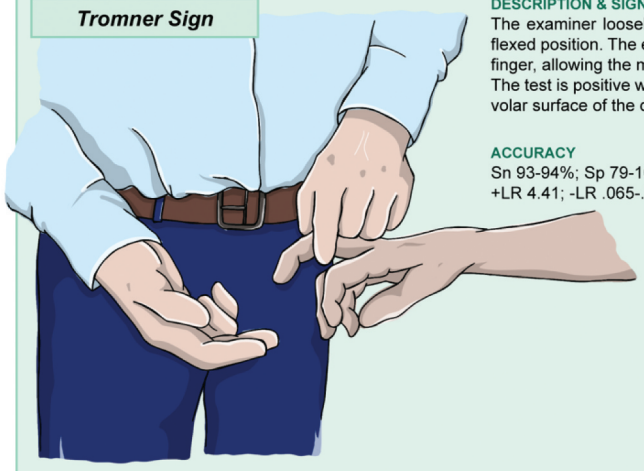
*Hoffmann Sign



DESCRIPTION & SIGNIFICANCE
The examiner loosely holds the middle finger just proximal to the distal interphalangeal joint and tap or rapidly flicks the fingernail downward, allowing the middle finger to flick upward reflexively.
The test is positive when the thumb and or index finger involuntary flex when the examiner flicks the fingernail of the middle finger down.

ACCURACY
Sn 31-89%; Sp 50-100%
+LR 1.15-10.50; -LR .011-.095

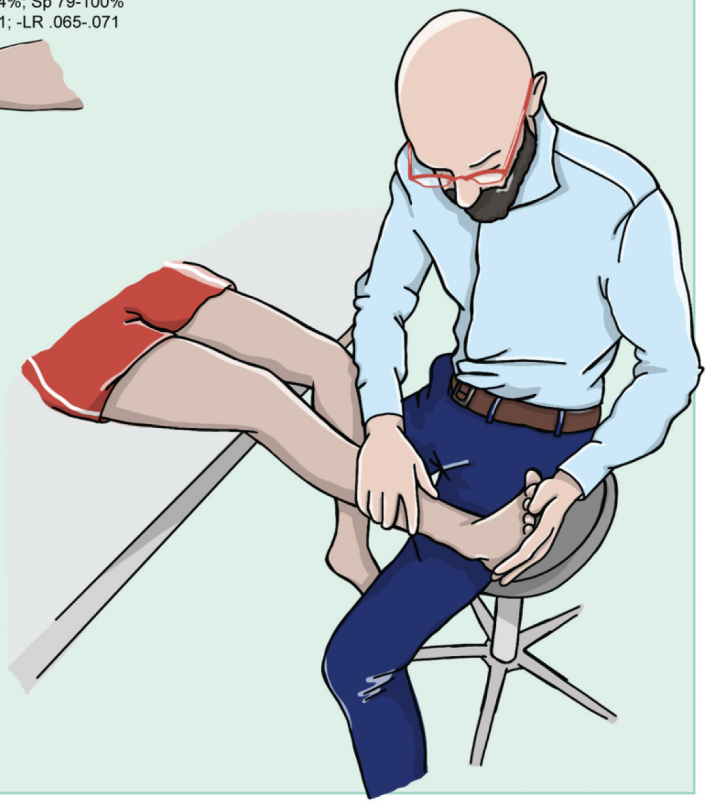
Tromner Sign



DESCRIPTION & SIGNIFICANCE
The examiner loosely holds the middle finger just proximal to the distal interphalangeal joint in a flexed position. The examiner then taps or rapidly flicks upward the volar surface of the distal middle finger, allowing the middle finger to flick upward reflexively.
The test is positive when the thumb and or index finger involuntary flex when the examiner flicks the volar surface of the distal middle finger.

ACCURACY
Sn 93-94%; Sp 79-100%
+LR 4.41; -LR .065-.071

Clonus



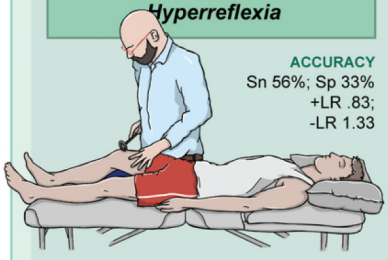
DESCRIPTION & SIGNIFICANCE
With the patient in sitting position with the feet off the ground or lying supine, the examiner applies a quick stretch to the Achilles tendon doing a passive rapid ankle dorsiflexion.
A positive response is observed when the patient's ankle "beats" in and out of dorsiflexion > 3 beats in succession.

ACCURACY
Sn 7-13%; Sp 96-100%
+LR 3.00-5.49; -LR .87-.94

Deep Tendon Hyperreflexia

Hyperreflexia at any deep tendon has moderate sensitivity (72%; -LR .35-1.22) but low specificity (43%; +LR .90-1.78).
For reflex testing, the patient is in a comfortable and relaxed position. The clinician strikes the tendon to be tested with a reflex hammer.
Upper extremities hyperreflexia can help to recognize the level of compression (e.g., pectoralis reflex may indicate a compression above C4).

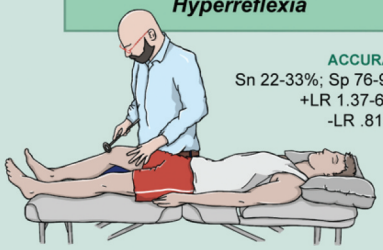
Suprapatellar Tendon Hyperreflexia



ACCURACY
Sn 56%; Sp 33%
+LR .83;
-LR 1.33

DESCRIPTION & SIGNIFICANCE
With the patient lying supine or sitting with feet off the ground, the examiner applies quick strikes with a reflex hammer the suprapatellar tendon.
The test is positive when an hyperreflexive knee extension is observed.

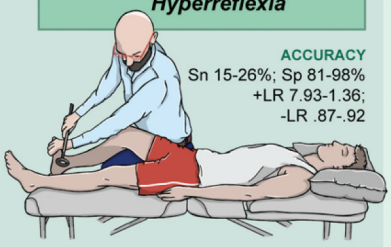
Patellar Tendon Hyperreflexia



ACCURACY
Sn 22-33%; Sp 76-97%
+LR 1.37-6.95;
-LR .81-.88

DESCRIPTION & SIGNIFICANCE
With the patient lying supine or sitting with feet off the ground, the examiner takes of the weight of one of the patient's knees and strikes the patellar tendon.
Test is positive if an exaggerated contraction of the quadricep's tendon causes extension of the knee.

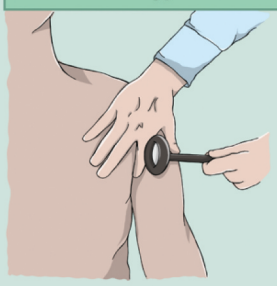
Achilles Tendon Hyperreflexia



ACCURACY
Sn 15-26%; Sp 81-98%
+LR 7.93-1.36;
-LR .87-.92

DESCRIPTION & SIGNIFICANCE
With the patient lying supine or sitting with feet off the ground or lying supine, the examiner flexes one of the patient's feet and strikes the Achilles tendon.
The test is positive if an exaggerated contraction of the gastrocnemius tendon causes plantarflexion of the foot.

Pectoralis Hyperreflexia



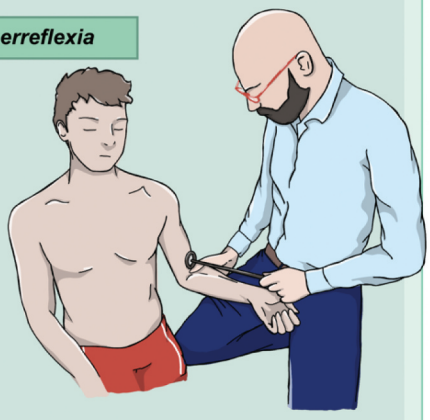
DESCRIPTION & SIGNIFICANCE
With the patient sitting the examiner places one finger lightly on the tendon of the pectoralis major at the deltopectoral groove and strikes the finger with the reflex hammer.
The test is positive when hyperreflexive adduction and internal rotation of the shoulder is observed.

ACCURACY
Sn 85%; Sp 97%
For compression above C4 level

Biceps Hyperreflexia

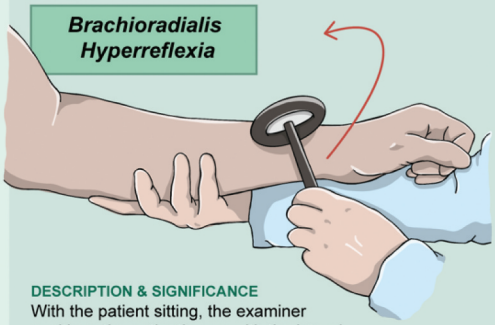
DESCRIPTION & SIGNIFICANCE
With the patient sitting or lying supine, the examiner slightly supinates the patient's forearm and tests the reflex by striking the biceps tendon.

The test is positive when an exaggerated biceps contraction causing flexion of the elbow is observed.



ACCURACY
Sn 18-62%; Sp 96-49%; +LR 4.88-1.20; -LR .79-.85

Brachioradialis Hyperreflexia



DESCRIPTION & SIGNIFICANCE
With the patient sitting, the examiner positions the patient's arm with the lateral side upwards and tests the reflex by striking the lower end of the radius, just proximal to the styloid process of the radius.
The test is positive when exaggerated supination (i.e, elbow flexion and wrist extension) is observed.

ACCURACY
Sn 21%; Sp 89%
+LR 1.90; -LR .89

Triceps Hyperreflexia

DESCRIPTION & SIGNIFICANCE
With the patient sitting or lying supine, the examiner places the patient's hand on the contralateral shoulder, keeping it in a flexed position and tests the reflex by striking the triceps tendon.
The test is positive when an exaggerated triceps contraction causing extension of the elbow is observed.

ACCURACY
Sn 36%; Sp 78%
+LR 1.66; -LR .82

+LR, positive likelihood ratio; -LR, negative likelihood ratio; Sn, sensitivity; Sp, specificity; N.R., not reported. References:
Cook C, Roman M, Stewart KM, Leithe LG, Isaacs R. Reliability and Diagnostic Accuracy of Clinical Special Tests for Myelopathy in Patients Seen for Cervical Dysfunction. Journal of Orthopaedic & Sports Physical Therapy. 2009;39(3):172-178.
Fogarty A, Lenza E, Gupta G, Jarzem P, Dasgupta K, Radhakrishna M. A Systematic Review of the Utility of the Hoffmann Sign for the Diagnosis of Degenerative Cervical Myelopathy. Spine. 2018;43(23):1664-1669.
Jiang Z, Davies B, Zipser C, et al. The value of Clinical signs in the diagnosis of Degenerative Cervical Myelopathy - A Systematic review and Meta-analysis. Global Spine Journal. Published online October 2023:21925682231209869.
Ver MLP, Gum JL, Glassman SD, Carreon LY. Assessment of standing balance in normal versus cervical spondylotic myelopathy patients. North American Spine Society Journal (NASSJ). 2020;3:100023.

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