

Hand Examination in Neurology: Recognizing Localizing Clinical Signs

Exame das mãos em neurologia: reconhecimento de sinais clínicos localizatórios

Helena Bethlem Mirow¹ , Amanda de Moraes Affonso¹ , Fernanda de Miranda Santos¹ , Roberto Pereira Santos² 
Victor Evangelista Rodrigues Pereira³ 

¹Medical Student, Estácio de Sá University (IDOMED), School of Medicine, Rio de Janeiro, Rio de Janeiro, Brazil;

²MD, PhD Substitute Professor of Neurology, School of Medicine, Federal University of Rio de Janeiro (UFRJ), Rio de Janeiro, Rio de Janeiro, Brazil;

³MD, MSc Neurologist, Clementino Fraga Filho University Hospital (HUCFF), Federal University of Rio de Janeiro (UFRJ), Rio de Janeiro, Rio de Janeiro, Brazil

ABSTRACT

Background: The hands provide a unique window into neurological disease and may reveal subtle findings that facilitate syndromic, topographic, and etiological diagnosis. Despite their clinical importance, neurological hand findings are seldom reviewed comprehensively using an anatomically based framework.

Objective: To review the principal neurological hand signs and postures, emphasizing their clinical appearance, neuroanatomical basis, diagnostic value, historical context, and main differential diagnoses.

Methods: We conducted a narrative review of PubMed, the Cochrane Library, and Google Scholar, with no lower publication-date limit and with the search updated through June 2026. Search concepts included neurological examination, hand signs, clinical semiology, movement disorders, myelopathy, brachial plexopathy, peripheral neuropathy, motor neuron disease, and neuromuscular disorders. Classic semiology textbooks, historical reports, and reference lists of selected publications were also examined.

Results: Findings were organized into manifestations related to cortical and interhemispheric networks, the corticospinal tract, basal ganglia and thalamus, spinal cord and proprioceptive pathways, brachial plexus, peripheral nerves, motor unit and muscle, and selected systemic or metabolic disorders. Findings not exclusive to the hand were retained only when their hand expression has recognized localizing or diagnostic value.

Conclusions: Careful inspection and examination of the hands provide diagnostic information that extends beyond assessment of strength and sensation. When integrated with the complete neurological examination, characteristic hand signs can improve lesion localization, narrow the differential diagnosis, and support early recognition of neurological and selected systemic disorders.

Keywords: Neurologic Examination; Hand; Physical Examination; Semiology; Neuroanatomical Localization.

RESUMO

Introdução: As mãos oferecem uma janela singular para as doenças neurológicas e podem revelar sinais sutis úteis ao diagnóstico síndrome, topográfico e etiológico. Apesar de sua relevância, os achados neurológicos observáveis nas mãos raramente são revisados de forma abrangente e organizada pela localização anatômica.

Objetivo: Revisar os principais sinais e posturas das mãos de interesse neurológico, descrevendo sua apresentação clínica, base neuroanatômica, valor diagnóstico, contexto histórico e diagnósticos diferenciais.

Métodos: Foi realizada revisão narrativa nas bases PubMed, Cochrane Library e Google Scholar, sem restrição de data inicial e com atualização da busca até junho de 2026. Foram utilizados termos relacionados ao exame neurológico, sinais das mãos, semiologia clínica, distúrbios do movimento, mielopatia, plexopatia, neuropatia periférica, doença do neurônio motor e doenças neuromusculares. Livros clássicos de semiologia, artigos históricos e referências dos estudos selecionados também foram examinados.

Resultados: Os achados foram organizados em manifestações relacionadas ao córtex e às redes inter-hemisféricas; trato corticoespinal; núcleos da base e tálamo; medula espinal e vias proprioceptivas; plexo braquial; nervos periféricos; unidade motora e músculo; e condições sistêmicas ou metabólicas. Alterações que não são exclusivas das mãos foram mantidas apenas quando sua expressão manual apresenta utilidade localizatória ou diagnóstica reconhecida.

Conclusão: A inspeção e o exame cuidadoso das mãos fornecem informações que ultrapassam a avaliação de força e sensibilidade. Integrados ao restante do exame neurológico, sinais manuais característicos podem melhorar a localização da lesão, estreitar o diagnóstico diferencial e favorecer o reconhecimento precoce de doenças neurológicas e sistêmicas selecionadas.

Palavras-chave: Exame Neurológico; Mão; Exame Físico; Semiologia; Localização Neuroanatômica.

ARTICLE INFO

Received: June 27, 2026

Received in this final form: August 04, 2026

Accepted: August 27, 2026

DOI: <https://doi.org/10.46979/rbn.v62i3.75250>

Corresponding Author:

Victor Evangelista Rodrigues Pereira

Federal University of Rio de Janeiro (UFRJ)

Rua Professor Rodolpho Paulo Rocco, 255 - Cidade Universitária, Rio de Janeiro - RJ, 21941-617, Brazil

Email: victorevangelista01@hotmail.com

Conflicts of Interest

The authors declare no conflicts of interest.

Funding

No funding was received.

Author contributions

Author contributions (CRediT): All authors contributed to Conceptualization, Methodology, Investigation, Writing – Original Draft, Writing – Review & Editing, and Visualization. All authors reviewed and approved the final manuscript. All authors contributed equally to this work.

INTRODUCTION

The human hand is an evolutionary marvel. It has played a pivotal role in human evolution, enabling key milestones such as tool use, written language, and fine-motor interactions with the environment. Functionally, the hand supports many human activities, from manipulation and exploration to communication and self-expression. Its motor capabilities underlie tasks such as writing, playing musical instruments, performing surgery, and countless other skilled actions that define human dexterity.

Hand function depends on the coordinated activity of multiple levels of the nervous system. Voluntary movement originates in cortical motor planning areas and the primary motor cortex, with descending commands traveling through the corticospinal tract to lower motor neurons in the anterior horn of the spinal cord. Their axons continue through peripheral nerves to the neuromuscular junction, activating the intrinsic and extrinsic muscles responsible for dexterity. Precise movement also requires continuous sensory feedback from cutaneous and proprioceptive receptors, allowing ongoing adjustment of motor output. Beyond voluntary movement, the hands provide diagnostic clues through involuntary motor phenomena and structural changes. Fasciculations, dystonia, myotonia, and tremor may be early manifestations of neurological disease, whereas muscle atrophy, clawing, and skin or nail abnormalities may reflect chronic denervation or systemic disorders with neurological involvement. Accordingly, lesions at many levels of the neuroaxis can produce characteristic abnormalities on hand examination, making it an essential component of the neurological assessment.

Within clinical reasoning, certain semiological findings provide high-yield diagnostic information, guiding syndromic, topographical, and etiological localization.^{1,2} Many of these clues are found in the hands, a readily accessible yet often underexamined region. Owing to their anatomical complexity and extensive representation throughout the neuroaxis, the hands may reveal abnormalities arising from lesions ranging from the cerebral cortex to the peripheral nerves. Despite their diagnostic value, reviews specifically focused on neurological signs of the hands remain limited. This narrative review addresses this gap by organizing clinically relevant hand signs according to lesion topography, providing a practical, anatomically based framework for bedside neurological assessment.

The objective of this review is to provide a practical, anatomically organized account of hand signs that support neurological localization or diagnosis, while distinguishing focal hand signs from broader motor phenomena whose expression in the hand remains clinically informative.

To assemble this narrative review, PubMed was selected as the principal biomedical indexing database, the

Cochrane Library was searched for evidence syntheses, and Google Scholar was used to identify historical descriptions, books, and older articles that may not be consistently indexed in modern databases. Searches combined the concepts “neurological examination,” “hand sign,” “clinical semiology,” “apraxia,” “dystonia,” “myelopathy,” “brachial plexopathy,” “peripheral neuropathy,” “motor neuron disease,” and the names and eponyms of individual signs. No lower date limit was applied; the search was updated through June 2026. Articles in English, Portuguese, and French were considered. Reference lists were screened manually, and classic semiology textbooks and landmark historical reports were included when they represented the original or standard description of a sign. Because this was a narrative review, no formal risk-of-bias instrument or quantitative synthesis was applied.

Signs were included when the hand was the principal site of observation or when a broader neurological disorder produced a recognizable manual manifestation with localizing, diagnostic, or differential-diagnostic value. Tremor, athetosis, tics, and formal bradykinesia and rigidity assessment were excluded because they require dedicated examination frameworks and are reviewed extensively elsewhere. Non-specific hand findings without recognized localizing or diagnostic relevance were also excluded.

All illustrative figures were generated as educational reconstructions using OpenAI’s image-generation tool in ChatGPT (accessed August 3–4, 2026), iteratively refined using author-selected clinical reference images, and manually reviewed, labeled, and assembled by the authors. They are not patient photographs and contain no identifiable patient data.

MAIN TEXT

Historical perspective

Neurological examination has long relied on characteristic hand postures and responses. Trousseau described carpopedal spasm in the nineteenth century; Babinski used hand contracture to distinguish organic from hysterical motor disorders in 1893; forced grasping and groping were formalized in the early twentieth century; Oppenheim’s “useless hand” linked loss of manual function to proprioceptive deafferentation; and Ono and colleagues later systematized the “myelopathy hand” in cervical cord disease.³⁻⁷ These descriptions remain clinically useful when interpreted alongside modern neuroanatomy and contemporary diagnostic testing.

1. Cortical and interhemispheric network disorders

The following phenomena are not simply fixed deformities of the hand. They are higher-order motor or agency disorders expressed prominently through manual behavior and are included because the hand often provides the clearest bedside clue to cortical or callosal dysfunction.

Hand Apraxia

Hand apraxia is a disorder of learned skilled movement in which patients are unable to perform purposeful actions despite adequate strength, coordination, primary sensation, comprehension, and cooperation. The deficit reflects impaired motor planning rather than elementary motor or sensory dysfunction and is most often associated with dominant frontal or parietal lesions, although subcortical disconnection may also contribute. Clinical subtypes include ideomotor apraxia, ideational apraxia, and limb-kinetic apraxia. Dressing apraxia is conceptually distinct and is more often related to non-dominant parietal dysfunction. Because apraxia is not exclusive to the hand, examination should compare manual gestures with other learned actions and should exclude weakness, sensory loss, ataxia, and impaired comprehension.^{2,8}

Grasping and Groping

Grasping and groping are stimulus-bound motor behaviors produced by impaired frontal inhibitory control. Grasping refers to involuntary closure of the hand after palmar contact, whereas groping describes exploratory or compulsive reaching toward nearby objects. These phenomena were emphasized in early twentieth-century descriptions of frontal lobe dysfunction and are most strongly associated with medial or anterior frontal lesions, although they may occur with more extensive hemispheric disease.^{3,9}

Alien Hand Phenomenon

Alien hand syndrome is characterized by involuntary but apparently purposeful hand movements accompanied by impaired sense of agency or ownership. Three practical variants are recognized: a frontal variant with grasping and utilization behavior; a callosal variant with intermanual conflict; and a posterior variant with limb levitation and cortical sensory abnormalities. The phenomenon may follow medial frontal, callosal, parietal, thalamic, or corticobasal network lesions. Distinguishing alien hand from dystonia, chorea, seizures, neglect, and functional movement disorders requires attention to agency, task context, and associated cortical signs.¹⁰⁻¹²

2. Corticospinal tract and upper motor neuron manifestations

Spasticity is not hand-specific; however, asymmetric finger postures and reflex responses may expose mild corticospinal dysfunction before conventional strength testing becomes abnormal.

Digitus Quinti Sign

The digitus quinti sign is a subtle indicator of corticospinal tract dysfunction, characterized by spontaneous abduction of the fifth finger during sustained arm extension (Figure 1A). It is elicited by asking the patient to extend both arms forward with the forearms pronated and the fingers adducted. A positive sign is defined by involuntary separation of one little finger, often preceding overt weakness in patients with mild pyramidal tract involvement. Reported diagnostic performance is modest, with a sensitivity of approximately 51% and a specificity of 70%¹³. Because bilateral fifth-finger abduction may represent a physiological variant, only asymmetric findings should be considered pathological. Although the initial appearance may be similar, the distinction from ulnar neuropathy is usually evident on neurological examination. Ulnar neuropathy is characterized by intrinsic hand muscle weakness and atrophy, sensory loss in the fifth and ulnar half of the fourth digit, and other signs of peripheral nerve dysfunction. In contrast, corticospinal tract lesions produce a pyramidal pattern of weakness, frequently accompanied by hyperreflexia and spasticity¹³⁻¹⁵.

Spastic Hand

The spastic hand is characterized by a flexed posture of the wrist, fingers, and thumb resulting from increased muscle tone after an upper motor neuron lesion. The thumb is typically adducted and flexed into the palm, contributing to the characteristic clenched appearance (Figure 1B). This posture is usually accompanied by a pyramidal pattern of weakness, hyperreflexia, and velocity-dependent spasticity, reflecting chronic corticospinal tract dysfunction.¹⁶

Hoffmann and Tromner Signs

The Hoffmann and Tromner signs are clinical maneuvers used to elicit a flexor response of the fingers. In the Hoffmann sign, a sudden flicking of the distal phalanx of the middle or ring finger induces involuntary flexion of the thumb and/or index finger. The Tromner sign, a variant of the same reflex, involves tapping the palmar surface of the distal phalanx, similarly triggering a flexor response of the thumb and fingers. Historically, these signs were considered by some authors as upper-limb equivalents of the Babinski sign and were often interpreted as markers of pyramidal tract disease. However, studies and clinical experience—particularly Robert Wartenberg's observations—have demonstrated that these responses can be present in healthy individuals without neurological pathology. Wartenberg emphasized that the Hoffmann and Tromner signs are best understood as alternative methods for

eliciting the deep tendon reflexes of the finger flexors, rather than as reliable indicators of corticospinal tract lesions. Their presence, especially when symmetric and not accompanied by other signs of pyramidal dysfunction, should not be interpreted as pathological¹⁷.

Synkinetic Wrist Extension Sign

Although wrist drop is not strictly a hand deformity, as it results from weakness of the forearm wrist extensors, it is a classic finding of the distal upper limb and an important diagnostic clue in neurological practice. Wrist drop may result from either central lesions, particularly involving the cortical hand knob area, or peripheral radial nerve dysfunction, making their distinction clinically relevant. The synkinetic wrist extension sign is a bedside maneuver that helps differentiate these conditions¹⁸. Normally, forceful finger flexion is accompanied by automatic activation of the wrist extensors, stabilizing the wrist during grip. The patient is first asked to extend the wrist voluntarily and then to clench the fist while the examiner observes for involuntary wrist extension. Preservation of wrist extension during fist closure despite impaired voluntary extension suggests a central lesion, reflecting preserved automatic motor synergies despite impaired corticospinal output. In contrast, both voluntary and synkinetic wrist extension are absent in radial neuropathy because the peripheral motor pathway is interrupted¹⁹.

Hand Contracture Sign

The hand contracture sign, originally described by Babinski in 1893, is a bedside maneuver that may help distinguish organic spasticity from functional hand posturing⁴ (Figure 1C). In functional neurological disorders, apparent flexion contracture often reflects active muscle contraction or inconsistent resistance, making passive opening of the hand difficult. In contrast, the flexed posture associated with corticospinal tract lesions usually allows gradual passive finger extension. The distinction should not rely on the hand posture alone but on the overall neurological examination, including the presence of pyramidal weakness, hyperreflexia, and consistent upper motor neuron findings. When interpreted in the appropriate clinical context, this maneuver may contribute to the differential diagnosis between functional and structural neurological disorders²⁰.

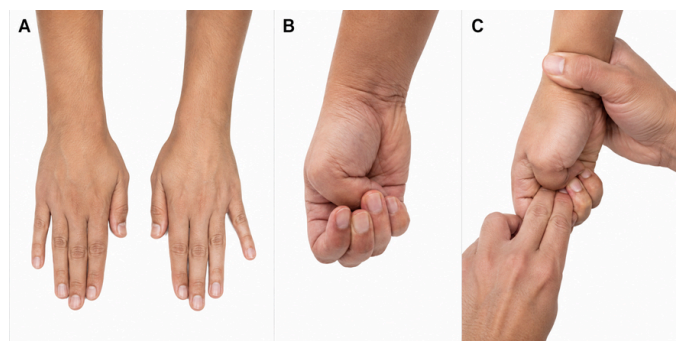


Figure 1. Corticospinal tract and upper motor neuron hand manifestations. (A) Digits quinti sign, characterized by asymmetric spontaneous abduction of the fifth finger while the remaining fingers are extended and adducted. (B) Spastic hand posture, with wrist and finger flexion and thumb adduction into the palm. (C) Babinski's hand contracture maneuver: the examiner attempts to introduce the fingers between the patient's tightly flexed fingers and palm to assess the nature of the apparent contracture. AI-generated educational reconstruction based on author-selected clinical references; not derived from identifiable patient photographs.

3. Basal ganglia, thalamus, and movement-disorder postures

Dystonia and chorea are generalized movement phenomenologies rather than diseases of the hand. They are included here only when a characteristic manual posture or task-specific finding contributes materially to diagnosis or differential diagnosis.

Thalamic Hand

The thalamic hand is a characteristic dystonic posture resulting from lesions of the posterolateral thalamus, typically involving the ventral posterior nuclei and adjacent structures. The wrist is mildly flexed and pronated, the metacarpophalangeal joints are flexed, the proximal interphalangeal joints extended, and the fingers assume an irregular, athetoid posture that may become more evident during voluntary movement. This abnormal posture reflects disruption of thalamic sensorimotor integration and is often accompanied by hemisensory loss, hemiataxia, and central post-stroke pain (Dejerine-Roussy syndrome). Recognition of this characteristic hand posture provides an important clue to a thalamic localization.²¹.

Dystonic Hand

Hand dystonia is characterized by sustained or intermittent muscle contractions that produce patterned, twisting movements or abnormal postures of the fingers and wrist. Common manifestations include paradoxical extension of one or more fingers, abnormal hand posturing

during voluntary movement, and task-specific dystonia, such as writer's cramp. A characteristic clinical feature is the geste antagoniste, in which a light tactile or proprioceptive stimulus transiently improves the abnormal posture. These manifestations reflect dysfunction of the basal ganglia-thalamo-cortical network²².

Dystonic hand postures may occasionally resemble spasticity, but the distinction is usually straightforward. Unlike dystonia, spasticity is associated with a pyramidal pattern of weakness, velocity-dependent hypertonia, hyperreflexia, and other upper motor neuron signs, whereas strength is typically preserved in primary dystonia.^{23,24}

Starfish hand

The starfish hand is a descriptive term for a distinctive dystonic posture characterized by abduction and extension of all five fingers, often accompanied by wrist extension, producing a flattened, starfish-like appearance (Figure 2A). The posture may be present at rest or become more prominent during voluntary movement and reflects abnormal co-contraction of agonist and antagonist muscles, a hallmark of dystonia. It is typically asymmetric and has been described in patients with corticobasal syndrome and, less commonly, following structural lesions involving the basal ganglia and related motor networks²⁵.

Striatal Hand

The striatal hand is a characteristic dystonic posture defined by flexion of the metacarpophalangeal joints with relative extension of the interphalangeal joints, often accompanied by ulnar deviation and flexion of the wrist²⁶ (Figure 2B). It reflects basal ganglia dysfunction and is most commonly observed in Parkinson's disease and atypical parkinsonian syndromes. Striatal hand deformity has been reported in approximately 8–10% of patients with Parkinson's disease and usually occurs on the side with more severe motor involvement. Recognition of this posture may facilitate the early identification of parkinsonism, particularly when accompanied by other extrapyramidal signs²⁷.

Pointing gun posture or Pistol sign

The pointing gun posture, also known as the pistol sign, is a distinctive dystonic hand posture characterized by extension of the thumb and index finger with flexion of the remaining digits (Figure 2C). It is most commonly observed during voluntary movement or while maintaining a posture and represents a form of focal hand dystonia. Although uncommon, this sign has been described in atypical parkinsonian syndromes, particularly progressive supranuclear palsy (PSP) and corticobasal syndrome (CBS),

where it may occur in up to 26% of patients with PSP^{28,29}. Its recognition should prompt careful evaluation for additional clinical features of atypical parkinsonism^{24,30}.

Spooning Hand

The spooning hand is elicited by asking the patient to extend both arms forward. The hand assumes a characteristic posture with hyperextension of the metacarpophalangeal joints and flexion of the wrist, producing a concave, "spoon-like" appearance (Figure 2D). In some patients, the posture is accompanied by excessive forearm pronation. This finding reflects hypotonia and is most commonly observed in cerebellar disorders and chorea. Although non-specific, recognition of the spooning hand may provide a useful clue to disorders associated with reduced muscle tone³¹.

Milkmaid's grip

Milkmaid's grip is characterized by an irregular, alternating squeezing and relaxation of the examiner's fingers when the patient attempts to maintain a sustained handshake or grip, resembling the rhythmic motion of hand milking³². The phenomenon reflects the inability to sustain a constant grip because of superimposed choreiform movements and is a classic bedside sign of chorea, particularly in Huntington's disease and Sydenham chorea. Although commonly associated with motor impersistence, it primarily reflects basal ganglia dysfunction disrupting sustained voluntary muscle contraction. Recognition of milkmaid's grip helps distinguish chorea from other hyperkinetic movement disorders³³.

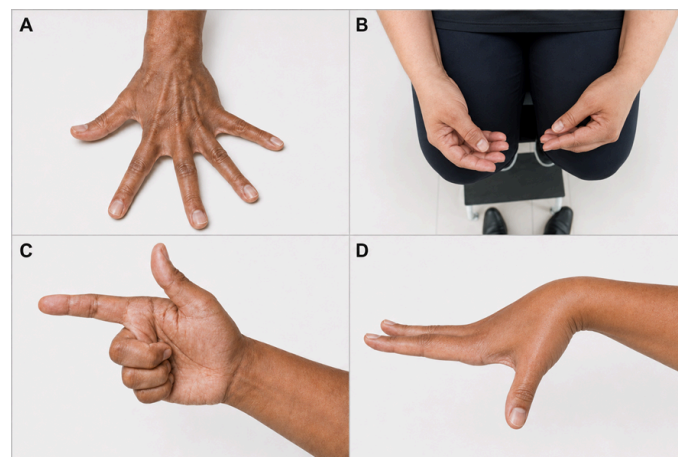


Figure 2. Basal ganglia and movement-disorder hand postures. (A) Starfish hand, characterized by marked extension and abduction of all five fingers. (B) Striatal hand, showing an asymmetric posture with metacarpophalangeal flexion, relative interphalangeal extension, ulnar deviation, and thumb adduction. (C) Pointing-gun posture or pistol sign, with extension of the index finger and thumb and flexion of the remaining fingers. (D) Spooning hand, characterized by wrist flexion and metacarpophalangeal hyperextension producing a concave, spoon-like contour. AI-generated educational reconstructions based on author-selected clinical references; not derived from identifiable patient photographs.

4. Spinal cord and proprioceptive pathway disorders

Ono's Myelopathic Hand

Ono's myelopathic hand is an early manifestation of cervical myelopathy, originally described by Ono and colleagues in 1987. The earliest finding is weakness of little-finger adduction, reflecting involvement of the intrinsic hand muscles (Figure 3A). As myelopathy progresses, weakness may extend to the ring and middle fingers, followed by impaired extension of these digits. These abnormalities are often accompanied by hyperactive triceps and finger flexor reflexes, supporting corticospinal tract involvement. Recognition of this characteristic pattern is particularly useful in patients with cervical cord disorders, in whom hand findings may precede more obvious signs of myelopathy⁵.

Marinescu's succulent hand (main succulente de Marinesco)

Marinescu's succulent hand (main succulente de Marinesco) is characterized by diffuse swelling of the hand with soft-tissue thickening, smooth, glossy skin, and tapering fingers, producing a plump or edematous appearance³⁴ (Figure 3B). The finding is thought to reflect trophic and autonomic changes resulting from chronic lower motor neuron denervation, including impaired vasomotor regulation. It is classically described in patients with syringomyelia but may also occur in other chronic disorders affecting the anterior horn cells or lower motor neurons. Recognition of this distinctive trophic pattern may provide an important clue to longstanding lower motor neuron dysfunction³⁵.

Useless Hand of Oppenheim

Oppenheim's useless hand describes the profound loss of hand dexterity caused by severe proprioceptive deafferentation despite preserved muscle strength. Without adequate joint-position sense, voluntary movements become inaccurate and poorly coordinated, rendering the hand functionally "useless," particularly when visual feedback is removed. Examination demonstrates marked impairment of position and vibration sense, often accompanied by pseudoathetosis that improves with visual guidance. The sign reflects disruption of proprioceptive pathways rather than primary motor dysfunction and is most commonly associated with lesions of the dorsal columns, particularly in the cervical spinal cord, although it may occur in any disorder producing severe proprioceptive loss⁶.

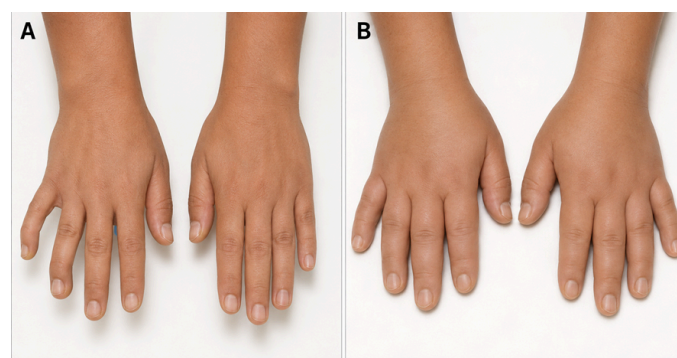


Figure 3. Spinal cord-associated hand manifestations. (A) Ono's myelopathic hand, characterized by impaired extension and adduction of the ulnar digits, particularly the little finger, during sustained hand extension. The contralateral hand is shown for comparison. (B) Marinescu's succulent hand (main succulente de Marinesco), showing bilateral broadening and thickening of the hands and fingers, with smooth, fleshy contours and reduced prominence of the underlying tendons and bony landmarks. AI-generated educational reconstruction based on author-selected clinical references; not derived from identifiable patient photographs.

5. Brachial plexus lesions and reinnervation phenomena

Erb-Duchenne Hand (The Waiter's Tip Posture)

Upper brachial plexus palsy (C5–C6), or Erb-Duchenne palsy, produces the characteristic waiter's tip posture, with the arm adducted and internally rotated, the elbow extended, the forearm pronated, and the wrist and fingers flexed (Figure 4A). Although the primary deficit affects the proximal muscles of the shoulder and arm, the hand assumes a flexed posture because of unopposed distal flexor activity. Intrinsic hand muscles are usually preserved, as they are predominantly supplied by the C8–T1 roots. In longstanding or congenital cases, however, secondary contractures and developmental disuse may lead to fixed hand deformities. Recognition of this posture is useful in localizing an upper brachial plexus lesion and distinguishing it from more extensive plexus injuries.²

Dejerine-Klumpke Palsy

Dejerine-Klumpke palsy results from injury to the lower brachial plexus (C8–T1) and is characterized by paralysis of the intrinsic hand muscles, producing a claw hand deformity with impaired grip and fine motor function. The metacarpophalangeal joints become hyperextended while the interphalangeal joints flex because of denervation of the lumbricals and interossei, often accompanied by thenar and hypothenar atrophy (Figure 4B). Unlike Erb-Duchenne palsy, shoulder and elbow function are largely preserved, whereas distal hand weakness predominates. When the T1 root is involved, ipsilateral Horner's syndrome (pseudoptosis, miosis, and anhidrosis) may also be present because of interruption of sympathetic fibers. Common causes include birth trauma, traction injuries, thoracic outlet syndrome, and apical lung tumors. Recognition of this characteristic hand posture helps localize the lesion to the lower brachial plexus.²

Breathing Hand Syndrome

The breathing hand is a rare form of respiratory synkinesis in which involuntary contractions of the intrinsic hand muscles occur synchronously with respiration, becoming most evident during deep inspiration. It has been described primarily in patients with severe brachial plexus injuries, particularly obstetric brachial plexopathy. The phenomenon is thought to result from aberrant reinnervation, whereby regenerating respiratory motor fibers—most likely from the phrenic or intercostal nerves—misdirect into the lower brachial plexus and reinnervate intrinsic hand muscles. Similar respiratory synkinesis involving the proximal upper limb ("breathing arm") is more common. Recognition of this unusual finding suggests a severe proximal brachial plexus lesion with preserved nerve continuity sufficient to permit aberrant axonal regeneration^{36,37}.

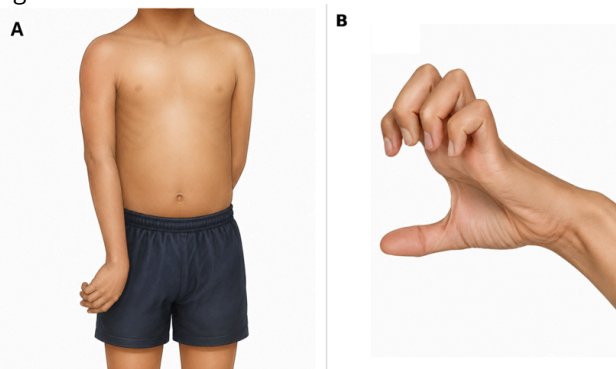


Figure 4. Upper-limb postures associated with brachial plexus lesions. (A) Erb-Duchenne palsy, showing the characteristic waiter's-tip posture, with shoulder adduction and internal rotation, elbow extension, forearm pronation, wrist flexion, and flexed fingers. (B) Dejerine-Klumpke palsy, showing a claw-hand posture characterized by metacarpophalangeal hyperextension and flexion of the proximal and distal interphalangeal joints due to lower brachial plexus involvement. AI-generated educational reconstructions based on author-selected clinical references; not derived from identifiable patient photographs.

6. Peripheral nerve lesions

Simian Hand (Ape Hand deformity)

The simian hand, or ape hand deformity, is characterized by wasting of the thenar eminence with loss of thumb opposition, causing the thumb to lie in the same plane as the fingers (Figure 5A). The deformity reflects paralysis of the thenar muscles innervated by the median nerve and is most evident during inspection and attempted thumb opposition or precision grip. It is classically associated with proximal median nerve lesions, in which denervation involves both the recurrent motor branch to the thenar muscles and the anterior interosseous branch. Common causes include traumatic injury, compressive neuropathies, and other proximal median nerve lesions. Recognition of this characteristic posture readily localizes the deficit to the median nerve³⁸.

Claw Hand

Claw hand is characterized by hyperextension of the metacarpophalangeal joints and flexion of the proximal and distal interphalangeal joints, predominantly affecting the fourth and fifth fingers (Figure 5B). The deformity results from denervation of the ulnar-innervated intrinsic hand muscles, particularly the interossei and the medial two lumbricals, leaving the long finger extensors and flexors unopposed. It is the hallmark of chronic ulnar neuropathy and is typically more pronounced in distal lesions at the wrist, where the flexor digitorum profundus remains intact. In proximal ulnar nerve lesions, weakness of the flexor digitorum profundus to the ring and little fingers reduces flexion at the distal interphalangeal joints, making the clawing less conspicuous—a phenomenon known as the ulnar paradox^{38,39}.

Benediction hand

The benediction hand is a dynamic deformity observed during attempted fist formation. In proximal median nerve lesions, the patient is unable to fully flex the thumb, index, and middle fingers because of weakness of the flexor pollicis longus, flexor digitorum superficialis, and the lateral half of the flexor digitorum profundus, with additional impairment of metacarpophalangeal flexion from paralysis of the lateral lumbricals. As a result, the ring and little fingers flex normally while the index and middle fingers remain relatively extended, producing the characteristic "hand of benediction." Unlike the ulnar claw, this posture is present only during active finger flexion and is not evident at rest. It is classically associated with traumatic or compressive lesions of the median nerve in the arm or proximal forearm^{31,38,40}.

Finger Drop Sign

Finger drop refers to selective weakness of the finger extensors, particularly the extensor digitorum communis and extensor indicis proprius, resulting in an inability to extend the fingers despite preserved wrist extension (Figure 5C). This pattern reflects most commonly lesions of the posterior interosseous nerve, a radial nerve branch^{41,42}. When a patient presents with isolated finger drop or asymmetric finger drop, it should raise suspicion for multifocal motor neuropathy. The condition is immune-mediated and strongly associated with anti-GM1 IgM antibodies. Recognition of this distinct motor pattern is crucial, as MMN responds well to IVIG therapy⁴³.

Kiloh-Nevin Syndrome

Kiloh-Nevin syndrome, or anterior interosseous nerve (AIN) syndrome, is a pure motor neuropathy involving the anterior interosseous branch of the median nerve. It causes weakness of the flexor pollicis longus, the radial portion of the flexor digitorum profundus (to the index and

often the middle finger), and the pronator quadratus. The hallmark finding is an abnormal "OK" sign: instead of forming a tip-to-tip pinch, the patient approximates the pads of the thumb and index finger because of impaired flexion of the thumb interphalangeal joint and the index distal interphalangeal joint (Figure 5D). Unlike more proximal median nerve lesions, AIN syndrome produces no sensory deficits. Causes include compression, trauma, and neuritis, and the syndrome may occur as part of neuralgic amyotrophy (Parsonage-Turner syndrome)^{44,45}.

Wartenberg Sign

Wartenberg's sign is characterized by persistent abduction of the fifth finger at rest due to weakness of the third palmar interosseous muscle, resulting in unopposed action of the extensor digiti minimi. Patients often report that the little finger catches on clothing or becomes caught when placing the hand in a pocket. The sign is most commonly associated with ulnar neuropathy, particularly at the elbow, and may represent an early manifestation of motor involvement. Unlike the claw hand deformity, Wartenberg's sign is a static postural abnormality rather than a deformity that develops during finger flexion. It should also be distinguished from the digiti quinti sign, which reflects corticospinal tract dysfunction. Associated intrinsic hand muscle weakness, interosseous atrophy, and sensory loss in the ulnar distribution support a peripheral ulnar nerve lesion.^{39,46}

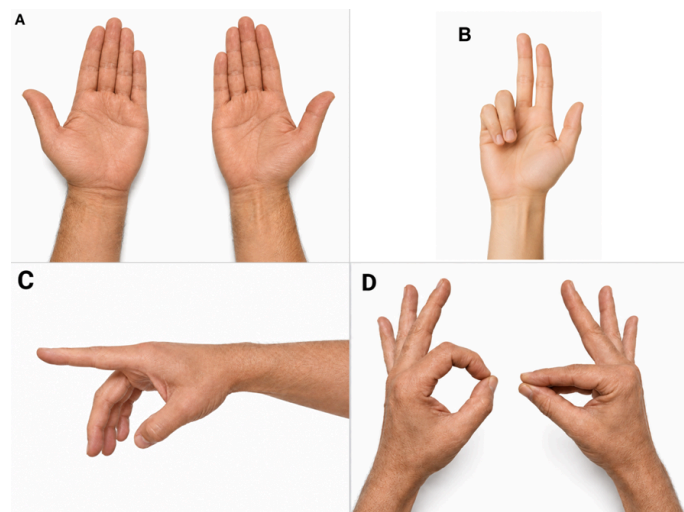


Figure 5. Characteristic hand postures associated with peripheral nerve lesions. (A) Simian hand, showing flattening and atrophy of the thenar eminence due to median nerve dysfunction, with preservation of thumb size. The contralateral hand is shown for comparison. (B) Ulnar claw hand, characterized by relative extension of the thumb, index, and middle fingers, with predominant clawing of the ring and little fingers. (C) Finger drop, showing preserved wrist and index-finger extension with variable dropping of the remaining fingers. (D) Kiloh-Nevin syndrome due to anterior interosseous nerve palsy. The normal hand forms a tip-to-tip "OK" sign, whereas the affected hand produces a flattened pulp-to-pulp pinch because of weakness of the flexor pollicis longus and the flexor digitorum profundus to the index finger. AI-generated educational reconstructions based on author-selected clinical references; not derived from identifiable patient photographs.

7. Motor neuron, neuromuscular, and muscle disorders

Myotonic Hand Phenomenon

The myotonic hand phenomenon is characterized by delayed relaxation after voluntary grip or percussion of the thenar or hypothenar muscles. It is a hallmark of myotonic disorders, including myotonic dystrophy type 1 and non-dystrophic myotonias such as myotonia congenita and paramyotonia congenita. The phenomenon results from sarcolemmal hyperexcitability caused by ion channel dysfunction, leading to persistent muscle contraction after activation. In myotonic dystrophy, grip myotonia is often accompanied by distal weakness and muscle wasting, whereas non-dystrophic myotonias typically present without fixed weakness. Electromyography demonstrates characteristic myotonic discharges. Recognition of delayed grip relaxation is an important bedside clue to myotonic disorders and may represent one of their earliest clinical manifestations^{47,48}.

Crab Hand Posture

The crab hand posture refers to the characteristic appearance produced by selective weakness of the finger flexors, particularly the flexor digitorum profundus of the index and middle fingers. During attempted grip, the index and middle fingers fail to flex adequately at the distal interphalangeal joints, creating a pincer-like appearance reminiscent of a crab's claw (Figure 6A). This selective pattern of finger flexor weakness, especially when accompanied by quadriceps weakness, is highly characteristic of IBM and helps distinguish it from other inflammatory myopathies. Recognition of this pattern should prompt consideration of inclusion body myositis, particularly in older adults with slowly progressive distal hand weakness.⁴⁹

Split-hand sign

The split-hand sign is characterized by selective wasting of the abductor pollicis brevis and first dorsal interosseous muscles, with relative preservation of the hypothenar muscles, producing a visible dissociation between the radial and ulnar aspects of the hand (Figure 6B). This characteristic pattern is strongly associated with amyotrophic lateral sclerosis (ALS) and reflects preferential involvement of motor neurons supplying the lateral intrinsic hand muscles. The finding may be unilateral or asymmetric early in the disease and often precedes more widespread upper and lower motor neuron signs. Because this pattern cannot be explained by a single median or ulnar nerve lesion, it is an important clue favoring a motor neuron

disorder. Electrophysiological studies typically demonstrate reduced compound muscle action potentials in the abductor pollicis brevis and first dorsal interosseous with relative preservation of hypothenar responses.^{50,51}

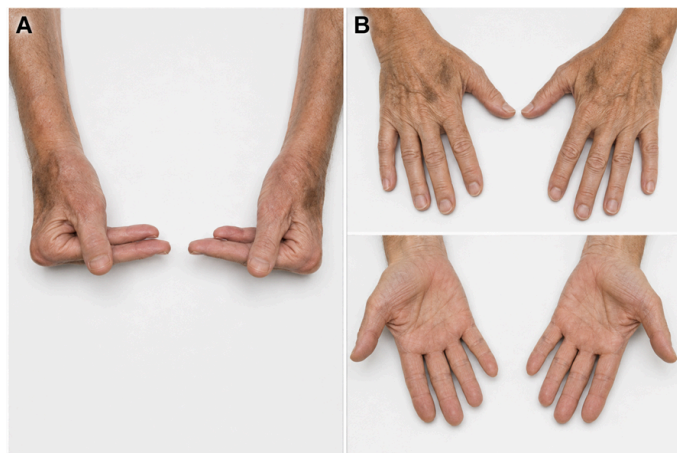


Figure 6. Characteristic hand findings in inclusion body myositis and motor neuron disease. (A) Crab-hand posture in inclusion body myositis, characterized by selective weakness of the flexor digitorum profundus to the index and middle fingers, which remain extended during attempted fist closure, with relative preservation of flexion of the ring and little fingers. (B) Split-hand phenomenon, shown in dorsal and palmar views, with preferential wasting of the thenar muscles and first dorsal interosseous muscle and relative preservation of the hypothenar eminence. Finger length and bulk are preserved because the atrophy predominantly affects the intrinsic hand muscles rather than the digits themselves. AI-generated educational reconstructions based on author-selected clinical references; not derived from identifiable patient photographs.

8. Systemic and metabolic disorders with characteristic hand findings

These findings do not arise from a focal lesion of the nervous system. They are retained because the hand posture or skin phenotype may provide an immediate clue to a systemic disorder with neurological manifestations or neuromuscular excitability.

Mechanic's Hands

Mechanic's hands are characterized by hyperkeratotic, fissured, and hyperpigmented skin involving the radial aspects of the fingers, particularly the thumb and index finger, giving the appearance of a manual laborer's hands. The finding is strongly associated with antisynthetase syndrome and dermatomyositis and may precede or accompany muscle weakness. It often occurs together with other cutaneous manifestations, such as Gottron's papules, and should prompt evaluation for myositis-specific autoantibodies, interstitial lung disease, and other features of idiopathic inflammatory myopathies. Histopathology is nonspecific, typically showing hyperkeratosis and acanthosis⁵².

Obstetrician's Hand (Trousseau's Sign)

The obstetrician's hand is the characteristic posture elicited by Trousseau's sign, a manifestation of latent tetany caused by hypocalcemia. After inflation of a

blood pressure cuff above systolic pressure for 2–3 minutes, patients develop a carpopedal spasm with wrist flexion, flexion of the metacarpophalangeal joints, extension of the interphalangeal joints, and thumb adduction into the palm. This posture reflects increased neuromuscular excitability resulting from reduced extracellular calcium. Trousseau's sign is more sensitive than Chvostek's sign for detecting hypocalcemia and is classically observed in hypoparathyroidism, vitamin D deficiency, acute pancreatitis, and following parathyroid surgery. Recognition of this characteristic hand posture should prompt evaluation for an underlying electrolyte disturbance.^{7,53}

LIMITATIONS

This review has several limitations. As a narrative review, study selection and interpretation were not governed by a preregistered systematic-review protocol and remain susceptible to selection bias. Many classical signs have not been evaluated in large prospective cohorts using contemporary reference standards; therefore, their evidentiary basis often consists of historical descriptions, expert observation, small series, or teaching reports. Terminology is also inconsistent across specialties, and some eponyms describe overlapping postures or broader syndromes rather than hand-specific abnormalities. Finally, no individual sign should be interpreted in isolation: diagnostic value depends on asymmetry, associated neurological findings, clinical context, and confirmation by appropriate ancillary testing.

CONCLUSION

Hand examination extends well beyond the assessment of strength and sensation. Careful observation of posture, spontaneous and stimulus-bound movement, muscle bulk, reflex responses, trophic changes, and task-specific performance can provide clues to lesion localization throughout the neuroaxis. Organizing these findings by cortical and interhemispheric networks, corticospinal pathways, deep gray structures, spinal cord, brachial plexus, peripheral nerves, motor unit, muscle, and systemic disease clarifies why some manifestations are hand-specific whereas others represent broader neurological dysfunction expressed through the hand. Integrated with the remainder of the neurological examination, these signs can sharpen diagnostic reasoning while preserving the historical and practical value of bedside semiology.

REFERENCES

1. Santos RP, Fernandes RCL. Clinical reasoning unveiled: demystifying heuristics and hermeneutics in neurological clinical practice. *Rev Bras Neurol.* 2024;60(3):16-23. doi:10.46979/rbn.v60i3.65939.
2. Ropper AH, Samuels MA, Klein JP, Prasad S. Adams and Victor's *Principles of Neurology*. 12th ed. New York: McGraw Hill; 2023. p. 68-69.

3. Adie WJ, Critchley M. Forced grasping and groping. *Brain*. 1927;50(2):142-170. doi:10.1093/brain/50.2.142.
4. Babinski J. Contracture organique et hystérique. *Bull Mem Soc Med Hop Paris*. 1893;10:327-343.
5. Ono K, Ebara S, Fuji T, Yonenobu K, Fujiwara K, Yamashita K. Myelopathy hand: new clinical signs of cervical cord damage. *J Bone Joint Surg Br*. 1987;69(2):215-219. doi:10.1302/0301-620X.69B2.3818752.
6. Wiblin L, Guadagno J. The useless hand of Oppenheim. *Pract Neurol*. 2017;17(6):464-468. doi:10.1136/practneurol-2016-001494.
7. Patel M, McMahon K, Hu EW. Trousseau sign. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK557832/>.
8. Mendez MF, Parand L. Limb apraxias and related disorders. In: Daroff RB, Jankovic J, Mazziotta JC, Pomeroy SL, editors. *Bradley and Daroff's Neurology in Clinical Practice*. 8th ed. Philadelphia: Elsevier; 2022.
9. De Renzi E, Barbieri C. The incidence of the grasp reflex following hemispheric lesion and its relation to frontal damage. *Brain*. 1992;115(1):293-313. doi:10.1093/brain/115.1.293.
10. Hassan A, Josephs KA. Alien hand syndrome. *Curr Neurol Neurosci Rep*. 2016;16(8):73. doi:10.1007/s11910-016-0676-z.
11. Mark VW. Alien hand: current research trends. *Curr Neurol Neurosci Rep*. 2025;25(1):63. doi:10.1007/s11910-025-01449-z.
12. Josephs KA, Rossor MN. The alien limb. *Pract Neurol*. 2004;4(1):44-45. doi:10.1111/j.1474-7766.2004.06-189.x.
13. Santos RP, Fernandes RCL. Exame neurológico essencial baseado em evidências. *Braz J Dev*. 2021;7(12):117286-117301. doi:10.34117/bjdv7n12-471.
14. Peris-Subiza J, Vidal-Notari S, Cuadrado-Godia E, Ois A, Rodríguez-Campello A, Guisado-Alonso D. Teaching Video NeuroImage: the Digits Quinti sign as the sole objective sign of mild hemiparesis. *Neurology*. 2023;101(22):e2340. doi:10.1212/WNL.0000000000207906.
15. Alter M. The digits quinti sign of mild hemiparesis. *Neurology*. 1973;23(5):503-505. doi:10.1212/WNL.23.5.503.
16. Dolbow J, El-Azzouni S, Zhang Y, Geiger C. A practical guide to assessing functional motor weakness: a review of validated techniques. *J Neurol*. 2025;272(6):427. doi:10.1007/s00415-025-13139-4.
17. Wartenberg R. *The Examination of Reflexes*. Chicago: Year Book Publishers; 1945.
18. Brigo F, Ragnedda G, Canu P, Nardone R. Synkinetic wrist extension in distinguishing cortical hand from radial nerve palsy. *Pract Neurol*. 2018;18(6):520-521. doi:10.1136/practneurol-2018-001971.
19. Hallett M, Stone J, Carson A, editors. *Functional Neurologic Disorders*. *Handb Clin Neurol*. Vol. 139. Amsterdam: Elsevier; 2016.
20. Maranhão-Filho P. Babinski's twelve signs: history of neurology and neurosemiology. *Rev Bras Neurol*. 2010;46(4):29-34.
21. Datta AK, Chakraborty U, Chandra A. The thalamic hand: an enigmatic sequela of thalamic stroke. *QJM*. 2022;115(4):239-240. doi:10.1093/qjmed/hcac050.
22. Albanese A, Bhatia K, Bressman SB, DeLong MR, Fahn S, Fung VS, et al. Phenomenology and classification of dystonia: a consensus update. *Mov Disord*. 2013;28(7):863-873. doi:10.1002/mds.25475.
23. Vives-Rodriguez A, Louis ED. Index finger pointing (likely a subtle form of hand dystonia): prevalence across movement disorders. *Front Neurol*. 2018;9:542. doi:10.3389/fneur.2018.00542.
24. Ganguly J, Mukherjee S, Singh N, Kumar H. Pointing index posture in progressive supranuclear palsy and corticobasal syndrome. *Ann Mov Disord*. 2025;8(1):74-75. doi:10.4103/aomd.aomd_57_24.
25. Ho BK, Morgan JC, Sethi KD. Starfish hand. *Neurology*. 2007;69(1):115. doi:10.1212/01.WNL.0000267410.94742.D4.
26. Brogren E, Arner M, Tsuboi Y, Hariz GM, Hariz MI, Wiberg M, et al. Striatal hand deformities in Parkinson's disease: hand surgical perspectives. *Mov Disord Clin Pract*. 2022;9(8):1047-1054. doi:10.1002/mdc3.13531.
27. Maranhão-Filho P, Maranhão ET. Mão estriatal. *Rev Bras Neurol*. 2010;46(4):35.
28. Barclay CL, Lang AE. Dystonia in progressive supranuclear palsy. *J Neurol Neurosurg Psychiatry*. 1997;62(4):352-356. doi:10.1136/jnnp.62.4.352.
29. LeWitt PA. Hidden gems in the neurological literature of progressive supranuclear palsy. *J Parkinsons Dis*. 2024;14(4):639-642. doi:10.3233/JPD-230301.
30. Marsili L, Bologna M, Kojovic M, Berardelli A, Espay AJ, Colosimo C. Dystonia in atypical parkinsonian disorders. *Parkinsonism Relat Disord*. 2019;66:25-33. doi:10.1016/j.parkreldis.2019.07.030.
31. Campbell WW, Barohn RJ. DeJong's The Neurologic Examination. 8th ed. Philadelphia: Wolters Kluwer; 2020. p. 913-971.
32. Kompolti K, Metman LV, editors. *Encyclopedia of Movement Disorders*. London: Academic Press; 2010.
33. Zomorodi A, Wald ER. Sydenham's chorea in western Pennsylvania. *Pediatrics*. 2006;117(4):e675-e679. doi:10.1542/peds.2005-1573.
34. Catala M, Poirier J. Georges Marinesco (1863-1938): neurologist, neurohistologist and neuropathologist. *Rom J Morphol Embryol*. 2012;53(4):869-877.
35. Tarawneh R, Galvin JE. Neurologic signs in the elderly. In: Fillit HM, Rockwood K, Woodhouse K, editors. *Brocklehurst's Textbook of Geriatric Medicine and Gerontology*. 7th ed. Philadelphia: Saunders Elsevier; 2010. p. 101-105.
36. Lam L, Engstrom J. Teaching Video NeuroImages: the breathing arm: respiratory brachial synkinesis. *Neurology*. 2010;74(16):e69. doi:10.1212/WNL.0b013e3181d9ed1e.
37. Friedenberg SM, Hermann RC. The breathing hand: obstetric brachial plexopathy reinnervation from thoracic roots? *J Neurol Neurosurg Psychiatry*. 2004;75(1):158-160.
38. Brazis PW, Masdeu JC, Biller J. *Localization in Clinical Neurology*. 7th ed. Philadelphia: Wolters Kluwer; 2016.
39. Bauer B, Chaise F. Correction of ulnar claw hand and Wartenberg's sign. *Hand Surg Rehabil*. 2022;41(1 Suppl):S118-S127. doi:10.1016/j.hansur.2020.11.012.
40. Aldossari RM, Galecio Castillo MC, Hameed S. Benediction sign. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025.
41. Severo ALM, Lemos MM, Duarte MP, Tagliari I. Síndrome interóssea posterior devido a schwannoma: um relato de caso. *Rev Bras Ortop*. 2024;59(Suppl 1):e91-e93. doi:10.1055/s-0042-1749614.
42. Bevelacqua AC, Hayter CL, Feinberg JH, Rodeo SA. Posterior interosseous neuropathy: electrodiagnostic evaluation. *HSS J*. 2012;8(2):184-189. doi:10.1007/s11420-011-9238-8.
43. Van Schaik IN, van den Berg LH, de Haan R, Vermeulen M. Intravenous immunoglobulin for multifocal motor neuropathy. *Cochrane Database Syst Rev*. 2005;(2):CD004429. doi:10.1002/14651858.CD004429.pub2.
44. Seba MCC, Bernardo HNSA, Pereira MG, Vecina DFC, Andari AC, Santana ET, et al. Killoh-Nevin syndrome: a literature review. *J Bras Neurocirurg*. 2024;35(2):211. doi:10.22290/jbnc.2024.350211.
45. Rabaut V, Vander Vyer V, Verstraete K. Killoh-Nevin syndrome. *J Belg Soc Radiol*. 2020;104(1):49. doi:10.5334/jbsr.2183.
46. Voche P, Merle M. Wartenberg's sign. *J Hand Surg Br*. 1995;20(1):49-52. doi:10.1016/S0266-7681(05)80016-6.
47. Howard R, Kullmann D, Werring D, Zandi M. *Neurology: A Queen Square Textbook*. 3rd ed. Oxford: Wiley-Blackwell; 2024.
48. Samuels MA, Feske SK. *Office Practice of Neurology*. 2nd ed. Philadelphia: Elsevier; 2003.
49. Nicolau S, Liewluck T, Milone M. Myopathies with finger flexor weakness: not only inclusion-body myositis. *Muscle Nerve*. 2020;62(2):144-154. doi:10.1002/mus.26914.
50. Zoccollella S, Giugno A, Logroscino G. Split phenomena in amyotrophic lateral sclerosis: current evidences, pathogenetic hypotheses and diagnostic implications. *Front Neurosci*. 2023;16:1100040. doi:10.3389/fnins.2022.1100040.

51. Benny R, Shetty K. The split hand sign. *Ann Indian Acad Neurol.* 2012;15(3):175-176. doi:10.4103/0972-2327.99700.
52. Didona D, Solimani F, Caposiena Caro RD, Sequeira Santos AM, Hinterseher J, Kussini J, et al. Dermatomyositis: a comprehensive review of clinical manifestations, serological features, and therapeutic approaches. *Ital J Dermatol Venereol.* 2023;158(2):84-98. doi:10.23736/S2784-8671.23.07458-3.
53. van Bussel BC, Koopmans RP. Trousseau's sign at the emergency department. *BMJ Case Rep.* 2016;2016:bcr2016216270. doi:10.1136/bcr-2016-216270.