

Bedside Clinics in Pediatrics Ready Reckoner



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Foreword
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Examination of the Central and Peripheral Nervous System

Pradip Prava Paria

PART A: CENTRAL NERVOUS SYSTEM EXAMINATIONS: CLINICAL APPROACH

■ INTRODUCTION

Infants and young children are not simply smaller versions of adults, and as such, traditional neurological examination methods are often not suitable for children under five years of age. Evaluating neurological function in this age group requires creativity, flexibility, and a playful, unstructured approach to gain the child's cooperation and obtain meaningful results. Like other organ systems, the central nervous system (CNS) in children is constantly growing and maturing. At birth, the brain weighs approximately 70% of its adult size, while the body constitutes only about 5% of adult body weight. Most brain growth takes place during infancy and the early preschool years. Certain neurological disorders, such as developmental delays caused by hypoxia, congenital malformations, and metabolic disorders, are primarily seen in pediatric populations. Additionally, in children, neurological symptoms are more frequently linked to systemic illnesses rather than primary diseases of the CNS.

Just as in adults, the *neurological evaluation in infants and young children* requires a comprehensive history, a relevant physical examination, and well-chosen diagnostic tests. The primary aim is to establish the anatomical diagnosis by pinpointing the lesion's location and the pathological diagnosis by identifying the cause of dysfunction.

■ WHERE IS THE LESION?

While history provides key etiological insights, the neurological assessment and imaging pinpoint the precise anatomical location of the symptoms. The brain and spinal cord, together with the cerebellum, peripheral nerves, and muscles, form a bilaterally symmetrical system aligned along the vertical axis of the CNS. Examination components include mental status assessment for the cortex, cranial

nerve testing for the brainstem, coordination evaluation for the cerebellum, and sensory/motor assessment for the spinal cord, muscles, and peripheral nerves (**Fig. 1**).

■ WHAT IS THE LESION?

A comprehensive history encompassing present illness, past medical events, family background, and social context enables the clinician to map the progression of the child's condition. Neurological disorders typically exhibit characteristic temporal patterns: acute (within seconds to hours), subacute (over hours to days), chronic (evolving over weeks to months), or paroxysmal (episodic with recovery between episodes) (**Table 1**).

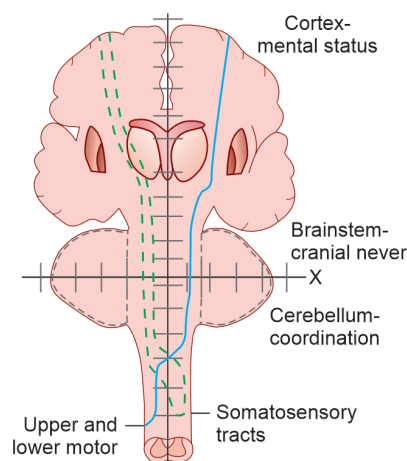


Fig. 1: Neuraxis of neurological localization.

TABLE 1: Temporal profile.

Acute	Subacute	Chronic	Paroxysmal
• Infarct • Hypoxia • Trauma	• Infections • Immune/inflammation • Toxic/metabolic	• Congenital • Degenerative • Neoplastic	• Seizure • Syncope • Pain/headache

AGE-APPROPRIATE NEUROLOGICAL ASSESSMENT IN CHILDREN

The foundation of pediatric neurological assessment lies in adult examination techniques, many of which can be applied to verbal children. However, rigidly enforcing these methods on infants and toddlers can lead to frustration. A flexible approach, considering the child's temperament and developmental level, allows for a more effective assessment, often through observing spontaneous behaviors.

Observe the Child for—

Any obvious dysmorphisms, facial asymmetry, the child's social interaction, presence of hyperactivity. Assess head control, posture, early hand preference, and presence of motor abnormalities. What sounds or words does he or she make?

Once the initial spontaneous observations are complete, the next phase of the examination begins. This is best performed with the infant in the parent's lap. To reduce anxiety and encourage cooperation in children, imaginative strategies, such as turning a reflex hammer into a toy animal, can be highly effective. Adding sound effects to it may make it a nonthreatening game when eliciting muscle stretch reflexes. Finger puppets or brightly colored toys may be offered to the child for playing, which maximizes the information for the clinician. The final stage of the assessment consists of tasks that might cause discomfort or anxiety in the child, such as removing clothing, examining the eyes and ears, evaluating the gag reflex, and checking head size.

RELEVANT HISTORY

A thorough history is essential for neurological assessment. Parents typically serve as the primary informants, but children older than 4 years can often share details about their condition and should be included in the discussion. The history must begin with the caregiver's main concern and be evaluated against developmental expectations for age (**Box 1**).

Next, the history of the present illness: A thorough and precise neurological history is essential, with symptoms reviewed in chronological order. Assessing the timeline and frequency of symptoms, along with their progression—whether advancing (neurodegenerative diseases), static, or recovering [cerebral palsy (CP)]—is crucial for diagnosis.

It is also important to determine whether the condition is acute, subacute, or chronic. Factors that alleviate or worsen symptoms, including medication effects, should be explored. Additionally, assessing how neurological symptoms interact with other organ systems can provide clues about the underlying condition, such as fever and vomiting in meningitis or urinary retention in spinal cord disorders.

Reviewing the *past medical history* is crucial as it may influence the current diagnosis. This includes a record of similar past illnesses, major health issues, surgical interventions, trauma, transfusions, and allergies, and prescribed medications (noting dosage and indication) with response to them.

This is followed by a complete *birth history*, with a review of the pregnancy, including the mother's age, immunization status, any complications during pregnancy [e.g., pregnancy-induced hypertension (PIH), autoimmune hepatitis (AIH), hydramnios, gestational diabetes, and febrile illnesses with rash], use of illicit drugs, cigarettes, or alcohol. Ask about whether the mother had regular antenatal check-ups, ultrasound examination,

BOX 1: Symptoms commonly associated with neurological disorders in children.

- Delayed development—single domain or multidomain, e.g., neurologic—cerebral palsy, non-neurologic—congenital hypothyroidism, and Down syndrome
- Regression of milestones, e.g., neurodegenerative disorder, muscular dystrophy, Rett's syndrome, and epileptic encephalopathy
- Weakness—hemiparesis, paraparesis, or quadriparesis
- Symptoms of raised intracranial tension—headache, vomiting, visual blurring, etc.
- Altered sensorium, e.g., encephalopathy
- Convulsion—motor, sensory, autonomic, provoked, or unprovoked
- Cranial nerve involvement—in the form of vision (II) or hearing impairment (VIII), diplopia, convergent/divergent squint (III, IV, and VI cranial nerves), deviation of the angle of the mouth (VII), dysphagia, drooling of saliva (IX), nasal intonation/ nasal regurgitation of food (XI), etc
- Symptoms of sensory disturbances—pain, paraesthesia, and complete loss of sensation in GBS and TM
- Autonomic disturbances—retention of urine/retention with overflow or fecal incontinence—in spinal disorders
- Loss of postural balance—in cerebellar disorders
- Involuntary movements, such as chorea, athetosis, dystonia, and tremors
- Headache
- Neuromuscular weaknesses

and iron and folic acid intake before or throughout conception. Inquiring about fetal movement, as decreased fetal activity can be associated with hypotonia due to CNS or neuromuscular disorders. The mother's labor history should address, as that was a term or preterm delivery, the place (home or institutional) and mode (normal vaginal vs assisted) of delivery. Birth weight and any complication, e.g., birth asphyxia (Apgar score), should be noted. The baby's consciousness level and history of neonatal issues like seizures or jaundice must be noted. For jaundice, clarify how soon after birth it began and what treatment was employed, typically phototherapy or exchange transfusion. A reliable account of the postnatal period is usually obtainable from the parents, complemented by a review of hospital documentation for any complications. Key areas of inquiry include the infant's general health, feeding and sleeping habits, level of activity, and the nature of the cry.

Evaluating *developmental milestones* (**Table 2**) is a fundamental aspect of neurological history-taking. It involves assessing multiple areas—social, cognitive, linguistic, and motor skills—to identify delays that are isolated or widespread. Any sign of skill regression warrants investigation for possible neurodegenerative or neurometabolic disease.

If parents have difficulty remembering milestone achievements, especially for second or third children, comparing them with the developmental timelines of siblings can be informative. In school-aged children, academic progress often reflects cognitive development, and abrupt changes in performance may signal a neurological problem.

Immunization history is vital as it protects against vaccine-preventable diseases in children. If parents could or cannot remember the vaccine given to the child, it is better to check the immunization card whether there are any missing doses.

Nutritional history plays a significant role in pediatric evaluation, especially in developing countries. Important factors include the mode of feeding (breastfeeding or formula), the introduction of solid foods, and weaning practices. It's better to go for calorie calculation in preillness and during the sickness period. Any form of picky eating (nutritional deficiency) and history of pica (lead poisoning) should be asked for.

Next, the *family history* must be reviewed. A comprehensive family history must include the age at which any neurologic disorders, like developmental delays, seizures, migraines, strokes, or genetic conditions, were diagnosed in close family members. Three generation pedigree charts should be drawn. History should include regarding consanguinity (the chance of metabolic and degenerative diseases is higher in consanguineous marriage) and ethnic background. It is necessary to obtain clear information about miscarriages or medical termination of pregnancy with specific indication, and also document the sex and gestational age of the relevant embryo or fetus.

A comprehensive *social history* should be obtained, particularly in resource-limited settings. This includes details about living arrangements, crowding, parental occupation and education, and contact with domestic or farm animals. Travel history is especially relevant in urban populations to assess the risk of zoonotic and parasitic

TABLE 2: Key developmental milestones.

Age (mo)	Gross motor	Fine motor and vision	Social	Language
3 months	Neck holding	Opens hand spontaneously	Recognizes mother	Coos
6 months	Sits with support	Transfer objects	Stranger anxiety	Monosyllables
9 months	Stands with support	An immature prince grasps	Waves bye-bye	Bisyllables
12 months	Stands without support	Releasing grasp in response to command	Responds to name being called	One to two meaningful words
15 months	Walks alone	A tower of 2 blocks	Jargon	
18 months	Runs	Feeds self from a spoon	Copies parents in the task	8–10-word vocabulary
24 months	Walks up and downstairs	Build a six-block tower	Engages in playing with peers	Two-to-three-word simple sentences

diseases. Additionally, recent stressors, such as family disruptions, sibling birth, and loss of a loved one, should be explored, as they may impact a child's emotional well-being. School and daycare records should also be reviewed to identify any significant changes in academic or social behavior.

■ GENERAL PHYSICAL EXAMINATION

General appearance, nature of disability, facies, decubitus, build, and nutrition (anthropometry) should be ascertained first, followed by vital signs. Record temperature, pulse, respiration, and blood pressure. Increased temperature and tachycardia may suggest possible infection, including meningitis or encephalitis. Bradycardia, increased blood pressure, and abnormal respiratory pattern may be features of increased intracranial pressure (Cushing response). Tachycardia, along with hypotension, is suggestive of septic shock, as in cases of meningococemia. Conversely, hypertension may be the cause of encephalopathy or a response to elevated intracranial pressure. An abnormal respiratory pattern may point toward different types of brain herniation.

Look for pallor (leukemia, lymphoma with CNS involvement, and intracranial bleed), cyanosis/clubbing (cyanotic heart disease with cerebral thrombosis or abscess), jaundice (hepatic encephalopathy), and edema.

Skin rash and petechiae in a child with altered sensorium may indicate infection with meningococcus, rickettsia, dengue, etc. Enlarged neck glands may indicate tuberculosis or malignancy with CNS involvement.

■ HEAD-TO-TOE EXAMINATIONS IN RELATION TO CENTRAL NERVOUS SYSTEM

- **Face:** Dysmorphic features—in Down syndrome or some chromosomal and metabolic disorders (mucopolysaccharidosis, GM1 gangliosidosis, and mucopolipidosis).
- **Eyes:** Strabismus, hyper- or hypotelorism, cataract (galactosemia), gaze abnormality, Kayser-Fleischer ring (Wilson's disease), telangiectasia (ataxia telangiectasia)
- **Ears:** Position of the ears (low-set ear in many syndromic children) and any ear tags or skin lesions.
- **Mouth:** Any deviation of the angle of the mouth, drooling, gum hypertrophy, or atrophy of the tongue.
- **Neck:** Check for the presence of neck tilt, webbed neck, implanted shunt, or any scars.

- **Back:** Signs suggestive of occult spinal dysraphism, like midline sacral dimples or localized hair tufts, kyphosis, and gibbus.
- **Skin/neurocutaneous lesions (Table 3):** Hair (kinky-Menkes disease).

Observe for abnormal postures such as hemiplegic limb positioning (**Fig. 2**), scissoring suggestive of cerebral palsy, and the frog-like posture typical of hypotonia. Identify contractures if present. For ambulant children, gait assessment is essential—look for signs like spastic gait, hemiparetic stride, stepage pattern from sensory deficits, or waddling due to myopathy. Check for hand dominance. Although most children establish handedness by 2 years, a strong preference before this age may be an early sign of asymmetrical motor function or limb weakness.

TABLE 3: Neurocutaneous marker.

Ash-leaf hypopigmented spots, facial angiofibroma, and shagreen patches	Tuberous sclerosis.
Café-au-lait macules along with axillary and groin freckling	Neurofibromatosis type I
Port-wine stain/Nevus flammeus	Sturge-Weber syndrome
Raised verrucous skin lesions in ovoid or linear plaques	Epidermal nevus syndrome



Fig. 2: Hemiplegic posture—arm-adducted and internally rotated at the shoulder; elbow is flexed; forearm semi-pronated, and the wrist and fingers are flexed. Lower limb—slightly flexed at the hip, extended at the knee with plantar-flexed and inverted foot.

■ OTHER SYSTEM EXAMINATIONS

Abdominal examination should include assessment for hepatosplenomegaly, which can be linked to neurometabolic conditions. A distended bladder, evidence of constipation, or undescended testes may point toward underlying neural tube anomalies. Respiratory functions are mostly compromised in chronic progressive neuromuscular disorders.

■ NEUROLOGICAL EXAMINATIONS

The neurological examination begins as soon as the interview starts. Observing the child's appearance and movements can provide important clues to underlying conditions. For instance, dysmorphic facial features, abnormal posture, or motor impairments such as hemiparesis and gait disturbances may be noticeable. The exam should take place in a relaxed, child-friendly environment, allowing the child to sit where they feel most at ease—whether on a parent's lap or the floor. The physician should approach gently, saving any invasive. More invasive assessments, such as measuring head circumference or testing the gag reflex, should be saved for the end of the examination. Observing the child's behavior during play and interactions with parents can also provide valuable insights. Typically, a child will play independently at the beginning of the visit but gradually engage in the interview. In contrast, a child with attention-deficit/hyperactivity disorder (ADHD) may display impulsiveness, while a child with autism may seem completely unaware of their surroundings (**Box 2**).

BOX 2: Key points in neurological examination.

- Mental status—(appearance, behavior, communication, delusion, hallucination, and illusion) and Higher mental function—consciousness, orientation, memory, attention, intelligence, and speech
- Cranial nerve examinations
- Motor system examination—muscle mass, tone, power, deep tendon reflexes and superficial reflexes, gait, and involuntary movements
- Sensory examination—lateral, spinothalamic tract mediating—pain, temperature posterior spinothalamic tract mediating—fine touch, vibration, joint position, cortical sensation (two-point discrimination, and tactile localization)
- Cerebellar functions
- Meningeal irritation signs—neck rigidity, Kerning sign, and Brudzinski sign
- Skull and spinal column

■ ESSENTIAL TOOLS

Instruments necessary for pediatric neurologic evaluation include colorful cubes, a reflex hammer, a bell, a rubber-adapted penlight, fiberglass tape, items for stereognosis assessment, tuning forks (128 Hz for sensation, 256 Hz for auditory testing), a two-point discrimination device, developmental kits, and an ophthalmoscope.

■ CONSCIOUSNESS AND HIGHER MENTAL FUNCTION

Scheme of examinations:

- Level of consciousness
- Higher mental functions

Appearance and Behavior

- Delusion/hallucination/illusion
- Intelligence
 - Attention/working memory
 - Language/reading/writing /visuospatial
 - ♦ Mood and affect
 - ♦ Orientation to place, person, and time
 - ♦ Speech
 - ♦ Sleep

The examination of selective elements will be discussed here.

The level of consciousness (LOC) refers to a person's awareness of themselves, their environment, and their ability to respond to stimuli. It is classified into two distinct stages—arousal and awareness. Arousal is a function of the reticular activating system (described as whether the sleep-wake cycle is preserved or not). And, awareness is a function of cortical or subcortical structures (expressed in interest in their toys, consolability, eye contact, etc.). Deranged consciousness is thus an impairment in arousal and awareness. Infants under 28 weeks of gestation often do not display predictable alert periods, whereas gentle physical contact can rouse older infants from sleep. In older children, it is particularly important in neurological, medical, and psychiatric evaluations. The levels range from full awareness to a complete lack of responsiveness and are typically described as follows:

- Fully conscious, a person is awake, alert, and oriented to time, place, and person. Normal responses to verbal and environmental stimuli.
- *Clouding of consciousness*: Mildly reduced awareness of the environment. The individual may be confused, distracted, or slow to respond.

- **Drowsiness/lethargy:** There is impaired attention and reduced consciousness; the child responds to verbal or tactile stimulation but drifts back to sleep once the stimulation stops.
- **Obtundation:** More severe reduction in alertness. It is a stage between drowsiness and stupor. The person requires loud verbal or physical stimuli other than pain to respond and may appear confused. Responses are slower and less purposeful.
- **Stupor:** The child responds only to persistent and strong painful stimulation. It is a cerebral response. The child can localize pain and tries to remove the noxious stimuli. Soon after the removal of stimuli, the child slips to the previous condition.
- **Coma:** No response is elicited from the child even with strong and repeated painful stimuli.
- **Delirium:** The child is confused and disoriented, unable to form coherent thoughts, and may be irritable, fearful, agitated, or experiencing hallucinations.

Grading of Consciousness

Glasgow Coma Scale (Table 4)

A numerical scoring system is often used to quantify the LOC in clinical settings.

- **Components:** Eye opening (E)—spontaneous to none (1–4).
- **Verbal response (V):** Oriented to no verbal response (1–5).
- **Motor response (M):** Obeys commands to no motor response (1–6).

TABLE 4: Glasgow Coma Scale.

Score	Eye-opening (E)	Verbal response (V)	Motor response (M)
1	None	No response	No response
2	To pain	Incomprehensible sound	Abnormal extension
3	To speech	Inappropriate words	Abnormal flexion
4	Spontaneous	Confused	Withdrawal from pain
5		Oriented	Localizes to pain
6			Obeys commands

Total score: 3 (deep coma) to 15 (fully alert).

An easy and efficient tool for assessing consciousness is the AVPU scale, often used in initial evaluations where: A stand for alertness, V for response to voice, P for response to pain and U for unresponsive to any stimulus.

Vegetative State and Minimally Conscious State

- **Vegetative state:** Awake but not aware. Retains sleep-wake cycles but lacks purposeful responses to stimuli. For example, a persistent vegetative state (PVS) following severe brain injury.
- **Minimally conscious state:** Severely altered consciousness but with occasional purposeful responses (e.g., following simple commands). For example, the recovery phase from a coma.

HIGHER MENTAL FUNCTION

Higher mental functions refer to the advanced cognitive abilities that distinguish humans from other species. The mental status examination evaluates the function of the cerebral cortex, the highest level of the neuroaxis responsible for integrating perception, cognition, and emotion, ultimately shaping thoughts, feelings, and behavior. In children, the cortex undergoes significant age-dependent development. At the time of birth, most of an infant's actions are controlled by reflexive responses. Mental status assessment should be adapted based on the child's developmental stage.

Appearance and Behavior

As you talk with the parents, casually observe the child for indicators such as cleanliness, responsiveness to the environment, facial affect, sustained eye contact, and overall activity.

Delusions and Hallucinations, Illusion

- **Delusion** is a firmly held mistaken belief that persists even when faced with clear evidence against it.
- **Hallucination** is a false sensory perception that occurs without an external stimulus, originating from the organ of special senses, and is commonly associated with psychiatric, neurological, and substance-related disorders.
- **Illusion** is the mistaken interpretation of what one sees, as if one mistakes a rope for a snake.

Orientation

For older children, orientation can be evaluated by asking them to state the current time, identify their location, and recognize familiar individuals.

Attention

Children with inattention often find it hard to concentrate on tasks requiring focus. Attention can be evaluated by asking the child to recall a sequence of numbers, beginning with two and gradually increasing in length, or to repeat a short list of items until mistakes occur. If a child struggles to repeat six or more numbers correctly, it may indicate attention difficulties. Another method is to ask the child to spell a word in reverse, though this may be influenced by memory problems or learning challenges.

Speech and Language Function

Assess the child's natural speech for clarity of articulation, speech flow, tempo, tone, and volume, while noting any atypical language patterns like made-up words.

Dysphasia refers to an impairment in language abilities, affecting either speech production, comprehension, or both. It usually occurs due to a lesion in the dominant cerebral hemisphere (mostly on the left side). Normally, sounds are recognized as language in the sensory (Wernicke's) area, which is connected to the motor (Broca's) area by the arcuate fasciculus (**Fig. 3**). The following patterns of aphasia can be recognized (**Table 5**).

Dysphasia Needs to be Distinguished From

Impairment of voice—dysphonia—may result from vagus nerve palsy or lesions affecting the larynx, altering voice pitch, tone, or volume. Dysarthria, however, is due to poor articulation control, often related to muscle disorders or cranial nerve damage (VII, X, and XII). Evaluating articulation can be done using the “ka, pa, ta” test: ‘Ka’ comes from the throat, ‘pa’ from the lips, and ‘ta’ from the tongue-palate region.

Disorders of Speech

- Stammering/fluency disorder or infantile speech results from psychological factors rather than neurological causes.
- Scanning or staccato speech, characterized by slow, slurred syllables resembling intoxication, is associated with cerebellar disorders.
- Spastic dysarthria, marked by strained or slow speech, is seen in many cases of cerebral palsy.
- Bulbar dysarthria due to bulbar palsy is marked by slurred speech, difficulty swallowing with nasal regurgitation, and pooling of secretions in the throat.
- Nasal speech is typically associated with palatal weakness or congenital anomalies like cleft palate.

Memory

From a clinical perspective, memory is categorized into immediate, recent, and remote types. It can be assessed in children over the age of 5 years. Informal evaluation includes observing how a patient recalls personal details, while formal testing involves asking them to register and recall three objects or perform a digit span test. Additionally, assessing cognitive functions can involve activities such as storytelling, drawing, or solving puzzles.

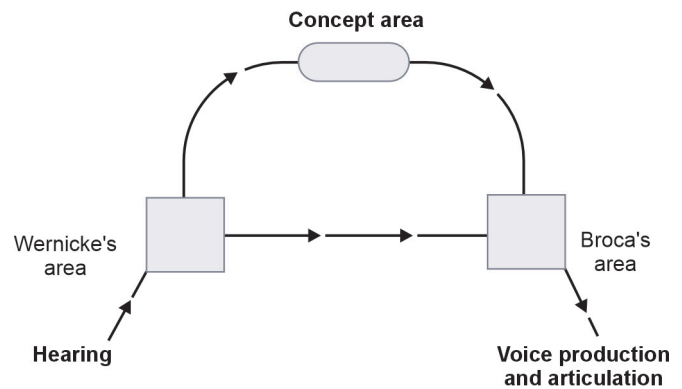


Fig. 3: Speech understanding and output.

TABLE 5: Pattern of aphasia.

Aphasia	Lesion	Characteristics
Wernicke's aphasia	Superior temporal gyrus	Fluent but nonsensical speech, poor comprehension, unaware of deficits
Broca's aphasia	Inferior frontal gyrus	Effortful speech, difficulty with grammar, and good comprehension
Conduction aphasia	Arcuate fasciculus	Fluent speech, preserved comprehension, and impaired repetition
Transcortical sensory	Temporoparietal junction outside Wernicke's area	As in Wernicke's aphasia, with preserved repetition
Transcortical moto	Frontal lobe outside Broca's area	As in Broca's aphasia, with preserved repetition

Immediate memory refers to the ability to recall information after just a few seconds. For example, a child can be asked to repeat a series of digits shortly after hearing them.

Recent memory involves recalling events from the same day, such as answering the question, “*What did you eat for breakfast today?*” It requires retrieving information after a few hours to days.

Remote memory pertains to the recollection of facts or events from months or years ago. This can be assessed by asking questions like, “*What is your teacher’s name?*” or “*Which school do you attend?*”

- **Intelligence:** Best assessed by an IQ test. The history of schooling of the child, if age-appropriate, indicates evidence of optimum intelligence. At bedside, interpretation or comprehension of power, problem-solving power can be tested, depending on the child’s age.
- **Language:** Age-appropriate language skills—identifying objects, understanding word meanings, and following instructions.
- Reading and writing.
- **Visuospatial:** Copying shapes, illustrating a human figure, and creating a clock drawing.

■ CRANIAL NERVES

There are 12 pairs of cranial nerves. Of all cranial nerves, I, II, and VIII are pure sensory, III, IV, VI, XI, and XII are purely motor, and the rest of the nerves are mixed in character. The lower part of the face, XI, XII, has cortical control from the opposite hemisphere only. All the other cranial nerves have bilateral cortical control. All the cranial nerves should be examined. However, in infants and children up to 3 years it may not be possible to test all of them.

Olfactory Nerve (Cranial Nerve I)

The olfactory nerves control the sense of smell and are usually tested only when indicated. Ask the child if they can detect odors normally or if they have noticed any changes. Ensure nasal passages are clear. To assess smell, have the child close their eyes and block one nostril at a time while identifying common household scents like chocolate, peppermint, or orange peel (**Fig. 4**). Avoid strong substances such as ammonia, which can trigger the trigeminal nerve. Smelling perception develops as early as the 32nd week of gestation in newborns.



Fig. 4: Cranial nerve I.

Abnormalities: Anosmia, or loss of smell, are often due to nasal congestion, head trauma, meningitis, frontal lobe tumors, or congenital conditions, such as Kallmann syndrome. Distorted smell perception, or parosmia, occurs when pleasant odors are perceived as unpleasant.

Optic Nerve (Cranial Nerve II)

These are the nerves of sight.

Assessment—acuity of vision, field of vision, color vision, and fundus.

- Acuity of vision—at birth—3/60 and by 2 years 6/6.
- **Below 3 years:** There is no standardized protocol for assessing visual acuity in children under 3 years of age. For younger verbal children, the examiner may ask them to identify small objects. In preverbal infants, visual fixation and tracking can be observed using a colored object. Various tools, such as Cardiff acuity cards (7 months to 3 years), tumbling E (3–5 years), or an animal picture Snellen chart, can aid in assessment.
- **Beyond 3 years:** Snellen’s chart (placed 6 meters away) is used for distance vision testing, while Jaeger’s chart (held 30 cm from the patient) evaluates near vision. Each eye should be tested separately by covering the other. The child’s visual acuity is recorded based on the smallest line they can read (e.g., 6/6). If vision is severely impaired, finger counting at 36 cm (14 inches)

is attempted, followed by checking for movement of the fingers, perception, and projection of light, if needed. For an uncooperative child, menace reflex (defensive eye blinking reaction when moving the examiner’s fingers suddenly and threateningly toward the patient’s eye) may be an option to test visual acuity.

Blindness occurring without an underlying eye disease is called amaurosis, whereas vision impairment resulting from an organic cause, such as cataract, strabismus, or anisometropia, is referred to as amblyopia.

- **Visual fields:** The normal visual pathway is given in **Figure 5**, with the field defect.

To evaluate visual fields, confrontation perimetry is used. Ideally, each eye should be tested individually, but for basic screening, testing both eyes together is acceptable. Position yourself about one meter from the child, making sure your eyes are aligned with theirs. Explain the procedure clearly. Ask the child to cover one eye and focus on your opposite eye. Close your eye corresponding to the child’s covered eye. Slowly bring an object from outside their field of vision and have them indicate when they see it. Repeat the process for the other eye. To check for sensory neglect, test both eyes simultaneously (**Fig. 6**).

To check visual fields in younger children, maintain a toy at the center of vision and slowly move a second, more engaging toy from behind into their peripheral sight. Observe if and when the child shifts their gaze toward the second toy.



Fig. 5: Examination of the visual field.

Localization of Field Defects

A visual field defect is called homonymous when the identical portion of the visual field is lost in both eyes. For instance, right homonymous hemianopia involves vision loss in the right eye’s temporal field and the left eye’s nasal field. Conversely, bitemporal hemianopia is a heteronymous defect, affecting the outer (temporal) visual fields of both eyes. When the visual field loss in both eyes matches exactly, the defect is described as congruous (**Table 6**).

- **Color vision:** Ishihara’s chart is used for formal testing. For clinical situations, one may give colored objects and ask the child to name the colors. A 3–4-year-old child will be able to say three colors. Red-green deficiency is the *most common* congenital color blindness. Color testing should be performed in children with acute or chronic vision loss, including optic nerve injury.

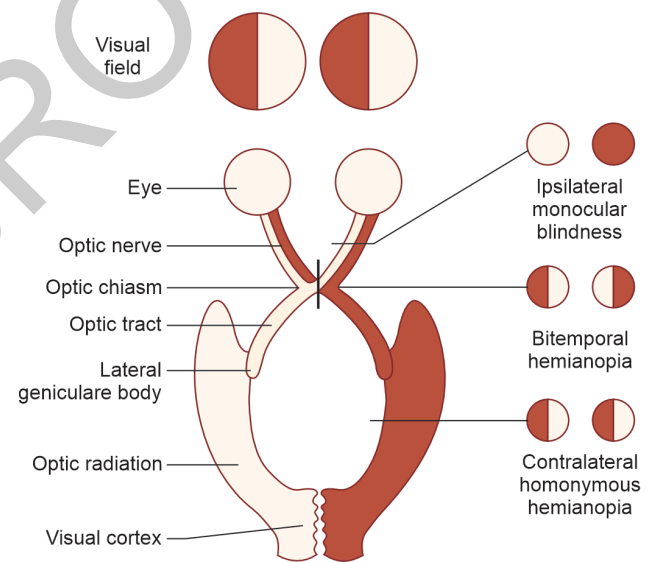


Fig. 6: Lesion in the visual pathway and corresponding visual field defects.

TABLE 6: Field defects with their localization.	
Field defect	Localization
Monocular	Anterior to optic chiasma
Bitemporal	Lesion in the optic chiasma
Homonymous field defect	Lesion behind the optic chiasm
Incongruous homonymous	Lesion in the optic tract
Congruous homonymous	Lesion behind the lateral geniculate body

- **Fundus:** If necessary (such as when the red reflex is absent), perform a fundoscopic exam at the end of the evaluation. To ease retinal inspection in infants, using a pacifier or gently turning the child's head to one side can be helpful. In children, the optic disc usually has a salmon-pink hue.

Fundoscopy

Dim the room lights by pulling the curtains. Explain to the child that you will be examining their eyes.

Ask the child to fix their gaze on a distant object. Hold the ophthalmoscope in your right hand and approach the child's right eye from the right side, using your right eye to examine. Start the exam about 20 cm away, focusing on the red reflex. An absent or white reflex could suggest serious issues such as cataract, retinoblastoma, and retinal detachment.

Approach the child's head and adjust the ophthalmoscope lens to bring the retina into clear focus. Once the optic disc is visible, evaluate the sharpness of its edges, color, arterial flow, venous pulsations, macula, and surrounding retina. Check for signs of abnormalities such as optic atrophy, papilledema, chorioretinitis, or a cherry-red spot. Then, examine the left eye by holding the ophthalmoscope in your left hand and using your left eye. Although mydriatic drops are usually not needed in older children, they may be necessary for infants.

Abnormalities: Swelling of the optic disc is known as disc edema, and when caused by increased intracranial pressure, it is called papilledema (**Figs. 7A and B**). Signs to look for are loss of venous pulsation, absence of the physiological cup, engorgement of veins, redness of the

disc, and flame hemorrhages. An atrophic disc appears pale yellow to white.

Examining the surrounding retina can help identify various childhood conditions, including congenital infections such as cytomegalovirus and toxoplasmosis, acquired infections such as miliary tuberculosis, and retinal hemorrhages. Retinal hemorrhages are observed in 30–40% of full-term newborns and typically resolve within 1–2 weeks. However, their persistence beyond the neonatal period should raise suspicion for nonaccidental trauma. Other conditions that may be detected include retinal dystrophies (e.g., rod/cone dystrophies) and neurocutaneous syndromes, such as tuberous sclerosis.

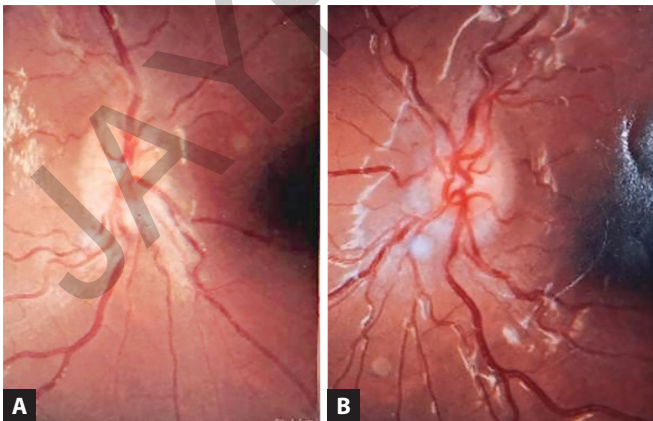
Evaluation of Vision In Newborns

- At 28 weeks of gestational age, the infant blinks to a bright light.
- At 32 weeks, the infant maintains eye closure until the light source is removed.
- At 29–32 weeks, the pupil reacts to bright light.
- At 37 weeks, the infant turns the head toward a soft light.
- At term—able to fix on and follow a target.

Oculomotor Nerve (Cranial Nerve III), Trochlear Nerve (Cranial Nerve IV), and Abducens Nerve (Cranial Nerve VI)

Nerves – to test for ptosis, nystagmus, strabismus, movement of the eyeball, and light reflexes.

These cranial nerves coordinate eye movements and are evaluated together. The oculomotor nerve (CN III) controls eye adduction (medial rectus), elevation (superior rectus and inferior oblique), depression (inferior rectus), and eyelid elevation (levator palpebrae superioris). It also carries parasympathetic fibers for pupil constriction. The abducens nerve (CN VI) innervates the lateral rectus muscle, allowing for eye abduction, while the trochlear nerve (CN IV) controls the superior oblique muscle, which facilitates downward and inward movement toward the midline (**Fig. 8**). The initial step in examining these cranial nerves involves watching the child's eyes when looking forward. Both eyes should be aligned, the corneal light reflexes should be symmetrical, pupils equal in size, and the eyelid openings (palpebral fissures) should be the same width.



Figs. 7A and B: Papilledema.

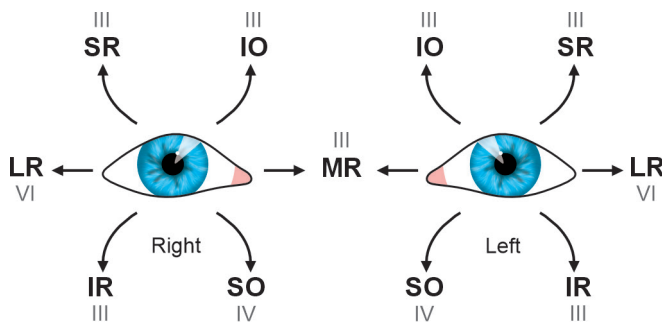


Fig. 8: Function of the extraocular muscles. (MR: medial rectus; LR: lateral rectus; SR: superior rectus; IR: inferior rectus; SO: superior oblique; IO: inferior oblique)



Fig. 9: Examination of movements of the eyeball.

MOVEMENT OF THE EYEBALL

The six extraocular muscles, controlled by cranial nerves III, IV, and VI, coordinate eye movements. To assess these movements in the four cardinal directions, follow these steps: Move your finger or an engaging object across the child's visual field.

Ask the child to track the movement using only their eyes, ensuring they do not move their head (you may need to stabilize it). Avoid extreme lateral gaze, as normal children may exhibit nystagmus. Observe whether eye movements are conjugated. Ask the child if they perceive double vision at any point. When the eye is abducted, the superior and inferior recti function purely as elevators and depressors, while the oblique muscles perform these functions in an adducted position (**Fig. 9**).

In premature infants beyond 25 weeks of gestation and in comatose patients, eye movement can be assessed

Positive oculocephalic reflex

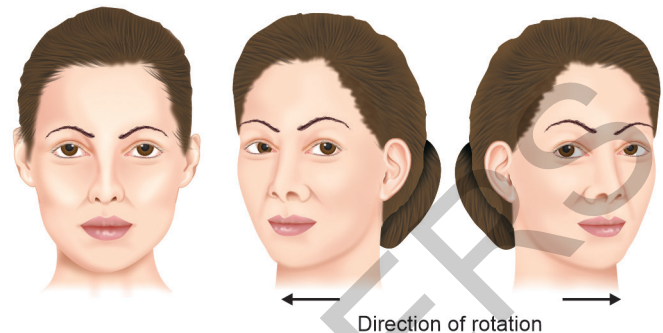


Fig. 10: Oculocephalic reflex.

using the oculocephalic (doll's eye) reflex. This is tested by rapidly rotating the patient's head horizontally or vertically, observing for reflexive eye movement. Normally, the baby's eyes lag behind, deviating in the opposite direction of the head movement, similar to a doll's eyes. In an awake, alert person, this reflex is absent as voluntary eye movements suppress it. A missing doll's eye response in a comatose child suggests brainstem dysfunction (**Fig. 10**).

Effects of Paralysis

In oculomotor nerve (CN3) palsy, the affected eye is positioned downward and outward, with restricted movement in adduction, elevation, and depression. Compression of CN3 leads to ipsilateral ptosis and pupil dilation, whereas ischemic CN3 palsy often spares the pupil and causes mild ptosis.

A trochlear nerve (CN4) palsy results in an upward and sometimes outward deviation of the affected eye. Diplopia is most pronounced when looking downward and toward the midline. To compensate, the child tilts their head to the opposite shoulder.

With an abducens nerve (CN6) palsy, the affected eye has a medial deviation and cannot fully abduct. To reduce double vision, the child turns their head toward the affected side.

Diplopia often results from lesions affecting cranial nerves III, IV, or VI. Misalignment of the eyes causes the image to strike different retinal areas, which the brain interprets as separate images.

Supranuclear palsy—lesions of the cortex or midbrain. Cause gaze palsy (paralysis of movement of both eyes in one direction or the other), without paralysis of individual ocular muscles. There is no diplopia. Thus, the presence of diplopia localizes the lesion to the nucleus or infranuclear location [lower motor neuron (LMN) lesion].

Conjugate deviation—means both eyes gaze toward one direction, to the right or left, down or up. A cortical irritative foci (in epilepsy) causes conjugate deviation of the eye to the opposite side, and a cortical destructive lesion (in stroke) causes conjugate deviation of the eyes to the same side. The exact reverse occurs in a pontine lesion.

Internuclear ophthalmoplegia (INO) results from a lesion in the medial longitudinal fasciculus (MLF) of the brainstem, which plays a crucial role in coordinating conjugated eye movements by linking CN VI on one side to CN III on the other. INO is characterized by impaired adduction of the affected eye (medial rectus weakness) and nystagmus in the abducting eye. Ocular motility testing helps distinguish INO from extraocular ophthalmoplegia. Extraocular ophthalmoplegia presents with paralysis of all extraocular muscles, ptosis, and normal pupillary function, whereas INO specifically involves impaired adduction with nystagmus in the contralateral abducting eye (**Fig. 11**).

Strabismus

Strabismus is a misalignment of the visual axes. If the corneal light reflex is asymmetrical, strabismus should be suspected. It can be classified as paralytic or nonparalytic. Paralytic strabismus results from cranial nerve palsies or brainstem lesions affecting the medial longitudinal fasciculus, whereas nonparalytic strabismus occurs due to eye misalignment with normal extraocular muscle function, often linked to genetic factors, hyperopia, or intraocular abnormalities. In infants younger than two

months, a slight resting eye misalignment is normal. When diplopia is present, two key principles help determine the underlying pathology: (1) Diplopia is most pronounced when looking in the direction of the affected muscle's pull, and (2) the displaced peripheral image comes from the affected eye.

To differentiate between paralytic and nonparalytic strabismus, both versions (testing eye movements in six cardinal directions with both eyes open) and ductions (testing each eye separately with the other covered) should be assessed. In paralytic strabismus, such as cranial nerve palsies, abnormal eye movement is evident in both versions and ductions. In nonparalytic strabismus, such as congenital strabismus and misalignment is observed in versions, but a full range of motion is preserved in ductions.

If ductions are restricted, a mechanical cause, such as thyroid eye disease, trauma, and congenital fibrosis, should be considered. Nonparalytic strabismus in children is rarely due to neurological conditions but can lead to amblyopia, which, if untreated, may cause long-term vision impairment.

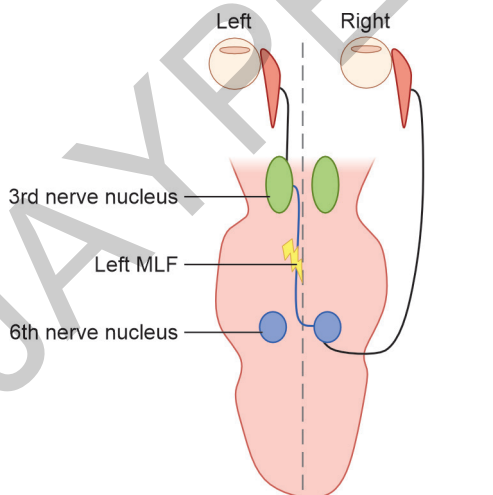
The cover-uncover test can help diagnose latent strabismus. Instruct the child to focus on your right eye. Cover the left eye, then uncover it while covering the right eye. If the uncovered eye readjusts to refixate, latent strabismus is present, which can be classified as convergent (inward deviation) or divergent (outward deviation) (**Fig. 12**).

Convergent squint, often due to lateral rectus paralysis (CN VI palsy), is normal in infants during the first few months of life. Divergent squint is usually associated with third nerve palsy (CN III).

When eye movement abnormalities are subtle, the red glass test can assist in identifying the affected muscle. A red glass is placed over one eye, and the patient is asked to follow a white light in various directions of gaze. In areas where eye movement is normal, the patient perceives a single red-white light. However, in the direction of the affected muscle's action, the images separate, with the greatest displacement occurring along the plane of the weakened muscle.

Ptosis

If an eyelid is lower than normal, this is referred to as ptosis. It can be partial or complete. It may be congenital, traumatic, myogenic (myasthenia gravis and myopathy), or neurogenic (III nerve palsy and Horner's syndrome).



Lesion in the left medial longitudinal fasciculus results in a left adduction deficit

Fig. 11: Internuclear ophthalmoplegia.

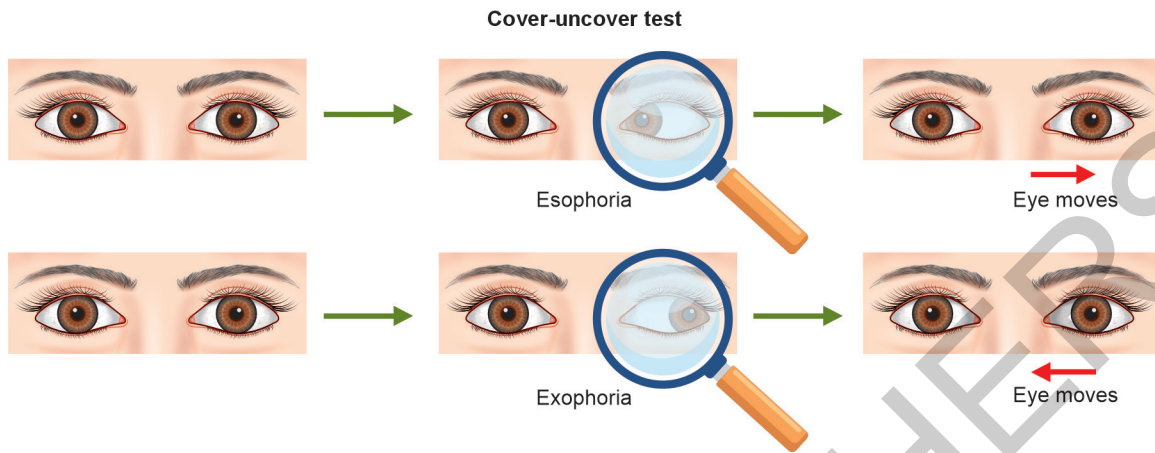


Fig. 12: Cover—uncover test.

Pupillary reflex

Look at the pupils and see if they are equal in size. Pupillary size is controlled by the sphincter pupillae, supplied by the III nerve, and the dilator pupillae, which is supplied by the cervical sympathetics. A constricted pupil is seen in morphine poisoning, organophosphorus exposure, pontine hemorrhage, Horner's syndrome, etc. On the other hand, dilated pupils are seen in optic nerve paralysis, third nerve paralysis, diphtheria, etc., but the most common cause is mydriatic eye drops.

To delineate the pupillary reflex, using a torch, shine the light into one eye to look for constriction (direct reflex). Afferents pass via the second nerve to the lateral geniculate body, and efferents pass from the Edinger-Westphal nucleus through the third nerve to the ciliary ganglion and subsequently to the pupil. Then shine the light in the same eye but look for constriction in the contralateral pupil (consensual reflex) (Fig. 13).

A difference in pupil size combined with an absent direct pupillary response suggests a lesion of the oculomotor nerve. Conversely, if the pupils are equal in size but one pupil shows reduced constriction to light, this indicates possible optic nerve dysfunction, causing diminished light perception. This condition, known as an afferent pupillary defect, is most reliably detected using the swinging flashlight test. During this test, a light moves slowly between the eyes. A pupil with an afferent defect will paradoxically dilate when the light is directed at the affected eye, indicating impaired optic nerve function (Fig. 14).

A fixed and dilated pupil is often seen in optic atrophy, while normal pupillary reactions are preserved in cases of cortical blindness.

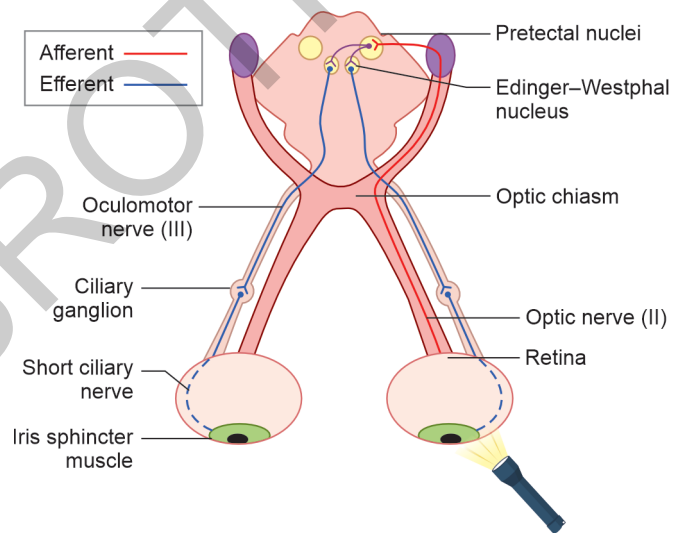


Fig. 13: Pathway of light reflex.

The convergence (accommodation reflex) is performed by asking the child to focus on an object in the midline, which is gradually brought closer to the eyes. This is accompanied by convergence of the eyes and pupillary constriction. The afferent component arises in the frontal lobe, and the efferent from the parasympathetic component of the III nerve.

Nystagmus

It is an involuntary, rapid eye movement that can be assessed by asking the child to follow the examiner's finger as it moves slowly in horizontal and vertical directions. It is classified as either pendular or jerky. Jerky nystagmus is further characterized based on the direction of its fast phase, which may be horizontal, vertical, rotatory, or mixed. A few beats of nystagmus at extreme lateral gaze

(end-gaze nystagmus) are common and usually not concerning. However, while horizontal nystagmus can be congenital or drug-related and often involves peripheral vestibular issues, vertical nystagmus is typically a red flag for structural disturbances in central areas like the brainstem or cerebellum. Blind children may exhibit coarse, slow, searching, pendular nystagmoid movements. In cerebellar nystagmus, the fast phase moves toward the side of the lesion.

- **Opsoclonus:** It is a rapid, chaotic, multidirectional eye movement seen classically in neuroblastoma. When these rapid jerky movements are only in the horizontal plane, the term ocular flutter is used.

Trigeminal Nerve (Cranial Nerve V)

These cranial nerves are among the largest and serve both sensory and motor functions. They provide primary sensory innervation to the face, head, oral and nasal cavities, and teeth, while their motor fibers control the muscles of mastication (**Fig. 15**).

Motor Function

Instruct the child to open their mouth against resistance and then clench their teeth. While they do so, palpate the masseter and temporalis muscles to assess strength. If there is unilateral weakness, the jaw will deviate toward

the affected side due to the unopposed action of the healthy pterygoid muscle, and lateral jaw movement will be impaired.

Sensory Function

With the child's eyes closed, assess sensation by gently touching areas corresponding to the ophthalmic, maxillary, and mandibular branches of the trigeminal nerve. Test for light touch, pain, and temperature perception.

Corneal Reflex

The corneal reflex is mediated by the trigeminal nerve (afferent limb) and the facial nerve (efferent limb). It is not routinely tested in children. If necessary, instruct the child to look upward and gently touch the cornea's lateral edge with a fine wisp of cotton, observing for a blink response.

Both the afferent and efferent limbs of the jaw jerk reflex are controlled by the trigeminal nerve. The test is performed by having the child relax with their mouth slightly open while the examiner places an index finger on the chin and taps gently with a reflex hammer. An exaggerated response indicates upper motor neuron (UMN) involvement above the pons.

Facial Nerve (Cranial Nerve VII)

Motor: As a primarily motor nerve, the facial nerve controls the muscles involved in facial expression. Examine the child's resting facial features, noting any unevenness in the palpebral fissures, eyelid closure, nasolabial creases, and the positioning of the lips.

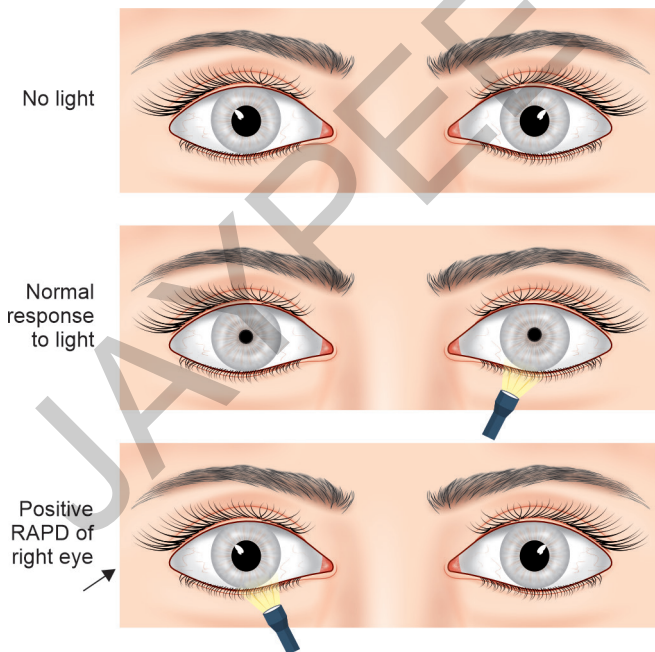


Fig. 14: Normal light reflex and relative afferent pupillary defect (RAPD).

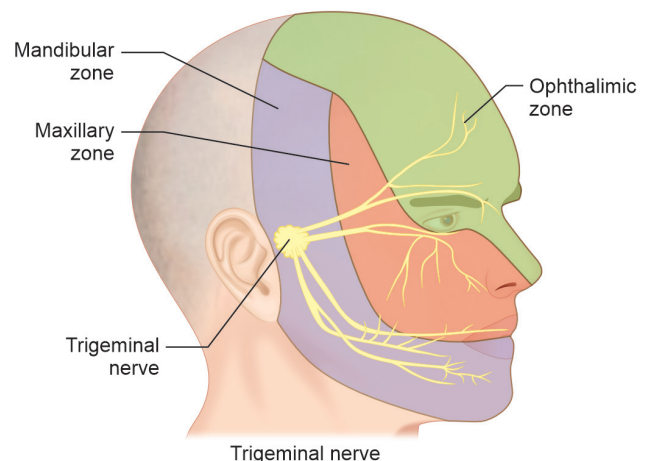


Fig. 15: Trigeminal nerve distribution.

To test, ask the child to raise their eyebrows and observe the forehead. Asymmetrical wrinkling or flattening on one side is a sign of upper facial muscle weakness.

Then ask the child to shut both eyes firmly. Failure to close one eye completely and an increased palpebral gap on that side are indicative of orbicularis oculi weakness. Bell's phenomenon—upward deviation of the eyeball—can also be observed.

Ask the child to show his teeth and to smile (orbicularis oris and levator anguli oris). A normal side can smile; the weak side usually has flattening of the nasolabial fold with drooping of that side of the mouth.

Air leaks on attempted blowing or tapping with lips closed and mouth full of air (buccinator).

When the nerve to the stapedius is compromised within the facial canal, it can result in heightened auditory sensitivity.

Sensory: The Facial nerve conveys taste from the front two-thirds of the tongue via the chorda tympani; if necessary, assess taste using natural flavors such as sweet, salty, or sour agents.

A parasympathetic component supplies the lacrimal gland.

Facial nerve dysfunction can be due to congenital factors, spontaneous idiopathic onset (Bell's palsy), trauma, immune-mediated demyelination (e.g., Guillain-Barré), infectious agents like HSV and HIV, infiltrative or granulomatous diseases, or neoplastic processes. A key differential diagnosis is congenital absence of the depressor anguli oris. Bilateral facial nerve palsy can be challenging to identify, often presenting as a mask-like, expressionless face. It is associated with conditions such as Guillain-Barré syndrome, Moebius syndrome, infantile botulism, and certain myopathies. In cases of LMN facial paralysis, the entire side of the face is affected, whereas in UMN paralysis, the forehead and eye muscles are spared due to bilateral cortical representation of the upper face. Emotional facial expressions remain intact in UMN lesions (Fig. 16).

Vestibulocochlear Nerve (Cranial Nerve VIII)

The vestibulocochlear nerve consists of two branches: the vestibular nerve, responsible for transmitting signals from the semicircular canals to the cerebellum to regulate balance and posture, and the cochlear nerve, which carries auditory information from the cochlea to the auditory cortex in the temporal lobe for sound perception.

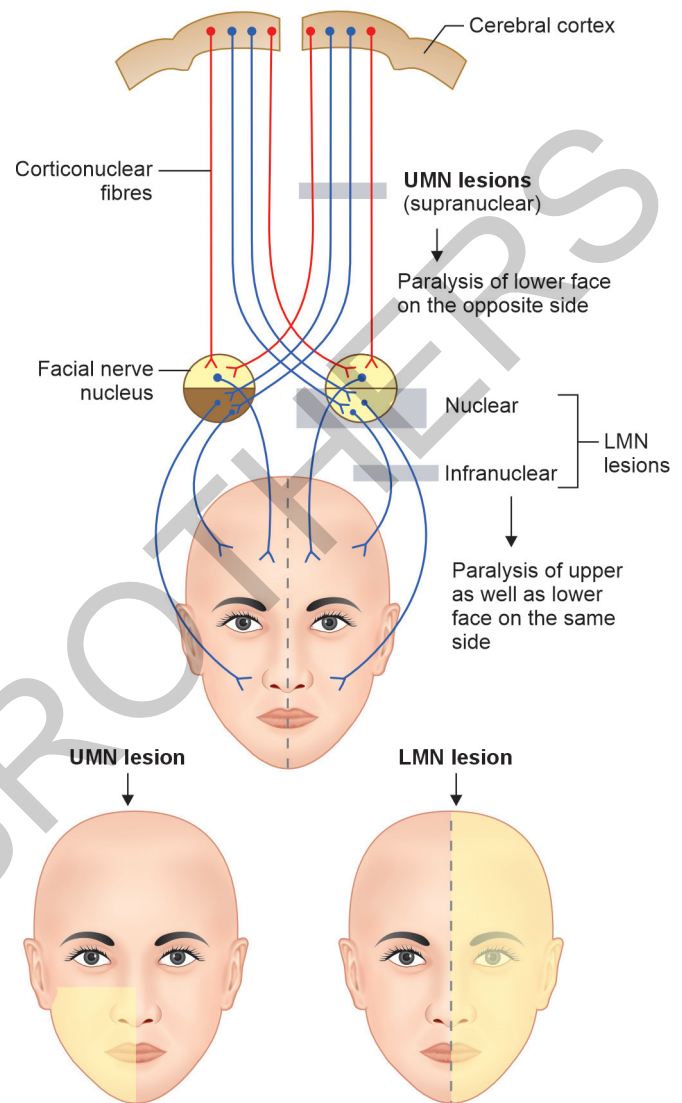


Fig. 16: Facial nerve lesion.

For infants and children, bedside cochlear nerve testing is most easily carried out by asking the parent if there have been any concerns about their child's hearing. A soft bell or rattle can be used to check for auditory response by noting if the child turns their head toward the source of the sound, or in a verbal child, by asking the child himself if he can hear the tickling of your watch. If you suspect a hearing deficit, look in both external auditory canals for wax, foreign bodies, or a perforated tympanic membrane.

Cochlear nerve: If hearing impairment is present, assess whether it is due to conductive or sensorineural causes using the Weber and Rinne tests (Fig. 17).

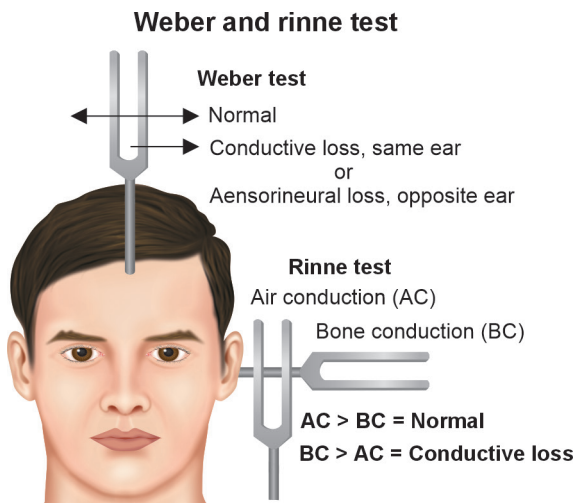


Fig. 17: Weber and Rinne test.

Rinne Test

To conduct the Rinne test, a tuning fork is applied to the mastoid bone until vibrations are no longer felt, then positioned near the ear to determine if air conduction surpasses bone conduction. Since air conduction is more efficient than bone conduction, the child should still perceive the sound produced by the vibrating tuning fork. A positive Rinne test indicates normal hearing, where air conduction exceeds bone conduction. In conductive hearing loss, bone conduction is perceived as louder (negative Rinne), whereas in sensorineural loss, air conduction is still greater than or equal to bone conduction.

Weber Test

In older children, the Weber test is performed by placing a vibrating tuning fork (256 or 512 Hz) on the center of the forehead or incisors and asking if the sound is louder in one ear. Normally, the sound is heard equally in both ears. In sensorineural hearing loss, it localizes to the unaffected ear, whereas in conductive hearing loss, it is louder in the affected ear. The Weber test is ineffective for diagnosing symmetrical hearing loss.

Vestibular

In vestibular dysfunction, the child may experience vertigo or dizziness. Examination often reveals nystagmus, which can be assessed using ice water caloric testing. The test should only be done if the ear canal is clear of wax and the tympanic membrane is intact. The child's head is

positioned at a 30° semireclined angle using pillows. Then, 10 mL of cold water (30°C) is gently introduced into the ear canal while nystagmus is recorded via electromyography. During formal testing, the slow phase of nystagmus moves toward the irrigated ear, indicating brainstem activity, whereas the fast corrective phase moves away, reflecting cerebral hemisphere involvement. Ice water caloric testing is commonly used to evaluate brainstem function in a comatose child. If the brainstem is intact, the eyes will turn toward the ear being irrigated, although the corrective nystagmus away from that ear will be absent. The fast, or corrective, phase of nystagmus is observed only in conscious individuals with preserved vestibular function. Absence of response during ice water caloric testing is among the key indicators for confirming brain death.

On Romberg and tandem gait testing, they tend to fall toward the abnormal ear.

Glossopharyngeal Nerve (Cranial Nerve IX) and Vagus Nerve (Cranial Nerve X)

The glossopharyngeal nerve (cranial nerve IX) provides motor innervation to the stylopharyngeus muscle and transmits sensory information from the posterior third of the tongue, pharynx, and tonsillar region. It is an essential component of both the swallowing mechanism and the gag reflex.

The vagus nerve has the widest distribution among cranial nerves, supplying all muscles of the soft palate (except the tensor veli palatini), as well as the pharynx and larynx. It provides both sensory and motor innervation to the respiratory tract, heart, and most abdominal organs through parasympathetic ganglia. These two nerves are closely linked, and it is uncommon for one to be affected without the other.

Assess the child's speech for any hoarseness, nasal resonance, or speech impairment. A combined IX and X nerve lesion can result in an absent swallowing reflex, leading to drooling and choking episodes.

Examine the uvula's movement by asking the child to say "Ahh." If there is asymmetry, the uvula will be pulled toward the stronger side. Also, you can assess the gag reflex by applying light pressure to the lateral oropharynx or soft palate with a tongue blade and noting symmetric palatal movement.

When the UMNs are affected, as in pseudobulbar palsy, the gag reflex becomes exaggerated, often observed in cases of diffuse brain injury or neurodevelopmental

disorders. On the other hand, in bulbar paralysis, the cranial nerves IX through XII or their brainstem nuclei in the medulla oblongata are involved. The loss of jaw jerk and gag reflex, along with speech and swallowing difficulties, are key clinical signs of bulbar paralysis, commonly seen in neurological conditions such as Guillain-Barré syndrome and poliomyelitis.

Unilateral vagus nerve dysfunction affects laryngeal function, causing voice hoarseness, while bilateral involvement can severely impact speech and airway control.

Accessory Nerve (Cranial Nerve XI)

The accessory nerve supplies the sternocleidomastoid (SCM) and trapezius muscles. The SCM muscle on one side turns the head to the opposite side, while both SCMs together flex the neck. The trapezius muscle functions to elevate the shoulder and assist in head and neck movement.

Observe the child for any signs of SCM muscle wasting or hypertrophy and palpate to evaluate muscle bulk. Stand behind the child and inspect the trapezius muscles for asymmetry, atrophy, or hypertrophy.

Assess SCM strength by asking the child to turn their head to each side while applying resistance with your hand (**Fig. 18**). Test the power of the trapezius by instructing the child to shrug their shoulders against resistance.

Muscle atrophy and weakness in these regions are commonly seen in skull base fractures or lesions, motor neuron disease, myotonic dystrophy, and myasthenia gravis; congenital torticollis, however, is associated with SCM enlargement.



Fig. 18: Examination of sternocleidomastoid.

Hypoglossal Nerve (Cranial Nerve XII)

- The hypoglossal nerve is responsible for tongue function.
- The tongue should be inspected for atrophy, fasciculations and tested for strength.
- Instruct the child to stick out their tongue and sweep it across both sides. Evaluate for asymmetry, fasciculations, or deviation toward the affected side.
- Next, instruct the child to press their tongue against the inner surface of each cheek while you apply resistance externally to evaluate tongue strength.
- Unilateral hypoglossal nerve (CN XII) dysfunction causes the tongue to deviate toward the affected side, while bilateral involvement results in an inability to protrude the tongue, often accompanied by dysphagia. In nuclear or infranuclear lesions, the tongue appears to be wasted and may exhibit fasciculations, with spinal muscular atrophy type 1 being a common cause in children. Proper assessment requires inspecting the tongue while it rests on the floor of the mouth, as the quivering movements seen in a crying infant may sometimes be mistaken for fasciculations.

■ CRANIAL NERVE EXAMINATION IN INFANT

Cranial Testing Nerve

- **CN2:** Assess visual awareness by noting blink response to bright light. Check for the presence and symmetry of red reflex using an ophthalmoscope.
- **CN 2 and 3:** Evaluate pupillary light reflex for both direct and consensual response.
- **CN3, 4, and 6:** Inspect ocular alignment and conjugate gaze at rest. Perform the oculoccephalic reflex ("Doll's eye" maneuver) by gently rotating the infant's head side to side and up and down, observing for compensatory eye movement in the opposite direction.
- **CN5:** Observe rooting and sucking reflexes during feeding. Assess corneal sensitivity using a soft stimulus near the eye and monitor for blinking or grimacing.
- **CN7:** Watch facial movements during spontaneous activity, such as crying and smiling, to assess symmetry.
- **CN8:** Evaluate auditory function by observing startle or turning response to sudden loud sounds.
- **CN9 and 10:** Assess swallowing and coordination during feeding. Listen to the cry quality for hoarseness or nasal tone. Test gag reflex using gentle posterior pharyngeal stimulation.

- **CN11:** Note head turning and shoulder elevation symmetry, especially during spontaneous movement.
- **CN12:** Inspect tongue movements during sucking or when crying; look for deviation, fasciculations, or weakness.

Motor Examination

Assessment of muscle bulk, tone, strength, and involuntary movements.

- **Bulk:** assess three S—(1) the size, (2) shape, and (3) symmetry of a muscle.
 - By inspection—Muscle atrophy, or decreased muscle bulk, can result from disuse or LMN palsy, with neurogenic atrophy typically being more severe than myogenic atrophy. It is important to determine whether the atrophy primarily affects proximal or distal muscle groups and whether it is symmetrical or asymmetrical. Limb circumference should be measured at consistent anatomical landmarks, such as the anterior superior iliac spine or olecranon, to ensure accurate assessment.
 - Muscle hypertrophy is typically a normal physiological adaptation, but in some cases, it can be misleading. Pseudohypertrophy occurs when muscle tissue is replaced by fat and connective tissue, leading to an apparent increase in size but a paradoxical loss of strength, as seen in Duchenne and Becker muscular dystrophies. When muscle weakness is present, palpation is crucial to assess texture and firmness. Muscle tenderness may indicate an inflammatory myopathy, such as dermatomyositis or postinfectious myositis.
- **Tone:** Resistance to passive movement of a joint. From a clinical perspective, muscle tone can be categorized into postural tone and phasic tone.

Muscle tone fluctuates significantly depending on the child's age and level of alertness.

In newborns:

- At 28 weeks of gestation, all four extremities are extended (**Fig. 19**).
- 32 weeks: Flexor tone in the lower extremities
- 36 weeks: Flexor tone in both upper and lower extremities
- A normal term infant's posture is characterized by flexion of all four extremities.

In infants, the range of motion can be quantified using angles (adductor angle, popliteal angle, dorsiflexion angle



Fig. 19: A preterm baby with universal extension posture.

of foot, and scarf sign), as described in the Amiel-Tison method.

Amiel-Tison (Figs. 20A to E):

- **Adductor angle**—in the supine position, the hips are abducted by holding on to the extended knee. The adductor angle is the angle between the thighs. It is $<75^\circ$ below 3 months of age.
- **Popliteal angle**—measured by flexing the hip to 90° while extending the knee as much as possible. The angle between the thigh and lower leg was recorded.
- **Ankle dorsiflexion**—angle between the dorsum of the foot and the anterior aspect of the leg when the foot is dorsiflexed.
- **Scarf sign**—in the supine position, the upper limb is pulled across the chest, holding the flexed elbow. The elbow does not cross the midline below 3 months of age.
- **Heel to ear maneuver**—lift both the legs of the supine infant without lifting the pelvis from the table and try to bring it to the ear.

Tests for assessing postural tone in infants: Traction response, vertical suspension, and horizontal suspension.

- **Traction response:** Gently holding the infant's hands and pulling them into a sitting position typically reveals a slight head lag, which is expected at this stage. As the infant reaches the upright position, the Head momentarily lags behind before falling forward. This response is considered normal in early infancy, reflecting developing neck and trunk control (**Fig. 21**).



Figs. 20A to E: (A) Adductor angle; (B) Popliteal angle; (C) Ankle dorsiflexion; (D) Scarf sign; and (E) Heel to ear maneuver.



Fig. 21: Traction response.



Fig. 23: Horizontal suspension.



Fig. 22: Vertical suspension.

- **Vertical suspension:** Lifting the infant by the axillae without applying pressure to the thorax should allow the infant to remain suspended, with the lower limbs maintaining a flexed position. In cases of hypotonia, the infant may struggle to maintain this posture and could slip through the examiner's hands due to insufficient muscle tone (**Fig. 22**).
- **Horizontal suspension:** Supporting the infant in a prone position with a hand placed under the abdomen should prompt the infant to lift the head and flex the limbs. However, in a hypotonic infant, poor muscle tone causes the body to drape over the examiner's hand in a U-shaped posture, indicating weakness (**Fig. 23**).

■ TONE ASSESSMENT IN ELDER CHILDREN

- **Posture:** Observe the spontaneous posture of the child when lying down in bed. A floppy hypotonic child lies in a frog-like position (abduction at the hip and flexion at the knee). A hypertonic child will have extended lower limbs and may have scissoring.
- **Feel the muscles:** Normal muscles feel firm. Hypotonic muscles feel flabby and soft. Hypertonic muscles feel stiff.
- **Resistance to passive stretching and flap ability—for the upper limb—**hold the child's hand and extend and flex the elbow several times. Clasp-knife spasticity will usually be easy to feel (initial stiffness followed by giving way). Then, still grasping the hand, alternate between pronation and supination and flexion and extension of the wrist for lower limb—tested in three ways. (1) Can roll legs from side to side (a good indicator of rigidity); (2) by placing your hand under each knee, abruptly lifting it upward. The heel should remain on the bed. If the heel raises, this indicates increased tone; (3) place one hand on the knee and the other hand around the feet, and unpredictably flex and extend the knee.

Muscle tone must be assessed bilaterally with the head in a neutral position to ensure symmetry. It may be normal, elevated (as in hypertonia—spasticity or rigidity), or diminished (hypotonia).

Abnormalities of Tone

- **Hypertonia:** Elevated muscle tone is often linked to UMN involvement or disorders affecting the basal ganglia.

- **Spasticity:** Spasticity is characterized by a sudden release of resistance during passive movement, referred to as the clasp-knife phenomenon. It results from UMN lesions and typically involves increased tone in flexor muscles of the arms and extensor muscles of the legs. In infants, spasticity in the lower limbs may lead to leg scissoring when the child is suspended vertically. Older children often show persistent commando crawling or walking on their toes due to this increased muscle tone.
- **Rigidity:** Associated with basal ganglia lesions, this condition presents as uniform resistance to passive movement in both flexor and extensor muscles, regardless of movement speed, resembling a “lead-pipe” rigidity. Individuals with spasticity or rigidity may develop opisthotonos, a condition characterized by pronounced spinal hyperextension due to excessive paraspinal muscle tone.
- **Hypotonia,** or reduced muscle tone, is the most frequently observed tone abnormality in neonates with neurological impairment. A hypotonic infant appears floppy and often rests in a frog-leg position. The underlying cause may involve the CNS (cerebral hemispheres, cerebellum, spinal cord) or the peripheral nervous system (anterior horn cells, peripheral nerves, neuromuscular junction, or muscle). In cases of central hypotonia associated with UMN disorders, deep tendon reflexes (DTRs) remain present and are usually exaggerated. Conversely, in neuromuscular disorders, hypotonia is accompanied by absent or diminished DTRs.

Power: Power is the force that can be voluntarily generated.

Formal motor power testing is generally feasible in cooperative older children.

Evaluating a child's posture and performing simple tests can aid in assessing muscle strength. In older children, muscle power can be observed through their movements around the examination room. For upper limb assessment, the child is instructed to extend both arms fully at shoulder height with palms facing upward and maintain the position (**Fig. 24**).

In cases of a contralateral pyramidal tract lesion, the affected forearm gradually pronates and drifts downward, a sign known as pronator drift. This finding suggests spasticity, and closing the eyes can enhance the effect by further revealing difficulty in maintaining the position.



Fig. 24: Pronator drift.

Formal muscle strength testing can typically be performed in children older than 3 years. The easiest way to do this is to put the child's arms into the position of the muscle strength you are testing. For example, if you are testing for shoulder abduction, in the sitting position, stabilize the shoulder, and ask the child to lift the arm. Apply resistance in the downward direction to prevent abduction. Likewise, in the case of the lower limb, to examine the power of the knee extensor, ask the child to extend the knee against the resistance in the downward direction applied by you. Know the root values for the different muscle actions.

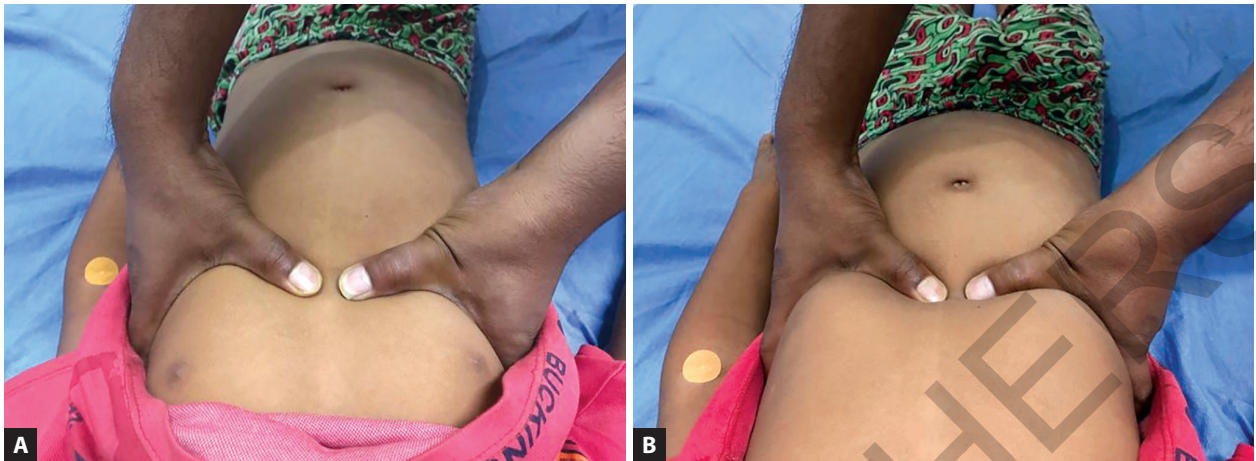
- **Upper limb:** Test shoulder adduction (C7, C8)/abduction (C5), elbow flexion (C5.6)/extension (C7, 8), wrist flexion/extension against resistance. To assess handgrips, request the child to grasp and squeeze the examiner's fingers when prompted. In a pyramidal lesion, flexors are stronger than extensors (the reverse from the legs) (**Figs. 25A to F**).
- **Lower limb:** Assess muscle strength for hip flexion (L1, L2) and extension (L5, S1), hip abduction and adduction, knee flexion (L3, L4) and extension (S1), ankle dorsiflexion (L4), plantar flexion (S1, S2), as well as foot inversion and eversion (**Figs. 26A to H**).
- **Neck:** Extend and flex the neck against resistance offered at the occiput and the chin, respectively.
- **Intercostal muscles and diaphragm**—splint the abdomen below the costal margin with the hand. This will restrict the movement of the diaphragm, and hence, if the intercostals are weak, there will be increasing dyspnea. For the diaphragm, splint the



Figs. 25A to F: (A) Shoulder abduction; (B) Shoulder adduction; (C) Elbow flexion; (D) Elbow extension; (E) Wrist flexion; and (F) Wrist extension.



Figs. 26A to H: (A) Hip flexion; (B) Hip extension; (C) Hip adduction; (D) Hip abduction; (E) Knee flexion; (F) Knee extension; (G) Ankle dorsiflexion; (H) Plantar flexion.



Figs. 27A and B: (A) Intercostal muscle weakness and (B) Diaphragm weakness.

chest by encircling the chest with the examiner's hand. Dyspnea will increase if the diaphragm is paralyzed (**Figs. 27A and B**).

- **Abdomen:** For the upper abdomen, ask the child to raise the head and chest in the supine position without any support. For the lower abdomen, to raise his extended leg without support. When testing the upper abdomen, if there is upward deviation of the umbilicus (Bevor sign), it indicates bilateral lower abdominal wall paralysis.

Muscle power is graded on a scale of 0–5 as follows (Medical Research Council power scale):

- 0 = No contraction
- 1 = Flicker or trace of contraction
- 2 = Active movement with gravity eliminated
- 3 = Active movement against gravity
- 4 = Active movement against gravity and some resistance
- 5 = Normal power.

Since infants and young children cannot engage in formal strength testing, functional assessments are the most effective approach. Proximal and distal upper limb strength can be evaluated by observing the child reach overhead for a toy and manipulate small objects, providing insight into their motor abilities. Infants with lower extremity weakness often exhibit reduced spontaneous leg movements and struggle to bear weight when held in an upright position. In older children, weakness may present as difficulty with activities such as climbing stairs, jumping, or hopping. When asked to rise from a prone position, they may push against their legs with their hands

to assist in standing, a characteristic movement known as Gowers' sign.

For subtle weakness—Ask the child to perform tasks such as hopping on one foot, walking forwards and backwards, toe walking, heel walking, and rising from a squatting or supine position. These activities can reveal subtle weaknesses in the limbs. Additionally, evaluate whether the weakness is predominantly affecting the proximal muscles—as seen in conditions like myopathy, dermatomyositis, or Guillain-Barré syndrome—or the distal muscles, which is more typical of peripheral neuropathies, and note whether the weakness is symmetrical or asymmetrical.

A special mention regarding myotonia, in which the contraction of the muscle persists even after Voluntary efforts have ceased. The child will have difficulty in releasing a handshake. Do not forget to shake hands with parents in those cases—as it may demonstrate myotonia in parents in case of myotonic dystrophy.

Involuntary Movements

Check for unusual movements and determine whether they are present or absent while the child is asleep. All involuntary movements disappear during sleep except hemiballismus and myoclonus.

Most involuntary movements, such as tics, dystonia, chorea, and athetosis, originate from basal ganglia dysfunction. Tremors, however, appear to be an exception, as they are believed to involve the cerebellothalamocortical pathways. Fasciculations, on the other hand, are characteristic of LMN lesions (**Table 7**).

TABLE 7: Involuntary movements in childhood.

Chorea	Involuntary, quasi-purposeful, continuous, writhing, flowing, and irregular movements. These movements primarily affect the proximal joints, becoming more pronounced with excitement and subsiding with purposeful action. Ex-Sydenham Chorea
Ballism	Involuntary, high-amplitude flinging movements, typically generated proximally. Lesion in the subthalamic nuclei.
Athetosis	Slow, writhing, or continuous involuntary movement. It most often affects the distal joints and occurs in association with chorea, hence the name choreoathetosis
Tics	Sudden, involuntary motor or vocal actions that are repetitive and irregular in pattern
Myoclonus	Rapid, shock-like contraction of muscle. Occurs due to lesions in the brainstem or reticular formation
Dystonia	Intermittent and sustained involuntary muscle contractions. Common causes—birth asphyxia, kernicterus, and encephalitis
Tremors	Repetitive, rhythmic shaking of a limb caused by alternating activity in antagonist muscle groups; may occur at rest or with movement
Fasciculations	Brief, involuntary flickers of muscle movement arising from single motor unit activation, typically noted in the lingual muscles of young children

REFLEXES DEEP TENDON REFLEXES AND THE PLANTAR RESPONSE

Involuntary muscle contractions occur when a tendon is tapped with a reflex hammer, causing a brisk stretch of the muscle. Tendon reflexes are heightened in individuals with pyramidal tract lesions, whereas they are diminished or absent in cases of LMN or muscle disorders. Observe for limb movements and visible muscle contractions during the assessment.

Deep tendon reflexes can usually be elicited easily in infants and children. When assessing reflexes in infants, it is crucial to keep the head in a midline position, as turning it to one side may influence reflex tone. Reflex responses are graded on a scale from 0 (absent) to 4+ (markedly hyperactive), with 2+ considered normal. Reflexes graded as 1+ or 3+ may also be normal if they are symmetrical. If a reflex is difficult to elicit, reinforcement techniques such as having the child clench their teeth or interlock their fingers (Jendrassik maneuver) can be used to enhance the response.

Examine for Superficial and Deep Reflexes

Superficial Reflexes

- To evaluate the abdominal wall reflex (T7–T12), gently stroke the abdominal skin from the side toward the midline and watch for contraction of the anterior abdominal muscles. This reflex may be diminished or absent in cases of LMN lesions affecting T7–T12 or UMN dysfunction (**Fig. 28**).

**Fig. 28:** Abdominal reflex.

- The cremasteric reflex (L1) is tested by stroking the inner thigh while observing for elevation of the ipsilateral testis. A normal response involves the testis rising on the same side as the stimulus. Anal reflex (S3, S4)—elicited by gently stroking the perianal skin. It results in the puckering of the anal orifice.
- The plantar response (S1, S2) is assessed by firmly stroking the lateral sole of the foot with a blunt object or thumbnail. A normal (negative) response consists of plantar flexion of the big toe. Upward movement (dorsiflexion) of the big toe, called a positive Babinski sign, indicates a UMN lesion but is considered normal in infants (**Fig. 29**).

Deep Tendon Reflexes (Figs. 30A to E)

- With the child in a relaxed state and the head positioned centrally, use a tendon hammer to gently tap each tendon, observing the expected muscle contraction and relaxation. Compare the reflex response on both



Fig. 29: Plantar response.

sides to assess for symmetry and any abnormalities (Table 8).

- Upper limb:* Biceps reflex, Triceps reflex, Supinator (brachioradialis) reflex.
- Lower limb:* Knee reflex, ankle reflex.

In newborns and infants, the middle finger can be used in place of a reflex hammer to elicit DTRs. Reflex responses tend to be brisk during infancy. When the knee-jerk reflex is tested on one side, a crossed adductor response may be observed, which is considered a normal finding in infants.

CLONUS

The child should be in a relaxed state, with the hips flexed and the knee and ankle in moderate flexion. Support the leg by placing one hand under the knee while grasping the foot from below, then rapidly dorsiflex the foot. In cases of clonus, rhythmic alternating contractions may occur. Sustained clonus is always pathological, but in infants under three months, 5–10 beats may be normal, while in older children, one to two beats can be considered normal if symmetrical.



Figs. 30A to E: (A) Biceps reflex; (B) Triceps reflex; (C) Supinator reflex; (D) Knee reflex; and (E) Ankle reflex.

TABLE 8: Deep tendon reflexes and their normal responses.

Reflex	Spinal level	Procedure	Normal Response
Biceps reflex	C5–C6	Tap the biceps tendon in the antecubital fossa	Elbow flexion
Supinator reflex	C5–C6	Tap the brachioradialis tendon near the radial styloid	Forearm flexion or supination
Triceps reflex	C7–C8	Tap the triceps tendon above the olecranon	Elbow extension
Knee reflex	L2–L4	Tap the patellar tendon below the kneecap	Knee extension
Ankle reflex	S1–S2	Tap the Achilles tendon while the foot is slightly dorsiflexed	Plantar flexion of the foot

TABLE 9: Primitive reflexes.

Reflex	Onset	Duration
Palmer grasp	28 weeks of gestation	2–3 months postnatal
Rooting	32 weeks	1 month postnatal
Moro	28–32 weeks	5–6 months postnatal
Tonic neck	35 weeks	6–7 months postnatal
Parachute	7–8 months postnatal	Remains throughout life

Developmental Reflexes/Primitive Reflexes (TABLE 9)

Newborns operate mainly through reflex actions, exhibiting primitive reflexes that are expected at birth. The absence of these reflexes may signal serious neurological issues. As the brain matures, these primitive reflexes typically diminish, disappearing by around 4–6 months of age. Meanwhile, postural reflexes start to appear, beginning at 3–4 months and completing development by 8 months. These postural reflexes help the infant maintain balance and prepare for walking. If primitive reflexes persist and postural reflexes fail to develop, this may indicate UMN dysfunction in infants.

Persistence or absence outside the normal developmental window suggests neurological impairment.

- **Moro reflex:** The reflex is elicited by holding the infant semi-erect and carefully dropping the head backward onto the examiner's palm. Normally, the infant responds by opening their hands, extending and abducting their arms, then bringing them back in with flexion, often crying. An uneven reaction could signal a clavicle fracture, injury to the brachial plexus, or hemiparesis. Absence of this reflex in a term of infant may indicate generalized CNS suppression (**Fig. 31**).

**Fig. 31:** Moro reflex.

- **Suck, root:** Observing or palpating the tongue's rhythmic stripping motion can help assess feeding coordination. The rooting reflex is elicited by gently stroking the infant's cheek near the mouth, causing the baby to open their mouth and turn their head toward the touch.
- **Galant (trunk incurvation):** Support the infant in a ventral suspension position and gently stroke one side of the lower back. A normal response involves the trunk and hips curving toward the side of the stimulus.
- **Grasp response:** Elicited by placing a finger in the open palm of each hand; at 37 weeks, the reflex is strong enough to allow lifting up of the infant from bed. Marked retention of these reflexes beyond 6 months is characteristic of athetoid cerebral palsy (**Fig. 32**).
- **Asymmetric tonic neck reflex:** This reflex is triggered by turning the infant's head to one side. The expected response is extension of the upper limb on the side the face is turned toward and flexion of the upper limb on the opposite side. A persistent or obligatory tonic neck reflex, where the infant remains fixed in the fencing posture, is always considered abnormal (**Fig. 33**).



Fig. 32: Grasp response.



Fig. 34: Finger-nose test.



Fig. 33: Asymmetric tonic neck reflex.

Postural Reflexes

- **Landau:** Age of appearance—4–5 months. When held in ventral suspension, the infant demonstrates extension of the head and legs.
- **Parachute reflex:** Hold the child in ventral suspension and suddenly lower him as if he or she were falling. The arms will extend to break the infant's fall. Absent or incomplete response is seen in cerebral palsy.

COORDINATION/CEREBELLAR SIGN

Coordination relies on visual and proprioceptive feedback to execute complex movement patterns and is controlled by the cerebellum. Unlike cortical lesions, cerebellar dysfunction produces ipsilateral rather than contralateral

signs. As coordination develops with age, younger children may naturally exhibit some clumsiness. Impairment of the cerebellum disrupts the smooth execution of voluntary movements, leading to ataxia.

- **Lesions to the cerebellar vermis:** Result in truncal ataxia, a broad-based gait, or difficulty performing tandem gait assessment.
- **Lesions of the cerebellar hemispheres:** Lead to appendicular ataxia, which becomes evident during tasks such as reaching for objects or performing finger-to-nose and heel-to-shin tests.

Cerebellar disorders often present with hypotonia, preserved muscle strength, and increased fatigability. Speech may be slurred, explosive, and jerky with scanning of syllables. There may be nystagmus and intention tremor.

Of Upper Limbs

Finger-nose test (Fig. 34): This test is done by instructing the child to touch the tip of their nose with their index finger, then reach out to touch the examiner's fingertip. The child should repeat the movement as quickly as possible while the examiner moves their finger to different positions. It is normal for children to do this maneuver repeatedly. But a child with cerebellar affection may have dysmetria or past pointing (errors in judging distance), dyssynergia (irregular, clumsy, uncoordinated motion), and intention tremor (the tremor appears when the child attempts to accurately reach for a target).

Rebound phenomenon: The examiner attempts to extend the patient's elbow while the biceps actively contract against resistance. If the wrist is suddenly released, the



Fig. 35: Dysdiadochokinesia.



Fig. 36: Heel-shin test.

hand may recoil abruptly and could strike the patient's face.

Dysdiadochokinesia: Instruct the child to rapidly alternate between palm-up and palm-down positions by tapping one hand against the other. Difficulty or inability to perform this movement smoothly is known as dysdiadochokinesia. Swaying or oscillation of the arms held outstretched suggests cerebellar dysfunction (**Fig. 35**).

For Lower Limbs

Heel-shin test: With the child lying on their back, ask them to slide the heel of one foot down the shin of the other leg repeatedly and rapidly. Observe the accuracy and smoothness of the movement (**Fig. 36**).

- **Reflexes:** Deep tendon jerks are pendular
- **Gait:** A broad-based, unsteady gait is characteristic of cerebellar ataxia.

Ask the child to walk heel-to-toe in a straight line, with the toes of the rear foot touching the heel of the front foot at every step. This tandem gait test is useful for identifying subtle balance problems.

- **Romberg's sign:** Although not a cerebellar sign, this test evaluates position sense loss (sensory ataxia) in the legs, often due to dorsal column, spinal cord, or peripheral nerve disorders. The child is asked to stand independently with their eyes closed, while the examiner stays close for safety. A positive test is indicated by swaying or loss of balance. In cerebellar ataxia, instability is present even with the eyes open.

■ STANCE AND GAIT

Stance refers to how a person stands, while gait describes how they walk. Typically, normal children can stand with their feet close together without swaying. During standing, observe for spinal abnormalities such as kyphosis, scoliosis, or lordosis.

Evaluate the child's ability to stand from a prone position; children under 3 years often turn prone before standing due to immature pelvic muscle strength. Beyond 3 years, if they need this maneuver, it indicates proximal muscle weakness (Gowers sign), wherein the child climbs up his legs to stand due to weakness of the glutei, classically seen in Duchenne's muscular dystrophy.

Evaluate walking and running—by age 5, a normal gait should follow a heel-toe pattern. A toe-heel gait (toe-walking) is often idiopathic but can indicate pyramidal tract dysfunction or pelvic girdle weakness. Subtle gait asymmetries can be detected using Fogs' test, which involves walking on the toes, heels, and the inner and outer edges of the feet.

Gait analysis is most reliable when the child is not self-conscious or aware of being examined. Look for arm swing, pelvic tilt, lurching to one or both sides, length of steps, any asymmetry, and associated movements of the upper limbs. Finally, make him walk in tandem steps and observe.

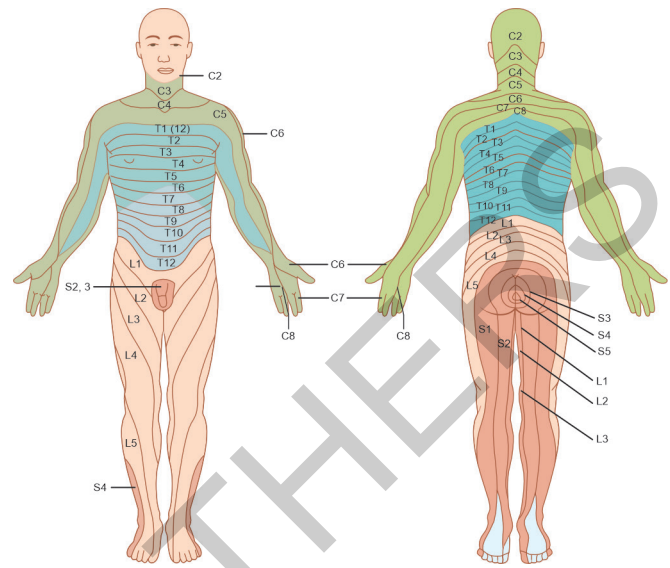
There are a variety of abnormal gaits (**Table 10**).

Sensory Examination

The spinal cord contains two main groups of sensory nerve fibers. The posterior column transmits sensory information from the same side of the body, including

TABLE 10: Abnormal gait.

Gait	Appearance	Condition
Antalgic	The stance phase is briefer than the swing	Musculoskeletal pain
Broad based	Staggering, wide-spread feet	Cerebellar ataxia
Stamping	Wide-based steppage gait	Sensory ataxia
Scissoring	Stiff-legged, drag and cross the midline	Diplegia
Circumduction	Circumduction of the leg, decreased arm swing on the affected side	Hemiplegia
Waddling	Drooping of the hip girdle on the contralateral side	Proximal myopathy
High-stepping	Stepping high to avoid injury to the feet	Lower motor neuron (LMN) foot drop

**Fig. 37:** Sensory dermatome.

stereognosis, proprioception, vibration, and the perception of weight, shape, and size. In contrast, the anterior and lateral columns carry sensory signals for touch, pain, and temperature from the opposite side of the body.

Sensory System Examination

Sensory testing involves assessing touch, pain, temperature, vibration, joint position (proprioception), and cortical sensory processing. These tests require the child to be old enough to cooperate and accurately describe sensations, making sensory examination challenging and often overlooked in young children. Observation can aid in identifying sensory deficits, such as changes in skin color, temperature, or sweating, with the skin appearing cool and dry below the level of injury. A basic assessment can be performed by engaging the child with a toy while gently touching different areas with a cotton swab. Normal infants and children typically respond by crying or withdrawing the affected limb, and isolated sensory disorders are rare in very young children.

- **Sensory level:** Identifying a sensory level is crucial for determining the location of a spinal cord lesion. On examination, the sensory deficit typically appears one to two spinal segments below the actual site of the lesion.
- **For older child:**
 - **Lateral column sensation:**
 - ♦ **Touch:** Assess light touch sensation by lightly stroking the skin with a piece of cotton. Ask

the child to keep their eyes closed and indicate when they feel the touch. Compare both sides for symmetry.

Remember these important landmarks for dermatomes (**Fig. 37**):

- C1 gives no supply to the skin.
- C2 The occiput.
- C7 supplies the middle finger.
- T3 in the axilla.
- T10 supplies the umbilicus.
- L3 supplies the knee.
- L5 outer aspect of the tibia
- S1 supplies the little toe.
- S3, 4, 5 around the anus.
 - **Pain:** Assess pain sensation by gently pricking the skin with a fresh pin.
 - **Temperature:** Evaluate the child's response to temperature changes by applying a cooled or heated metal surface. You may also use a cold spray or body warmth as simpler alternatives.

Posterior column sensation:

- **Joint position (proprioception):** Hold the child's big toe by stabilizing the middle segment with one hand and gently gripping the tip from the sides with the other. Move the distal phalanx up and down while explaining the directions to the child. Have the child close their eyes, then repeat the movement several times, pausing at either the up or down position. Ask the child to

identify the position of their toe. Repeat the test in various positions.

- **Vibration:** Testing vibration sense in children can be challenging. Use a 128 Hz tuning fork and place it on the distal phalanx of the big toe, just below the nail bed. Vibration should also be assessed over the small joints of the fingers and toes. If perception is impaired, move proximally until normal sensation is detected.

Cortical localization:

Cortical localization includes:

- **Two-point discrimination:** A caliper or a pair of compasses with sharp ends can be used to evaluate two-point discrimination. Begin with a wider distance to establish communication and ensure the child's cooperation. Randomly alternate between touching one or two points, asking the child to distinguish between them. Normal discrimination distances are approximately 2–4 mm on the fingertips, 4–6 mm on the dorsal fingers, 8–12 mm on the palm, and 20–30 mm on the dorsum of the hand.
- **Astereognosis:** (The child is unable to recognize objects of different shapes by touching alone without visual or other sensory input). Use familiar items such as a coin, sharpener, pencil, button, or safety pin. Test each hand separately (**Fig. 38**).
- **Graphesthesia:** Assess the child's ability to identify letters or numbers drawn on the palm by tactile sensation alone (**Fig. 39**).

Significant impairments in graphesthesia and stereognosis are commonly associated with contralateral hemispheric lesions, particularly in the parietal lobe.

- **Point localization:** Ability to localize the area touched with eyes closed on the face, arm, hand, leg, and foot. The child is asked to point to the area being touched or tell the area verbally. This is the parietal lobe function.
- **Sensory inattention:** Simultaneous stimulation is performed by pricking two corresponding spots on opposite sides of the body. A patient with a sensory deficit may not perceive the prick on the affected side. In cases of parietal lobe lesions, when both a proximal and distal area (such as the cheek and hand) are touched at the same time, the patient may fail to recognize the distal stimulus due to sensory extinction.

Meningeal Irritation Sign

Meningeal signs are present when the meninges are inflamed from infection or the presence of blood.

Two closely related terms are important in this regard:

- **Meningismus** is the presence of meningeal signs, found in meningitis, whereas **Meningism** is the presence of neck stiffness in the absence of meningeal inflammation
- Check for signs of meningeal irritation when appropriate.
- **Neck stiffness:** With the child in a supine position, support the head with one hand and gently attempt to flex the neck. Alternatively, ask a conscious child to bend their neck forward and touch their chin to their chest. Increased resistance or discomfort may indicate meningeal irritation. Neck stiffness is an ominous sign in children suffering from meningitis and meningoencephalitis. However, it may also be

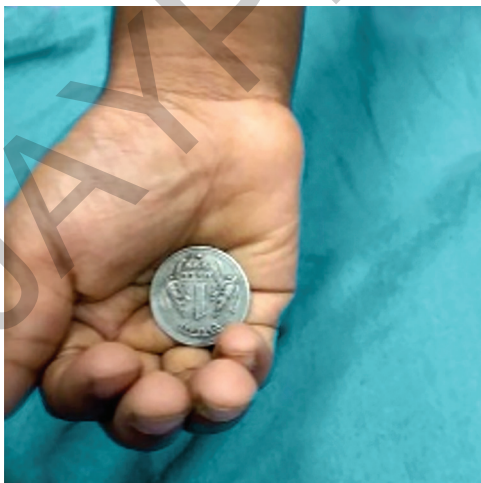


Fig. 38: Stereognosis.

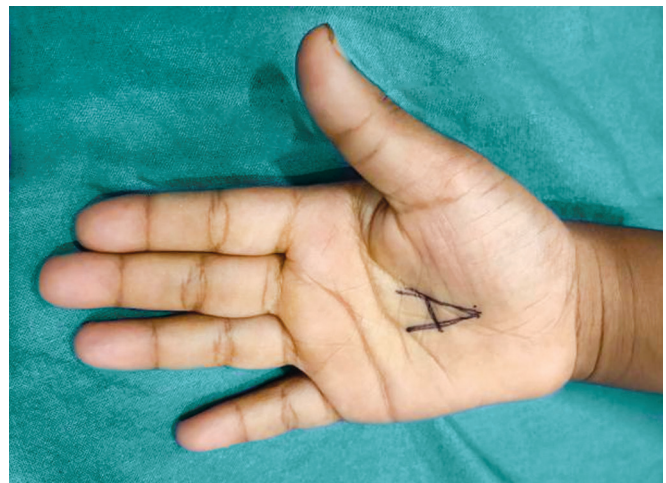


Fig. 39: Graphesthesia.

seen in some children with tonsillitis and cervical lymphadenopathy. It may be absent in the presence of meningitis in young infants and very sick children. To test neck rigidity in a struggling infant, let the head hang off the edge of a table.

Signs Associated with Neck Stiffness

- **Kernig sign:** Position the child supine and flex one hip and knee. While maintaining hip flexion, gently extend the knee. Pain in the posterior thigh and difficulty extending the knee suggests meningeal irritation. Kernig's sign is typically absent if neck stiffness results from local causes rather than meningeal inflammation.
- **Brudzinski sign:** With the child lying supine, support the head with one hand while placing the other hand on the chest to restrict movement. Gently attempt to flex the neck. A positive sign is demonstrated by spontaneous flexion at the hips and knees, pointing toward meningeal inflammation (**Fig. 40**).

AUTONOMIC FUNCTIONS

The autonomic nervous system regulates involuntary functions by controlling smooth muscles and glands. It consists of central components, including cortical areas, the amygdala, hypothalamic, and brainstem nuclei, as well as peripheral components such as the sympathetic thoracolumbar and parasympathetic craniosacral outflows. These functions operate independently of conscious control.

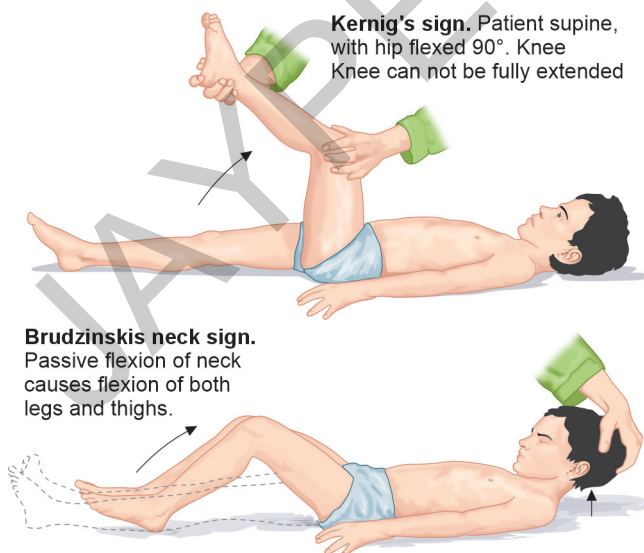


Fig. 40: Kernig and Brudzinski sign.

Symptoms—complain of symptoms related to:

- Orthostatic hypotension—lightheadedness, syncope, and palpitations, which become worse with standing.
- Abnormalities of sweating—excessive or reduced sweating, areas of skin with blanching, etc.
- Dysfunction of the gastrointestinal or genitourinary tracts. Inadequate or absent salivation or lacrimation, alternate diarrhea or constipation, and sphincteric disturbances.

Measure the pulse rate at rest and after six maximal deep breaths. A normal response is a decrease of >15 beats/min, whereas in autonomic dysfunction, the decrease is less than 10 beats per minute. Additionally, in patients with autonomic dysfunction, a drop of >20 mm Hg in systolic blood pressure upon standing from a lying position indicates orthostatic hypotension.

SKULL AND SPINE

The bony framework surrounding the CNS, including the skull and spine, requires close inspection. Look for variations in size, shape, symmetry, swellings, and abnormalities in sutures or fontanelles.

- **Head size:** Head circumference should be measured at every visit for children under 3 years, as it serves as an indicator of brain growth. Then it should be recorded on a suitable growth chart to look for any aberrations. To measure, a plastic nondistensible tape is placed anteriorly over the nasion and supraorbital ridge and posteriorly over the external occipital protuberance surrounding the head (**Fig. 41**). When a patient presents with an abnormal head circumference, it is



Fig. 41: Occipital frontal circumference (OFC) measurement.

necessary to document the head circumferences of family members, including parents and siblings.

In healthy premature infant's rate of growth of head circumference:

Increases by 0.5 cm per week during the first 2 weeks, 0.75 cm in the third week, and 1.0 cm in the fourth week, continuing at 1 cm weekly until the 40th week

- Head circumference of a term infant: 34–35 cm at birth, thereafter the rate of growth of head circumference:
 - First 3 months @approximately 2 cm/month
 - 3–6 months @1cm/mt
 - 7–12 months @0.5 cm/month
 - 1–3 years @1cm/ 6 months
 - 3–5 years @1 cm/year: The adult head circumference is achieved at around 5–6 years of age.
- *Abnormal size:*
 - *Macrocephaly (large head):* OFC > 2 SD above the mean for age and sex, most commonly familial or may be a disturbance in growth if there is megalencephaly (enlarged brain, or hydrocephalus)
 - *Microcephaly (small head):* An OFC less than three standard deviations below the mean for age and sex may result from primary reduced brain growth or secondary premature suture closure. It can also be associated with craniosynostosis (premature fusion of cranial sutures).
- *Shape—alteration occurs in:*
 - *Plagiocephaly:* While some skull flattening is normal in infants, it is more pronounced in hypotonic cases.
 - *Craniosynostosis*—cranial sutures fuse prematurely, may be in the form of brachycephaly (premature closure of coronal suture), dolichocephaly (closure of sagittal suture), trigonocephaly (metopic suture), oxycephaly (multiple sutures).
 - *Hydrocephalus*—frontal bulging
 - *Box-shaped heart*—chronic subdural effusion, rickets.
- *Fontanels:*
 - Two fontanelles are normally present in an infant at the time of birth.
 1. *Anterior fontanel:* A soft, diamond-shaped area found where the frontal and parietal bones meet, usually measuring around 2 × 2 cm. It generally closes between 9 and 18 months and is most accurately evaluated when the infant is calm, such as during sleep or feeding in an upright position.

Normal anterior fontanel: The fontanel is normally slightly depressed and pulsatile. A bulging fontanel may signal increased intracranial pressure (ICP). If the anterior fontanelle is very small or absent at birth, conditions such as craniosynostosis and microcephaly should be considered. Several conditions in infants, including hypothyroidism, rickets, achondroplasia, and Zellweger syndrome, present with large and late-closing fontanels. A depressed fontanel may indicate dehydration, while a full, bulging fontanel is a sign of increased intracranial pressure.

2. *Posterior fontanel:* Triangular in shape, this fontanel is located at the junction of the parietal and occipital bones and typically closes between 6 and 8 weeks. It is usually small enough to admit the tip of a finger or may already be closed at birth. If it remains open beyond the expected time frame, it may indicate hydrocephalus or congenital hypothyroidism.

- *Shiny skin with prominent, distended veins*—in hydrocephalus, increased intracranial pressure, sinovenous thrombosis.
- *Palpation of a newborn's skull*—may reveal.
 - Overriding sutures (molding)—caused by the pressure applied to the skull as it passes through the pelvis during birth.
 - Premature fusion of cranial sutures leading to restricted skull growth (craniosynostosis)
 - Cranial defects.
 - Thinning and softening of the parietal bones, known as craniotabes.
 - Macewen's sign (a “cracked pot” sound upon skull percussion) should be assessed. It is considered normal in early infancy due to open sutures.
 - *Transillumination:* Place a beam of bright torch light on the skull in a darkened room to look for a halo around the light. if there is hydrocephalus, there will be generalized transillumination in the entire skull.
 - Transillumination may also be positive in subdural effusion.
- Auscultation of the skull—cranial bruit best heard using the diaphragm of the stethoscope. This may appear at the anterior of fontanel, temporal areas, or in the orbital regions.

- Soft, symmetrical bruits can sometimes be heard in healthy children under 4 years old and during fever.
- A loud bruit may indicate severe anemia, raised intracranial pressure, or vein of Galen malformation.
- Spine—to look for any tuft of hair or localized swelling over the spine indicating underlying spina bifida, any sinus, gibbus, kyphosis, or scoliosis. Check for surgical scars on the spine, which may indicate prior to meningocele repair, or on the scalp, which may suggest ventriculoperitoneal shunt placement.

Bedside Clinics in Pediatrics Ready Reckoner

Salient Features

- The book contains case discussion system-wise by experts in the field of pediatrics and pediatric subspecialties. The case is discussed in a question–answer pattern to help overcome the practical examination with a model answer
- Provides oral tables like X-rays to identify, vaccines as ready reckoner, and instruments to identify and the probable questions to be asked in oral examination
- Includes OSCE which has become a part of both undergraduate and postgraduate examinations. It contains around 50 OSCEs. Although it is difficult to cover the whole subject in 50 questions, it will guide you to understand the pattern of OSCEs
- Special emphasis has been given on history taking as well as system-wise general examination of the particular system.

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