Neurologic examination of the newborn

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INTRODUCTION

A comprehensive neurologic assessment should be performed in any newborn suspected to have central or peripheral nervous system dysfunction, either based upon history (eg, perinatal asphyxia) or a physical finding detected during the routine neonatal assessment (eg, a weak and immobile upper extremity). (See "Assessment of the newborn infant", section on 'Neurologic examination'.)

There are several publications that describe the newborn neurologic examination [1-15]. The discussion on the neonatal neurologic assessment presented here is based upon a review of the literature and the experience of the author. Other aspects of the routine assessment of the newborn are discussed separately. (See "Assessment of the newborn infant".)

OVERVIEW

The goals of the neurologic examination are to recognize emergent and treatable issues like status epilepticus, assist in localization of the disturbance, establish a diagnosis, and, to some extent, help predict the long-term outcome [16].

The following factors may influence the results of the neurologic examination and must therefore be taken into consideration:

- Conceptional age (CA; ie, gestational age [GA] plus postnatal age) of the infant – The normal neurologic examination changes with maturation. In particular, passive tone and posture vary with GA and are used by the Ballard score to assign GA (figure 1 and table 1). (See "Postnatal assessment of gestational age", section on 'New Ballard score' and 'Passive tone/posture' below.)
- Level of alertness – The examination findings may vary depending upon the level of alertness of the infant. (See 'Level of alertness' below.)
- Experience of the clinician – Many neonatal neurologic maneuvers are dependent upon the skill of the examiner (eg, elicitation of tendon reflexes).
- Serial examinations are sometimes necessary to reliably establish the neurologic findings.
- Sedation from medications or anesthesia.
- Hypothermia, including therapeutic.

The neurologic examination includes the following components:

- General assessment, including determining pulse, blood pressure, temperature, the level of alertness, and examination of the skin, head, and spine
- Motor function
- Cranial nerves
- Reflexes (deep tendon, superficial, and developmental)
- Sensory examination
- Behavioral assessment

GENERAL ASSESSMENT

Key components of the general examination include:

- Conceptual age (CA) and gestational age (GA) (figure 1 and table 1), determination of whether the infant's birth weight is appropriate for gestation

- Careful examination of the head and spine, and identification of dysmorphic features that suggest a congenital anomaly or inborn errors of metabolism.

- Vital signs (pulse, blood pressure, and temperature) are essential in assessing critically ill infants. Newborns on a therapeutic hypothermia protocol for perinatal hypoxic ischemic encephalopathy may be on a cooling blanket, with core body temperature being maintained at 34°C. Pharmacologically induced skeletal muscle paralysis may be utilized for ventilatory management and can influence the assessment of eye movements, motor function, or reflexes.

- Pain assessment by identifying clinical markers for pain, such as changes in facial expression or crying, and elevations in heart rate or blood pressure, is imperative. Close to 20 percent of newborn infants experience pain during the delivery process or shortly thereafter. This aspect of the newborn assessment is frequently overlooked, yet can impact feeding, sleep, and parent-child interactions [17]. (See "Assessment of neonatal pain", section on 'Pain assessment'.)

A collection of videos depicting elements of the neurologic examination in infants can be viewed on the Pediatric NeuroLogic Exam website [15].

Other components of the general newborn assessment are discussed separately. (See "Assessment of the newborn infant".)

Observation — A brief period of observation before handling the infant is recommended to determine the level of alertness, presence of involuntary movements [11], resting posture, and respiratory rate. (See 'Passive tone/posture' below.)

Level of alertness — The neurologic examination varies depending upon the following five levels of alertness (state).

- Quiet sleep (equivalent to nonrapid eye movement sleep in older infants)
- Active sleep (equivalent to rapid eye movement sleep in older infants)
- Awake/drowsy (eyes open without gross movements)
- Alert
- Crying

As an example, muscle tone is diminished during active sleep compared with quiet sleep. It increases in the alert infant and is maximum in a hungry, crying infant.

The optimal time for neurologic assessment is when the infant is quiet and awake, which is typically after feeding but before the infant has fallen back to sleep. In extremely preterm infants (GA before 28 weeks), it may be difficult to differentiate between the various behavioral states [14].

An altered level of alertness is commonly observed with acute neurologic dysfunction in the neonate [14]. It may manifest as diminished or absent response to arousal stimuli, such as persistent and gentle shaking, perioral tactile stimulation, pinching, shining a light in the eyes, or ringing a bell.

The following classification of altered alertness states for infants greater than 28 and 30 weeks gestation is based upon the neonatal response to arousal maneuvers and a more noxious stimulus, such as sternal rub or pressure on the supraorbital nerve [14].

- Normal – The infant appears awake, and has normal arousal and noxious stimuli responses.
- Mild stupor (lethargic) – The infant appears sleepy, with slightly diminished arousal and noxious stimuli responses.
- Moderate stupor – The infant is unresponsive, with moderately diminished arousal and noxious stimuli responses.
- Deep stupor – The infant is unresponsive, with an absent arousal response and a markedly diminished reaction to noxious stimuli.
- Coma – The infant is unresponsive, with absent arousal and noxious stimuli responses.

Encephalopathy — Altered levels of alertness are seen in infants with cortical dysfunction, including neonates with perinatal hypoxic ischemic encephalopathy. For these patients, initial assessment should be carried out quickly to determine whether or not they qualify for therapeutic hypothermia. (See "Clinical features, diagnosis, and treatment of neonatal encephalopathy", section on 'Rapid assessment'.)

Head — The examination includes measuring the occipitofrontal circumference, noting the skull shape, presence of abnormal hair patterns, scalp defects, and unusual lesions or protuberances such as caput succedaneum or cephalohematoma. Additional evaluation includes transillumination with a bright light source and auscultation with a stethoscope for bruits. (See "Assessment of the newborn infant", section on 'Head'.)

Head size — The occipitofrontal circumference (OFC) should be measured at its maximum using a cloth tape. The OFC should be measured at its maximum circumference using a cloth tape. The tape should be positioned in the front just above the nasion (ie, bridge of the nose where the frontal and nasal bones of the skull meet), and in the back, across the external occipital protuberance (picture 1). At 40 weeks gestation, the average OFC is 35 cm (10th to 90th percentile, 33 to 37 cm).

Microcephaly, defined as an OFC more than 2 standard deviations (SD) below the mean (<3rd percentile), is usually due to a genetic abnormality or toxic, metabolic, or intrauterine infections (eg, Zika virus or toxoplasmosis) that affect cell proliferation and migration and myelination (table 2 and table 3). (See "Microcephaly in infants and children: Etiology and evaluation", section on 'Microcephaly' and "Microcephaly in infants and children: Etiology and evaluation", section on 'Etiology'.)

In contrast, macrocephaly is defined as an OFC greater than 2 SD above the mean (>97th percentile). Macrocephaly is caused by an increase in size of any of the components of the cranium, such as the brain parenchyma, cerebrospinal fluid, blood, or bone, or may be associated with increased intracranial pressure (table 4). (See "Macrocephaly in infants and children: Etiology and evaluation", section on 'Etiology'.)

Fontanel — The fontanels should be palpated, preferably with the infant in the sitting position. The following findings may be associated with a specific neurologic condition.

- Anterior fontanel tends to be small in infants with microcephaly, and enlarged in those with hydrocephalus, megalencephaly, hypothyroidism, or peroxisomal disorders, such as cerebro-hepato-renal syndrome.

- A tense and bulging fontanel in an infant who is placed in the sitting position and is not crying may indicate raised intracranial pressure, which can occur in patients with subdural hematoma, bacterial meningitis, or cerebral edema. (See "Neonatal birth injuries", section on 'Subdural hemorrhage' and "Bacterial meningitis in the neonate: Neurologic complications".)

Sutures — The principal sutures of the skull (sagittal, coronal, lambdoid, and metopic) should be palpated. Passage through the birth canal may result in moulding, a temporary asymmetry of the skull caused by overlapping or overriding of the sutures. However, an asymmetric skull that persists for longer than two to three weeks after birth, or a persistent

palpable ridge along the suture line is abnormal, and might suggest craniosynostosis. (See "Overview of craniosynostosis", section on 'Categorization of cranial deformities'.)

Scalp swelling — Edema or bleeding into various compartments within the scalp and skull may be due to injuries that occur during delivery, particularly vacuum assisted extraction. These injuries include caput succedaneum, cephalohematoma, and subgaleal hematoma, and are discussed separately (figure 2). (See "Neonatal birth injuries", section on 'Extracranial injuries'.)

Auscultation — Auscultation of the head may detect a venous hum, which is a benign finding. However, an asymmetric systolic-diastolic bruit that is heard over the eyeballs and anterior fontanel may indicate the presence of an arteriovenous malformation involving the vein of Galen. The bruit is best heard by using the bell of the stethoscope. Vein of Galen malformations may present with high-output cardiac failure.

Transillumination — Transillumination of the head is performed by placing a bright light source with a dark rim on to the scalp surface in a darkened room. The normal zone of transillumination is generally 1 to 2 cm from the rim of light, and is somewhat larger over the frontal than the parieto-occipital regions. Increased transillumination suggests a fluid-filled, intracranial lesion such as hydrocephalus, hydranencephaly, or porencephalic cyst.

Spine — The spine is inspected and palpated to detect abnormalities, such as a neural tube defect. Myelomeningocele is a readily apparent neurulation defect that is most often localized to the lumbar region, and less frequently at the thoracic or cervical level. (See "Pathophysiology and clinical manifestations of myelomeningocele (spina bifida)".)

The gluteal folds should be separated to determine presence of a sacrococcygeal sinus, asymmetric gluteal cleft, lipoma, hemangioma, or sacral dimple suggestive of a congenital dermal sinus (picture 2). Other findings indicative of a post-neurulation defect include a tuft of hair (picture 3), or discoloration of the skin over the spine. Post-neurulation defects also include tethered cord syndrome and diastematomyelia (split cord). (See "Assessment of the newborn infant", section on 'Trunk and spine'.)

Associated findings — The following findings may be associated with a congenital disorder with neurologic abnormalities:

- Hypopigmented skin patches may be associated with tuberous sclerosis. (See "Tuberous sclerosis complex: Genetics, clinical features, and diagnosis".)

- Facial hemangioma involving the ophthalmic and maxillary divisions of the trigeminal nerve distribution is suggestive of the Sturge-Weber syndrome. (See "Sturge-Weber syndrome".)

- The combination of petechiae and hepatosplenomegaly may suggest congenital cytomegaloviral infection. (See "Congenital cytomegalovirus infection: Clinical features and diagnosis", section on 'Clinical manifestations'.)

- Congenital brain anomalies may be associated with dysmorphic facial features such as hypotelorism, hypertelorism, low set ears, narrow palpebral fissures, and cleft lip and/or palate. (See "Congenital cytogenetic abnormalities" and "Primary (congenital) encephalocele".)

- Atrophy or hypoplasia of the orbicularis oris muscle may accompany congenital myopathies and may present as an inverted U-shaped appearance of the upper lip. (See "Congenital myopathies".)

- Arthrogryposis (contractures around multiple joints) may be associated with congenital myasthenia gravis, myopathies, or anterior horn cell disease. (See "Spinal muscular atrophy", section on 'Arthrogryposis multiplex congenita'.)

MOTOR FUNCTION

The evaluation of motor function is based upon the assessment of passive tone and posture, and active muscle activity.

Passive tone/posture — Passive tone is assessed by observing the resting neonatal posture, and may be measured by the resistance to passive movement of the limbs. The normal resting posture and passive tone of the neonate vary with conceptional age (CA) (figure 1 and table 1) [1,15].

The normal resting posture is evaluated with the head placed in midline. It should be symmetric in all four extremities and varies with CA as follows (figure 1):

- ≤28 weeks CA – All the infant's limbs are passively extended
- 32 weeks CA – Slight flexion starting at the knee, then the hip with extended upper extremities

- 34 weeks CA – Increasing flexion of the knee and hip with extended upper extremities
- 36 weeks CA – Weak flexion of all four extremities
- 40 weeks CA (term infant) – Strong flexion of all four extremities

Passive tone can be measured in a supine infant starting at 28 weeks CA by using the following passive maneuvers (figure 1) [1,3]. During these manipulations, the head should remain in the midline position.

- Popliteal angle – At 28 weeks CA, extension of the lower extremity at the knee (while the hips are still in contact with the bed) is approximately 150 degrees (popliteal angle), whereas in the 40 week CA term infant, the range of the popliteal angle diminishes to 80 degrees.

- Scarf sign – The scarf sign measures the range of shoulder adduction. At 28 weeks CA, it is possible to adduct the shoulder and bring the elbow to touch the opposite shoulder, whereas by 40 weeks CA, passive adduction decreases so that the elbow only reaches midline.

Active muscle function — By 32 to 34 weeks gestational age (GA), infants should exhibit symmetric, smooth, and spontaneous movements in all extremities. Persistent asymmetry may indicate weakness of the side that is less active. As an example, the affected upper limb is generally immobile in infants with a brachial plexus lesion. (See "Brachial plexus syndromes", section on 'Neonatal brachial plexus palsy'.)

In most normal term infants, small amplitude, choreo-athetoid movements of the hands are seen. Common physiological findings during active sleep include fragmentary myoclonus that migrates from one limb to another, facial twitches, and irregular respiratory rate. Although jitteriness or tremulousness can occur in normal awake infants, sustained tremulousness beyond the fourth day of life may suggest marker of cortical dysfunction [4].

Active motor tone and strength can be measured in the axial and appendicular musculature by the following:

- Stepping response can be obtained in infants born ≥32 weeks CA by holding the infant in a vertical position with his/her feet in contact with a flat surface [15]. This initiates a slow alternate stepping action of flexion and extension of the legs (figure 3).

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Vertical suspension measures the strength of the neonate's shoulder girdle. The examiner holds the infant in an upright position by placing the hands under the arms and around the chest with feet unsupported (figure 4). Vertical suspension is also helpful in evaluating for subtle increases in muscle tone in the lower extremities. In the presence of lower extremity hypertonicity, there may be adduction at the hips combined with hyperextension at the knees and ankles. The lower extremities may appear to cross over each other (scissoring posture).

- Head control – By 40 weeks CA, the infant has sufficient neck and truncal strength to maintain the head in line with the trunk for one to two seconds while being pulled from the supine to sitting position (figure 5).

- Ventral suspension measures the strength of the infant's trunk and neck. The infant is held in a suspended prone position in the air by placing a hand under the chest. A normal term infant will keep his/her head in the horizontal plane momentarily with flexion of both the upper and lower extremities (figure 6). Passive, limp extremities and lack of neck extension may be seen with neuromuscular disorders.

Prechtl system — The quantity and quality of gross body movements in both preterm and term infants can be studied using the assessment system developed by Prechtl, based on postmenstrual age for the preterm infant [18]. At birth, infants normally demonstrate "writhing." By six to nine weeks of age, movements change to a fidgety pattern that includes small amplitude circular movements of the neck, trunk, and extremities that are optimally observed when the infant is supine, awake, and quiet. Fidgety movements evolve concurrent with neuromaturation and resolve by 20 weeks of age. Absence of fidgety movements at a time when they should be normally present is predictive of major long-term neurologic sequelae. Ideally, it is recommended that there be integration of the Prechtl assessment and the traditional neurologic examination [19]. (See "Hyperkinetic movement disorders in children", section on 'Benign neonatal sleep myoclonus'.)

Abnormal motor exam — Hypotonia (decreased muscle tone) is the most common motor abnormality. When associated with weakness it is suggestive of a congenital myopathy, a disorder of the anterior horn cells of the spinal cord such as infantile spinal muscular atrophy, or a neuromuscular junction disorder such as myasthenia gravis. When hypotonia is associated with relative preservation of mobility and with tendon reflexes that are brisk or exaggerated, it is likely that hypotonia is of central nervous system (CNS) origin (eg, Down

syndrome). Of note, it is difficult to identify a motor abnormality in extremely preterm infants below 28 to 30 weeks GA because of physiologic low tone, immature posture, and decreased motility.

Hypotonia — The hypotonic term infant lies supine in a frog-like position with the hips abducted and the limbs abnormally extended [6]. Spontaneous activity is decreased. Decreased muscle tone can also be recognized when the following are observed:

- Vertical suspension – Decreased tone of the shoulder girdle allows the infant to slip through the examiner's hands, and the legs are extended (figure 4).
- Ventral suspension – The infant appears limp with the extended limbs and the head drooping (figure 6).
- Head control – The head lags behind as the infant is pulled from the supine to sitting position and continues to lag when the sitting position is reached (figure 5).

Neonatal hypotonia is caused by a variety of conditions that affect the CNS (brain or spinal cord), peripheral nervous system, or skeletal muscles (table 5). These disorders and the approach to an infant with hypotonia are presented separately. (See "Overview of peripheral nerve and muscle disorders causing hypotonia in the newborn" and "Approach to the infant with hypotonia and weakness".)

Hypertonia — Hypertonia is less common than hypotonia. It is associated with dysfunction of the pyramidal or extrapyramidal systems. Spasticity is a form of hypertonia that accompanies pyramidal tract dysfunction. It is characterized by an abnormal lengthening-shortening reactions of muscles. It is most apparent in the distal portion of the extremities. On passive flexion-extension of the limb, a "clasp knife" type of resistance is felt. Rigidity is associated with increased resistance to movement throughout the range of motion at the joint. It may be proximal or distal, and has the feel of bending a "lead pipe" or a "cog wheel". Sometimes it is difficult to determine whether the newborn hypertonia is due to spasticity or rigidity. Congenital brain anomalies and hypoxic-ischemic lesions are the most common etiologies of hypertonia.

Opisthotonus (ie, persistent arching of the neck and trunk) is caused by decreased cerebral cortical inhibition of the labyrinthine-brainstem-spinal cord motor projections that mediate extension of the trunk and proximal extremities. It is associated with moderate to severe acute bilirubin encephalopathy or a severe, diffuse disturbance of cortical function. In

general hypotonia, with brisk tendon reflexes suggests CNS dysfunction, whereas hypotonia with diminished to absent tendon reflexes suggests a neuromuscular etiology. (See "Clinical manifestations of unconjugated hyperbilirubinemia in term and late preterm infants" and "Congenital cytogenetic abnormalities".)

Movement disorder — Movement disorders include the following:

- Jitteriness comprising of rapid, to and fro movement of the muscles that is abolished by manually relaxing the affected muscle group. Jitteriness is often present in neonates with drug withdrawal or with mild hypoxic encephalopathy. It is often confused with seizures.

- Seizures in the neonate are generally accompanied by nystagmoid eye movements, stereotyped chewing movements, or twitching of one corner of the mouth that cannot be suppressed. Seizures are observed in neonates with encephalopathy, structural brain injuries, metabolic disturbances, and central nervous and systemic infections. (See "Clinical features, evaluation, and diagnosis of neonatal seizures", section on 'Clinical features'.)

- Hemiparesis is suggested in a neonate with diminished spontaneous movements of one side of the body.

- Unilateral immobile upper extremity may be indicative of a brachial plexus lesion. (See "Neonatal brachial plexus palsy".)

- A thoracic or lumbar spinal cord lesion may be suspected in a patients with normal upper extremity motor function but diminished lower extremity movements.

- Fragmentary myoclonus (twitches and jerks that migrate from one part of the body to another) may be physiologic and accompany normal rapid eye movement sleep. However, if unusually prominent, myoclonus may be a marker for a metabolic encephalopathy.

- Persistent choreoathetoid movements beyond the second or third week of life may be due to bilirubin encephalopathy, a result of perinatal hypoxic-ischemic encephalopathy, or an inborn error of metabolism.

CRANIAL NERVES

CN I and XI — Although a response to peppermint smell in the form of alerting or stereotyped sucking can be elicited as early as 32 weeks conceptional age (CA), smell (I) is rarely tested in the neonate [20]. Consistent anosmia (complete loss of smell) may accompany arhinencephaly, which is a disorder of failure of development of the olfactory bulbs and tracts. Cranial nerve (CN) XI is also rarely tested because it is difficult to assess the function of the sternocleidomastoid muscle in the newborn.

Cranial nerve II — Visual responses mature with CA [14]:

- At 26 weeks CA, infants consistently blink to light.
- At 32 weeks CA, infants begin to show signs of fixation.
- At 34 weeks CA, most infants can track a fluffy ball of red wool.
- At 37 weeks CA, infants will turn their eyes towards a soft light.

A response to an opticokinetic stimulus is present in the majority of infants by 36 weeks CA and can be observed by using commercially available drums or with tapes made of alternating black and white stripes.

It is difficult to directly examine the optic fundi in the newborn because of the small pupil size. Indirect ophthalmoscopy can be requested dependent upon the clinical setting.

Intact CNs II and III are necessary for a pupillary light response, which is consistently present by 35 weeks gestation [14].

CN III, IV, and VI — Extraocular movements can be evaluated by observation of spontaneous eye movements or by the use of the Doll's eye phenomenon, which assesses the vestibulo-ocular response (VOR).

The Doll's eye maneuver test can be performed as early as 25 weeks CA [14]. The test entails moving the head and neck from side to side, which leads to eye deviation to the opposite side. The conjugate deviation of the eyes in the opposite direction demonstrates intact eye adduction (III) and abduction (VI). Another method to demonstrate VOR is for the examiner to spin in one direction while holding the infant upright and supporting the neck. The eyes should deviate in the opposite direction to the spin. The neck can be flexed and extended in a similar manner to the Doll's test to produce vertical eye movements (IV).

Abnormal eye movements — The following abnormal eye movements may be associated with neurologic problems:

- Spontaneous, horizontal, unidirectional, jerky eye movements of abrupt onset and offset may be associated with a seizure originating in the opposite frontal lobe.
- Tonic horizontal deviation of the eyes to one side may suggest a destructive central nervous system (CNS) lesion in the hemisphere towards which the eyes are deviated.
- Tonic downward gaze deviation due to dysfunction of the pretectal region of the brainstem may indicate raised intracranial pressure.
- Skew eye deviation, in which the eyes are at different horizontal planes, suggest metabolic brainstem dysfunction. It is commonly observed after hypoxic ischemic encephalopathy.
- A unilaterally dilated and poorly reactive pupil in a comatose patient is a marker for ipsilateral uncal herniation.
- A unilaterally constricted pupil on the side of a flaccid, immobile upper extremity may indicate pupillary sympathetic paralysis due to brachial nerve injury involving the C8-T1 nerve roots (ie, partial Horner syndrome). (See "Horner syndrome".)

Cranial nerve V — The response to tactile stimuli, such as tissue paper or pinprick, over the face assesses CN V. This includes the corneal reflex in which gentle, tactile stimulation of the cornea with a tissue paper results in an ipsilateral and consensual blink response.

Cranial nerve VII — The normal newborn is able to close both eyelids during vigorous crying. In the presence of a peripheral VII nerve lesion, the ipsilateral eyelid may not close, and the nasolabial furrow may be less prominent on the affected side. In addition, sucking may be less vigorous with drooling of saliva or milk from the affected side.

Cranial nerve VIII — By 28 weeks gestation, the infant startles or blinks to a sudden loud noise [14]. During quiet wakefulness, term infants respond to the sound of a bell, rattle, hand clap, or voice by a startle response or an increase in their state of alertness. The consistent absence of a response in a quiet environment may suggest a possible hearing loss. (See "Hearing loss in children: Screening and evaluation" and "Screening the newborn for hearing loss".)

Sucking/swallowing — Muscle function for sucking and swallowing is mediated through branches of CNs V, VII, IX, X, and XII. Sucking patterns that are similar to term infants begin to appear around 33 and 34 weeks gestation. (See "Neonatal oral feeding difficulties due to sucking and swallowing disorders".)

Cranial nerves IX and X — Impairment of CNs IX and X is associated with difficulty in swallowing, diminished soft palatal movements, and a weak or absent gag reflex.

Cranial nerve XII — The tongue is innervated by CN XII. Atrophy and fasciculations of the tongue (slow undulating movement of the surface of the tongue) suggest degeneration of the hypoglossal nucleus, which is seen in patients with type I (infantile) spinal muscular atrophy. Deviation of the tongue to one side usually implies ipsilateral XII nerve palsy. (See "Spinal muscular atrophy".)

REFLEXES

Testing of reflexes provides information on the integrity of the central and peripheral nervous system.

Tendon reflexes — In the newborn, tendon stretch reflexes may be difficult to obtain and interpret. This aspect of the examination is most relevant when the reflexes are consistently absent, exaggerated, or asymmetric. As with all parts of the neurologic examination, the information should be interpreted in the context of other findings. As an example, the absence of a biceps reflex is likely not significant when the infant has a normal resting posture of flexion, and symmetrical spontaneous movement.

The following tendon reflexes can be elicited in the newborn infant generally after 33 weeks gestation. An examiner's finger that is placed over the tendon to be tested can be lightly struck with a percussion hammer to elicit the reflex.

- Jaw – Tapping the chin with the mouth slightly open leads to slight jaw closure.
- Biceps – With the elbow flexed, tapping the biceps tendon in the antecubital fossa leads to flexion at the elbow.
- Brachioradialis (supinator) – Tapping above the wrist on the radial aspect of the forearm leads to flexion at the elbow.
-

Knee (patellar) – Tapping the quadriceps tendon below the patella leads to extension of the knee.

In the newborn, it may be difficult to elicit the triceps reflex because of strong flexion of the elbows in the normal neonatal resting posture.

Lower motor neuron lesions are generally associated with diminished to absent tendon reflex responses, while upper motor neuron lesions are followed by exaggerated responses. Altitudinal dissociation in the amplitude of the tendon reflexes is helpful in determining the level of a spinal cord lesion. For example, an intact jaw jerk combined with absent biceps, brachioradialis, knee, and ankle reflexes in a flaccid immobile infant suggests a cervical spinal cord lesion. Similarly, the combination of normal jaw, biceps, and supinator reflexes with an absent patellar response may indicate a thoracic or lumbar spinal cord lesion.

Superficial — The following superficial reflexes can be elicited in the newborn:

- Abdominal reflexes in each of the four quadrants of the abdomen are elicited by gentle stroking in an axial to peripheral direction, resulting in contraction of the abdominal wall. The absence of response may be consistent with an ipsilateral pyramidal tract lesion.
- In males, the cremasteric reflex can be demonstrated by stroking the inner thigh area in an anterior to posterior direction, which results in ipsilateral scrotal retraction and testicular rise due to contraction of the scrotal dartos muscle. The lack of response or an asymmetrical response may be consistent with a corticospinal tract abnormality.
- Anal wink reflex is elicited by gentle stroking of the perianal region, which results in contraction of the perianal muscle. The absence of an anal wink reflex may be consistent with a spinal cord lesion.
- Corneal reflex (see 'Cranial nerve V' above).
- In the newborn infant, an extensor plantar response (Babinski) may be physiologic. It is elicited by stroking the lateral plantar surface of the foot with a pointed but not sharp object. An extensor plantar response may, however, indicate a corticospinal tract lesion, if it is consistent and persistent on one side, while on the opposite side there is a plantar flexor response.

Developmental reflexes — Developmental reflexes are also called primitive reflexes. They are mediated at the brainstem or spinal cord level and are generally present at birth (table

6) [15,21-25]. They resolve in a fairly set time course with maturation of descending, inhibitory projections from the cerebral cortex to subcortical motor systems. These reflexes may reappear later in life in the presence of degenerative disorders.

Developmental reflexes are abnormal if they are:

- Absent during the neonatal period
- Asymmetric (suggesting hemiplegia or monoplegia)
- Persist beyond the age by which they should have normally disappeared

The developmental reflexes include the following:

- Moro reflex – Moro reflex is elicited by the sudden dropping of the infant's head in relation to the trunk. It results in abduction and extension of the infant's arms and opening of the hands, followed by flexion (figure 7). It is present starting at 32 weeks gestation, well-established by 37 weeks gestation, and disappears by three to six months of age [14].

- Stepping reflex is obtained by holding the infant in a vertical position with the feet in contact with a flat surface [15]. This initiates a slow alternate stepping action of flexion and extension of the legs (figure 3). It is present starting at 32 weeks gestation and disappears by one to two months of age [14].

- Grasp reflexes (palmar and plantar) are well established by 32 weeks of age [14]. The palmar grasp reflex is generally present until three months of age in full-term infants, whereas the plantar grasp reflex is generally present until age six months in full-term infants (figure 8). The absence of the plantar grasp reflex in the term newborn has been reported to be associated with an increased risk of developing cerebral palsy [26].

- Asymmetrical tonic neck reflex (ATNR) is characterized by extension of the upper and lower extremities on the side to which the head and neck is turned with flexion of the contralateral upper extremity (fencing posture) (figure 9). This reflex is due to decreased cerebral cortical inhibition of the labyrinthine-brainstem-spinal cord pathway that subserves limb extension. ATNR that is seen as a resting posture rather than being elicited is never normal. ATNR appears at 35 weeks gestation, is well-established by one month postnatal age, and disappears by three to four months of age in a term infant, concurrent with maturation of descending inhibitory projections from the cerebral cortex [14].

- Galant reflex (trunk incurvation) is obtained by placing the baby in ventral suspension and then stroking the paravertebral region from the thorax to the lumbar area. This will elicit movement of the infant's trunk and hips towards the side of the stimulus.

SENSORY

The neonatal assessment of sensation is challenging because it is difficult to accurately determine an infant's response to sensory stimuli. As a result, sensory assessment is limited or not generally performed as part of the neonatal neurologic examination.

Perioral tactile sensation can be evaluated by the rooting response in which gentle stroking of the cheek results in the infant turning towards the stimulus with an open mouth ready to latch onto the object [23]. In a patient with a spinal cord lesion, there may be loss of pinprick sensation on the trunk or neck, below the level of the lesion. This is probably the one situation in the newborn where testing sensation with a pinprick provides useful information. Otherwise, testing in the newborn with pinpricks provides little useful information.

Assessment of pain is also difficult in the newborn and is discussed separately. (See "Assessment of neonatal pain".)

BEHAVIORAL EVALUATION

Evaluation of neonatal behavior assesses higher cortical function. Several behavioral neonatal assessment tools have been developed, including the Neonatal Behavioral Assessment Scale (NBAS). The NBAS includes 27 behavioral responses and 20 reflex items, and takes approximately 20 minutes to administer [2].

The following two key elements from the NBAS are incorporated into the neurologic examination:

- Consolability – is easiest to assess in a crying infant. A number of techniques are used to try to calm the infant within 15 seconds. They include infant sucking on a pacifier or the examiner's finger, holding, rocking, placing a hand on the infant's belly, restraining one or both arms of the infant, soothing by voice, or a combination of a soothing voice and the examiner's face approaching the infant. Infants with brain injury are more difficult to console than a normal infant.

- Habituation – Habituation is a marker of cortical inhibitory function and measures the infant's ability to learn to diminish his/her response to repetitive stimuli. The use of repeating light or auditory stimuli are used to test for habituation. The shining of a soft light initially elicits a blinking response that diminishes in intensity over the first four to five trials, and afterwards ceases with subsequent trials. Similarly, auditory stimuli, such as hand claps, elicit a startle response to the first four or five stimuli, after which there is little to no response. The lack of habituation is compatible with cortical dysfunction, which is exemplified by the behavior of an infant with prenatal substance abuse exposure.

INTERPRETATION AND PROGNOSIS

The newborn neurologic examination has some limitations in its specificity and its ability to predict long-term outcome, in part due to a number of intrinsic and extrinsic factors. The exam has limited utility in extremely preterm infants (gestational age <28 weeks) and those who are heavily sedated or receiving skeletal muscle paralytic agents for ventilator management. It is important to maintain a low threshold for obtaining a bedside electroencephalogram (EEG) whenever a disturbance of the central nervous system is suspected.

Nevertheless, abnormal neurologic findings can be useful in guiding care and predicting outcome in the following manner:

- They can define the site and extent of injury, which guides the choice of further management decisions. As an example, the neurologic examination is an important component in making the diagnosis of encephalopathy and determining its severity. The severity of encephalopathy guides management decisions, including the use of therapeutic hypothermia. (See "Clinical features, diagnosis, and treatment of neonatal encephalopathy".)

- Persistent neurologic dysfunction is associated with an increased risk of long-term neurodevelopmental deficits. As an example, preterm infants with abnormal neurologic examination at the time of discharge from the newborn nursery compared with those with a normal examination have an increased risk of long-term motor impairment (eg, cerebral palsy) [27,28]. The risk of cerebral palsy increases in infants with persistent hypotonia, weak cry, poor sucking, and decreased level of activity. (See "Long-term

neurodevelopmental outcome of preterm infants: Epidemiology and risk factors" and "Cerebral palsy: Epidemiology, etiology, and prevention".)

- Persistent asymmetric findings often are associated with an underlying abnormality such as stroke, and should be further evaluated.

- Composites of neurologic findings appear to be better predictors of outcome than isolated neurologic findings. As an example, the Prechtl test, which is based upon classifying gross body movements, is reported to be a good predictor of neurologic outcome at two years of age [4,29]. In one study of preterm infants (gestational age [GA] <30 weeks), the group with an abnormal Prechtl test was most likely to have abnormal Bayley Mental and Psychomotor Developmental Index scores at 24 months corrected age, followed by those with suspect results, and the normal group was least likely to have abnormal Bayley scores [29]. (See 'Prechtl system' above.)

SUMMARY AND RECOMMENDATIONS

A comprehensive neurologic assessment should be performed in any neonate who is suspected to have a neurologic abnormality either based upon history (eg, perinatal asphyxia) or a finding during routine neonatal assessment. (See "Assessment of the newborn infant".)

Findings on the neurologic examination vary with gestational age (GA) and the infant's level of alertness. Serial examinations are sometimes necessary to reliably confirm a neurologic finding. (See 'Overview' above and 'Level of alertness' above.)

The newborn neurologic examination can be useful in guiding care and predicting outcome. However, there are limitations in its specificity and prognostic ability, partly due to the number of factors that affect the results of the examination. (See 'Interpretation and prognosis' above.)

The neurologic examination includes the following components:

- General assessment includes the vital signs, determination of the conceptional age (CA) of the infant and whether the infant's birth weight is appropriate for CA, a thorough examination of the head and spine, and identification of abnormalities suggestive of an

underlying congenital disorder or perinatal event with associated neurologic problems. (See 'General assessment' above.)

- Motor function assessment is based upon evaluation of passive tone and posture, and active motor activity. Hypotonia is the most commonly identified motor abnormality, and is due to a variety of neurologic and muscular disorders (table 2). (See 'Motor function' above.)

- Cranial nerve (CN) development varies with CA. In general, CNs I and XI are not tested in the neonates. The assessment of extraocular movements (CNs III, IV, VI) is based upon observation of spontaneous eye movements or the infant's response to the Doll's eyes test. Observation of the infant's sucking and swallowing assesses CNs V, VII, IX, X, and XII. Impaired gag reflex and decreased movement of the soft palate suggest dysfunction of CNs IX or X. Fasciculations over the tongue are seen with degeneration of the hypoglossal (CN XII) nucleus. (See 'Cranial nerves' above.)

- Reflex examination provides information on the integrity of the central and peripheral nervous system, and includes evaluation of deep tendon, superficial, and developmental (also referred to as primitive) reflexes. Developmental reflexes (eg, Moro, stepping, grasp, and asymmetrical tonic neck reflex) require coordination of multiple muscles and nerves (figure 7 and figure 3 and figure 8 and figure 9). (See 'Reflexes' above.)

- Sensory – Sensory assessment is not generally performed as part of neonatal neurologic examination because it is difficult to accurately determine an infant's response to sensory stimuli. (See 'Sensory' above.)

- Behavioral – Evaluation of neonatal behavior, including consolability and habituation, assesses higher cortical functions. (See 'Behavioral evaluation' above.)

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