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Дифференциальная диагностика вялых парезов и параличей верхних конечностей у детей первых месяцев жизни (обзор литературы)

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В статье проанализированы данные литературы, посвященной вялым парезам и параличам верхних конечностей у детей первых месяцев жизни. Указанная патология представляет собой гетерогенную группу заболеваний, имеющих различный этиопатогенез: поражение спинного мозга, плечевого сплетения, периферической нервной системы до уровня плечевого сплетения, а также изолированное повреждение периферического нерва. По срокам возникновения вялые парезы и параличи можно разделить на три группы: антенатальные, интранатальные и постнатальные.

Основным механизмом возникновения данной патологии является интранатальная травма. Более редко вялые парезы и параличи верхних конечностей возникают вследствие антенатальных состояний диспластического и травматического генеза, а также постнатального поражения периферической нервной системы травматического или инфекционного генеза. Врожденные контрактуры верхних конечностей в сочетании с вялыми параличами характерны для ряда генетически детерминированных заболеваний нижнего мотонейрона и врожденных миопатий, внутриутробных повреждений плечевого сплетения и периферических нервов. В статье подробно рассмотрены топическая и дифференциальная диагностика данной патологии, клинические проявления, характерные для каждого периода жизни ребенка, и прогноз заболевания. Данное исследование полезно не только врачам-неврологам, но и специалистам смежных специальностей: врачам-ортопедам, реабилитологам, неонатологам для правильной диагностики патологического состояния, назначения адекватного лечения, а также прогнозирования его результатов.

Ключевые слова: интранатальная травма; вялый парез; паралич; парез Эрба; плечевое сплетение; периферические нервы.

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Differential diagnosis of flaccid palsy of the upper extremities in children first months after birth (Literature review)

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This article analyzes the literature related to flaccid paresis and paralysis of the upper extremities in children during the first months of life. This pathology is a heterogeneous group of diseases with different etiopathogenesis. There are various courses of flaccid paresis and paralysis of the upper extremities in children: damage to the spinal cord, brachial plexus, peripheral nervous system to the level of the brachial plexus, and isolated damage to peripheral nerves. According to the time of occurrence, flaccid paresis and paralysis can be divided into three groups: antenatal, intranatal, and postnatal pathology.

The main mechanism of occurrence of this pathology is intranatal trauma. More rare causes of flaccid paresis and paralysis of the upper extremities are antenatal conditions of dysplastic and traumatic origin, postnatal damage to the peripheral nervous system due to trauma or infection. Congenital contractures of the upper extremities combined with flaccid paralysis are connected with genetically determined diseases of the lower motor neurons and congenital myopathies, intrauterine injuries of the brachial plexus peripheral nerves. This article discusses the issues of topical and differential diagnosis of this pathology, the clinical picture suitable for each period of the child's life, and the prognosis of the disease. This research will be useful not only for neurologists, but also for specialists of related specialties: orthopedists, physiotherapists, and neonatologists for making correct the diagnosis, providing adequate treatment, and predicting its results.

Keywords: intranatal trauma; flaccid palsy; Erb's palsy; brachial plexus; peripheral nerves.

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BACKGROUND

Flaccid palsy and paralysis of the upper limbs among children in the first months of life represent a heterogeneous group of diseases with different etiologies and pathogeneses. Their occurrence can be attributed to the damage in the spinal cord, brachial plexus, peripheral nervous system to the level of the brachial plexus, and an isolated damage to the peripheral nerve.

With respect to the timing of the onset, flaccid palsy and paralysis can be divided into three groups: antenatal, intranatal, and postnatal.

An intranatal trauma of the brachial plexus is mainly responsible for this pathology in young children. Congenital contractures of the upper extremities in combination with flaccid paralysis characterize many genetically determined diseases of the lower motor neurons and congenital myopathies [1], along with intrauterine injuries of the brachial plexus and peripheral nerves [2–4]. Secondary postnatal lesion of the peripheral nerves present in the upper extremities can be a complication of both mechanical damage and infectious diseases during the neonatal period [5–10].

Antenatal injuries, as a rule, include embryofetopathies with damage to the structures of the spinal cord, brachial plexus, and nerve trunk, as well as to the joints and muscles of the upper limb (amyoplasia, congenital varicella syndrome, antenatal damage to the brachial plexus and peripheral nerves, etc.) [1, 11–17].

Amyoplasia is a classic form of congenital multiple arthrogryposis, which is characterized by damage to the motor neurons of the anterior horns of the spinal cord and multiple deformities of the limbs. The clinical presentation may resemble bilateral Erb's palsy, which is combined with congenital rigid contractures of the joints, muscle hypoplasia or aplasia, and skin retraction in the joint area [1].

Congenital varicella syndrome (embryopetopathy) is registered in about 2% of newborns from women who have had varicella during pregnancy. The typical symptoms of this pathology are as follows: zigzag-shaped scars on the skin; shortening and deformity of the limbs; and damage to the eyes, brain, palsy, and hypoplasia of the muscles of the limbs. Clinical signs of damage to various organs and systems can be expressed to varying degrees; however, some of them may be absent. The relationship of palsy with intrauterine varicella can be assumed based on the history and the presence of other manifestations. Virological data can be positive, mainly in the early postnatal period [11].

Antenatal damage to the brachial plexus is clinically manifested by a presentation of flaccid paralysis or palsy in combination with the congenital underdevelopment of the upper limb, a decrease in its size and joint contractures. Neurophysiological examination reveals signs of limb denervation in patients with this pathology in the first weeks of life, in contrast to patients with intranatal brachial plexus injury. In addition, signs of bone demineralization of the affected limb are noted in these children who are already in the early stages after birth [12, 13].

The literature presents cases of flaccid palsy and paralysis in children born by cesarean section, which is associated with intrauterine damage to the brachial plexus, but the exact pathogenesis of this pathology is yet to be established [13–15]. One of the probable causes of intrauterine damage to the brachial plexus is its ischemia, which develops because of insufficient placental blood circulation [16]. Amniotic adhesions can also cause the intrauterine compression of the brachial plexus and, consequently, the formation of flaccid palsy and paralysis of the upper limbs [17].

A simultaneous damage to the radial, ulnar, and median nerves is noted in the presence of congenital adhesions; in this case, differential diagnostics to determine damage in the brachial plexus is performed [5]. However, the characteristic skin manifestations (adhesions) along with atrophy of the extremities already at the time of birth enable the correct diagnosis during a clinical examination of the patient. According to a large number of authors, congenital amniotic adhesions accompanied by radial nerve palsy are associated with a worse prognosis than isolated neuropathy [18–21]. The literature describes cases of congenital adhesions causing muscle weakness or complete paralysis because of the compression of peripheral nerves [20–27]. H. Meyer et al. (1941) were the first to describe a newborn with a congenital adhesion on the upper limb and a "wristdrop" [24]. P.M. Weeks (1982) reported a case of ulnar, median, and radial nerve neuropathies in a patient with a congenital adhesion of the upper limb. Despite the early elimination of the adhesion, there was no significant improvement in the patient. Plastic surgery and decompression of the nerves also did not cause the restoration of function in the long term [20]. N.F. Jones et al. (2001) monitored three patients with congenital adhesions and symptoms of complete paralysis of the ulnar nerve, confirmed by electrophysiological examination. In one patient, this pathology was detected by ultrasound examination at week 18 of pregnancy. This pathology was detected immediately after birth in two children. Despite early surgical treatment, which comprised nerve grafting (two children underwent surgery at the age of three months and one child at the age of six months), the examination of patients seven months after the surgery revealed the presence of ulnar nerve neuropathy [23].

The flaccid palsy and paralysis of the upper limb in the antenatal period are caused by congenital tumors and tumor-like diseases, such as namely congenital hemangiomatosis, rhabdoid tumors, neurofibromatosis, cervical myofibroma, and the first rib exostosis, which lead to the compression of

the brachial plexus or peripheral nerves [5, 28, 29]. At the same time, the patients had no history of intranatal trauma; in some cases, palsy developed during the first month of life and had a progressive course [5, 29]. C. de Turckheim (1991) described two cases of the first rib exostosis in combination with damage to the brachial plexus and localized at the fascicle level [9].

Volkmann's congenital ischemic contracture caused by the development of compartment syndrome is a rare cause of compression-ischemic effect on the forearm nerves [30]. The cause of this pathological condition is associated with fetal hypokinesia and pathology of the fetal blood coagulation system, but the exact etiopathogenesis of the disease is unknown. As a rule, at birth, most children with neonatal compartment syndrome have edema and necrosis of skin areas, thereby indicating ischemia and the onset of the pathological process *in utero*. Determining the occlusion of the main vessels of the limb, a characteristic of neonatal gangrene, is the most crucial element of differential diagnostics in this condition. R. Ragland et al. (2005) noted residual nerve damage in 11 of 16 patients with Volkmann's congenital ischemic contracture in the long-term period after birth [30].

Congenital radial nerve palsy, which is an isolated congenital radial nerve palsy, is very rare [18, 31–42]. At the same time, damage to the brachial plexus is characterized by the high variability of outcomes; moreover, isolated radial nerve palsy is fully restored, despite the severity of clinical manifestations. Because of the rare occurrence and spontaneous recovery, the true prevalence of congenital radial nerve palsy is unknown.

X. Song, J.M. Abzug (2015) identified 55 cases of congenital radial nerve palsy [42]. The patients were characterized by changes, such as ecchymosis, depression, subcutaneous nodes, erythema, and induration, in the skin of the middle third area of the shoulder along the lateral surface. The majority of patients had changes in the skin color in the middle third area of the shoulder on the affected side, with some patients reporting subcutaneous nodules [18, 31–39, 41]. There are also known cases of the

Table 1. Differential diagnostics of flaccid palsy and paralysis of the upper limbs that developed in the antenatal period

Pathology	Clinical manifestations	Diagnostics (methods)
Amyoplasia [1]	Flaccid palsy and atrophy of the upper limb at birth, multiple joint contractures, bilateral nature	Neurophysiological (ENMG), somatosensory-evoked potentials
Congenital varicella syndrome [11]	Flaccid palsy and atrophy of the upper limb at birth, zigzag-shaped scars on the skin, shortening and deformities of the limbs, damage to the eyes and the brain	Neurophysiological (ENMG), somatosensory-evoked potentials; virological research (in the first weeks of life); clinical and anamnestic (the history of varicella in the mother during pregnancy)
Antenatal brachial plexus lesions [12–17]	Flaccid palsy and atrophy of the upper limb at birth, joint contractures	Neurophysiological (ENMG), somatosensory-evoked potentials; X-ray (osteoporosis of the bones on the affected side)
Exostosis of rib 1 [5, 9, 29]	Flaccid palsy and atrophy of the upper limb at birth or it develops in the first months of life	X-ray; neurophysiological (ENMG), somatosensory-evoked potentials
Congenital tumors and tumor-like diseases [5, 29]	Flaccid palsy and atrophy of the upper limb at birth or develops in the first months of life	Neurophysiological (ENMG), somatosensory-evoked potentials; detected through MRI; ultrasound scans
Congenital ischemic Volkmann contracture [3, 4, 30]	Edema and necrosis of the forearm skin at birth. Upper flaccid distal palsy at birth	Neurophysiological (ENMG), somatosensory-evoked potentials; dopplerography; angiography
Congenital radial nerve palsy [18, 31–42]	Upper flaccid palsy at birth. Typical skin manifestations in the middle third area of the shoulder along the lateral surface: ecchymosis, depression, subcutaneous nodes, erythema, induration	Neurophysiological (ENMG), somatosensory-evoked potentials
Congenital adhesion syndrome [5, 18–27]	Flaccid palsy and atrophy of the upper limb at birth, amniotic adhesions	Neurophysiological (ENMG), somatosensory-evoked potentials

Note. ENMG, electroneuromyography; MRI, magnetic resonance imaging; US, ultrasound examination.

spontaneous complete restoration of nerve function without treatment [19, 31]. According to F.S. Alsubhi et al. (2011), in 72% of patients, a complete recovery occurred during the treatment usually at week 8 after the birth [37].

Table 1 presents the differential diagnostics of flaccid palsy and paralysis of the upper limbs in the antenatal period.

Differential diagnostics of flaccid palsy and paralysis of the upper limbs that developed in the intranatal period

Intranatal injuries such as traumatic, ischemic, or hemorrhagic injuries to the spinal cord, nerve roots, plexuses, and peripheral nerves occur because of birth trauma and hypoxia.

Intranatal spinal cord injury is usually caused by traction, hyperextension, and rotation of the spinal cord during labor. The injury of the lower cervical and upper thoracic regions characterizes the breech presentation, whereas the upper and middle cervical regions are affected with the parietal presentation of the fetus. Acute spinal cord injury is caused by hemorrhages (predominantly epidural), intraspinal injuries, and spinal cord edema. Fractures or dislocations of the vertebrae and damage to the dura mater can rarely occur. The combination of flaccid tetraparesis with respiratory failure and low APGAR scores suggests intranatal spinal injury, which is diagnosed by exclusion. MRI or CT myelography are used as additional methods of examination that assist in determining the nature of the injury (edema, hemorrhage, or ischemia) [43–45].

Intranatal trauma to the brachial plexus is the most common cause of flaccid palsy and paralysis of the upper limbs among newborns. This type of injury can be combined with other types of injuries such as clavicle fracture (10%), shoulder fracture (10%), injury to the cervical spine (5%), spinal cord injury (<5%), palsy of the facial, hypoglossal, and recurrent laryngeal nerves, which determine the aspects of the clinical presentation of the disease (10%) [46]. The incidence of damage to the brachial plexus in childbirth

Table 2. Clinical signs of intranatal brachial plexus injury

Symptoms	Level of the brachial plexus injury		
	Upper, sometimes middle trunk of the brachial plexus, C_5–C_7 roots (Erb–Duchenne palsy/ paralysis)	**Lower brachial plexus trunk, C_8–Th_1 roots (Dejerine–Klumpke palsy/ paralysis)**	**C_5–Th_1 roots (total palsy/ Kehrer's paralysis)**
Flaccid palsy/paralysis of the muscles of the upper limb	Proximal regions: deltoid, supraspinatus/infraspinatus muscle; forearm flexors (biceps), supinators. C_7 – extensors of the forearm (triceps), fingers	Distal sections: interosseous and lumbrical muscles of the hand, finger flexors	Muscles of the proximal and distal sections
Upper limb position	Adduction and internal rotation of the shoulder	The arm hanging along the body, bird arm–shaped hand	The arm hanging along the body
Active movements	Abduction and external rotation of the shoulder are absent or limited, as well as supination and flexion of the forearm, extension of the forearm, hand and fingers is possible (C_7). Retained in the fingers	Absent or limited in the hand and fingers. Retained in the shoulder and elbow joints	Absent or limited in all joints of the upper limb
Passive movements	Free, painless		
Assessment of muscle tone	Atony or hypotension in the proximal regions	Atony or hypotension in the distal regions	Atony or hypotension of the entire limb
Deep reflexes	Absent or reduced with biceps and triceps muscles	Carporadial reflex is absent or reduced	Absent
Reflexes of newborns (congenital)	The Babkin's reflex, Moro's reflex, and grasping reflex are absent or reduced		
Sensitivity	Can be reduced along the outer surface in the proximal regions	Can be reduced along the inner surface in the distal regions	Reduced in all the regions
Concomitant symptoms	Righting torticollis, Horner's syndrome, phrenic nerve paralysis, trophic disorders on the affected side	Horner's syndrome, trophic disorders on the affected side	Horner's syndrome, trophic disorders on the affected side

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Table 3. Differential diagnostics of flaccid palsy and paralysis of the upper limb in the postnatal period

Pathology	Clinical manifestations	Diagnostics (methods)
Syndrome of central muscle hypotension in perinatal hypoxic–ischemic encephalopathy [59]	In the early period, hypoxic–ischemic brain damage can mimic the presentation of flaccid paralysis of the upper limbs in children, but it is characterized by diffuse muscle hypotension involving both the upper and lower extremities, with preserved or increased tendon and periosteal reflexes. In severe cases, the atonic infantile cerebral palsy is subsequently diagnosed	NSG, MRI, ENMG
Peripheral muscle hypotension syndrome [59]	Werdnig–Hoffmann amyotrophia type 1: generalized muscle hypotension (atony), fasciculations, absence of tendon and periosteal reflexes, decreased general motor activity	EMG, ENMG, genetic testing
	Congenital structural myopathies: generalized muscle hypotension, the absence of tendon and periosteal reflexes, muscle atrophy, structural skeletal abnormalities, decreased general motor activity, and respiratory failure	EMG, ENMG, genetic testing, muscle biopsy
	Congenital muscular dystrophies: generalized muscle hypotension and asthenia, the absence of tendon and periosteal reflexes, early muscle atrophy with fibrosis and the hypertrophy of adipose tissue	EMG, ENMG, genetic testing, muscle biopsy
	Hereditary metabolic diseases: generalized muscle hypotension, the absence of tendon and periosteal reflexes, atony, drowsiness, respiratory failure, frequent seizures, vomiting, water deprivation	EMG, ENMG, laboratory (clinical, biochemical)
Pseudoparalysis (the incidence in newborns is 5.9%) [38]	They arise because of pain or deformity of the upper limb and simulate damage to the brachial plexus. Parrot's disease in congenital syphilis: pain and limitation of passive and active movements in the joints of the upper limb are associated with multiple microfractures of the humerus	Clinical and anamnestic (maternal history of syphilis); laboratory (RW); X-ray
Fracture of the clavicle [40]	Limitation of the amplitude of active movements in the shoulder and elbow joints, edema, deformity in the clavicle, anxiety during palpation and passive movements in the shoulder joint, hand and forearm functions are not impaired, and there are no pathological positions of the upper limb	X-ray
Fracture of the humerus [40]	Lack of active movements and sharp crying during passive movements in the shoulder and elbow joints, along with deformity and swelling of tissues in the fracture area	X-ray
Osteomyelitis of bones, arthritis of the upper limb joints [40]	Pain and limitation of the range of motion in the joints of the upper limb, antalgic posture of the limb, local symptoms (edema, hyperemia, hyperthermia and tenderness of soft tissues, symptoms of intoxication, fever)	X-ray; laboratory (clinical and biochemical blood tests); bacteriological
Sprengel disease (malformation of the shoulder girdle, high position of the scapula) [40]	Asymmetry of the position of the scapula, deformity of the scapula, limitation of the amplitude of passive abduction of the upper limb. Active movements in the shoulder joint are preserved, but limited (mainly abduction)	X-ray

Note. ENMG, electroneuromyography; MRI, magnetic resonance imaging; NSG, neurosonography; EMG, electromyography.

ranges from 0.4 to 4 cases per 1000 newborns [13, 47–49]. Mild injuries are observed in 70%–92% of patients, which spontaneously recover within 1–2 years of life [13, 50]. There are three main clinical types of damage to the brachial plexus: upper Erb–Duchenne, lower Dejerine–Klumpke, and total Kehrer's damage. Table 2 presents the clinical manifestations of each of these types [40].

Other less common types of brachial plexus lesions include:

1) injury of individual fascicles or trunks of the brachial plexus, wherein the patient has weakness of one or more muscle groups of the upper limb because of the damage in the small groups of motor fibers of the brachial plexus [5, 51];
2) bilateral lesion to the brachial plexus, which, as a rule, has an asymmetric clinical presentation [5, 51–53].

In cases when the function of the biceps brachii is restored after the child reaches the age of 3 months, the limb function is rarely fully restored without the loss of muscle strength or movement in the joints. The degree of recovery of obstetric palsy also correlates with the topic of the lesion, and the proximal palsy is associated with a better outcome than total or distal ones [54].

An isolated lesion of a peripheral nerve may clinically resemble damage to the fascicles of the brachial plexus. The main clinical diagnostic criteria for this type of injury are the absence of Horner's symptoms, weakness and denervation of muscles innervated by other nerves from this segment of the spinal cord, as well as the data of electrophysiological studies (electromyography, electroneuromyography) [5]. For diagnostic purposes, electromyography, electroneuromyography, and somatosensory-evoked potentials are performed from the first days of a child's life. The indication for their implementation in early infancy is the need to clarify the level and extent of damage to the roots of the spinal cord, brachial plexus, and individual peripheral nerves [5, 7, 8].

Differential diagnostics of flaccid palsy and paralysis of the upper limbs that developed in the postnatal period

Most often in the postnatal period, the isolated palsy of the peripheral nerves of the upper limb, mainly of traumatic origin, is registered. The isolated palsy of the radial nerve, causing unilateral limitation of active movements in the hand, is noted most often. Distinctive signs of radial nerve palsy from intranatal brachial plexus injury are active movements in the shoulder, flexion in the elbow joints, and the presence of a gripping reflex without extension of the hand and fingers [1, 2, 4]. Isolated damage to the ulnar nerve most often occurs because of local trauma, and there is a decrease of strength in the adductor and abductor muscles of the fingers; in severe cases, "bird arm" deformities are manifested. However, in most cases, neurological deficits in children are resolved within 10 days to 3 months during conservative treatment [55].

Iatrogenic damage to the radial nerve is possible because of the improper placement of the BP cuff while measuring blood pressure due to its compression in patients with undifferentiated connective tissue dysplasia, as well as nerve injury during intramuscular injections into the shoulder [40, 56, 57].

The literature describes isolated cases of nontraumatic mononeuropathies. S.K. Mahapatra et al. (2014) reported four cases of lesions of the shoulder joint with sepsis in combination with neuropathy of the radial nerve. Because of the anatomical proximity of the radial nerve to the shoulder joint capsule during its inflammation, the capsule is stretched while compressing the nerve, which is clinically manifested by the neuropathy of the radial nerve. All patients had a loss of active extension of the hand and thumb, and three patients had no extension of the threephalangeal fingers. After 10–21 days from the inception of treatment, the condition improved. By day 18–35, the extension of the hand and fingers was fully restored [58].

Table 3 presents differential diagnostics of flaccid palsy and paralysis of the upper limb in the postnatal period.

CONCLUSION

When flaccid palsy and paralysis of the upper extremities are detected in the children of the first months of life, the possible causes of this pathology must be determined for deciding the approach to treatment because the outcome and prognosis of the disease depend on these decisions. An early electrophysiological examination enables the determination of the level, severity of nerve damage, and differentiate antenatal damage from intranatal and postnatal damage. Neuroimaging and laboratory studies are required in the case of an atypical clinical presentation, including lesions of the skin, subcutaneous fat, muscle atrophy, hypoplasia and contractures of the extremities, the progression of palsy, as well as the absence of a history of trauma. Because of the variability of the outcomes of palsy and paralysis of the upper limbs in young children, a multidisciplinary approach to the treatment of this pathology is used with the involvement of specialists such as neurologists, neurosurgeons, orthopedists, and rehabilitation specialists in the team.

ADDITIONAL INFORMATION

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Author contributions. *O.E. Agranovich* developed the research design, reviewed publications on the topic of the article, analyzed the material, and wrote the text of the manuscript. *G.A. Ikoeva* and *E.L. Gabbasova* performed a review of publications on the topic of the article, analyzed the material, wrote, and edited the text of the manuscript. *E.V. Petrova, V.M. Kenis, A.V. Sapogovskiy,* and *E.V. Melchenko* performed the review of publications on the topic of the article and wrote the text of the manuscript.

All authors made significant contributions to the research and preparation of the article, read, and approved the final version before publication.

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