Maternal and neonatal nerves injuries during delivery

Valentin Varlas\textsuperscript{1,2}, Vlad Dima\textsuperscript{2}, Mihaela Plotoge\textsuperscript{3}, Roxana Georgiana Bors\textsuperscript{4}

\textsuperscript{1}Department of Obstetrics and Gynecology, Filantropia Clinical Hospital, Bucharest, Romania
\textsuperscript{2}“Carol Davila” University of Medicine and Pharmacy, Bucharest, Romania
\textsuperscript{3}“Nicolae Malaxa” Clinical Hospital, Bucharest, Romania
\textsuperscript{4}Department of Neonatology, Filantropia Clinical Hospital, Bucharest, Romania

**ABSTRACT**

Peripheral nerve injuries in the mother and newborn during delivery represent two obstetrical challenges, for which we try to find the best results both from the point of view of diagnosis and therapeutic strategy. The mother’s lesions can be due to obstetric trauma and neuraxial anesthesia, while fetal injuries are mainly caused by obstetric trauma due to instrumental vaginal delivery but also secondary to abnormal presentations, macrosomia, and deficiencies regarding perinatal monitoring during spontaneous vaginal birth. In most cases, these lesions resolve spontaneously, or if they persist, conservative treatment or surgical correction is necessary. Peripheral nerve injuries in the mother and the newborn continue to remain a challenge addressed to obstetricians and neonatologists, as in-depth, randomized studies are needed to develop clinical guidelines that can be applied.

**Keywords:** peripheral nerve injuries, maternal nerve injuries, phrenic nerve injuries, lumbosacral plexus palsy, neonatal nerves injuries, neonatal brachial palsy, neonatal facial palsy

**INTRODUCTION**

Peripheral nerve injuries in obstetrics meet with a variable frequency due to poor reporting. Special attention is paid to iatrogenic injuries, which occur during delivery and associated maneuvers.

During labor and delivery, both nerve damage to the mother as well as intrinsic damage to the newborn can occur. Neurological complications due to obstetrics are 4-6 times more frequent than those due to anesthesia [1]. In nulliparous pregnant women, nerve damage occurs in cephalopelvic disproportion after prolonged labor or assisted vaginal delivery. The mechanisms of nerve damage during vaginal birth are determined either by the compressive effect exerted by the fetal head or by the motor block secondary to epidural anesthesia that makes it difficult to reposition the pregnant woman in labor. The consequence is the appearance of a focal lesion of the demyelinating nerve without axonal lesions. The latter can appear in prolonged compressions or in the case of pre-existing neuropathy. The symptoms are remitted on average after 6-8 weeks, sometimes with partial improvement [1,2].

Clinical examination and paraclinical investigations (MRI, electromyography, urological investigations) are important in specifying the diagnosis and establishing the prognosis. Correct obstetric evaluation of the pelvis, frequent repositioning of the lower extremities during labor, avoidance of prolonged labor, extreme abduction, exaggerated external rotation, and prolonged thigh flexion are prophylactic methods to avoid maternal nerve damage [3].

Neonatal palsies of the peripheral nerves (brachial plexus, facial nerve) are due to obstetric trauma encountered most frequently in spontaneous or instrumental birth and, to a lesser extent, in cesarean section.

The identification of risk factors, the increase in the rate of cesarean operations, and the training of obstetricians regarding instrumental vaginal birth and birth assistance in a pelvic presentation have decreased the incidence of obstetric paralysis.

**MATERNAL NERVES INJURIES DURING DELIVERY**

Maternal peripheral neuropathies that occur during pregnancy and in the postpartum period are secondary to the phenomena of stretching or compression on the nerves. The most common are the neuropathies of the lower extremities, Bell's palsy,
and carpal tunnel syndrome. Postpartum obstetric neuropathy occurs in approximately 1% of births [2]. They remit spontaneously, instead, the prolongation or worsening of symptoms requires additional investigations (electromyography and imaging) and appropriate treatment. In addition, comorbidities (diabetes or preeclampsia) increase the risk of nerve damage [4].

Women may have neuropathy, radiculopathy, plexopathy, or polyneuropathy during pregnancy and postpartum. Those with pre-existing neuromuscular impairments require careful monitoring and counseling regarding possible complications [5].

The mechanisms by which peripheral nerve injuries can occur include compression, stretching, direct trauma, wounds, neurotoxic substances, vascular changes that induce hypoperfusion, and then ischemia. The etiology is not known in all cases, so the most frequent situation is hypoperfusion at the level of the vasa nervorum, followed by ischemia, hemorrhage, or edema. These lead to processes of cell destruction, demyelination, and modification of axoplasmic transport, with the impairment of nerve impulse transmission directly proportional to the degree of nerve damage and the duration of the ischemic process. The risk of peripheral nerve damage can be determined by the patient's position under anesthesia, the duration of the intervention, arterial hypotension, hypovolemia, hypothermia, pre-existing peripheral neuropathy, and hydroelectrolytic disorders [6].

a) Peripheral nerve injuries encountered during anesthesia

Neurological lesions of the lower extremities secondary to neuraxial anesthesia are rare compared to obstetric ones. Common causes are direct trauma, epidural hematoma, spinal block, and very rarely epidural abscess and spinal cord ischemia [7].

During neuraxial anesthesia, the possible traumas are due to the needle's high positioning, the catheter's placement, and the anesthetic substance's injection into the nerve root or the medullary cone. Common symptoms are weakness, radicular pain, paraesthesia, numbness, or spinal cord syndrome [8]. Another complication is the epidural hematoma found especially in patients with known coagulopathy or under anticoagulant or antiplatelet therapy. If it is not an obstetric emergency, timing is preferable. Instead, in the case of severe neurological damage, surgical decompression of the hematoma is practiced [9].

b) Postpartum peripheral nerve injuries

i. Postpartum neuropathy in the lower extremities

Postpartum incidence of obstetric neuropathies does not exceed 1% of births [1]. In an observational study, Richards et al. highlighted that the sensorimotor complications in the lower extremity immediately after labor and delivery are rare and transient through the involvement of the lumbosacral nerve plexus or the sacral cutaneous nerves [10].

The symptomatology generated by these nerve injuries (femoral nerve, genitofemoral nerve, lateral femoral cutaneous, the lumbosacral plexus, and sciatic, obturator, pudendal nerves) is supported by the anamnesis, clinical examination, neuroimaging (ultrasonography, MRI, CT), and neurophysiological investigations (neurography and electromyography) [9].

The risk factors for postpartum neuropathy in the lower extremities are obesity, short stature, prolonged labor, labor positioning, nulliparity, instrumental delivery, cephalopelvic disproportion, dystocic presentations, and neuraxial anesthesia through position changes. The production mechanism is stretching or compression of the nerves, with focal demyelination processes without axonal damage. Drug therapy addresses the painful symptoms of neuropathy, being compatible with breastfeeding. Thus, local lidocaine patches and non-steroidal anti-inflammatory drugs can be used. To improve motor function, physical exercises are recommended. The prognosis is good, and the injuries recover in 6-8 weeks [9].

ii. Lumbosacral plexus injuries

Damage to the lumbosacral plexus presents a series of symptoms, such as asymmetric motor deficit, hypesthesia in the lower limbs, and sphincteric function disorders (urinary and defecation complications). Affecting the lumbosacral roots has the following causes: fetal macrosomia after prolonged labor, mid-forceps rotation, and epidural anesthesia catheter [11].

Clinical examination, MRI, electromyography, urodynamic evaluation, or anorectal manometry can investigate lumbosacral plexopathy. Urological investigations reveal a flaccid neurogenic bladder, secondary to relaxation of the detrusor muscles and continuous excitation of the internal urethral sphincter muscle. The lack of paraspinal innervation is identified by the electromyogram. EMG changes are observed approximately 2 weeks after injury. Evidence of neurological damage in the first week after birth attests to a possible pre-existing maternal neuropathy. Evaluation of anorectal function by defecography is disputed [1,12].

Anorectal incontinence due to the denervation of the anal sphincter occurs as a result of the pudendal nerve injury during vaginal birth. This mechanism can be explained by stretching the pudendal nerve or compression by the fetal head [1].

iii. Postpartum obturator neuropathy

Obturator neuropathy is very rare due to the deep protection of the nerve in the pelvis at the level of the
obturator foramen. Postpartum symptoms include weakness or pain in the groin and medial thigh, leg adduction difficulty, and reduced adductor muscle reflexes. A possible mechanism would be due to the patient’s position during regional anesthesia through the excessive angulation of the obturator nerve. The initial MRI of the lumbosacral spine is normal; later, secondary to the denervation, an increased signal is found on T2-weighted images in adductor muscles and external obturator. Electromyography shows the denervation of the large adductor muscle. The therapeutic behavior is conservative, the recovery being good [13,14].

NEONATAL NERVES INJURIES DURING DELIVERY

The incidence of neonatal nerve injuries due to obstetric causes is approximately 0.181%, 98% of which are represented by brachial plexus injuries (0.1%) and facial nerve injuries (0.074%) [15]. Rehm et al., in a retrospective study over 17 years using logistic regression analysis on 87,461 live births, identified an incidence of neonatal obstetric nerve injuries of 0.085% and a brachial plexus injury rate of 0.051% [16].

The risk factors of neonatal obstetrical paralysis are abnormal presentations, instrumental vaginal delivery, deficiencies regarding perinatal monitoring, and macrosomia. Neonatal nerve injuries (brachial plexus, facial nerve) are most frequently encountered in spontaneous or instrumental birth, but also in newborns by cesarean section, when the indication was for cephalo-pelvic disproportion or dystocic presentations [15].

c) Phrenic nerve injury

Phrenic nerve injury secondary to trauma during vaginal birth can cause paresis or paralysis of the diaphragm. It is frequently associated with paralysis of the ipsilateral brachial plexus, and, from the point of view of symptoms, it is accompanied by severe respiratory dysfunction, going up to respiratory failure in serious cases. The mechanism of occurrence is secondary to the phenomenon of stretching of the phrenic nerve that motor innervates the diaphragm [17]. Thus, the excessive lateral hyperextension of the neck (of the C3-C5 nerve roots) during birth secondary to a cephalo-pelvic disproportion, a modified pelvis, a breech presentation, or an instrumental delivery (vide extraction, forceps) is the cause of phrenic nerve injury. The coexistence of phrenic nerve and brachial plexus injury occurs in 75% of cases, being more frequent on the right side. Reiter et al. highlighted the extremely rare possibility of contralateral paralysis of the diaphragm [18].

The symptomatology is dominated by severe respiratory dysfunction and excessive activity of the normal hemidiaphragm, followed by progressive cyanosis in the case of bilateral paralysis. Late complications are caused by growth deficit, lung infections, and finally, the death of the newborn. The paraclinical investigations are represented by the chest X-ray, which observes the elevation of the affected hemidiaphragm and areas of atelectasis at the base of the ipsilateral lung.

The therapeutic management of these newborns requires either waiting to assess the degree of damage to the phrenic nerve or active management through the use of ventilatory support. After the initial assessment, ventilatory support consisted of continuous positive airway pressure (CPAP) or mechanical ventilation. In the case of severe injuries, surgical correction of the diaphragm is performed to improve the prognosis. The decision-making moment regarding therapeutic conduct is not yet fully known. Thus, Rizeq et al. report a 2% incidence of phrenic nerve injuries with diaphragmatic paralysis, of which 23% required surgical correction [17]. Surgical techniques for plication for diaphragmatic evagination used in neonates and infants are minimally invasive, laparoscopic, and thoracoscopic surgery [19]. Initially, mini-thoracotomy with plication was applied, accompanied by complications, so today, thoracoscopic surgical plication of the diaphragm is used [20,21,22,23].

This technique is reserved for severe injuries with a decrease in pulmonary ventilatory capacity and should not be applied immediately after the diagnosis of diaphragmatic paralysis because there is spontaneous remission in the first two months [24]. Other studies highlighted good respiratory results when thoracoscopic plication was performed in the first month of life, thus reducing the secondary morbidity of prolonged ventilatory support [22,25].

d) Obstetric brachial plexus lesions

Erb-Duchenne palsy is a brachial plexus lesion produced by sectioning the ventral branches of the C5-C8 spinal nerves and the T1 thoracic nerve, mainly caused by shoulder dystocia. Erb’s palsy secondary to shoulder dystocia occurs in 80% of posterior arm injuries. The lack of shoulder dystocia in the case of Erb’s palsy occurs in two-thirds of the posterior arm lesions [26,27]. Obstetric brachial paralysis has an incidence of 1.74 per 1000 live births, with equal distribution between the sexes, affecting mainly the upper part of the right arm [28,29]. The main fetal risk factor is humeral dystocia in macrosomic babies, while maternal factors include diabetes and obesity. The mechanism of producing the obstetric brachial paralysis is by blocking the anterior shoulder under the pubic symphysis of the mother, and excessive traction on the fetal head, which will cause stretching and twisting of the brachial plexus.
Anatomical classification of injuries (Sunderland, 1951) [30]:
- Grade 1 – Simple elongation with axonal integrity and rapid recovery.
- Grade 2 – Rupture of axons followed by the simultaneous contraction of agonist and antagonist muscles, with partial recovery.
- Grade 3 – Complete rupture of the nerve root without restoration of the involved territory.
- Grade 4 – Avulsion of the medullary root without the possibility of direct repair.

Obstetric paralysis of the brachial plexus depends on the type of presentation and the mode of delivery. In the cranial presentation, due to the fetal head’s excessive traction, external rotation, and the wrong handling of the shoulder, nerve stretching/rupture or nerve root avulsion can occur. In the pelvic presentation, by excessive traction of the arms in extension, with the retention of the head in the uterus, avulsions of the roots of the C5-C7 nerves can occur. A particular form of paralysis of the brachial plexus is Klumpke’s paralysis, obtained by avulsion of the roots of the nerves C8 and T1, achieved by excessive traction of the upper limb with the shoulder in abduction, the result being paralysis of the hand [31].

The physical examination after birth is particularly important for evaluating neurological lesions, the most common sign being the internal rotation of the flaccid upper limb. In newborns, muscle function is difficult to analyze, the reflex of the affected muscle being appreciated. C5, C6 plexus paralysis occurs with the arm in internal rotation without affecting the hand and wrist; the association of the C7 nerve injury is accompanied by damage to the wrist and with the fingers flexed in ulnar deviation [32].

Total paralysis of the brachial plexus with involvement of the C8 and T1 nerves presents the most severe damage in which the upper limb is limp, with an immobile hand and wrist. The classification proposed by Narakas et al. identifies four situations: I – C5-C6 involvement with paralysis of the shoulder and biceps; II – C5-C7 damage with paralysis of the shoulder, biceps, and wrist and finger extension; III – complete damage C5-T1 total paralysis of the upper limb; IV – total paralysis of the limb with radicular avulsion in the case of Claude Bernard-Horner syndrome [33,34].

Paraclinical investigations involve chest X-ray, MRI, ultrasonography, electromyography (after 3 weeks), and myelography in the case of root avulsions. The gold standard is now represented by MRI.

Most children have a good prognosis in the case of upper paralysis, which spontaneously and completely remits up to 3-4 months and is successful in the total ones. If the symptoms persist in the case of incomplete paralysis, surgical correction will be performed after 3-4 months, while in total paralysis, the correction of the sequelae can be performed after 4-5 years [31].

The treatment can be conservative (rehabilitation) or surgical. Conservative therapy combines upper limb physiotherapy to maintain muscle strength and joint mobility. The conservative treatment represented by the early immobilization of the elbow allowed a complete functional recovery in 83% of cases after a follow-up for 6 months [35]. The surgical treatment consists in correcting the nerve damage and subsequently the sequelae. In incomplete paralysis in the absence of recovery after 3 months, nerve grafting is performed. In total paralysis, nerve grafting must be performed in the case of Claude Bernard-Horner syndrome or the case of loss of sensitivity and hand motor function [36].

To improve the prognosis of these patients with brachial plexus paralysis, Wilson et al. developed a decision tree prediction algorithm to select the cases requiring surgical intervention and their timing [37].

Surgical treatment in brachial plexus paralysis is palliative due to the impossibility of achieving a complete functional correction of the paralyzed limb. The technique consists of resection of the neuroma, followed by nerve or root repair by interposition, sometimes with neurolysis of spared roots. In the case of nerve root avulsion, a nerve transfer (neurotization) is performed [38].

Reconstructive surgery in these patients is performed after a selection 3 months after birth based on the Toronto test score that quantifies the function of the upper extremity. The primary intervention consists of neuroma excision, selective motor nerve transfer close to the neuromuscular junction, and sural nerve grafting. Borschel and Clarke observed in these operated patients a decrease in perioperative morbidity rate and a functional motor recovery up to 3-4 years postoperatively [39].

Currently, reconstructive microsurgery through peripheral nerve transfers is frequently used for neuroma excision and sural nerve autograft, which have been the standard treatment for brachial plexus palsy [38,40].

Postoperative recovery begins 6-12 months and continues for 2-3 years in partial paralysis and 4-5 years in total paralysis. Sequelae surgery is applied when nerve recovery is stopped, muscle transfer is performed after 3-4 years, and bone surgery at puberty [31].

e) Neonatal facial paralysis

Facial paralysis found in newborns can be caused by obstetric trauma or congenital developmental anomalies. The incidence of facial nerve injuries varies between 0.074-0.21% [15,41]. In a retrospective study conducted on 44,292 infants, Falco and Erikson
found a 0.18% incidence of facial nerve paralysis due to forceps applications in 91% of cases, in the conditions that during the study period (1982-1987), 19% of births were instrumental. Furthermore, the recovery of newborns with traumatic facial paralysis was complete in 89% of cases. The mechanism of damage to the facial nerve is realized by its compression at the level of the stylomastoid foramen, with the appearance of temporary partial neurapraxia [42].

Currently, the prevalence of instrumental vaginal delivery is 10.3%, with geographic variations between 1.5% and 15% of all births. The proportion of forceps vs. vacuum extractor applications within instrumental vaginal delivery varied from 4:1 to 1:4, which caused a decrease in the percentage of facial nerve palsies [43]. The most common clinical form is facial asymmetry, marked deviation of the labial commissure, with feeding difficulties. The functional sequelae are quickly remitted under physical therapy procedures [41].

CONCLUSIONS

Early diagnosis and properly applied treatment of peripheral nerve injuries both in the mother and in the newborn during birth allow rapid recovery and improve the prognosis of these patients. Future research carried out on a larger number of cases will improve these patients’ quality of life.

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