Chapter

Prevention of Neurological Sequels in Infants with Perinatal Brain Damage Using Katona's Neurohabilitation Procedure

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Abstract

We aim to describe evaluations and early treatments to prevent neurological sequels in the outcome. Preterm and term infants with prenatal and perinatal risk factors for perinatal brain damage were studied. MRI examinations showed that 80% of these infants with risk factors have abnormal structural brain findings suggesting brain damage. This fact suggested that they must be treated as soon as possible. Katona's neurohabilitation procedure was described, and the results obtained with different samples of term and preterm infants showed that its application prevented neurologic sequels. The outcome for the infants between 70 and 80% was favorable. The conclusion was that infants with prenatal and perinatal risk factors for brain damage should be treated immediately.

Keywords: perinatal brain damage, prenatal risk factors, perinatal risk factors, MRI, Katona's therapy

1. Introduction

Perinatal brain damage (PBD) is a term used for brain lesions observed in term and preterm newborns. The main pathologies are hypoxic-ischemic encephalopathy (HIE) and encephalopathy of prematurity (EOP). The effects of these pathologies on the brain are very different and characterized by neurological sequels, such as cerebral palsy (CP) and other motor insufficiencies, sensorial (blindness, deafness), perceptual and cognitive problems (intellectual retardation, learning difficulties, and attention problems), and adverse neuropsychiatric development [1]. The most severe sequel is CP, characterized by motor and mental deficiencies. However, there are other motor sequels, such as deficits in coordination and minor motor difficulties. The most frequent cognitive deficit is attention deficit hyperactivity disorder (ADHD).

Evidence suggests that brain development may be particularly vulnerable to factors such as maternal nutrition, infection, and stress during pregnancy. This review discusses how maternal factors can affect brain development and outcomes in offspring.

The literature states that perinatal brain lesions and neurodevelopment problems are frequently due to prenatal and perinatal risk factors [2, 3]. Chorioamnionitis has been associated with a higher risk for hypoxic-ischemic encephalopathy (HIE) and cerebral palsy (CP) in both preterm and term-born neonates. In addition, maternal stress and inflammation during any trimester, in the absence of direct fetal brain infection, can negatively affect neurodevelopmental outcomes [4].

Unfortunately, these antecedents in the presence of apparently "normal" newborns are often dismissed in the newborn period and not considered for a follow-up of the child. This occurs in many undeveloped countries or some populations of rich countries. Late clinical observations by parents or medical professionals, as delays in developmental milestones, are the first elements to diagnose the brain lesion. It is too late to make use of brain plasticity. Brain plasticity decreases after the newborn period, while the brain lesions are not yet clinically expressed (See **Figure 1**).

Therefore, there is a period when brain plasticity is high, and the lesion has not been expressed to begin the therapy. This period is used in Katona's therapy [5].

To dismiss the presence of antecedents of risk factors and begin therapy once the lesion is clinically expressed (delayed presence of developmental milestones) are one of the causes of the high rate of neurologic disabilities due to these factors. A report on the global prevalence of disabilities among children and adolescents [6] born before 33 weeks of gestation indicates that 291.3 million (11.3%) children younger than 20 years have mild-to-severe disabilities, according to [7].

Perinatal brain injury affects infants born at all gestational ages, but its incidence and morbidity increase with decreasing gestational age [8–10]. In another meta-analysis, preterm infants were found at an increased risk for language, cognitive, sensory, and motor deficits [11]. Other causes of brain traumas are due to improper obstetric instrumental techniques such as forceps delivery and vacuum extraction. These procedures increase the ease of the descendant baby during labor but may produce brain injuries [12, 13]. These obstetric errors may be attributable to poor health services and lack of mothers' education.

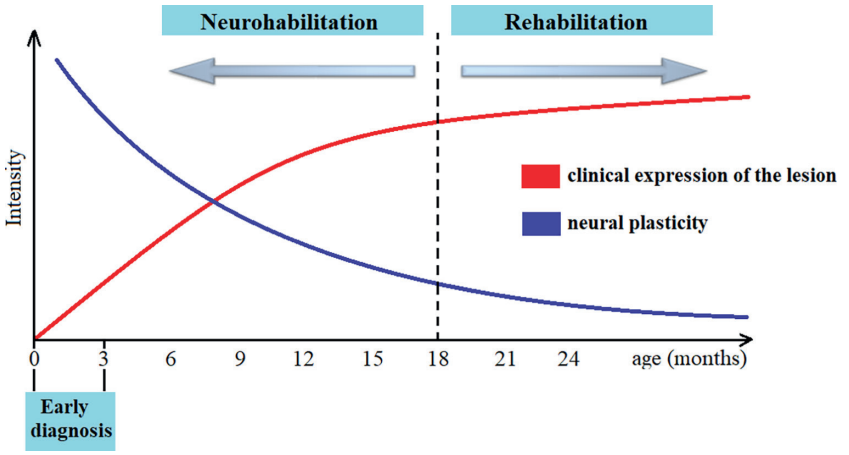

Figure 1.
This figure shows the trend of brain plasticity (red line) and the clinical expression of the lesion (blue line). The x-axis shows the infant's age in months, and the y-axis shows the intensity of these processes. During the first three months, the diagnosis or findings of antecedents of prenatal or perinatal risk factors for brain damage should be made. Katona's neurohabilitation therapy should begin, which ends when the infant's independent walking is reached.

1.1 Prenatal and perinatal risk factors

In recent years, the survival of preterm newborns has increased due to advanced medical procedures; however, this has resulted in a rising number of infants with long-term developmental problems. Preterm infants are at considerably increased risk of mortality as well as respiratory and non-respiratory morbidity. Equally, there is evidence that these infants may be at increased risk of long-term neurocognitive and behavioral problems and reduced school performance [14].

In a recent review by [4], acquired brain injuries during the different trimesters of pregnancy and in the postnatal brain are referred to. White matter injury and germinal matrix hemorrhage/intraventricular hemorrhage in preterm infants and hypoxic-ischemic encephalopathy in term infants are the main pathologies studied by these authors. During the first trimester, it is very important to ensure maternal health. Infections may affect the placenta and fetal neurodevelopment, producing neural tube defects in 0.74 per 1000 births. Folate deficiency is the main nutritional risk factor for neural tube defects, which can be prevented by prenatal folate intake. During the second trimester, the cerebral cortex is formed, and insults such as maternal alcohol use, tobacco, or other drugs of abuse in utero may produce cortical structural alterations. These facts may also alter the development of the corpus callosum. In the third trimester, myelination, synaptogenesis, and axonal pruning begin. Fetal brain infarcts or hemorrhages produce severe structural defects. Maternal infections can lead to neonatal encephalopathy [4].

Prolonged labor lasting more than 20 hours for the first delivery and more than 14 hours in those who have previously given birth, fetal distress, and perinatal asphyxia are other factors that may produce fetal brain deficiencies. Congenital neural tube anomalies are also due to multifactorial causes, including prenatal and perinatal risk factors [15]. Other important prenatal and perinatal risk factors for perinatal brain damage are the mother's age, previous abortions, toxemia, growth restriction in utero, lower weight at birth for the gestational age, congenital heart anomalies, anemia, necrotizing enterocolitis, and respiratory distress [16].

Lower Apgar Score (<7) affects neurodevelopment in infants born small for gestational age [17], and it is used routinely to detect problems in newborns.

In 2017, [18] published a Table of prenatal and perinatal risk factors in 262 infants from 25 to 40 weeks of gestational age (WGA). In the groups of 25 to 27 and 28 to 29 WGA, the most frequent factors were sepsis, asphyxia, and very low weight at birth (VLW). From 30 to 31 WGA, toxemia, VLW, and metabolic problems were the most frequent. Maternal infections, VLW, asphyxia, and sepsis were frequently observed between 32 and 34 WGA. At term, maternal infections and asphyxia were the most frequent risk factors.

We may conclude that following mothers during gestation and newborns during labor is very important. Now, according to [19], "pregnancy-related deaths and diseases remain unacceptably high. In 2015, an estimated 303,000 women died from pregnancy-related causes, 2.7 million babies died during the first 28 days of life, and 2.6 million babies were stillborn. While substantial progress has been made over the past two decades, increased access to, and use of, higher-quality health care during pregnancy and childbirth can prevent many of these deaths and diseases, as well as improve women and adolescent girls' experience of pregnancy and childbirth. Globally, however, only 64% of women receive antenatal care four or more times throughout their pregnancy".

1.2 MRI findings

The term "encephalopathy of prematurity" refers to encephalic gray matter abnormalities and WMA in preterm infants during the perinatal period [20]. Periventricular Leukoencephalopathy (PVL) and neuronal axonal injury are the hallmarks of this condition [21, 22].

Enlarged extracerebral space is a relatively common abnormal finding. In 2003, [23] found that very preterm infants with moderate-to-severe disability at 12 months of age (based on clinical examination) had reduced cortical and subcortical gray matter volumes and increased cerebrospinal fluid volumes compared to infants without disability or with mild disability. Enlarged extracerebral space is a relatively common abnormal finding. Anderson [24] described that in the 1998–2000 Christchurch cohort, 51% of very preterm infants had a mild enlargement of the extracerebral space, while 17% had a moderate-to-severe enlargement **Figure 2**.

MRI findings in extremely and very preterm showed that 50–80% have diffuse white matter abnormalities (WMA), which have been related to significant neurological and psychological deficits [25–27]. In preterm infants, MRI findings include diffuse and cystic white matter abnormalities, germinal matrix hemorrhage / intraventricular hemorrhage, neuronal loss, and gliosis of the gray matter (subcortical gray matter and cerebellum are the most affected). For a review visit [27].

Another characteristic of MRI in preterm babies is the volume decrease of the corpus callosum, as shown in **Figure 3**.

1.3 Katona's neurohabilitation

Katona [5] proposed the term neurohabilitation to differentiate it from rehabilitation. The main difference is that neurohabilitatory treatment should begin before the sequels of the lesion have been established, during the first 1–4 months after birth,

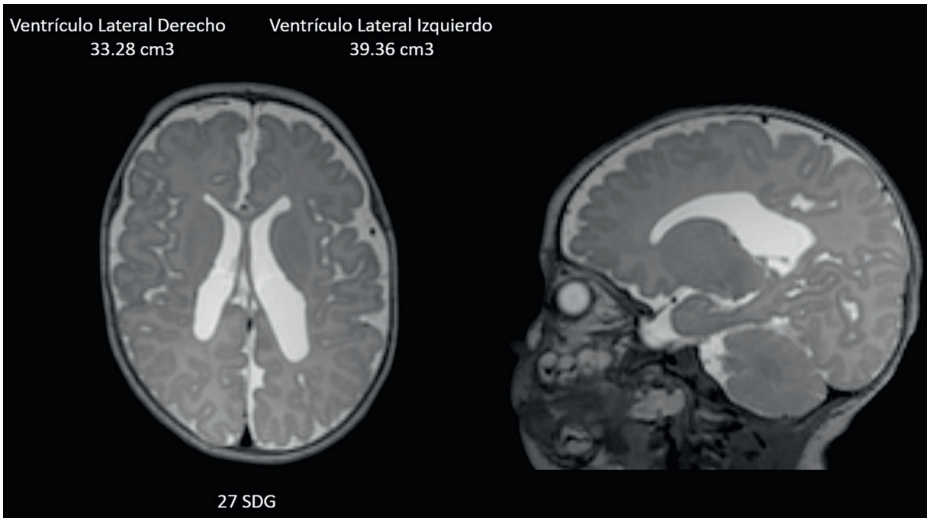

Figure 2.
Longitudinal and sagittal MRI of a preterm child of 27 weeks of gestational age. The enlargement of the lateral ventricles can be observed. The volume of the right lateral ventricle is 32.28 cm3 and 39.36 cm3 of the left lateral ventricle.

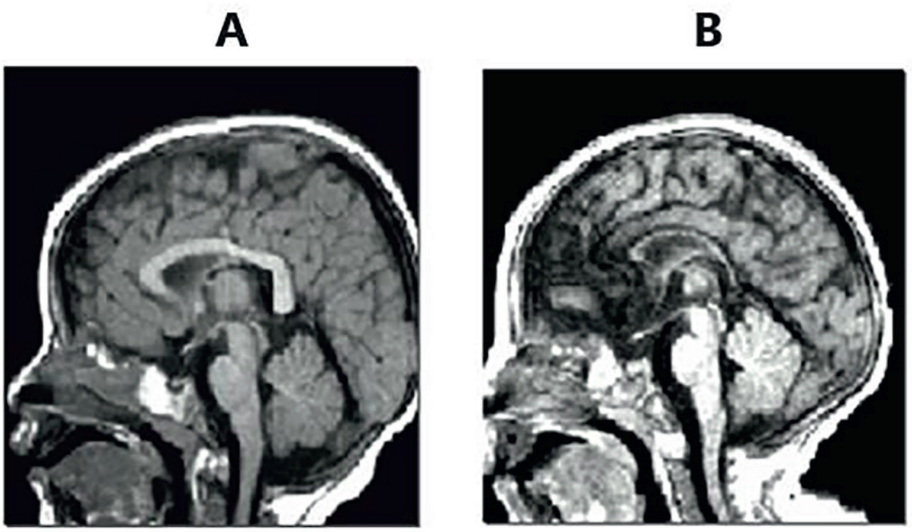

Figure 3.
Sagittal MRI of two infants at one-year-old. A corresponds to a normal child with adequate corpus callosum volume. B is the image of a preterm infant (32 weeks of gestational age) with a small corpus callosum volume.

to try to decrease or abolish the neurological sequels that perinatal brain damage may produce.

This procedure is both diagnostic and therapeutic. It evaluates muscle tone, body symmetry, attention, eye tracking, and auditory monitoring.

According to Katona [5], the therapy uses "elementary motor patterns," which are human-specific and present from 24 weeks of gestational age to 6 months. These early integrated complex movements are chains of processes in which the neck, trunk, and extremities perform complex and continual movements in certain repetitive patterns. These motor patterns have high organization, persistence, and stereotypy [28]. The developing subcortical structures control these movements. They can be activated by determined head and body positions that trigger the activation of the vestibular nuclei and their projections to the spinal cord, reticular formation, thalamus, cerebellum, and basal ganglia, whose tracts are already myelinated. Later, all these structures project into the sensorimotor cortex by myelinated axons [29]. **Figure 4** shows the neural pathways after the activation of the semicircular channels by the movements of the head.

A therapeutic program consists of training a series of neuromotor patterns each day for a certain time. Katon described 40 maneuvers to trigger the elementary motor patterns. The different positions to generate the specific neuromotor patterns are described in [30]. The repeated generation of these movements produces brain engrams that improve motor development. At the initiation of the treatment, specialized therapists conducted the Katona evaluations to obtain a diagnosis and to program 5 to 6 maneuvers that parents should learn to apply to their infants at home during the first month of therapy. After a month, the therapist evaluates the infant and selects the maneuvers for the next month, and this happens every month until the infant reaches independent walking. The therapy is intensive and specific for each infant.

Three to five maneuvers (as shown in **Figure 5**) are repeated five to six times in one therapeutic session that lasts 45 minutes and should be repeated 3 or 5 times daily. Parents learn how to perform the exercises correctly since they will treat the infant at

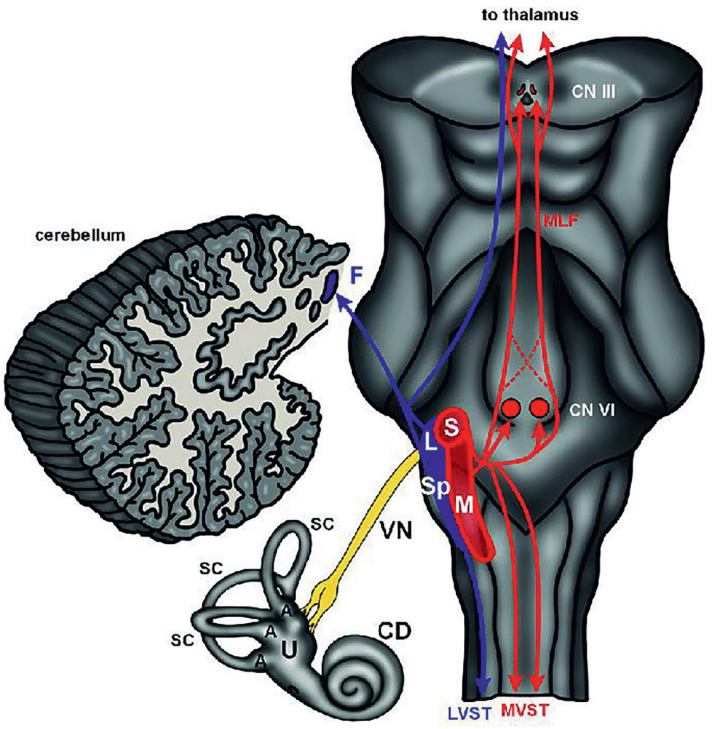

Figure 4.
Different positions of the head activate the semicircular channels (SC) that send afferent fibers by the VIII pair (Vestibular nerve, VN) to the vestibular nuclei (L, SP, S, M) in the medulla oblongata. These nuclei send descendant fibers to the spinal cord (LVST and MVST) and ascendant fibers to the nuclei of the abducens nuclei, the oculomotor nuclei, the cerebellum, and the thalamus. The Centro median thalamic nuclei send projections to the striatum and the cortex.

home. Therapy must be integrated into the infant's schedule and divided into periods according to sleeping and wakefulness patterns and feeding and nursing schedules. Each month, the infant was examined, evaluating both motor performance and visual and auditory attention and the ages at which the infant mastered various developmental milestones **Figures** 5 and **6**.

2. Results

In the Neurodevelopmental Research Unit, where we work with infants, we perform multidisciplinary evaluations of the infants. The inclusion criteria are a corrected age (CA) of 2 months or less and prenatal and/or perinatal risk factors for brain damage. The exclusion criteria are genetic factors associated with brain damage, cardiovascular pathology, brain malformations, and/or chromosomal aberrations. In the first step, a pediatrician confirms these criteria. Immediately and in parallel, the multidisciplinary evaluations (pediatric, neuropediatric, psychological, electrophysiological (EEG, visual, and auditory evoked potentials), and brain MRI) and Katona's therapy begin. We follow up with infants up to 8 years old. During this period, several multidisciplinary evaluations are done. If, in those evaluations, some motor, sensory, and/ or cognitive incapacity is detected, it is immediately treated. **Figure** 7 shows the different steps.

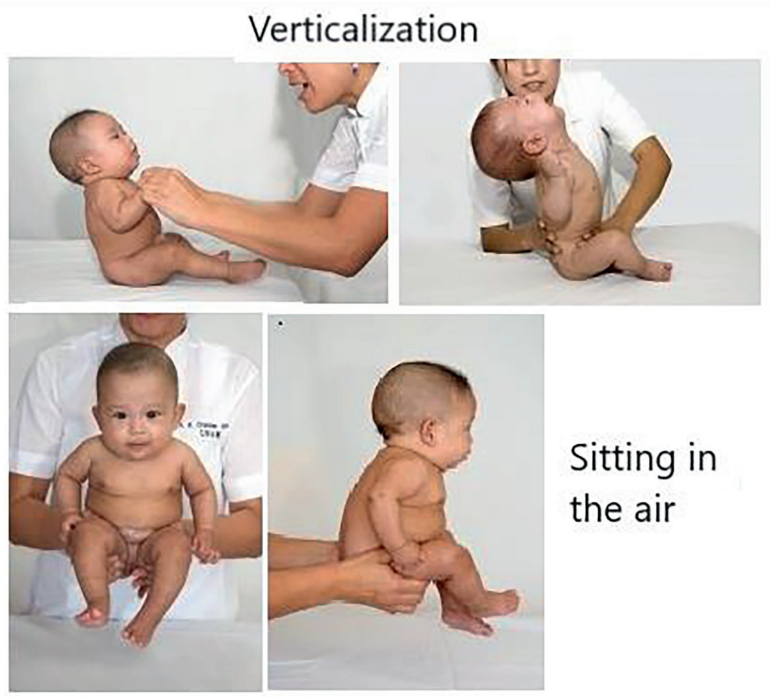

Figure 5.
Katona's positions for verticalization of the body. In the upper images are shown how the therapist should place his/her hands to activate the vertical position of the baby. The lower images show the other hand positions in the inferior extremities of the baby to obtain the "sitting in the air" position.

Figure 6.
Positions of the baby and hands of the therapist to improve crawling in a horizontal plane and in an ascendent ramp. It is also shown how the therapist helps the infant up a step.

The results of applying Katona's neurohabilitation have shown in several infant samples that it prevents neurological and cognitive sequels in infants with prenatal and perinatal risk factors [31–35].

Harmony [18] studied a group of 262 infants from 25 to 40 WGA with prenatal and perinatal risk factors for brain damage. In this group, the MRI showed that 80% of the subjects had some abnormal findings, particularly increased volumes of the subarachnoidal space and the lateral ventricles. Decreased volumes of the corpus callosum were also observed. Application of Katona's methodology showed that at three years old, the Mental (MDI) and Psychomotor Developmental (PDI) Indices were normal at 62% and 80%, respectively. In this sample, if those infants with slight delay were added to the normal infants, around 90% of subjects could have appropriate behavior among their peers. See **Figure 8** for these results.

In another publication related to prenatal and perinatal risk factors, where 82% of the infants had some abnormal MRI findings, we used Katona's procedure. The outcome of children at eight years old showed that 78, 76, and 78% of extremely preterm, very preterm, and late preterm, respectively, had normal neurodevelopment [36].

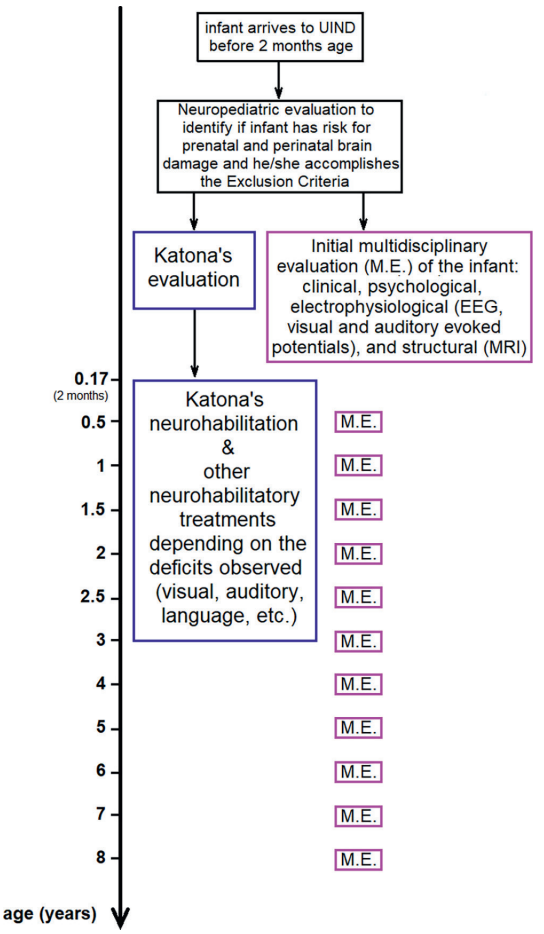

Figure 7.
This figure shows the main steps followed by the Developmental Research Unit (Unidad de Investigación en Neurodesarrollo, UIND) for the multidisciplinary evaluation and treatment of infants and their follow-up.

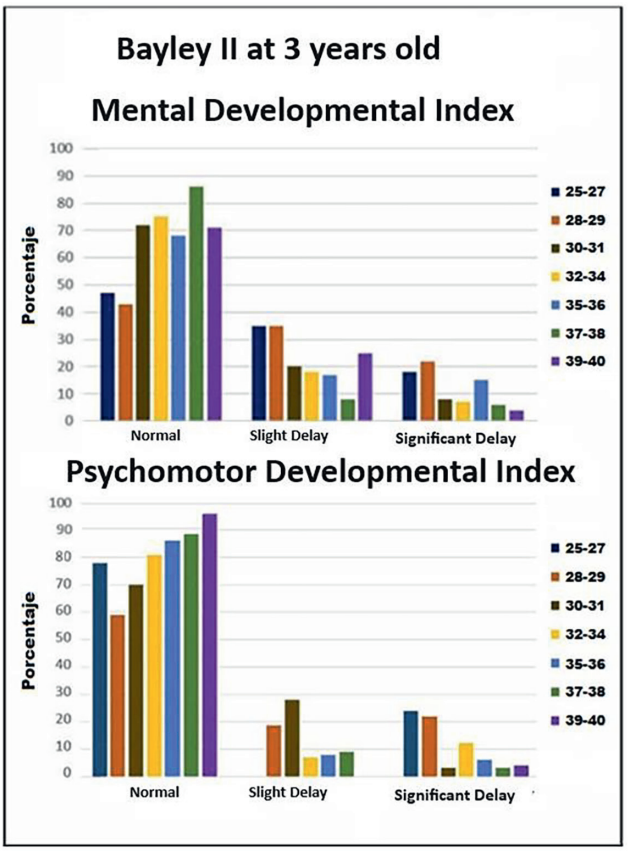

Figure 8.
Results of Bayley's II test at 3 years old of 262 preterm infants who followed Katona's therapy from 2 months to 24 months. The colors indicate the gestational age at birth in the right column. If indices of all infants (25 to 40 weeks of gestational age) were added, the total Mental Developmental Index is Normal + Accelerated = 68% + Mildly delayed 89%. The total Psychomotor Developmental Index is Normal + Accelerated = 80% + Mildly delayed 91%.

In a recent publication, we compare the outcome of 166 preterm infants with prenatal and perinatal risk factors and MRI showing structural abnormalities in 87% of the infants that were treated with Katona's procedure and a group of infants with similar brain abnormalities where parents did not approve the treatment. The parents of 128 infants accepted and received Katona's neurohabilitation treatment. The remaining 38 infants did not receive treatment. Bayley's II scales were applied in both groups at three years old. The treated infants showed normal values (100 on the MDI scale and 104 on the PDI scale), and the nontreated children were 79 on the MDI and 81 on the PDI. The differences between groups were very significant [16].

3. Discussion

The application of Katona's neurohabilitation is based on the work at home by their parents or somebody who, for at least one year, dedicated the whole day to attending the infant in care. This is necessary for the infant to receive 3 to 4

stimulation periods with the method each day. The method should continue once the infant walks independently. The exit of treatment is due to regularly administered sessions of 45 minutes several times a day. If this is not accomplished, the neurodevelopmental outcome will not be reasonable. It is important to give feedback to the parents in each evaluation. If the therapist observes that the infant is not developing reasonably, he/she should discuss the child's future with the parent engaged, which depends on adequate adhesion to the therapy.

Barrera [30] published a Spanish-language manual for this therapy, including exercise instructions. This manual has been used in our laboratory with exit.

4. Conclusions

1. Newborns with prenatal and/or perinatal risk factors have, in 80% of the cases, abnormal MRI findings suggesting brain injury.

2. Therefore, all infants with this type of risk factor should be treated immediately, as soon as possible, to decrease neurologic sequels.

3. Katona's therapy has shown to be useful in the prevention of neurological sequels in preterm babies, as well as in term infants with perinatal brain damage.

4. Katona's therapy depends on the parents' work or someone dedicated to the infant until the infant walks independently.

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