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I. NEONATOLOGY

A FORM OF DANDY-WALKER SYNDROME AS A PART OF A MULTIPLE MALFORMATIVE SYNDROME. LIMITATIONS OF THE ULTRASOUND SCAN.

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Abstract

The most impressive issue of this special case is that although standard ultrasound scan correctly recommended and performed during the pregnancy do not always identify some of the cerebral malformations. When dealing with this specific type of malformations, they can “escape” the prenatal ultrasound screening and therefore they get diagnosed postnatally.

In this particular case the hydrocephalus was identified not only through a clinical examination or ultrasound scan but mostly by magnetic resonance.

Key-words: Dandy-Walker Syndrome, ultrasound scan.

History

First carrying 32 years old woman who followed precisely the doctor's recommendations regarding medical examinations, clinical follow-up and ultrasound scans during the pregnancy; the risk score for the 21st trisomy ranged 1.1674 (embriofetal biometry, free serological β HCG and α FP).

The ultrasound scan performed at 20 weeks of gestation did not identify any foetal malformations, the intracranial structures appeared normal, the cerebellum's vermix was obvious (while using the standard incidence), the cisterna magna seized 6 mm, normal figure of the ventricles.(fig.1)



Fig. 1. Ultrasound scan at 20 weeks of gestation – normal aspect of the ventricles.

At 31 weeks of gestation there is noticed a premature imminence of the delivery. That was successfully stopped using tocolisis, uterosedatives and antispastics drugs. Accordingly, it was decided to administer corticosteroids in order to prevent the hyaline membrane disease (Celestone VI f). On the ultrasound scan there was evident a bordered placenta praevia.

Birth: at 33 weeks of gestation a minor bleeding episode intervened being shortly after followed by a massive haemorrhage. The caesarean section was the best

choice of that moment and so it was performed as an emergency. There it was, a new born baby girl, weighing 2140 grams, Apgar scored 6/7 at 5 minutes who was in obvious need of bag-and-mask ventilation using 100% O₂ than 40%O₂. The obstetrician confirmed his suspicion of accrete bordered placenta praevia.

The new born baby was admitted in the Neonatal Intensive Care Unit undergoing continuous monitoring. The first chest X-ray performed soon after birth showed a fixed lifting of the right hemi diaphragm which rose the question

of a possible congenital paresis of the right frenic nerve. The peribronchovascular image sustained a lack of amniotic fluid resorbption (considered at that time as a neonatal transient tachypnea). The baby's condition aged 24 hours was worsening. She was experiencing a moderate respiratory distress syndrome (scoring 4/5 on Silverman scale) and so the neonatologist decided to intubate her for starting conventional mechanical ventilation. The chest X-ray performed in this stage undoubtedly showed a right

pneumothorax and pneumomediastine (fig.2,3,4). The symptoms could be kept under control while receiving the mechanical ventilation via the endotracheal tube and while performing the aspiration puncture on the right hemidiaphragm. But not for so long and there was a need for a second puncture. At 4 days of age, undergoing conventional mechanical ventilation and pleural drainage, there were no signs of respiratory distress whatsoever.



Fig. 2,3,4. Right pneumothorax and pneumomediastine

Starting the 2nd week of life, there was noticed a progressive increase of the cranial circumference, from 31,5 cm at birth to 34 cm ageing 2 weeks, 35 cm at 3 weeks of age and 40 cm at 8 weeks of age.

Transfontanellar ultrasound (baby girl aged 8 days): well designed cerebral tissue, right lateral ventricle

21 mm, left lateral ventricle 19 mm, with nonomogenous transsonic insights, anterior horns with hypereccogenous walls, moderately dilated 3rd ventricle with massa intermedia present. All of the above stand for evolutive bilateral hydrocephalus (moderate form). No signs of intraventricular hemorrhage.(fig.5)

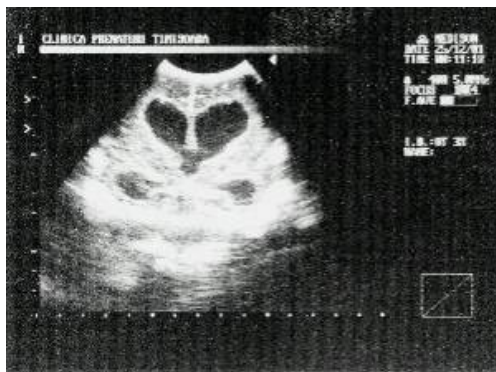


Fig. 5. Transfontanellar ultrasound – no signs of intraventricular hemorrhage.

Hydrocephalus is most frequently due to Sylvius's aqueduct stenosis, obstruction of the foramina of Luschka or Magendie. It is seldom related to an increase secretion of cerebral spinal fluid, to a decreased reabsorption in the subarachnoid space or to an arterial-venous malformation. Isolated hydrocephalus is more often caused by a congenital obstruction of Sylvius's aqueduct which makes the connection between the 3rd and the 4th ventricles. Hydrocephalus is a common feature of meningocele associated with Arnold Chiari malformation. When detected postnatal, the hydrocephalus might be following a bleeding process or a cerebral infectious episode.

First evaluation:

- Premature baby girl (gestational age 33 weeks)

- Caesarean section for bordered placenta praevia
- Respiratory distress syndrome-neonatal transient tachypnea
- Pneumothorax secondary to the therapy and to the right frenic nerve paralysis
- Evolutive hydrocephalus starting the 2nd week of life; first considered as a post bleeding process and then confirmed by the lumbar puncture (cerebral spinal fluid analysis) and the transfontanellar ultrasound

Question to be raised at this point: early postnatal evolving hydrocephalus associated to the unilateral frenic nerve paralysis might conclude to a complex syndrome? In order to answer to this question (baby's age being 3 weeks), it was asked for a magnetic resonance imagery (severe

ectasies of the intracerebrum liquid spaces on bilateral overtentorium level with obvious compression on the cerebrum tissues; possible identification of an arterial-venous malformation of approximately 3/3/2 cm at the

cortico / subcortical level, just anterior to the frontal corn of the right lateral ventricle; drainage vessels and bloody regional clots- using T₁ hypo signal, T₁ hyper signal)(fig.6)

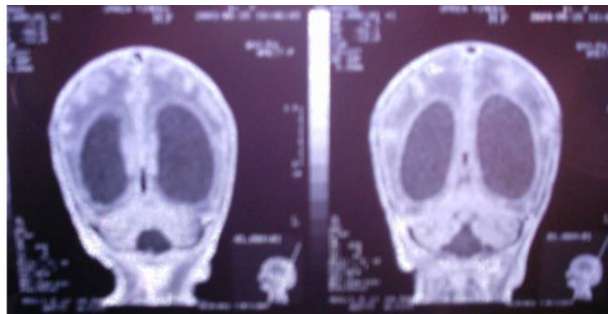


Fig. 6. Magnetic resonance aspect.

▪ Hypoplastic inferior cerebellum's vermix with moderate ectasies of the 4th ventricle and it's large communication with cisterna magna; low insertion of the tentorium; intracerebral vessels- slightly ectasies, sinuous shaped; hypophysis gland and orbits-normal (form of Dandy-Walker Syndrome)

Conclusions

1. severe triventricular hydrocephalus
2. right arterial-venous malformation with bloody regional clots (delayed sub acute status)
3. possible form of the Dandy-Walker Syndrome – partially agenesis of the anterior and inferior sector of the cerebellum's vermix

4. postnatal diagnosis via magnetic resonance imagery
5. standard incidences on the prenatal ultrasound scans of the cerebellum **DO NOT IDENTIFY** this possibility
6. an increasing need of adopting new incidences over the cerebellum while performing the prenatal ultrasound scan which might be borrowed from the magnetic resonance techniques (including transverse and oblique subcerebellar incidences)
7. **THE TRANSFONTANELLAR ULTRASOUND HAS IT'S OWN LIMITATIONS** related to the equipment itself, the techniques used, the examiner's abilities and of course the anatomical peculiarities identifiable in every baby at a certain age.

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NEONATAL HEPATITIS WITH CYTOMEGALOVIRUS

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Abstract

Hepatic disorder is frequent during congenital infection with cytomegalovirus. The clinical expression ranges from subclinical to serious, even fatal forms of disease. The hepatic manifestations can dominate the clinical symptomatology or, within the multisystemic involvement clinical or only biochemical signs of hepatitis can occur. The evolutive forms of disease, progressing towards the chronic stage, have an important potential towards chir.

Key words: cytomegalvirus infection, neonatal hepatitis

Introduction

Infection with cytomegalovirus (CMV) represents one of the most frequent congenital infection in Europe, with a rate of 4-5 to 1,000 births.

The newborn liver is particularly predisposed to transplacental or perinatal infection with CMV, whereas in other ages the hepatic disease occurs rarely and only in certain circumstances (e.g. immunity deficiency)

Hepatic disorder is mainly manifested as a hepatocellular disease (hepatitis), but the CMV infection has been associated, in many studies, with three other clinical-pathological distinct entities:

- inflammation and reduplication of the biliary duct in the portal spaces, which in some circumstances results in paucity of the biliary duct;
- lesions of the main intrahepatic biliary ducts with evolution towards sclerosing cholangitis;
- diseases of the extrahepatic biliary ducts, mainly biliary atresia (2)

In all these cases there is a stereotype response, neonatal cholestasis, which creates difficulties in diagnosis, and the primary pathological impact can be common, respectively an inflammatory cholangiopathic process, with various degrees of involving the hepatocytes and the biliary ducts.

Aim of study

The authors target the evaluation of hepatic involvement in the congenital infection with CMV, as well as the study of clinical manifestations, of the biological modifications and of the evolution in the neonatal hepatitis with CMV.

Material and method

A number of 11 newborns were under study, that had been diagnosed with maternal-fetal infection with CMV, in the Maternity of Reșita Hospital, during 1996-2004.

The serological diagnosis of the congenital infection with CMV was carried out by determining the IgM type CMV antibodies, with the ELISA method. In 2 cases the diagnosis was confirmed by the detection of pp65 antigen, specifically CMV in leucocytes and the isolation of the CMV infected cells in urine. The presence at birth of the IgM type CMV antibodies indicated the intrauterine infection. Diagnosis of neonatal hepatitis with CMV was based on clinically documented hepatopathy and/or by altering the hepatic tests.

Results

Out of the 11 analysed cases, 8 presented clinical symptoms at birth or during the early neonatal period and 3 cases were asymptomatic, the diagnosis being exclusively serological, based on the presence of CMV IgM type antibodies. The asymptomatic cases have been included in the study for the following up of subclinical forms of disease.

In order to study the occurrence of the hepatic clinical manifestations in the CMV congenital infection, the 8 cases of children with symptomatic congenital infection were analysed.

The clinical signs in the CMV congenital infection were: jaundice, in 5 cases (62,50%), hepatomegaly present in 5 cases (62,50%), splenomegaly in 3 cases (37,50%) hypoxic-ischaemic encephalopathy in 3 cases (37,50%) convulsions in 1 case (12,50%) petechiae in 4 cases (50%) ascites in 1 case (12,50%) the hemorrhagipary syndrome 1 case (12,50%), microcephaly in 1 case (12,50%), chorioretinitis in 3 cases (37,50%), pneumonia in 3 cases (37,50%). The clinical signs of neonatal hepatitis were present in 5 of the 8 cases of transplacental infection with CMV

For the study of subclinical forms of disease, 1 asymptomatic case was included in the study, case where the hepatic disease was proved by altering the biological hepatic tests. According to the cases' genders, age of pregnancy, Apgar score, birth weight, it comes out that the male gender was affected to a degree of 66.66%. According to the age of pregnancy, as determined by anamnesis and Ballard score, 16,66% were born premature. According to the placement on the intra-uterine growing curves, 5 newborns (83,33%) were appropriate for gestational age, 1 children (16,66%) was small for gestational age. According to the Apgar score, 2 cases (33,33%) presented obstetrical asphyxia.

Clinical manifestations in neonatal hepatitis with CMV

Jaundice was present in 5 cases. The early onset of jaundice was present in 2 cases: 1 case in the first 24 hours,

respectively, 1 case in the first 36 hours. In most of the cases, the jaundice was a colestatic type (4/5), of medium intensity and associating hyperchrome urine (2 cases), and hypocolic stools (1 case). In all cases, jaundice was prolonged, with a winding evolution present in 2 cases and progressive evolution, towards worsening, noticed in 2 cases. Hepatomegaly was present in 5 cases, associated with splenomegaly in 3 cases. The associated digestive

symptoms were: biliary vomiting, difficulties in sucking, low digestive tolerance, anorexia, abdominal distension with meteorism. Within the systemic disease, the hepatic symptoms were associated in 2 of the cases with pathological neurological symptomatology: hypotonia, apnea, convulsions. Other general symptoms: marble-like skin, fever were also assessed.

Table1. Clinical findings in CMV neonatal hepatitis

Symptoms	Nr. of cases	Occurrence
Jaundice		
-average intensity	4	80%
-intens	1	20%
Hepatomegaly		
< 2 cm	2	40%
2-4 cm	2	40%
> 4 cm	1	20%
Splenomegaly	3	60%
Hypocolic stools	1	20%
Hyperchrome urine	2	40%
Hemorrhagipary syndrome	1	20%
Ascites	1	20%
Biliary vomiting	1	20%

Hepatic biological investigations in the neonatal hepatitis with CMV

- Hepatocytolytic syndrome

Hepatocytolysis was pointed out by high levels of serum transaminase (ALAT/SGPT and ASAT/SGOT) in all the studied cases. In 4 cases mild elevation of transaminase were noticed, important cytolysis (TGP over 200 UI/l, upon repeated analysis) occurred in 1 case and was associated

with intense jaundice, important hepatosplenomegaly and colestasis. Slightly increased levels of transaminase (1.5xN) were recorded in 1 asymptomatic case. Minor increased levels of transaminasis, upon repeated analysis, associated with slightly modified levels of direct bilirubin, led, by means of additional investigation, to diagnosis of clinically asymptomatic transplacental CMV infection. (table 2)

Table 2. Hepatocytolytic syndrome

SGPT			SGOT		
<50 UI/l	50-100UI/l	>100UI/l	<70UI/l	70-100UI/l	>100UI/l
-	4 66,66%	2 33,33%	-	5 83,33%	1 16,66%

- Hepatoprive syndrome

Prolongation of prothrombin time (PT) ocured in a single case, with a value of IP 83%. The hepatic synthesis deficit was proved by carrying out the Koller test (the intravenous administration of K vitamin and repeating the PT after 2 days). The hepatocellular dystrophy, affecting the capacity of synthesis, revealed the decrease in the total proteins under 60 g/l in 1 cases (proving the lowering of hepatic synthesis). The albuminemia was normal in all 6 cases. The alfa-1 globulin levels were normal in 4 cases. Even though this parameter is not considered accurate in assessing the hepatoprive syndrome, due to its unspecificity, the slightly increased values occurred in 2 cases were included in this inflammatory context.(table 3)

Table 3. Hepatoprive syndrome

P.T.	P.I	Proteines	Albumines
≥20s	≤50%	<60g/l	≤50%
1 16,66%	1 16,66%	1 16,66%	- -

- Cholestatic syndrome

The cholestatic syndrome was revealed by higher values of conjugated bilirubinemia in all cases, and mixed hyperbilirubinemia was present in 1 case. Higher values of

colestasis enzymes (alkaline phosphatase and gamma-glutamyl-transpeptidase) were noticed in 2 cases. (table 4)

Table 4. Colestatic syndrome

Bilirubin		A. Ph.	gama-GT
D> 0,3mg%	T>12mg%	>400U/L	>130U/L
6 100%	1 16,66%	2 33,33%	2 33,33%

- Immunologic investigations

The quantitative finding of immunoglobulins: IgA, IgM and IgG, according to the nephelometric procedure indicated:

- normal values for IgA in all new-born babies
- higher values for IgG in 1 new-born baby (16,66%)
- higher values for IgM in 3 new-born babies (50%).

The changes noticed at the immunoglobulins levels were interpreted as a reaction to the persistent viral stimulus, with an important IgM reaction. The determination of lymphocyte subpopulations with the Beckton-Dickinson flowcytometry in 4 cases did not indicate cellular immune deficit.

Evolution

Out of 6 infants, 4 (66.66%) presented a favourable evolution, towards healing, with the improvement of the clinical symptomatology and normalisation of the biological values until the age of 6 months. In one patient, hepatomegaly and the colestatic jaundice lasted until the age of 7 months.

The evolution towards chronicity occurred in 2 infants (33,33%), with the persistency of clinical and inflammatory laboratory signs, hepatic disfunction and cholestasis. Decease was recorded in one case (16,66%), at the age of 8 months, due to a progressive evolution towards biliary cirrhosis.

Treatment of neonatal hepatitis with CMV

1. Etiological treatment. The treatment with Ganciclovir, dosage of 6 mg/body kg, intravenous, in one

hour perfusions each 12 hours, for 6 weeks, was carried out together with the monitoring of possible side effects (especially, the myelosuppressive effect, firstly severe thrombocytopenia and neutropenia). Two cases of new-born babies with severe forms of disease, with systemic symptomatology, were administered the treatment.

2. Treatment of cholestasis. Phenobarbital was used in a dosage of 3-5 mg/day, as an inductor of mitochondrial enzymes and stimulator of the biliary flow, acting independently from the biliary acids.

3. Prevention and fighting the main complications of the prolonged cholestasis:

For lipidic malabsorption the diet was modified by lowering the long chain triglycerides and by supplementing medium chain triglycerides. Substitution of liposoluble vitamins: A vitamin (10,000 units/month, intramuscular), D3 vitamin (25-OH) 800 units/day orally), K vitamin (5-10 mg x 2/week, intramuscular), E vitamin (5-300 units/day) was performed. Administration of 0.5 g of Calcium per day was done.

Conclusions:

1. The occurrence of hepatic manifestations of the congenital CMV infection was in this study of 54,54%. The clinical forms ranged from subclinic forms, present in 16,66% of newborns, mild forms in 33,33% of the cases, medium forms in 33,33% of the cases and severe forms in 16,66% of the cases.

2. In the neonatal CMV hepatitis the hepatic clinical manifestations were associated, in 33,33% of the cases, within the systemic disease, with pathological neurologic symptoms.

3. In 66.66% of the cases the evolution of CVM hepatitis was favourable, towards healing. Improving the clinical symptomatology and normalisation of the biological values were achieved, in most cases, until the age of 6 months. The chronic evolution was noticed in 33,33% of the cases, with the persistency of clinical and inflammatory laboratory signs, hepatic disfunction and cholestasis. Decease was recorded in 16,66% of the cases at the age of 8 months, due to a progressive evolution towards biliary cirrhosis.

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PERINATAL LEUKODYSTROPHY CLINICAL CASE

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Abstract

In this study the authors want to present a case of a premature newborn who presented an extremely rare disease for medical practice. Low prevalence of the perinatal leukodystrophy, the difficulty of the clinical diagnosis and the echography resemblance with other diseases of the periventricular white matter is the subjects of this presentation.

Key words: premature newborn, perinatal leukodystrophy, echography.

Introduction

Perinatal leukodystrophy is a rare cerebral disease which is part of the cerebral white matter diseases and in most of the cases starts in prenatal period. In fact this disease represents the progressive destruction of the white matter from the very proximity of the lateral ventricles. It is hard to make the difference from other affections of the white matter, especially the periventricular leukomalacia. Both diseases are characterized by initial increasing of the cerebral echogenity followed, in the next step, by the appearance of cystic formations.

In severe forms, in both affections, the area which results from white matter destruction can be occupied in compensation by cerebrospinal fluid leading finally to a hydrocephaly *ex vacuo*.

The progressive degeneration of the cerebral tissue, during the myelinating process is rare. Detection by

MRI or transfontanellar ultrasound is difficult because normal echogenity in this period (due to high water content) is hard to differ from the pathological echogenity increasing. Therefore in this situations comparison between clinical data and ultrasound and MRI data is needed.

Case presentation

We present the case of H.A.M., female, 3 weeks old hospitalized in our department for hypotonia, rhythm and respiratory disorders, myoclonia. The new born comes from a pathological pregnancy with repeated addmitances in the hospital for bleedigs and uterine contractions. Gestational age was 31/32 weeks, birth weight 1700 g, Apgar score 7 at 1 minute and 9 at 5 minutes, born by cesarean section for breech presentation.

At birth the new born presented a generally altered state and resuscitation manuevres were needed, also oxygen input by bag-and-mask ventilation. In early neonatal period the infant presented neonatal adaption disorders.

During the hospitalization she presented generalized hypotonia, myoclonus, nystagmus. Paraclinical investigation emphasized frequent metabolic acidosis, hydroelectrolitical disorders. The serum doseing of the immunoglobulin does not reveal Ig M increasing; genetic investigation-normal karyotype. The transfontanellar ultrasound revealed the presence of inhomogeneous, wide spread, hyperechogene formations, in parietal-occipital area (Fig.1), especially in the right side, with multiple transonic areas between 3/3mm and 11/9mm (Fig.2).



Fig.1. Inhomogeneous, wide spread, hyperechogene formations, in parietal-occipital area.

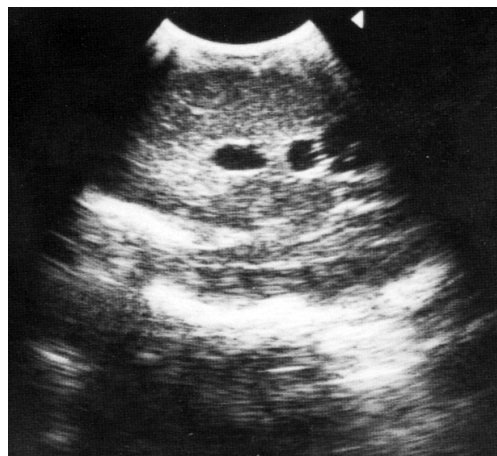
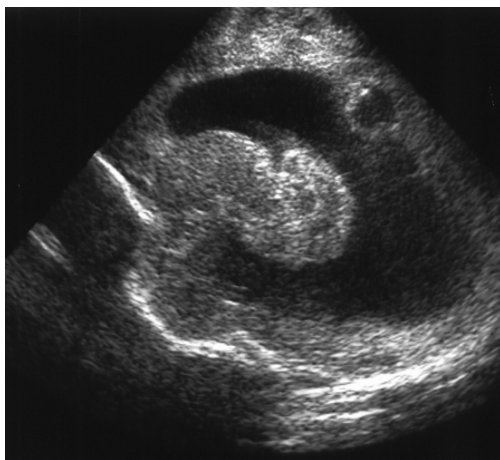


Fig.2. Multiple transonic areas between 3/3mm and 11/9mm.

Ventricular system had normal dimensions, with inhomogeneous hyperechogene formations, with transonic content within the bilateral lateral ventricles (Fig.3).



MRI was not performed yet, investigation being refused by the parents.

Fig.3. Ventricular system with transonic content within the bilateral lateral ventricles.

Discussion

Echographical changes of the white matter, intense increasing of echogeneity, are the result of a dystrophic process of the white matter occurred especially during intrauterine life. There are multiple causes of this process and there are involved genetical, infectious, hypoxic factors, isolated or associated.

Typical placement in the white matter requires differential diagnosis with several other disorders, with early start of the white matter especially periventricular leukomalacia; in perinatal leukodystrophy these echographic densities (hyperechogenities) are placed under or above the front horns of the lateral ventricles. Typical injuries are bilateral but asymmetrical (they are symmetrical only when associated with a component of focomatosis and gliomatosis). This aspect is extremely rare in periventricular leukomalacia where the typical placement of the injury is in the very proximity of the lateral ventricles walls or at the level of lateral ventricles external angles. Another different element is the persistence of the hyperechogene injuries: in perinatal leukodystrophy the stage of the hyperechogenities lasts for month while in periventricular leukomalacia lasts 3-4 weeks. Both disorders can determine a ventriculomegaly. Revealing of the fine, granular, inside the injuries calcifications suggests a chronic intrauterine infection which can be the base of the disorder (more frequent periventricular leukomalacia, extremely rare perinatal leukodystrophy).

Both affections have a resembling clinical symptomatology: hypotonia or hypertonia, seizures, growth disorders. Therefore certain establishing of the diagnosis is difficult in the clinical stage. In the case we present the prenatal antecedents and the clinical signs led us to neurological disorder. The presence of the intense hyperechogene injuries above the lateral ventricles, the persistence of this formation also hyperechogene for a period bigger than two month led us to the suspicion of perinatal leukodystrophy.

In the literature there are presented some series of myelinating disorders (leukodystrophies), which are hard to differ, based on the clinical and imagistic data, both between themselves and from periventricular leukomalacia. Generally leukodystrophies are: lysosomal storage diseases, peroxisomal disorders and diseases caused by mitochondrial dysfunction.

Lysosomal storage diseases are characterized by accumulation in the lysosomes of several substances. Based on clinical and imagistic signs the following affections can be considered as differential diagnosis in our case:

- Krabbe disease is an autosomal recessive disorder caused by a deficiency of galactocerebrosidase β -galactosidase, an enzyme that degrades cerebroside, a normal constituent of myelin. The infantile forme manifests a hyperirritability, increased tonus of muscle, myoclonus, opisthotonus and nystagmus. The disease is rapidly progressive and fatal. Hyperechogene injuries are initially seen in the area of thalamus and caudate nucleus, symmetrically, and then they spread in the periventricular white matter.
- Mucopolysaccharidosis is caused by a deficiency of the various lysosomal enzymes involved in the degradation of glycosaminoglycans. Clinically and imagistically occur various degrees of hydrocephalus, atrophy and white matter changes.

The second big category of leukodystrophies is peroxisomal dysfunctions. Differential diagnosis in our case can be Zellweger syndrome named also cerebrohepatorenal syndrome, which is an autosomal recessive disorder caused by multiple enzyme defects and characterized by neurological syndrome (hypotonia, psychomotor retardation, craniofacial dysmorphism, liver dysfunction with jaundice and cyst formations placed in renal cortical and cerebral white matter).

Leukodystrophy caused by mitochondrial dysfunctions is the third category involved in differential

diagnosis of the perinatal leukodystrophy (or possible etiology). Therefore from this category we can discuss the Leigh syndrome or the subacute necrotizing encephalomyelopathy. Clinically it appears with hypotonia, psychomotor deterioration, swallowing difficulties and ataxia. Ultrasonographic hyperechogenicity can be identified in early stage in the area of caudate nucleus and putamen while in the late stages hydrocephaly occurs associated with persistence of the hyperechogenic formations in the striated area.

Mitochondrial encephalopathy was met at the children with neonatal progeria syndrome (Wiedemann-Rautenstrauch); neurological injuries with antenatal debut and manifest with progressive and generalized ventriculomegaly determining degeneration of the white matter.

Other affections which has to be differentiate with the present case are: Aicardi-Goutieres Syndrome - is a clinical entity which contains leukodystrophy with striocerebellos calcification and subependymal cyst; Alexander disease with early debut manifested by macrocephaly, psychomotor retardation and seizures. Echogenicity is placed mostly in the white matter of the frontal lobe.

As mentioned leukodystrophy can occur as a result of a neonatal asphyxial affection and also as a result of a genetic and metabolic disease. It is difficult to make a

difference between these two types but the early start in the early neonatal period, clinical symptomatology (not so suggestive) and imaging data led us to the perinatal leukodystrophy diagnosis from hypoxic cause. Genetic and metabolic investigations could certainly decide the diagnosis but besides the karyotype – normal - they were not performed. Besides the above mentioned regarding the placement and intensity of the lesions, we can consider that the injury from leukodystrophy is deep and affects even the middle, the gyral center while in periventricular leukomalacia there is no affection of the gyral middle of the periventricular white matter.

Conclusions

- Perinatal leukodystrophy from hypoxic cause is part of the cerebral white matter diseases with early start, most of the times in antenatal period.
- Leukodystrophy (progressive destruction of the white matter) is difficult to differentiate from the periventricular leukomalacia and also from the other types of leukodystrophia, especially in genetic and metabolic diseases.
- Differentiating from periventricular leukomalacia is made on the base of the echogenicity placement, hyperechogenic injury spreading and the length of the hyperechogenicity stage, appearance of the cyst formations and their long persistence.

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SEVERE POSTASPHYXIC SYNDROME FOLLOWING A MASSIVE FETOMATERNAL BLEEDING

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Abstract

The fetomaternal bleeding might be an important cause of anaemia in the neonate. It occurs in 8% of all pregnancies and in 1% of them the volume may be as large as 50 ml. These losses may be acute or chronic. If the blood loss is recent, it may lead to a neonatal hypovolemic shock along with it's drastic consequences. If the bleeding is chronic, the baby develops an iron deficiency anaemia in which situation he is pale, the hematocrit level is low but his peripheral pulse and perfusion are good.

The normal level of hematocrit does not put aside a massive acute haemorrhage or a chronic fetomaternal bleeding on it's way to burst!

ABO incompatibility may have a masking effect over a neonatal anaemia due to the foetal bleeding. This is why it is mandatory to take into consideration this possibility.

The fetomaternal bleeding is confirmed by the Kleihauer – Betke preparation of the mother's blood. A 50 ml loss of foetal blood into the maternal circulation will show up as 1% foetal cells in the maternal circulation. Be aware that the test is considered non valid when there is evidence of a maternal hemoglobinopathy. There are some records that the foetal anaemia is frequently associated with a particular situation – the umbilical cord wrapped around the baby – and this concludes with a neonatal anaemia due to a possible compression of the umbilical vein and secondary the decrease of foetal venous blood flow.

Key words: fetomaternal bleeding, Kleihauer – Betke test.

There has to be emphasized the major role of the severe ischemia and hypoxia that come along together. They add a burden on the medical judgement in the aspects of diagnosis and therapy to be chosen. At this moment, there is necessary to pinpoint some issues:

- The diagnosis is not always revealed from the very beginning
- The complete evaluation is difficult
- The treatment is controversial
- The prognosis (ethical and medical) is complex

CASE REPORT

The baby P.I. is a male, born on the 10th of July, 2003 at 11.30 am. His birth weight was 2900gr, cranial circumference – 32 cm, thoracic circumference – 31cm and

47 cm in length. His mother, P.O. was 24 years old, with no history of other pregnancies or abortions, 0I, Rh +, married to the healthy father aged 29 years. She was a house wife and they live in the city. Her first visit to the doctor took place when she was two months pregnant and she kept on going monthly afterwards. The date of her last period was the 10th of October, 2002 so the gestational age of her baby was approximately 39 weeks. At the moment of her admittance in our hospital, she was considered to do well, having no fever, blood pressure 120/80 mmHg, the uterus's cervix was 3 cm dilated, the amniotic membranes were ruptured 3 hours prior to this moment. The obstetrician decided to continuously monitor her and her baby and started Ocitocine iv. She was closely watched during this procedure so that the foetal distress that rapidly underwent (persistent bradychardia) could be traced from the very beginning. There was no delay in deciding to put to an end to this pregnancy by performing an emergency caesarean section.

The baby boy delivered by caesarean section, head first scored Apgar as follows: 1 at 1 minute, 1 at 5 minutes, 3 at 15 minutes and 4 at 20 minutes. There were no evidences of macroscopic placental pathology (malformation or sectioning), no evidence of umbilical cord pathology and no evidence of maternal bleeding.

First evaluation:

- the baby was pale,
- non reactive, limp
- no respiratory effort, bradycardic (heart rate < 60 bpm),
- fixed mydriasis,
- no peripheral pulse, impossible to detect the blood pressure.

Gathering the information we could get so far in the delivery room, we had established the *first diagnosis*:

1. term newborn baby boy with intrauterine growth restriction
2. neonatal shock probably due to a hypovolemic process

Resuscitation in the delivery room: the baby was placed under a radiant heater (to reduce heat loss by radiation and conduction), he was then dried and wrapped in pre-warmed towel (to prevent evaporative heat loss) meanwhile assessing his colour, muscle tone, heart rate and breathing efforts. The airway was clear but yet there was no effective breathing so the baby was intubated using an

endotracheal tube. He was given inflation breaths, receiving 100% O₂ with continued support, breathing efforts were almost satisfactory but the heart rate did not respond. Giving the very slow heart rate and not increasing despite good lung inflation as judged by good chest movement, it was necessary to start chest compressions (to provide means of moving oxygenated blood from the lungs to the heart and coronary arteries). Nevertheless, these resuscitative measures were not enough to produce an increase in heart rate so we chose the next step – drugs (Adrenaline via endotracheal tube, repeatedly). In order to have a rapid venous access, there was placed an umbilical venous catheter and several boluses of normal saline 0,9% and sodium bicarbonate were pushed in.

Second evaluation (after 20 minutes of resuscitation):

- extremely pale,
- non reactive, no spontaneous breathing,
- heart rate >100 bpm, blood pressure 60/30 mmHg,
- O₂ saturation 80-85% while receiving 100% O₂ via endotracheal tube,

- Astrup parameters (arterial blood sampled): pH=7,15, P_aO₂=35, P_aCO₂=65, BE=-15, HCO₃⁻=18,
- Referred to the Neonatal Intensive Care Unit for assisted ventilation (IPPV) with the start parameters as follows: PIP=24-26-29 cm H₂O, PEEP=4cmH₂O, ET/IT=0,3/0,9, volume=7l/minut, respiratory rate=55-57rpm, FiO₂=100% (they were adjusted according to the Astrup values and the chest X-ray performed, sequentially)
- The main goal was to sustain a viable circulation for the baby giving him Dopamine + Dobutamine along with prophylactic antibiotics (Ampicilline + Gentamycine), drugs to prevent further bleeding, vitamin E and several transfusions of packed RBCs of the same group and Rh as the baby's.

Second diagnosis (after admitting in the Neonatal Intensive Care Unit):

1. term newborn baby boy with intrauterine growth restriction
2. neonatal shock- severe acute hypoxia
3. multiorganic failure
4. acute post bleeding anaemia.

Differential diagnosis between neonatal asphyxia and acute blood loss.

FEATURES	NEONATAL ASPHYXIA	ACUTE BLOOD LOSS
Heart rate	Low	High
Respiratory rate	Low	High
Intercostal retractions	Present	Absent
Colour of the skin	Pale with cyanosis	Pale, no cyanosis
Response to O ₂ or assisted ventilation	Improving	No significant changes

Third evaluation

- the neurological examination stated the 2nd / 3rd stage of SARNAT and SARNAT, the baby being lethargic/comatose, mildly hypotonic, absent complex reflexes, miosis, spontaneous respirations but occasionally apnoeic, heart rate normal/tachycardia, generalized tonic - clonic seizures (day 2).
- Because there was no identifiable blood loss coming from a placental cause, an umbilical cord pathology and no post delivery bleeding, we

targeted our attention towards a foetal bleeding.

So, there were performed additional investigations:

- Hb=9,6g%, Ht=30%, H=2630000/mm³, L=11000/mm³, BIII, Rh (-)
- Astrup –severe metabolic acidosis
- low Ca level in the serum, urea=53U/L, Creatinine=0,9mg%, Na⁺=126mEq/l, K⁺=3,88mEq/l, blood sugar level 181mg%....40mg%
- chest X-ray – cardiomegalia (cardiothoracic index>0,67), increased vascular shadow on the pulmons (Fig. 1)



Fig. 1. Chest X-ray: cardiomegalia, increased vascular shadow on the pulmons.

- transfontanellar ultrasound – ischemic encephalopathy, multifocal cortical necrosis with cerebral oedema
- the maternal blood sample is coloured using acid fluids; the foetus's erythrocytes turn dark while the mother's do not and this is why these last ones are named "phantom cells"
- the volume of fetomaternal transfusion = (number of foetal erythrocytes/number of maternal erythrocytes) x 2400

The outcome:

-day 2-extremely reduced activity, generalized seizures which did not ceased after triple anticonvulsant therapy (Phenobarbitone + Valproic Acid + Midazolam).

-day 4-stupor/comatose, absent spontaneous respirations, acute renal failure-rapidly progressing, repeatedly decreases of O₂ saturations in need of bag-and-mask ventilation and *finally cardio-respiratory arrest*.

Anatomo-pathological aspects:

- severe cerebral oedema with plane cerebral circumvolutions, interhemispheric patterns roughly recognizable; pointish haemorrhages on the cerebral mass, cerebellum, cerebral trunchus; pulmonary tissues evidenced shock, liver and spleen were affected by the stasis phenomenon; the kidneys had extended areas of necrosis on their cortex and medulla (hemorrhagic necrosis).

Conclusions:

- The fetomaternal haemorrhage with acute onset may mimic a severe obstetrical asphyxia
- Continuously monitoring the mother and her foetus was beneficial for choosing the wise moment to perform the caesarean section
- Tough target it was to identify in the delivery room the initial cause of the severe foetal distress!
- Initially, the Hb level of the baby might be slightly low because of the vasoconstrictive process

- The clinical aspects of a respiratory distress (pallor, tachycardia, tachypnea, weak pulse, low blood pressure) may as well be signs of an obstetrical asphyxia
- BE AWARE when you have a pale baby with no evidence of cyanosis, at the very beginning of a respiratory distress and KEEP IN MIND the possibility of a fetomaternal bleeding especially if the baby in his poorest condition does not respond to O₂ therapy or to conventional mechanical ventilation (high parameters)
- The poor outcome of this case may be explained by an antenatal asphyxia because of a massive acute blood loss certified by Kleihauer-Betke test
- The major 3 factors involved in the asphyxic process (hypoxia due to anaemia, hypercapnia, mixed acidosis) led to consequences affecting multiple areas: pulmonary level, cerebral level (cerebral oedema, signs of hypoxic-ischemic encephalopathy), renal tubular necrosis with acute renal failure, mesenteric hypo perfusion, lactic acidosis, glycaemia, electrolyte and thermal disorders

NOTA BENE

**this was a case of massive acute fetomaternal bleeding, demonstrated by the Kleihauer-Betke test (HbF>4% states for a foetal loss of 200ml);

**the pregnant woman was closely monitored during her labour;

**even if the foetal distress was captured from the very first possible moment, it's consequences were devastating;

**giving Ocitocin in order to sustain the labour of the mother associated with a wrapped umbilical cord might increase the blood loss from the foetus to his mother.

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II. PEDIATRICS

HALLOTHERAPY - AN ADDITIONAL METHOD IN THE TREATMENT OF THE RESPIRATORY DISEASE IN CYSTIC FIBROSIS

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Abstract

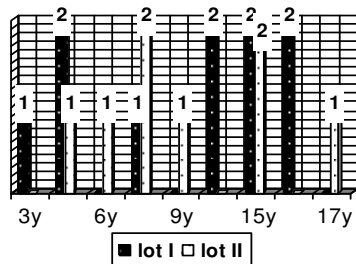
Cystic fibrosis (CF) is one of the most common lethal genetic disorders affecting Caucasian population. The respiratory disease from CF is the main factor which influences the prognosis. The concentrated NaCl solutions in aerosols as well as the courses of treatment performed in regions rich in Na ions (saline, seaside) have been observed to be beneficial for as an adjuvant in the treatment of CF. The paper present the efficiency of "Salin" device in halotherapy in CF.

Key words: cystic fibrosis, halotherapy, „Salin”, children

Premise of the paper

The respiratory disease from CF is the main factor which influences the prognosis (5). Setting up of a well organized therapeutic plan appropriate to the patient's age and clinical state, is the only chance for ameliorating the prognosis of these patients. The daily home therapy with mucolytic substances in aerosols represents one of the compulsory steps of the treatment. rh-DNAse is the best option from this point of view. Unfortunately, in the countries with a low economical standard as Romania, there is a very low possibility to have access to the rh-DNAse treatment because of the very high costs (5). The concentrated NaCl solutions in aerosols as well as the courses of treatment performed in regions rich in Na ions (saline, seaside) have been beneficial for as an adjuvant in the treatment of CF (1,2). Starting from the favorable effect of the NaCl treatment, „Tehno Bionic” – Buzau, Romania, planned and built the "Salin" device for the forced ionization of the indoor air. Principle of the method: *forced passing of the air through microcrystal salt deposit plates.*

Fig. 1 The age of patients



This procedure leads to changes of the air composition and quality by salt sublimation (4,6).

Aim of the paper

The aim of this paper is to check the therapy's efficiency upon the patients with CF and chronic respiratory disease by forced ionization of the indoor air.

Material and method

This study has been realized within a 6 months interval on two lots of CF children and teenagers followed up by the Center of CF from Timisoara (Fig. 1).

Lot I: 10 patients (4 male and 6 female) with their age between 3 and 16 years (average 10,3 years) where we applied forced ionization of the indoor air (living room, bedroom).

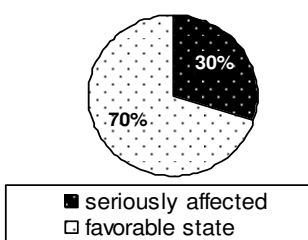
Lot II (control lot): 8 patients (3 male and 5 female) with their age between 5 and 17 years (average 10,3 years) where the device worked without the salt plates.

The device worked approximately 8-10 hours/day, at 9 V voltage. All patients followed the appropriate treatment during this period. The studied parameters: the general clinical state of the patient by subjective self-appreciation at the young people, respectively the parents' appreciation at the younger children, clinical examination of the respiratory system, FEV1 value at older children.

The selection criteria for both lots have been (Figure 2)

- Patients that have been seriously affected by the disease (3 from lot I, 2 from lot II), respectively: infection with *Pseudomonas aeruginosa* and/or *Staphylococcus aureus*, bronchiectasis, FEV1 < 50% - Patients with a favorable or mild clinical state (7 from lot I, 6 from lot II), respectively: without associated infection, FEV1 > 50-60%

Fig. 2 Clinical status of patients



Results

In lot I we noticed a significant improvement of the clinical state, respectively:

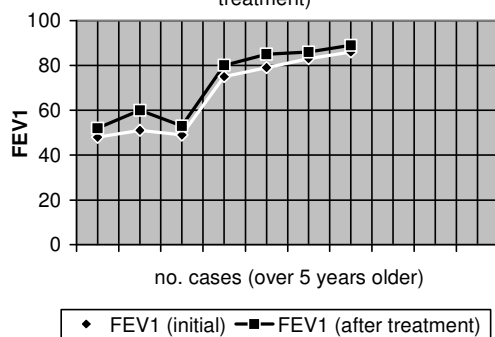
- The subjective estimation “for better” seen by the patients, respectively by the parents especially in those that have been more seriously affected;
- Improvement of the objective symptoms of the disease: increase of the sputum elimination within a first stage followed by a significant reducing of its quantity,

improvement of the respiratory functional syndrome, reducing of the crackles at auscultation, FEV1 improvement (Fig.3).

From the therapy beginning no patient showed other acute episodes of the respiratory disease that should require another hospitalization.

In lot II there were no changes similar with those from lot I (Fig.4)

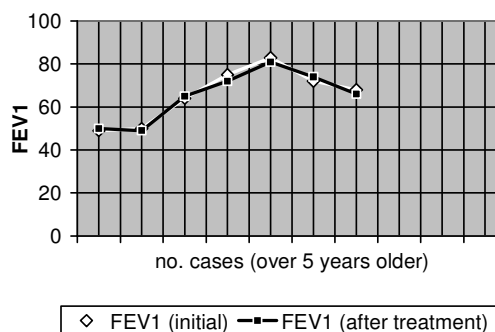
Fig. 3 Lot I (Values of FEV1, before and after treatment)



Conclusions

Forced ionization of the indoor air by salt sublimation represents an efficient method of the respiratory disease treatment in CF.

Fig. 4 Lot II (Values of FEV1, before and after treatment)



The method is an adjuvant, it does not exclude classical therapy.

It is a natural method of therapy adapted to the living space and it does not involve any risk.

It is a method of therapy quite cheap.

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THE VALUE OF ESOPHAGEAL pH MONITORING FOR THE DIAGNOSIS OF GASTROESOPHAGEAL REFLUX

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Abstract:

We studied the gastroesophageal reflux in a group of 31 infants using 24 hours esophageal pH monitoring. Including criteria: suspicion of gastroesophageal reflux in infants with extradigestive clinical manifestations, gastroesophageal reflux persistent after treatment, no prokinetic medication 48 hours before the examination. Excluding criteria: length of pH monitoring shorter than 18 hours, graphics with artifacts. Our results proved several significant differences for reflux parameters in symptomatic patients: subjects with chronic cough have higher reflux index (RI) and Euler score (ES) than those without cough, subjects with recurrent pneumonia have higher values for the length of the longest episode of reflux (LLER) than those with no pneumonia, subjects presenting wheezing have higher number of episodes of reflux (NER) than those with no airway obstruction symptoms, subjects with malaise have a higher ES than those without malaise, subjects showing growth failure have higher number of episodes of reflux longer than 5 minutes (NER5) than those with normal somatic development.

Key words: gastroesophageal reflux, pH monitoring, infant

Background:

We aimed to evaluate the correlations between different parameters of esophageal pH monitoring and clinical manifestations. Gastroesophageal reflux (GER) is common in humans; its incidence varies with age and gender.

In infants, GER may be the first noticed “pathological” sign¹, over 67% of the healthy children having more than one regurgitation during the day. Perceptible (visible) regurgitation occurs only in 20% of the episodes of GER detected by pH monitoring or gastric scintiscan⁷. The highest incidence of symptomatic GER, either physiological or pathological, is around the age of 4 months¹. Gastroesophageal reflux affects 1 of 300 cases according to American authors³, or 1 of 500 cases according to French literature⁴.

Quantitative esophageal pH monitoring allows the assessment of pH variations according to certain parameters. Physiologically, the esophageal pH ranges from 4.0 to 7.0. GER is defined by the decreasing of esophageal pH below 4.0. An episode of reflux is defined as beginning at the first decreasing of pH below 4.0 and ending at the first increasing of pH above 4.0⁵. The assessed parameters are:

1. **Reflux index (quotient) (RI/RQ)** – indicates the percentage of time when the pH is below 4.0; it demonstrates the exposure of the distal esophageal epithelium to gastric acid contents, but cannot specify the mechanism of reflux.
2. **Number of episodes of reflux (NER)** – is significant in assessing the function of the lower esophageal sphincter (LES).
3. **Number of episodes of reflux longer than 5 minutes (NER5)** – offers details on the esophageal clearance.
4. **Length of the longest episode of reflux (LLER)** – offers details on the esophageal clearance.

The most frequent used is **Euler score (ES)** = $NER + 4 \cdot NER5$, with a normal value below 50.

The most valuable parameter for assessing the GER in children is RI⁶, while the other ones are useful for a more accurate interpretation⁷.

Material and methods:

We have studied retrospectively a group of 31 infants (18 boys and 13 girls), most of them being of 5 to 11 weeks old, followed-up in our hospital from November 1999 through June 2002. All these patients have been investigated by 24 hours esophageal pH monitoring.

The equipment used to perform the pH monitoring consisted of:

- simple pH probe (with external reference electrode) made of stibium
- solutions for the standardization of the probes, prepared by the Institute of Chemistry “Raluca Ripan” of Cluj-Napoca
- pH meter (type D x C – 91 multi ion meter pH), provided by Datronix Computer in 1999.
- software for analyzing the information and generating the graphic of pH evolution, which is then printed on a paper.

During the continuous 24 hours esophageal pH monitoring should be recorded on a piece of paper all the sleep and the wake whiles (periods), meal whiles and any clinical manifestation.

The diagnosis of pathological acid GER has been established considering the RI for pH=4.0, its upper limit being 5%, just like in the study of Vanderplas^{8,9}. Euler score has also been used. Data provided by the pH graphics have been processed using descriptive and analytic statistics¹⁰. Data have been expressed as mean value \pm standard deviation (SD). Reflux parameters (RI, NER,

NER5, LLER, ES) have been analyzed with Student test. The differences have been considered statistical significant if $p < 0.05$, respectively having a very good statistical significance if $p < 0.01$.

Results:

Comparison of reflux parameters between subjects with pathological acid GER and those with physiological acid GER:

	Pathological acid GER M \pm SD	Physiological acid GER M \pm SD	p value
RI	11.58 \pm 4.76	3.30 \pm 0.82	2,93 E-07
NER	66.6 \pm 19.13	19.27 \pm 4.15	3,59 E-09
NER5	6.55 \pm 5.08	2.45 \pm 2.16	0,0084
LLER	25.04 \pm 12.24	8.04 \pm 5.52	0,0422
ES	90.95 \pm 27.14	28.90 \pm 9.40	2,46 E-08

Correlations among reflux parameters in subjects with pathological acid GER:

	IR	NER	NER5	LLER	ES
RI	0	0,5372	0.6936	0.4650	0.8271
NER	0.5372	0	0.0159	0.1411	0.6936
NER5	0.6936	0.0159	0	0.3686	0.7366
LLER	0.4650	0.1411	0.3686	0	0.2013
ES	0.8271	0.6936	0.7366	0.2013	0

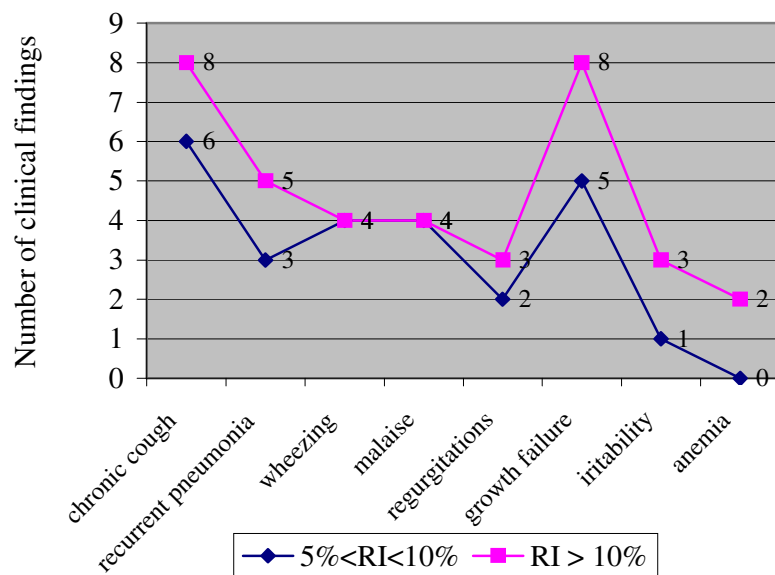
Correlations among reflux parameters in subjects with pathological acid GER:

	RI	NER	NER5	LLER	ES
RI	0	0.4096	0.6517	0.7474	0.6138
NER	0.4096	0	0.0962	0.2241	0.5463
NER5	0.6517	0.0962	0	0.6428	0.8775
LLER	0.7474	0.2241	0.6428	0	0.4897
ES	0.6138	0.5463	0.8775	0.4897	0

Comparison of reflux parameters in subjects with pathological acid GER, according to RI value:

	5% < IR < 10% M \pm SD	RI > 10% M \pm SD	p value
RI	7.96 \pm 1.42	15.48 \pm 3.48	0.0082
NER	57.9 \pm 17.29	75.3 \pm 17.47	0,0191
NER5	4.2 \pm 4.77	8.9 \pm 4.4	0,0173
LLER	10.49 \pm 6.39	19.59 \pm 15.15	0,0486
ES	7.51 \pm 11.43	106.8 \pm 29.44	0.0026

Comparison of clinical findings in subjects with pathological acid GER, according to RI value:



Prevalence of clinical findings:

Clinical findings		Pathological acid GER	
		YES	NO
Chronic cough	YES	14	5
	NO	6	6
Recurrent pneumonia	YES	8	6
	NO	12	5
Wheezing	YES	8	4
	NO	12	7
Malaise	YES	8	1
	NO	12	10
Regurgitations	YES	5	6
	NO	15	5
Growth failure	YES	13	10
	NO	7	1
Irritability	YES	4	0
	NO	16	11
Anemia	YES	2	3
	NO	18	8

Comparison of reflux parameters in subjects with pathological acid GER considering the presence or the absence of chronic cough:

	YES M ± SD	NO M ± SD	p value
RI	13 ± 4.95	8.28 ± 1.93	0.0192
NER	70.7 ± 18.92	57 ± 17.36	0.0731
NER5	7.64 ± 5.38	4 ± 3.75	0.0731
LLER	16.37 ± 13.89	11.91 ± 7.15	0.2350
ES	98.35 ± 29.17	73.66 ± 8.68	0.0300

Comparison of reflux parameters in subjects with pathological acid GER considering the presence or the absence of *recurrent pneumonia*:

	YES M ± SD	NO M ± SD	p value
RI	12.02 ± 3.46	11.34 ± 5.45	0.3844
NER	67.28 ± 15.55	66.23 ± 21.4	0.4550
NER5	6.42 ± 3.64	6.615 ± 5.85	0.4700
LLER	22.28 ± 16.69	11.13 ± 7.14	0.0245
ES	88.85 ± 20.37	92.07 ± 30.91	0.4039

Comparison of reflux parameters in subjects with pathological acid GER considering the presence or the absence of *wheezing*:

	YES M ± SD	NO M ± SD	p value
RI	13.81 ± 5.96	10.38 ± 3.96	0.0637
NER	86 ± 18.86	61.53 ± 7.95	0.0412
NER5	8.14 ± 6.46	5.69 ± 4.21	0.1583
LLER	17.85 ± 8.56	13.52 ± 13.91	0.2326
ES	109.14 ± 26.58	91.15 ± 14.17	0.0616

Comparison of reflux parameters in subjects with pathological acid GER considering the presence or the absence of *malaise*:

	YES M ± SD	NO M ± SD	p value
RI	13.27 ± 5.95	10.45 ± 3.62	0.1015
NER	73.12 ± 23.47	62.25 ± 15.16	0.1110
NER5	8.5 ± 6.45	5.25 ± 3.67	0.0835
LLER	11.66 ± 6.67	17.29 ± 14.72	0.1635
ES	105.6 ± 31.6	81.16 ± 19.40	0.0224

Comparison of reflux parameters in subjects with pathological acid GER considering the presence or the absence of *regurgitations*:

	YES M ± SD	NO M ± SD	p value
RI	11.4 ± 2.96	11.73 ± 5.30	0.1422
NER	51.6 ± 22.81	71.6 ± 15.52	0.0527
NER5	9 ± 4	5.73 ± 5.25	0.1285
LLER	21.9 ± 20.18	12.74 ± 8.03	0.0558
ES	81.8 ± 12.35	94 ± 30.27	0.1034

Comparison of reflux parameters in subjects with pathological acid GER considering the presence or the absence of *growth failure*:

	YES M ± SD	NO M ± SD	p value
RI	12.41 ± 5.17	10.56 ± 4.27	0.2008
NER	64 ± 23.52	69.77 ± 12.52	0.2582
NER5	8.09 ± 3.35	4.66 ± 2.91	0.0185
LLER	18.55 ± 15.12	10.74 ± 5.70	0.0806
ES	97.36 ± 31.43	83.11 ± 19.73	0.1266

Comparison of reflux parameters in subjects with pathological acid GER considering the presence or the absence of *irritability*:

	YES M ± SD	NO M ± SD	p value
RI	11.6 ± 3.21	11.58 ± 5.61	0.4973
NER	56 ± 23.7	69.25 ± 17.69	0.1122
NER5	9.5 ± 4.43	5.81 ± 5.08	0.1011
LLER	31.4 ± 9.27	13.45 ± 8.25	0.0611
ES	86.75 ± 6.34	92 ± 30.32	0.3697

Comparison of reflux parameters in subjects with pathological acid GER considering the presence or the absence of *esophagitis*:

	YES M ± SD	NO M ± SD	p value
IR	16.02 ± 3.49	10.10 ± 4.23	0.0158
NER	74.6 ± 11.63	63.93 ± 20.67	0.1461
NER5	9.6 ± 4.15	5.53 ± 5.06	0.0620
LLER	28.02 ± 18.07	10.71 ± 5.47	0.0315
ES	107.2 ± 17.97	85.53 ± 27.96	0.0625

Discussions:

We encountered the following clinical manifestations in our study group: irritability (24%), growth failure (18%), chronic cough (15%), recurrent pneumonia (11%), wheezing (9%), regurgitations (9%), malaise (7%), anemia (4%).

We found several significant differences for reflux parameters in symptomatic patients:

- subjects with chronic cough have higher RI (13 ± 4.95) and ES (98.35 ± 29.17) than those without cough (RI = 8.28 ± 1.93, p<0.05; ES = 73.66 ± 8.68, p<0.05)
- subjects with recurrent pneumonia have higher LLER (22.28 ± 16.69) than those with no pneumonia (11.13 ± 7.14), p<0.05

- subjects presenting wheezing have higher NER (86 ± 18.86) than those with no airway obstruction symptoms (61.53 ± 7.95), p<0.05
- subjects with malaise have a higher ES (105.6 ± 31.6) than those without malaise (81.16 ± 19.4), p<0.05
- subjects showing growth failure have higher NER5 (8.09 ± 3.35) than those with normal somatic development (4.66 ± 2.91), p<0.05

There are evidences of the involvement of GER in exacerbating the airway reactivity¹¹. The other parameters may be considered of poor outcome, imposing a more accurate treatment: increasing the lower esophageal sphincter tone, administering antacids, H₂-receptor blockers or hydrogen pump inhibitors in infants with higher NER5 and LLER, or stimulating gastric emptying in those with higher NER.

In patients with esophagitis due to reflux, RI (16.02 ± 3.49) is higher and so is LLER (107.2 ± 17.97). These results are contradictory to some published articles, which sustain that esophageal pH monitoring does not correlate to the endoscopic image of esophagitis due to reflux¹².

Conclusions:

1. Esophageal pH monitoring is useful to assess gastroesophageal reflux in infants. RI is the most valuable parameter. NER, NER5 and LLER allow to distinguish between physiological and pathological acid GER.
2. Euler score is useful to evaluate gastroesophageal reflux in infants and allows to distinguish between physiological and pathological acid GER.

3. In infants with pathological acid GER, there is a very significant correlation between RI and NER, NER5 and ES respectively. There is also a correlation, but not so significant, between RI and LLER. No correlation could be established between NER5 and LLER.
4. In infants with physiological acid GER, there is a very significant correlation between RI and NER5 and ES respectively. There is also a correlation, but not so significant, first between RI and LLER, and then between NER5 and LLER. No correlation could be established between RI and NER.
5. Infants with pathological acid GER having RI above 10% show more clinical manifestations than those having RI between 5 and 10%.
6. Infants with pathological acid GER presenting esophagitis have higher RI and LLER compared to those without esophagitis.

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MALLORY-WEISS TEAR IN AN 11 YEARS OLD PATIENT WITH ACUTE LYMPHOBLASTIC LEUKEMIA

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Abstract:

We describe the case of an 11 years old boy followed-up in our hospital for acute lymphoblastic leukemia (ALL) type L1, who presented vomiting during the specific chemotherapy. The antiemetic drugs were ineffective and the esophagogastroscope showed a Mallory-Weiss tear. After prokinetic treatment, antacid medication and hydrogen pump inhibitors, his evolution was favorable, with the cessation of vomiting and the healing of esophageal tear.

Key words: leukemia, vomiting, Mallory-Weiss tear

Background:

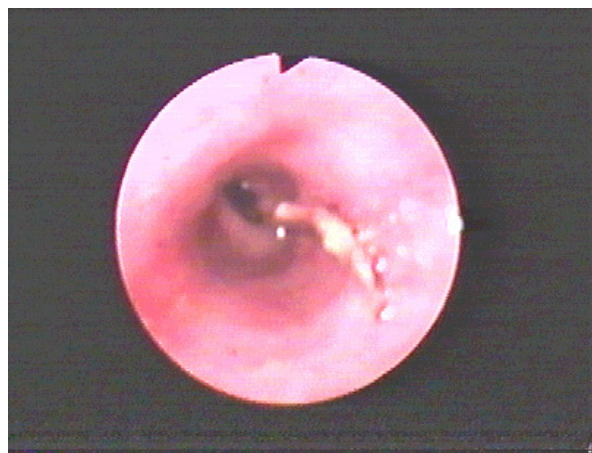
Mallory-Weiss syndrome is an uncommon cause of upper gastrointestinal bleeding in adults, several studies estimating a prevalence of 5-10% of the total cases of upper gastrointestinal bleeding¹. In children, the prevalence of upper gastrointestinal bleeding due to Mallory-Weiss syndrome varies with age², apparently being less than in adults³.

Case report:

P.C. aged 11 years, male, has been followed-up in our hospital since January 2004 with the diagnosis of acute lymphoblastic leukemia type L1, CD 10+, medium risk group. He received induction chemotherapy according to

BMF 95 Non B ALL, MRG protocol, his clinical evolution being favorable. In November 2004, while the outpatient received maintenance therapy with Methotrexat po and Purinetol, he presented vomiting which could not be controlled by the administered medication, the patient being unable to tolerate Metoclopramide. The vomiting became incoercible even when the cytostatic agents were stopped. After three weeks with persistent vomiting, the patient came in our service for investigations. Clinical findings at admission consisted of malaise, fatigue, intense paleness, stable hemodynamic, biliary vomiting, polakiuria and disuria due to a hemorrhagic cystitis on a scleroatrophic urinary bladder.

After the routine hematological, biochemical and bacteriological exams, esophagogastroscope was performed using an Olympus Exera CV 160 videoendoscope, preceded by sedation with Midazolam (Dormicum). The endoscopy revealed intense paleness of the esophageal mucosa, no lesions in the upper two thirds, but one longitudinal tear pericardial, in the upper superior quadrant, with minimum bleeding, covered by fibrin and blood clots. At the same time, a gastric prolapse and a minimum hiatal hernia were noticed. The entire gastric cavity could be examined and no pathological features were found, excepting the paleness of the mucosa and minimum stasis (gastric liquid, bile), but without any sign of bleeding. (Fig.1,2).

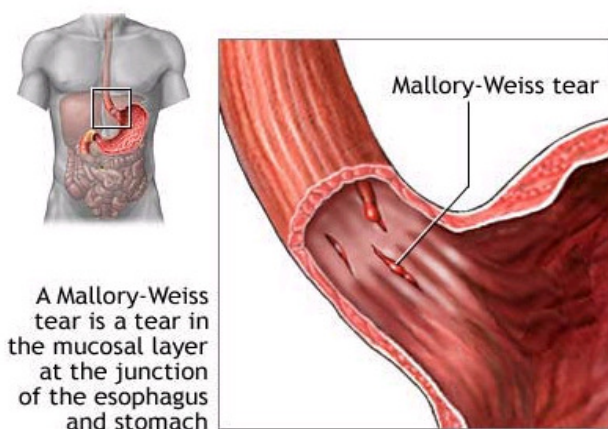


The persistence of vomitings for three weeks correlated to the endoscopic aspect (described above) led us

to the diagnosis of Mallory-Weiss tear in a patient receiving chemotherapy.

Treatment included a proton pump inhibitor – Pantoprazole (Controloc) 40 mg/day iv, an antacid – Sucralfat (1 g po tid) and Erythromycine in prokinetic dose of 5 mg/kg/day po. With this therapy, vomiting remitted progresively and the endoscopy performed 7 days later showed the healing of the esophageal lesion.

The ultimate diagnosis was: Acute lymphoblastic leukemia, Mallory-Weiss tear, Hiatal hernia, Scleroatrophic urinary bladder, Hemorrhagic cystitis.



The most important presenting clinical features include: nausea, vomiting effort, hematemesis. History is typical in just 50% of the cases, so that the diagnosis is established by esophagogastrosocopy^{8,9}.

Discussions:

Mallory-Weiss tear consists in longitudinal or ellipsoidal ruptures of the lower esophageal mucosa, close to the gastro-esophageal junction^{4,5,6}, which classically occur after a vomiting effort, although this can be absent sometimes. Bleeding appears when the rupture reaches to the esophageal venous or arterial plexus (Fig.3). It occurs especially after alcohol drinking, chemotherapy or some drugs administration. Patients suffering of portal hypertension may develop more severe Mallory-Weiss tears⁷.

In adults, the incidence of Mallory-Weiss tear varies by different authors. According to American Society of Gastrointestinal Endoscopy Bleeding Survey, this lesion is the seventh cause of upper gastrointestinal bleeding¹ (Table 1).

Table 1: Etiology of upper gastrointestinal bleeding in adults (*American Society of Gastrointestinal Endoscopy Bleeding Survey*)¹

Cause	Number of patients	Incidence
Gastric erosion	620	29.6 %
Duodenal ulcer	477	22.8 %
Gastric ulcer	457	21.9 %
Varices	323	15.2 %
Esophagitis	269	12.8 %
Duodenitis	191	9.1 %
Mallory-Weiss tear	168	8.0 %
Neoplasm	78	3.7 %
Esophageal ulcer	46	2.2 %
Stomal ulcer	39	1.9 %
Telangiectasia	10	0.5 %
Other	152	7.3 %

In pediatric patients, large trials showed a less incidence of Mallory-Weiss syndrome as a cause of upper gastrointestinal bleeding, 0.3% respectively³. In children, the incidence of upper gastrointestinal bleeding due to Mallory-Weiss tear varies with age^{2,4,5}. It is supposed that the lesion does not occur in neonate. For children aged 1 to 24 months it is the fourth cause. For the ages of 2 to 7 years

Mallory-Weiss tear is the second cause of upper gastrointestinal bleeding, being more frequent than esophagitis, ulcers or esophageal varices. In 7 to 17 years patients, Mallory-Weiss is less frequent than gastritis, esophagitis and ulcers, but more frequent than esophageal varices. (Table 2).

Table 2: Etiology of upper gastrointestinal bleeding in children ²:

0-1 month	1-24 months	2-7 years	7-17 years
Esophagitis	Esophagitis	Gastritis	Gastritis
Gastritis	Gastritis	Mallory-Weiss tear	Esophagitis
	Ulcers	Esophagitis	Ulcers
	Mallory-Weiss tear	Ulcers	Mallory-Weiss tear
		Esophageal varices	Esophageal varices

In most cases bleeding stops spontaneously in 24 to 48 hours^{9,6}. But, because of the potential persistence of vomiting, of restarting the bleeding, or even of the esophageal rupture (Boerhaave syndrome) it is important to promptly remove all the vomiting causes and to carefully monitor the patient.

Whether the hemorrhage is active, the treatment may include Vasopresine (0.1-0.4 U/min iv³) administration or endoscopic measures⁹ Adrenaline 1/10000, thermal probe or angiographic embolization.

As cited in several articles^{10,11,12} the hemorrhage did not become visible in our case until the admission in our

hospital, when stool exam proved positive Gregersen reaction.

Conclusions:

Even if the prevalence of Mallory-Weiss tear seems to be less than in adult pathology, the large number of upper gastrointestinal bleeding conditions in children, their potential of dramatic outcome and the possibility of endoscopic examination in young children, impose a prompt cessation of vomiting and performing esophagogastrosocopy.

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THE CORELATION OF THE RICKETS INCIDENCE WITH THE RISK FACTORS

The risk factors- individual study

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Summary

The vitamin D-deficiency rickets, a well defined clinical entity, still represents an important risk factor for the infantile morbidity and mortality. As it is known for more than 10 years, the vitamin D-deficiency rickets has multiple causes. Until now, there was no prospective study undertaken in order to establish the real incidence of the disease.

The analysis performed through this study sustains that the whole process of the primary medical practice consists in the knowledge of the rickets epidemiology with the purpose to improve the efficiency of the general and individual prevention measures of this disease.

Key words: vitamin D-deficiency rickets, child, incidence, risk factors, study.

Introduction

The vitamin D-deficiency rickets is a well defined clinical entity and remains the most frequent vitamin deficiency during the childhood and especially in the infant period.

The frequency of the disease continues to remain high in our country, this representing an important risk factor for the infantile morbidity and mortality (4). In other parts of the world (Canada, USA, Scandinavian countries,

some countries from Asia) the high incidence of the disease led to settled measures of public health, a great importance was given to the nutritional factors (6, 7, 8, 9, 12, 14).

Rickets etiology studies performed for more than 10 years in other countries showed that the disease has multiple causes. (13)

So far, no prospective study was undertaken in order to establish the real incidence of the disease. The evaluation is complicated due to the fact that many rickets cases are diagnosed and treated in the out patient departments without being reported. That is why it is necessary once more a prospective follow-up study of the rickets cases.

Material and method

In order to achieve our goal, we followed a group of children aged from 0 to 16 years recorded in the G.P. Children Consulting Room – Dr. Boangiu from Timisoara, during the period 1.01.1997- 31.12.2003. During each year we divided the patients group in 2 subgroups: subgroup 1 - children aged 0-2 years - and subgroup 2 - children aged 3-16 years. The subjects repartition during the study was the following:

The study period	Total analyzed subjects	Subjects repartition on age groups
1. 1.01.1997-31.12.1997	1464	subgroup1: 153 subgroup 2: 1311
1.01.1998-31.12.1998	1448	subgroup1: 149 subgroup2: 1299
1.01.1999-31.12.1999	1493	subgroup1: 143 subgroup2: 1350
1.01.2000-31.12.2000	1490	subgroup1: 128 subgroup2: 1362
1.01.2001- 31.12.2001	1156	subgroup1: 136 subgroup2: 1331
1.01.2002-31.12.2002	1481	subgroup1: 155 subgroup2: 1326
1.01.2003-31.12.2003	1453	subgroup1: 138 subgroup2: 1315

To these patients we followed the appearance of the clinical signs and the laboratory findings in correlation with the physiological antecedent history (the pregnancy evolution, the gestational age, the physiological jaundice duration, dairy nourishment during infancy), the somatic and the health state evolution, the child feeding and the caring environment, the moment of the rickets prevention initiation, the manner of its applying, the types of the used vitamin D preparations. The rickets diagnosed cases were

treated through the Specialty Out-Patient Department of the Pediatrics Clinic II Timisoara.

2.Results and discussions

Depending on the disease form, the diagnosed cases were divided in:

- the classical form of the infant and toddler (the common vitamin D-deficiency rickets);
- the delayed onset form.

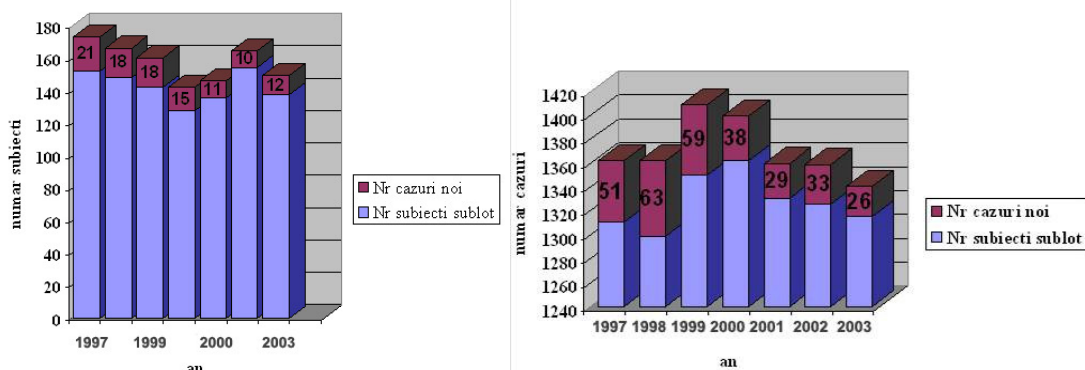


Fig 1 The specific morbidity at the analyzed group

At the analyzed group we followed the rickets incidence in relation with the risk factors, starting from the premise that the decisive factors in this disease are the nutritional vitamin D-deficiency (exogenous deficiency) and the lack of exposure to sunrays (endogenous deficiency). The risk factors taken into the study were: the season, dairy nourishment during infancy, maternal vitamin D-deficiency, prematurity, the prolonged jaundice evolution in the newborn period.

The rickets incidence during the 7 years study in the presented group was of 4.02%. The percentage analysis deducted on the study-years led to the conclusion that the peak incidence occurred in 1998 and was followed by a progressive decrease until the end of the year 2003. We consider that this favorable evolution of the rickets incidence curve during the 7 years study is due to the up to date prevention scheme of the disease beginning with 1998.

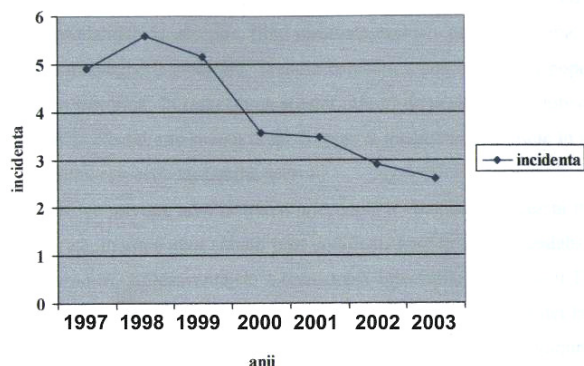


Fig. 2. The rickets incidence during the years of study.

Another factor that might have contributed to this favorable evolution was the large scale accessibility of the commercially available formulas that are supplemented with the adequate vitamins, including the vitamin D necessary.

Comparing with this favorable evolution, the rickets incidence at the studied group was high, this situation sustaining the multiple etiology of the disease.

The rickets incidence depending on the clinical form was of 1.04% for the classical form of the infant and toddler and of 2.97% for the delayed onset form. The appearance risk of the delayed onset form is much higher

because the development rhythm at this age is more accelerated.

The season incidence of the rickets in the followed group was of 3% in the non-sunny months, comparing with 1,01% during the sunny months, this aspect confirming the importance of the environment factor which should be taken into account for the adjustment of the prevention vitamin D doses (2,5).

We chose for this study the risk factors that we were able to demonstrate through experimental, clinical and statistical studies and that could cause, promote or aggravate the disease. We used the statistical method after Ancusa et al. from the bibliographical studied material (1).

The infant diary nourishment evaluation as a risk factor for the rickets, through the obtained results, confirms the data sustained by other authors from abroad. (7,11)

The vitamin D-deficient human milk represents a risk factor for the rickets. Despite this result, the breastfeeding's value remains absolute and indisputable even in the rickets prevention (3).

The data used for the evaluation of the gestational age as a risk factor showed in all the 7 years of the study

that the prematurity represents a very important risk factor, which should be considered in adjusting the vitamin D prevention dose for this infant category(10).

The vitamin D maternal deficiency showed that ignoring the antenatal prevention might represent a risk factor for the disease.

Conclusions

1. The rickets incidence found in the study-group sustains the importance that should be given to this public health problem.
2. The simultaneous action of all etiological factors requires a detailed knowledge, the aim being the improvement of the general and individual prevention measures.
3. Fighting this disease as a necessary premise for the whole primary medical process is correlated with the competent medical surveillance of the pregnant woman and of her child from the first days of life until the end of the growth process.

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THE ECHOGRAPHICAL EXAMINATION IN CHILD CHOLESTATIC SYNDROME

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Summary

The sonographical exploration of the child cholestatic jaundice brings its real contribution to diagnosis when the children's age and pathology particularities are taken into account by the sonographer.

The patients group with cholestatic syndrome taken into study is significant (20 cases) and the diagnostical criteria (clinical examination and laboratory tests) are scientifically and accurate argued.

The abdominal echographical examination completed with the Doppler techniques of pulsatory emission and colour codified, made sensitively evident specific paediatric diseases such as: bile cyst stone, bile cyst hydrops, cholestatic chronic hepatitis, biliary atresia, choledochocyst etc.

Through the supplied informations, the ultrasonography has become the essential method for the child hepatic disease, allowing in the same time the dynamical supervision of the existing pathological elements. The Doppler techniques have a remarkable contribution and prove a very high accuracy in the etiological diagnosis of the cholestatic syndrome in Pediatrics.

Key words: cholestatic syndrome, liver diseases, child, abdominal echography, Doppler techniques

Introduction

The appearance and the development of the ultrasonography has improved the study of the child cholestatic syndrome, with the mention of informing the sonographer of the age and pathology particularities.

The cholestatic jaundice sonographical examination must include a liver, biliary tract and the proximity organs global examination, and must indicate: the liver size and structure, the intra- and extra hepatic bile tract size and appearance, the bile cyst size and the wall thickness, the pancreas and spleen appearance, the evidence of the portal hypertension signs.

Thus, the ultrasonography became the lead method for the bile cyst and biliary tract visualization, having a high accuracy for the obstructive feature specification of the icteric syndrome and in the differentiation between intra- and extra hepatic cholestasis.

Material and method

The study was performed in the Pediatrics Clinic II Timisoara during a period of 5 years (1997-2002), on a 20 cases group with cholestatic syndrome. The cases selection was made on the basis of the well defined clinical and laboratory criteria:

-Clinical manifestations: nausea, vomiting, flatulence, right hypochondrium pains, pruritus, hyperchrome urines, acholic stools, fever, asthenia, jaundice.

-Laboratory investigations:

- Enzyme tests (SGOT, SGPT, LDH, Alkaline phosphatase, γ GT, \pm α fetoprotein)
- Biochemical tests (bilirubin, serum iron, cholesterol, triglycerides, electrophoresis, PT);
- Hematological tests
- Immunological tests: immunoassay, viral markers, antinuclear antibodies, specific antibodies for hepatitis, cytomegalovirus and mononucleosis
- Cholecystoangiography was performed at 5 subjects
- Fibroscopy was performed in 2 cases
- Computerized tomography was needed in 5 cases
- Hepatic bioptic puncture was performed on an empty stomach, after a premedication administrated to prevent abdominal meteorism, using 3.5 and 5 MHz probes upon the size of abdomen and the explored zone. We used pulsing and color mapping Doppler method to differentiate biliary ducts of blood vessels

Results

Sex and age distribution of the cases are represented in Fig. 1 and Fig. 2:

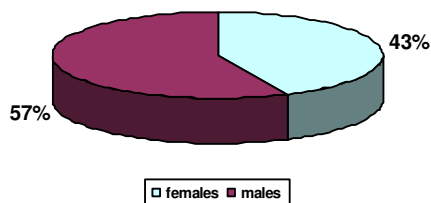


Fig. 1. Sex distribution of the patients.

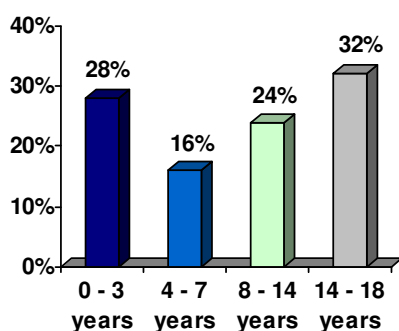


Fig. 2. Age distribution of the patients.

The echographic classification of the diseases was: 6 cases with vesicular pathology, 14 cases with primary impairment of the biliary tracts.

Obstructive pathology of the biliary system revealed: 4 children with vesicular lithiasis, 2 cases with

vesicular hydrops, 6 cases with chronic cholestatic hepatitis, 2 patients with biliary atresia, 4 cases with neonatal hepatitis, 1 case with choledocal cyst, 1 case with primary sclerotic cholangitis.

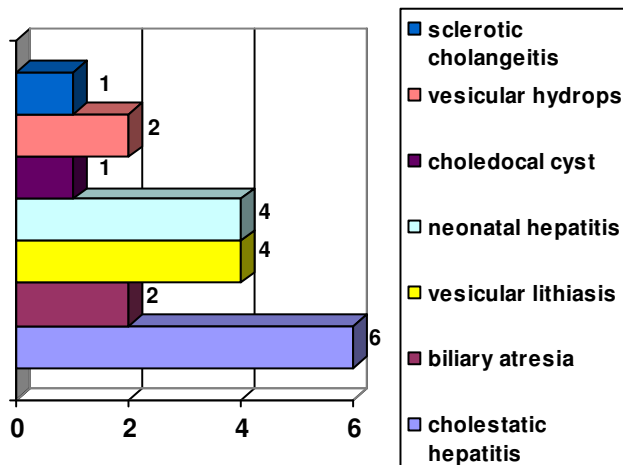


Fig.3. Classification on morbidities.

Using Doppler techniques we could perform a precise differentiation of the biliary tract dilatation from the venous or arterial dilatations.

Color Doppler ultrasonography visualized the specific venous and arterial blood flow, the direction, velocity and turbulence of the sanguine flow through different colors.

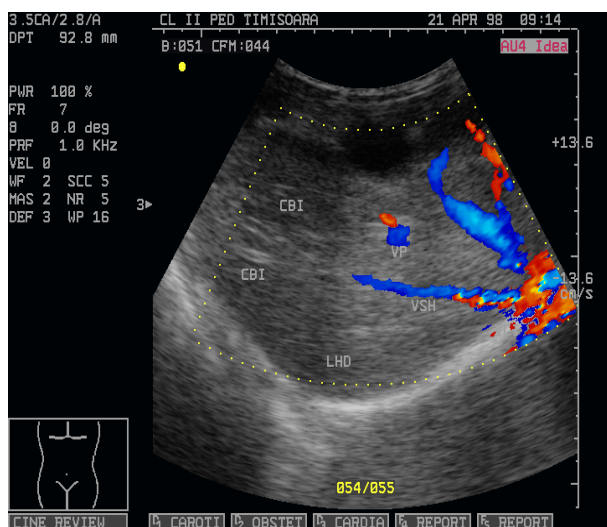


Fig.4 Doppler techniques for the diagnosis of the biliary tract dilatation from the venous or arterial dilatations

The dilated intrahepatic biliary tracts had been visualized as tubular elongated images, with hiperreflective walls, parallel with the intrahepatic port branches, which converged thru the hill, without Doppler pulsations and no color.

The liver is easy to visualize due to his topographic position, and it must be explored entirely on the screen. To perform a precise localization of the pathologic elements it is very useful the segment division of the liver lobes.

We obtained the following echographic aspects:

1. In the vesicular lithiasis we visualized hyperechogenic images with declive localization, with posterior shadow conus, mobile with patient position. We met solitary or multiple calculi. The biliary vesicle wall was hyperechogenic, over 3 mm thick, dilated and torsioned. The precision of ultrasonography to diagnose lithiasis is approximately 95%. In one case we noticed the migration of a 3 mm wide calculi in choledocus, which dilated the biliary extrahepatic vessels.

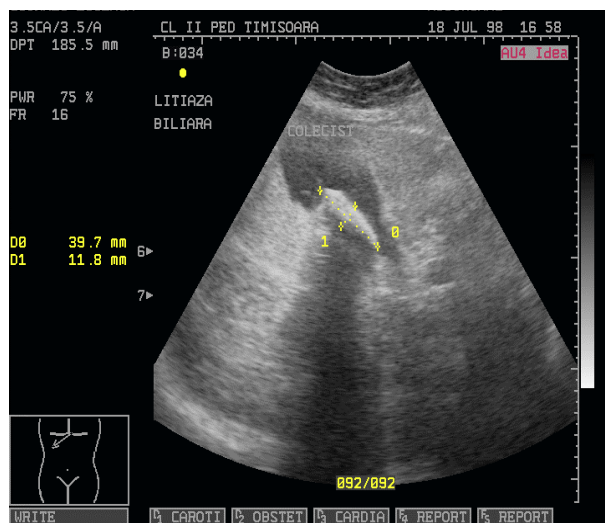


Fig.5 Vesicular lithiasis

2. Vesicular hydrops was defined by an augmentation of the cholecystic volume with the diameters over 10/4 cm, resulting in a globular shape, with hyperechogenic images in the infundibular region double contoured ticked wall, with focal hypoechogenities

(micro abscesses) or focal hyperechogenities (hemorrhages, necrosis) and irregularities of the wall contour (ulceration) with floating intraluminal membranes and sludge. The Murphy sign was positive.

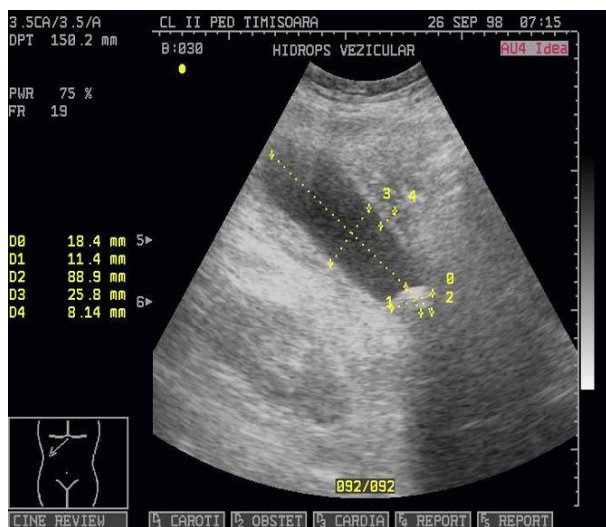


Fig.6 Vesicular hydrops

3. Cholestatic chronic hepatitis presented: hepatomegaly with nonhomogenous structure, marked hyperechogenicity with posterior attenuation, intrahepatic

dilated biliary vessels, different grades of splenomegaly, shape and parietal modifications of the cholecyst.

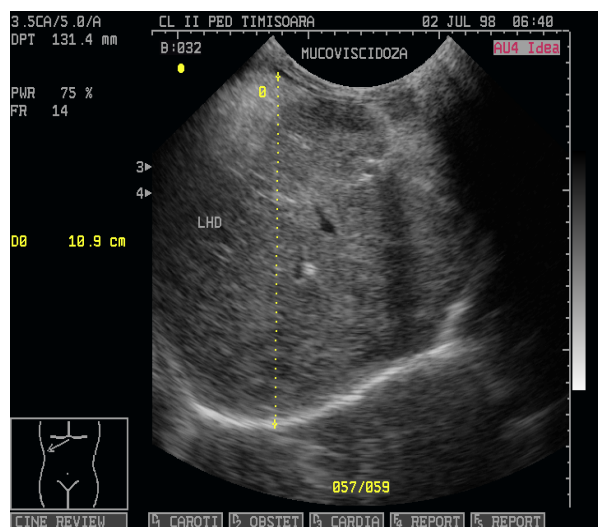


Fig. 7 Intrahepatic cholestasis. Right subcostal oblique sonogram.

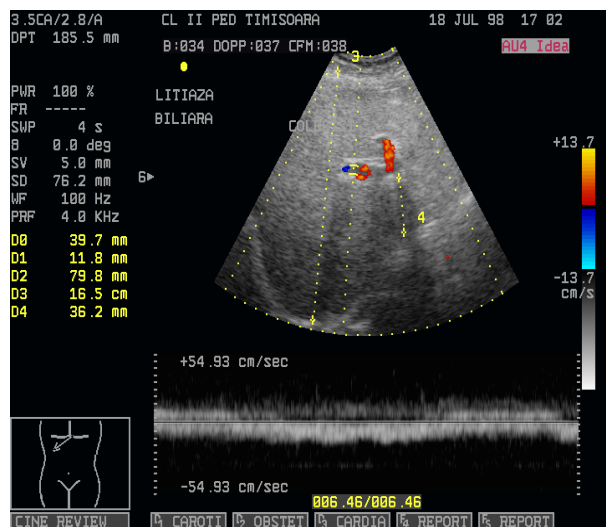


Fig. 8 Common biliary duct dilated. Right oblique sonogram.

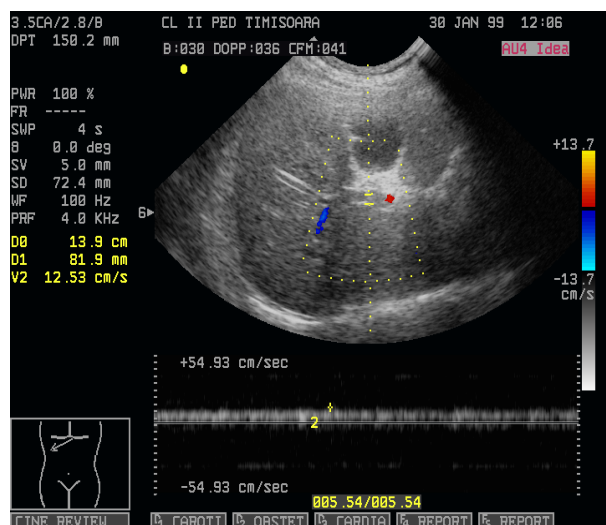


Fig. 9 Dilated intrahepatic biliary tract. Right oblique sonogram.

4. Biliary atresia was discovered in one new born with severe progressive cholestatic jaundice, who presented dilated intrahepatic biliary vessels being impossible to visualize the lumen of the main biliary way and cholecyst.

5. The choledocal cyst clinically presented transient episodes of pain and jaundice and was illustrated

through a marked pseudocystic choledocal dilatation, generating the aspect of choledochocel. In one case we noticed segmental cystic dilatations of the intrahepatic biliary vessels (Caroli disease).

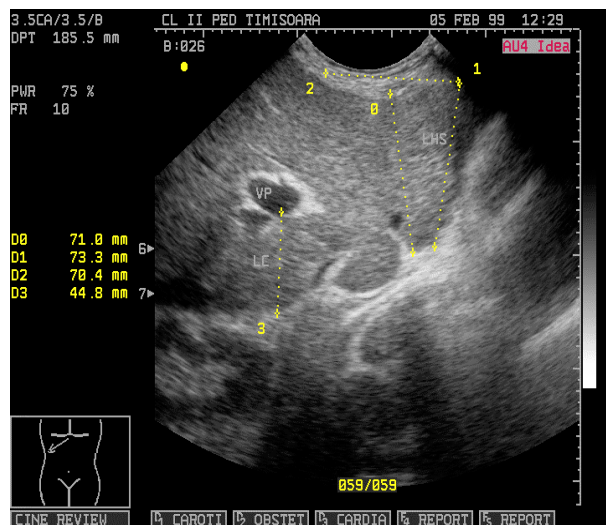


Fig.10 Choledocal cyst

6. Idiopathic sclerotic cholangitis was identified through the thickness of the intra- and extrahepatic vessel

walls, with doubled contour and narrowing of the main biliary way lumen and an increased periportal echogenicity.

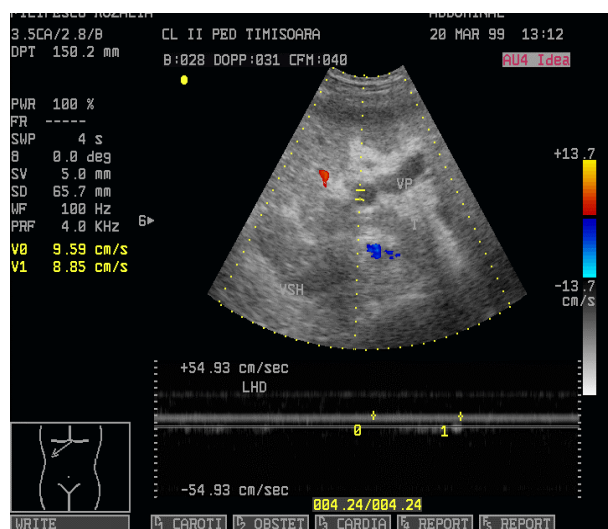


Fig.11 Cholangitis

Conclusions

1. Ultrasonography became the main method to evaluate children's liver disease, permitting a dynamic surveillance of the existing pathological elements.

2. Echography is as efficient as the new imagistic techniques to evaluate the children's cholestatic syndrome. It is preferred because has no invasiveness, can be repeated and has a low cost.

3. It also permit echographic guided biopsy of the liver formations and the histopathological examination confirms the diagnosis.

4. Using ultrasonography for the cholecyst pathology we can describe: position, shape and volume anomalies, focal or diffuse modifications of cholecyst content or wall, the biliary obstruction site: intrahepatic or extra hepatic cholestasis.

5. Doppler techniques are more accurate to describe biliary tract dilatations of venous or arterial dilatations.

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PERINATAL HYPOXIA-ISCHEMIA MAJOUR COUSE OF SYSTEMIC DISFUNCTION IN NEWBORNS

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Abstract

The aim of this work to show the role that hypoxic ischemia has in inducing newborns manifestations (neurological, cardiovascular, digestive, respiratory dysfunctions) and a short time evolution affected newborns. The evolution of the hypoxia-ischemia depends on the number of affected organs, influenced by the functional maturity grade and the individual genetic heritage. The lot of study included 88 from 237 newborns hospitalized in First Pediatric Clinic between 01.Jan.2003 to 01.July.2004. The newborns studied were all affected by different grades of hypoxic-ischemia without other infections.

Key words: hypoxic-ischemia, newborns.

Introduction

Increasing hypoxemia, leads to fetal compromise due to tissue Hypoxia, anaerobic metabolism and a metabolic acidosis. Tissue hypoxia of particular degree and duration will cause multiple organ damage including brain.

Material and methods.

The lot of study included 88 from 237 newborns hospitalized in First Pediatric Clinic between 01.Jan.2003 to 01.July.2004. The newborns studied were all affected by different grades of hypoxic-ischemia without other infections.

We formed 3 lots of newborns based on the time of delivery: preterms-26, intrauterine growth retardation (IUGR)-29 and 33 on term newborns.

We analyzed the functional response in each organ and cases evolution.

Results and discussions

Regarding the time criteria of illness and the onset of hypoxic-ischemia stress:

- at 19(22%) cases studied hypoxic-ischemia reached chronic level in intrauterine phase: disgravidia with inevitable abortion, obstetrical anomalies, vicious behavior (smoking, intense physical effort, coffee and alcohol consumption), maternal affections (anemia, hypertension, spasmofilia and physical trauma), twins pregnancy
- at 25(28%) cases the hypoxic-ischemia installed acute – at delivery: fetal distress during labor, not medical assisted delivery, abnormal fetus outcome
- 20 (23%) cases associated chronic and acute perinatal asphyxia: abnormal outcomes, umbilical cord anomalies, hemolytic disease and aspiration pneumonia
- at 11(13%) the hypoxic-ischemia stress appeared after birth (heart malformations, pneumotorax, heart rate modifications);

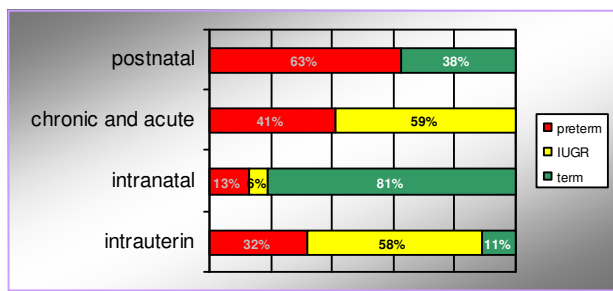


Fig.1-time criteria of illness and the onset of hypoxic-ischemia stress

only 6 pregnancies had a medical long term observation so the onset of hypoxic-ischemia stress had been measured based on post-delivery clinical, biological and paraclinical examsl.

Affected organs and body systems (descending order): central neural system (CNS) (88 cases), acido-basic status (86 cases-98%), cardiovascular (57 cases-65%),

respirator tract (48 cases-55%), gastro-intestinal and hepatic system (43 cases-52%), hemo-coagulation (41 cases-47%) renourinal tract (26 cases-30%).

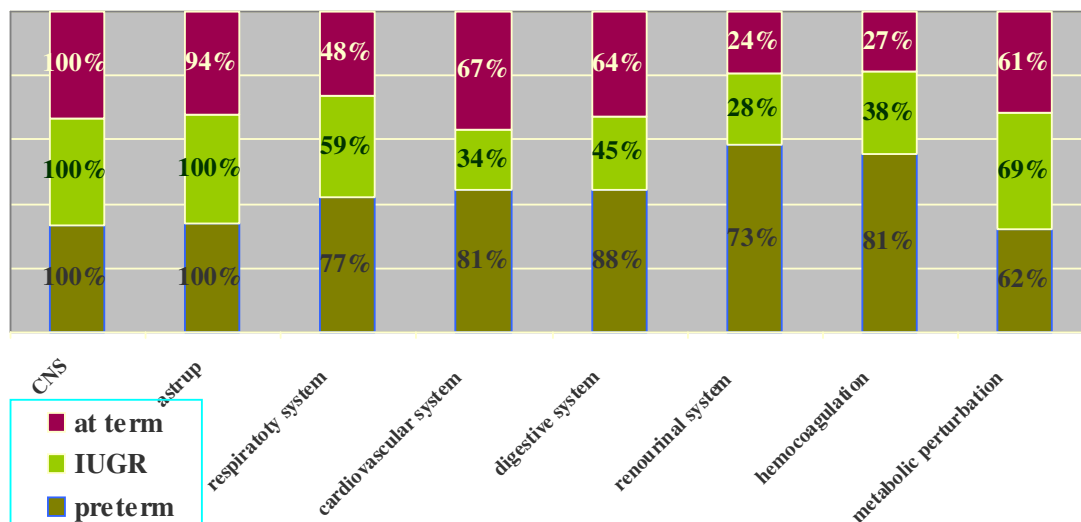


Fig.2. Organ and system distress (type of disease in %) appear at the hypoxic-ischemic affected newborns.

Vaso-motor disorders were found more often at the term newborns.

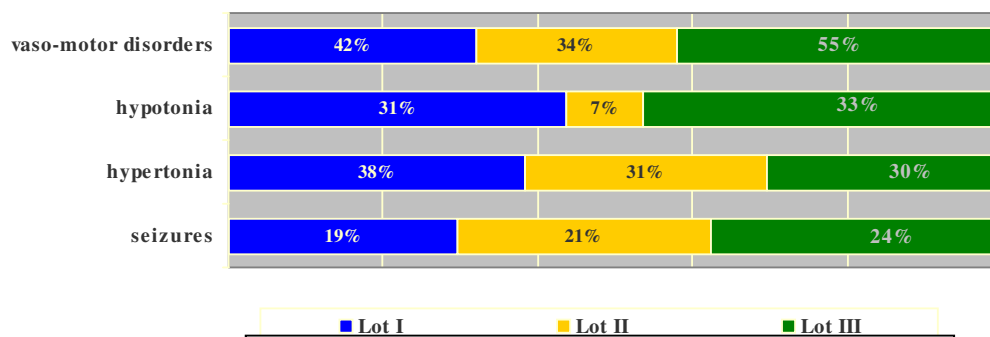


Fig.3. Neurological disorders found at the subjects (in percentage).

Echo transfontanelar

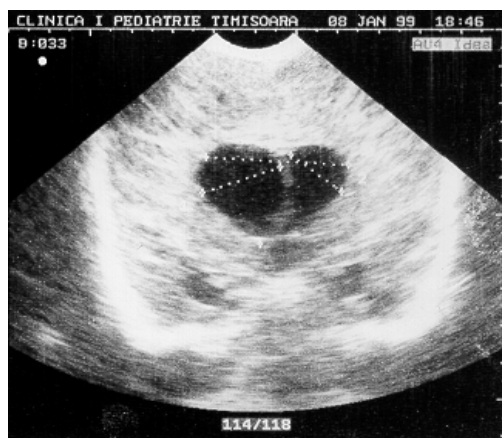


Fig.4. Periventriculare and coroids plexus hemorrhage.



Fig.5. Hydrencephaly as a follow-up of intraventricular hemorrhage.

Heart rate proved to be a very good clinical criteria for hypoxic-ischemia split into the following forms: easy- tachycardia; medium – bradycardia; sever tahy-bradycardia.

ECG shows a high rate of repolarization dysfunctions (50 from 88 cases).

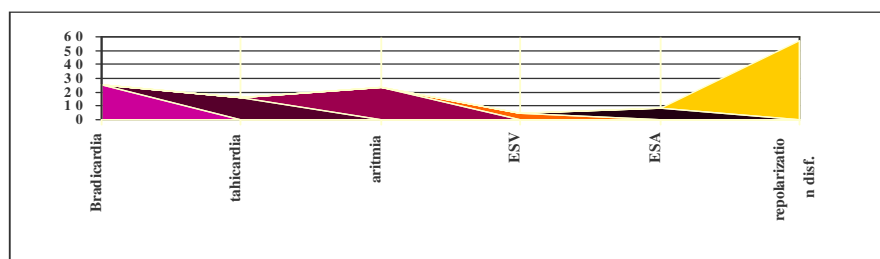


Fig.6. Heart dysfunctions a clinical criteria for hypoxic-ischemic affection.

The respiratory dysfunctions showed a higher rate of the apnea (48%).

Apnea being in our cases a dominant central symptom.

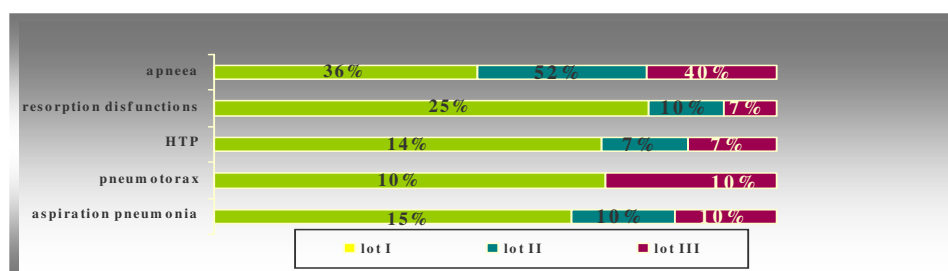


Fig.7. Respiratory dysfunctions appears at the 88 cases (in percentage).

The digestive function proved to be difficult to all the 3 lots. The preterm lot had the slowest adaptation; 6(23%) of them developed ulcero-necrotic enteritis (EUN)

< that wasn't found at the IUGR lot > 14(54%) vomiting, 20(77%) gastric residuum and 18(69%) abdominal distension.

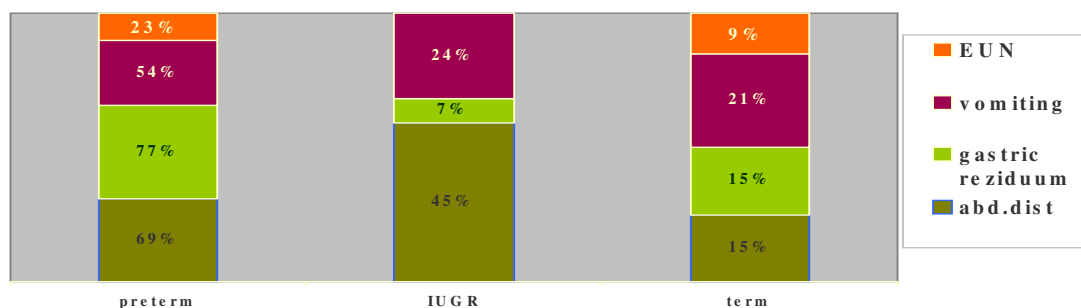


Fig.8. Digestive dysfunctions at the 88 newborns studied.

The modification of the acido-bazic status and cytolyses enzymes (**LDH**, TGP, TGO) represents both a

marker and a prognostic factor in hypoxic-ischemia.

Lot	Acidosis	Cytolysis	BI	CID	Azotes retention	hypoCa	Hypoglic.	hyperglyc
I	100%	100%	31%	23%	35%	50%	42%	9
II	79%	69%	28%	3%	17%	41%	52%	3
III	88%	61%	12%	21%	21%	55%	18%	7

Fig.9. Biological modifications appeared at the 88 newborns studied.

Case evolution—no preterm had a good evolution, 31% died in the first 2 weeks-4% of them showing complex heart malformations, 50% continued to experience

neurological dysfunctions, 3(8%) were transferred to Cardiology, the IUGR had the best evolution (79%).

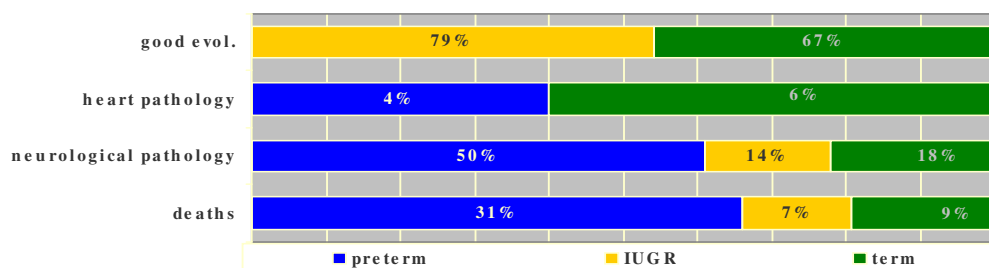


Fig.10. First month evolution (in percentage).

The evolution of the hypoxia-ischemia depends on the number of affected organs, influenced by the functional maturity grade and the individual genetic heritage. Two

preterm (8%) with 2 organs affected died. High rate of decease appears at the newborns with more then two organs affected.

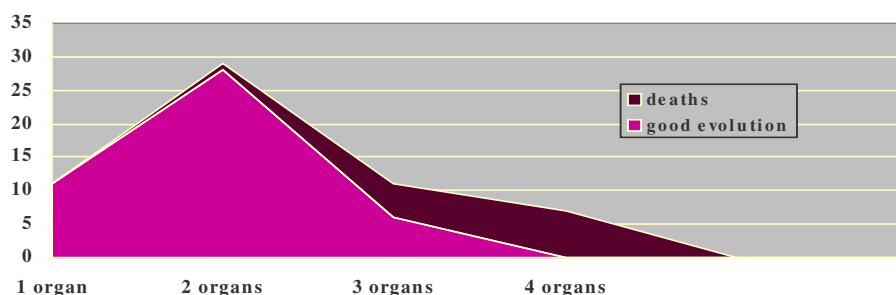


Fig. 11. Corelation between short term and number of affected organs.

Conclusions:

- Clinical trials of interventions must address the phase of injury therapy for reperfusion injury must be given before the insult or early during the insult

- Neuronal rescue strategies to arrest apoptosis to reduce the inflammatory response and to suppress post asphyxia seizures seem most promising

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III. PEDIATRIC SURGERY

CONSIDERATIONS UPON A CASE OF SPONDYLOLYSIS IN A PRE-SCHOOL AGED INFANT

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Abstract

Movement and implicitly traumas of various intensities and etiologies are constant features of childhood.

The affections of vertebral spine are not rare within this period.

Although the clinical picture is not very rich: inconstant pain, especially at mobilization, spastic contracture followed by antalgic posture, the anatomical structure concerned is the vertebral osteoarticular, especially the one from the lumbar area, the pathological substratum being given by phenomena of spondylolysis and spondylolisthesis.

Key words: spondylolysis, spondylolisthesis

Case presentation:

Two years ago, the child G.P., now 6 years of age, male, from Timisoara, came for a medical orthopedic examination because of a painful discomfort in the lumbar area, without recognizing an important traumatic history in the period preceding the clinical examination. The clinical examination shows painful sensitivity at palpation in the L5 region, pain at the dorsal flexia.

It is recommended a lumbar spine radiography, front and side, which does not emphasize anatomical modifications.

It is recommended physical rest, orthopedic surveillance. At the next follow up examination (at three months from the first examination) it is told about the persistence of the painful discomfort that appears during playing, leading to its interruption (when returning from the forward bending position, to rotation moves). The x-ray made in oblique incidence confirms the suspicion of L5 level spondylolysis.

Definition

The spondylolysis represents the interruption of the intra-articular area of the vertebral arch.

Spondylolisthesis = the anterior glide of a vertebral body compared the other one, followed by a possible

anterior throw and the description of a kyphotic deformation.

Spondylos = vertebra, olisthesis = glide

The spondylolysis and spondylolisthesis from the lumbosacral region represent a consequence of the biped propagation. At mammals, this affection is not known, the lumbar spine of four-footed presenting a kyphotic curve. The lordosis from the level of the human lumbar spine seems to be the premise for the appearance of this affection.

Spondylolysis may be accompanied, but not compulsory determined by the spondylolisthesis. The spondylolisthesis is not compulsory determined by the spondylosis. It may be given by dysplastic, degenerative, congenital and traumatic causes.

The etiology stipulates as determinant causes the mechanical and genetic factors.

The mechanical particularities refers to the intra-articular component which in case of a hyperextension lead to an increased closeness of the L4 inferior articular face with the L5 intra-articular component. If this trauma is repetitive, as it happens when practicing sport activities that involve hyperextensions of the lumbar region, there may occur fractures of the intra-articular area, spondylolysis.

Another possible mechanical cause that may lead to the spondylolysis is given by the particular situation from the caudal end of a longer fusion path. A frequent association of the spondylolysis is with the Scheuermann disease, due to the fact that there is at the level of thoracic spine an exaggerated kyphosis that generates in turn an exaggeration of the lumbar lordosis.

The genetical factors show a high incidence of affecting the intra-articular area at the 1st degree relatives. Also, it is observed a high incidence within some populations (Eskimos).

The traumatic causing agent may be both unique and repetitive; when it is unique it generates a spondylolysis situated above L5.

The frequency is located within the white population at 6.4%, within the Afro-American population at 1.1% and at Eskimos at approximately 50%.

A high frequency is also observed at teenagers and among those who practice various sports: artistic gymnastics, ballet, javelin throw, weight lifting, soccer, and skydiving.

The highest incidence of spondylolysis is in early age of childhood.

In patients with cerebral palsy, who present hip flexion contracture, the incidence of spondylolysis increases up to 21% due to compensation of the hip flexion contracture with lumbar hyperlordosis and with the existence of an exaggerated movement, with the purpose of stabilizing the upper body.

Diagnosis, clinical picture

A very small number of patients become symptomatic. When the symptoms appear, they are given by pain of the lumbar spine, that appear during the day, after orthostatism or extended sitting position. Pain is related to typ of motion.

Especially the reclination movement is very painful.

At clinical examination there is an increased local sensitivity at pressing and with easy hitting of the specific area corresponding to the spinal process, usually L5.

If spondylolysis is also associated with spondylolisthesis typical pain occurs at the level of ischiocrural muscles.

By emphasizing the kyphotic changes between L5 and S1, previous migration of the body mass center occurs. In order to correct the anteversion of the pelvis an sustained and extended contracture of the ischiocrural muscles follows, they shorten and become painful.

Also at the level of lumbar paravertebral muscles a painful contracture occurs, caused by the instability of the interrupted vertebral arch.

One of the tests of clinical examination refers to the painful reaction induced by maximum reclination. Another important clinical sign is the lack of painful accuses during forward bending, with the appearance of intense pain when resuming the initial position, pain located in the lumbosacral area.

Radiological diagnosis

In the initial phase, on conventional x-rays, the spondylolysis often cannot be discovered.

On the scintigram there is an increased accumulation area, even before the appearance of the spondylolysis area on x-ray. On the profil x-ray, spondylolysis can be seen when the image was focused on the lumbosacral area and the lysis area is large enough.

The best image of spondylolysis is obtained from oblique incidence.

The most frequent location of spondylolysis is in 95% of cases at L5 level, sometimes associated with olisthesis between L5 and S1. Rarely, within teenagers the location is at level L1-L4 and usually has a traumatic cause. In adult, spondylolysis is located between L4 and L5 and has degenerative causes.

The severity of associated spondylolisthesis is given by the Meyerding classification (that refers to the slide angle and the kyphosis degree).

The kyphosis degree is given by the angle formed of the tangent at the posterior edge of the sacrum bone and the line that passes through the inferior edge of L5 vertebra.

1 st degree	under 25%
2 nd degree	25-50%
3 rd degree	51-75%
4 th degree	over 75% glide, with total glide representing spondyloptosis

Evolution

Spondylolysis frequently remains asymptomatic during the existence even when associated with spondylolisthesis.

The greatest risk for agraveting spondylolisthesis is situated between 10 and 15 years of age, in this period the patient should be clinically and radiologically supervised for an early discovery of lumbar pathology.

Frequently, a scoliosis occurs, usually a lumbar one, probably given by the spastic muscular contracture.

Rarely, spontaneous neurological lesions accompanie spondylolisthesis.

Treatment

In asymptomatic patients with an olisthesis degree of up to 30%, clinical and radiological monitoring is recommended, without limiting sport activity. With the appearance of painful symptoms, the activities that involve more intense physical effort are suspended for a limited period of time. If the symptoms persist an elastic support belt is recommended(for a period of 3-6 months).

In symptomatic patients, the following therapeutic measures may be used: physical therapy, belt, cast, surgical intervention.

The purpose of physical therapy is to relax the spastic muscles and then to work them up. It may not stop the progression of the spondylolisthesis phenomenon.

The movements that lead to the exacerbation of the lumbar lordosis should be avoided.

The belt treatment is recommended in recent cases of spondylolysis in order to give local stability and positive influence to local pain(belt with a light kyphotic orientation).

The patient G.P. underwent kinetotherapeutic treatment, resulting relaxation and consolidation of lumbar area muscles. He was clinically monitored, returning to foolow up examinations at 6 months period. The evolution was positiv, the painful symptomatology was fading until its disappearance. At the radiological follow up examination, there were no signs of spondylolisthesis not even after one year since positiv spondylolysis diagnosis.

Surgical intervention

It is not recommended in milder cases, meaning spondylolisthesis of 1st and 2nd degree. In other circumstances surgery is needed, using the direct osteosynthesis with bolt, the posterolateral spondylodesis or

the ventral spondylodesis, or for spondylolisthesis fusion in situ or repositioning followed by fusion with dorsal

instrumentation.

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EVOLVING SURGICAL TECHNIQUES FOR PECTUS EXCAVATUM CORRECTION

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Abstract

The principles of the surgical treatment for pectus excavatum were established more than 50 years ago, still new treatment techniques and methods are being developed at the present time. Many different materials have been used by orthopedic surgeons for the repair of this thoracic deformity. The present study is a review of both old and new techniques that were designed for a better surgical correction of pectus excavatum.

Keyword : pectus excavatum, surgical procedures, chest deformity.

It is the most common malformation of the thorax, usually diagnosed soon after birth [18].

The first case report of pectus excavatum was made by Bauhinus [19] in 1494, who described the congenital deformity in a seven years old child. Since the beginning of the century, many authors described in detail the anatomy, pathophysiology and the treatment of P.E. [20]

The main characteristic of this deformity is a depression of the anterior wall of the thorax.

P.E. occurs in 86% of cases after the first year of life. It is three times more frequent in boys than girls. 15% of patients have scoliosis and 11% have a family history of thorax malformation. The deformity can be symmetric or asymmetric. Seldom it is associated with Marfan syndrome. [23] There are several theories regarding the aetiology of P.E.:

- the theory of the fibrous sternal-vertebrous ligament of Bauhinus;
- the theory of secondary hypoplasia and atrophy of the sternal-diafragmatic muscles postulated by Brokin;
- the theory of costal dyscondroplasia, which nowadays is most accepted, according to which the deformity of the sternum in P.E. or pectus

carinatum occurs due to an excessive growth of the costal cartilages.

In 1913, Sauerbruch performed the first surgical procedure and was followed by many others who managed to improve the technique, so that nowadays postoperative results are almost perfect. [4] Surgical intervention indications are:

A. Age - experience shows that the procedure can be performed safely in children. The younger the patient, the easier the recovery. This is why surgery is indicated whenever the deformity is severe or progressive. In children, persistence of sternum depression during forced exhalation is as important as the depth of depression or paradoxical movements of the xyphoid [23]. According to many surgeons, the appropriate age for intervention is between 2-6 years, though controversies still exist.

In a study including 12 patients aged under 4 with P.E. who underwent surgery, Haller evaluates complications due to thorax development. Results mention that growth failure of the thorax is the consequence of resection of costal growth center [27]. The author of this article emphasised on the need of delaying surgical correction till the age of 6-8. The younger the patient, the smaller the resection of the chest wall should be. Five patients out of twelve had to accept reintervention for expansion of thorax cage because of developmental failure [27]. Examining the results related to age at operation, several things are clear : the younger the patient at the time of correction, provided he is at least 3 years of age, the better his chances are for an excellent cosmetic result. (Sbokos et al.) [24] The optimum age for correction of P.E. seems to be between 6 and 10 years of age. If the patient is younger than 3 or older than 20 he has less chance of an excellent result. The tendencies are to operate on patients at the extremes of age more for relief of symptoms than in the hope of an excellent cosmetic result. [24] Results obtained in a study including 72 patients are shown in the following table:

Age	Number	Excellent	Good	Poor
<3	6	2	2	2
3-5	17	15	1	1
6-10	23	23	0	0
11-20	21	12	6	3
>20	5	1	3	1
Total	72	53	12	7

Table 1. Age at correction/ long term results relationship [28].

The problem of optimum age for intervention remains controversial, different authors sustaining different opinions:

- Haller obtained good results at different groups of age; 85-90 % are good or very good in children younger than 5 and older than 15;
- Skobos has the best results in children aged between 6 and 10 and the worst in subjects aged under 3 or over 20;
- Von der Oelnitz has the worst results in children aged between 3 and 5, respectively 9 and 12, suggesting optimum age for intervention over 12.

B. Depression severity

- Swenson indicates surgery when sternum depression exceeds 3 cm; between 2-2.5 cm depth the patient should be reevaluated in a year; less than 2 cm - there is no need for intervention.
- Haller indicates surgery if pectus index exceeds 3.25.
- Coexistence with other malformation demands a precise diagnosis, followed by deciding the need of surgery and the order of priorities.

Surgical management of chest wall deformities begins around 1911, when Meyer and Sauerbuch first attempt the correction of P.E. In 1929, Brown, Ochner and DeBakey recommend fixation of sternum after surgery by external traction (Jacobs` frame). In 1945 Lester and

Ravitch suggest radical resection of costochondral cartilages, as well as a posterior transverse sternotomy. The sternum was fixed by lifting it to the level of the second costochondral cartilage. In 1958 Welch recommends that the whole perichondrium be left in place, sternum osteotomy without fixing it with metal plate. To avoid postoperative recurrences, surgeons use several methods to maintain the sternal bone in place (metal plates, Kirschner wire, Steinman nails) [23]. Most of surgical techniques try to solve the cause of thorax deformities, that is the excessive growth of costochondral cartilage. In 1974, Haller fixes costal cartilages at the level of the second rib in overcorrected position, in order to avoid postoperative recurrences. Haller introduces the so-called tripod support cerated to sustain the sternum. This method is based on an oblique incision (condrotomy) on the normal ribs (the last pair of unmodified ribs), followed by overlapping of these segments on the normal ribs and suturing them.

In 1980, Japan, Wada [59] presented his technique called “ sternoturnover”, in which the sternum was used among large numbers of patients as a 180° rotated free graft fastened to the costal cartilages. This technique was abandoned because of postoperative complications, the most undesirable being sternal bone necrosis. [59]

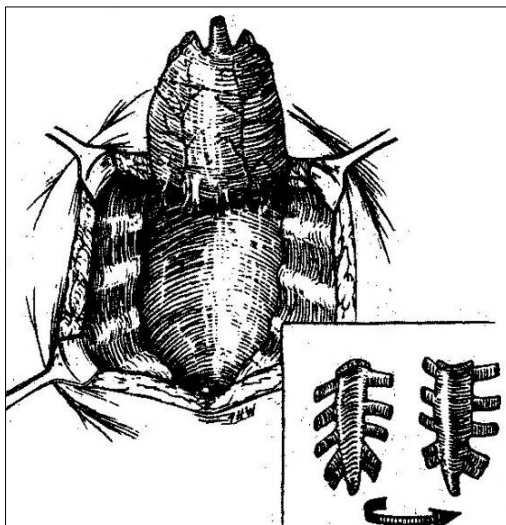


Fig. 1. Sternoturnover-Wada [59]

The so-called „sternoturnover” procedure performed by various surgeons turned out to have poor results, due to sternum and muscle necrosis. Taguchi [41] presents a modified „sternal turnover” technique; he maintains the bone’s blood supply by keeping the internal mammary arteries on both sides. This procedure is mostly recommended to patients older than 15 years. The operative technique consists of :

- Vertical submammary incision is made in boys, transverse incision in girls;
- Dissection of pectoral muscles and exposure of skeletal chest wall;

- The rectus abdominis muscles are divided and the retrosternal space is prepared, thus creating a tunnel in the anterior mediastinum and separating at the same time the pleura from the anterior skeletal chest wall;
- From the first to the third ribs are divided bilaterally at their junction with the cartilage;
- The sternal bone is freed at the level of the third rib by a transverse sternotomy, keeping the internal mammary arteries;
- Internal mammary arteries are carefully prepared 5 cm cranially and 2 cm caudally ; the deformed sternum is turned 180° over, taking care not to section arteries;

afterwards, the costal cartilage is fastened to the edge of the sternum with wire or dexon.

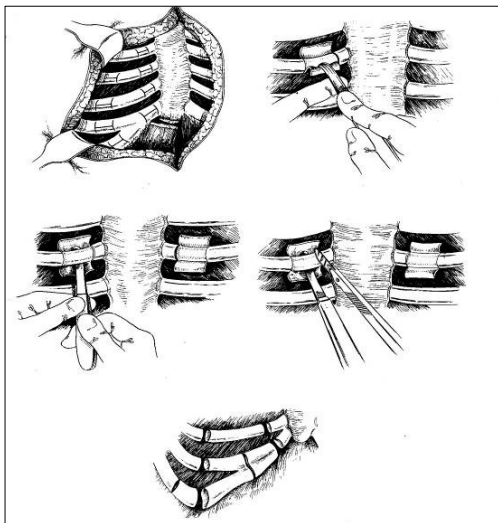
It must be emphasised on the importance of checking the blood supply after sternum turnover. Finally, the excess of bony tissue at the level of the sternum should be removed, otherwise the thorax would look like some kind of a pectus carinatum.

Eric Fonkalsrud published a study over 25 years including 252 patients who underwent surgery using Ravitch technique, modified by Welch and Haller [33]. The operative technique consists of :

- Submammary transverse incision ;
- Elevation of cutaneous flap from muscles, avoiding major bleeding by electrocoagulation;
- Pectoral muscles are dissected free from the sternum and costal cartilages are reflected sideways;
- The perichondrium is incised longitudinally, bilaterally on its anterior surface at the level of the last 4-5 costochondral cartilages; deformed cartilages are resected subperichondrially with the aid of an elevator;
- The xyphoid process is detached from the sternal bone;
- The perichondrium and intercostal muscles are resected medially to the internal mammary arteries;

- The retrosternal space is mobilized;
- The pleura is incised on the right and a drain tube is inserted;
- A transverse sternotomy is made at the level of the last normal rib;
- The posterior tabula externa of the sternum is fractured, without completely interrupting its blood supply, thus enabling to handle the sternal bone in desired position;
- The anterior sternotomy is sutured;
- In children aged under 5, the perichondrium from the 5th to the last rib is sutured on the midline;
- The xyphoid process and the perichondrium **teci** are reinserted;
- The pectoral and rectus abdominis muscles are sutured in normal position;
- The wound is closed in layers, and the drain is removed 3 days after surgery.

Average surgical time required in forty interventions was three hours and average time for hospital admission was about five days, during which antibiotics were given to patients. Attentive skin suture reduces the incidence of inaesthetic scars.



Picture 2. The surgical procedure [35]

Professor C. Coman from Romania describes another procedure for the management of P.E. [25]:

On one hand, midline incision is abandoned because of poor aesthetic results, thus avoiding delayed healing and scars. On the other hand, transverse incision is considered insufficient to expose the interested area. Specific for this procedure are the two parasternal incisions made along the costal arch, leaving the sternum covered and allowing at the same time removal of cartilages in good circumstances.

The main difficulty encountered by the author is the failure of preserving the perichondrium.

Nevertheless, the following technique seems to offer better results:

-procedure starts with incision at the costochondral junction with the special mention that the perichondrium must be entirely left in place.

-at this level, the cartilage is severed up to the posterior perichondrium.

-cartilage is lifted and the posterior perichondrium is digitally dissected to its junction with the sternum.

-the cartilage is mobilized from its joint with the sternal bone, lifting its sternal end and thus freeing the anterior perichondrium. This technique allows preservation of the perichondrium, having a decisive role in postoperative results. Resection of xyphoid process is not compulsory, thus avoiding detachment of rectus abdominis muscles, which might lead to postoperative hypotonia. It ought to be mentioned that in case of a severe deformity

where the xyphoid process complicates the correction, its resection must be carried out. Next, the retrosternal space is dissected by blunt finger dissection, starting at the resected cartilages. The sternum is mobilized and brought to overcorrected position by transverse pyramid-shaped sternotomy at the level of the second or third intercostal space. Sternum is supported by retrosternal stainless steel

plates (C. Carpinisan et al.) In case of asymmetric deformities, the sustaining plate is obliquely, medially and upwards oriented. Symmetric deformities, usually occurring in patients with increased longitudinal thorax diameter, are solved by inserting two parallel metal plates. Drain tubes are inserted in the retrosternal space and at the site condrectomy was performed.

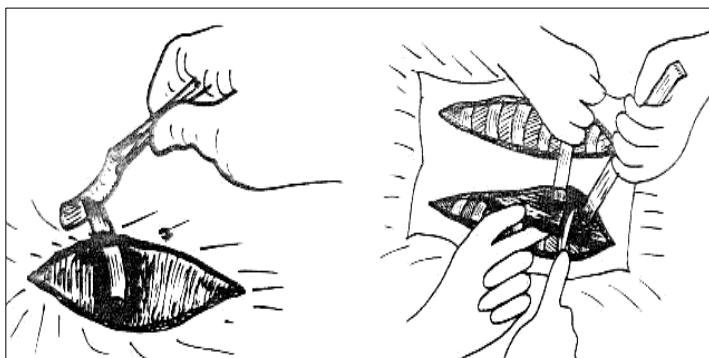


Fig.3 Coman technique [25]

Kobayashi describes a minimal invasive procedure for the management of P.E., coming up with a partial costal cartilage resection and sternal osteotomy, the entire intervention being carried out through a 2.5-4 cm skin incision. [37]

The incision is located just above the xyphoid process, the procedure being assisted by an thoracoscope.[37]

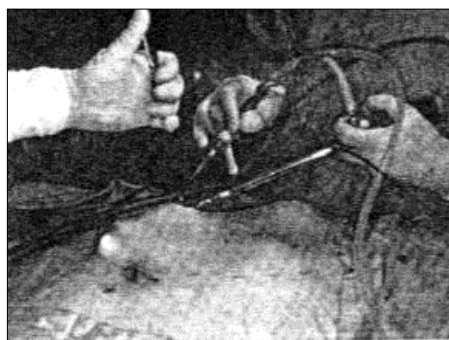
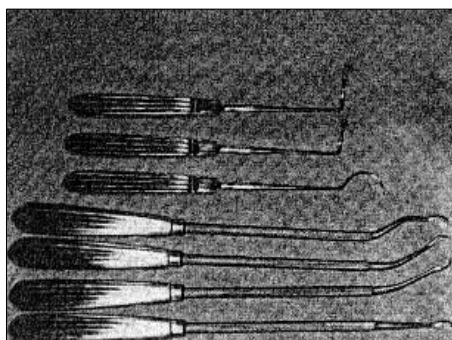


Fig 4 Instruments used by Kobayashi [37]

In addition, stab wounds of less than 3 mm in diameter are also made, in some cases, for insertion of the surgical instruments to facilitate the operation. The ribs and rib cartilages are exposed beyond the affected area through a prexyphoid incision under thoracoscopic control. The mobility of the skin incision site is thus enhanced, and it is possible to perform most of the subperichondrial partial costal cartilage resection under direct visualization. [37]

A retrosternal support is also used for keeping the sternum in desired position. The authors use specially designed curved perichondrial elevators for perichondrium resection.[37]

The pectoralis major muscle and the rectus abdominis muscle are incised where they attach to the sternum and ribs. Next, the subperichondrial space is

prepared, followed by an oblique incision of the costal cartilages and removal of excess cartilages. [37]

The authors mention the difficulty of resecting the superior-most costal cartilages (third or fourth) due to the low location of the initial incision. [37]

The costal cartilage resection with the endoscope is facilitated by the use of a surgical arm that holds the endoscope. Finally, a wedge osteotomy of the sternum is performed at the point of maximum curve deformation of the sternum. The deformed sternum is corrected manually by performing a green stick fracture at the osteotomy site. The elevated sternum is maintained by a strut that is inserted into the retrosternal space. Finally, the divided xyphoid process and muscle are sutured to their original point of attachment. Skin is closed in two layers, and two suction drains are inserted into the subcutaneous and

prepleural space. The thoracic cage correction results obtained using this procedure were the same as those obtained using the conventional open method, except for the minimized operative scars. Specifically, the operative time was longer for those patients in whom subperichondrial dissection of the superior-most third or fourth rib cartilage was needed, given the fact that this area is hardly accessible through a prexyphoid incision. [37]

The long transverse or longitudinal incision scar observed among patients who have undergone surgical treatment of P.E. is unfortunately inevitable when correcting a deformed thoracic cage using conventional methods. This is why the authors of this study recommend the endoscopic procedure whenever is possible, provided there are proper instruments and an experienced working team. [37]

The advantages of this technique include minimal operative scars and the ability to free the pleura from the sternum under endoscopically magnified visualization,

which prevents rupturing of the pleura in the retrosternal area. The disadvantages of the technique are increased surgical time, possible pleural perforation and an increased blood loss in elder children. Postoperative results after long-term follow-up (18 months) were good. [37]

The use of the Marlex mesh for the support of the sternum after PE surgery was introduced by Francis Robicsek . A transverse cuneiform osteotomy of the sternum at the beginning of it's abnormal downward curve is performed . Care is taken to do the osteotomy in a line that falls to an intercostal space rather than at a chondrosternal junction.

The xiphoid process is detached from the sternum and is allowed to retract downward. The tip of the sternum is then lifted with a towel clip and with blunt and sharp dissection , the sternum is freed of its mediastinal, perichondrial and intercostal attachment.

The bone is then bent forward to a slightly overcorrected position.

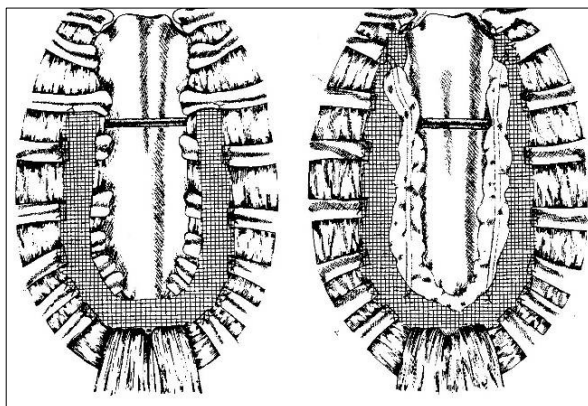


Fig 5 Marlex mesh support [38]

The right pleural cavity is entered deliberately and drained through an intercostal water-sealed catheter to prevent accumulation of blood in the wound itself and to assure undisturbed wound healing. [38]

To stabilize and maintain the sternum in its corrected position , a piece of Marlex mesh is cut to approximate size, placed under it and sutured under slight tension to the distal ends of the divided costal cartilages with nonabsorbable heavy filament. If the pectoralis muscles are well developed, they are joined together in the midline, above the sternum . If they are hypoplastic, they are reattached to the sternal edge. [38]

Long-term follow-up was obtained for 161 patients who underwent a modified Ravitch repair of pectus excavatum. In all cases, the substernal strut was fashioned from a Dacron vascular graft. [39]

A piece of Dacron vascular graft , usually 8 or 10 mm in width, was sutured taut, to the 4th and 5th ribs, to form a substernal strut, using Vicryl or Dexon sutures. The wound was closed in the standard fashion, over subcutaneous drain. [39]

The follow-up period was 12 months to 21.5 years. One hundred thirty-three patients (83%) had satisfactory chest contour; 17 had a fair result, and 11 had frank

recurrence. Risk factors for poor contour were an asymmetrical defect, a severe defect, and associated congenital anomalies. [39]

The use of a Dacron strut is relatively easy and complication-free. It can be left permanently in situ. Long-term results have been good, except in patients with an extensive or asymmetrical deformity, and in those with other congenital anomalies, especially Marfan's syndrome. [39]

Isakov et al. introduced in 1980 a new technique for the repair of PE with the aid of permanent magnets.[40] The rib cartilages are mobilised after moving sternal muscles aside Double chondrotomy (near sternum and along the external boarder of the deformation) of deformed rib cartilages is performed when the deformation is flat .If the deformation is “deep “ the rib perichondrial resection of rib cartilages is performed . [40]

In all cases cross sternotomy is made by Gigli saw, at the level of the upper boarder of deformation. The magnet plate is put into the retrosternal space so that the upper edge is situated at the level of the sternotomy and it's lower one does not protrude from beneath the sternum more then 0.5-1.0 cm. [40]

The wound is sutured without drainage. The brace with external magnetic system is put on when the patient is on the operating table. The external magnetic system is set up so that the sternum is on the state of moderate hypercorrection and it does not float during breathing .

The extension of sternum is made from one month in younger patients to 1.5 month in older ones. The patient is allowed to sit up 3 days after the surgery and walk after 5 days .The plate is removed between the 6 and 8th month after the operation. The number of patients operated with this technique is 35. The results according to the author were good in 33 patients. [40]

In 1992 M.L. Bentz publishes an article in the British Journal of Plastic Surgery in which he underlines the fact that the vast majority of patients with PE undergo surgical treatment because of esthetical reasons and analyses the opportunity for plastic surgery approach for the treatment of this condition.

An approach for the treatment of PE with regards to esthetical appearance is proposed by plastic surgeons in cases in which no respiratory or cardiac symptoms are associated. The first case reported belongs to Murray in 1965 when he describes the use of silicon implants.

Chavoin [42] reports great results on short term as well as on long terms using the silicon implant for the treatment of PE.

The procedure begins with the measurement of the skin thickness in the area of the sunken chest using a special clipper . This is a very important step because of the fact that the thickness of the skin varies as we move towards the lateral side .If this fact is not taken into consideration the implant might not have a correct position on the sternum. [42]

A cast of the sunken region of the chest is made with plaster and shaped manually into the desired shape. The final implant is made of silicon. [42]

For the positioning of the implant a small incision is made at the level of the xiphoid, anterior to the insertion of the abdominal muscles. The muscular mass is dissected until the costal cartilages are evident and the pectoral muscles are detached up to the second rib. [42] The implant is then inserted into the obtained pocket under the muscles and anchored in place. If the muscles are well developed they can be sutured on top of the implant hence giving it a better anchorage, if they are hypoplastic then they are anchored laterally.

External compression is sometimes required for short amounts of time after surgery using sand bags. [42] The results according to the authors are very good.

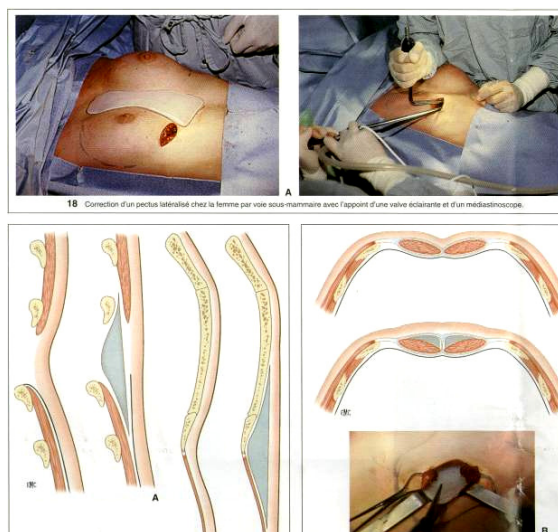


Fig. 6 Silicon implant for the correction of PE [42]

Yamamoto et al. introduced in 1995 the technique for the correction of PE using instead of soft tissue augmentation of the depressed chest a deepithelialized latissimus dorsi myocutaneous flap. The muscle was harvested from the left side because the majority of patients were right handed. [43] The skin at the anterior borders of the latissimus dorsi muscle was incised and the thoracodorsal vessels and nerve were preserved . The whole body of the muscle with overlying adipose tissue was

harvested ,and the insertion of the muscle was detached from the humerus . [43]

The skin islands was deepithelialized , and the pedicle was transferred to the anterior chest through a subcutaneous tunnel . Then the flap was adequately tacked with 4-0 nylon to the pectoral and abdominal fascia to repair the sternal depression through bilateral periareolar incisions , a small skin incision at the supraumbilical area, and the skin incisions where the latissimus dorsi muscle flap was harvested. [43]

The most important problem with this technique are the atrophic changes that take place in the transferred muscle.

Reasonable certainty of an excellent results can be ensured only if a generous overcorrection of greater than 50% volume is transferred to the pectoral region. [43]

Hayashi et al. uses a vascularized rib strut for the repair of PE. [48] A vertical midline skin incision is made to obtain maximum exposure. A transverse inframammary incision may be used in young female patients. The procedure of correction of PE is basically a modification of the Ravitch technique. This includes subperichondral removal of all abnormal costal cartilages, suprasternal incision and manual mobilization of the substernal space, division of the intercostal bundles, oblique transection of the costal cartilage at the lowest normal rib, transverse anterior cuneiform sternal osteotomy and tripod fixation of the sternum.

The authors dissect the intercostal bundles medial to the IMA on one side to preserve the communication between the IMA and the anterior intercostal branches and lateral to the IMA at the other side to maintain the circulation to the sternum. [48]

After correction of the PE deformity the seventh rib, usually on the left side, is harvested as a vascularized rib strut. A 5cm lateral chest incision just above and parallel to the seventh rib is made and the periosteum is exposed for a length of 15 cm. In the upper margin of the rib, the intercostal muscles are dissected and then the space just anterior to the parietal pleura is divided bluntly to the next lower intercostal space. [48]

An intercostal muscle cuff of 15 mm width is attached to the lower margin of the rib to avoid injuring the intercostal vessels. A flap is raised from the chest wall after the rib is cut posteriorly, and then the internal mammary vessels are severed distal to the bifurcation of the corresponding anterior intercostal branch. The rib strut is positioned under the distal portion of the mobilized sternum and is anchored bilaterally to the fifth ribs with wire sutures. Wire fixation of the sternum to the underlying strut is recommended. The incisions are closed in layers with a suction drain tube placed behind the sternum. [48]

Over the past 25 years, Arnold S. Leonard designed a new operation which does not violate the chest and is combined with a bracing technique. This operation can be carried out in children between 45 minutes and an hour and in adults between 1 and 1 hour and 15 minutes and only requires 1 day hospitalization.

The incision is a bilateral transverse curvilinear incision beneath the breasts, which gives a good cosmetic scar. The lower 4-5 cartilages are removed and the perichondrium or the covering of the cartilages is left in place. Then a wedge osteotomy or wedge is taken out of the sternum and depending on whether there is asymmetry the sternum is tailored obliquely according to the defect. A sheathed wire then is placed behind the sternum and then brought out through the muscles and skin and later attached to a modified brace for a period of 12-15 weeks depending on severity. During that period of time, the cartilages reform in the new position and the defect, thus, is

completely corrected. The wedge osteotomy is sutured appropriately. The patients are fit with a brace prior to surgery which is a light vest to which the wire is attached at surgery. Patients can return to work within a week after surgery and children may go back to school within that period of time. Blood administration is unnecessary. The complete healing period is 3 months after which individuals can return to their normal activities. Recurrence is very unusual with this operation because of the wedge osteotomy and the holding of the position by the wire and vest.

In 1994 Matsui and M.Kitano [51] propose a new technique for the repair of PE using a poly-L-lactide plate. Poly-L-lactide, a polymer of lactic acid, shows slow degradation in living tissue. Poly-L-lactide plate of high molecular weight maintains more than 90% of its initial mechanical properties for more than 3 months after implantation. Using struts made from poly-L-lactide plate, he performed chest wall reconstruction in 56 patients: for postoperative chronic sternal dehiscence in 23 and sternal elevation for pectus excavatum in 33 cases. [51]

The postoperative external appearances of the anterior chest were improved in comparison with the preoperative state in all cases. The internal features were evaluated by computed tomographic scan. Neither postoperative wound infection nor respiratory complication was observed, and no tendency for regression of the anterior chest occurred in any of the patients.

In 3 of 56 cases (5.4%; one in the sternal dehiscence group and two in the pectus excavatum group), it was necessary to remove part of the strut because of overgrowth of granulation tissue around the implanted material after 4, 12, and 13 postoperative months, respectively.

In the pectus excavatum group, the computed tomographic evaluations showed that poly-L-lactide strut maintained sufficient strength to support the thoracic wall 5 months after implantation. These findings suggest that the bioabsorbable poly-L-lactide strut is a promising material for surgical treatment of chest deformity. [51]

In 1999 Donald Nuss [55] presents to the world probably the most efficient surgical technique for the repair of PE without the resection of the costal cartilage, with minimal blood loss and with record operating time. Prior to surgery, a stainless steel bar (Walter Lorenz Surgical) is bent to conform to the contour of the patient's chest at the level of the deepest part of excavatum. The bar is selected so that its length is sufficient to extend from the mid-axillary line on one side of the patient's chest to the mid-axillary line on the other side. For the operation, the patient is supine with the arms abducted. Incisions are made on the sides of the chest between the anterior and posterior axillary lines. Large subcutaneous pockets are created anteriorly and posteriorly to accommodate the bar. A clamp is inserted through the intercostal space in line with the deepest point of the concavity and passed across the mediastinum directly behind the sternum. The passage is monitored with a thoracoscope. The point of the clamp is pushed through the corresponding intercostal space on the opposite side of the chest.

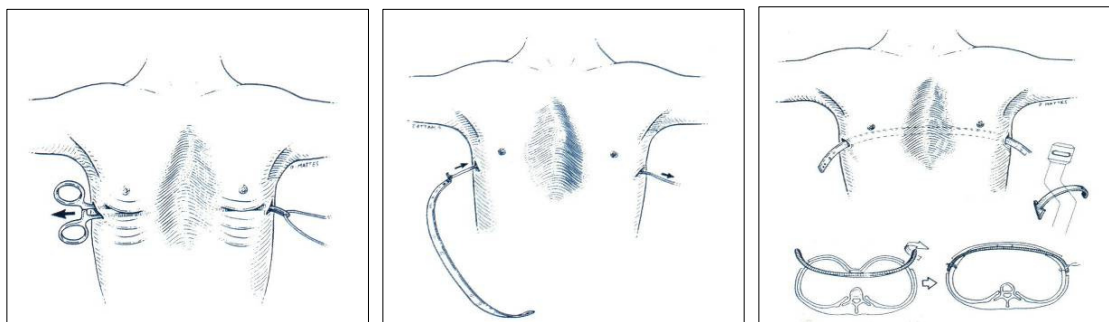


Fig. 9 Nuss procedure [55]

An umbilical tape is then tied to the bar, and the bar is guided across the mediastinum. The convex side of the bar faces posteriorly as it traverses the mediastinum. The bar is then rotated 180 degrees with a vise grip or rotational device so that the convex surface elevates the sternum. Before the incision is closed, a positive end expiratory pressure of 5 cm of water is added to eliminate air from the chest. The surgical wounds are then closed in layers. Analyzing Till's results [56] with the Nuss technique we noticed a minimum blood loss, an average surgery duration of 57 minutes, no complications during surgery, very good esthetic results, no trauma for the patient with a fast recovery and without wide incisions. Thus, it can already be affirmed that this is the actual

golden standard for the surgical treatment of pectus excavatum.

Conclusions

Today, high efficiency means an early discovery of the growth disturbances of chondrocostal cartilage and their immediate solving, preferable with minimum invasive procedures. Both the open technique and that minimum invasive– Nuss, require periodic and systematic reevaluations with the purpose of increase in surgical performance, meaning a maximum reduction of application difficulties as well as a decrease in complication during and after surgery.

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IV. PSYCHOLOGY

THE PSYCHO-AFFECTIVE LIFE OF THE CHILD BEFORE AND AFTER BIRTH

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Abstract

Many observations suggest that the psycho-affective life of the child begins before birth, being dominated by the fetomaternal and even the fetoparental relationships and after birth the mother's speech based on its sonorous qualities, gains an emotional significance and it is affectively linked to the child. The child develops naturally, passing through stages that succeed in constant order.

Key words: psycho-affective life, fetomaternal relationships, fetoparental relationships

There are already many observations which suggest that, in fact, the spiritual life of the child begins before birth, being dominated by the fetomaternal and even the fetoparental relationships.

We can give some examples: the clear and differentiated reaction of the fetus to some sounds - the music of Antonio Vivaldi and that of Wolfgang Amadeus Mozart has a calming, relaxing effect on the fetus, while the music of Ludwig van Beethoven and that of Johannes Brahms causes movements and striking with the foot even at the most peaceful fetuses.

Observations along centuries stress the repercussions which anxiety and fear experienced by the pregnant woman could have upon the fetus: for example fire could disturb the pregnancy and the child's temperament.

The relaxing, cheering-up and enlivening effect a lullaby has upon the child, mainly if it is the same lullaby the fetus heard from the mother around his 7th intrauterine month of life, is also a good example.

The first who understood the importance of these terms related to prenatal psychology was Leonardo da Vinci who, in “Cahiers”, wrote with a remarkable intuition: “The same soul rules the two bodies - the things the mother wishes are often imprinted in the child she carries, every wish or fear of the mother and every spiritual pain touch heavily the child.”

If a mother listens to some musical works daily during her pregnancy, the child will develop a constant taste for music, which may cause excitement and restlessness. The case of the conductor Boris Brett from the Hamilton Philharmonic Orchestra (Ontario) who said that music was part of him already before birth (his mother was also a musician - she played the cello) and that he could recall musical works from the first age is a very good example. Other musicians as Arthur Rubinstein, Yehudi

Menuhin have said that their taste for music had already been woken when they were sucking from their mothers.

After 1960, the medical technology has progressed and has brought verifiable proves that can certify that the fetus is a human being capable of auditive, sensorial and affective reactions. These discoveries have given a new authority to prenatal physiology and psychology.

For example, at the age of 6 months the fetus moves his arms, head, back, using primitive gestural speech to express taste and dislikes through foot kicks. At this age, the ear of the fetus is always on “watch”. The noise that dominates his universe is represented by the rhythmic, muffled heartbeats of the mother. As long as the noises are harmonious, steady, the fetus feels save.

The eyesight develops more slowly because of obvious reasons. When the mother is exposed to sun, when the sun rays touch the mothers abdomen, the fetus turns his head. Cardiac rhythm variation at the fetus have also been obtained through the projection of sparkling light on the pregnant woman's abdomen.

The fact that the fetus is capable of sensorial reactions shows that he has the necessary elements to be able to learn. The first elements of memory begin to arise in the brain of the fetus in his 3rd trimester of intrauterine life.

In a study of the Czech psychiatrist, Stanislav Graf a case of a girl is presented who (under the effect of medicine) has described precisely her fetal body. The girl said she heard noises and sounds from the exterior (laughter, joyful yells and trumpets). When asked the girl's mother confirmed those said by the girl adding that the excitement during the carnival caused her premature delivery.

It is known that starting with the 8th month of pregnancy, before delivery, some memory schemes are functioning following recognizable models.

The fact that we are not capable to remember precise events or situations from the period before birth does not mean that they are irretrievably lost. Even profound and old memories maintain an emotional resonance. If they escape of being kept in the voluntary memory, it is because of a substance of the organism called “ocitocine” which rises during delivery and which, together with other effects, causes amnesia.

Our ability to find again these memories later is related to the natural production of ACTH, another

substance that could have an opposite effect to that of ocitocine.

As in nature, where everything takes place from simple to complex, the child in uterus passes on to an emotional level from the small age fetus - incapable of sensations, to a human being capable to record and decode feelings and shaded emotions. This developing process is named the personality or "self" - forming. The "self" represents a sum of what an individual thinks and feels about himself. The personality begins to function in the intrauterine life.

Affectivity has characteristical phases. At the age of 2-3 months, after the appearance of the smile, new features appear on the child's face but, at the same time, he has new demands.

At the age of one year the child asserts his "self" and shows opposition through the word "no". The first steps done by him will enlarge his autonomy, he will have own experiences that he will repeat and enrich. He will turn toward everything that is sparkling, colored, tasteful, striking.

This period is prolific, but also fragile. A tendency of referring everything to himself (egocentrism), a very serious phenomenon, becomes visible in this period, a phase necessary for building personality. In time, the child learns to offer in order to get, he learns to sacrifice pleasure in order to please others. This improvement has long-term effect.

In normal life and normal educational conditions, the child differentiates numerous sounds, he retains them and knows the mother's voice because this is related to the satisfaction of his needs. After the 2nd month of life and in the following months the sounds of the child are characterized by specific modulations which imitate the musicality of the mother's speech.

The mother's speech based on its sonorous qualities, gains an emotional significance and it is affectively linked to the child.

Using sonorous games, toys, the activity of the ear can be stimulated (ex. xylophone). The music from the radio can unleash positive emotions if it is simple, balanced, joyful, being at the same time a good training for the child's hearing, for a more and more subtle differentiation of sounds.

After the age of one year, the psychic activity of the child has a huge development, it is the period of fast gain of words with meaning for the child. Now the child pronounces clearly about 10-20 words, related to diverse objects. He knows the name of many objects, but he cannot reproduce them yet.

Between 1½-3 years of age, the thinking and the speech of the child develop in a fast rhythm, both in the field of understanding the speech of the adults and in that of the number of words he already masters. He also learns grammatical structures. The child understands better and more exactly the adults' speech, which is related to events, to life and his own experience. In order to understand easier the meaning of the words it is necessary that they are related to images and objects. At this age, the child thinks acting.

After he walks and when he learns to speak his relations enlarge. He comes in contact with a larger number of adults and children, he establishes contact with those around him through verbal communication. The word becomes the way to communicate with those around him.

Labels of ideas, words have been modeled so that people can understand each other. Through their function words note what we have in common, what we already know, terms which they can through a common and agreed designation recall in each of us. Hobbs has defined the word as: "symbol used by people in order to serve as a sign that can rise in mind a similar thought as one known before and which, put in speech and told to others, is a sign indicating the idea of the person who speaks."

Words had to be kept away from the affective fog of our interior life, of everything that meant emotion, individual reaction, in order to keep just its central core, with an infinitely smaller volume but which compensates through its fermity and clearness, it corresponds to delimited and controllable experience: a restoration of the senses or the general idea that could be detached out of it. Speech, as intelligence, had to be consecrated to the field of the object to find a solid ground.

Human ingenuity has striven to live the gift of words of expressing more than just the simple fact or the abstract idea, they hide, tends to transmit, as Proust wrote to Anton Bibescu: "a certain quality of the vision, a revelation of the intimate universe that each of us sees and the others can not see."

The spiritual field exceeds ideas and cannot be reduced without losing its deep value. If a time ago the rationalist spirit ignored the unconscious and its right to expression, the modern spirit penetrated by positive civilization, hardly admits that the spiritual is not to be mixed up with the intellectual and sometimes it can not be reduced to the last being superior to it. This contains the peaks of our interior life, those in which we see spaces where thinking weakens.

The unconscious and the spiritual, the essence of the individuals interior life, are damned to silence because of the lack of common language, if it is not freed and it does not show art and its images. Just art, poetry, can detach them from the everyday life and give them significance, exterior transposition. This is why art has always been chosen for religious relevance and intimate confessions of everything that exceeds the sensorial or rational evidence which alone belong to the word. Religion, as Malraux said "is based on states of the soul... and an essential fact - the Sumerian statues speak to us, although the resonance the universe had upon the soul of a Sumerian priest disappeared forever, because a lecture of religions from Sumer and Egipt don't tell us anything, anymore. A religion, a believe are "the music of the hymns that was maintained through metamorphoses just in great works."

The physiological education prepares directly the psychic education, the perfectioning of the sense organs, the nervous ways of protection and association. Indirectly, the adapting of the individual to the environment is obtained because the childhood of the nowadays humanity is prepared. The population of the present civilization is an

observer of the environment because they must use its treasures as much as possible.

Both science and art are based on the observation of the truth. It is necessary that the education of the senses be started during childhood if we want to perfectionate them later through education and to apply them to the particular forms of culture.

It is sometimes said that intelligence becomes useless if it lacks practice and this practice is almost always the education of the senses.

At adults the sensorial education is difficult as it is difficult to educate a hand of an adult who wants to become a pianist. A physician can be well prepared theoretically without being a good practitioner. A good practitioner needs long exercise, a good education of the senses: visual, hearing, sensitive in order to observe phenomena and to be able to distinguish them in practice.

Lacking the capacity of discriminating sensorial stimuli, study and will are not enough. This is the cause of discourage, disillusion of young people in practicing a profession with such high responsibility.

Esthetical and moral education are closely related to sensorial education. Multiplying sensations and developing the capacity to assess the small quantitative differences between stimuli develop sensibilities, which increase satisfaction.

Esthetical harmonies in nature and in art are not observed at those with low education. In the environment, there are springs of esthetical joy that some people pass by indifferently, looking for tough sensations, the only ones that are easy to access for them. Often gross pleasures are the source of vicious habits, because strong pleasures do

not sharpen, on the contrary, they blunt the senses and that is why stronger stimuli are always needed.

Senses are organs for "catching" signals from the exterior world, necessary for intellectual knowledge, as the hand is the organ to catch material things necessary for the body. But senses and hand can reach a level of refinement that exceeds the existence of simple tasks, becoming aristocratic instruments of the huge motor that puts them in their job.

Education that rises intelligence must do the same with the two means susceptible of unlimited perfecting.

In his works concerning pedagogy J.J.Rousseau said: "it is not enough to have organs, we must know .to use them and the more developed these organs are the more precise and delicate the work done by them is, the period necessary to make them functionable is longer. Function is one of the conditions of forming structure, these two aspects are inseparable."

Both for Rousseau and for other authors after him, development implies stimulation, a continuous exercise of the organs in order to be developed. The period reserved by nature for this is childhood, so we must respect it. "Let nature work much before interfering to work instead it as not to vex its operations. Observe nature and follow the way it goes. Nature wants children that are children before being men. If we want to change this order we will produce fruits before it is their time, and they will have neither maturity nor taste."

The child develops naturally, passing through stages that succeed in constant order. These steps synthesize those passed by the spirit of humanity.

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MANUSCRIPT REQUIREMENTS

The manuscript must be in English, typed single space, two columns (equal width – 8,5 cm, line between and spacing – 0,8 cm) on A4 paper, with margins: top – 3 cm, bottom – 2,26 cm, left – 1,5 cm, right – 1,7cm. A 10-point font Times New Roman is required.

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