

Newborn surgery

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The main issues of newborn surgery are inflammatory illnesses, injuries, tumours and the most important **congenital developmental defect** by which suffer approximately **?%** of all newborns.



The main issues of newborn surgery are inflammatory illnesses, injuries, tumours and the most important **congenital developmental defect** by which suffer approximately **2%** of all newborns.



Dividing of newborn surgery

- Newborn neurosurgery
- Neck surgery
- Thoracic surgery
- Cardiac surgery
- Abdomen chirurgy
- Urinology
- Musculoskeletal system surgery

Classification of CDD



Simple abnormality

- **Malformation** (development of organ or tissue was stopped or was abnormally exerted – e.g. defect of atrial septum, cleft lip, ...)
- **Disruption** (external factor consequences – Cocaine – vascular supply of exerted structures suppression – enteric atresia)
- **Deformation** (mechanical entrapment of organ - pes equinovarus with oligohydramnion)
- **Dysplasia** (abnormal cell organization to the tissues – multicystic renal dysplasia)

Multiple anomaly

- **Sequences** (consequences of cascade of developmental changes activated by one anomaly or by mechanical entrapment – Pierre Robin Sequence – primarily, lower jaw anomaly leads to the lack of space for tongue development which expels cranially and gothic palate up to cleft palate is being created)
- **Syndrome** (multiple malformation pathogenetically connected with one cause – e.g. Down's syndrome chromosome aberration)
- **Association** (nonrandom occurrence of types of malformations which are not pathogenetically connected
- e.g. VACTERL association during omphalocele)

Serious CDD	- cardiovascular	1%
	- CNS	1%
	- GIT	0,4%
	- Limbs	0,2%
	- Urinogenital	0,4%

Less serious 10%

preauricular excrescence
 lacrimal canaliculus stenosis
 umbilical hernia
 hydrocele
 hollow in sacral area
 syndactyly of 2nd and 3rd toe
 supernumerary nipple
 ...



„Zdravý určitě nejste, protože dnes už je medicína tak pokročilá, že zdravý člověk neexistuje.“

„You are not definitely healthy because the medicine is so advanced that there no longer exists a healthy human.“

Causes of CDD

Genetic

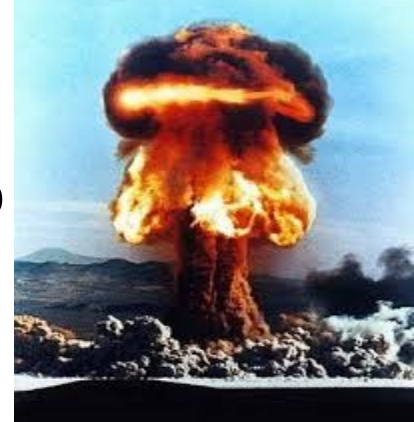
30-40%

- Chromosomal
- Disfunction of one gene
- Multifactorial



External environment influence 5-10%

- Drugs and chemicals
- Infection
- Physical factors



Unknown

50%



CDD Prognosis

Depends on the type of the defect, not always estimated

In general, at serious VVV is not encouraging

25% dies at the early infantile age

25% proves after-mental or physical disability

50% has acceptable or good prognosis during right therapy

Anatomical differences

(find 10 differences)





Anatomical differences



uf...

**Head – great, facial part to cerebral part 1:8,
floating sutures, fontanelle**

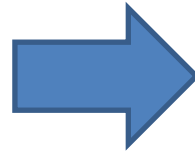
**Chest – without noticeable sulcus,
chest/abdomen crossing is not sensible, ball
shape heart, thymus, more mediastinum in
thoracic cavity**

**Abdomen – the biggest width in the liver
level, great livers, canal gallbladder, higher
position of caecum, large intestine dilated by
meconium, larger adrenal gland, small
omentum, musculature is not noticeable
externally, transverse dermal sulcus in lower
area, panniculus adiposus in pubic area**

Limbs – relatively short



- Significantly smaller measurement
- Delicate fragile tissue
- Blood loss !
- Thermoregulation !
- Restricted options of vascular access
- Small manipulative space



- **Special technical equipment in the workplace**
- **Informed team of doctors and paramedical staff**
- **Specific surgery technique**
- **Specific postoperative care**

We are having a baby

it could happen to you as well



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20.t.g – US screening

- Labour in 40 t.g., ph 3050/50cm
- AS 8-9-10
- Postnatal adaptation in normal
- 4 cm defected abdominal wall, which prolapse skin livid intestinal villis clenched to solid adhesions
- Suspicious eventration of fetus intestinal villis prenatally according to the US



Gastroschisis , omphalocele

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gastroschisis



- Defected abdominal wall from funis (90%dx)
- Changing intestinal wall (both anatomically and functionally)

omphalocele



- Defected abdominal wall in umbilical cord area
- 30-70% + VACTERL
- Anatomy and function of intestine in normal

Gastroschisis+omphalocele therapy

Loop reposition to the abdominal cavity and its closure

- **Small defects** - primordial closure
- **Great defects** with small abdominal cavity – often surgeries with progressive organ reposition (due to abdominal compartment sy.)

Conservative procedure with unstable newborns only – temporary hanged omphalocele bag packing with physiological saline after stabilized surgery state.

Gastroschisis

1st stage



Gastroschisis

1st stage



Gastroschisis

2nd stage



Gastroschisis

2nd stage



Gastroschisis

3rd stage



Gastroschisis

3rd stage



Gastroschisis

final state after two weeks



Omphalocele one time surgery



Gastroschisis one time surgery



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- Labour in 39tg, ph3500g/50 cm
- AS 9,5,4
- Progressive poor breathlessness and cyanosis
- Heavily breathless newborn after 15 minutes
- Weak bilateral breathing
- The state is getting worse after breathing with mask



Diaphragmatic hernia

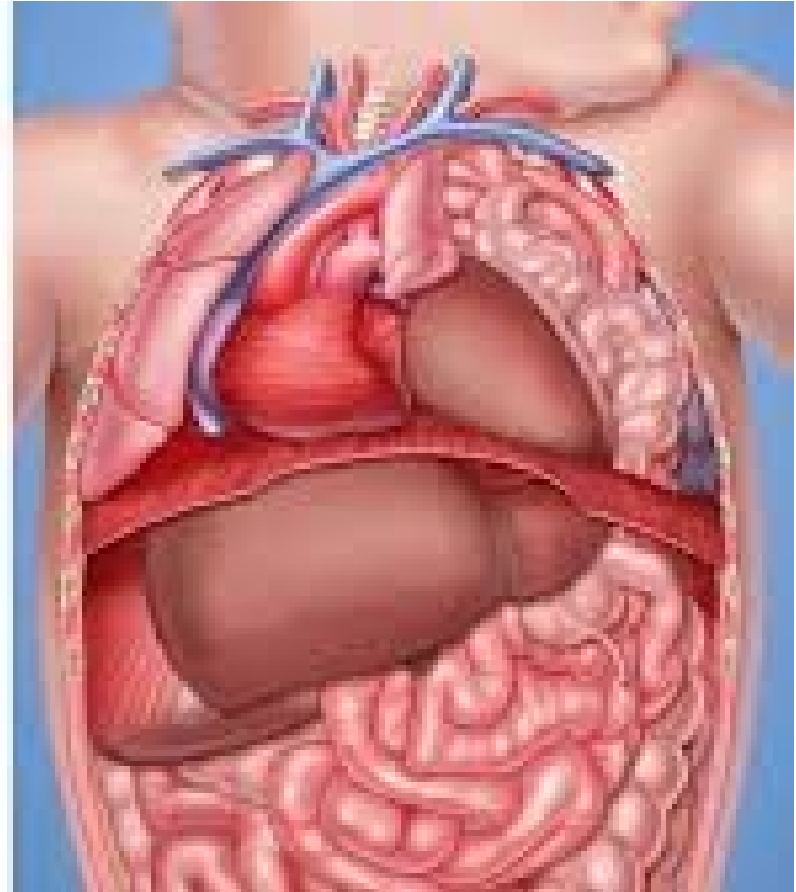
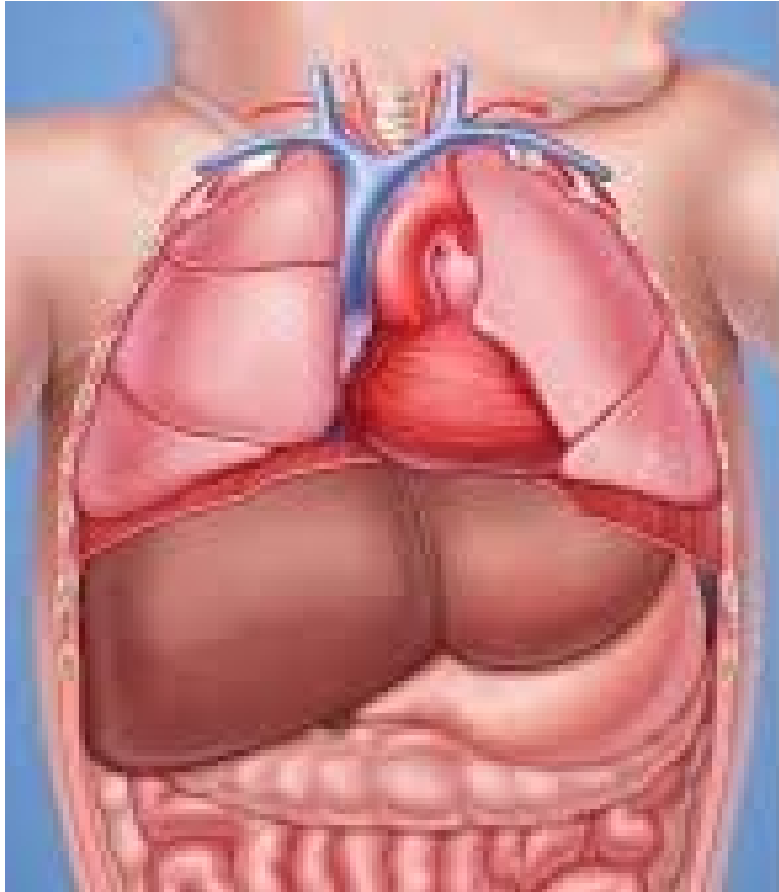
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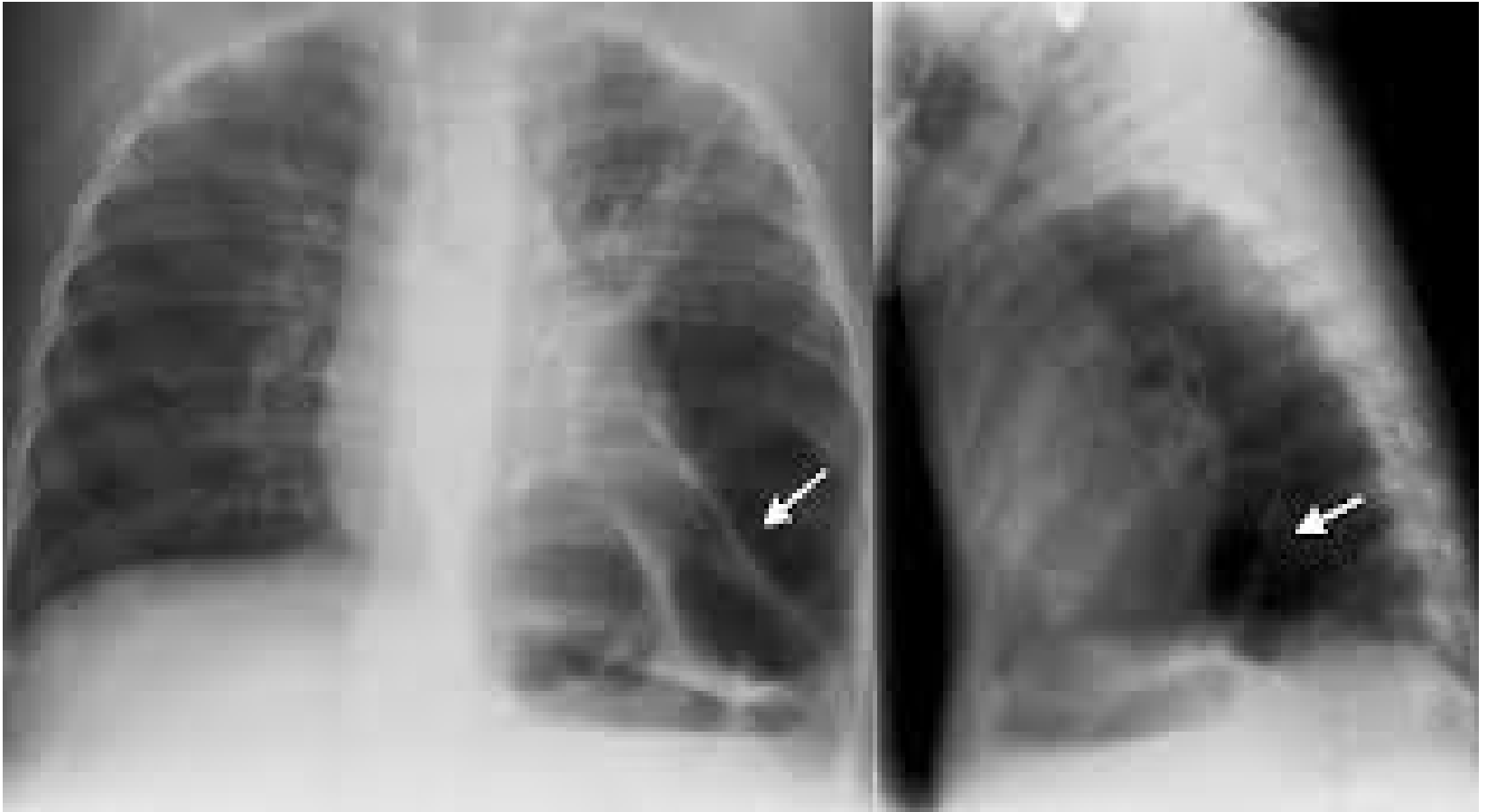
Diaphragmatic hernia

- Malformation affects diaphragm development
- The hole to which are organs transformed from the abdominal cavity to the chest cavity
- Lungs development endangered
- Sinistrous 85%, right-hand, bilateral
- True, false 80-90%
- Noticeable prenatally on US, MRI

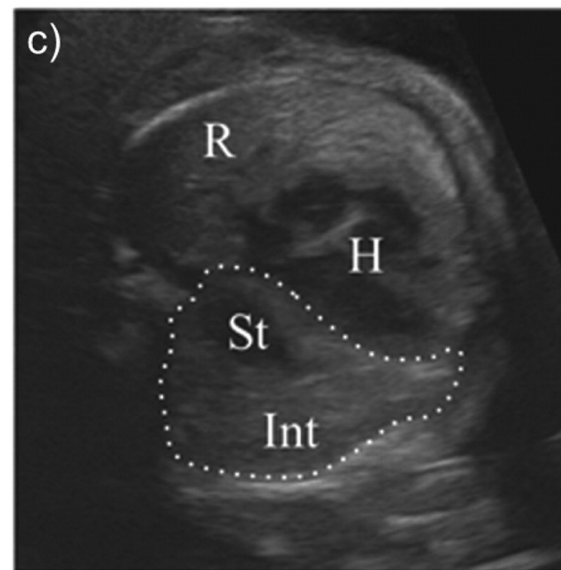
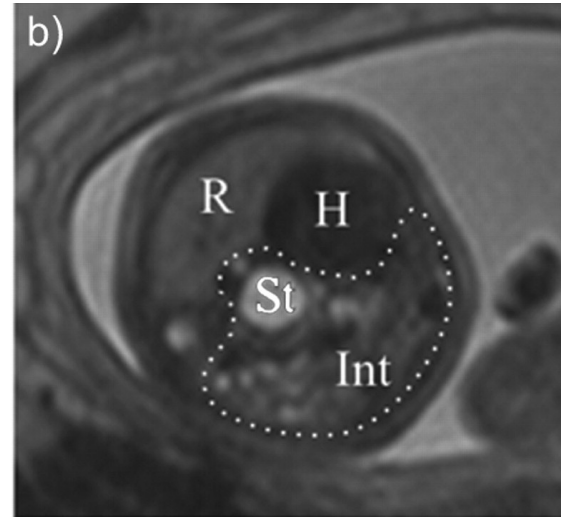
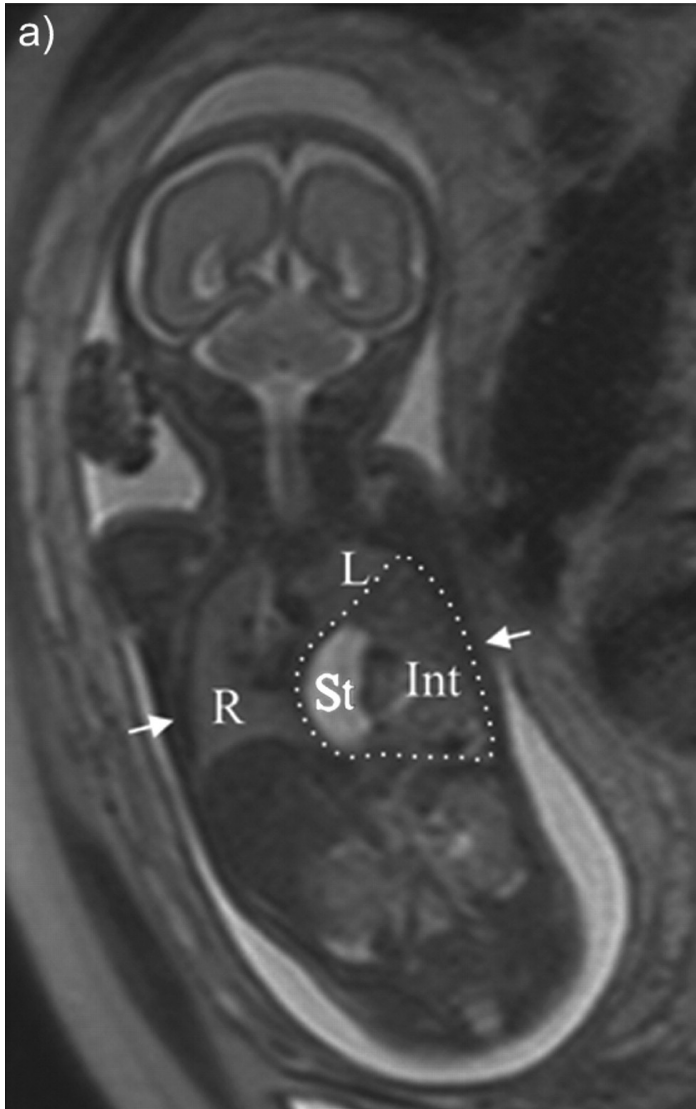
Diaphragmatic hernia



Diaphragmatic hernia



Diaphragmatic hernia



Diaphragmatic hernia

Clinical signs

Progressive aggravation of respiratory insufficiency

- Serious dyspnoea
- Heart sounds transferred to the other side
- Intestinal peristalsis in thoracic cavity

Diaphragmatic hernia

Therapy

Immediate treatment of respiratory insufficiency

- Immediate intubation
- NG tube
- Intravenous cannula
- Airway, Breathing and Circulation monitoring
- Deaden the newborn
- Transport to the child surgery workplace

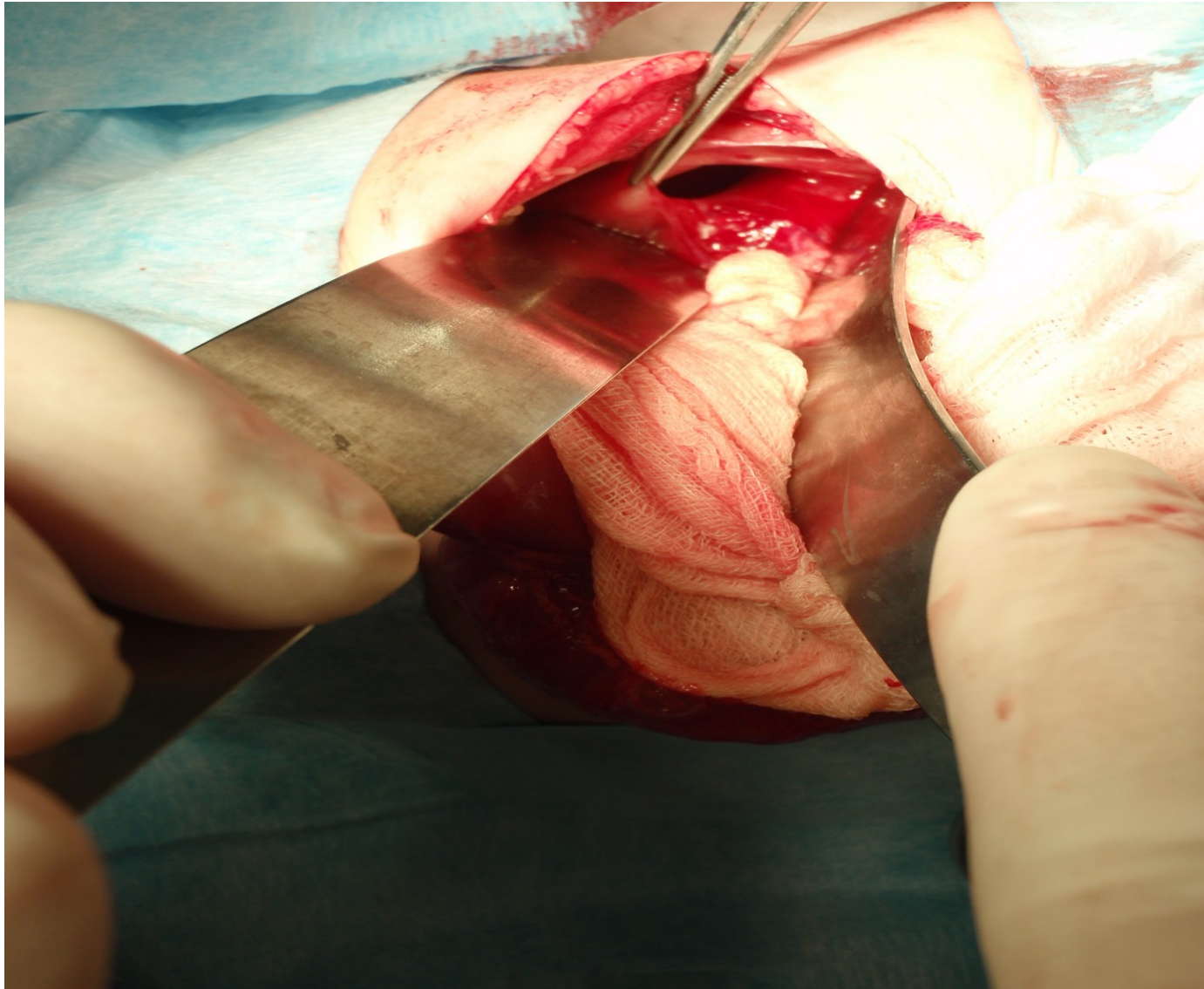
Diaphragmatic hernia

Therapy

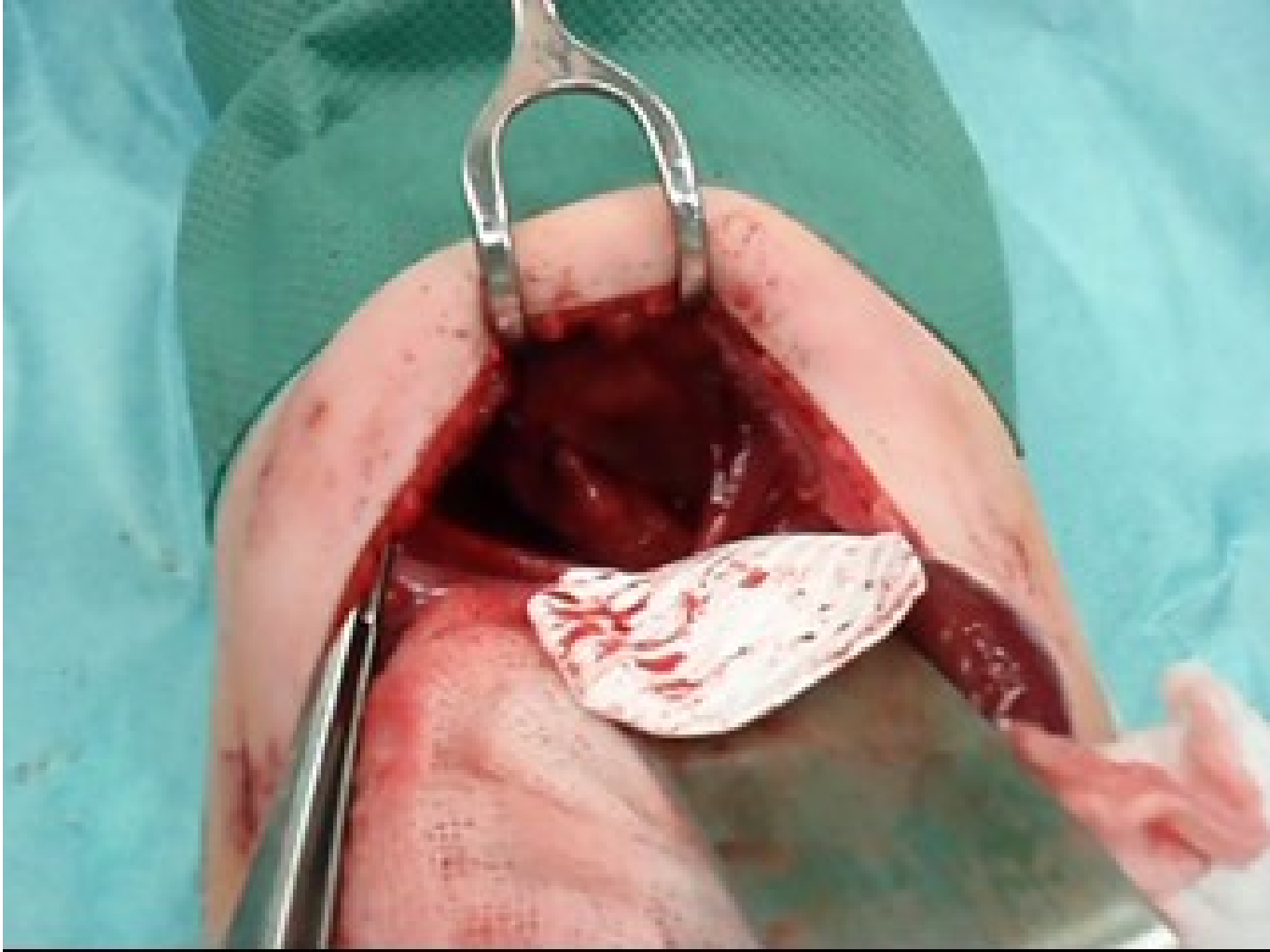
Creating septum between the thoracic and abdominal cavity

- **Small defects** – simple suture defect
- **Great defects** – synthetic material diaphragm compensation (Gore-Tex membrane)

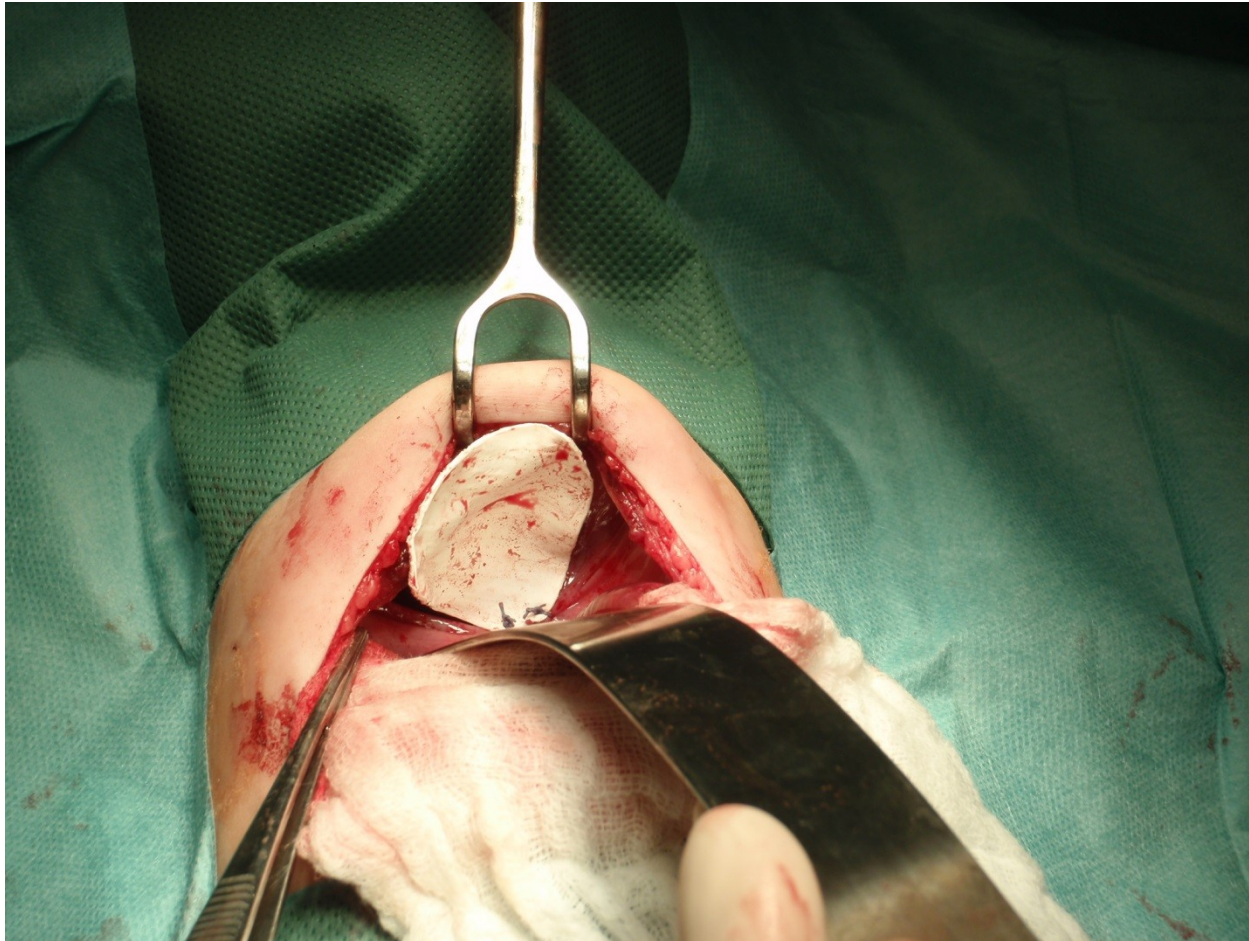
Diaphragmatic hernia



Diaphragmatic hernia



Diaphragmatic hernia



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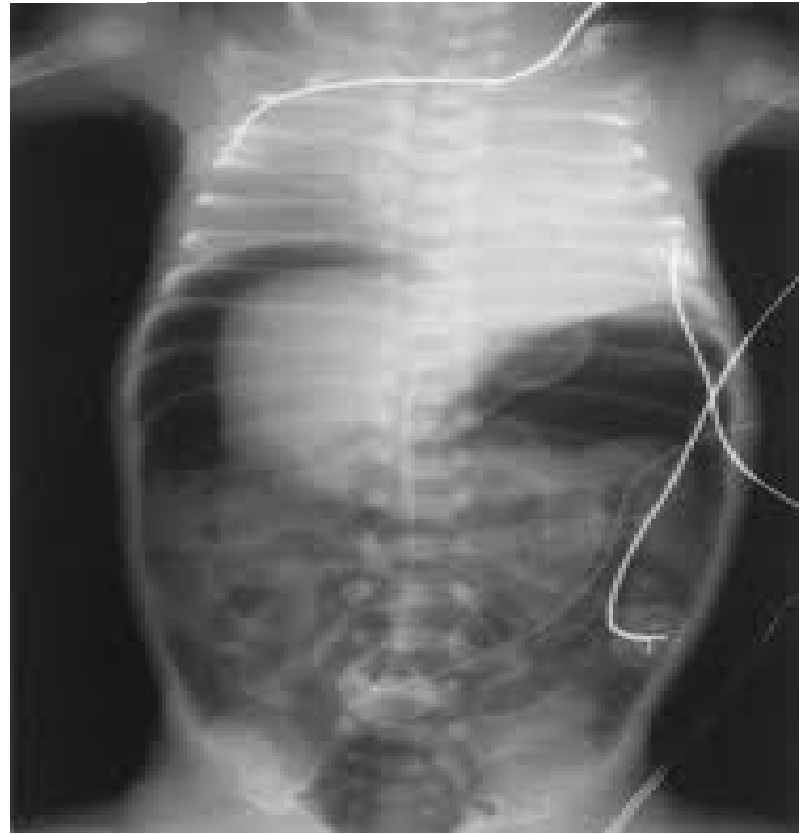




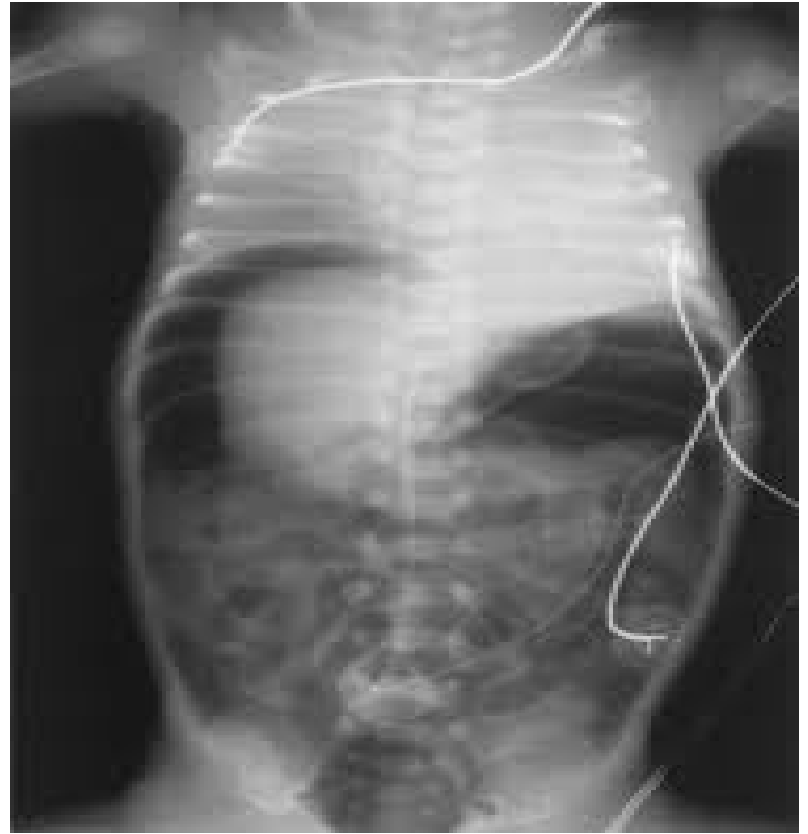
- Labour in 32.tgSC, ph 1060g/40cm
- AS 5,8,9
- Ventilated for 4 days due to RDS
- Detected hypotension, circulatory support by dopamine for 48 hours
- ATB therapy , clin. and lab. signs of sepsis regression after 72 hours
- Fed by tube + parent. nutrition
- 12th day of aggravation state, billowing abdomen, tachycardia, apneic intermission, impure blood in stool, hypotension



X-ray



Necrotizing enterocolitis



Necrotizing enterocolitis

- One of the most serious GIT newborns disease
- Affects mostly **immature newborns** with pH under 1500g (10-20% affects mature newborns with risk factors – Congenital Heart Defect..., exceptionally full term infant without CDD)
- Causes – multifactorial.
 - immature GIT **ischemic** reaction (perinat. asphyxia, pneumopathie, shock, ductus arteriosus patens, polycythemia, ...)
 - food** loading of unsuitable composition (cow milk proteins, hyperosmolar food)
 - immune** systém immature
 - **bacterial infection** – nosocomial flora colonization
- Terminal ileum and proximal colon frequently affected, however, the whole intestines necrosis might happen (up to 20% of patients)

Necrotizing enterocolitis

clinical count

Mild course  Peracute course

- Temporary food intolerance
- Abdomen distension
- Rectum occult bleeding
- Fully spread sepsis with apnea count
- Bradycardia
- Unstable temperature
- Unstable circulation
- Massive enterorrhagia
- Shock

Stage	Systemic symptoms	GIT symptoms	X-ray symptoms
1A – suspected NEC	Unstable temperature, bradycardia, mild apnea, lethargy	Gastric residuum, mild abdomen distension, vomiting, occult bleeding	Mild loop dilitation up to subileus
1B – suspected NEC	The same	Clearly red rectum blood	The same
2A – confirmed NEC, mild alteration	The same	The same + deaf peristalsis, +/- sensitive abdomen	ileus, pneumatosis intestinalis
2B - confirmed NEC, middle alteration	The same + mild metab. acidosis, mild trombocytopenia	The same + clear abdomen pain, +/- abdomen wall cellulitis or right lowe quadrant resistance	The same + gas v port. bed, +/- ascites
3A – advanced NEC, serious alteration, intestine infarction	The same+ hypotension bradycardia,deep apnea, DIC, neutropenia	The same + diffuse peritonitis signs, breakthrough pain and abdomen distension	The same + overt ascites
3B – advanced NEC, serious alteration, perforated intestine	The same	The same	The same + pneumoperitoneum

Necrotizing enterocolitis

Diagnosis

- Clinical finding
- Lab tests—neutropenia, thrombocytopenia, inflammation messenger growth, metabol. acidosis, hyperglycemia
- Imaging methods –X-ray (sub-ileus, pneumatosis intestini, gas in portal bed, pneumoperitoneum)
- US (ascites)

Necrotizing enterocolitis

Thrapy

- Mild form – **nursing intervention** – ATB, discontinued enteral nutrition, GIT decompression by NG tube, TIBC
- From 2B stage – **surgical treatment** – abdomen revision, affected intestine removal, primordial anastomosis (rather exceptionally), stoma
- Drainage abdominal cavity succesive by second look surgery at unstable patients in critical state or during pancolitis only.

Necrotizing enterocolitis abdomen distension



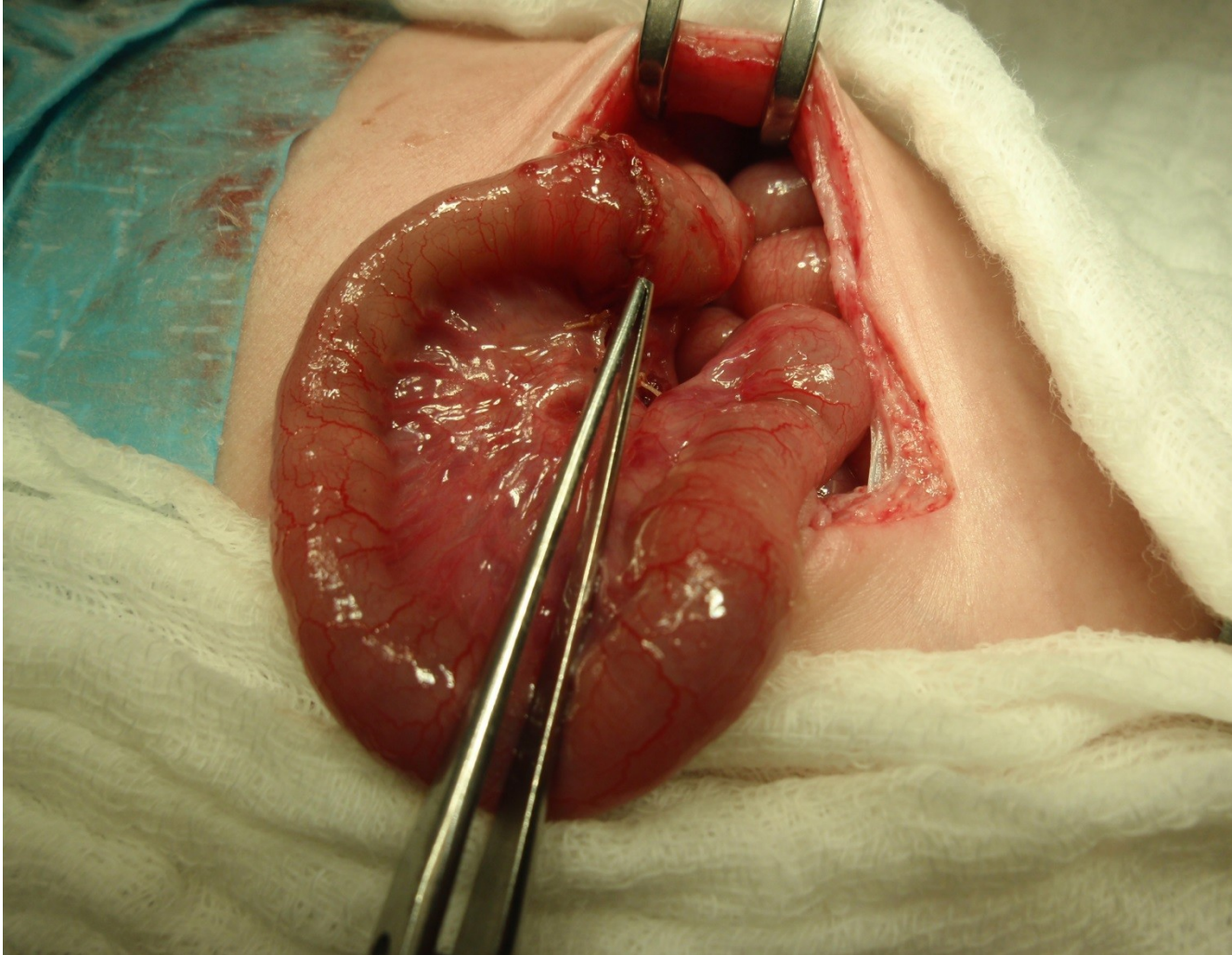
Necrotizing enterocolitis pneumatosis intestini



Necrotizing enterocolitis caecum necrosis + colon ascendens



Necrotizing enterocolitis primary anastomosis



Necrotizing enterocolitis pancolitis



Necrotizing enterocolitis stoma



We are having a baby

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- Spont. labour, 2500g/47cm
- Respiratory insufficiency progression after labour
- Billowing abdomen, meconium is not gone
- Anus created normally, RR can be inserted for 5 cm, no meconium impression
- Sono finding of small amount of floating liquid in abdominal cavity



Meconium ileus

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- Respiratory insufficiency progression after labour
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Meconium ileus

- Affects 10-15% of newborns with cystic fibrosis (mucoviscidosis)
- Rigid meconium similar like rubber arises because of exocrine gland affect
- Termin. ileum as the most frequent obstruction

Meconium ileus

newborns meconium passages casebook

Simple meconium ileus	Termin. ileum obstruction by meconium viscosity, CF+
Complicated meconium ileus	Intestine obstruction by meconium viscosity, CF+, intestine atresia at the same time, volvulus, stenosis, perforation, intestine necrosis, peritonitis
Meconium plugs syndrome	Rectosigma obstruction by meconium, another disease unproven
Immaturity ileus	Termin. ilea obstruction by meconium, children with less than 1500g
Intestine innervation dysfunction	M.Hirschsprung, totální aganglionosis, intestine hypoperistalsis
Other causes	Congenital hypothyroidism, mother's narcotic abusus

Meconium ileus

Clinical count

- No meconium leaving
- 1st-2nd day of vomiting and progressive abdomen distension
- Sometimes palpable indefinite resistance in the right hypogastrium
- Progressive ileus and sepsis

Meconium ileus

Diagnosis

- Abdomen X-ray in eyesight – ileu count, sometimes noticeable meconium shadows
- Irrigography – narrow colon count (microcolon)
- Sono – distended small bowel loops, termin. ileum distended by content without liquid
- Elevated chloride values in sweat
- Genetic examination CF

Meconium ileus

- „Dry“ ileus X-ray



- Irrigography - microcolon



Meconium ileus

Therapy

- Simple form – **conservative** – clysis (alternatively with mucolytic), start with feeding after clearance by GIT (special milks + pancreat. enzymes substitution)
- Complicated forms – **surgical th.** – in case of conservat. th. failure or at či GIT perforation – the most frequent temporar stoma.

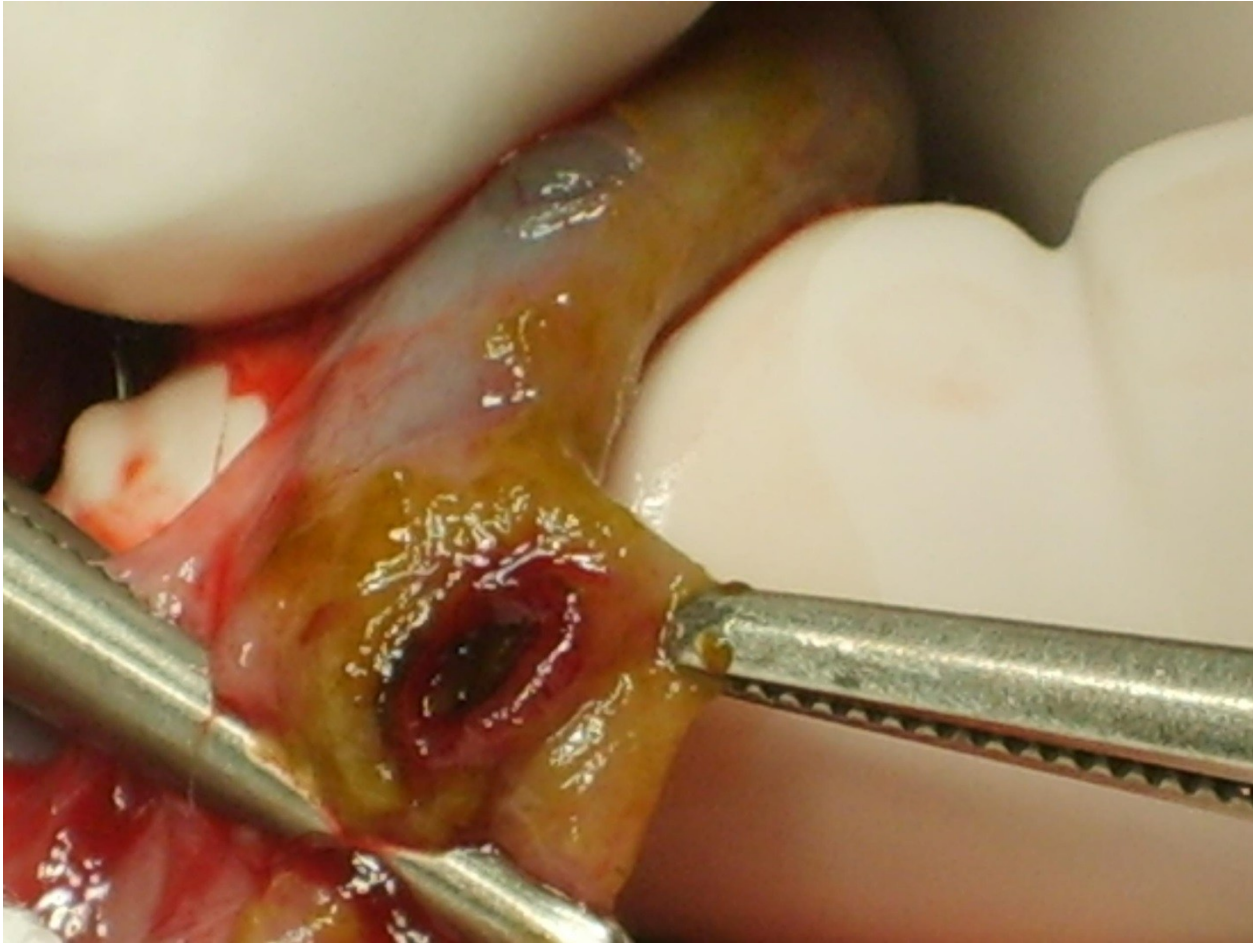
Meconium ileus



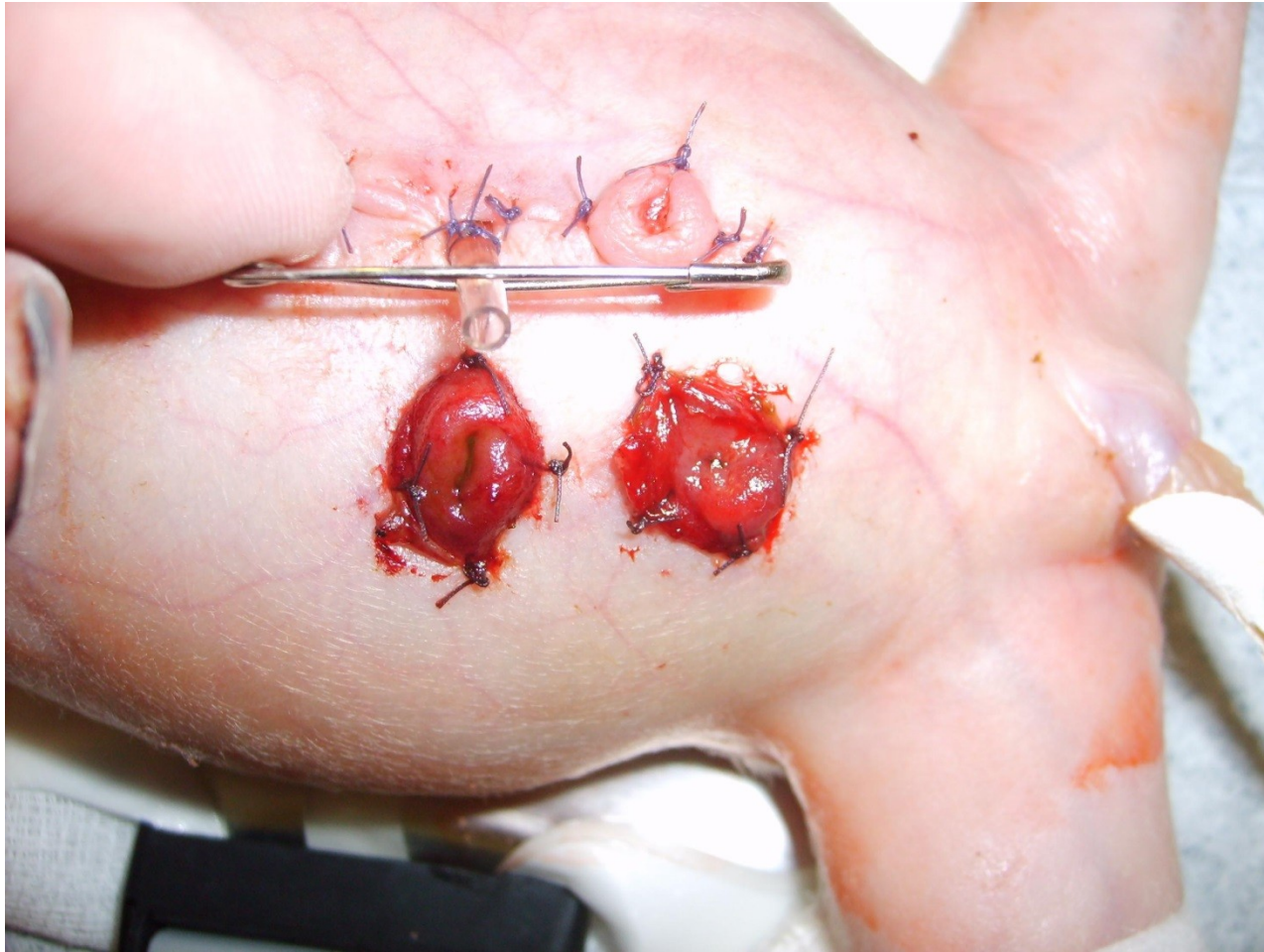
Meconium ileus



Meconium ileus



Meconium ileus



We are having a baby

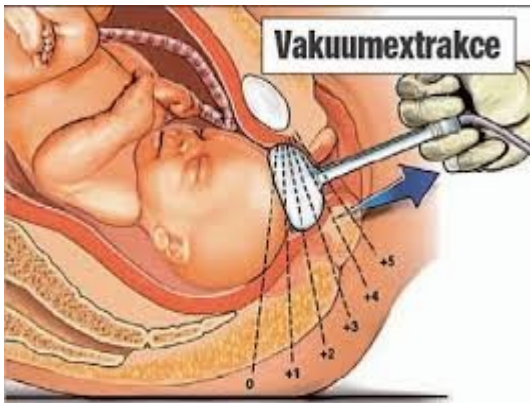
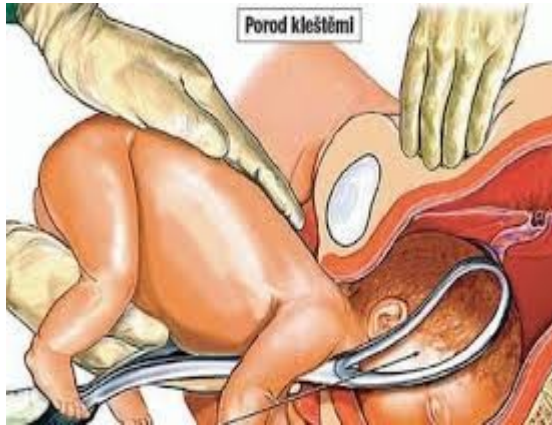
it could happen to you as well



Is it going to be smooth?



Sometimes not...



Newborn's labour injury

Risk factors

- Immature fetus
- Breech birth
- Instrumental delivery
- Great fetus delivery, cephalopelvic disproportion
- Rapid delivery
- Slacken delivery
- Abnormally positioned fetus
- CDD of fetus

Newborn's labour injury

Soft tissue injury (petechia, cephalhaematoma, subconjunctival and retinal haemorrhage, m.sternocleidomastoideus injury)

Skeleton injury (skull, long bones)

CNS and periph. nerves injury(epidural, subdural or subarachnoid bleeding, n. facialis palsy, brachial plexus palsy)

Intra-abdominal injury (liver and spleen tear, suprarenal bleeding)

Newborn's labour injury

Clinical symptoms

- Depends on the location of the injury
- From slight behavioural change to difficult life endangering state
- Painful manipulation
- Hematoma
- Limb disfigurement



- Painful manipulation with LA
- Asymmetry in collar bone area
- Palpable resistance in left collar bone area



Clavicle fracture

- Painful manipulation with LA
- Asymmetry in collar bone area
- Palpable resistance in left collar bone area



Clavicle fracture



Clavicle fracture



Thigh bone fracture - callus



Thigh bone fracture— one month after



Thigh bone fracture



Humerus fracture



Humerus fracture





- Prolonged vag. delivery with shoulder dystocia
- Fetus size and parturient passage disproportion
- Restricted mobility of LA
- Pronated position of LA

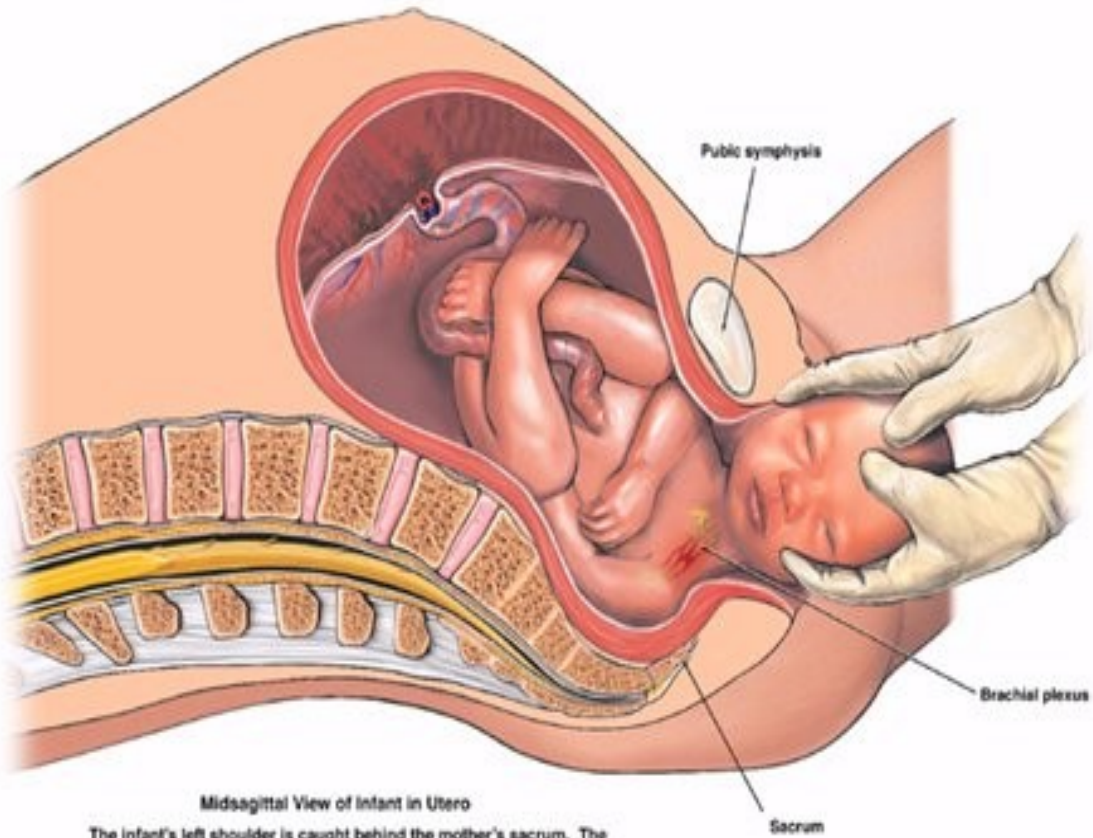


Brachial plexus paresis

- Prolonged vag. delivery with shoulder dystocia
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- Restricted mobility of LA
- Pronated position of LA



Brachial plexus paresis

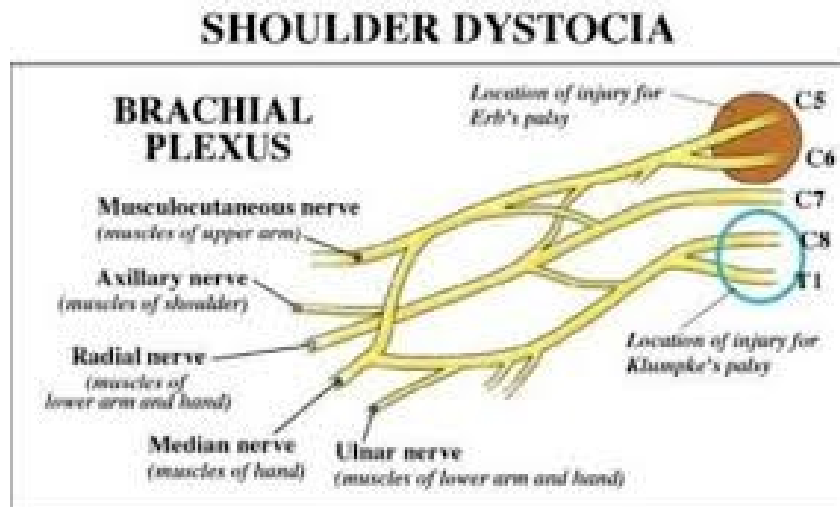


Brachial plexus paresis

At labour of great fetuses together with abnormal position

Superior type - 90% of paresis, C5-6 injury, UL poorly hangs in adduction and inner shoulder rotation, prehension reflex is possible to equip, n.phrenicus paresis and unilateral diaphragm paresis attend with C4 injury at the same time

Inferior type – 10% of paresis, C7-8 injury, distal part affected, pressured fingers, arm can be livid, Horner's syndrome at the same time with Th1



Brachial plexus paresis

Therapy

- Pat. in neurologist's and RHB doctor's care
- Start of RHB **in time** (if no other CI – e.g. collar bone fr., ABC functions unstable)
- Exercising according to neurophysiology – connection between movement and nervous system – Vojta's method
- Well conducted exercising for 4x day
- Baby crying – suppression of personal freedom manifestation, no pain
- If the elevation of UL fails within a period of next 3 months – mostly never successful
- 90% of the disabled in 1 year is without neurological deficiency

Thank you for your attention



The goal of our work is being sincerely interested in our entrust patients, have a friendly relationship with them, treat them with kindness. Give them their health back and save their lives.