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ASPHYXIA NEONATORUM. STAGES OF RESUSCITATION. REHABILITATION. PROGNOSIS

Methodical recommendations
for students 5th course of medical faculty

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The methodical recommendations give detailed recommendations for the provision of resuscitation assistance to newborns in asphyxia. These guidelines include traditional sections: the relevance, causes of asphyxia, the classification and clinical picture of asphyxia, the steps of resuscitative care for newborns with asphyxia, further rehabilitation and the prognosis of the disease. Determining the state of the newborn in the delivery room, determining the indications for the primary resuscitation of newborns, the ability to conduct initial steps of primary resuscitation of newborns, to design a plan for future monitoring of the newborn is an integral part of training the future doctor**.**

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1. LIST OF ABBREVIATIONS

AAP – American academy of pediatrics
AHA – American Heart Association
AN – asphyxia neonatorum
AVL – artificial ventilation of the lungs
CBC – complete blood count
CT – computer tomography
DIC – disseminated intravascular coagulation
EEG – electroencephalography
HIE – hypoxic ischaemic encephalopathy
HR – heart rate
MAS – meconium aspiration syndrome
MRI – magnetic resonance imaging
NEC – necrotizing enterocolitis
PPV – positive-pressure ventilation

2. ACTUALITY, TASKS AND METHODS

Asphyxia neonatorum(AN), also called birth or newborn asphyxia, is defined as an inability to start regular respiration within a minute of birth. Asphyxia neonatorum is a neonatal condition as it may cause to hypoxia (lowering of oxygen supply to the brain and tissues) and possible damage to the brain or death if not properly managed. A newborn normally start to breathe without assistance and usually cry after birth. One minute after delivery most infants are breathing well. If an infant fails to establish sustained respiration after birth, the infant is diagnosed with AN. Normally infants have good muscle tone at birth and actively move their arms and legs, while those with asphyxia are completely limps and not move at all. If not correctly treated, AN will cause hypoxia and possible brain damage or death.

Asphyxia neonatorum is one of the causes of newborn deaths in developing countries, in which 4 to 9 million cases of newborn asphyxia take place each year, accounting for about 20 % of the infant mortality rate(according to the World Health Organization).

Task of neonatal resuscitation programs – to improve implementation of these recommendations through the provision of necessary knowledge and practical skills of cardiopulmonary resuscitation of newborn.

3. CAUSES OF ASPHYXIA NEONATORUM

There are many causes of AN, the most common are following :

– insufficiency of hemoperfusion of the maternal part of the placenta: it is widespread when pregnancy is complicated with preeclampsia (hypertension, high levels of protein in urine and edema);

– hypoxemia and hypoxia of the mother;

– fetal anemia: this can happen when there is a severe immunization, mostly an immunization between the Rhesus factors. The fetal erythrocytes will be destroyed, so the result is an increasing anemia;

– twin transfusion. One twin is anemic and the other has a high hemoglobin;

– fetal hypoxia: because of a heavy smoking mother;

– shock, cardiovascular and /or respiratory failure;

– complication with the umbilical cord such as a real knots;

– placental separation (infarcts, calcifications, edema and inflammatory changes, placenta hemorrhage, premature);

– impaired placental function;

– sepsis can be associated with premature rupture of the membranes and fever;

– prolapses of the umbilical cord with cessation of the blood flow to the child;

– breech presentation;

– trauma: narrow pelvis and/or prolonged labor;

– drugs: sedatives, analgesics or general anesthetics (opiate), antihypertensive, antidepressants, antihistamines;

– congenital malformations of the brain, cardiovascular and respiratory system; obstructing the airway or preventing lung expansion (choanal atresia, congenital diaphragmatic hernia, meconium aspiration syndrome);

– prematurity and immaturity: surfactant deficiency and/or muscle weakness;

– postmaturity;

– unphysiological labour (induction);

– malpresentation including vasa praevia;

– primary muscle disease;

– unknown factors: about 20 % of cases with a low Apgar were not expected before delivery.

<u>High–risk for asphyxia neonatorum include:</u>

– maternal age of less than 16 years old or over 40 years old;

– low socioeconomic level;

– maternal illnesses, such as diabetes, hypertension, Rh-sensitization, anemia;

– mothers with previous abortions, stillbirths, early neonatal deaths, or preterm birth;

– lack of prenatal care;

– abnormal fetal presentation or position;

– alcohol abuse and smoking by the mother;

– severe fetal growth retardation;

– preterm labor;

– reduction of O_2-transport function of the mother's blood.

4. PATHOPHYSIOLOGY OF ASPHYXIA NEONATORUM

Asphyxia is a pathological state, characterized by functional and morphological changes into the body, cell, mitochondrial, molecular and genetic level, which cause to dysfunction of vital organs and systems of a child. The ability of the fetus or infant to survive episodes of asphyxia is related to the mechanism that regulates blood flow to the organs of the body. These

mechanisms are designed to maintain oxygen delivery to the vital organs during periods of hypoxia. The passage from the fetal to the extrauterine environment requires two major cardiorespiratory changes: removal of fluid from unexpanded alveoli to allow ventilation and redistribution of cardiac output to provide lung perfusion. Incorrect ventilation or inadequate perfusion leads to shunting, hypoxia, and ultimately reversion to fetal physiology.

The blood flow to the brain is increased during periods of hypoxia or hypercapnia. The result is a stable delivery of oxygen to the brain to meet metabolic demands and maintenance of a normal intracellular pH.

During periods of mild asphyxia**,** adaptive changes in blood flow permit correct oxygen delivery to the brain, heart, and adrenal gland. Blood flow to the skin, muscle, kidney, and gastrointestinal tract is sacrificed to maintain perfusion of the vital organs. The development of hypoxia in fetus leads to period of rapid respiration. If hypoxia continue, the respiratory movements stop, heart beats decries and fetus entrance to period of primary apnea.

During periods of severe or prolonged asphyxia, the sacrificed tissues and organs gradually become acidosis due to anaerobic metabolism and lactic acid production, causes to myocardial depression and a gradual decrease in blood pressure so that blood fails to perfuse the vital organs, with resultant permanent tissue damage in such organs, the deep rapid respiration-gasping takes place. The heart beats and AP are decreasing.

A brief period of rapid breathing followed by “primary apnea”. Is the initial reaction to asphyxia. The initial response to asphyxia during primary apnea, stimulation such as drying or slapping the feet will restart breathing. If the apnea is prolonged, the infant loses muscle tone and becomes cyanotic and then bradycardic. After a several gasping respirations, the neonate enters “secondary apnea”.

5. CLASSIFICATION OF ASPHYXIA NEONATORUM

In Ukraine, a clinical and statistical classification of diseases has been developed, in which highlighted the paragraph "Separate conditions arising in the perinatal period". Asphyxia refers to the item "Disorders of the respiratory and cardiovascular systems in the perinatal period" and is distributed to such forms:

R.21. Birth asphyxia.

R.21.0. Severe birth asphyxia.

R.21.1. Mild or moderate birth asphyxia.

R.21.9. Birth asphyxia, unspecified.

6. CLINICAL MANIFESTATION OF ASPHYXIA NEONATORUM

The ability to recognize the clinical signs and symptoms of asphyxia requires knowledge of the risk factors for the development of asphyxia, as well as the prenatal and postnatal symptoms of asphyxia.

During labor:

– loss of beat-to-beat variability;

– variable or late deceleration pattern;

– yellow, meconium-stained amniotic fluid.

After birth:

– infants are depressed and fail to breathe spontaneously.

Diagnosis can be objectively assessed using the Apgar score a recording of the physical health of a newborn infant, determined after examination of the adequacy of respiration, heart action, muscle tone, skin color, and reflexes.

Apgar Score of the newborn

SIGN	SCORE		
	0	1	2
Heart rate	Absent	<100 beats/min	>100 beats/min
Respiratory effort	Absent	Weak, irregular	Strong, regular
Muscle tone	Flaccid	Some flexion	Well flexed
Reflex irritability (response to catheter in nostril)	No response	Grimace	Cough or sneeze
Skin colour	Blue, pale	Body pink, extremities blue	Entire body pink

Each vital sign is given a score of 0 or 1, or 2. The best possible Apgar score is 10 and the worst 0. An infant with a score of 0 shows no sign of life.

Normally the Apgar score is from 7 to 10. Infants who have a score between 4 and 6 have moderate depression of their vital signs while newborns with a score of 0 to 3 have severely depressed vital signs and are at great risk of dying unless actively resuscitated. Premature newborns often have a lower Apgar score and usually require ventilation right after the birth. To record the infant's clinical condition after birth the Apgar score should be performed on all infants at 1 minute after complete delivery. The aim is to identify infants who need resuscitation. If the 1 minute Apgar score is below 7, then the Apgar score should be repeated at 5 minutes to document the success or failure of the resuscitation efforts. If the 5 minute Apgar score is still low, it should be repeated every 5 minutes till a normal Apgar score of 7 or more is achieved.

Criteria of severe asphyxia:

– severe metabolic or mixt acidosis pH ≤7/00 in arterial blood of umbilical vessels;

– assessment by Apgar is 0–3 during more than 5 minutes;

– neurological symptoms such as general hypotonia, lethargy, coma, seizures;

– damage of vital organs (lungs, heart and other) in fetus or newborn.

Complications associated with Asphyxia:

– Hypotension;

– seizures;

– persistent pulmonary hypertension;

– hypoxic cardiomyopathy;

– hypoxic encephalopathy;

– necrotizing enterocolitis;

– acute tubular necrosis;

– adrenal hemorrhage and necrosis;

– polycythemia;

– hypoglycemia;

– disseminated intravascular coagulation.

Perinatal asphyxia may cause hypoxic ischaemic encephalopathy (HIE). This is graded according to the classification of Sarnat and Sarnat:

Grade 1: mild encephalopathy with infant hyperalert, irritable, and over-sensitive to stimulation. There is evidence of sympathetic over-stimulation with tachycardia, dilated pupils and jitteriness. The EEG is normal.

Grade 2: moderate encephalopathy with the infant displaying lethargy, hypotonia and proximal weakness. There is parasympathetic overstimulation with low resting heart rate, small pupils, and copious secretions. The EEG is bnormal and 70 % of infants will have seizures.

Grade 3: severe encephalopathy with a stuporous, flaccid infant, and absent reflexes. The infant may have seizures and has an abnormal EEG with decreased background activity and/or voltage suppression.

One third or more of infants with HIE will have 2 or more organ systems involved, which may include lung, heart, liver, brain, kidneys.

7. DIAGNOSIS OF ASPHYXIA NEONATOROM

Principles of the assessment include history of maternal and intrapartum risk factors for problems that may affect the infant including pre-existing medical conditions in the mother, problems of pregnancy, antenatal abnormalities in the fetus, the presence of meconium stained liquor,

CT abnormalities, scalp pH, maternal indicators of infection, presentation and method of delivery. The following prognostic factors may be used as guides in management and counseling:

– Apgar score by the 1 and 5 minute;

– CBC with differential;

– serum glucose level;

– arterial blood gas analysis: acidosis, hypoxia and hypercarbia at birth – profound metabolic or mixed acidosis (pH less 7,0) in the blood of he umbilical artery;

– serum electrolyte, Ca^{+2}, phosphorus and magnesium levels;

– renal evaluation: blood urea nitrogen, creatinine, FE-Na, urine analysis (β2 microglobulin) & renal ultrasound;

– cardiac evaluation: serum troponin and creatine kinase;

– hepatic evaluation: serum transaminases, albumin, bilirubin and ammonia levels, prothrombin time and activated partial thromboplastin time;

– brain imaging: ultrasound, CT and MRI. They are NOT used routinely;

– computerised tomography: hypodensities may take 10–14 days to develop. Abnormalities include haemorrhages and hypodensities;

– ultrasound abnormality: may detect haemorrhage. Infants with small or poorly visualised ventricles, and hypoechogenicities are at increased risk of abnormal neurodevelopment;

– EEG: to evaluate seizure activity and to define abnormal background activity;

– brain stem function responses (prognostic significance).

8. STAGES OF RESUSCITATION, TREATMENT

The treatment of AN is resuscitation of the newborn. All medical delivery rooms have adequate resuscitation equipment in case when an infant cannot breathe well at delivery. There are some necessary routine procedures for newborn.

The principles include evaluation of the airway, establishing effective respiration and adequate circulation; the guidelines also highlight the assessment and response to the neonatal heart rate and the management of infants with meconium-stained fluid.

Immediately after the birth, an infant in need of resuscitation ought to be placed under a radiant heater and dried (to avoid hypothermia), positioned head down and slightly extended, the airway cleared by suctioning and gentle tactile stimulation provided. Immediately after birth, infants who

are breathing and crying may undergo delayed cord clamping. However, until more evidence is available, infants who are not breathing or crying should have the cord clamped (unless part of a delayed cord clamping research protocol), so that resuscitation measures can start promptly.

About 60 seconds ("the Golden Minute") are aconsigned for completing the initial steps, reevaluating, and beginning ventilation if required. The decision to progress beyond the initial steps is determined by simultaneous assessment of 2 vital characteristics: respirations (apnea, gasping, or labored or unlabored breathing) and heart rate (less than 100/min). Once positive-pressure ventilation (PPV) or supplementary oxygen administration is started, assessment should consist of simultaneous evaluation of 3 vital characteristics: heart rate, respirations, and oxygen saturation, as determined by pulseoximetry. The most sensitive indicator of a successful response to each step is an evaluation of the heart rate.

The initial steps of newborn resuscitation are:

– to maintain normal temperature of the infant;

– to put infant in a "sniffing" position;

– clear secretions if needed with a bulb syringe or suction catheter;

– to dry the infant (unless preterm and covered in plastic wrap);

– to stimulate the infant to breathe.

– The temperature of nonasphyxiated newborns is recommended to be maintained between 36.5°C and 37.5°C after birth through admission and stabilization.

<u>The steps in neonatal resuscitation follow the ABCs:</u>

A. Anticipate and establish a patent airway by suctioning and, if needed, performing endotracheal intubation;

B. Initiate breathing by using tactile stimulation or positive-pressure ventilation with a bag and mask or through an endotracheal tube;

C. Maintain the circulation with chest compression and medications, if needed.

If no respirations are noted or if the heart rate is below 100/min, positive pressure ventilation is given through a tightly fitted face mask and bag for 15–30 sec , endotracheal intubation should be performed in infants with severe respiratory depression who do not respond to positive pressure ventilation via bag and mask. Ventilation is continued and chest compression should be initiated over the lower third of the sternum at a rate of 120/min if the heart rate does not improve after 30 sec with bag and mask (or endo-tracheal) ventilation and remains below 100/min. The ratio of compres-sions to ventilation is 3:1. If the HR remains less 60 despite effective compressions and ventilation, administration of epinephrine should be considered. Persistent bradycardia in neonates is usually due to hypoxia

resulting from respiratory arrest and often responds rapidly to effective ventilation alone.

A loosely fitted mask, poor positioning of the endotracheal tube, intraesophageal intubation, airway obstruction, insufficient pressure, pleural effusions, pneumothorax, excessive air in the stomach, asystole, hypovolemia, diaphragmatic hernia, or prolonged intrauterine asphyxia may cause poor response to ventilation.

Equpment for neonatal resustitation:

– radiant warmer;

– stethoscope;

– oxygen source with warmer and humidifier;

– suction source, suction catheter, and meconium "aspirators";

– nasogastric tubes;

– apparatus for bag-and-mask ventilation;

– ventilation masks Laryngoscope (handles, blades, and batteries);

– endotracheal tubes (2.5, 3.0, 3.5, and 4.0 mm);

– intravenous fluids (10 % dextrose, normal saline, and Ringer's lactate);

– drugs: Epinephrine (1:10,000 solution) Naloxone hydrochloride (0.4 or 1.0 mg/mL) Sodium bicarbonate (0.5 mEq/mL);

– volume expanders (5 % albumin, O-negative whole blood [cros-smatched against the mother's blood]);

– clock;

– syringes, hypodermic needles, and tubes for collection of blood samples;

– equipment for umbilical vessel catheterization;

– micro-blood gas analysis availability;

– warm blankets.

Additional equipment setup: all of the above plus the following:

– pressure manometer for use during ventilation;

– oxygen blender;

– heart rate and blood gas monitoring equipment;

– umbilical vessel catheter setup (ready to insert);

– transcutaneous oxygen tension or saturation monitor;

– blood gas laboratory immediately available;

– apgar timer;

– camera;

– plastic bags for "micro-preemies".

Preparation for a high-risk delivery is often the key to a successful outcome. Cooperation between the obstetric and pediatric staff is important. An estimation of weight and gestational age is useful in calculation of drug dosages and in choosing of the appropriate endotracheal tube and umbilical

catheter size. While waiting for the infant to arrive, it is useful to think through potential problems, steps that may be undertaken to correct them, and which member of the team will handle each step. Provided there is both time and opportunity, resuscitative measures should be discussed with the parents.

The Apgar score is assigned at 1, 5, and, occasionally, 10–20 min after delivery. It gives a fairly objective retrospective idea of how much resuscitation a term infant required at birth and the infant's response to resuscitative efforts. It is rather useless during resuscitation. During those long, tense moments, simultaneous assessment of HR, skin color, and respiratory activity provides the quickest and most accurate evaluation of the need for continuing resuscitation. For preterm infants, Apgar scores may be particularly misleading (even in assessment of the response to resuscitation) because of developmental differences in tone and response to stimulation.

A. Heart rate. The HR is ideally checked by a cardiotachometer with electrodes taped to the chest. Most often, evaluation is done by listening to the apical beat or feeling the pulse by lightly grasping the base of the umbilical cord. The evaluator should tap out each beat so that all team members can hear it. If no heart rate can be heard or felt, ventilatory efforts should be halted for a few seconds.

B. Skin color. Assessment of skin color may be difficult when there is severe bruising, especially in preterm infants. Bluish coloring indicates central cyanosis, and oxygen supplementation or assisted ventilation is needed. Pinkish membranes indicate normal oxygen levels, and resuscitation may not be needed.

C. Respiratory activity. Respiratory activity is assessed by observing chest movement or listening for breath sounds. If there is no respiratory effort or the effort is poor, the infant needs respiratory assistance by either manual stimulation or bag-and-mask ventilation.

Technique of resuscitation.

A. Ventilation:

1. General measures:

a) Suctioning. First, nasal and oropharyngeal secretions should be partially removed with a brief period of suctioning using either a bulb syringe or a suction catheter. More prolonged suctioning delays resuscitation and may lead to a vagal response in the infant .

b) Mechanical ventilation. Most infants can be adequately ventilated with a bag and mask under condition that the mask is of the correct size with a close seal around the mouth and nose and there is appropriate flow of oxygen to the bag. The stomach should be emptied during and after prolonged bag-and-mask ventilation.

c) Endotracheal intubation. Endotracheal intubation should be performed when indicated. Absolute indications for aggressive ventilatory support with endotracheal intubation are difficult to list here because institutional guidelines and clinical situations vary widely. Time limit: should be completed within 20 seconds.

Steps of intubation:

– stabilize the newborn's head in the "sniffing" position;

– deliver free flow oxygen during the procedure;

– cut the tube to a shorter length (13–15 cm);

– slide the laryngoscope over the right side of the tongue, pushing the tongue to the left side of the mouth, and advancing the blade until the tip lies just beyond the base of the tongue, lift the blade slightly and raise the entire blade (not just the tip).

– look for landmarks; vocal cords should appear as vertical stripes on each side of the glottis or as an inverted letter "V";

– suction, if necessary, for visualization;

– hold the tube with the right hand, insert into the right side of the mouth with the curve of the tube lying in the horizontal plane, and then pass it between the vocal cords ≈ 2 cm below the glos (the p of the tube is inserted until the vocal cord guide is at the level of the cords). If the vocal cords are closed, wait for them to open;

– visualize the glottis before inserting the tube, watch the tube enter between the vocal cords;

– hold the tube firmly against the infant's palate while removing the laryngoscope, and hold the tube in place while removing the stylet (if used);

– estimate the proper depth of insertion by:

weight (kg) + 6 cm = insertion depth at lip in cm.

Weight (g)	Gestational age (weeks)	Endotracheal tube size, inside diameter (mm)	Depth of insertion (cm from upper lip)
<1000	<28	2.5	6–7
1000–2000	28–34	3.0	7–8
2000–3000	34–38	3.5	8–9
>3000	9 > 38	3.5–4.0	9–10

Based on guidelines from Bloom RS, Cropley C: Textbook of Neonatal Resuscitation. American Heart Association/American Academy of Pediatrics, 1995;

– confirm the position of the tube by:

a) Observing symmetrical chest wall movement;

b) Listening for bilateral equal breath sounds;

c) Confirming absence of gastric inflation;

d) Watching a fog of moisture in the tube during exhalation;

e) Noting improvement in HR, color & activity;

f) Chest X-ray confirmation, if the tube is to remain in place past initial resuscitation.

2. Specific measures

Term infant with meconium staining

Meconium staining of the amniotic fluid may be an indication of fetal stress; therefore, personnel skilled at endotracheal intubation and resuscitation should be present at the delivery.

Infants born through thick meconium may aspirate this inflammatory material in utero (gasping), during delivery, or immediately after birth. The sickest of these infants have usually aspirated in utero and generally also have reactive pulmonary vasoconstriction. The AAP and the AHA recommended endotracheal suctioning when meconium is present in the amniotic fluid and the infant is not vigorous (eg, without good muscle tone, good respirations, and heart rate >100 beats/min). Clinical judgment is always important in deciding whether or not aggressive endotracheal suctioning is necessary.

– Hypopharyngeal suctioning should be started as soon as the head is delivered, before the infant has started to cry. Deep suctioning should be avoided because it may result in acute laryngospasm.

– Endotracheal suctioning. Subsequently, endotracheal intubation is performed, and suction is applied directly to the endotracheal tube. Suctioning with a negative pressure of 80–100 mm Hg can be done directly from the wall unit via a connector (meconium aspirator) to the endotracheal tube. Suction is applied as the endotracheal tube is slowly withdrawn (Kattwinkel, 2000).

– If meconium has been suctioned "below the cords," suctioning should be repeated after reintubation. Prolonged or repeated suctioning is not recommended because it will exacerbate the preexisting asphyxial insult.

– The procedures just described may be continued for up to 2 min after delivery, but then other resuscitative measures (particularly ventilation and oxygenation) must be started.

– Supplemental oxygen. Infants born through thick meconium may have experienced prolonged partial asphyxia in utero as well as pulmonary vascular constriction, leading to pulmonary hypertension after delivery. Generous amounts of supplemental oxygen to these infants should be provided.

– If meconium-stained fluid is reported at <34 weeks' gestation, one of the following situations should be suspected.

• The fetus is a growth-retarded term infant.

• The fluid can actually be purulent (consider Listeria or Pseudomonas).

• The fluid can actually be bile stained (consider proximal intestinal obstruction).

Term infant with perinatal asphyxia

A term infant with a HR of <100 beats/min and no spontaneous respiratory activity requires immediate lung expansion and supplemental oxygen provided by bag-and-mask ventilation. The lungs should be slowly expanded (5–10 breaths) with high peak inflating pressures (30–40 cm H_2O). If there is no effect in stimulating spontaneous respiratory effort or an improved heart rate, the ventilation rate should be increased to 40–60 breaths/min and peak inflating pressures should be adjusted as necessary to expand the lungs. In case when bag-and-mask ventilation is ineffective or prolonged positive-pressure ventilation is necessary, endotracheal intubation is indicated.

If effective spontaneous respiratory effort results, the infant may be extubated and closely examined while breathing supplemental oxygen.

A term infant with a HR of >100 beats/min but with poor skin color and weak respiratory activity requires stimulation (rubbing the back is often useful), supplemental oxygen blown across the face, and occasionally bag-and-mask ventilation to expand the lungs. Majority of these infants will react with improved skin color and good spontaneous respiratory effort by 5 min of age.

Resuscitation with room air is also effective and may decrease the risk of hyperoxia, with decreased cerebral blood flow, and generation of oxygen free radicals. Currently 100 % O_2 is recommended. Room air may become the preferred initial gas for neonatal resuscitation in the future; if the neonate does not achieve normal oxygen saturations within 90 sec, increasing concentrations of oxygen should be blended in (up to 100 % oxygen) till normal oxygen saturations are achieved. If pulmonary hypertension is suspected (meconium aspiration, diaphragmatic hernia) one may consider 100 % oxygen as the initial gas for resuscitation.

Although the 1st breath normally requires pressures as low as 15–20 cm H_2O, pressures as high as 30–40 cm H_2O may be necessary. Following breaths are given at a rate of 40–60/min with a pressure of 15–20 cm H_2O. Noncompliant stiff lungs secondary to respiratory distress syndrome, congenital pneumonia, pulmonary hypoplasia, or meconium aspiration may require higher pressures. Successful ventilation is determined by normal chest rise, symmetric breath sounds, improved pink color, HR >100/min, spontaneous respirations, presence of end-tidal CO_2, and

improved tone. Various tools to detect exhaled CO_2 and to confirm accurate placement of an endotracheal tube are commercially available. A laryngeal mask airway may be an effective device to establish an airway, especially if bag mask ventilation is ineffective or intubation is unsuccessful.

Preterm infant

Preterm infants weighing <1200 g usually require immediate lung expansion in the delivery room. Ventilatory support measures should be taken as described for the asphyxiated term infant, with several important differences:

– If intubation is required, a smaller (2.5 – or 3mm internal diameter) endotracheal tube is chosen ;

– Although high peak inflating pressures may initially be needed to expand the lungs, as soon as the lungs "open up" the pressure should be quickly decreased to as low as 10–15 cm H_2O by the end of the resuscitation if the clinical course allows ;

– If available, one of several forms of liquid surfactant may be administered intratracheally as prophylaxis for hyaline membrane disease. However, surfactant is not a resuscitative medication and should be administered only to a stable neonate with a correctly placed endotracheal tube.

B. Cardiac resuscitation.

During delivery room resuscitation, efforts should be aimed to assisting ventilation and providing supplemental oxygen. A sluggish heart rate will usually respond to these efforts.

1. If the HR continues to be <60 beats/min by 30 s of age in spite of ventilatory assistance, chest compression should be started.

• Methods:

– <u>two-thumb encircling hands method</u>: stand at the infant's foot and grip the chest in both hands; the 2 thumbs press at the junction of the middle and lower thirds of the sternum (just below an imaginary line joining the nipples); with the fingers wrapped around and supporting the back;

– <u>two-finger method</u>: stand at the infant's side and compress the lower third of the sternum with the index and third fingers of one hand; with the other hand supporting the back.

Thumbs or fingers remain in contact with the chest at all times, and chest compressions and ventilation are well coordinated. The thumbs are put on the midsternum just below a line connecting the nipples, while the palms of the hands encircle the torso and suppor. The sternum is compressed ½–¾ in (1.3–1.9 cm) at a regular rate of 90 compressions/min, while ventilating the infant at 30 breaths/min. The HR should be checked regulary and chest compression discontinued when the HR is >60 beats/min.

2. An infant with no heart rate (a true Apgar of 0) who does not respond to ventilation and oxygenation may be considered stillborn. Prolonged resuscitative efforts are a matter for ethical consideration (Jain, 1991; Kattwinkel, 2000).

C. Drugs used in resuscitation.

Medications are rarely required but should be administered when the HR is <60/min after 30 sec of combined ventilation and chest compressions or during asystole. The umbilical vein can generally be readily cannulated and used for immediate administration of medications during neonatal resuscitation. If intravenous access is not available and/or for naloxone hydrochloride the endotracheal tube may be used for the administration of epinephrine.

a) Epinephrine may be required during resuscitation when adequate ventilation, oxygenation, and chest compression have failed and the heart rate is still <60 beats/min. This drug causes peripheral vasoconstriction, enhances cardiac contractility, and increases HR. The dose is 0.1–0.3 mL/kg of 1:10,000 solution given intravenously or by endotracheal tube. It can be repeated every 3–5 min. If an endotracheal tube is used, the solution should be diluted 1:1 with normal saline.

b) Volume expanders. Hypovolemia should be suspected in any infant requiring resuscitation, particularly when there is evidence of acute blood loss with extreme pallor despite adequate oxygenation, poor peripheral pulse volume despite a normal heart rate, long capillary refill times, or poor response to resuscitative efforts. Appropriate volume expanders include O-negative whole blood (cross-matched against the mother's blood), 10 mL/kg; Ringer's lactate, 10 mL/kg; and normal saline, 10 mL/kg. All are given intravenously over a 5 to 10-min period.

c) Naloxone hydrochloride. Naloxone (Narcan) is a narcotic antagonist and should be administered to an infant with respiratory depression unresponsive to ventilatory assistance whose mother has received narcotics within 4 h before delivery. One major exception to this recommendation is the newborn infant of a drug-addicted mother. These infants should never receive Narcan because acute withdrawal symptoms may develop. The intravenous or intratracheal dosage for Narcan is 0.1 mg/kg. Two concentrations of naloxone are available: 0.4 mg/mL and 1.0 mg/mL. The dose can be repeated every 5 min if necessary. It should be emphasized that the half-life of Narcan is shorter than that of narcotics.

d) Dextrose. The blood glucose concentration should be checked within 30 min after the birth in asphyxiated term infants, infants of diabetic mothers, and preterm infants, especially those whose mothers received tocolysis with ritodrine. Large boluses of dextrose should be avoided, even

when the blood sugar is <25 mg/dL. To avoid wide swings in blood glucose, add a small bolus of 10 % dextrose in water (1–2 mL/kg intravenously) and then start an intravenous infusion of 10 % dextrose at a rate of 4–6 mg/kg/min (80–100 mL/kg/day).

e) Sodium bicarbonate is usually useless during the acute phase of neonatal resuscitation. Without adequate ventilation and oxygenation, it will not improve the blood pH. After prolonged resuscitation, however, sodium bicarbonate may be used in correcting documented metabolic acidosis. Give 1–2 mEq/kg intravenously (usually over a period of 30 min).

f) Atropine and calcium. Although previously used during resuscitation of the asphyxiated newborn, atropine and calcium are no longer recommended by the AAP. Current evidence does not support their effectiveness during delivery room resuscitation.

D. Other supportive measures:

1. Temperature regulation. Although some degree of cooling in a newborn infant is desirable because it provides a normal stimulus to respiratory effort, excessive cooling increases oxygen consumption and exacerbates acidosis. This is a problem especially for preterm infants, who have thin skin, decreased stores of body fat, and increased body surface area. Heat loss may be prevented by the following measures.

a) Dry the infant thoroughly immediately after delivery.

b) Maintain a warm delivery room.

c) Place the infant under a prewarmed radiant warmer.

Cover preterm infants with plastic wrap or a plastic bag up to the neck.

2. Preparation of the parents for resuscitation. Initial resuscitation usually occurs in the delivery room in presence of one or both parents present. It is useful to prepare the parents before hand, if possible. Describe what will be done, who will be present, who will explain what is going on, where the resuscitation will happen, where the father should stand, why crying may not be heard, and where the infant will be taken after stabilization.

Severe asphyxia may also depress myocardial function and cause cardiogenic shock despite the recovery of heart and respiratory rates. Dopamine or dobutamine administered as a continuous infusion (5–20 μg/kg/min) and fluids should be started after the initial resuscitation effort to improve cardiac output in an infant with poor peripheral perfusion, weak pulses, hypotension, tachycardia, and poor urine output. Epinephrine (0.1–1.0 μg/kg/ min) may be indicated for infants in severe shock that does not respond to dopamine or dobutamine. Less severe degrees of poor cardiopulmonary transition in the delivery room can usually be managed by brief periods of bag and mask ventilation. Chest compression

and medications are not required for most neonates who have mild to moderate birth depression. Regardless of the severity of asphyxia or the response to resuscitation, asphyxiated infants should be monitored carefully for signs of multiorgan hypoxic-ischemic tissue injury. Routine intubation of the lungs of vigorous infants born through meconium-stained fluid is not recommended to apply. Depressed infants (those with hypotonia, bradycardia, fetal acidosis, or apnea) should undergo endotracheal intubation, and suction should be applied directly to the endotracheal tube to remove meconium from the airway. The risk of MAS in these circumstances is higher than the risk associated with laryngoscopy and endotracheal intubation (bradycardia, laryngospasm, hypoxia, posterior pharyngeal laceration with pseudodiverticulum formation).

9. REHABILITATION OF CHILDREN WITH ASPHYXIA NEONATORUM

Clinical management is directed at appropriate and rapid resuscitation, and preventing hypoxia, hypercarbia and acidosis. Early arterial blood gas and blood sugar level should be performed and acidosis and hypoglycaemia treated. The infant's cardiorespiratory status should be monitored and signs of multiorgan system dysfunction sought and treated where appropriate.

1. Correction of hypoglycaemia.

2. Correction of acidosis: obtain early ABC and correct respiratory acidosis (hypercarbia and acidosis) with appropriate ventilatory support. Correct persistent severe metabolic acidosis with bicarbonate over 30–60 minutes. Using bicarbonate to an infant not adequately ventilated (either spontaneously or mechanically) is forbidden as it causes hypercarbia and acidosis.

3. Treatment of seizures: Treat seizures initially with phenobarbitone. Failure to control with phenobarbitone – add phenytoin. Persistent seizures – add clonazepam (infant will require ventilation – see seizures).

4. Temperature: maintain core temperature 36–37 ^{0}C, skin temperature 36–36.3 ^{0}C. Avoid hyperthermia.

5. Respiratory status – monitor for hypoxia, acidosis and hypercarbia. Respiratory distress may have multiple aetiology including acidosis, meconium aspiration, sepsis or persistent pulmonary hypertension. Aim for normocarbia (Pco^2 35–45). Avoid hypoxia and hypocarbia.

6. Cardiac status – blood pressure is a poor predictor of low cardiac output. Cardiac ECHO may identify hypovolaemia or poor myocardial contractility and low flow states – see hypotension. Use inotropes (dobutamine or dopamine) early if hypotension present or low flow states documented on ECHO.

7. Fluid therapy and renal impairment – Infants with anuria / oliguria should receive 40–60 mls / kg /day until adequate urine output documented. Regular assessment of fluid balance, electrolytes and creatinine should be performed – (watch for fluid overload and hyperkalaemia).

8. DIC – if evidence of bleeding or petecchiae: perform platelet level and a coagulation profile. Give vitamin K and replace clotting factors.

9. Gastrointestinal – feeding: the decision to feed will depend on a clinical assessment of the severity of asphyxia and associated system dysfunction (respiratory distress, encephalopathy, hypotension and renal impairment). Feed intolerance is common and NEC may complicate perinatal asphyxia. Breast milk is preferred.

Discontinuation of resuscitation may be appropriate if no signs of life (no HR and spontaneous breaths) in an infant 15 min of complete and adequate resuscitation effort, with no evidence for other causes of newborn compromise.

10. PROGNOSIS OF ASPHYXIA NEONATORUM

Prognosis is difficult because of the inability to establish the precise extent and duration of cerebral insult and injury. At the time of delivery low delayed Apgar scores between 0 and 3 at 10, 15 and 20 minutes' of age are associated with significantly increased mortality and morbidity, e. g. cerebral palsy. The single most useful prognostic factor is the severity of the neonatal neurological syndrome. The prognosis for asphyxia neonatorum depends on how long the new born is unable to breathe. For example, clinical researches show that the outcome of infants with low five-minute Apgar scores is significantly better than those with the same scores at 10 minutes. With prolonged asphyxia, brain, heart, kidney, and lung damage can result and also death, if the asphyxiation lasts longer than 10 minutes.

There are generally no long term abnormalities reported following mild encephalopathy, whereas essentially all infants with severe encephalopathy have either died or developed multiple neurological sequel.

The overall prevalence of neurological sequel following hypoxicischemic encephalopathy observed at 3 1/2 years of age is approximately 17 %.

Prolonged duration of encephalopathy with an abnormal neurological examination after 7 days of age is associated with an increased incidence of long-term sequel. Outcome is related to the severity and duration of the asphyxia insult and to the adequacy of compensatory mechanisms, resuscitation procedures, and specific treatment of multiorgan system involvement.

Neurological outcome is the best related to the degree of hypoxic encephalopathy and EEC activity in the neonatal period, and to findings on physical examination of the infant at 9–12 months of age.

11. CONTROL TESTS

1. In a child apnea is persistent, heart rate is less than 100 contractions per minute, in 15–30 sec auxiliary ventilation of lungs is provided. It is neccesary:

A. To continue ventilation of lungs by a mask and sack
B. To begin indirect massage of heart
C. To inject medicine
D. To conduct intubation of trachea
E. To conduct haptic stimulation
Standard response: A.

2. Injection of drugs during the procedure of primary reanimation of newborn is necessary, if:

A. Heart rate after the indirect massage of heart is less than 80 per minute
B. Heart rate after AVL with 100 % oxygen under positive pressure is less than 80 contractions per minute
C. Heart rate after AVL with 100 % oxygen under positive pressure with the indirect massage of heart is 60 per minute or less
D. Heart rate after AVL with 100 % oxygen under positive pressure in combination with the indirect massage of heart is 80 contractions per minute
E. All answers are correct
Standard response: C.

3. The newborn in asphyxia is carried by ventilation with a bag and mask. Excursion of the chest is satisfactory. When assessing the condition noted: heart rate 90 per minute, total cyanosis of the skin, muscle hypotension. The signs of the effectiveness of ventilation for this child do you expect?

A. Increase in heart rate
B. Restoration of independent breathing
C. Improvement of the color of the skin
E. Improvement of muscle tone responses
Standard response: A.

4. At birth, a newborn infant is noted to have the following findings: heart rate – 70/min, respiratory effort – poor and irregular, limp, no reflex irritability, blue all over the body. The Apgar score of the baby at this point is?

A. 3
B. 5
C. 7
D. 1
Standard response: A.

5. A newborn by cesarean section showed signs of a narcotic depression of breathing, cyanotic skin, apnea. Indicate the preparation of primary care.

A. Adrenaline hydrochloride
B. Sodium hydrogen carbonate
C. Naloxone
D. Albumin
E. Calcium gluconate
Standard response: C.

6. Assessment of newborn by Apgar should be perfomed at the:
A. 1 and 5 minutes of life
B. 1 and 10 minutes of life
C. 5 and 10 minutes of life
D. 1 and 20 minutes of life
Standard response: A.

7. The third step of reanimation of newborn must include:
A. Airways' clearing
B. Tactile stimulation
C. Tracheal intubation
D. External chest compression
Standard response: D.

8. Which criterion corresponds about moderate asphyxia:
A. Evaluation by Apgar score 4–6 points:
B. pH 7.1 of urine;
C. Increased movements of fetus;
D. Hepatomegaly
Standard response: A.

9. After birth a child was pale and had arrhythmical breathing. Oxygen therapy didnt have any effect. Pulse was weak and rapid. It was difficult to measure arterial pressure accurately. There were no edemata. What is the most likely reason for these symptoms?

A. Intracranial haematoma
B. Congenital pneumonia
C. Intrauterine sepsis
D. Asphyxia neonatorum
Standard response: D.

10. The concentration of epinephrine hydrochloride, which is used for neonatal resuscitation:

A. 1:10
B. 1:100
C. 1:1000
D. 1:10000
Standard response: D.

12. REFERENCES

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