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Resuscitation of the newborn

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RESUSCITATION OF THE NEWBORN

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In this paper I propose to present a review of causes exclusive of intracranial accidents and treatment of fetal and neonatal asphyxia in the otherwise normal infant.

Bograd (1) has defined asphyxia neonatorum as that condition in which spontaneous respiration is not established with sufficient promptness or force to maintain life.

During a five year period from 1941-1946 the infant mortality rate at Chicago Lying-In-Hospital was 2.5% for infants weighing over 1000 grams. This mortality was almost equally divided between still-births and deaths during the first 10 days of life. The mortality among newborn weighing over 2500 grams was only 0.5%. The principal cause of death both before and after birth was anoxia resulting from interference with placental circulation; chiefly due to premature separation of the placenta. After birth the principal cause of death is an anatomic and physiologic immaturity which is incompatible with an independent existence.

Potter and Diekman (2) state that 63% of the still-births in the series they investigated were due to causes operating prior to the onset of labor, the chief demonstrable cause being anoxia with premature detachment of the placenta and cord knots and entanglements being the

principal causes. Prevention here is still in the realm of discussion since the underlying factors causing premature placental separation are unknown. The only hope for successful delivery is the early recognition of the condition and relief by caesarian section. Here failure to recognize such signs of fetal distress as change in heart rate and excessive fetal movements can lead only to disaster.

The prognostic signs of impending asphyxia during delivery (Flagg)(3) are:

(1) Bradycardia between labor pains with irregularity of heart sounds.

(2) Loud umbilical souffle.

(3) Excessive movement of the baby.

(4) Meconium in the amniotic fluid due to increased peristalsis.

During delivery the infant is most apt to suffer from what Flagg calls stagnant anoxia. This is most often due to extrinsic factors superimposed upon an otherwise normal delivery.

Fetal respiratory movements have been demonstrated in the human as well as in other animals. Therefore, depression or failure of respiration at birth must be regarded as the suppression of previous activity rather

than the failure of some new mechanism to begin functioning.

Normal regulation of fetal respiration depends on oxygen and carbon dioxide levels in the fetal blood. Oxygen want in the fetus depresses or abolishes fetal respiratory movements (Snyder)(4).

The obstetrician is faced with the problem of how to give his patient the relief she demands without endangering her child.

Henderson, Foster and Eno(5) analized a series of nine-hundred and seventy-five unselected deliveries from the standpoint of factors which might produce asphyxia in the newborn, concluded that:

"(1) Ninety and six tenths percent of babies born when the mother is under the influence of analgesia show no evidence of clinical asphyxia.

(2) Causes other than the use of analgesia or anesthesia are found in two-thirds of the cases that are clinical asphyxiated and may be present in others.

(3) General anesthesia definitely decreases the respiratory response of the newborn.

(4) When properly supervised and in the hands of those familiar with their use, analgesics per se do not increase the incidence of asphyxia."

Lund(6) reviewed One-thousand nine-hundred and eighty-two consecutive deliveries from the aspect of asphyxia neonatorum in relation to inhalation analgesic and anesthetic agents with the following results:

"(1) Nitrous oxide, ethylene and cyclopropane, when used as analgesic agents, did not materially influence the incidence of asphyxia neonatorum.

(2) Nitrous oxide, properly administered was given for long periods of time without significant effects on fetal asphyxia.

(3) Cyclopropane and other agents when used in concentrations sufficient for anesthesia by operative delivery were accompanied by an increase in the incidence of fetal asphyxia.

(4) The incidence of asphyxia neonatorum varied directly as the duration of administration of cyclopropane. (See Note)

(5) There seemed to be no relationship between asphyxia neonatorum and the type of anesthetic technic.

(6) Prematurity, complications of pregnancy and labor, method of delivery and misuse of analgesic agents were apparently of greater significance in asphyxia neonatorum than the various inhalation agents when properly administered."

Others, as Cole, Kimbal and Daniels(7), take a different stand. They state:

"Sedatives in any amount definitely increase the incidence of asphyxia in the baby in direct proportion to the amounts given."

"General anesthesia in any amount definitely increases the incidence of asphyxia in the baby in direct proportion to the duration of the anesthesia."

Sullivan(8) is in general agreement with this viewpoint favoring the use of only enough anesthetic to dull the patient's nerve sense, (keep her in the room).

The individual factors which may accompany any delivery must be taken into account in considering asphyxia, that is fetal size, position and presentation, abnormalities of maternal pelvis or soft parts, the expulsion force of the uterus, the use of uterine stimulating drugs. The recognition and management of these factors depend upon the obstetricians skill and judgement and will not be considered further in this paper.

Factors in asphyxia which become important with the delivery of the infant can be divided into two basic groups; those due to respiratory obstruction and those due to circulatory collapse.

The most common respiratory obstructive factor is

mucous and debris in the nasal and pharyngeal passages through which the infant is unable to make its initial respiratory effort. This condition may be complicated by fluid and mucous in the bronchial tree if the infant has suffered any degree of distress in the course of labor. This is especially important in breech and caesarian deliveries since natural compression forces in the course of delivery do not act to force fluid from the trachea. The position in which the newborn is held is also a factor in asphyxiation since the trachea of the newborn is very flexible and easily collapsed by flexions of the head on the chest or pressure on the chin.

The circulatory factors include hemorrhage from the umbilical vessels and circulatory collapse due to post delivery exposure. Post delivery exposure can hardly be considered a sole factor in production of neonatal asphyxia, however its prevention is a simple matter of routine which may give the asphyxiated infant that little support it needs in the struggle for life.

Flagg(3) has divided asphyxia into three stages according to the reflex state of the infant; stage of depression, stage of spasticity, stage of flaccidity. In the stage of depression, respirations are slow and

may be irregular but reflexes are present. The infant's jaws will close on a finger introduced into the mouth. In the stage of depression the color varies from duskiness to recurring cyanosis, never pallor since circulation is active in this stage.

With continued asphyxia the infant passes into the stage of spasticity with irregular gasping respirations, muscle tone is good, mucous membranes cyanotic and skin blotchy to pale. Reflexes are present varying from active to sluggish.

This is followed by the stage of flaccidity in which reflexes and muscle tone are gradually lost. The infant offers no resistance to exposure of pharynx. In this stage there may be cyanosis or pallor, depending on the activity of the infant circulation, cyanosis indicating an active circulation. Apex beat may or may not be demonstrable.

In general prevention of fetal asphyxia is dependent upon a recognition of the conditions which produce it and of the signs which indicate its approach. Obstetrical judgement dictates as to the advisability of rupturing membranes and administration of oxytoxics. Premature separation of placenta is unpredictable. In all conditions leading to antipartum asphyxia the early

recognition of fetal distress is important. Treatment is directed at relief of the condition by administration of oxygen to the mother, and early delivery.

Pending delivery of an asphyxiated infant it is important that equipment for resuscitation be immediately available and in working order, that is, present at every delivery, not packed away in a closet. The delivery room attendant who fails to have a full tank of oxygen avilable is as guilty as a hit-and-run driver.

Resuscitation begins with the aspiration of nasal pharyngeal passages immediately upon delivery of the head. This is best carried out by use of soft rubber syringe. Follow completion of delivery, clearing of airway is aided by holding the infant by the feet and gently stroking the throat toward the mouth.

A warm sterile cotton blanket should be available to receive the infant. In depression due to the forces of labor this is all the resuscitative effort necessary. However administration of oxygen and carbon dioxide can do no harm and is indicated until cause of depression is determined.

In the stage of spasticity the above measures are carried out first. Reflexes are still present. Bograd[1]

advocates stroking the hard palate, thus producing a delayed reflex involving the abdominal muscles. Could this be a pharyngeal gag reflex? Oxygen and carbon dioxide should be administered to an infant in this stage of asphyxia. Care should be taken to hold the infant's chin well forward in order that an open airway be maintained. Inhalation should be continued until the child is pink, respirations are regular and the child cries lustily.

In the stage of flaccidity the initial steps of clearing the upper air passages and keeping the child warm should be carried out. Inhalation oxygen and carbon dioxide are of no value until respiratory efforts are made, since they don't reach the lung without the inspiratory movement. This is the only stage of asphyxia in which tracheal intubation should be attempted since complete relaxation of pharynx and larynx is necessary in order that intubation be carried out without injury to the vocal cords and a possible traumatic reaction which would be worse than the initial asphyxia. Technic of intubation is dependent upon the equipment available and the experience of the operator. Flagg(3) recommends intubation by direct vision using an infant laryngescope, while

others use the blind method of intubation following a finger introduced into the pharynx.

There is still some difference of opinion on whether oxygen should be used alone or in conjunction with five percent carbon dioxide in resuscitation.

Coryllos(9) points out that carbon dioxide diffuses out of blood faster than oxygen is absorbed and in an organism compensated for a high carbon dioxide level breathing of pure oxygen may produce true alkalosis with a resultant depression of the respiratory center and apnea.

On the other hand carbon dioxide is a respiratory stimulant and in concentrations of five percant carbon dioxide stimulates the deep respiratory movements desired to expand the lung and thus increase the alveolar bed available for oxygen absorption without greatly reducing the amount of available oxygen.

Following resuscitation the child should be treated like a premature, that is, handled as little as possible, placed in an incubator under close observation.

Use of a mechanical respiratory is recommended by Coryllos(9), and Flagg(3) for infants who continue to show respiratory difficulty following initial

resuscitation. It is felt that these machines are less traumatic than manual manipulations but it must be remembered that they cannot function against an obstructed airway. They do not take the place of careful observation. Machines recommended by the two authors include the Drinker and Shaw, the E & J, the Kreiselman and the Flagg equipment.

How much pressure should be used with insufflation? FTagg(3) suggests an attempt at stimulation of the Hering-Brewer reflex by use of an initial maximum pressure of 25 mm. of mercury. Coryllos states that 14 cm. of water, approximately 10 mm. of mercury, were necessary to inflate the atelectatic lung of a dog. Most resuscitation apparatus can be adjusted to deliver up to 16 mm. mercury pressure.

Schwab, Eastman and Etsten(10) investigated a series of thirty-three newborn from the standpoint of roentgenogram changes before and after initiation of respiration. They found that the rapidity of full airation of the chest was dependent on the activity of the infant, varying from five minutes to two weeks.

The use of drugs as respiratory stimulants is advocated by some authors but has never been generally accepted. Drugs alone have not been advocated and it

is difficult to determine whether they every materially alter the effects of the resuscitative methods given above. Litchfield(12) uses 1/20 grain of alpha-lobeline hydrochloride injected into the umbilical vein and repeated in ten to fifteen minutes as necessary. Snyder and Kha Tilim(13) experimenting with alpha-lobeline, coramine, caffeine, metrazol and cyanide in newborn rabbits concluded that range between effective dose and convulsive dose was so narrow that they could not support the use of any of these drugs in the newborn infant.

Sufficient oxygen can be administered through intubation to maintain life without active respiratory movements. However, persistant atelectasis accompanied by pulmonary edema makes the infant very prone to develope pneumonia.(MacGregor)(11).

Use of mouth to mouth breathing is a time honored method of resuscitation which should not be forgotten in any emergency. Its dangers of course lie in over dilatation of the infant lung since it is difficult to gauge the force being applied. (I have as yet found no figures on the incidence of this) A gauze filter should be used to protect both obstetritian and infant from an interchange of infectious agents.

Manual manipulations such as compression of the lower ribs does more harm than good in the asphyxiated newborn. Before the initial respiratory effoft has taken place the thoracic cage is completely filled with tissue. A modification of the Eve tilt method of resuscitation seems more logical since it is dependent on the weight of the abdominal viscera pulling upon the diaphragm to produce a change in pressure within the thoracic cage. However, with the increased vascular permeability the frequency of intercranial hemorrhage accompanying asphyxia is too great to be further encouraged by such excessive handling.

NOTE

Snyder and Rosenfeld (14) state that experiments with rabbits at term revealed that full surgical anesthesia could be attained with cyclopropane without interruption of fetal respiratory rate. This is not true of other inhalation anesthetics of IV anesthetics. Clifford (15), on the other hand, makes the statement that cyclopropane may produce fetal asphyxia due to its effect of marked capillary dilatation, blood circulating though the capillary bed of placenta so fast that it is not properly oxygenated.

There is obvious need for more work here since if one accepts Snyder and Rosenfeld's state that oxygen want depresses fetal respiratory movements there either must be some change in fetal respiration under cycloporpane or the fetal asphyxia which Clifford attributes to that agent, must be produced by some other mechanism.

SUMMARY

(1) Prematurity, though seldom a sole factor, is the most important single factor in the etiology of neonatal asphyxia.

(2) Anoxia due to premature separation of the placenta is the commonest cause of death both before and after birth.

(3) Use of sedative and analgesic agents should be kept at a minimum.

(4) General anesthesia increases the incidence of fetal asphyxia.

(5) Asphyxia can be divided into three stages according to the reflex state of the infant: Stage of depression, Stage of Spasticity, Stage of flaccidity.

(6) Initial treatment in any stage consists of:

(a) Securing an open airway.

(b) Maintainence of body warmth.

(7) Tracheal intubation is indicated only when all reflex irritability of pharynx and larynx is absent.

(8) Administration of carbon dioxide five percent and oxygen is advocated for relief of anoxia.

(9) The use of drugs as respiratory stimulants is not recommended.

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