Resuscitation of the Newborn

George A. Gregory, M.D.*

The birth process is associated with profound changes in both circulation and respiration. Most infants make these changes without difficulty. However, about 10 percent fail to make the transition smoothly and require assistance. In the subsequent pages we consider the causes and effects of cardiopulmonary insufficiencies in the newborn and discuss currently practiced techniques for resuscitation.

Intrauterine and Natal Physiology

Respiration

The fetal lung begins as a simple budding of the foregut at 24 days’ gestation. By five months, the airways are lined with cuboidal epithelium and pulmonary capillaries arise from the mesenchyme. By 26 to 28 weeks, the capillaries are closely approximated to the developing airways, and extraterine life is possible. In the ensuing four weeks the cuboidal epithelium flattens and thins, a process that is hastened by administering steroids to the mother. Significant amounts of surface-active material are present intracellularly by 22 weeks, but do not reach the alveolar surface until 26–28 weeks’ gestation. Surfactant, too, can be released from cells by maternal steroids.

The fetal lung contains an ultrafiltrate of plasma (30 mg/kg body weight) which is not amniotic fluid. However, if the depth of fetal breathing is stimulated by stress, amniotic fluid may be carried into the lung, as evidenced by meconium and squamous cells in the lungs of asphyxiated and stillborn infants. During vaginal delivery the vagina and pelvic floor “squeeze” the fetal chest and force approximately two thirds of the fluid from the lung (Fig. 1). The remainder is removed after birth by lymphatics and capillaries. Small premature infants and those born by cesarean section receive no “vaginal squeeze,” possibly accounting for their increased difficulty in establishing respiration.

Most infants establish air breathing within 30 seconds of birth and rhythmic breathing within 90 seconds. The outward recoil of the chest wall following vaginal delivery fills the lungs with air, and brain-stem respiratory centers initiate rhythmic respiration. Many factors sustain ventilation, including pain, cold, touch, noise, cord clamping, acidosis, hypoxia, and hypercarbia. Severe acidosis, hypoxia, CNS injury, and maternal drugs (narcotics, local anesthetics, barbiturates, magnesium and alcohol) depress ventilation.

Circulation

The fetal circulation of man is unknown, but inferred to be similar to that of animals. It differs from that of adult man in that it is in parallel rather than in series (Fig. 2). The right ventricle accounts for two thirds of the combined ventricular output and the left ventricle for one third. Well-oxygenated blood from the inferior vena cava returns primarily to the left atrium and left ventricle. Poorly oxygenated blood from the superior vena cava returns primarily to the right

Abbreviations

CNS = central nervous system
DA = ductus arteriosus
FO = foramen ovale
MAP = mean arterial pressure
PBF = pulmonary blood flow
PVR = pulmonary vascular resistance
RDS = respiratory distress syndrome
SD = standard deviations
UA = umbilical artery
UV = umbilical vein

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ventricle. This division of venous return assures reasonable oxygenation of the heart and the brain. Only 7 per cent of the combined cardiac output is distributed to the fetal lung, because of intense pulmonary vasoconstriction. At birth pulmonary vascular resistance (PVR) decreases in response to increased pH, alveolar and arterial oxygen, and lung expansion. Hypoxia, acidosis, and cold increase PVR and decrease pulmonary blood flow (PBF). The combination of hypoxia and acidosis reduces PBF more than either alone. The decrease in PVR reduces pulmonary arterial blood pressure below systemic pressure, preventing right-to-left shunting of blood through the ductus arteriosus (DA). The increase in PBF is reflected in an increased left atrial pressure that functionally closes the foramen ovale (FO) and prevents right-to-left shunting of blood at this level. Anatomic closure of the FO may not take place for months, if ever.

The DA closes in response to oxygen, acetylcholine, and parasympathetic nerve stimulation. Premature animals do not constrict the DA even at $P_{aO_2}$ as high as 300 torr, while term animals do so when $P_{aO_2}$ is 60–100 torr. Functionally, the DA is closed immediately after birth, but it may not close anatomically in term infants for 10 to 12 days and in preterm infants for months. During the first one to two weeks of life, hypoxia, cold and acidosis may re-establish the fetal circulatory pattern.

![Diagram of fetal circulation](image_url)

**Fig. 2. Circulation of the fetus. Numbers in boxes are percentages of combined ventricular output.**

Courtesy of Drs. A.M. Rudolph and M. Heymann.
Figure 3 shows the effects of asphyxia on a newly born monkey. The initial period of rapid gasping is followed by primary apnea. The heart rate decreases and blood pressure falls after initially rising. Gasping is re-established following 2 minutes of apnea. Approximately 7 minutes later (following the last gasp), terminal or secondary apnea occurs, and persists until death or resuscitation. Brain damage begins after approximately 8 minutes of total asphyxia and is maximal after 12–13 minutes.

The oxygen content of blood decreases to near zero during the first two minutes of asphyxia. Cardiac output is then sustained by anaerobic metabolism. Plasma pH decreases in response to both respiratory and lactic acidosis, the latter resulting primarily from cardiac metabolism. The administration of glucose alone fails to improve circulation, but glucose and buffers administered together improve intracellular pH, heart rate, and cardiac output. Increasing pH towards normal reduces brain damage and prolongs the time to the last gasp. The maintenance of cardiac output during asphyxia is essential, because it delivers glucose to and removes hydrogen ion from heart and brain so that they continue to function. Hypothermia and hyperbaric oxygenation have little effect on reducing brain damage from hypoxia. Barbiturates protect the CNS but are not recommended as therapy because of their depressive side effects.

During asphyxia the brain's sodium pump fails, allowing potassium to leak from cells and edema fluid to accumulate intracellularly. This may depress ventilation and cause convulsions, but both are usually reversible if the asphyxia is short-lived.

**Assessment of the Fetus at Birth**

With the advent of fetal monitoring of ECG and acid–base balance, the severity of birth asphyxia should be reduced. These monitors give us early warnings of fetal difficulty and thus lead to earlier delivery and decreased neonatal depression.

The 1-minute Apgar score, if done correctly, is still a useful guide to neonatal well-being and resuscitation. It correlates well with acidosis and fetal survival. The 5-minute score may possibly correlate with brain damage. However, vigorous resuscitation reverses the latter trend. Each variable of the Apgar score (heart rate, respiratory effort, muscle tone, reflexes, and color) must be evaluated separately and each scored 0 to 2 (table 1).

**Heart Rate**

The heart rate at birth is usually 120 to 160 beats/min. Rates below 100 are frequently associated with severe asphyxia and low cardiac output. Low rates may rarely be associated with congenital heart lesions.

**Respiratory Effort**

Respiratory efforts normally begin before 30 seconds of age and are sustained by 90 seconds. The respiratory rate is between 30
TABLE 1. The Apgar Scoring System*

<table>
<thead>
<tr>
<th>Sign</th>
<th>0</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>Absent</td>
<td>Less than 100/min</td>
<td>More than 100/min</td>
</tr>
<tr>
<td>Respiratory effort</td>
<td>Absent</td>
<td>Slow, irregular</td>
<td>Good, crying</td>
</tr>
<tr>
<td>Muscle tone</td>
<td>Limp</td>
<td>Some flexion of extremities</td>
<td>Active motion</td>
</tr>
<tr>
<td>Reflex irritability</td>
<td>Absent</td>
<td>Grimace</td>
<td>Cough or sneeze</td>
</tr>
<tr>
<td>(response to catheter in nose)</td>
<td>Blue, pale</td>
<td>Body pink, extremities blue (acrocyanosis)</td>
<td>Completely pink</td>
</tr>
</tbody>
</table>

* Each sign is evaluated individually and scored from 0 to 2 at both 1 and 5 minutes of life. The final score at each time is the sum of the individual scores. (From Apgar V: A proposal for a new method of evaluation of the newborn infant. Curr Res Anesth 32:250–257, 1953.)

and 60/min. Apnea or bradypnea is often associated with severe asphyxia, acidosis, maternal drugs, infection, and CNS damage. Tachypnea (>60 breaths/min) is associated with hypovolemia, hypoxia, metabolic acidosis, CNS hemorrhage and pneumothorax, pneumomediastinum, and pulmonary disease (RDS, aspiration, etc.).

**MUSCLE TONE**

Most infants, even preterm infants, are active at birth and move all extremities. Asphyxia, maternal drugs, CNS damage, amyoctonia congenita, myasthenia gravis, or severe prematurity may cause flaccidity or poor muscle tone. Flexion contractures and absent finger and joint creases are signs of intrauterine central nervous system (CNS) damage.

**REFLEX IRRITABILITY**

The normal response to flicking the feet or inserting a nasal catheter is active movement. Failure to respond may be a sign of hypoxia or congenital muscle or CNS lesions.

**COLOR**

All infants are blue at birth but “pink up” when ventilation is established. Most are “pink centrally” by 90 seconds of age but may still have acrocyanosis (coldness and cyanosis of feet, hands and lips). If still generally blue by 90 seconds of age, low cardiac output, methemoglobinemia, polycythemia, cyanotic congenital heart disease, or pulmonary disease (RDS, airway obstruction, hypoplastic lungs, etc.) should be considered, especially if the patient fails to “pink up” with oxygen.

The infant who is pale at birth is generally vasoconstricted in response to hypovolemia, severe acidosis, aortic stenosis, meconium aspiration, or anemia. Those who are bright pink even while breathing room air may be magnesium or alcohol intoxicated or alkalotic. Those who are red may be polycythemic.

**Procedure**

Before delivery, someone other than the obstetrician should be available to evaluate and resuscitate the infant. If there is known intrauterine asphyxia or any of the conditions listed in table 2, two persons should be available for resuscitation, one to assist ventilation and the other to insert umbilical catheters and correct acid-base and volume deficits.

If the infant is severely depressed, the umbilical cord should be cut immediately, and the infant dried and transferred to a resuscitation cart equipped with a servo-controlled infrared heater. Otherwise, the infant should be held at the introitus until respiration has been established and then the cord cut.
NEWBORN RESUSCITATION

Stripping the umbilical cord of blood or clamping it after the onset of respiration causes the infant's blood volume,\(^{24}\) respiratory rate,\(^{25}\) lung water,\(^{26}\) pulmonary arterial blood pressure, and \(P_aCO_2\), to be higher\(^{27}\) and lung compliance, functional residual capacity\(^{28}\) and \(P_aO_2\),\(^{27}\) to be lower than those in infants whose cords are clamped earlier.

The 1-minute Apgar score is a guide to resuscitation, but should not be adhered to slavishly. If the infant is severely depressed, we should not wait the 1 minute to determine the score before instituting resuscitation. Figures 4A and 4B are guides to resuscitation of the newborn. With delivery of the head, the mouth and nose are suctioned. The umbilical cord is clamped, the infant is dried to reduce evaporative heat loss, and transferred to a resuscitation table, where he is kept warm with infrared heat. The mouth and nose are again suctioned briefly with a bulb-syringe, as prolonged suctioning may cause asphyxia, vomiting and dysrhythmias. The 1-minute Apgar score is determined and used to guide resuscitation.

**APGAR SCORE 8–10**

Approximately 90 per cent of infants fall into this category. They require nothing except upper-airway suctioning, their skin dried, and warmth. They must be carefully re-evaluated at 5 minutes, as some infants hypoventilate and become hypoxic once stimulation ceases.

**APGAR SCORE 5, 6, 7**

These infants have generally suffered some mild terminal asphyxia, i.e., asphyxia occurring just prior to birth, and respond to

<table>
<thead>
<tr>
<th>Maternal conditions</th>
<th>17) History of previous neonatal death</th>
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</thead>
<tbody>
<tr>
<td>1) Elderly primigravida (more than 35 years of age)</td>
<td>18) Prolonged rupture of membranes</td>
</tr>
<tr>
<td>2) Diabetes</td>
<td>Conditions of labor and delivery</td>
</tr>
<tr>
<td>3) Hypertension</td>
<td>1) Forceps delivery other than low elective</td>
</tr>
<tr>
<td>4) Toxemia</td>
<td>2) Vacuum extraction delivery</td>
</tr>
<tr>
<td>5) Maternal treatment with any of the following:</td>
<td>3) Breech presentation and delivery or other abnormal presentation</td>
</tr>
<tr>
<td>a) glucocorticoids</td>
<td>4) Cesarean section</td>
</tr>
<tr>
<td>b) diuretics</td>
<td>5) Prolonged labor</td>
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<tr>
<td>c) antimetabolites</td>
<td>6) Eclampsial umbilical cord</td>
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<tr>
<td>d) reserpine, lithium</td>
<td>7) Cephalopelvic disproportion</td>
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<td>e) magnesium</td>
<td>8) Maternal hypotension</td>
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<td>f) ethyl alcohol</td>
<td>9) Sedative or analgesic drugs given intravenously within 1 hour of delivery or intramuscularly within 2 hours of delivery</td>
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<td>g) (\beta)-adrenergic drugs (to stop premature labor)</td>
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<tr>
<td>6) Abnormal estriol levels</td>
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<tr>
<td>7) Anemia (hemoglobin less than 10 g/100 ml)</td>
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<tr>
<td>8) Blood type or group isoimmunization</td>
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<tr>
<td>9) Previous birth of child with a hereditary disease</td>
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<tr>
<td>10) Current maternal infection or infection during pregnancy with rubella, herpes simplex, syphilis</td>
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<tr>
<td>11) Abruptio placenta</td>
<td></td>
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<tr>
<td>12) Placenta previa</td>
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<tr>
<td>13) Antepartum hemorrhage</td>
<td></td>
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<tr>
<td>14) History of previous infant with jaundice, thrombocytopenia, cardiorespiratory distress, congenital anomalies</td>
<td></td>
</tr>
<tr>
<td>15) Narcotic, barbiturate, tranquilizer or psychodelic drug use</td>
<td></td>
</tr>
<tr>
<td>16) Ethyl alcohol intoxiciation</td>
<td></td>
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<tr>
<td>17) History of previous neonatal death</td>
<td></td>
</tr>
<tr>
<td>18) Prolonged rupture of membranes</td>
<td></td>
</tr>
</tbody>
</table>

**Fetal conditions**

1) Multiple births
2) Polyhydramnios
3) Meconium-stained amniotic fluid
4) Abnormal heart rate or rhythm
5) Acidosis (fetal scalp capillary blood
6) Decreased rate of growth (uterine size)
7) Premature delivery
8) Amniotic fluid surfactant test negative or intermediate within 24 hours of delivery

**Neonatal conditions**

1) Birth asphyxia
2) Birth weight inappropriate for gestational age
3) Meconium staining of the skin, nails or umbilical cord
4) Signs of cardiorespiratory distress
vigorous stimulation and oxygen blown over their faces. If they fail to do so within 60 seconds, assisted ventilation with an oxygen-enriched mixture is instituted. These infants are generally well by 5 minutes of age.

APGAR SCORE 3, 4

These infants are moderately depressed, cyanotic, and may have poor respiratory efforts. However, because their asphyxia is moderate, they generally respond to bag-and-mask ventilation alone. The acid–base status of umbilical cord artery and vein blood should be determined, as one occasionally finds severe metabolic acidosis despite what appears clinically to be adequate ventilation and perfusion. If pH is less than 7.20, pH and PaCO₂, of blood from a warmed heel or from an artery should be determined 10 minutes later. If, at this time, the pH has not improved or is lower, buffer therapy may be necessary (see below).

![Diagram of birth resuscitation and Apgar score evaluation](http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931527/)

**Fig. 4A.** Guide to ventilatory and acid–base resuscitation of the newborn. Therapy is based in part on the one-minute Apgar score. If thick meconium is present at birth, the trachea should be suctioned before stimulating the patient to cry or breathe. If the patient’s condition does not improve, therapy should move to the next panel to the right. The volume of sodium bicarbonate (ml) required for total correction of base excess (BE) is obtained by multiplying BE x wt in kg x 0.5. During IPPB PaCO₂ is maintained between 30 and 45 torr. PaO₂ is maintained between 80 and 80 torr. Base deficit is greater as it becomes more negative.
APGAR SCORE 0–2

These infants have suffered severe asphyxia, are usually apneic, and require immediate resuscitation.

Resuscitation

PULMONARY RESUSCITATION

Endotracheal intubation should be accomplished quickly and positive-pressure ventilation instituted. It is often difficult to ventilate these patients with a bag and mask. Since it is impossible to determine whether the apnea is primary or secondary, delay in establishing ventilation may result in brain damage.

Adequacy of Ventilation

The adequacy of ventilation is determined by assurance that both sides of the chest, especially the apices, expand equally, and heart rate and color improve. The only absolute way to assure adequacy of ventilation is to measure blood gases and pH. Equal breath sounds are no assurance of bilateral ventilation because breath sounds are well transmitted in these small chests and may be present despite pneumothorax or atelectasis. The breath sounds should be louder over the chest than over the stomach.

Ventilatory Pressures

A pressure of 25–30 cm H₂O is adequate to ventilate most asphyxiated infants. However, those with stiff lungs (erythroblastosis or congenital anomalies of lung) may require much higher pressures, and are prone to develop pulmonary air leaks during ventilation. If this occurs, the intrapleural gas should be drained by tube thoracostomy.

Tracheal Intubation

To facilitate visualization of the infant’s anterior larynx, the head is placed in a neutral, “sniffing” position, neither flexed nor extended (fig. 5). The laryngoscope is held between the thumb and first finger of the left hand and the chin grasped firmly with the second and third finger of that hand. This “welds” the hand and chin into one, decreasing the likelihood of pharyngeal lacerations if the child moves its head. The small finger of the left hand applies pressure over the hyoid bone to move the larynx posteriorly and expose the vocal cords. Heart rate and rhythm should be monitored during laryngoscopy, as dysrhythmias or bradycardia are common. The endotracheal tube should be inserted approximately 2 cm below the glottis and the lungs gently expanded with an anesthesia bag or by mouth. If Pao₂ rises above 100 torr, Fio₂ should be reduced, so

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B. Guide to volume expansion. If the infant has an Apgar score of 0, 1, or 2, and is cyanotic and depressed, blood pressure should be measured by umbilical artery catheter or doppler and blood pressure device (see text). The volumes suggested for blood volume expansion are initial volumes only. More may be needed subsequently.
long as $P_{ao2}$ remains in the normal range (50 to 80 torr). This is especially important in preterm infants, where a $P_{ao2}$ of 150 torr for 2 to 4 hours may cause retrolental fibroplasia.1

Routine Tracheal Suctioning

Sixty per cent of patients born following meconium staining of the amniotic fluid have meconium below the vocal cords which is pulled into the periphery of the lung with the onset of respiration.28-30 Of these, 18 per cent have respiratory difficulties during the first few days of life. Therefore, infants born following parturition or "pea-soup" meconium staining require immediate tracheal suctioning to remove this material from the lung. Thin meconium does not require suctioning. Meconium or other large particulate material is best removed by using the endotracheal tube as a straw and sucking on it with one's mouth while withdrawing the tube from the treachea. If meconium is retrieved, suctioning is quickly repeated and then the lungs gently re-expand. The absence of meconium at the larynx does not preclude its presence below the vocal cords. Heart rate should be monitored continuously during airway suctioning.

1Phibbs RH: Unpublished data.

Vascular Resuscitation

Vascular resuscitation is a much neglected aspect of infant resuscitation. Few textbooks even mention it, despite the fact that need occurs commonly, especially in infants who are born before term.31

If depression is severe or the infant is hypotensive and fails to respond immediately to ventilation, an umbilical-artery catheter should be inserted to measure blood gases, pH, and blood pressure, and to administer drugs. The technique for inserting these catheters has been well described.32 In many instances little more than ventilation is necessary to improve peripheral and pulmonary circulation, since ventilation expands the lung, raises $P_{ao2}$, decreases pH, reduces PVR, and improves PBF.18 However, 20-30 per cent of those infants severely depressed are hypovolemic and/or severely acidic, and survival depends upon expansion of blood volume and correction of the acidosis (unpublished data).

Correction of Acidosis

The respiratory component of acidosis is corrected by ventilation, the metabolic component by sodium bicarbonate or Tham. Sodium bicarbonate is less expensive and does not produce the severe hypoglycemia, hypocalcaemia, hypokalemia and apnea occasionally seen with Tham.

If the Apgar score is 2 or less at 2 minutes, or 5 or less at 5 minutes, despite oxygen, stimulation and assisted ventilation, 2 or 3 mEq of NaHCO$_3$ per kg body weight are given through the umbilical artery, or through the venous catheter if the latter's tip lies in the inferior vena cava. Buffers given into the liver may cause hepatic necrosis.33 A sample of blood for determination of oxygen and carbon dioxide tensions and pH is obtained before giving buffers. If the pH is less than 7.1, one fourth of the base deficit is corrected with sodium bicarbonate.$\dagger$ If the pH is more than 7.1, assisted ventilation is

$\dagger$ mEq NaHCO$_3$ = 0.6 x weight in kg x base excess, the infant's bicarbonate space being 60 per cent of body weight.
continued and blood-gas and pH measurements repeated within 5 minutes, for many infants will spontaneously correct pH's above 7.2 and require no further buffers.

Figure 6 shows the effects of acid-base correction on $P_{ao}$ when ventilation is maintained constant. No significant increase in $P_{ao}$ occurred until $pH$ rose above 7.1 to 7.2, the point above which PVR rapidly decreases. It is unclear whether this increase in $P_{ao}$ is due to an increase in pH or expansion of vascular volume.

**Volume Expansion**

Approximately 60 per cent of preterm infants asphyxiated in utero are volume-depleted at birth. Because many are also acidic, they are peripherally constricted and have normal mean arterial pressures (MAP) (Fig. 7). When the acidosis is relieved, hypotension ensues. Unless blood pressure is monitored as part of our routine delivery room care, hypotension will be missed.

**Detection of hypovolemia:** Hypovolemia is best detected by blood pressure measurements, patient color, perfusion (capillary filling), and pulse volume. After 24 hours of age, urinary output and specific gravity are also useful.

Arterial pressure is measured either by a doppler system or from the umbilical artery. Figure 8 shows the normal MAP ± 2 SD.

**Fig. 6.** The effects of rapid administration of sodium bicarbonate on $P_{ao}$ and pH when ventilation is held constant. Note: $P_{ao}$ increased when pH rose above 7.1 to 7.2. Courtesy of W. H. Tooley.

Below 2 SD the infants are considered hypotensive and in most cases, hypovolemic. Table 3 shows the normal mean, systolic, and diastolic pressures. If transducers are not available for intravascular pressure measurements, the umbilical-artery catheter is connected to a venous-pressure manometer and the height of the column of water divided by 13.6 (the density of mercury). The value obtained is the MAP.

**Intrathoracic** venous pressures (right or left atrial) are also useful in determining hypovolemia. The right and left atrial pres-

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**Fig. 7.** The effects of sodium bicarbonate on blood pressure, heart rate and hematocrit. Note: Hypotension developed following administration of NaHCO$_3$. The hematocrit (hct) decreased when fluid was "pulled" into the vascular space to compensate for the hypovolemia present prior to administering NaHCO$_3$ (HR > 200/min, decreasing hematocrit and BP). The heart rate decreased and the hypotension was relieved following administration of albumin, 1 g/kg body weight. ($P_{ao}$ stands for mean arterial pressure.) From Phibbs RH: In Problems of Neonatal Intensive Care Units. Report of 59th Ross Conference on Pediatric Research. Edited by JF Lucey. Columbus, 1969.
Hypovolemia is suspected and an arterial catheter cannot be inserted, a catheter should be placed in the left atrium. Hypovolemic infants are usually pale and have poor perfusion and capillary filling. Their extremities are cool and their pulses (especially radial and posterior tibial) weak or absent. Those intoxicated with alcohol or hypermagnesemia are frequently peripherally vasodilated, pink, and grossly hypotensive.

Treatment of hypovolemia: Volume expansion is the key to treatment of this physiologic abnormality. This is best done through an umbilical-artery or venous catheter. If one has advance warning of a potentially hypovolemic infant (any premature or asphyxiated fetus), O = negative low-liter blood should be cross-matched against the mother’s blood so it is available in the delivery room at the time of birth. If the situation is urgent, one can clean the placenta with iodine and withdraw blood from the umbilical artery or vein. The syringe should contain 1 unit of heparin for each ml of blood withdrawn. This blood belongs to the infant and has good clotting factors. Placental blood can be given in an emergency but should not be used routinely because of the potential problem of sepsis. If blood is unavailable, the patient is given

Table 3. Average Systolic, Diastolic, and Mean Blood Pressures (Torr) during the First 12 Hours of Life in Normal Newborn Infants Grouped According to Birth Weight

<table>
<thead>
<tr>
<th>Body weight</th>
<th>1 Hour</th>
<th>2 Hours</th>
<th>3 Hours</th>
<th>4 Hours</th>
<th>5 Hours</th>
<th>6 Hours</th>
<th>7 Hours</th>
<th>8 Hours</th>
<th>9 Hours</th>
<th>10 Hours</th>
<th>11 Hours</th>
<th>12 Hours</th>
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<td>1,001–2,000 g</td>
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<tr>
<td>Birth weight</td>
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<td>&gt;3,000 g</td>
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<tr>
<td>Systolic</td>
<td>70</td>
<td>67</td>
<td>65</td>
<td>65</td>
<td>66</td>
<td>66</td>
<td>67</td>
<td>67</td>
<td>68</td>
<td>70</td>
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<tr>
<td>Diastolic</td>
<td>44</td>
<td>41</td>
<td>40</td>
<td>41</td>
<td>40</td>
<td>41</td>
<td>41</td>
<td>41</td>
<td>44</td>
<td>43</td>
<td>41</td>
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<tr>
<td>Mean</td>
<td>53</td>
<td>51</td>
<td>50</td>
<td>50</td>
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<td>50</td>
<td>50</td>
<td>51</td>
<td>53</td>
<td>54</td>
<td>51</td>
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</tbody>
</table>

1–2 g albumin or 10–15 ml physiologic saline solution or plasma per kg body weight. The volume of fluid or blood required to expand blood volume may be enormous. At times it may exceed 50 per cent of the predicted blood volume (85 ml/kg) (fig. 9). The blood pressure tracing may provide an additional clue to hypovolemia, in that there is a large fall in pressure with each inspiration when hypovolemia is present. Correction of blood volume returns the pressure tracing to normal (fig. 9).

Other causes of hypotension: Pulmonary air leaks (pneumothorax or pneumomediastinum) occur in 1 per cent of normal infants and 10 per cent of those born following meconium aspiration. These may be associated with hypotension if they interfere with venous return. A reduced cardiac output and hypotension may occur if the infant is hypoglycemic (blood glucose <20 mg/100 ml or <30 mg/100 ml in preterm and term infants, respectively). Hypoglycemia should be treated with 5 ml of 20 per cent dextrose in water and hypocalcemia with 100 mg calcium gluconate/kg body weight. These doses may require repeating.

Infants who are hypermagnesemic will respond to volume repletion and/or calcium infusions.

Cardiac Massage

If the heart is not beating or the rate is less than 100 beats/min at 1 minute, ventilation should be assisted and closed-chest cardiac massage instituted. Both thumbs are placed on the body of the sternum at the junction of the lower and middle thirds and the back supported with the fingers (fig. 10). The sternum is compressed approximately two thirds the distance to the vertebral column 100 times per minute. The effectiveness of the cardiac massage is monitored through the umbilical-artery catheter and by pupil size (fig. 11).

Ideally, with massage, the systolic pressure should be 60–80 torr. This will maintain the diastolic pressure at 15–20 torr, a level probably adequate for coronary perfusion.

RESUSCITATION DRUGS

Table 4 provides a list of drugs useful for cardiovascular resuscitation. The effectiveness of vasopressors is decreased in an acidic medium. Therefore, drug effectiveness is improved when increasing pH towards normal. Care must be taken to control the volume of fluid infused during administration of drugs. However, to accomplish this with drugs such as isoproterenol they must be given in high concentrations; the deadspace of the catheters may contain large amounts of drugs that could cause dysrhythmias or cardiac arrest. The deadspace should thus be cleared before additional drugs or fluids are injected.

WHEN TO DISCONTINUE THERAPY

The decision when to stop resuscitation remains a personal one, based on one's own experience and with consideration given to the parents' feelings. In making this decision we must always consider the probability of severe neurologic damage and the chances of even semi-normality of the infant. If the chances are poor, resuscitation should not be pursued.

**TABLE 4. Drugs Used during Resuscitation**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Indication</th>
<th>Dose</th>
<th>Route</th>
<th>Response</th>
<th>Complication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td>Bradycardia</td>
<td>0.03 mg/kg</td>
<td>iv</td>
<td>Increased heart rate</td>
<td>Marked tachycardia, diminished cardiac output</td>
</tr>
<tr>
<td>Calcium gluconate</td>
<td>Low cardiac output</td>
<td>100 mg/kg over 5–10 minutes (ECG monitoring)</td>
<td>iv</td>
<td>Improved cardiac output</td>
<td>Bradycardia, dysrhythmias</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>&quot;Flat line&quot; ECG</td>
<td>0.1 ml/kg of a 1:10,000 solution</td>
<td>iv</td>
<td>&quot;Flat line&quot; ECG converted to some rhythmic response</td>
<td>Hypertension; ventricular fibrillation</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>Bradycardia, hypotension, low cardiac output</td>
<td>4 mg/250 ml 5 per cent dextrose in water, until heart rate increases</td>
<td>iv</td>
<td>Increased heart rate, improved cardiac output</td>
<td>Dysrhythmias, low cardiac output if heart rate more than 180–200/min</td>
</tr>
<tr>
<td>Dopamine†</td>
<td>Low cardiac output</td>
<td>5 μg/kg/min, increased to 50 μg/kg/min as necessary</td>
<td>iv</td>
<td>Improved cardiac output, slightly increased heart rate</td>
<td>Dysrhythmias</td>
</tr>
</tbody>
</table>

* Doses given are, in general, starting doses, and may have to be increased. Most drugs tend to be more effective when pH > 7.15.
† Useful only when blood volume is adequate.
References


