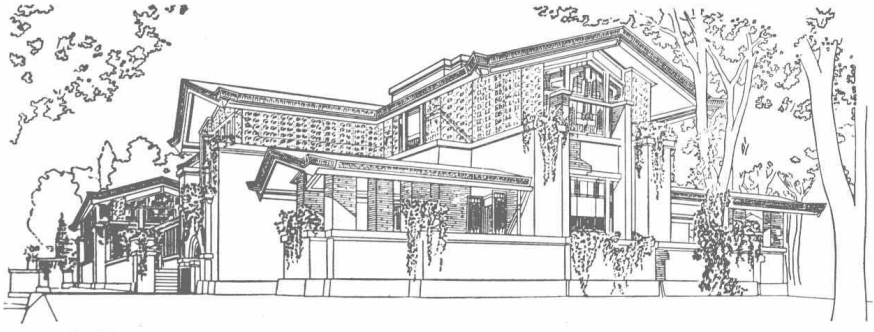
Resuscitation
OF THE NEWBORN

JOSEPH D. RUSS, M.D.

Resuscitation
OF THE NEWBORN

By JOSEPH D. RUSS, M.D., F.A.A.P., *Assistant Professor of Pediatrics, Tulane University School of Medicine, Senior Pediatrician Touro Infirmary, New Orleans, Louisiana*



CHARLES C THOMAS · PUBLISHER
Springfield · Illinois · U. S. A.

CHARLES C THOMAS • PUBLISHER
BANNERSTONE HOUSE
301-327 EAST LAWRENCE AVENUE, SPRINGFIELD, ILLINOIS, U.S.A.

Published simultaneously in the British Commonwealth of Nations by
BLACKWELL SCIENTIFIC PUBLICATIONS, LTD., OXFORD, ENGLAND

Published simultaneously in Canada by
THE RYERSON PRESS, TORONTO

Library of Congress Catalog Card Number: 53-8690

Printed in the United States of America

PREFACE

NEONATAL ASPHYXIA is a condition which has long perplexed the obstetrician and pediatrician. It is the major source of death in the first 24 hours of life. The medical profession must recognize the fact that neonatal asphyxia can be prevented, and also treated if it occurs, that the neonatal mortality from asphyxia can be materially lowered by proper knowledge in advance of the birth of the baby. This book is based upon my own personal work of some 2000 resuscitations since 1938 and a considerable amount of published information. It is the purpose of this book to correlate these two.

The information is divided into five major chapters, each of which will contain detail sufficient for reference and teaching. These chapters are:

1. Definitions, classifications, statistics, pathology.
2. Various causes of neonatal asphyxia.
3. Treatment of neonatal asphyxia.
4. The after-effects of neonatal asphyxia.
5. The teaching and training of medical students, residents, and interns in the proper methods of prevention and treatment.

Many physicians have contributed largely to my effort in this particular field, too many to mention individually. It is my honor and privilege to dedicate what good this book may accomplish to my wife, without whom, any effort could not have succeeded; to Dr. Arthur A. Caire, who initiated my work and has continued to be a constant

source of assistance; to Dr. Robert A. Strong, whose advice and inspiration really encouraged me to go further with this work after its first start; to Dr. Ralph A. Platou, who has materially assisted me in properly coordinating the many things which have arisen, and who is outstanding in the teaching of the prevention and treatment of neonatal asphyxia.

J.D.R.

CONTENTS

RESUSCITATION
OF THE NEWBORN

CHAPTER 1

DEFINITION AND CLASSIFICATIONS

Neonatal asphyxia has been defined as the failure of respiration in the newborn or imperfect breathing in the newborn. It is stated that asphyxia really means an insufficient amount of oxygen in the circulating blood stream. Etymological analysis of this particular term reveals that asphyxia really means cessation of the heart beat or absence of throbbing. By common misusage, the word now refers to the failure of respiration; the proper term should be apnea neonatorum.

Potter feels that primary anoxemia is always intrauterine and that the term asphyxia should be retained only for those conditions in which live born infants died who had suffered in utero from primary lack of oxygen. The presence of neonatal apnea may be stated to exist whenever the baby has failed to take a spontaneous respiratory movement within 30 seconds of severing the cord. Schrieber has shown that periods longer than 30 seconds of insufficient oxygenation of the blood by lack of respiration will tend to produce certain irreversible changes in the cerebral cortex. In order to be on the very safe side, it is necessary to state that asphyxia does exist immediately after the 30 second period and that it then becomes imperative to initiate interference in order

to insure adequate oxygenation so that cerebral damage might be prevented.

Neonatal asphyxia has been variously classified by many men. One of the oldest classifications has been that of:

(a) Asphyxia livida.
(b) Asphyxia pallida.

In the former condition, the asphyxia is defined to be that in which a baby fails to breathe; the color of the child is a bluish-red cyanosis, some muscle tone is present and some reflexes are present to varying degrees. In the latter condition of asphyxia pallida, the child is limp, gray-greenish and has little or no muscle tone and is in a state of impending death. This particular classification is quite outmoded and does not bear the scrutiny of close clinical evidence. In most cases, asphyxia pallida and asphyxia livida represent poor clinical classification because they do not adequately express either the degree of cyanosis which exists, or the amount of asphyxia which is present. It has been my experience, augmented by experiences of others, that many cases of asphyxia livida are even more severely shocked and closer to death than those presenting asphyxia pallida. This particular classification, I believe, should no longer be taught, because it is not only a poor pathological terminology, but it is not properly borne out by clinical evidence.

Tow has preferred to divide asphyxia into the following classifications:

1. *Asphyxia of peripheral origin:* By which is meant asphyxia resulting from some interference with the entrance of oxygen into the circulation. This occurs with any interference of the circulation of the umbilical cord or placenta, such as premature separation of the placenta;

kinks, knots of the cord; and profound anemia of the mother. It may also occur from the wrapping of the cord around the baby's neck. It may occur following birth when interference is produced by the aspiration of foreign material into the bronchial or tracheal passages.

2. *Asphyxia of central origin:* Appears when the function of the infant's respiratory center is disturbed. This condition is caused by direct injury to the brain, toxicity, shock, or the reaction of narcotics, anesthetics, and analgesics.

Because the true pathology of asphyxia is really the production of cerebral anoxia, it is necessary to follow Tow in his classification of cerebral anoxia. These are:

1. *Anoxic:* The blood is insufficiently saturated with oxygen.
2. *Anemic:* The oxygen capacity of the blood is abnormally low.
3. *Stagnant anoxia:* Less than the normal quantity of blood is circulating.
4. *Histotoxic anoxia:* The Hemoglobin oxygen tension is perfectly normal, but the tissue cells of the brain are in such condition that they cannot use the oxygen properly.

This classification is a splendid one based on pathological and clinical findings. But its use for clinical purposes is impractical in order to analyze the amount of asphyxia and the potential cause. It is really a classification of asphyxia which can be used when the entire clinical pattern has been depicted and a review of all conditions may be surveyed.

Potter and Adair have outlined a pathological classification of asphyxia which is quite adaptable for this subject:

CONDITIONS INTERFERING WITH ESTABLISHMENT OF NORMAL RESPIRATION

A. Abnormalities of the central nervous system.

1. Malformation of the brain.
2. Depression of the respiratory center by:
   a. Pressure from intracranial hemorrhage.
   b. Cellular injury from anoxemia.
   c. Cellular injury from drugs.
3. Immaturity of the cells of the respiratory center.

B. Abnormalities of the lungs.

1. Immaturity of the parenchyma with insufficient alveolar development to permit gaseous exchange.
2. Hyperplasia of lung tissue caused by a defect of germ plasm.
3. Obstruction of bronchi and alveoli.
   a. Aspiration of amniotic fluid, meconium, blood or mucous.
   b. Intrauterine pneumonia.

C. Mechanical compression of lungs.

1. Subdiaphragmatic pressure.
   a. Polycystic kidneys.
   b. Intestinal distention.
   c. Peritoneal effusion.
2. Intrapleural pressure.
   a. Diaphragmatic hernia.
   b. Massive cardiac hypertrophy.
   c. Plural effusion.

This excellent classification is one which is of most concern in a pathological report.

Probably the best clinical classification is the following one advocated by Flagg:

1. *Mild asphyxia:* The infant resists movement of the head and limbs. Mucous plugs and amniotic fluid fill the

mouth and pharynx. Muscle tone is good. Conjunctival reflexes are present.

2. *Moderate asphyxia:* Muscle tone is absent. There is no resistance to opening the mouth. No reflex irritation is induced by aspiration or by stimulation of the glottis.

3. *Severe asphyxia:* The infant does not respond to attempts of resuscitation. He appears livid or pallid. There is absence of any respiratory movement and only an occasional flicker of the cardiac impulse may be detected through the thoracic wall. The upper respiratory tract has become a collapsed tube. All reflexes are naturally absent.

It is unfortunate that there has not been a greater emphasis upon the proper diagnosis of the degree of asphyxia based upon clinical evidence and the pathological cause. In practically all statistical summaries, it is impossible to obtain such information. In the subsequent chapters on the causes of asphyxia, I shall attempt to indicate, based upon my own personal experience, what degree of asphyxia usually exists with each causative factor. In this way the very method of making a diagnosis of the degree of asphyxia will also provide a clue indicating the possible cause.

NEONATAL MORTALITY

In reviewing the causes of neonatal mortality, the importance of asphyxia as a cause of death in the first few hours of life is becoming more and more paramount.

Potter and Adair, reviewing autopsies of 526 fetuses and infants from 1931 to 1938 found asphyxia (or anoxemia) the cause of death in 18.5 per cent. This was the greatest cause of death exceeding malformations, cerebral hemorrhage, and prematurity.

Holland and Lane-Claypon in reviewing 376 deaths found that 24.8 per cent were from complications of labor, including obstruction to the cord. Of the causes of death, the majority were from asphyxia.

Adair, *et al.* found that suffocation and aspiration produced death in 17.18 per cent of 483 deaths.

Bundesen, *et al.* in reviewing the necropsy of 1043 infants less than 14 days of age found that asphyxia was the cause of death in 6.2 per cent of the babies.

Improvement in the use of analgesia and anesthesia, refinements of technique in the methods of delivery, increasing use of Caesarean Section in cases which might produce cerebral damage to the baby have certainly lowered the number of deaths from cerebral hemorrhage. Upon this basis alone, asphyxia causing death has necessarily taken an increase in the past several years so that it now occupies first place among the causes of death. As indicated by Potter and others, the major cause of death in newborn infants is now asphyxia and is based on a review of 2000 cases causing death in 20.3 per cent of cases. My own personal statistics based on an analysis of 327 deaths at Touro Infirmary, of which 168 were autopsied, revealed that neonatal asphyxia may be determined as the primary cause of death in 17.8 per cent. It is interesting to note that of the remaining 159 cases which were not autopsied, 22 per cent were diagnosed according to the various terminologies indicating neonatal asphyxia. Statistics on infant mortality under 28 days as provided by the Children's Bureau Statistical Series Number 9, 1951, reveals that post-natal asphyxia and atelectasis is the major cause of death — 3.6/1000 live births.

It is definitely indicated that as neonatal asphyxia is the most common cause of death in the neonatal period it must be investigated extremely thoroughly to de-

termine its causes, to provide for proper prophylaxis, and to properly treat it once it occurs. In the last 20 to 25 years, the increasing use of analgesics and anesthetics has increased the actual number of cases of asphyxia. The increasing technical improvements and proper knowledge of proper resuscitation during this same period has probably caused a decrease in the amount of deaths of asphyxiated babies, but it has not held pace with the decreasing number of deaths in the neonatal period. This is based not on percentage figures but on actual quantitative figures alone.

PATHOLOGY

The pathology of anoxia is neither specific or constant. The findings are few and varied. Edema of the brain; petechial hemorrhages of the brain, lungs, abdominal organs; passive congestion of the lungs; dilatation of the right ventricle; all of the foregoing are common findings but need not necessarily be present. In fact, in many cases of death from asphyxia no particular pathological finding can be discovered.

The so-called "asphyxial membrane" is a fairly constant finding in cases of asphyxia occurring after aspiration of tracheal contents. There has been some discussion, of late, that the asphyxial membrane does not represent a true membrane arising from inhaled detritus but rather a true reaction of the alveolar walls – preventing absorption of oxygen. Greater numbers of observers, however, believe that the asphyxial membrane represents an artificial membrane composed of amniotic fluid, blood, meconium and lanugo covering the alveolar walls or filling the alveolus; in either event absorption of oxygen is impossible. The asphyxial membrane occurs clinically in those cases which live from 12 to 48 hours after birth,

with recurrent periods of apnea – usually following traumatic deliveries, prolonged labors, delivery by Caesarean Section, and in cases in which the mouth, pharynx, trachea, and bronchial tree are filled with a viscous meconium stained mucus.

In past months, discussion of the nature of the hyaline-

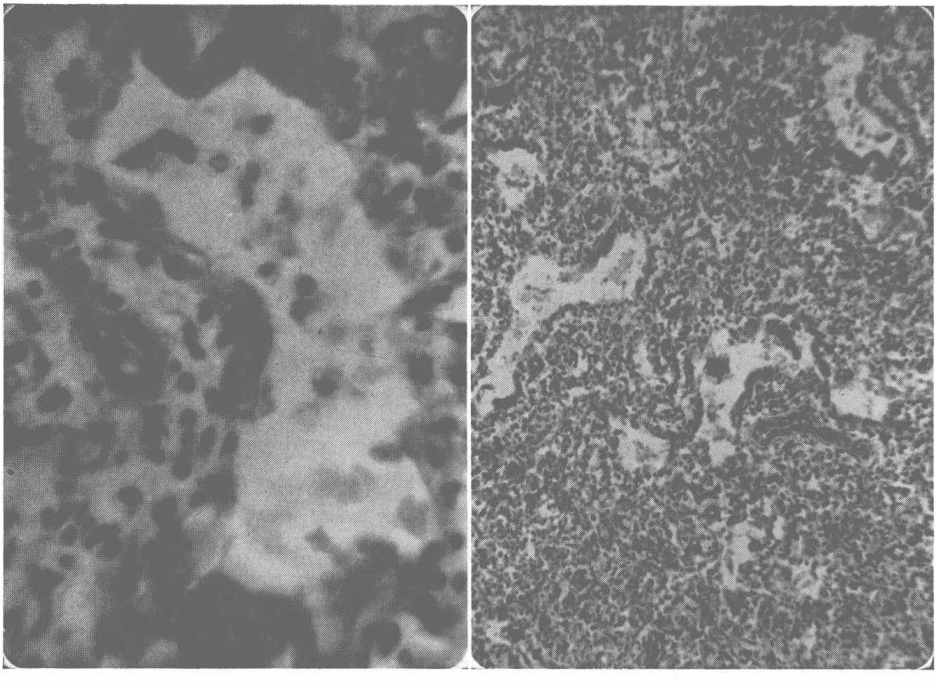


Figure 1. Asphyxial membrane. (Courtesy, Dr. John Dent.)
Figure 2. Asphyxial membrane. (Courtesy, Dr. John Dent.)

like membrane (asphyxial membrane) has been widespread. Statistics and research have pointed out that the membrane is probably not a product of extraneous material introduced into the alveoli from the upper respiratory tree, but rather a true product of the alveolar epithelium caused by irritation, trauma, oxygen deprivation, or some unknown cause. The unknown cause has also been