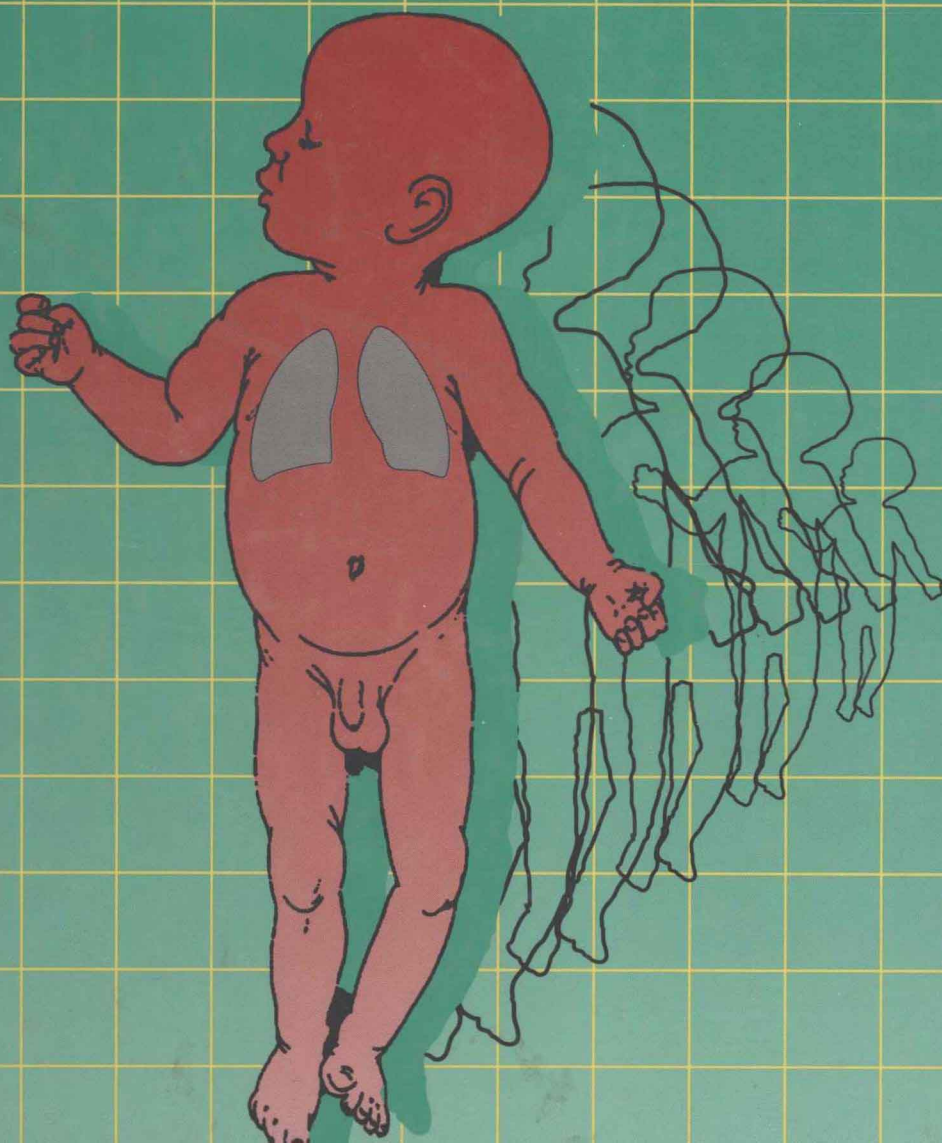


NEONATAL^{and} PEDIATRIC RESPIRATORY CARE

PATRICIA BECK KOFF • DONALD EITZMAN • JOSEF NEU

Second Edition



SECOND EDITION

Neonatal and Pediatric Respiratory Care

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Neonatal and Pediatric Respiratory Care

To health care providers around the globe who have provided us with their insight and consistently enabled us to see beyond our current capabilities.

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PREFACE

We are pleased to provide a second edition of this text, and we thank the respiratory therapists, physicians, and nurses who used the first edition. Several advances have occurred in our specialty area in the last 5 years that make it appropriate to provide an update. Artificial surfactant has met with approval, extracorporeal membrane oxygenation (ECMO) has been developed further, and various other treatment regimens have been expanded.

The philosophy and format of this second edition remain similar to the first. We believe a solid base of understanding regarding neonatal and pediatric patients is required before therapy can be rendered, to avoid the erroneous approach of treating children as “just small adults.” With that in mind, we again have organized the book into four broad areas: general introductory concepts, disease states, equipment and therapy, and outcomes.

In the introductory section we have expanded Chapters 1 and 3 to include additional information on surfactant, teratogens, assessment techniques, and prenatal problems. Chapters 5 through 13 address the various related diseases and include updated information that has affected understanding and treatment of problems in the past 5 years. In the equipment and therapy section almost every chapter has been expanded significantly. The chapter on oxygen therapy has been expanded with new tables and a segment on monitoring. Pharmacology now addresses more maternal substance use and abuse, new medications, and potential health risks that care giv-

ers should note. Airway care includes additions in tracheostomy care and innovative means of providing chest physiotherapy. The chapters on ventilation incorporate new ventilators and techniques as well as high-frequency ventilation.

The first edition “Novel Methods” chapter has been eliminated in favor of expanding the mechanical ventilation chapters and adding two additional chapters, on surfactant and ECMO, respectively. The home care chapter also has been expanded, as has the developmental outcomes chapter, which now includes early data related to outcome and surfactant, high-frequency ventilation, and ECMO use. Additional tables have been added throughout the book to enhance the summarizing of issues and, it is hoped, to facilitate the use of the text in teaching settings.

All of these revisions required the assistance of a number of authors, and we wholeheartedly thank them. Their willingness to revise and expand their chapters and remain flexible so that their material could be incorporated into the overall text was much appreciated. In addition, we thank Randy Wilkening for his review of the neonatal portion of pharmacology, and Pat Pike for her ongoing coordination with manuscript production, mailing, and computer magic. Thanks also are due Robert Kacmarek and Dean Hess for providing Patricia Koff with editorial direction.

The Mosby–Year Book team of David Marshall, Julie Tryboski, and Wendy Sweetland also deserve

unquestionable thanks. David long will be remembered for providing clear answers, suggestions, and directions, even in somewhat unclear situations. Julie and Wendy always kept things moving.

Special thanks from P. Koff to David and Teresa Crichton of Woodmoor, Colorado, who warmly shared their great mountainview home during production of this second edition.

Finally, we thank our spouses (Bob, MaryLou, and Carol), who consistently keep us going with their love and encouragement, regardless of the obstacles presented to us.

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CONTENTS

Preface

1 / Development of the Cardiopulmonary System	1
<i>P.B. Koff</i>	
2 / Physiologic Development	16
<i>D.V. Eitzman</i>	
3 / Patient Assessment	31
<i>M. Behnke and P.B. Koff</i>	
4 / Radiographic Evaluation	53
<i>J.L. Williams</i>	
5 / Neonatal Parenchymal Diseases	75
<i>D. Burchfield and J. Neu</i>	
6 / Pediatric Parenchymal Diseases	92
<i>K. McCoy</i>	
7 / Obstructive Airway Diseases in Infants and Children	102
<i>C.D. Kurth and S.R. Goodwin</i>	
8 / Surgical Lesions of Pediatric Airways and Lungs	128
<i>C.E. Bagwell</i>	
9 / Congenital Heart Disease	155
<i>M.J. Polak and R.L. Bucciarelli</i>	
10 / Apnea of Prematurity	178
<i>E.C. Eichenwald and A.R. Stark</i>	
11 / Sudden Infant Death Syndrome and Apnea Disorders	186
<i>M.H. Wagner and S.E. Chesrown</i>	

12 / Respiratory Care of the Neurologically Injured and Neuromuscular Impaired Child	196
<i>K.J. Kelly</i>	
13 / Adult Respiratory Distress Syndrome in Children	212
<i>R. Vidal, N. Kissoon, L. DeNicola, and T.B. Rice</i>	
14 / Oxygen Therapy	226
<i>B.G. Wilson and D.A. Desautels</i>	
15 / Pharmacology	246
<i>P.B. Koff and A.G. Durmowicz</i>	
16 / Continuous Positive Airway Pressure	265
<i>M.P. Czervinske</i>	
17 / Airway Care and Chest Physiotherapy	285
<i>A.A. Scott and P.B. Koff</i>	
18 / Arterial Blood Gas Analysis and Other Cardiopulmonary Monitoring	303
<i>M.P. Czervinske</i>	
19 / Mechanical Ventilation	324
<i>P. Betit, J.E. Thompson, and P.K. Benjamin</i>	
20 / Mechanical Ventilators	345
<i>D.A. Desautels and P. Blanch</i>	
21 / Transport	374
<i>L.R. Roy, W. Cunningham, and K. Heitzman</i>	
22 / Surfactant Replacement Therapy	390
<i>C.L. Bose and M.S. Wright</i>	
23 / Extracorporeal Membrane Oxygenation	402
<i>J.E. Thompson and N. Perlman</i>	
24 / Pediatric Home Care	414
<i>D.D. Cimo and S. vanBeuzekom</i>	
25 / Developmental Outcomes	440
<i>F.D. Eyler</i>	
Appendix	462
Index	510

1

Development of the cardiopulmonary system

P. B. KOFF

Objectives

At the completion of reviewing this chapter the reader will be able to:

1. Describe the placenta and discuss its role in fetal blood flow and gas exchange.
 2. Compare the blood gas values of umbilical arteries and the umbilical vein.
 3. Differentiate between the stages of lung development based on gestational age and structures present.
 4. Describe surfactant, including source, appearance in developmental stage, composition, and significance to respiration.
 5. Compare the developing heart structures with those in extrauterine life.
 6. Diagram fetal blood flow and differentiate between areas of high and low oxygen levels.
 7. Relate oxygen levels to the developing brain.
 8. Explain why only approximately 10% of the fetal blood flow perfuses the lungs.
 9. Describe the anatomy and physiology of the foramen ovale and the ductus arteriosus.
 10. Discuss maternal influences on the developing cardiopulmonary system.
-

This chapter discusses fetal cardiopulmonary development, with emphasis on the relationship between gestational age, lung maturity, and cardiac development. At birth the neonate is required to make a rapid transition from placental gas exchange to respiration that is dependent on the lung. The level of lung development greatly influ-

ences the success of this transition, and allows prediction of the possible need for supportive care.

Study of early embryonic development also is enlightening as regards the development of the cardiac system. To understand congenital heart defects, the intricate development of the heart during the first few weeks of gestation must be understood. The de-

developmental curve shown in Figure 1-1 provides an overview of the stages of development of the embryo and fetus.¹ Teratogenesis, or the development of abnormal structures, is discussed briefly as it relates to the cardiopulmonary system.

PLACENTAL GAS EXCHANGE

In the adult, the pulmonary system carries out the gas exchange function: oxygen is absorbed and carbon dioxide is eliminated via a sophisticated, expansive alveolar capillary membrane. This is in contrast to the fetal state, in which the primary center for gas exchange is the placenta. Blood can easily flow through the placenta, making it a low-resistance circulatory system. The developing lungs are high in resistance and thus do not easily allow blood to flow through them. It is estimated that 10% of the fetal blood, an amount important primarily for nourishing developing lung tissue,² circulates through the lungs.

The circulatory systems of the mother and fetus

are completely separate with the placenta serving as the only point at which the two circulations communicate. The placenta is approximately 6 to 8 inches in diameter and 1 inch thick, and weighs approximately 1 pound.³ It provides for the exchange of nutrients and waste products, including oxygen delivery and carbon dioxide elimination.

The umbilical cord enters the placenta from the fetus. The cord is composed of two fetal arteries that carry deoxygenated blood to the placenta from the fetus, one large fetal vein that carries oxygenated blood to the fetus, and Wharton's jelly, which is a gelatinous tissue surrounding the vessels.³

Blood from the maternal system enters the placenta via uterine spiral arteries. It spreads out into intervillous spaces, which are irregular spaces surrounding fingerlike tissue projections called *chorionic villi*. The capillaries from the fetus are inside the villi (Fig 1-2).

As the maternal blood makes the passage through these structures above and eventually communicates

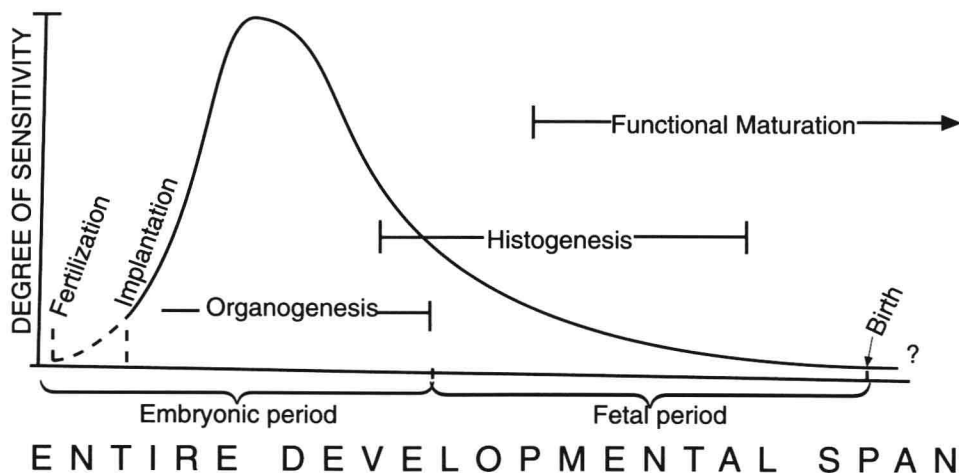


FIG 1-1.

Curve approximating the susceptibility of the human embryo to teratogenesis from fertilization throughout intrauterine development. The highest sensitivity, at least to structural deviation, occurs during the period of organogenesis, from about days 18 to 20 until about days 55 to 60, although the absolute peak of sensitivity may be reached before day 30 postconception. As organogenesis is completed susceptibility to anatomical de-

fects diminishes greatly, but probably minor structural deviation is possible until histogenesis is completed late in the fetal period. Deviations during the fetal period are more likely to involve growth or functional aspects because these are the predominant developmental features at this time. (Redrawn from Wilson JG: *Environmental sciences: Environment and birth defects*, New York, Academic Press, 1973.)

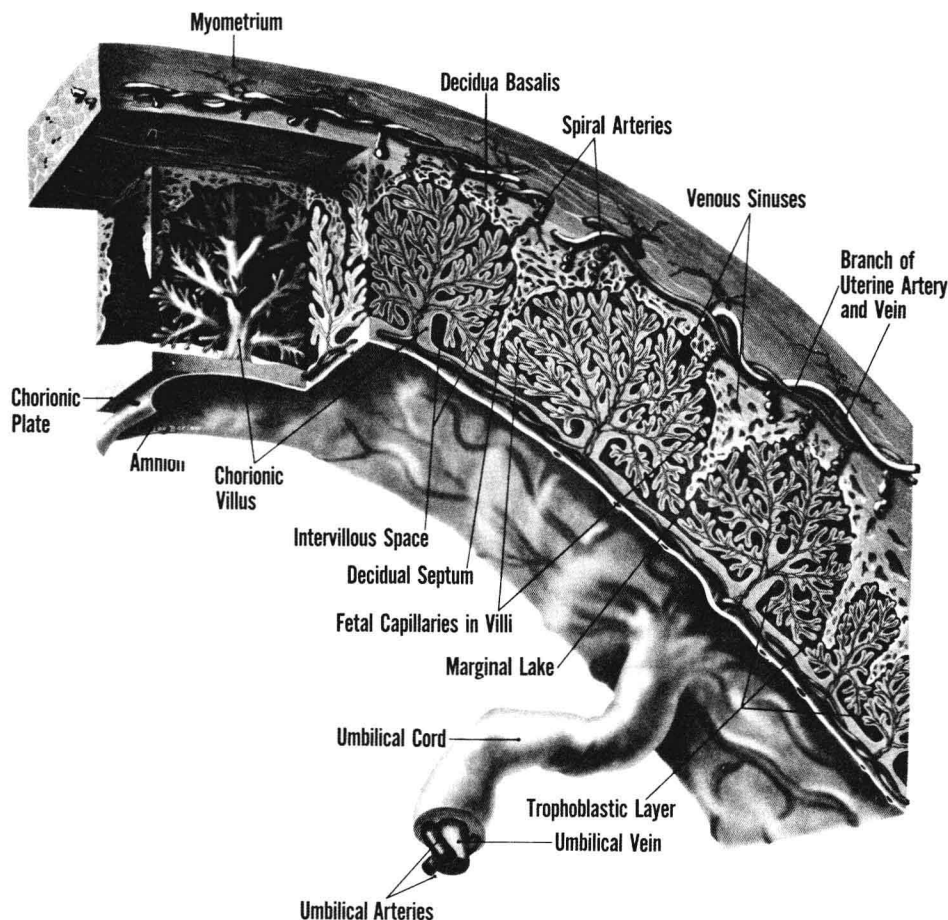


FIG 1-2.

Placental circulation. See text for description of blood flow pathways. (From Clinical education aids, vol 1, Columbus, Ohio, Ross Laboratories, 1986. Used by permission.)

with the fetal circulation at the level of the chorionic villi, gas exchange occurs. In the term fetus the umbilical vein and umbilical arteries will have the blood gas values shown in Table 1-1.

Given that the mother most likely began with an arterial P_{O_2} of 85 to 100 mm Hg, the oxygenated umbilical vein value seems low. This large partial pressure difference between the two circulations is a result of a number of factors, summarized in Table 1-2.⁴ Diffusing capacity is affected by the surface area and thickness, both of which vary within the placenta. Maternal blood flow is not evenly distrib-

uted and may even occur in intermittent spurts.⁵

Even though there is a significant reduction in arterial P_{O_2} values between the uterine arteries and umbilical veins, sufficient oxygenation of the fetus does occur. The large maternal-fetal P_{O_2} gradient promotes the transfer of oxygen; the higher hemoglobin concentration in fetal blood facilitates increased oxygen-carrying ability; and the fetal hemoglobin (HbF) provides a greater affinity for the oxygen to bind the hemoglobin. Fetal hemoglobin has a higher percentage of saturation for the same P_{O_2} than adult hemoglobin and facilitates the transfer of oxy-

TABLE 1-1.

Fetal Blood Gas Values*

	Average age 38 weeks		Average age 25 weeks	
	Umbilical Artery	Umbilical Vein	Umbilical Artery	Umbilical Vein
Po ₂	26	32	28	43
Pco ₂	43	37	35	35
pH	7.36	7.39	7.37	7.41

*Adapted from Nicolaides KH, Economides DL, Soothill PW: *Am J Obstet Gynecol* 161:996, 1989.

TABLE 1-2.

Factors Affecting Gas Transfer Across Placenta*

- Diffusion limitation
- Shunts present in maternal and fetal circulation
- Oxygen consumption within placenta
- Uneven distribution of maternal blood flow
- Uneven distribution of diffusing capacity to blood flow

*From Longo LD, Power GG: *J Appl Physiol* 26:48, 1969. Used by permission.

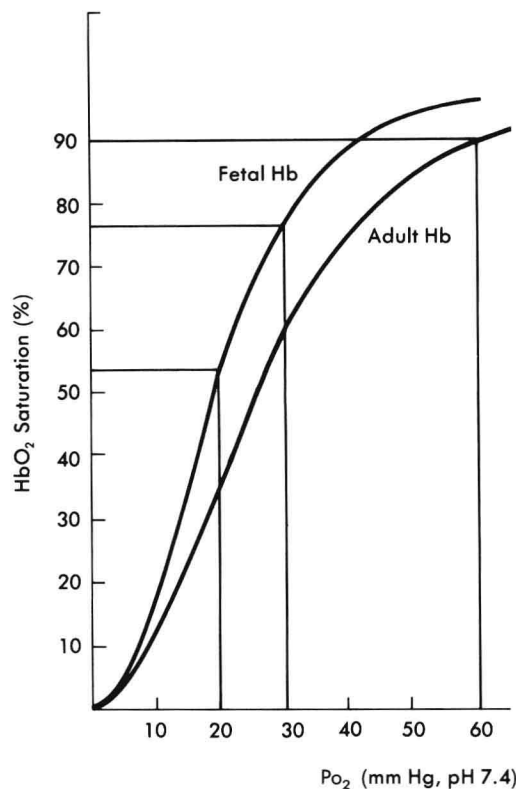
gen from maternal to fetal circulation, which is represented by a left shift of the fetal oxyhemoglobin dissociation curve (Fig 1-3).

PHASES OF LUNG DEVELOPMENT

While the placenta carries out gas exchange for the fetus, the pulmonary system is developing. Conceptually, this development is thought to occur in approximately four stages: (1) embryonic, which covers primitive development; (2) pseudoglandular, in which the conducting airway system is developed; (3) canalicular, in which the respiratory portion begins to develop and vascularization occurs; and (4) terminal sac, in which well-defined gas exchange areas develop.⁶ There are various opinions as to the naming of these stages and the developmental age at which they occur.⁷

Embryonic stage: primitive development (first 5 weeks of gestation)

The earliest formation of the lung begins at approximately 26 days gestation. An outpouching of the foregut, the lung bud, develops at this time from

**FIG 1-3.**

Fetal hemoglobin produces left shift of oxyhemoglobin curve.

the endoderm, the innermost layer of the three primary germ layers. The lung bud divides, continues to grow, and becomes enveloped within the mesoderm (middle layer). As it develops into the right and left mainstem bronchi (Fig 1-4), the lung bud carries mesoderm with it that adapts to the shape of the bronchial tree and differentiates into muscle, connective tissue, cartilage, and supportive structures.

During this first stage of pulmonary development the primary muscle of ventilation, the diaphragm, is also developing. Diaphragm development is complete by approximately the seventh week of gestation.⁸

Primitive pulmonary arteries and veins also begin appearing during the embryonic stage at about 32 to 37 days of gestation.⁶

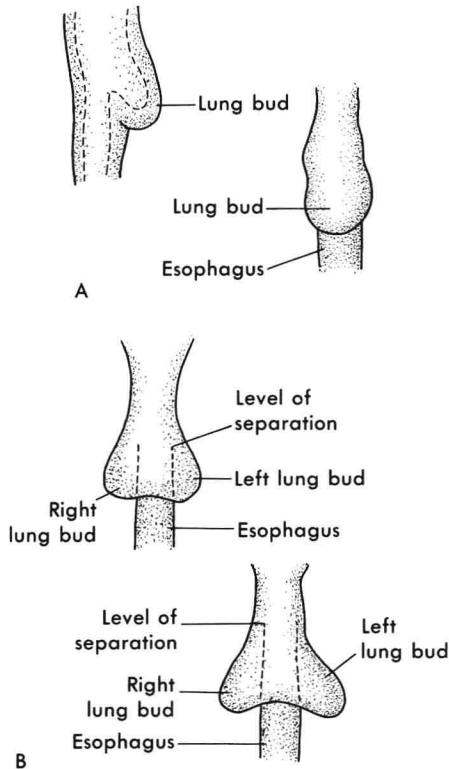


FIG 1-4. Earliest formation of lung. **A**, outpouching of foregut. **B**, expansion of lung bud into right and left mainstem bronchi. (Adapted from Hodson WA: Development of the lung, New York, 1977, Marcel Dekker.)

Pseudoglandular stage (6–16 weeks gestation)

Asymmetric dichotomous branching of the bronchial tree occurs at this stage and gradually progresses from about 4 to 25 generations of branches at 16 weeks of gestation.⁶ The lung at this point resembles a gland and gives the stage its name. By 16 weeks, the prenatal formation of new bronchi is nearly complete.

Mucous glands also begin to appear during this stage. As early as the tenth week submucosal glands are present in the trachea; by 16 weeks gestation they are noted in the bronchi.⁶ Goblet cells are also present by 10 weeks.⁹

Smooth muscle also appears during this phase. Sometime between the sixth and eighth week of ges-

tation it can be identified in the trachea, primary bronchi, and lobar bronchi.

Ciliated cells appear as early as 7 weeks gestation in the upper airway.

Fetal breathing movements also begin during this stage, probably as early as 12 weeks of gestation¹⁰; however, mature breathing movements with balanced inspiration and expiration are not present until after 30 weeks.¹¹ The fetal breathing movements may serve as conditioning exercises for the respiratory muscles and appear to be necessary for lung development.

Canalicular stage (16–26 weeks gestation)

The first identifiable components of the respiratory units appear at about the 16th week of gestation, after the development of the conducting airways is complete. The terminal bronchioles, respiratory bronchioles, and clusters of closely branched buds that eventually become alveolar sacs develop as the capillary network also begins to appear. Capillary loops begin to surround the respiratory portions, but adequate gas exchange in extrauterine life is unlikely because the interalveolar walls are still thick. The alveolar capillary membrane begins to develop, but expansive surface area is not yet available.

During the canalicular phase other key elements in pulmonary development appear. The alveolar epithelium begins differentiating into type I and type II cells. Type I cells, essential to the development of the alveolar capillary membrane, line a relatively large portion of the alveolar surface (Fig 1–5) and are characterized by small perinuclear bodies with cytoplasmic extensions. Type II cells are described as larger and rounder than type I cells and contain lamellar bodies, which are surfactant storage sites. Type II cells are involved in the complicated process of surfactant synthesis.

Surfactant is essential in reducing the surface tension present in the liquid-air interface within the lungs. According to the LaPlace equation $P = \frac{2T}{r}$, surface tension at the alveolar wall produces a pressure that tends to collapse the alveoli, where P represents the distending pressure within the alveolus, T is the surface tension, and r is the radius of the alveolus. The significance of this is illustrated with two inflated spheres that are connected. The smaller