

## OXYGEN AS A SUPPORTIVE THERAPY IN FETAL ANOXIA

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THE most secure insurance against fetal anoxia is adequate, spontaneous respiration [of the newborn] immediately after birth. The absence of normal ventilation and crying has led to urgent and painstaking efforts to substitute artificial respiration with oxygen by a large variety of methods. It cannot be overemphasized that intermittent pulmonary inflation with oxygen at safe pressures is the most effective therapeutic means available in overcoming fetal anoxia. It is the purpose of the present communication, however, to examine the role of oxygen in the physiological recovery of the fetus and the therapy of fetal anoxia from the dual aspect of some recent newer knowledge and a critical appraisal of the results of current research.

In spite of a steady improvement in neonatal mortality during the past twenty years, there has been almost no decrease in the deaths during the first twenty-four hours after birth.<sup>1</sup> Is this because we do not understand the fundamental causes of death during this period or because we do not apply what is already known? For a variety of reasons, oxygen lack plays the major role in over half the deaths, and the basic treatment for oxygen want is well known. The widespread application of such treatment is sadly lacking. The junior house officer, who is usually left to handle the resuscitative problem, has received instruction only from his immediate superior in the principles of resuscitation, or not at all. It should be the joint responsibility of departments of obstetrics, pediatrics and anesthesiology to offer instruction to the internes and residents in all three departments, irrespective of which one takes the initiative for such teaching. Too often emphasis is placed on a certain type of equipment or on certain stimulating drugs while the basic principles of prompt action, the maintenance of a free airway and getting oxygen into the newborn baby are overlooked. Our own specialty has been negligent in exhibiting an interest in this fascinating field and too often obstetrical anesthesia is left to a most inexperienced group.

The anoxia problem has been outlined and discussed most clearly and unemotionally by Smith<sup>2</sup> in Volume III of *Advances in Pediatrics*. The only practical method of recognizing fetal anoxia before delivery is through the occurrence of a bradycardia. Other commonly-used diagnostic signs, such as tachycardia and increase in fetal movements, have not been substantiated experimentally.<sup>3</sup> Some method of recording the fetal heart rate continuously in human subjects would be a material aid in recognizing fetal anoxia, but so far neither electrocardiographic nor electronic auscultatory methods have been successful. Waters and Harris,<sup>4</sup> Lund<sup>5</sup> and Barcroft<sup>6</sup> have all demonstrated clearly that the administration of oxygen to the mother between contractions will correct to a large degree the bradycardia of the fetus during labor pains. This simple treatment is not present-day practice in large, busy obstetrical clinics, but should be instituted especially in situations in which neonatal mortality is especially high, i.e., prematurity, multiple births and toxemias of pregnancy.

At the time of delivery, an infant who is apneic, breathes feebly or with occasional deep gasps needs oxygen. The presence of cyanosis, pallor, flaccidity and bradycardia merely corroborates this need. The use of gravity and gentle pharyngeal suction should precede the administration of oxygen. Actual pulmonary inflation can be obtained in countless ways, from the ever-available mouth-to-mouth insufflation to the latest type of mechanical device. Endotracheal suction and inflation are rarely necessary but should be performed quickly and atraumatically by someone well accustomed to the technique. Obviously, the training program mentioned previously should include practical experience with the technique of these procedures.

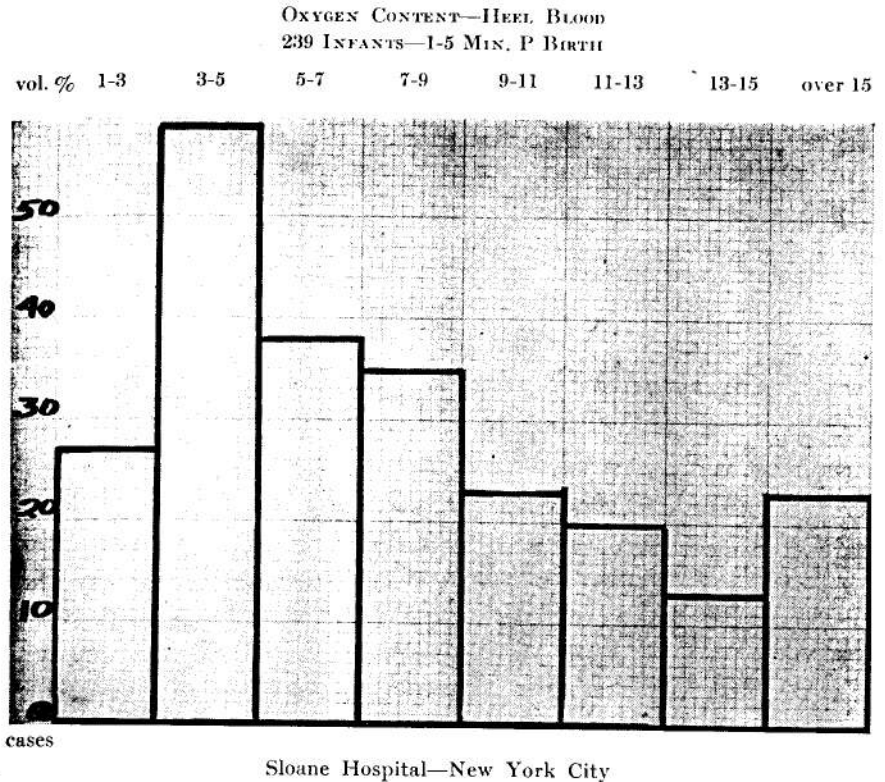
The damage caused by a period of anoxia at birth has been the subject of many papers. True results are elusive because of the nature of the complications. Psychological maldevelopment is indeed hard to evaluate unless the series is large and the controls are ample. The infants who are adequately oxygenated at birth should be followed as assiduously as those who are anoxic. Environmental and hereditary considerations further complicate interpretation. Objective neurological signs are more accurately interpreted but again a large series of cases is needed. Prolonged studies such as those begun in several cities, notably Washington and New York, will be enlightening and may produce other unexpected signs of damage. It is surprising that no metabolic complica-

tions have been noted as a result of fetal anoxia. Negative evidence will be as valuable as positive, and reports of the development of a brilliant intellect in spite of anoxia at birth, as in a case cited by Barcroft,<sup>6</sup> will need elucidation.

The remarkable resistance of the fetus to anoxia has been demonstrated in many ways in several species, and the explanation of this fact is nearing completion. In the rabbit, dog and guinea pig the survival time of fetuses of mothers breathing 100 per cent nitrogen was related to the stage of development rather than to the species.<sup>7</sup> Removal of the medulla is less quickly fatal to newborn small laboratory animals than to the adult.<sup>8</sup> The pattern of oxygen consumption in various parts of the newborn animal's brain is the opposite of that of the adult, the cortical metabolic rate reversing itself from a low to a high figure.<sup>9</sup> It is probable that some mechanism for anaerobic oxidation exists, for the survival time of fetal animals is not improved under anoxic conditions if the anaerobic use of sugar is withheld. The existence of such a mechanism would help to explain why even more damage does not occur after fetal anoxia, but in no way does its presence excuse negligence in administering oxygen to a newborn infant when the need is clinically indicated. No data at all are available to explain the greater tolerance of the *human* fetus to prenatal anoxia.

The numerous difficulties of embarking upon a study in human beings are obvious. The obstetrical and anesthetic problems are inversely proportionate to the skill of the operator. Detailed records of the prenatal problems and an accurately kept chart of the events occurring during delivery call for additional personnel. Simultaneous studies of the maternal and fetal blood are desirable. The meticulous technique needed for the study of samples of intact cord blood, well described by Barcroft,<sup>6</sup> are not applicable to the usual delivery. A deep heel puncture in the infant supplies a blood sample so closely similar to arterial blood samples that this method has proven practicable. The Roughton-Scholander<sup>10</sup> analysis of the blood sample drawn under oil is relatively reliable. Possibly correction for the type of anesthetic gas in use should be made.

The accompanying chart reveals the distribution of oxygen content in volumes per cent, as determined from an initial sampling of blood by the method just described, made within five minutes of delivery of the head in 239 consecutive, unselected vaginal deliveries. If ten volumes per cent is the true average figure for the human newborn infant, the causes



for our lower mean value 8.0 per cent must be uncovered and corrected. Follow-up studies on these infants in the Pediatric Clinic is planned for a period of five years.

#### SUMMARY

The problem of fetal anoxia has been only partially solved in clinical practice. The explanations for the persistence of a relatively higher anoxic mortality in the first day of life are twofold. First, there is the failure to employ adequate methods of artificial ventilation with oxygen promptly after birth. Second, there is still a great deal to learn about the physiology of respiration during and after the birth process. Some information based upon the oxygen content of newborn blood has been added in this presentation. The importance of the low values observed is not entirely clear at the present time. It is possible that they suggest the presence of effective anaerobic metabolism.

## REFERENCES

1. Dublin, L. and Spiegelman, M. *The Record, Am. Inst. Actuaries*, 1941, 30:31.
2. Smith, C. A. Effect of birth processes and obstetric procedures upon the newborn infant, *Advances in Pediatrics*, 1948, 3:1.
3. Lund, C. J. Recognition and treatment of fetal heart arrhythmias due to anoxia, *Am. J. Obst. & Gynec.*, 1940, 40:946.
4. Waters, R. M. and Harris, J. W. Carbon dioxide and oxygen problems in obstetric anesthesia, *Anesth. & Analg.*, 1931, 10:59.
5. Lund, C. J. Prevention of asphyxia neonatorum, *Am. J. Obst. & Gynec.*, 1941, 41:934.
6. Barcroft, J., Kramer, K. and Milliken, G. A. Oxygen in carotid blood at birth, *J. Physiol.*, 1939, 94:571.
7. Glass, H. G., Snyder, F. F. and Webster, F. The rate of decline in resistance to anoxia of rabbits, dogs, and guinea pigs from the onset of viability to adult life, *Am. J. Physiol.*, 1944, 140:609.
9. Feldmann, W. M. *Principles of antenatal and post-natal child physiology, pure and applied*. London, Longmans, Green & Co., 1920.
9. Himwich, H. E., Fazekas, J. F. and Alexander, F. A. D. Comparative studies of metabolism of brain of infant and adult dogs, *Proc. Soc. Exper. Biol. & Med.*, 1941, 46:553.
10. Scholander, P. F. and Roughton, F. J. W. Micro gasometric estimation of the blood gases, *J. Biol. Chem.*, 1943, 148:551; 573.