APNEA NEONATORUM

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TERMINOLOGY

ALTHOUGH questions of nomenclature are usually of academic interest only, occasionally a term is encountered which is so inept that it leads to incorrect thinking and an erroneous concept of the condition designated. Deeply entrenched as "asphyxia neonatorum" is in obstetric terminology, there is reason to believe that it belongs in this unhappy category.

Few words have undergone such radical changes in meaning as asphyxia. The term comes from the Greek ἀσφυκτία (ἀ, not + σφύκεων, to throb) and means literally absence of pulsation. It was used in this sense by Galen to describe the state of an artery distal to a tourniquet and, as late as the eighteenth century, seems to have carried no other meaning. Thus, in 1706 Phillips described asphyxia as "a cessation of the pulse throughout the whole body; which is the highest degree of Swooning and next to Death"; while Quincy in his New Medical Dictionary, published in 1730, defined the term as a "deficiency or privation of the pulse in some cases where it stops for a time." By 1778 the word was being used in a broader sense to convey the idea of apparent death, as from drowning, and in that year an essay by T. Brandt carried the title, "The Cure of Asphyxia, or Apparent Death by Drowning." In a later edition of Quincy's dictionary published in 1794, the earlier definition is entirely deleted and in its place we read that asphyxia "happens from a long failure of vital and animal power, as from drowning." With the beginning of the nineteenth century the import of the word centered more and more on the suffocation which results from drowning, and at the same time its connotation became extended to include suffocation from other causes, such as strangulation and noxious gases. This meaning, of course, has persisted to the present; but when it is recalled that the pulse in asphyxiated animals continues to beat long after all signs of respiratory action have ceased, it becomes apparent that our word asphyxia represents a most curious infelicity of etymology.

From a practical viewpoint a much more important objection to the term is the fact that obstetricians have taken further liberties with it and customarily refer to any baby who does not breathe at birth as "asphyxiated," whether the cause be lack of oxygen, cerebral hemorrhage, congenital defect or whatnot. This use of one etiologic condition to denote the whole syndrome of apnea at birth, from whatever cause, is not only illogical but slights some of the most important etiologic factors. It ignores entirely the gravest cause of apnea, birth trauma, as well as the commonest cause of temporary apnea in modern practice, namely, narcosis. Moreover, by focusing attention solely on
the acute anoxic episode at birth, it tends to circumscribe unduly the scope of the problem; in this connection it should be remembered that careful prenatal care and judicious conduct of labor will do more to save babies from apnea at birth than all the resuscitating measures in the world.

In view of these facts, the simple term *apnea neonatorum*, which is merely descriptive and carries no implication in regard to etiology, seems preferable to the older designation. The use of the word *asphyxia* could then be restricted to those cases in which apnea was actually due to lack of oxygen as in, for instance, prolapse of the umbilical cord and premature separation of the placenta.

**ETIOLOGY AND PREVENTION**

*Cerebral Hemorrhage.*—There is general agreement that the most common cause of fatal apnea at birth is cerebral hemorrhage. Statistics also concur in showing that such hemorrhage is most often consequent upon traumatic operative delivery. The procedures which are most likely to produce it are, in order, version and extraction, midforceps delivery and breech extraction (it being assumed that high forceps, which would otherwise head the list, is an abandoned operation). The prevention of grave apnea neonatorum, accordingly, consists very largely in the avoidance, when possible, of these operations.

Although clinical and necropsy evidence shows that trauma is the deciding factor in most of these hemorrhages, it is well known that all newborn infants show an especial tendency to bleed and it seems probable that this diathesis plays an auxiliary role, at least, in some of these cases. A rational explanation for this hemorrhagic tendency was advanced in 1917 by Brinkhous, Smith and Warner who showed that the plasma prothrombin level of babies at birth is exceedingly low, ranging from 14 to 39 per cent of the usual adult value. During the past two years Hellman and Shettles, of the Johns Hopkins Hospital, have been exploring the possibility of raising the low plasma prothrombin of the newborn infant by administering vitamin K antenatally to the mothers. They have shown, beyond peradventure, that the plasma prothrombin level of the baby can be raised severalfold in this way, even when the vitamin is given as late as four hours before delivery. The question at once arises, of course, as to the practical value of such a procedure. Will it diminish stillbirth and neonatal mortality by preventing a certain number of deaths from cerebral bleeding? It is realized, of course, that massive cerebral hemorrhage, resulting from severe birth trauma, cannot be prevented by this measure; but it must be remembered that the most common site of cerebral hemorrhage in the newborn is subtemporal, that is, within a small, confined space near the vital centers where a minute hemorrhage may be fatal. To date, Hellman and Shettles have administered vitamin K to more than 500 expectant mothers and are finding that this prophylactic procedure definitely reduces the incidence of all types of hemorrhage in the newborn. Details of the study will appear in an early issue of this Journal.
Narcosis.—As we have indicated, the commonest cause of temporary apnea neonatorum is anesthesia and analgesia. In my experience, nitrous oxide oxygen pushed (without ether) to the point of surgical anesthesia is a more frequent offender than realized, because of the resultant fetal anoxia. The time element here is most important, and pure nitrous oxide, administered for four to five breaths to produce analgesia, probably causes less anoxia than a mixture of 85:15 continued for five minutes. So far as operative obstetrics is concerned, it seems plain that when nitrous oxide oxygen is given in concentrations of 90:10 or stronger over periods which exceed five minutes, marked degrees of anoxia are produced in about one baby out of every three. The anoxia may not prove harmful, it is true, but in an occasional case it may be associated with profound and even fatal apnea. An adequate saturation of the fetal blood with oxygen can be guaranteed only if the mother receives fifteen parts of oxygen to every 100 parts of the gas mixture, and to satisfy this end, ether should be added to the gas mixture if the latter, in proportions of 85:15, does not suffice. After prolonged labors, in particular, babies seem to withstand anoxemia poorly, and if an operation is necessary in such cases, we prefer ether on an open mask to insure liberal oxygenation of the child’s blood. The same is true of breech extractions in which there is an inherent tendency to fetal anoxia due to the impingement of the child’s shoulders and head on the umbilical cord.

Since ether passes readily through the placenta, it naturally exerts an anesthetic effect upon the child, and it is this influence apparently, and not anoxia, which causes the occasional “etherized” baby to be slow in breathing. Provided there is no cerebral injury, however, we find that these babies react well to time and gentle stimulation. Certainly, in our opinion, their prognosis is immeasurably better than those in which the apnea is the result of profound anoxia.

We now come to one of the most controversial questions in modern obstetrics, namely: Does the apnea of the newborn which commonly follows the use of modern analgesic programs (barbiturates, paraldehyde, scopolamine, etc.) so jeopardize the baby as to condemn the employment of these drugs? Beyond question, these sedatives, given in amnesic doses, do inhibit the onset of respiration in 40 to 60 per cent of cases, the duration of the apnea varying from a few seconds to half a minute, as a rule. Expediency, moreover, very often dictates the use of outlet forceps in these cases, so that inhalation anesthesia to the surgical degree is superimposed on the drug action, with well-known synergistic effects. If modern analgesia is to be evaluated on the basis of sound obstetrics (dismissing for the time being humanitarian considerations), the following two questions must be answered: Are the babies permanently harmed by the temporary apnea? Do any advantages accrue to the baby as the result of such analgesia? Obviously, the first question can best be answered in terms of actual results, that is, by statistics. It is my impression, based on available reports as well as on my own material, that the ultimate outcome for the baby born under analgesia, intelligently administered, is just as good as for one born under no analgesia, provided the infant is mature. Space does
not permit marshaling the huge mass of factual data bearing on this question, but anyone familiar with recent statistical studies of the problem will find this conclusion inescapable. Respiration in the premature baby is at best a precarious business and should not be hampered, in our opinion, by depressant drugs. Turning now to possible beneficial effects conferred on the infant by analgesics, there is a growing conviction on the part of experienced observers that the necessity for difficult midforceps operations arises less frequently in patients who have been given sedation. This is ascribed to the fact that such women enjoy more rest throughout labor and are sometimes better able to rotate the head and bring it to the perineum than a patient exhausted by pain and loss of sleep; such women, moreover, are less likely to suffer from premature operative interference since they are not crying constantly with pain; in other words, the obstetrician is more disposed to give the patient additional time. Whatever the explanation may be, several clínics, including our own, report a diminished incidence of midforceps in this group.

Anoxia.—If oxygen determinations are done on the umbilical vein blood at birth, it will be found that most apneic babies (narcosis cases excluded) show very low oxygen levels. This anoxia may be the primary and determining cause of the apnea, as in prolapse of the umbilical cord, for instance; or it may be secondary to cerebral hemorrhage and a number of other conditions which interfere with the fetal circulation and thus prevent proper oxygenation of the blood in the placenta. Since, conversely, anoxia is a common cause of small cerebral hemorrhage, the situation is avowedly a complicated one and it is often difficult to determine positively the primary cause of the apnea in a given case. However this may be, from a practical viewpoint, the most important thing to bear in mind is that most apneic babies are anoxic; as we shall see presently, this is the dominant consideration in the treatment of the condition.

Prematurity and Congenital Malformations.—Although prematurity is the most common cause of neonatal death, it is seldom responsible for actual apnea at birth unless narcosis or cerebral hemorrhage is superimposed. Similarly, congenital malformations are a more common cause of early neonatal death than of apnea neonatorum.

TREATMENT

In the presence of anoxia, apnea is resistant to all types of treatment other than correction of the anoxia itself. In a recent study of experimental anoxia by Kreiselman and myself, even convulsive doses of alpha-lobeline, metrazol and ecoramine, whether injected intravenously or directly into the carotid artery, were found to have no effect whatsoever on anoxic apnea; on the other hand, a few insufflations with oxygen produced immediate breathing. In other words, there is only one way in which respiration can be initiated when suppressed by anoxia and that is by the administration of oxygen. It is our opinion, therefore, that insufflation with 100 per cent oxygen transcends all else in the treatment of apnea at birth. Attempts to stimulate respira-
tion by adding carbon dioxide to the oxygen are not only futile (since all forms of stimulation are futile in the presence of anoxia), but may even be dangerous; the same applies to the drugs mentioned above.

The main desiderata in the treatment of apnea at birth would seem to be four in number: (1) Warmth. These babies are in a state of vascular collapse and should be treated as is any patient in shock. (2) Posture. The head should be declined about 30 degrees to favor gravity drainage of fluids in the trachea, but should not be placed so directly downward as to augment a pre-existing cerebral hemorrhage. (3) Aspiration of mucus. Clear air passages are essential, and mucus must be removed by means of a catheter, employing either mouth suction or an electric aspirator. (4) Delivery of 100 per cent oxygen to the pulmonary alveoli, by adequate apparatus such as the Kreiselman resuscitator. Mouth-to-mouth insufflation, provided it is done gently (never over 20 cm. of water pressure), is usually a satisfactory substitute.