carried out to determine the effect on the composition of the inspired gases of the long connecting tube and nosepiece tubes usually employed in dental anesthesia. It was thought that the additional dead space thus produced would considerably lower the oxygen content of the inspired gas when rebreathing was taking place. During the investigations, however, other important facts emerged.

"These were: (1) That resistance of the narrow nosepiece tubes had important effects on respiration by impeding respiratory movements. (2) That owing to hyperventilation, due to the increased resistance, acapnia tends to develop. (3) That the percentage of oxygen inhaled during the average gas-oxygen anaesthesia in unpremedicated dental patients is so low that this form of anaesthesia used for more than two or three minutes is physiologically unsound; the alkalaemic tendency produced by (2) further reducing oxygen availability. In view of the fact that prolonged gas-oxygen anaesthesia is being used increasingly by dentists this last point is of great importance. . . .

"We would suggest that: (1) Nasal gas-oxygen should never be used for procedures lasting more than five minutes, without the addition of ether or vinesthene and a higher oxygen content or alternatively after premedication with morphia or a barbiturate. (2) Carbon dioxide should be added to gas-oxygen mixtures or failing this efficient partial rebreathing should be used for all patients except those with a low alkali reserve. This will combat the acapnia and help to diminsh any harmful effects of the low oxygen tensions. (3) All tubes leading from the gas machine to the nosepiece should be of wide bore to eliminate the resistance to the flow of gases. Theoretically no tube or orifice in the nosepiece should be smaller in calibre than the trachea. Further these tubes should be arranged as a circuit with the inspiratory gases passing up one tube and expiratory gases down the other in order to eliminate the artificial increase of dead space which would be considerably more marked with wide bore tubes than the present narrow ones."

J. C. M. C.

WINDLE, W. F., AND BECKER, R. F.: *Role of Carbon Dioxide in Resuscitation at Birth After Asphyxia and After Nembutal Anesthesia: An Experimental Study in the Cat and Guinea Pig.* Am. J. Obst. & Gynec. **42**: 852–858 (Nov.) 1941.

"The observations to be reported are part of a broader study of the physiology of respiration in the fetus and newborn. No attempt will be made to correlate the dosages of nembutal we have used with those commonly or uncommonly employed in the human being. . . . Last year Dr. Yandell Henderson . . . suggested anarehapnea, containing the Greek roots 'archon' (governor) and 'pneuma' (breath), to indicate a state of uncontrolled respiration encountered after excessive use of certain narcotic drugs, notably the barbiturates. The government of respiration by carbon dioxide appears to be abolished in deep barbiturate narcosis. The responsiveness of the respiratory center is lowered, the volume of breathing diminishes, hypercapnia manifesting itself as carbon dioxide displaces oxygen in lung alveoli, and anoxia ensues. The use of carbon dioxide diluted in oxygen for resuscitation in asphyxia of the newborn has been debated for some years. . . . We have studied intrauterine respiratory activities in several hundred fetuses of several species during the last few years. Respiration at birth under normal and asphyxial conditions has engaged our attention in at least 50 litters of kittens and guinea pigs. The effects on the fetus of nembutal admin-

istered to the mother have been observed in 25 pregnant cats with 88 fetuses, for the most part within five days of birth, and about a dozen guinea pigs at full term. . . .

"When a fetus is born normally or is delivered without anesthesia, the stimulus of the anoxia associated with interruption of placental exchange serves as an exaggeration of the stimulus of the intrauterine anoxemia associated with uterine relaxation; the fetal heart slows, the fetus squirms, and breathing starts at once. The blood becomes well oxygenated again due to pulmonary breathing, but the newborn does not lapse into apnea as it did in utero. It seems that the changed environment with markedly increased afferent stimulation lowers the threshold of the respiratory center. When a cesarean section is performed in a decerebrated cat without general anesthesia and the uterine vessels are clamped to bring about anoxia in the fetus, the fetal heart beat slows to half or less than half its normal rate, motor activities are exaggerated and deep respiratory movements are initiated. These cause aspiration of the amniotic contents. Such an asphyxia can be prolonged in the cat for fifteen or twenty minutes and in the guinea pig for five to seven minutes. Respiratory movements become deep gasps at ten to forty-second intervals. It has been our experience that the fetus must be delivered before these cease, to ensure spontaneous respiration. When this is done, breathing is established by the gasps, and normal respiratory movements are later superimposed upon the gasp rhythm as oxygenation of the blood takes place. Although no special measures were usually necessary, oxygen-air mixtures appear to speed resuscitation. When small quantities of carbon dioxide were added, the depth and sometimes the rate of the respirations were observed to increase. Recovery from simple asphyxia was usually complete within an hour or two in the cat, but symptoms of central nervous system damage, persisting for several days (even weeks in one case), were observed in a few animals, especially in the guinea pigs. A very different picture of respiration at birth presented itself when nembutal was given to the mothers. . . . An anesthetic dose for adult animals was considered to be 28 mg. per kilo of body weight. . . .

"After a full anesthetic dose of nembutal had been administered to the pregnant cats, the fetuses were profoundly narcotized. Some could not be induced to breathe. Others gasped feebly at rates of less than four times per minute. Most of them died within two hours. They were completely flaccid and appeared to draw little air into their lungs. When approximately 20 mg. of the drug per kilo had been used, the fetuses were completely narcotized. They did not often begin to breathe for several minutes after delivery. . . . When one-fourth to one-half of an adult anesthetic dose of nembutal had been administered, the fetuses nearly always began to breathe within one or two minutes. . . . The effects of nembutal upon the newborn cat and guinea pig fetuses lasted from several hours to two days, depending on the depth of the narcosis. . . .

"As soon as possible after delivery and when it had become evident that the narcotized newborn animals had established regular, though often very slow rhythms of breathing, they were placed in warm chambers of 70 liters capacity. In one chamber, oxygen was added to the air; in another a mixture of oxygen (90 per cent) and carbon dioxide (10 per cent) was added. In a few experiments the carbon dioxide content was raised further by adding a small quantity of the undiluted gas to the latter mixture. The mixtures of

carbon dioxide in oxygen had no visible beneficial effect on the respiration of any narcotized cat fetus for the first hour or two after birth. A few of the guinea pigs, not so heavily narcotized and already executing more normal rhythms of breathing, did respond to the carbon dioxide. Heavily narcotized cat fetuses died when left in the carbon dioxide mixtures for several hours, although several recovered in atmospheres containing a high percentage of oxygen alone. Cyanosis was relieved and regularity of breathing improved as recovery from the narcosis gradually took place. The newborn animals delivered after doses of about one-half anesthetic value were especially interesting. Litter mates were delivered simultaneously and placed in the warm chambers, one in oxygen-air mixture and the other in oxygen-carbon dioxide-air mixture. No difference in the rate or depth of their respirations could be observed for more than two hours. It was perfectly clear that their breathing was unaffected by the powerful respiratory stimulant, carbon dioxide. As soon as the narcosis began to subside and the animals began to move about, we could observe an increase in depth of respirations of the specimens in the carbon dioxide mixture. Later this was occasionally followed by an increase in the rate as well. The respirations of the animals in the oxygen-air mixtures increased in rate but decreased in depth at the time the narcosis subsided. Ultimately they became equal to those of the non-narcotized control animals."

J. C. M. C.

TYNES, A. L.; NICHOL, W. W., AND WIGGIN, S. C.: *Anesthesia for Military Needs.* War Med. 1: 789–798 (Nov.) 1941.

"In war one is deprived of the conditions and equipment available for managing anesthesia during peacetime. One must be prepared to take care of a large number of casualties in a relatively short time. . . . Every effort . . . will be made both in the battalion aid station and in the collecting station to provide for the comfort of the wounded and the prevention of shock. Both morphine and rapidly acting barbiturates will be available and will be given as needed. . . . For short emergency operations in the surgical trailer attached to the casualty-clearing station, the anesthetic of choice will be pentothal sodium given intravenously in fractional doses. Intravenous anesthesia has the decided advantage of rapid induction and recovery, so that there need be no delay in the further evacuation to the rear. For the more severely shocked patients who already give evidence of tissue anoxia, oxygen will also be available and can be administered by a small portable apparatus. When the mobile surgical unit is set up farther to the rear as a part of the surgical hospital, more time can be given to each case. The anesthetist will have an opportunity to devote more attention to preoperative medication. For several years, the method in general use in most of the Army hospitals has been to give 3 grains (0.19 Gm.) of sodium amytal or 1.5 grains (0.09 Gm.) of pentobarbital sodium two hours before the operation followed by morphine and atropine or scopolamine one-half hour before operation. In this connection it has been shown that shock developed much more slowly in anesthetized dogs subjected to trauma when the animals had previously been given adequate doses of sodium amytal or pentobarbital sodium. . . . Thus, if an early operation is anticipated the preoperative use of one of these barbiturates seems highly desirable not only in the surgical hospital but farther forward, in the collecting station or the battalion aid station. . . .

"Since in this unit there is no longer