"rush" of opioid administered iv, from the enforced immobility on a narrow operating table, from oral discomfort due to mouth breathing of dry gases, and from the encouragements to stay awake, at first partially masked the morphine effect. After one to two hours, accommodation plus morphine sedation overcomes these arousal mechanisms. The latter view is compatible with placebo response reported by Lambertsen, Wendell and Longenhagen.[10] In that study, isohypercapnic ventilation tended to increase several liters per minute, peaking at about half an hour and tending to return toward control values thereafter.

References

1. Constantine JW, Konick D, Williams M: Benzquinamide: A centrally mediated antagonist of histamine, and acetylcholine induced bronchoconstriction. Arch Int Pharmacodyn 154:148–154, 1965
2. Burstein CL: Treatment of halothane hypotension with benzquinamide. Anesth Analg (Cleve) 44:120–124, 1965
3. Scriabine A, Weissman A, Finger KF, et al: "Benzquinamide": A new anti-anxiety drug. JAMA 184:276–279, 1963
4. Burstein CL: Respiratory effects of benzquinamide during anesthesia. Anesth Analg (Cleve) 42:435–437, 1963
5. Lambertsen CJ, Wendell H: An alveolar P_{CO_2} control system: Its use to magnify respiratory depression by meperidine. J Appl Physiol 15:43–48, 1960
6. Collier CR: Determination of mixed venous CO_2 tensions by rebreathing. J Appl Physiol 9:25, 1956
7. Smith TC: Rapid continuous measurement of mixed expired carbon dioxide concentration. ANESTHESIOLOGY 29:1037–1039, 1968
8. Steen SN, Yates M: The effects of benzquinamide and prochlorperazine, separately and combined, on the human respiratory center. ANESTHESIOLOGY 36:519–520, 1972
9. Hoffman JC, Smith TC: The respiratory effects of meperidine and propiomazine in man. ANESTHESIOLOGY 32:325–331, 1970
10. Lambertsen CJ, Wendell H, Longenhagen JB: The separate and combined respiratory effects of chlorpromazine and meperidine in normal men controlled at 46 mm Hg alveolar P_{CO_2}. J Pharmacol Exp Ther 131: 381–393, 1961

Obstetrics

ABNORMAL PREGNANCY AND SURFACTANT MATURATION In a random sample of 134 pregnancies, amniotic fluid lecithin/sphingomyelin (L/S) ratios of 2.0 or more were not associated with the development of neonatal respiratory distress syndrome (RDS). With lower ratios, RDS occurred regardless of gestational age and birth weight. The L/S ratio reached 2.0 at approximately 35 weeks' gestation in normal pregnancies. A study of 147 pregnancies with maternal, fetal, or placental disease states revealed alterations in rate of maturation of fetal lungs away from the 35-week norm. Toxemia, hypertensive renal disease, severe diabetes, and retroplacental bleeding cause much earlier achievement of L/S ratios of 2.0 or above, while mild diabetes, chronic nonhypertensive glomerulonephritis and hydrops fetalis delayed pulmonary surfactant maturation beyond 35 weeks' gestation. The authors conclude that chronic intrauterine stress generally causes early maturation of pulmonary surfactant and decreases the risk of neonatal RDS despite the higher incidence of low birth weight and prematurity. *(Gluck, L., and Kulovich, M.: Lecithin/Sphingomyelin Ratios in Amniotic Fluid in Normal and Abnormal Pregnancy. Am J Obstet Gynecol 115: 539, 1973.)*