delivered by every anesthesia system. This is what we have endeavored to accomplish with the vaporizer described in our article.

JACK CHALON, M.D.
Department of Anesthesiology
Albert Einstein College of Medicine
Yeshiva University
Bronx, New York 10461

REFERENCES

1. Chalon J, Loew DAY, Malebranche J: Effects of dry anesthetic gases on tracheobronchial ciliated epithelium. ANESTHESIOLOGY 37:338–343, 1972
2. Weeks WB, Broman KE: A method of quantitating humidity in the anesthesia circuit by temperature control. Anesth Analg (Cleve) 49:292–296, 1970

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Experience Using the F. & P. Humidifier for Pediatric Patients

To the Editor:—The description by Spence and Melville (ANESTHESIOLOGY 36:89–93, 1972) of the F. & P. humidifier prompts us to describe its use in pediatric intensive care. This humidifier has been used for all infants needing mechanical ventilation in the pediatric intensive care unit at The Prince of Wales Hospital, Sydney, in the last 12 months. Most patients so treated have been newborn infants with respiratory distress. The gas flow in these circumstances was lower than those described by Spence and Melville. The humidifier has been used with the Starling Ideal Pump, the Harvard Dog Respirator, and the Drager Spiromat with an infant head. It has been necessary to place a length of tubing between the end of the heated delivery hose and the point where the tubing enters the humidicrib, to allow temperature to fall to dewpoint. The temperature of the gas is continuously monitored using a thermocouple at the end of the heated delivery tube and another just before the tubing enters the humidicrib. It has been necessary to adjust the temperature of the heater plate and humidifier chamber so that the temperature at entry to the humidicrib when condensation is just visible at this point is about 36 C. The thermometer at the end of the delivery hose is checked to ensure that the temperature there is well above dewpoint.

Under these circumstances, once the apparatus is satisfactorily adjusted, the temperature of the gas delivered to the humidicrib has remained remarkably stable for days on end, in contrast to our experience using the more conventional heated humidifiers. Although the internal gas volume is much greater than that of the apparatus described by Epstein (ANESTHESIOLOGY 35:532–536, 1971), this has not prevented satisfactory ventilation with the ventilators we have used. The humidifier compartment and delivery tube in these patients was replaced only at weekly intervals. Attempts to culture organisms from the apparatus at the time of change have been unsuccessful.

The humidifier has also been used in a similar fashion to humidify the fresh gases supplied to the Jackson Rees modification of an Ayre's T-piece in the operating theater during anesthesia for neonates and infants.

G. C. FISK, F.F.A., R.A.C.S.
J. B. VONWILLER, F.F.A., R.A.C.S.
Departments of Anaesthesia and Paediatrics
The Prince of Wales Hospital
Randwick, N.S.W. 2031
Australia

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The Advantages of Giving *d*-Tubocurarine before Succinylcholine

To the Editor:—Three studies recently published in ANESTHESIOLOGY have attempted to evaluate the disadvantages of preceding succinylcholine (SCh) with a small dose of non-depolarizing muscle relaxant.[1-3] The differences between two of these studies were emphasized in an editorial by Wollman.[4] I believe, however, that the similarities in all

studies, pointing out the minimal disadvantages and major advantages of this method of relaxant administration, deserve similar emphasis.

The fear that a desensitization block and prolonged apnea may follow SCh when preceded by a nondepolarizing muscle relaxant seems to be groundless, since this was not observed in the 183 patients studied.[1-3, 5] That the administration of a nondepolarizing muscle relaxant before SCh necessitates larger doses of SCh for adequate relaxation is substantiated, and full therapeutic advantages can be obtained if the proper doses of both relaxants are chosen. To varying degrees, all three studies demonstrated that paralysis by SCh will be decreased when it is preceded by 3 mg of *d*-tubocurarine. However, Freund and Rubin showed that adequate relaxation can be restored by increasing the dose of SCh by 70 per cent.[1] So, in a 70-kg patient, the usual dose of SCh of about 60 mg would have to be increased to 100 mg. Since the additional 30–40 mg of SCh does not result in a prolonged neuromuscular block, this seems to be a small price to pay for all the benefits of preceding SCh with *d*-tubocurarine. The benefits include decrease or elimination of: SCh-induced postoperative muscle pains; elevated intraocular and intragastric pressures; elevated serum creatine phosphokinase; myoglobinuria; and possibly hyperkalemia.

In summary, the similarities of these studies suggest that preceding SCh, 1 to 1.5 mg/kg, with 3 mg of *d*Tc or 20 mg of gallamine is a reasonable clinical approach.

RONALD D. MILLER, M.D.
Department of Anesthesia
School of Medicine
University of California
San Francisco, California 94122

REFERENCES

1. Freund FG, Rubin AP: The need for additional succinylcholine after *d*-tubocurarine. ANESTHESIOLOGY 36:185–187, 1972
2. Cullen DJ: The effect of pretreatment with nondepolarizing muscle relaxants on the neuromuscular blocking action of succinylcholine. ANESTHESIOLOGY 35:572–578, 1971
3. Miller RD, Way WL: The interaction between succinylcholine and subparalyzing doses of *d*-tubocurarine and gallamine in man. ANESTHESIOLOGY 35:567–571, 1971
4. Wollman H: The elephant's ears can be misleading (editorial). ANESTHESIOLOGY 35:566, 1971
5. Stovner J, Lund I: The muscle relaxants and their antagonists. Br J Anaesth 45:235–248, 1970

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Measuring Cerebral Blood Flow by the Nitrous Oxide Method

To the Editor:—The excellent review by Drs. Smith and Wollman (ANESTHESIOLOGY 36:378–400, 1972) requires some comments based on my own experiments.

Several values of CBF measured during hypothermia are cited in their review. Three of them [1, 2, 3] were measured by the Kety-Schmidt nitrous oxide method or a modification. Bering *et al.*,[4] as well as Adams *et al.*,[5] slightly modified the original nitrous oxide method by lengthening the nitrous oxide inhalation time to 15 minutes so that sufficient equilibration of the nitrous oxide between arterial or cerebral venous blood and cerebral tissue would take place. Zingg and Bender [6] pointed out that erroneously low values for CBF would be found if the solubility of nitrous oxide increased at lower temperatures; however, they actually used the original method and a partition coefficient of 1.0 for calculating CBF. Changes in the partition coefficient or the solubility of nitrous oxide were not taken into consideration in other reports.[1, 2, 7]

We investigated the uptake of nitrous oxide in cerebral tissue and blood at normothermia and moderate hypothermia. Nitrous oxide did not reach complete equilibrium between the cerebral tissue and blood (arterial as well as cerebral venous at 30 C and 34 C after 30 minutes of inhalation of 50 per cent nitrous oxide in oxygen. The distribution ratio (the ratio of nitrous oxide in cerebral tissue to that in cerebral venous blood, or relative rates of uptake by brain compared with uptake by cerebral venous blood) would be less than 1.0.

The nitrous oxide method, based on the assumption that the brain/blood partition coefficient of nitrous oxide is 1.0, cannot be ap-