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Correspondence

Dextromethorphan for Intubation

To the Editor:—A drug which would specifically suppress tracheal reflex irritability and increase the patient's ability to tolerate an endotracheal tube without any other effects would find wide use for clinical practice. In the past, we resorted to deeper levels of anesthesia, narcotics, or muscle relaxants to achieve this end, but all these affect functions other than cough. Recently I have used dextromethorphan intravenously to lessen or eliminate the bucking response to the endotracheal tube in 41 patients. Doses of 20 to 30 mg intravenously seem to effect a marked diminution or abolition of bucking without depressing respiration. Transient hypotension may occur following intravenous administration. At the doses used, the tracheal reflex response to suctioning and extubation is still present.

A method for determining objectively the tendency to react on the endotracheal tube is still being developed. In obtaining these preliminary observations, no topical anesthetic has been used, and intubation has been performed after a thiopental–succinylcholine sequence. Anesthesia is maintained with cyclopropane and oxygen delivered at a rate of 3 l/min in a semiclosed circle system. In this setting, very few patients have tolerated the tube at 10 per cent cyclopropane, but many tolerated it at 20 per cent cyclopropane. If the concentration of cyclopropane is lowered stepwise every 15 minutes, a point at which the patient will not tolerate the tube will be found. Injection of 10–20 mg dextromethorphan intravenously at this time will restore the patient's tolerance of the tube, and cyclopropane may then be further reduced. After dextromethorphan, many patients have tolerated the tube at 10 per cent cyclopropane and a few have tolerated it at 5 per cent cyclopropane. Tolerance of the tube by conscious patients has also been improved by intravenous administration of dextromethorphan.

Following these observations, I began to use similar doses before intubation and continued to obtain clinically satisfactory results. More recently, I have given the drug before induction. Patients become sedated, and they may remark that they feel "itchy" or "hot" in part or all of their bodies. There have been no other untoward effects either during or after this treatment.

The disturbing possibility that overly vigorous suppression of the cough reflex during the operating period may leave the patient's trachea relatively unprotected and vulnerable to aspiration in the postoperative period remains, however. For this reason, it would be highly desirable to have an antagonist to the antitussive effects of dextromethorphan. In animal experiments, neither levallorphan nor naloxone reversed the antitussive effects of dextromethorphan. I hope that a dextro analog of levallorphan or naloxone will be available for trial as an antogonist to dextromethorphan. If effective, it would be of great theoretical as well as practical interest.

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Ketamine-induced Convulsions

To the Editor:—An editorial by Winters [1] and lead article by Kayama [2] have emphasized CNS excitation by ketamine. Winters' proposed classification of anesthetics lists ketamine along with nitrous oxide and trichloroethylene as drugs which "include Stage I catalepsia only." This is in contrast to another group of drugs (phencyclidine, enflurane, and gamma-hydroxybutyrate) which "continue from Stage II to generalized convulsions." Kayama

closes his article with "the ketamine-induced EEG seizure does not lead to generalized convulsions. . . ." Both articles, however, give evidence for and suggest the possibility of generalized convulsions during ketamine anesthesia.

During a study of anesthesia for outpatient surgery, we observed a generalized grand mal seizure in a 26-year-old woman two minutes after an intravenous induction dose of 130 mg ketamine given for elective abortion. Treatment with oxygen by mask and diazepam, 10 mg, intravenously, was given immediately. This patient had no history of serious disease or convulsive disorder, and denied taking any drugs. Hypoxia by airway obstruction or hypoventilation could not account for the convulsion. She was alert and talking within 30 minutes and was discharged two hours later. A more complete neurologic evaluation was not carried out.

We have observed sporadic myoclonic jerks of arms or legs in several other patients during ketamine anesthesia. We now believe that generalized convulsions must be considered a potential complication of this drug.

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2. Kayama Y, Iwama K: The EEG, evoked potentials, and single-unit activity during ketamine anesthesia in cats. ANESTHESIOLOGY 36:316–328, 1972

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Postoperative Hypoxemia and Age

To the Editor:—We read with interest the paper by Dr. Kitamura and others (ANESTHESIOLOGY 36:244, 1972) on the contribution of age to postoperative hypoxemia. We would like to report some relevant measurements of our own.

A study of 42 male patients (mean age 45.1 years ±12.3 SD) who underwent vagotomy and pyloroplasty through a right paramedian incision was conducted. Their physical status was ASA grade 1. They were premedicated with morphine, 10 mg, dehydrobenzperidol, 5 mg, and atropine, 0.6 mg. Anesthesia was induced with thiopental and maintained with 70 per cent nitrous oxide in oxygen alone (16 patients), or supplemented by either trichloroethylene (12 patients) or morphine, 10 mg (14 patients). Muscle relaxation was achieved with pancuronium bromide and respiration controlled with a respirator. End-tidal P_{CO_2} was maintained in the range of 35–42 mm Hg. The operations lasted one to two hours. Atropine, 1.2 mg, and neostigmine, 2.5 mg, were administered during skin closure. In most patients, the adequacy of neuromuscular transmission was assessed with a peripheral-nerve stimulator.

The patients breathed room air for the first 30 minutes in the recovery room, after which arterial blood was sampled. Patients then breathed oxygen-enriched air from an Edinburgh mask with a 3 l/min O_2 flow. This provides an inspired oxygen concentration of 35–40 per cent.[1] After 30 minutes on this system, a second arterial sample was taken. Blood gases were measured using Radiometer electrodes. The P_{O_2} electrode was calibrated with nitrogen and room air and, when appropriate, values were corrected for temperature of the patient using the Severinghaus slide rule.[2] No correction was applied for differences in electrode responses to gas and blood.

During measurements, the patients appeared comfortable and could be roused. None developed shivering, cardiac arrhythmias, or hypotension.

Significant correlations between age and arterial P_{O_2} were found during breathing of both air ($r = -0.312$, $P = 0.05$) and oxygen-enriched air ($r = -0.490$, $P < 0.002$). The regression equations were:

$$Pa_{O_{2AIR}} = 81 - (0.285 \times \text{age})$$

$$Pa_{O_{2\,40\text{–}45\%\,O_2}} = 166 - (1.1204 \times \text{age})$$