related well with changes in plasma concentrations of the muscle relaxants.\textsuperscript{7}

Average plasma K\textsuperscript{+} levels in all groups decreased from 4.3 mEq preoperatively to 3.8 mEq during bypass. Low plasma K\textsuperscript{+} is known to decrease twitch height.\textsuperscript{8} With either agent, twitch heights in the normothermic group showed a continuous steady recovery during bypass. This suggests that the effects of changes in plasma K\textsuperscript{+} were not significant in our study.

The enhancement of the neuromuscular blockade produced by \textit{d}-tubocurarine or pancuronium during hypothermia might have been due either to a direct effect of hypothermia on the myoneural junction or to a direct effect on the action of muscle relaxant, or to a combination of the two. When the myoneural junction is exposed to hypothermia, quantal release of acetylcholine is increased and the motor end-plate becomes more sensitive to acetylcholine.\textsuperscript{9,10} These two factors can produce significantly increased isometric twitch tension of both directly and indirectly stimulated muscle. In spite of this increase in twitch tension, the neuromuscular blockade induced by either neuromuscular blocking agent was increased by hypothermia, implying that hypothermia probably affected the action of the neuromuscular blocking agent. The mechanisms involved in this phenomenon, however, are not yet clear. Further study will be needed to delineate the exact cause.

In summary, our study indicates that lowering the temperature enhances, while rewarming antagonizes, the neuromuscular blocking actions of \textit{d}-tubocurarine and pancuronium. It is, therefore, recommended that usual doses of \textit{d}-tubocurarine or pancuronium be reduced during hypothermia, and that muscle temperature of the patient be kept normal to insure adequate recovery from the muscle relaxant.

\textbf{REFERENCES}


8. Foldes FF: Factors which alter the effects of muscle relaxants. Anesthesiology 20:264–504, 1959


\textbf{Table 1. Twitch Height Changes at the Start of the Pump and during Hypothermia and Rewarming}

<table>
<thead>
<tr>
<th>Time</th>
<th>\textit{d}-Tubocurarine</th>
<th>Pancuronium</th>
</tr>
</thead>
<tbody>
<tr>
<td>At the start of the pump</td>
<td>18.5 ± 4.9</td>
<td>24.8 ± 11.9</td>
</tr>
<tr>
<td>During hypothermia (lowest) 30 minutes</td>
<td>7.3 ± 1.9</td>
<td>13.5 ± 6.7</td>
</tr>
<tr>
<td>before start of warming</td>
<td></td>
<td></td>
</tr>
<tr>
<td>At start of warming</td>
<td>10.6 ± 4.7</td>
<td>16.7 ± 7.9</td>
</tr>
<tr>
<td>30 minutes after start of warming</td>
<td>12.1 ± 4.1</td>
<td>22.8 ± 11.8</td>
</tr>
<tr>
<td></td>
<td>59.8 ± 14.7*</td>
<td>63.8 ± 24.4*</td>
</tr>
</tbody>
</table>

Mean ± SEM.

* Recoveries in twitch height during the 30 minutes after the start of warming were significantly greater than those during the corresponding 30-minute period before warming for both \textit{d}-tubocurarine and pancuronium.

\textbf{Postpartum Uterine Pressures with Different Doses of Ketamine}

GERTIE F. MARX, M.D.,* HYAN S. HWANG, M.D.,† PRASANTA CHANDRA, M.B., B.S.‡

The effects of ketamine on uterine tone and activity in term-pregnant women have been controversial, as both increases\textsuperscript{1,4} and decreases\textsuperscript{3,4} have been observed. Recently, results obtained in late-first-trimester and obstetrics, Albert Einstein College of Medicine, Bronx, New York. Accepted for publication June 9, 1978. Address reprint requests to Dr. Marx: Department of Anesthesiology, Albert Einstein College of Medicine—J-1224, 1300 Morris Park Avenue, Bronx, New York 10461.
early-second-trimester pregnancies were extrapolated to relate to the uterus at term and served to support the postulation that hazardous increases in uterine resting (base) pressure could develop.

As we have been impressed with both the maternal and the fetal effects of low-dose ketamine in obstetric practice, we felt that delineation of the uterine effects of this drug at term might settle the controversy surrounding its use. Accordingly, we employed our previously reported technique of postpartum uterine pressure measurements to determine the effects of four doses of ketamine on uterine activity in the immediate puerperium.

**METHOD**

Our studies were undertaken between 09.00 and 14.00 hours on primiparous or secundiparous whose labors had started after midnight, at least five hours after the last food or fluid ingestion, and who had received an oral antacid every three hours following admission. All patients delivered spontaneously with use of pudendal block (chloroprocaine, 1 per cent) with or without nitrous oxide–oxygen analgesia, or with nitrous oxide–oxygen analgesia alone. After expulsion of the placenta, a Csapo microballoon was inserted into the uterine cavity, filled with 0.8 ml of saline solution and connected, by way of a semirigid tube, to a pressure transducer (Statham P-1000A) and Physiograph recorder. Following a control period of at least 10 min each patient received a single intravenous injection of ketamine in one of the following doses: 25 mg (four patients), 50 mg (six patients), 75 mg (six patients), and 100 mg (six patients). Following injection of ketamine, oxygen was administered by use of a tight-fitting face mask, with special attention to maintenance of an open airway. Each parturient's postpartum weight was determined the next morning for calculation of the mg/kg ratio of ketamine.

When the effect of ketamine on uterine activity had disappeared, oxytocin, 10 milliunits (mIU), was administered intravenously, followed, after a short interval, by a continuous infusion of a solution containing oxytocin, 10 IU in 1,000 ml, at 30 drops (1.75 ml)/min; in seven cases, the bolus injection was omitted.

Statistical analysis was performed using the Student *t* test for paired data. Results are expressed as means ± standard errors.

Informed consent was obtained from all patients on admission to the labor suite.

**Table 1. Uterine Activity* before and after Various Doses of Ketamine (Means ± SE)**

<table>
<thead>
<tr>
<th>Ketamine (mg)</th>
<th>Number of Patients</th>
<th>Resting Pressure (torr)</th>
<th>Frequency of Contractions</th>
<th>Intensity of Contractions (torr)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Control†</td>
<td>Ketamine†</td>
</tr>
<tr>
<td>25</td>
<td>4</td>
<td>25.0 ± 4.6</td>
<td>2.8 ± 0.5</td>
<td>102.5 ± 22.6</td>
</tr>
<tr>
<td>50</td>
<td>6</td>
<td>22.5 ± 2.8</td>
<td>2.7 ± 0.2</td>
<td>93.3 ± 15.4</td>
</tr>
<tr>
<td>75</td>
<td>6</td>
<td>26.7 ± 4.0</td>
<td>2.4 ± 0.2</td>
<td>110.0 ± 10.7</td>
</tr>
<tr>
<td>100</td>
<td>6</td>
<td>24.0 ± 3.3</td>
<td>2.8 ± 0.5</td>
<td>102.1 ± 7.8</td>
</tr>
</tbody>
</table>

* Resting uterine pressure remained unchanged after ketamine in all patients.
† Ten-minute period following ketamine.
‡ Ten-minute period preceding ketamine.
§ Significant difference, *P* < 0.01.
RESULTS

The uterine pressure recordings for 22 parturients showed the same variability regarding frequency, intensity and duration of contractions observed previously. Ketamine, 25 mg (0.3–0.5 mg/kg) did not alter the contractile patterns in three of the four patients, and decreased the frequency of contractions in one. At 50 mg (0.6–1.0 mg/kg), there was no change in one patient, while increases in the intensities of the first contractions following the injection were seen in five women (fig. 1); these increases were similar to those obtained with intravenous injection of oxytocin, 10 mIU (21.7 ± 3.3 torr in 15 patients). Ketamine, 75 mg (1.0–1.4 mg/kg) led to marked increases in intensity of the following one to two contractions, and ketamine, 100 mg (1.5–1.8 mg/kg) increased the intensity of the following one to three contractions; again, the increases were similar to those found with oxytocin, 10 mIU. Both the 75 and 100 mg also increased the frequencies of contractions over the next 10 min (table 1).

No patient had an increase in resting (base) pressure of the uterus after either ketamine or oxytocin, 10 mIU. This was in contradistinction to the action of the oxytocin infusion, which led, without exception, to an increase in uterine resting pressure in addition to increases in the frequency and intensity of contractions (fig. 1).

DISCUSSION

The effects of drugs and anesthetic agents on uterine function have been studied in isolated uterine muscle strips, in pregnant animals, and, by external or internal tocology, in pregnant women at different stages of gestation. Of these methods of study, internal tocology of the term-pregnant uterus provides the most accurate data, as muscle mass, pressure and hormonal response of the uterus differ significantly in different species as well as at different stages of gestation. Thus, the muscle mass of the human uterus increases from a mean weight of 150 g at conception to 300–400 g at the midpoint of pregnancy and to approximately 1,100 g at term. During the first trimester, uterine resting and active pressures are low (resting pressure below 5 torr, mean active pressure 2.2 torr), and oxytocin response is suppressed. During the second trimester, all three begin a gradual increase, which is accelerated markedly from the thirty-sixth week on. At term, resting pressure is at least 10 torr, active pressure exceeds 40 torr, and oxytocin response is at its maximum.

Immediately following expulsion of the placenta, the internal uterine walls are opposed so that the cavity becomes almost obliterated. Transmission of pressure from the uterine wall to the microballoon used in our study is direct and unimpeded by uterine contents. Although radius and tension have decreased, the uterus continues to behave like a sphere, and obeys the law of LaPlace; uterine contractions resemble those of the first and second stages of labor so far as form and magnitude are concerned. Since hormonal milieu is unchanged, and concern for the infant no longer applicable, the postpartum uterus appears to provide optimal conditions for evaluating the effects of drugs used in obstetric analgesia–anesthesia. With this model, we have found that ketamine produces dose-related changes in uterine activity: a 25-mg injection causes no increase, while 50, 75, and 100 mg lead to transient increases in intensity and frequency of contractions; however, none of these doses induces an increase in the resting pressure.

The injection of oxytocin in a "physiologic" dose, i.e., 8–10 mIU/min, at term pregnancy, initiates uterine contractions of the same intensity, frequency and resting pressure as those seen in spontaneous labor. This dose has, therefore, been employed for comparative purposes. An "unphysiologic" dose, in contrast, increases frequency, intensity, and resting pressure. In our patients, the ketamine-induced increases in the intensities of contractions were similar to those recorded after one "physiologic" dose of oxytocin, and resting pressure remained unchanged. We conclude that ketamine, 100 mg, or less, has no unphysiologic effect on the term-pregnant human uterus under normal conditions. However, during situations in which any increase in uterine activity may be harmful, such as tetanic contraction, abruptio placentae or cord prolapse, the dose of ketamine should not exceed 25 mg. This is approximately the maximum recommended for analgesia in vaginal delivery (0.2–0.4 mg/kg maternal body weight), whereas the maximum used for induction of endotracheal anesthesia should not exceed 1 mg/kg, or 100 mg.

REFERENCES


* Schulman H: Personal communication.
Epidural Anesthesia in a Pediatric Patient with Congenital Tracheal Stenosis

Brendan T. Finucane, M.D.*

An 8-year-old child was scheduled for an ileoconduit operation. Congenital tracheal stenosis was incidentally found during induction of anesthesia. The case report describes in detail the subsequent anesthetic management of this patient by use of continuous epidural anesthesia.

REPORT OF A CASE

An 8-year-old white boy, weighing 23 kg, sought medical attention because of recurrent urinary-tract infections. The history included surgical correction of numerous urologic anomalies discovered at birth. These procedures had all been carried out with general anesthesia, without complication.

The patient was scheduled for a cystoscopic examination with general anesthesia. The anesthesia was uncomplicated, and consisted of thiopental, nitrous oxide, oxygen, and halothane by mask. An ileoconduit diversion was deemed necessary. On this occasion some problems were encountered. After four attempts at endotracheal intubation with tubes of decreasing diameters, a 4.0-mm uncuffed endotracheal tube was advanced into the trachea. A tracheogram revealed diffuse hypoplasia of the trachea with moderate stenosis of the distal two-thirds (fig. 1). The process extended into the left main-stem bronchus. There was no evidence of laryngeal involvement.

The operation was deferred. After careful consideration, continuous lumbar epidural anesthesia was selected, and the operation was rescheduled several days later.

Precordication with morphine sulfate, 5 mg, im, had the desired effect. On the patient's arrival in the operating room, an intravenous infusion of dextrose, 5 per cent, in lactated Ringer's solution was commenced.

The patient was placed in the left lateral decubitus position and the skin over the lower lumbar area was prepared. An 18-gauge Hustead needle was placed in the epidural space at the level of L4 by use of the loss-of-resistance technique. Following a 1.5-ml test dose with lidocaine, 1 per cent, 5 ml of 0.75 per cent bupivacaine with epinephrine, 1:200,000, were injected slowly through the needle. A 20-gauge radiopaque Teflon catheter (Deseret) was advanced with ease through the needle. The needle was removed and the catheter was securely taped. The patient was placed in the supine position and the sensory level was assessed. A more profound block had developed on the left side. A further 6 ml of 0.75 per cent bupivacaine was then injected through the catheter. Soon thereafter, an upper level to T3 was evident bilaterally.

The operation commenced about 15 minutes later and continued for a further five and a half hours. Additional sedation was given at intervals. Oxygen with varying concentrations of nitrous oxide (not >50 per cent) was administered throughout.

At two-hourly intervals further increments of bupivacaine, 0.75 per cent, with epinephrine, were injected. The object was to maintain an even level of anesthesia throughout. Towards the end of the procedure the surgeons encountered difficulty in closing the peritoneum. Lidocaine, 2 per cent, with epinephrine, 1:200,000, was selected at this time because of its shorter latency. Within minutes muscle relaxation was adequate. The patient received a total of 142 mg bupivacaine, 0.75 per cent, and 115 mg lidocaine over a six-hour period. Vital signs remained stable throughout the procedure. There was no episode of hypotension. Recovery was uneventful, and the patient was discharged several days later.

DISCUSSION

Most pediatric operations involve general anesthesia. The reasons for this have been reviewed by Eather. In the present case, it was clear that there were few alternatives. The duration of operation was expected to exceed four hours; consequently, a continuous method of regional anesthesia was selected. Contin-