We chose nasopharyngeal insufflation of inhaled anesthetics, an old technique, for induction of anesthesia for several reasons. First, preoperative evaluation demonstrated the only upper airway to be the left nostril with a maximum caliber of 3.5 mm. Secondly, the tumor stalk was friable and pulsatile and filled the right side of the mouth. Finally, surgical exposure required a transoral route or right mandibular flap route, thus compromising any oral airway. Preoperative examination also demonstrated easy glottic visualization by direct laryngoscopy on the left of midline.

In conclusion, perioperative management of newborn pharyngeal teratomas should include careful preoperative evaluation of the upper airway, exclusion of midline CNS lesions, provision for a secure perioperative airway, complete surgical excision, and early postoperative evaluation for airway edema, distortion, or recurrent hemorrhage. Prognosis in properly managed and uncomplicated cases is excellent.

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Anesthesiology
61:610–613, 1984

Cardiovascular Effects of Ketamine Given to Relieve Penile Turgescence after High Doses of Fentanyl

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Penile turgescence during attempted Foley catheterization after induction of anesthesia with high doses of fentanyl has been observed in approximately 1% of male patients undergoing cardiac surgery at my institution. Ketamine is touted as an agent effective in the treatment of penile turgescence. But induction doses of ketamine increase heart rate, blood pressure, cardiac output, pulmonary artery pressure, and intrapulmonary shunt. We present four cases in which the prior administration of fentanyl 40–50 μg·kg⁻¹ and pancuronium 0.08 mg·kg⁻¹ blocked these cardiovascular effects when ketamine 0.5–1.8 mg·kg⁻¹ was given to induce detumescence.

REPORT OF FOUR CASES

All patients were premedicated with morphine 0.1 mg·kg⁻¹ im and lorazepam 0.05 mg·kg⁻¹, po, 1.5 h before induction of anesthesia. Diazepam 4–10 mg iv was administered in 2-mg increments over 15 min during the insertion of arterial and pulmonary artery catheters. Arterial blood pressure, pulmonary artery pressure, and central venous pressure were recorded continuously. While breathing oxygen, metocurine 4 mg iv was given. Anesthesia was induced with fentanyl 40–50 μg·kg⁻¹ iv over a 5-min period. Pancuronium 0.08 mg·kg⁻¹

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Key words: Anesthetics, intravenous: fentanyl; ketamine. Complications: penile turgescence.
was administered 1 min after initiation of the fentanyl infusion. No muscle rigidity was encountered. End-tidal CO₂ was monitored and maintained at 4.5% ± 0.5% with controlled mask ventilation. Laryngoscopy and endotracheal intubation were accomplished 6-7 min after induction without technical difficulty. Five minutes after endotracheal intubation, F(IO₂) ranged from 35 to 38 mmHg, with an

Turgescence occurred in each patient while the penis was being prepped for Foley catheterization 10 min after induction of anesthesia. There were no cardiovascular signs of light anesthesia, i.e., hypertension or tachycardia, and no sweating or tearing. Ketamine 0.5-1.8 mg·kg⁻¹ iv produced sufficient detumescence within 4 min to permit catheterization and surgical draping. No changes were observed in ECG leads II and V₅. The hemodynamic variables measured before and 4 min after ketamine administration are presented in table 1.

The operations proceeded successfully. All patients received additional fentanyl and diazepam before terminating cardiopulmonary bypass. The tracheas of patients were extubated the following morning without the use of naloxone or phystostigmine. No patient had intraoperative recall or postoperative hallucinations, dysphoria, or unpleasant dreams.

Patient 1 was a 25-year-old 74 kg man with a bicuspid aortic valve and aortic insufficiency who presented for aortic valve replacement (AVR). Patient 2 was a 34-year-old 93-kg man with unstable angina, ischemic ECG changes, and a single proximal LAD lesion who presented for a single vessel coronary artery bypass. Patient 3 was a 21-year-old 56-kg man with a bicuspid aortic valve and aortic insufficiency who presented for AVR. Patient 4 was a 49-year-old 98-kg man with unstable angina who presented for a triple bypass. No patient had propranolol preoperatively on the morning of surgery. Patients 2 and 4 were on a continuous infusion of nitroglycerin 3 μg·kg⁻¹·min⁻¹ from the time they arrived in the operating room until cardiopulmonary bypass was initiated.

### DISCUSSION

Ketamine produces its sympathomimetic actions primarily by direct stimulation of the central nervous system. Roizen et al. observed that anesthetic agents, including ketamine, produce dissimilar changes in the neurotransmitter content of brain nuclei controlling cardiovascular function. Ngai et al. demonstrated that ketamine, enfurane, and halothane affect acetylcholine turnover differently in various areas of the brain. Thus, the cardiovascular effects of ketamine administered to anesthetized patients will vary from agent to agent and will be different from those seen in unanesthetized individuals. For example, prior dosing with diazepam 0.4 mg·kg⁻¹ enable ketamine 2 mg·kg⁻¹ to be given without producing changes in heart rate, blood pressure, or vascular resistance. Ketamine 2 mg·kg⁻¹ administered to patients anesthetized with halothane-nitrous oxide (1 MAC) produced no change in heart rate but lowered blood pressure and cardiac output and raised systemic vascular resistance.

The cardiovascular consequences of ketamine given to patients who have received high doses of fentanyl have not been reported. Ketamine interacts with opiate receptors as an agonist, and there are opiate receptors in those areas of the brain associated with cardiovascular regulation. Furthermore, fentanyl preloading blunts both the hemodynamic and neuroendocrine response to a rapid sequence induction of anesthesia, normally a potent stimulus for the sympathetic nervous system. Thus, our observation that no hyperdynamic changes occurred in hemodynamic variables after ketamine administration in patients receiving large doses of fentanyl is not surprising. We were able to supplement high-dose fentanyl-oxygen anesthesia with an agent associated with significant amnesia and analgesia without producing cardiovascular depression, as is seen when nitrous oxide, diazepam, or isoflurane are added. The cardiovascular stability observed with ketamine administration in this setting may be advantageous in patients in whom recall, bronchospasm, or penile turgescence present as intraoperative problems.

High doses of fentanyl are not expected to block those cardiovascular effects that result from direct action of anesthetic agents or adjuvants on the peripheral nervous system or vascular smooth muscle. For example, when pancuronium is administered during high-dose fentanyl anesthesia, tachycardia occurs. Ketamine, too, has peripheral vascular effects. It selectively inhibits nicotinic-receptor mediated responses while sparing the muscarinic ones. It is a direct depressant of cardiac and vascular smooth muscle. Finally, it blocks intraneuronal and extraneuronal uptake of norepinephrine by postganglionic adrenergic neurons. However, the effects of ketamine on the reuptake of norepinephrine

<table>
<thead>
<tr>
<th>Table 1. Hemodynamic Variables Immediately before and Four Minutes after Ketamine Administration in Patients Induced with High-Dose Fentanyl and Pancuronium</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient No.</td>
</tr>
<tr>
<td>-----------------</td>
</tr>
<tr>
<td><strong>Diazepam mg·kg⁻¹</strong></td>
</tr>
<tr>
<td><strong>Ketamine mg·kg⁻¹</strong></td>
</tr>
<tr>
<td><strong>Heart rate</strong></td>
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<tr>
<td>(beats/min)</td>
</tr>
<tr>
<td><strong>BP (mmHg)</strong></td>
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<tr>
<td><strong>(systolic/diastolic)</strong></td>
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<tr>
<td><strong>PAP (mmHg)</strong></td>
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<tr>
<td><strong>(systolic/diastolic)</strong></td>
</tr>
<tr>
<td><strong>PCWP (mmHg)</strong></td>
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<tr>
<td><strong>CVP (mmHg)</strong></td>
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<tr>
<td><strong>CO (l/min)</strong></td>
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<tr>
<td><strong>(l/min)</strong></td>
</tr>
<tr>
<td><strong>CVP (mmHg)</strong></td>
</tr>
<tr>
<td><strong>5.6</strong></td>
</tr>
<tr>
<td><strong>Pco₂ (mmHg)</strong></td>
</tr>
<tr>
<td><strong>484</strong></td>
</tr>
</tbody>
</table>

The first number in the paired rows is the value observed before ketamine administration but after fentanyl 40-50 μg·kg⁻¹ and pancuronium 0.08 mg·kg⁻¹. The second is the value observed 4 min after ketamine administration. BP = blood pressure; PAP = pulmonary artery pressure; PCWP = pulmonary capillary wedge pressure; CVP = central venous pressure; CO = cardiac output.
may not be clinically significant at the plasma concentrations achieved with 1 mg · kg⁻¹ iv. At plasma concentrations high enough to block the uptake of norepinephrine, the direct depressant effects of ketamine appear to predominate. The net effect of ketamine on vascular resistance is the result of four processes: central sympathetic stimulation, inhibition of ganglionic transmission, blockade of norepinephrine reuptake at the neuroeffector junction, and direct smooth muscle depression.

If the central stimulation is blocked by fentanyl and if the effect on norepinephrine reuptake is not clinically significant, then the depressant effects should predominate. That we did not observe a fall in vascular resistance suggests that complete suppression of sympathetic stimulation did not occur, that the depressant effects were not clinically significant in these patients at the plasma concentrations obtained, or that pancuronium contributed to the blockade of norepinephrine uptake at the neuroeffector junction. If any one of these occurs, then clinical scenarios can be envisioned wherein ketamine administration with high doses of fentanyl and pancuronium may not be benign. Patients with poor left ventricular function or patients on drugs that depress vascular smooth muscle may show a fall in cardiac output, blood pressure, or resistance. Patients manifesting ventricular irritability may demonstrate an increase in ectopy. Ischemia may be triggered in patients with variant angina. Patients with ischemia may show a worsening of ischemic signs. However, in patient 2 there was no change in ST segments as a result of ketamine administration.

The circulatory changes associated with the penile erectile cycle may be divided into four phases: I—flaccidity in which the erectile tissue of the corpus cavernosa is contracted; II—tumescence, or tumescence, in which the erectile tissue is relaxed to permit an increase in the volume of blood in the penis; III—full erection in which the pressure of the blood in the cavernous bodies increases to 5–10 mmHg below systolic aortic pressure; and IV—detumescence in which the pressure and volume of blood in the cavernous bodies falls rapidly as venous outflow acutely rises. Priapism is the abnormal persistence of phase III. There are four sets of smooth muscle structures involved in the erectile cycle: 1) arterial pads that are contracted in phases I and IV and relaxed in II and III; 2) arteriovenous shunts that are open in phases I and IV and closed in II and III; 3) cavernous erectile tissue that is contracted by noradrenergic mechanisms in I and IV and relaxed by vasoactive intestinal peptide released from postganglionic parasympathetic neurons in II and III; and 4) venous smooth muscle that is relaxed in I and IV and contracted in II and III. The most significant of these smooth muscle structures appears to be the cavernous erectile tissue. Neither acetylcholine nor alpha-blockade and beta-blockade will produce an erection. Muscarinic blockade with atropine will not prevent an erection, but ganglionic blockade does. The stimulus either for psychogenic or for reflex erections is mediated through sacral parasympathetic outflow.

Anesthesia with fentanyl may increase the incidence of penile erections because it centrally increases vagal tone. Ketamine is an effective detumescence agent because it decreases central vagal outflow, because it blocks transmission through parasympathetic ganglia, or because it blocks reuptake of norepinephrine at the neuroeffector junction in cavernous erectile tissue. However, ketamine does not always produce detumescence.

In summary, prior administration of pancuronium and high-dose fentanyl completely blocked the stimulating effect of ketamine on the cardiovascular system. Ketamine 0.5–1.8 mg · kg⁻¹ given intravenously produced detumescence in all four patients without electrocardiographic evidence of ischemia or dysrhythmia and without changes in hemodynamic variables.

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Acute Intermittent Obstruction of a Björk–Shiley Mitral Valve: A Case Report

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Mechanical obstruction of a prosthetic valve can be caused either by intrinsic valve-related defects or extrinsic mechanisms.1,2 Although acute obstruction of the mitral valve markedly increases left atrial pressures,1,2 we observed a patient with acute prosthetic valve dysfunction with almost no increase in the pulmonary artery occlusive pressure (PAOP).

REPORT OF A CASE

A 54-year-old man had mitral incompetence for 10 years. Two months before valve replacement was performed, he developed cardiac failure. Despite therapy, the patient remained in New York Heart Association class III. The ECG showed atrial fibrillation and generalized ST segment depression. By roentgenogram he had a cardiothoracic ratio of 0.6, indicating cardiac enlargement. The left atrial dimension on the echocardiogram was 51 mm. Cardiac catheterization revealed diffuse hypocontractility of the left ventricle, a grade IV mitral regurgitation, and a very enlarged left atrium.

After premedication with morphine 10 mg im and before induction of anesthesia, an iv line, radial artery catheter, and a pulmonary artery catheter were inserted. The mean pressures in mmHg were...