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infant airway, but it does represent an alternative method in cases of anticipated as well as unexpected difficult tracheal intubation.

Should blind tracheal intubation through the LMA prove unsuccessful, the airway can be maintained with the LMA in place. If the airway requires further protection, the fiberoptic bronchoscope may be used to facilitate intubation using the LMA as a guide.<sup>2,6-8</sup>

We describe two cases in which successful blind tracheal intubation was achieved using the LMA as a guide in infants with difficult airways. In the event that a difficult neonatal or pediatric airway is encountered, whether expectedly or unexpectedly, this simple technique may prove life-saving.

The authors thank Jacques E. Chelly, M.D., Ph.D., for his assistance and review of this case report.

Anesthesiology  
1996; 84:1511-3  
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## Cardiogenic Shock after Electroconvulsive Therapy

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MODERATE cardiac changes due to myocardial stunning<sup>1,2</sup> and acute neurogenic pulmonary edema<sup>3</sup> after electroconvulsive therapy (ECT) have been reported. We report a case of neurogenic pulmonary edema and fulminant cardiogenic shock after ECT in a patient receiving concurrent  $\beta$ -adrenergic blockade.

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Submitted for publication September 25, 1995. Accepted for publication February 12, 1996.

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Key words: Complications, electroconvulsive therapy; cardiogenic shock; pulmonary edema; stunned myocardium.

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## Case Report

A 41-yr-old woman was admitted for major depression, dissociative disorder, and self-mutilation. She had no prior history of cardiopulmonary disease, and her physical examination results were significant only for obesity (112 kg, 173 cm). Preoperative electrocardiogram and routine laboratory test results were normal. The patient's medications included 80 mg paroxetine daily, 80 mg propranolol long-acting daily, 30 mg propranolol four times daily, and 100 mg naltrexone twice daily. After failure of intensive psychotherapy and antidepressant medications, ECT was instituted. Anesthesia was induced with 100 mg methohexital and 100 mg succinylcholine. Ventilation by mask, using an ambubag, was initiated easily, and  $Sp_{O_2}$  was 99%. Baseline blood pressure was 130/80 mmHg, and heart rate was 60 beats/min. Bifrontotemporal ECT was initiated (Thymatron DGX, Somatics, Lake Bluff, IL) with bidirectional brief pulse square wave at 35% energy. A generalized tonic/clonic seizure was induced, and blood pressure increased to 190/120 mmHg, with a heart rate of 100 beats/min and  $Sp_{O_2}$  of 99%, with easy manual ventilation. As the patient awakened and spontaneous respirations resumed, she became agitated and confused, and hemoglobin oxygen saturation ( $Sp_{O_2}$ ) began to decrease. Assisted ventilation with 100%  $O_2$  was continued, and the patient began to expectorate copious amounts of pink frothy



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fluid. Oxygen saturation decreased to less than 90%, and the trachea was intubated. Auscultation revealed widespread bilateral crackles throughout the chest, and the chest radiograph showed fulminant pulmonary edema with a normal cardiac silhouette. Treatment of acute pulmonary edema with furosemide, morphine, and nitroglycerin was instituted. Despite aggressive therapy, mechanical ventilation with 100% O<sub>2</sub> and paralysis with a neuromuscular blocking agent, SpO<sub>2</sub> continued to decrease, and the patient's blood pressure began to decrease. During mechanical ventilation, with intermittent mandatory ventilation rate 12, tidal volume 800 ml, positive end-expiratory pressure 15 cmH<sub>2</sub>O, and FiO<sub>2</sub> 1.0, an arterial blood gas showed pH 7.32, P<sub>CO</sub><sub>2</sub> 42 mmHg, P<sub>O</sub><sub>2</sub> 56 mmHg, and 86% saturation. An electrocardiogram demonstrated minor nonspecific ST and T-wave changes, and a portable 2-D echocardiogram revealed a mildly dilated left ventricle with severe global hypokinesia and an ejection fraction of less than 20%. The patient was transferred to the medical intensive care unit, where her condition continued to deteriorate, and a pulmonary artery catheter was inserted. Initial hemodynamic values showed cardiac index 1.3 l · min<sup>-1</sup> · m<sup>-2</sup>, pulmonary capillary wedge pressure 18 mmHg, pulmonary artery pressures 34/16 mmHg, and systemic vascular resistance 1,874 dynes/cm<sup>2</sup>. Her blood pressure at that time was 76/58 mmHg, and her heart rate was 121 beats/min, with an SpO<sub>2</sub> of 95%. The blood pressure continued to decrease, and serial addition of inotropic drugs including dopamine, dobutamine, and epinephrine and use of an intraaortic balloon pump were instituted. Over the next 5 h, the patient began demonstrating slow reversal of her hemodynamic abnormalities, and on the second day after ECT, vasopressor support and the intraaortic balloon pump were withdrawn. A repeat echocardiogram at that time revealed an ejection fraction of 40% and no dilatation of the left ventricle, with significantly improved wall motion of the septum, apex, and posterior segments. Her electrocardiogram showed reversal of the nonspecific changes. A myocardial infarction was ruled out by measurement of enzymes and serial electrocardiograms. The trachea was extubated on the fourth day after ECT. The following week, coronary angiography and cardiac catheterization showed normal coronary arteries, normal ventricular function, ejection fraction of 77%, and cardiac index of 3.0 l · min<sup>-1</sup> · m<sup>-2</sup>.

## Discussion

ECT is performed routinely during general anesthesia. Published studies have demonstrated the relative safety of ECT even in patients with severe cardiovascular disease.<sup>4,5</sup> Nevertheless, cardiovascular events can be the principal cause of morbidity and mortality in patients after ECT.<sup>6</sup> Messina *et al.*<sup>7</sup> described the effects of ECT on the electrocardiogram and cardiac function and noted regional wall motion abnormalities, including global left ventricular hypokinesia. Although only a few cases like ours and none with cardiogenic shock have been described, a case of pulmonary edema after ECT was reported and ascribed to a purely neurogenic mechanism secondary to increased intracranial pressure from the ECT-induced seizure. In addition, a case of neurogenic pulmonary edema secondary to grand mal epilepsy<sup>3</sup> was described.

Pulmonary edema has long been recognized as an occasional complication of epileptic seizures,<sup>8</sup> and acute pulmonary edema may be induced experimentally in animals by increasing intracranial pressure.<sup>9</sup> The connection to ECT-induced seizure and pulmonary edema was made, noting that increasing cerebrospinal fluid pressure has been demonstrated to cause  $\alpha$ - and  $\beta$ -adrenergic stimulation, leading to peripheral vasoconstriction and hypertension, and increase of pulmonary arterial and venous pressures. The resultant increase in pulmonary blood flow shifts blood to the central circulation, leading to the development of neurogenic pulmonary edema. In the earlier report of neurogenic pulmonary edema after ECT,<sup>3</sup> a normal electrocardiogram result was obtained, however, no other cardiac function studies or discussion of cardiac involvement was mentioned. The increased myocardial oxygen demand, if severe enough to cause stunning, could initiate changes that would exacerbate pulmonary edema. Experimental evidence suggests that stimulation of adrenergic receptors may increase capillary permeability directly, giving an additional mechanism for pulmonary edema.<sup>10</sup>

The sequence postulated for myocardial stunning generally includes a brief episode of severe ischemia leading to prolonged myocardial dysfunction, with a gradual return to contractile activity.<sup>11,12</sup> This ischemia may be related to decreased myocardial oxygen supply or increased oxygen demand. In patients undergoing ECT, the increased myocardial oxygen demand appears to be predominant. The physiologic etiology for myocardial stunning has not been definitively elucidated, although studies with positron emission tomography<sup>13</sup> and ultrasonic tissue characterization<sup>14</sup> have been able to differentiate between irreversible ischemic injury and stunning of the myocardium.

Our case has some similarities to the previously noted case reports with acute pulmonary edema developing immediately after ECT, probably from neurogenic causes combined with stunned myocardium-induced pulmonary edema and cardiogenic shock. The severity of this case may have been related to the patient's preoperative  $\beta$ -blockade. A large dose of propranolol has been suggested for the treatment of anxiety, impulse control, and in particular, rapid personality switching in patients with multiple personality disorders<sup>15</sup> and was being used in our patient for this purpose. Furthermore,  $\beta$  blockade, including propranolol, has been recommended for the attenuation of the hypertension and tachycardia after ECT.<sup>16</sup> Labetalol<sup>6,17</sup> and



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esmolol<sup>17,18</sup> have been demonstrated to attenuate ECT-induced hypertension and tachycardia. Labetalol, however, has  $\alpha$ -adrenergic blocking properties in addition to its  $\beta$  blockade, which is more ideal, given the  $\alpha$ -adrenergic stimulation accompanying ECT. Although esmolol, which is a  $\beta$ -1-selective agent, would not block  $\alpha$ -adrenergic mediated responses, it does not have significant peripheral  $\beta$  blockade, so it would not be expected to lead to unopposed peripheral  $\alpha$  activity. Our patient, receiving propranolol, a nonselective  $\beta$  blocker, may have been at increased risk because of unopposed  $\alpha$  stimulation exacerbating the cardiac and pulmonary responses after the ECT. Clinicians should be aware that patients undergoing ECT who are receiving nonselective  $\beta$ -adrenergic blockers may be at increased risk for hemodynamic instability.

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Anesthesiology  
1996; 84:1513-5

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Lippincott-Raven Publishers

## Transient Muscular Spasm after a Large Dose of Intrathecal Sufentanil

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Received from the Service d'Anesthésie-Réanimation Chirurgicale, Hôtel-Dieu, Nantes, France. Submitted for publication December 27, 1995. Accepted for publication February 13, 1996.

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Key words: Anesthetics, opioids: sufentanil. Anesthetic technique, spinal: intrathecal anesthesia. Complications: neurotoxicity.

INTRATHECAL sufentanil produces analgesia without motor or sympathetic blockade. When used as sole spinal agent, it can relieve labor pain at intrathecal doses of between 5 and 15  $\mu\text{g}$ .<sup>1,2</sup> However, a report of muscular spasm in the lower limbs after administration of intrathecal solution containing epinephrine added to sufentanil<sup>3</sup> suggests that irritative effects can occur.