the authors presented, the measured Po2 at 50% saturation (p50) was normal and the oximetry was low. This suggests the dissociation curve was not shifted and the low oximetry seen in their patient was due to the fraction of the abnormal hemoglobin not binding oxygen. Secondly, the authors state that a rightward shift in the oxygenhemoglobin dissociation curve results in a higher p50. However, they incorrectly state that a rightward shift in the oxyhemoglobin dissociation curve occurs with blood transfusion, reduced levels of 2,3-diphosphoglycerate (DPG), hypophosphatemia, and hypothyroidism. Reduced levels of 2,3-DPG result in a leftward shift of the curve and increased affinity of oxygen binding to hemoglobin (lower p50).\textsuperscript{6} Reduced levels of 2,3-DPG are known to occur with hypothyroidism and hypophosphatemia.\textsuperscript{3} As well, stored blood has reduced levels of 2,3-DPG, potentially resulting in a leftward shift of the curve after transfusion.\textsuperscript{4}

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Intubating ICU Patients With Ketamine

Adverse Effects That Can Occur

To the Editor:

We read with interest the article by Dr. Walz et al\textsuperscript{1} and wish to enlighten the readers to a misconception about a drug that is mentioned and used in the ICU setting. The review article is excellent but requires additional explanation about the drug ketamine.

According to the authors, ketamine, when used in the ICU for intubation, stimulates the CNS system, thereby causing an increase in BP and heart rate. This phenomenon is usually expected by anesthesiologists, surgeons, and critical care specialists alike. Intubating conditions are also improved. That may well be, but the increase in BP and heart rate are not routinely achieved.

On the contrary, critically ill patients with minimal physiologic reserve do not always manifest this condition, but rather the opposite can occur: hypotension and bradycardia. Not only can it happen in the operating room but also in the ICU setting.

Ketamine is thought to stimulate cardiovascular functions by several mechanisms. First, it directly stimulates the sympathetic nervous system, resulting in the release of catecholamine.\textsuperscript{2} Second, ketamine increases tissue and circulating norepinephrine levels by inhibiting their neuronal and extraneuronal re-uptake.\textsuperscript{3} Elevated serum cortisol levels during ketamine anesthesia were demonstrated in elective (noncritically ill) patients, suggesting that the agent produced adrenocortical stimulation. Critically ill patients with minimal physiologic reserve are maximally compensating for hypovolemia, hypoxemia, fluid-electrolyte, acid-base, and nutritional problems.

According to Lippmann et al\textsuperscript{4} there was an early progressive increase in heart rate, cardiac index, arterial and venous pressure, stroke work and oxygen delivery, oxygen consumption, and oxygen extraction. In critically ill patients, however, ketamine did not produce uniform responses and was not without some adverse effects. There were a diversity of responses in mean arterial pressure, heart rate, cardiac index, oxygen consumption, oxygen extraction, and venous admixture. In some cases, ketamine may even cause maldistribution of systemic blood flow resulting in inadequate tissue oxygenation. Patients in this series were severely stressed and critically ill with depleted catecholamine and adrenocortical stores. Moreover, prolonged severe stress blunts sympathetic and/or adrenocortical stimulation by ketamine.

Lippmann et al\textsuperscript{5} concluded that the variability of ketamine responses in these patients was largely attributable to the balance between a direct myocardial depressant effect and a stimulatory sympathomimetic action of ketamine. This is altered in the critically ill patient whose compensatory responses may be affected to different degrees. For example, the hypovolemic patient may respond to sympathetic stimulation with tachycardia but be unable to respond with increased cardiac output. Those with limited myocardial reserves and increased demands may respond to ketamine with reduced cardiac output. They also conclude that although ketamine may be the agent often used in emergencies, whether it be in the operating room/ICU, its side effects, even in small doses, may lead to unanticipated severe adverse effects. Even though ketamine may facilitate intubation conditions, the adverse effects must be weighed carefully in critically ill patients.

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Bronchiectasis in Acute Pneumonia . . . Pseudobronchiectasis

To the Editor:

I read with interest in a recent issue of CHEST (June 2007)\textsuperscript{1} the case report “A 53-Year-Old Man With Fever, Clubbing,