Critical Hemoglobin Desaturation Will Occur before Return to an Unparalyzed State following 1 mg/kg Intravenous Succinylcholine

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THE American Society of Anesthesiologists (ASA) difficult airway algorithm recommends that if initial attempts at tracheal intubation after the induction of general anesthesia are unsuccessful, the practitioner should "consider the advisability of awakening the patient." With respect to the use of muscle relaxants, "awakening" is assumed to mean return to an unparalyzed state that permits life-sustaining spontaneous ventilation (hereafter referred to as functional recovery). The advisability of attempting and the likelihood of achieving functional recovery before life-threatening hemoglobin desaturation occurs depends on the initial alveolar fraction of oxygen (F\textsubscript{A}O\textsubscript{2}) and the minute ventilation (V\textsubscript{E}). When the minute ventilation is zero (e.g., a complete nonpatent airway), the advisability of waiting for functional recovery to occur is based on a comparison of the time to functional recovery versus the time to critical hemoglobin desaturation. The purpose of this analysis is to show that critical hemoglobin desaturation with V\textsubscript{E}= 0 occurs before the time to functional recovery for various patients receiving 1 mg/kg of intravenous succinylcholine.

Time to Significant Hemoglobin Desaturation

The time to hemoglobin desaturation is derived from the apnea (V\textsubscript{E}= 0) model of Farmery and Roe and is shown in figure 1 for various types of patients (the author will supply the values used for the major physiologic variables [surface area, hemoglobin concentration, blood volume, cardiac output, alveolar volume, oxygen consumption, shunt fraction, initial F\textsubscript{1}O\textsubscript{2} and F\textsubscript{1}CO\textsubscript{2}] required by the model on request). Because it would be dangerous to obtain time to significant hemoglobin desaturation data in humans, the model of Farmery and Roe is uniquely useful for analysis below S\textsubscript{a}O\textsubscript{2} = 90%. For example, when the endpoint for measuring desaturation versus time curves in children is defined as S\textsubscript{a}O\textsubscript{2} = 90%, there is as great as a 30% incidence of overshoot to S\textsubscript{a}O\textsubscript{2} = 70% and a 50% incidence of overshoot to a S\textsubscript{a}O\textsubscript{2} between 71% and 80%. During the time required to reach the various levels of hemoglobin desaturation, it is assumed that the airway is nonpatent (a reason why V\textsubscript{E}= 0) so that apneic insufflation of oxygen is not possible.

Figure 1 shows that for a healthy 70-kg adult, the moderately ill 70-kg adult, a healthy 10-kg child, and an obese 127-kg adult, S\textsubscript{a}O\textsubscript{2} = 80% is reached after 8.7, 5.5, 3.7, and 3.1 min, respectively, and S\textsubscript{a}O\textsubscript{2} = 60% is reached at 9.9, 6.2, 4.3, and 3.8 min, respectively. Critical hemoglobin desaturation is defined as S\textsubscript{a}O\textsubscript{2} ≤ 80% and decreasing; for the patients shown in figure 1, at S\textsubscript{a}O\textsubscript{2} ≤ 80% the range in rate of decrease is 20–40%/min.

To validate the model, we evaluated published data on desaturation. Table 1 shows that the predicted apnea time to reach a specific S\textsubscript{a}O\textsubscript{2} (89–91%) agrees reasonably well with actual data from patients whose weight and degree of normalcy and preoxygenation are reliably known. The most probable reason the model slightly overpredicts the apnea time to a specific oxyhemoglo-
bin endpoint in the majority of comparisons in table 1 (7 of 11) is that the model assumes that preoxygenation and denitrogenation are complete, whereas in actual patients neither of these preoxygenation conditions may be fully realized.10

The much shorter time to hemoglobin desaturation

Table 1. Apnea Time to a Specific $S_{\text{ao}}_{2}$ in Actual Patients under General Anesthesia Compared to Predicted Time from Model of Farmery and Roe20

<table>
<thead>
<tr>
<th>Reference, #</th>
<th>Type of Patient*</th>
<th>Time of Preoxygenation (min)</th>
<th>$S_{\text{ao}}_{2}$ Endpoint (%)</th>
<th>Time to Reach Endpoint (min ± SD)</th>
<th>Predicted Time to Reach Same $S_{\text{ao}}_{2}$ Endpoint from Model (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Xue, #3</td>
<td></td>
<td>2</td>
<td>90</td>
<td>2.00 ± 0.10</td>
<td>3.0</td>
</tr>
<tr>
<td>7.4 ± 1.2 kg</td>
<td></td>
<td></td>
<td>90</td>
<td>2.82 ± 0.12</td>
<td>3.5</td>
</tr>
<tr>
<td>12.3 ± 0.9 kg</td>
<td></td>
<td></td>
<td>90</td>
<td>4.10 ± 0.20</td>
<td>4.0</td>
</tr>
<tr>
<td>24.4 ± 2.5 kg</td>
<td></td>
<td></td>
<td>90</td>
<td>2.0 ± 0.15</td>
<td>3.3†</td>
</tr>
<tr>
<td>Patel, #4</td>
<td>7–24 months</td>
<td>3</td>
<td>91</td>
<td>6.8 ± 0.6</td>
<td>7.0‡</td>
</tr>
<tr>
<td>Teller, #5</td>
<td>52 ± 10 year, 20 ± 15 pack years</td>
<td>3</td>
<td>91</td>
<td>8.9 ± 1.0</td>
<td>8.0</td>
</tr>
<tr>
<td>Gambee, #6</td>
<td>28 ± 5 year, 58 ± 13 kg Nonsmokers</td>
<td>3</td>
<td>91</td>
<td>8.9 ± 1.0</td>
<td>8.0</td>
</tr>
<tr>
<td>Drummond, #7</td>
<td>48 years, Normal weight</td>
<td>0</td>
<td>88.8</td>
<td>1.0</td>
<td>1.1</td>
</tr>
<tr>
<td>Bhatia, #8</td>
<td>Normal 58 kg</td>
<td>2.7</td>
<td>92</td>
<td>5.0 ± 1.3</td>
<td>6.8</td>
</tr>
<tr>
<td>Jense, #9</td>
<td>Normal weight</td>
<td>5</td>
<td>90</td>
<td>6.1 ± 0.4</td>
<td>8.0</td>
</tr>
<tr>
<td>Weight &gt;20% ideal but &lt;45 kg above ideal</td>
<td>5</td>
<td>90</td>
<td>4.1 ± 0.3</td>
<td>6.0</td>
<td></td>
</tr>
<tr>
<td>Weight &gt;45 kg above ideal</td>
<td>5</td>
<td>90</td>
<td>2.8 ± 0.3</td>
<td>2.7</td>
<td></td>
</tr>
</tbody>
</table>

* = The physiologic characteristics of the patients in the references were considered normal for size, except where indicated.
† = Assumes a weight = 10 kg.
‡ = Assumes a $Q_{s}/Q_{t}$ = 0.1 (because of smoking history).
for a healthy 10-kg child compared with a healthy 70-
kg adult is largely the result of a relatively high oxygen
consumption and low alveolar volume with respect to
weight. The much shorter time to hemoglobin desatura-
tion for the morbidly obese patient compared with a
healthy 70-kg adult is largely a result of a much de-
creased alveolar volume with respect to weight. The
moderately ill patient (20% decreased alveolar volume,
20% increased oxygen consumption, 10% shunt, 20% 
decreased cardiac output, and 30% decreased hemoglo-
bin concentration) has nearly as short a time to hemo-
globin desaturation as an obese 127-kg adult and a
healthy 10-kg child.

Time to Functional Recovery

To compare the hemoglobin desaturation data with
rates of recovery from succinylcholine, it is first neces-
sary to examine published data on recovery. Recovery
may be defined as return to any one of various percent
of the control single twitch height of the adductor pol-
licis muscle after ulner nerve stimulation. From six ref-
ences, the mean times (range of mean times) to 10%,
50%, and 90% recovery of the control single twitch
height from 1 mg/kg or 40 mg/m² intravenous succinyl-
choline are 6.8 (5.6–7.2), 8.5 (7.8–10.1), and 10.2
(9.3–12.1) min.11–16 The slope of recovery from 10%
to 90% twitch height for succinylcholine is steep, and
the mean ± SD (range) in the previously noted six
studies11–16 was 24.8 ± 2.2%/min (20.0–28.7%/min).
The time to 50% recovery (8.5 min), which is similar for
adults and pediatric patients,15 should permit adequate
spontaneous ventilation with FiO₂ = 1.0 if the airway
was patent.13,17 we define time to 50% recovery as the
time to functional recovery and will use this time for
comparison with hemoglobin desaturation data.

Comparison of Time to Critical Hemoglobin
Desaturation with Time to Functional
Recovery

The mean time to 50% of control single twitch height
(functional recovery) was 8.5 min; for the best case
example of a healthy 70-kg adult (i.e., longest time to
hemoglobin desaturation) SaO₂ equals 83% at 8.5 min
(fig. 1); half of this population will be more and half
will be less hypoxicemic at this time. Beyond 9 min,
the SaO₂ rapidly approaches zero for the healthy 70-kg
group of patients. Use of any greater time for functional
recovery (e.g., return to 75% or 90% of control twitch
height) results in predictions of much greater degrees
of hypoxemia and periods of danger. With a mean func-
tional recovery (50% of control twitch height) time of
8.5 min, all other types of patients in figure 1 (10-kg
child, obese 127-kg adult, and moderately ill 70-kg pa-
ient) are either profoundly hypoxicemic or, in all likeli-
hood, dead at this time.

Clinical Implications of Comparison of
Time to Functional Recovery Versus
Hemoglobin Desaturation

These findings have several important implications for
the clinical treatment of a patient with a difficult airway.
First, because in a given patient it is unknown how
quickly succinylcholine will be metabolized, our anal-
ysis shows that the recommendation that the patient be
allowed to awaken (achieve functional recovery) can
only be logically pursued if there is some level of V̇e.
Thus, if V̇e = 0 (i.e., a complete "cannot ventilate/can-
not intubate" situation), then a rescue option should be
pursued immediately (e.g., insert laryngeal mask airway,
Combitube (Sheridan Corp., Argyle, NY), institute tran-
stracheal jet ventilation, surgical airway). Second, this
analysis ignored the central respiratory depressant ef-
effects of all concomitantly administered general anesthesi-
( which, of course, should be present). Therefore,
from this point of view, this analysis should be regarded
as an underestimation of the time to functional recovery
and the period of danger. The underestimation of the
time to functional recovery and the period of danger
strengthens the first two conclusions immediately
above. Third, if a healthy 70-kg patient were to suffer
either a decreased cardiac output, decreased initial alve-
olar volume, decreased hemoglobin concentration, or
an increase in oxygen consumption after 1 mg/kg intra-
venous succinylcholine, then the rate of arterial hemo-
globin desaturation would even be greater than shown
in figure 1. Consequently, from this point of view, the
analysis also may be an underestimation of the negative
difference between time to desaturation versus time to
functional recovery. Finally, our analysis only included
global physiologic variables that impacted on time to
hemoglobin desaturation and did not consider regional
pathologic factors, such as critical vessel stenoses, that
could increase the duration and intensity of the danger
for the region.

In summary, this analysis shows that in the large ma-

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iority of patients with 1 mg/kg intravenous succinylcholine-induced apnea, significant-to-life-threatening hemoglobin desaturation will occur before functional recovery. However, the intensity and duration of danger may have been underestimated by this analysis because of the assumptions that alveolar denitrogenation is complete, that functional recovery includes return of a patent airway, that concomitantly administered anesthetics may effectively delay functional recovery, that physiologic variables may change in a way that decreases the time to hemoglobin desaturation, and that regional pathologic variables may increase the degree of danger. Consequently, if $V_e$ is thought to be 0, or near 0, achievement of functional recovery before significant desaturation is not a realistic possibility, and a rescue option should be instituted aggressively and early. In most patients, the risk–benefit analysis will favor insertion of an laryngeal mask airway or Combitube before instituting transtracheal jet ventilation or performing a surgical airway.18

References


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