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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 (FAO2) and the minute ventilation (V_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 $\mathring{V}_E = 0$ occurs before the time to functional recovery for various patients receiving 1 mg/kg of intravenous succinylcholine.

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Time to Significant Hemoglobin Desaturation

The time to hemoglobin desaturation is derived from the apnea ($\mathring{V}_E = 0$) model of Farmery and Roe² 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 FAO2 and FACO2] 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 $S_pO_2 = 90\%$. For example, when the endpoint for measuring desaturation versus time curves in children is defined as SpO2 = 90%, there is as great as a 30% incidence of overshoot to $S_PO_2 \le 70\%$ and a 50% incidence of overshoot to a S_PO₂ between 71% and 80%.³ During the time required to reach the various levels of hemoglobin desaturation, it is assumed that the airway is nonpatent (a reason why $\dot{V}_E = 0$) 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, $S_aO_2 = 80\%$ is reached after 8.7, 5.5, 3.7, and 3.1 min, respectively, and $S_aO_2 = 60\%$ is reached at 9.9, 6.2, 4.3, and 3.8 min, respectively. Critical hemoglobin desaturation is defined as $S_PO_2 \le 80\%$ and decreasing; for the patients shown in figure 1, at $S_PO_2 \le 80\%$ 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_PO₂ (89-91%) agrees reasonably well with actual data from patients whose weight and degree of normalcy and preoxygenation are reliably known.³⁻⁹ The most probable reason the model slightly overpredicts the apnea time to a specific oxyhemoglo-

TIME TO HEMOGLOBIN DESATURATION WITH INITIAL $F_AO_2 = 0.87$

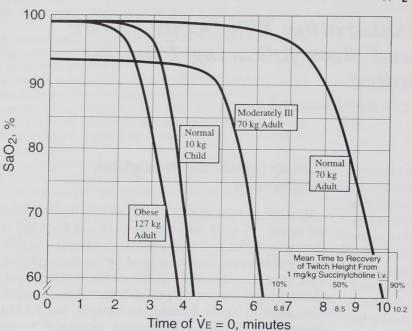


Fig. 1. SaO₂ versus time of apnea for various types of patients. The physiologic characteristics of these patients can be obtained from the author on request. The SaO2 versus time curves were produced by the computer apnea model of reference 2. The mean times to recovery from 1 mg/kg intravenous succinylcholine (lower right hand corner) are taken from references 11-16.

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_pO_2 \ in \ Actual \ Patients \ under \ General \ An esthesia \ Compared \ to \ Predicted \ Time \ from \ Model \ An esthesia \ Compared \ to \ Predicted \ Time \ from \ Model \ An esthesia \ Compared \ to \ Predicted \ Time \ from \ Model \ An esthesia \ Compared \ to \ Predicted \ Time \ from \ Model \ An esthesia \ Compared \ to \ Predicted \ Time \ from \ Model \ An esthesia \ Compared \ to \ Predicted \ Time \ from \ Model \ An esthesia \ Compared \ to \ Predicted \ Time \ from \ Model \ An esthesia \ Compared \ to \ Predicted \ Time \ from \ Model \ An esthesia \ Compared \ to \ Predicted \ Time \ from \ Model \ An esthesia \ Compared \ to \ Predicted \ Time \ from \ Model \ An esthesia \ Compared \ to \ Predicted \ Time \ from \ Model \ An esthesia \ Compared \ to \ Predicted \ Time \ from \ Model \ An esthesia \ Compared \ to \ Predicted \ Time \ from \ Model \ An esthesia \ Compared \ to \ Predicted \ Time \ from \ Model \ Time \$ of Farmery and Roe(2)

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

^{* =} The physiologic characteristics of the patients in the references were considered normal for size, except where indicated.

^{† =} Assumes a weight = 10 kg.

^{‡ =} Assumes a Qs/Qt = 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 desaturation for the morbidly obese patient compared with a healthy 70-kg adult is largely a result of a much decreased 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 hemoglobin concentration) has nearly as short a time to hemoglobin 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 necessary 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 pollicis muscle after ulner nerve stimulation. From six references, 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 succinvlcholine 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 $24.8 \pm 2.2\%$ /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 $FiO_2 = 1.0$ 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 hypoxemic 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 functional 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 patient) are either profoundly hypoxemic or, in all likelihood, 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 analysis 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 \mathring{V}_{E} . Thus, if $\mathring{V}_E = 0$ (i.e., a complete "cannot ventilate/cannot intubate" situation), then a rescue option should be pursued immediately (e.g., insert laryngeal mask airway, Combitube (Sheridan Corp., Argyle, NY), institute transtracheal jet ventilation, surgical airway). Second, this analysis ignored the central respiratory depressant effects of all concomitantly administered general anesthetics (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 alveolar volume, decreased hemoglobin concentration, or an increase in oxygen consumption after 1 mg/kg intravenous succinylcholine, then the rate of arterial hemoglobin 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-

jority 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 \mathring{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 larvngeal mask airway or Combitube before instituting transtracheal jet ventilation or performing a surgical airway. 18

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