REVIEW

Preoxygenation and general anesthesia: a review

G. BOUROCHE, J. L. BOURGAIN

Service d'Anesthésie Gustave Roussy, Villejuif, France

ABSTRACT

Because intubation can potentially become a lengthy procedure, the risk of arterial oxygen (O_2) desaturation during intubation must be considered. Preoxygenation should be routine, as oxygen reserves are not always sufficient to cover the duration of intubation. Three minutes of spontaneous breathing at FiO_2=1 allows denitrogenation with FAO_2 close to 95% in patients with normal lung function. Tolerable apnea time, defined as the delay until the SpO_2 reaches 90%, can be extended up to almost 10 minutes after 3 minutes of classic preoxygenation. Eight deep breaths within 60 seconds allow a comparable increase in O_2 reserves. For effectiveness, the equipment must be adapted and tightly fitted. Inadequate preoxygenation (FeO_2 <90% after three minutes tidal volume breathing) is frequently observed. Predictive risk factors for inadequate pre-oxygenation share overlap with criteria predictive of difficult mask ventilation. In cases of respiratory failure, oxygenation can be improved by positive end expiration pressure or by pressure support. In morbidly obese patients, preoxygenation is enhanced in a seated position (25°) and by use of positive pressure ventilation. O_2 can also be administered during the intubation procedure; techniques include pharyngeal O_2, special oxygen mask, or even pressure support ventilation for patients with spontaneous ventilation or positive pressure ventilation to the facial mask for apneic patients. Clinicians (especially anesthesiologists trained in ENT and traumatology) must be prepared to handle life-threatening emergency situations by alternate methods including trans-tracheal ventilation. The availability of equipment and training are two essential components of adequate preparation. *(Minerva Anestesiol 2015;81:910-20)*

Key words: Intubation, intratracheal - Cell respiration - Laryngeal masks - Ventilation - Anesthesia.

Preservation of oxygenation during intubation is essential because lack of control of O_2 intake can cause life-threatening complications. Anesthetic induction usually leads to apnea, when tissue oxygenation is maintained by consumption of the oxygen reserve and by continuous administration of O_2. In the majority of cases, rigorous preoxygenation and face mask ventilation are provided until muscle relaxation is sufficient to facilitate intubation in good conditions. In some cases oxygenation cannot be maintained, either due to pulmonary disease or to mask ventilation or intubation difficulties. These critical situations can often be foreseen and avoided by preparation for alternative methods of oxygenation following a validated algorithm.[1]

Pathophysiology of oxygenation

During anesthesia, oxygenation primarily depends on three parameters: alveolar ventilation (VA), distribution of ventilation/perfusion ratio, and consumption of O_2 (VO_2).

Oxygen reserves

During apnea, tissue oxygenation is maintained at the expense of the body's O_2 reserves,[2] which are very low in quantity and are mainly situated in lungs, plasma, and hemoglobin. Upon breathing ambient air, the lung O_2 reserve is calculated as follows, for a functional residual capacity (FRC) of 3000 mL: 0.21x3000=630 mL. After complete pre-oxygenation, the alveo-

lar fraction of O_2 (FAO_2) is close to 0.95 and the reserve increases as follows: 0.95x3000=2850 mL. These theoretical figures are maximal values; the FAO_2 is lower in practice because the ventilation/perfusion ratio is heterogeneous.

The plasma O_2 reserve of a subject breathing in ambient air (PaO_2=80 mmHg) with a plasma volume of 3 L is calculated as 0.003x80x3x10=7 mL. At a PaO_2 of 500 mmHg, this plasma reserve amounts to 45 mL. The hemoglobin O_2 reserve is calculated as follows for a concentration of hemoglobin of 12 g. 100 mL^{-1} and a total blood volume of 5 L: 1.34x0.98x12x10x5=788 mL in ambient air (saturation=98%). The value increases to 804 mL with a FiO_2 of 1 (saturation=100%). In cases of anemia, hyperoxic ventilation increases the utilizable O2 by augmenting the dissolved O_2.[3]

Considering the three main physiological O_2 reserves, the total O_2 reserve is about 1450 mL when breathing in ambient air and it rises to approximately 3700 mL when breathing pure O_2. This increase (approximately 2250 mL) is mainly due to the rise FAO_2 in FRC. The theoretical values were confirmed experimentally by measurement of oxygen uptake breath-by-breath in healthy volunteers during preoxygenation. The mean expired fraction of O_2 (FEO_2) after 3 minutes of breathing O_2 was 0.92±0.01, and the mean additional oxygen taken up was 2.23±0.85 L. This value closely agrees with the physiological model.[4]

Several factors influence O_2 availability: the initial rise in $PaCO_2$ (Haldane effect), FRC, FAO_2, fraction of shunt, VO_2, hemoglobin concentration, and cardiac output. Replacement of nitrogen by O_2 in the lung reservoir during preoxygenation obeys an exponential law.[2] The change in O_2 reserve over time is linear in both blood and tissue compartments.

O_2 consumption

The O_2 consumption of an awake subject is about 300 mL per min and it falls about 15% in the elderly. After ventilation in ambient air, O_2 reserves allow, at maximum, 3 minutes of apnea without serious impact on O_2 transport. This time can be doubled by correctly performed preoxygenation. The duration of apnea tolerated is additionally decreased if O_2 reserves are low due to decreased FRC, low PAO_2, and/or high VO_2.

Ventilation/perfusion mismatch

Preoxygenation leads to increased shunt and micro-atelectasis after anesthetic induction.[5] High FiO_2 is not the only mechanism responsible because atelectasis has also been observed when a FiO_2 0.4 is used.[6] The use of a FiO_2 of 0.8 does not prevent the appearance of microatelectasis, and it results in a considerably shortened margin of time before unacceptable desaturation compared with the use of 100% oxygen.[7] Microatelectasis are reversible by application of an alveolar recruitment maneuver (tracheal pressure >30 cm H_2O for 15 seconds) and they can be prevented by the addition of a positive end expiration pressure (PEEP) of 10 cm H_2O.[8] In morbidly obese patients and in parturients, shunt can exceed 20% and even increasing FiO_2 to 1 does not provide correction of the hypoxemia. Implementation of a microatelectasis prevention strategy of alveolar recruitment maneuvers and PEEP limits the extent in elderly [9] and obese patients.[10]

Epidemiology of arterial desaturation during induction and intubation

Anesthetic induction

Before upper airway control, arterial O_2 desaturation occurs when the O_2 reserves are insufficient to support the O_2 consumption during the apnea period. There are three mechanisms responsible (Figure 1): quantitative decrease in reserves (decrease in FRC, impairment of gas exchange), increase in VO_2 (parturient, fever), and prolonged apnea. Four high-risk situations deserve special mention:

— rapid induction sequence in which mask ventilation increases the risk of inhalation of gastric fluid (although this has never been demonstrated to occur);

— predicted difficulty with face mask ventilation;

— predicted difficulty with intubation due

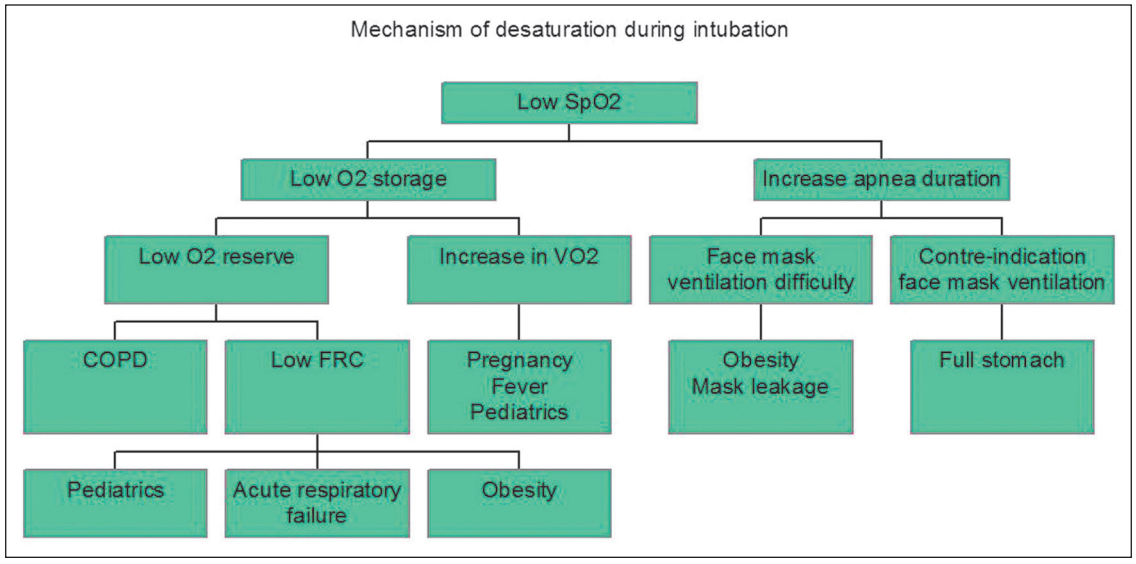

Figure 1.—Mechanism of arterial desaturation in O_2 during anesthetic induction.

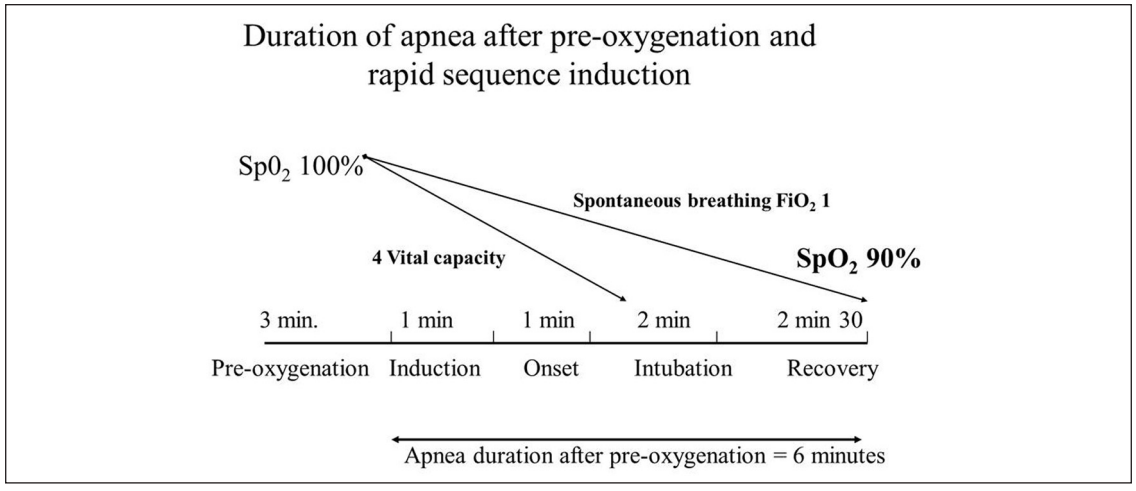

Figure 2.—Duration of apnea after pre-oxygenation and rapid induction sequence.
The durations in minutes were estimated from the literature for a rapid sequence induction data and have shown in timeline with pre-oxygenation conditions and duration of apnea up to SpO_2<90%. In some cases the apnea time will be less than the duration of action of anesthetic agents and that an alternative method for oxygenation will become necessary.

to anatomical abnormality or specific technical considerations (*e.g.*, double lumen tube);

— obesity or pregnancy.

After rapid sequence induction, the resumption of spontaneous ventilation does not occur fast enough to allow recovery after a failed intubation procedure, and saturation falls below 90% in 11% of patients (Figure 2).[11] After induction by propofol (2 mg.kg^{-1}) and fentanyl (2 µg.kg^{-1}), the administration of succinylcholine (0.56 mg.kg^{-1} and1 mg.kg^{-1}) increases the risk of desaturation and apnea duration compared to placebo.[12] In a pharmacodynamic study of succinylcholine (from 0.3 to 1 mg.kg^{-1}), intubation conditions were found to be excellent at dosages above 0.5 mg.kg^{-1} (Table I), but the delay in resumption of spontaneous breathing rose from 4.0 to 6.16 minutes after administration

Table I.—*Period of apnea and onset of desaturation after rapid sequence induction. Naguib used propofol and fentanyl and Heier thiopental 5mg.kg-1.*

		Placebo	Succinylcholine 0.65 mg.kg-1	Succinylcholine 1 mg.kg-1
% patients with desaturation	Naguib 2005 12	45%	65%	85%
	Heier 2001 70			42%
Times mean±SD in minutes to spontaneous of diaphragmatic movements	Naguib 2005 12	2.7±1.2	4.8±2.5	4.7±1.3

of 0.6 and 1 mg.kg^{-1}, respectively.[13] Reversal of profound high-dose rocuronium-induced neuromuscular block used for rapid sequence induction (1.2 mg/kg) with sugammadex (16 mg/kg) was significantly faster than spontaneous recovery from succinylcholine (1 mg/kg): 6.2±1.8 minutes versus 10.9±2.4 minutes respectively.[14] Rapid sequence induction with rocuronium followed by reversal with sugammadex allowed earlier re-establishment of spontaneous ventilation than with succinylcholine (216 seconds versus 406 seconds respectively).[15] Thus, the choice of the rocuronium would increase the margin of safety for a resumption of spontaneous ventilation after a rapid sequence induction.

Anesthesia and spontaneous breathing

Outside of pediatrics, no study has evaluated the risk factors for desaturation during induction with spontaneous ventilation. For most hypnotics, alveolar hypoventilation exhibits a dose-dependent effect.[16] In the absence of sedation, airway local anesthesia decreases inspiratory flows for a duration of approximately 45 minutes.[17]

Desaturation in pediatrics

Desaturation episodes occur commonly in children, with a frequency of 4-10% during induction and 20% during tracheal intubation.[18] Desaturation occurs much more rapidly when the child is young,[19, 20] and the duration of apnea before desaturation is linearly correlated with the age of the patient. The less the child weighs, the higher the incidence of severe arterial desaturation after reinstitution of manual ventilation with 100% oxygen. It has been suggested that a SpO_2 of 95% might be the safe limit for apnea during induction of pediatric anesthesia.[21] Infection of the upper respiratory tract is noted to increase the risk of desaturation during induction.[18]

Resuscitation and prehospital emergency

Variability exists among published incidences of desaturation in various studies [22, 23] but it is reported to reach 60% during prehospital intubation.[22] In emergency medicine, desaturation occurs frequently, even in patients who are not difficult to intubate and for whom the intubation process is relatively rapid. Decreased FRC related to lung pathology (pulmonary edema, pneumonia, pulmonary contusion) is a determining factor. Pulmonary aspiration and esophageal intubation are responsible for some cases of severe desaturation during the intubation process.[24] In a prospective study, preoxygenation was found to be effective (achieved PaO_2>100 mmHg) in 7 of 8 cases in which the indication was the protection of the airway (coma) and in 5 of 34 cases (15%) in which intubation was indicated due to respiratory or cardiac failure.[23]

Preoxygenation

In cases in which there is a potential risk of desaturation before securing the airway by endotracheal intubation, pre-oxygenation is highly recommended during induction of anesthesia. In its absence, the risk of desaturation is increased.

Preoxygenation techniques

The equipment must be adapted and tightly fitted to the patient, particularly the face mask. A morphological mismatch between the mask and the face of the patient (e.g., inappropriate mask size, presence of beards or moustaches)

Table II.—*Comparison of the different techniques of pre-oxygenation in normal subjects.*

Study	N.	Endpoint	Preoxygenation technique used				
			TVB	4 DB 30s	8 DB 60s	TVB with PEEP	AI + PEEP
Gambee AM [29]	12	DAWD to 90% min	8.9±10*	6.8±1.8			
Fleureaux O [33]	17	DAWD to 95% min	3±1*	1.87±0.99			
		Pa O_2 mmHg	397±49*	293±86			
Baraka AS [34]	24	DAWD to 95% min	3.73±0.76	2.78±0.39	5.21±0.96*		
Herriger A [30]	40	DAWD to 90% min	9.98±2.25*			7.83±2.63	
Gold MI [71]	22	Pa O_2 mmHg	350.4±35.8	339±33.9			
Rooney MJ [32]	24	FeO_2 %	91.9±3	90.8±3			
Nimmagadda U [72]	24	FeO_2 %	88±5*	80±5	87±3*		
Pandit JJ [4]	5	FeO_2 %	92±1*	83±9	91±4*		
Gagnon C [73]	20	FeO_2 %	89±3*	76±7			
Tanoubi I [36]	20	FeO_2 %	89±6				94±4*

TVB: tidal volume breathing; DB: deep breaths; DAWD: duration of apnea without desaturation.

prevents a perfect seal and can cause failure.[25] The mask must be applied securely on the face of the patient; 20% dilution of O_2 by ambient air occurs when the mask is not tightly applied, and 40% dilution occurs when it is held close to the face.[26] The circle system with fresh gas flow (5 L.min^{-1}) is used as the standard for comparison in anesthesia studies evaluating the effectiveness of different circuits because it allows higher inspiratory flow rates.[27] Some open circuit (Bain or Magill) systems have been shown to be much less effective.[27] Before preoxygenation, the circuit and the reservoir should be filled with O_2. Three preoxygenation techniques are used: spontaneous breathing at FiO_2 of 1 for 2 to 5 minutes, the "four vital capacities" method, and deep breaths (Table II).

Spontaneous breathing at FiO_2 of 1

The following technique of pre-oxygenation that was initially proposed by Hamilton in 1955 is still the reference standard: 3 minutes of spontaneous breathing at FiO_2 of 1. In patients with normal lung function, this provides denitrogenation with an FAO_2 of close to 95%. The denitrogenation is effective from the first minute of the preoxygenation; nevertheless, circuit leakage cancels these effects by a rapid decrease of the FiO_2.[28] Breathing pure O_2 for longer than a minute appears to have little benefit in terms of SpO_2 or denitrogenation alveolar, but positively influences the duration of apnea before arterial desaturation.[29] In experiments performed with healthy subjects, apnea time (with the exception of an insufflation to verify tracheal intubation) that is maintained until the SpO_2 reaches over 90%, can be extended to almost 10 minutes after 3 minutes of classic pre-oxygenation. The apnea time can be increased by an additional two minutes by application of positive pressure during the preoxygenation and by ventilation to the mask after induction.[30]

Vital capacity maneuvers

The four vital capacities method is used in cases in which patient cooperation is lacking. The duration of apnea without desaturation is shorter after four capacities maneuvers than with spontaneous breathing. Technical requirements are responsible for the limitations of this technic: bag capacity, inspiratory flow and room gas inspiration. They are partly resolved by the addition of an additional 2 liter bag and a non-rebreathing "Ambu" valve. The vital capacity maneuver preferably begins with a forced expiration to optimize the elevation of FeO_2.[31] To be fully effective, the inspiratory O_2 flow should be greater than the peak inspiratory flow, which is attained by activating the O_2 system "by-pass" during inspiration; 4 or 5 forced breaths of pure O_2 were found to be as efficient as conventional pre-oxygenation assessed on the FeO_2.[32] However, these results were not confirmed when PaO_2 was used for comparison; PaO_2 was observed to

be lower after the four vital capacity maneuver (293±86 mmHg) than after spontaneous ventilation in pure O_2 (397±48 mmHg) (Table II).[33]

Deep breathing method

Eight deep breaths within 60 seconds at an oxygen flow of 10 L per min constitutes a simple method of preoxygenation. This technique results in a mean arterial oxygen tension of 369±69 mmHg, which is not significantly different from the value achieved by 3 minutes of tidal volume breathing at an oxygen flow of 5 L per minute.[34] The voluntary hyperventilation technique (1 minute at FiO_2 1 followed by 2 minutes of voluntary hyperventilation) has been proposed to prevent post-apneic hypercapnia. $PaCO_2$ after intubation was similar, compared to a control, when either hyperventilation before induction or 3 min normal breathing was used as the pre-oxygenation technique.[35]

Pressure support ventilation

In healthy volunteers, PSV has been shown to improve the quality of pre-oxygenation by two mechanisms: acceleration of nitrogen washout and better contact between the mask and the face. In a healthy volunteer study,[36] FEO_2 after 3 minutes of preoxygenation was higher ($p<0.001$) with PSV 4 cm H_2O/PEEP 4 (94±3%) and PSV 6 cm H_2O/PEEP 4 (94±4%) than with the standard technique (89±6%). One hundred percent and 90% of the participants reached 90% FEO_2 with PSV 4 and 6 cmH_20 respectively *vs.* 65% with spontaneous breathing at FiO_2 of 1 ($P=0.0013$). Clinical tolerance was impaired at the highest level of pressure tested.

Preoxygenation failure

Inadequate preoxygenation, defined as an $FeO_2<90\%$ after three minutes of tidal volume breathing, is seen frequently in practice (56% in a sample of 1050 patients).[37] The effective FiO_2 delivered was observed to be lower in patients with a $FeO_2<90\%$. Risk factors for inadequate preoxygenation were determined to be bearded male, beardless male, ASA>1, lack of teeth, and age >55 years. These predictive factors overlap with those previously associated with difficult mask ventilation.

While SpO_2 measurement is not informative regarding the quality of pre-oxygenation maneuvers, it is essential to identify oxygenation problems. The FeO_2 depends on the tidal volume; small tidal volumes increase the difference between FeO_2 and FAO_2, leading to overestimation of FAO_2. The CO_2 wave shape is informative regarding the quality of the ventilation and the tightness of the circuit. A FeO_2 of <90% indicates incomplete denitrogenation at the FRC level. In a study of 40 volunteers,[25] 9 subjects were unable to attain $FeO_2>90\%$. Even if the mechanisms that cause incomplete denitrogenation are not identified, this monitoring method has utility in routine practice.[2] If the FeO_2 cannot be increased above 90%, PSV may be proposed to improve preoxygenation quality.[38] In emergency medicine, the monitoring of preoxygenation is typically based on SpO_2 measurement and pre-oxygenation duration, as the FeO_2 is not usually available.

Morbidly obese patients

In the obese, the decrease in FRC, increase in O_2 consumption, and heterogeneity of the ventilation/perfusion (V/Q) ratio result in a decrease in the time required for alveolar denitrogenation [39, 40] and a decrease in O_2 stores, which in turn reduce the duration of apnea tolerance.

After 3 minutes of classic preoxygenation, obese patients can tolerate apnea of 3 minutes duration while maintaining SpO_2 higher than 90%, and the time required to increase saturation above 96% after desaturation is 37 seconds, which is longer than the 22 seconds required in healthy subjects.[40] Pulmonary abnormalities are correlated to Body Mass Index and are responsible for early desaturation, before complete muscle relaxation and intubation.[39] The effectiveness of spontaneous ventilation and eight deep breaths as preoxygenation methods are comparable in the obese when regarding Fe O_2 and the duration of apnea before the SpO_2 reaches 95%.[41] Continuous Positive Airway Pressure (CPAP) (7.5 cm H_2O versus Mapleson

circuit) during spontaneous ventilation in pure O_2 were observed not to improve the duration of apnea (240 and 203 seconds CPAP versus zero end expiratory pressure, respectively).[42] When CPAP (10 cm H2O) is followed by a PSV with PEEP, post-intubation PaO_2 is significantly improved.[43]

Because lung volume reduction is the main factor causing impaired oxygenation in the obese patient, use of PSV with PEEP has been proposed. PSV improves the quality of preoxygenation in the obese, probably due to improved alveolar ventilation and recruitment.[44] Compared to five minutes of spontaneous ventilation with FiO_2 of 1, PSV results in increased FeO_2 (96.9%±1.3% *vs.* 94.1%±2.0%) and acceleration of nitrogen elimination (185.3±46.1 *vs.* 221±41.5 s).[45] However, this gain was not associated with an increase in the duration of tolerable apnea, defined by a 95% SpO_2 cutoff value. This is due to the small increase in O_2 reserve, which can be estimated at 58 mL O_2 for a 2000 mL FRC (2000x2, 8%=58 mL). When associated with a recruitment maneuver, the effectiveness of PSV is statistically significant regarding the arterial oxygenation.[46] In morbidly obese patients, CPAP of 5 cm H_2O combined with PSV of 5 cm H_2O during preoxygenation resulted in better oxygenation compared with neutral-pressure breathing, and it prevented desaturation episodes.[47] The postintubation PaO_2 was significantly higher in the CPAP/PSV group (32.2±4.1 kPa) than in the control group (23.8±8.8 kPa) (P<0.001). In the control group, the nadir of oxygen saturation was lower (median 98%, range 83-99%) than in the CPAP/PSV group. The PaO_2 is greater and the time of apnea extended when preoxygenation (either CPAP for 3 minutes [48] or 8 deep breaths) [49] is performed in a 25° head-up position compared to a supine position. After preoxygenation in the head-up position, the time to a SpO_2 of lower than 92% was always measured at longer than 3 minutes.

Preoxygenation in pregnancy

In the parturient, the time required for complete denitrogenation (FeN_2=2%) is shorter than in non-parturient young women as follows: 104±30 seconds between 13-26 weeks of pregnancy, 80±20 seconds between 26-42 weeks, and 130±30 seconds in controls, due to the reduction in the FRC during pregnancy.[50] Spontaneous ventilation in FiO_2 of 1 for 3 minutes and the four vital capacities method for 30 seconds give comparable results, whether judged by PaO_2 [51] or apnea duration.[52] Some women were observed to have a tolerable apnea duration of only approximately 60 seconds; this short delay carries obvious risk.[52] The shortening of the time to FeO_2 of 90% (average 107 seconds) is a good argument supporting recommendation of the 8 deep breaths technique during obstetric emergencies.[53]

Preoxygenation and chronic obstructive pulmonary disease

In patients with chronic obstructive pulmonary disease, the time needed to decrease the alveolar fraction of nitrogen (FAN_2) from 78% to 2% can exceed 30 minutes, as it is inversely proportional to the peak expiratory flow rate.[54] $FetO_2$ monitoring is used to evaluate the time necessary for preoxygenation in such patients.[55]

Preoxygenation in pediatrics

In pediatrics, the respiratory physiology of young children is particularly age-specific. The inhibition of intercostal tonus by general anesthesia is responsible for a reduction in FRC. Hypoxemia arises more quickly in children because of a higher VA/FRC ratio, a higher O_2 consumption, and lower O_2 reserves. Children exhibit a delay before reaching FeO_2 close to 90% of approximately 80 to 90 seconds when breathing at FiO_2 of 1.[56] While young children show a rapid drop in saturation, they also reach a FeO_2 of 0.9 more quickly.[56] After a period of at least 2 minutes breathing at FiO_2 of 1 and after muscle paralysis, the duration of apnea before the SpO_2 reaches 90% is found to be 96.5 seconds in children less than 6 months of age, 160.4 seconds in 2- to 5-year-olds, and 382.4 seconds in 11- to 18-year-olds.[57] In children younger than 6 months, even shorter apnea time limits, on the order of 70-90 seconds, have

been reported.[19] The duration of apnea required to reach a SpO_2 of 98%, 95%, or 90%, is significantly increased when the preoxygenation is extended for 1 to 2 minutes, but no benefit was found by extension past 3 minutes.[21] When the gas mixture used during pre-oxygenation passes from an average FiO_2 of approximately 93% to 39%, the duration of apnea until a 95% SpO_2 decreases from 210 to 71 seconds.[58]

Intensive care patients and emergency medicine

In emergency medicine, all of the patients can be considered to be at risk for desaturation during airway control and thus preoxygenation should be recommended as part of routine practice. In emergency nonsurgical intubation, preoxygenation is difficult to achieve. The benefit of the pre-oxygenation is probably greater in patients who do not have respiratory illness at the time of intubation.[23] Thus, all patients who are intubated for neurological distress should benefit from a careful preoxygenation of at least 3 minutes in duration, even if a lack of patient cooperation limits its effectiveness. During intubation of hypoxemic patients, pre-oxygenation using PSV is more effective at reducing arterial desaturation than the usual method.[59] At the end of the preoxygenation period, SpO_2 was higher in the PSV group than in the control group (98±2 *vs*. 93±6%, P<0.001). During the intubation procedure, lower SpO_2 values were observed in the control group (81±15 *vs*. 93±8%, P<0.001). Twelve (46%) of the patients in the control group and two (7%) in the PSV group had a SpO_2 below 80% (P<0.01).

Apneic oxygenation

It is possible to maintain oxygenation during a long period of apnea by administering 10 to 15 L per min of continuous oxygen into the pharynx. However, this method is only effective following a complete preoxygenation. Apnea of longer than 30 minutes in duration has been reported to result in severe hypercapnia (>150 mmHg) without damage to the patient. Apneic oxygenation failures are related to failure of the preoxygenation procedure and to reduced FRC.[60] An indirect method of apneic oxygenation is the administration of O_2 during intubation attempts, and the administration of O_2 at a rate of 3 L per min by a naso- or oro-pharyngeal catheter can significantly delay the onset of arterial O_2 desaturation.[61] Similar results have been reported more recently in ASA 1-2 patients after preoxygenation. This method is easy to apply and it confers a definite advantage in patients without respiratory pathology[62] and probably in morbidly obese patients as well.[63]

Management of failures of preoxygenation and oxygenation

As the risk factors for pre-oxygenation failures and difficult mask ventilation are similar, such at-risk patients should be identified and carefully monitored. If FeO_2 is lower than 0.9 after preoxygenation, alternative methods of oxygenation should be immediately available. Knowing that none of techniques is 100% reliable, it is essential to be able to provide several methods. The equipment must be immediately available and the team must be familiar with its use. The most popular device is the intubating laryngeal mask airway (ILMA). The ventilation is of good quality in the vast majority of cases and oxygenation failures are rare when using this device.[64] However, only the No. 3 size exists for use with child patients, and little data are available about ILMA use in pediatrics.[65]. For patients of less than 30 kg, the standard laryngeal mask is used, with the awareness that implementation is more difficult and not always successful.

In the event of ventilation failure with the facial or laryngeal mask, rescue trans-tracheal oxygenation is to be considered. Inter-crico-thyroid membrane puncture is straightforward in 98% of non-emergency cases.[66] Because the use of transtracheal ventilation in emergency medicine is very rare, studies on this subject include only very few patients. The success rate of the emergency puncture procedure is unknown.[67]

Jet ventilation is administered using a manual injector with operator control or using a jet ventilator with control of the driving pressure. The major risk is the possibility of pulmonary barotrauma by lung overdistension, the impact

of which can be serious in this context.[68] It is important to closely monitor the quality of expiration and keep in mind that the outflow of a 14 gauge catheter to a driving pressure of 3 bars is approximately 600 mL per second.[69] With O_2 consumption being approximately 300 mL per minute, the injection duration and respiratory rate are limited to a minimum.

Conclusions

It is of particular importance to consider the issues related to oxygenation because O_2 reserves are low and the difficulties of intubating the patient and providing adequate ventilation are often associated. The situation becomes critical when O_2 reserves are insufficient. Efficient technique and FeO_2 monitoring can improve the effectiveness of the pre-oxygenation and thereby increase the margin of safety. After pre-oxygenation, supplemental O_2 increases the duration of tolerable apnea in most cases, and this very simple measure should not be neglected. Failures of pre-oxygenation must be identified and alternative methods of oxygenation should be available for rapid and facile implementation. To this end, these methods should be taught and practiced on models or during simulation courses, so teams are prepared if the need arises.

Key messages

— Effective preoxygenation (FeO_2 >90%) is essential to avoid hypoxemia during airway management.

— Preoxygenation can be improved by use of a seated position (20° to 30°), PSV, and/or PEEP, especially in obese patients.

— Some induction situations present higher risk: pregnancy, obesity, rapid induction sequence and require special attention.

— Those situations at risk may be anticipated by identifying risk factors.

References

1. Bourgain JL, Chastre J, Combes X, Orliaguet G. [Oxygen arterial desaturation and upholding the oxygenation during intubation: question 2. Societe Francaise d'Anesthesie et de Reanimation]. Ann Fr Anesth Reanim 2008;27:15-25.
2. Campbell IT, Beatty PC. Monitoring preoxygenation. Br J Anaesth 1994;72:3-4.
3. Lauscher P, Mirakaj V, Koenig K, Meier J. Why hyperoxia matters during acute anemia. Minerva Anestesiol 2013;79:643-51.
4. Pandit JJ, Duncan T, Robbins PA. Total oxygen uptake with two maximal breathing techniques and the tidal volume breathing technique: a physiologic study of preoxygenation. Anesthesiology 2003;99:841-6.
5. Reber A, Engberg G, Wegenius G, Hedenstierna G. Lung aeration. The effect of pre-oxygenation and hyperoxygenation during total intravenous anaesthesia. Anaesthesia 1996;51:733-7.
6. Serafini G, Cornara G, Cavalloro F, Mori A, Dore R, Marraro G *et al*. Pulmonary atelectasis during paediatric anaesthesia: CT scan evaluation and effect of positive endexpiratory pressure (PEEP). Paediatr Anaesth 1999;9:225-8.
7. Edmark L, Auner U, Enlund M, Ostberg E, Hedenstierna G. Oxygen concentration and characteristics of progressive atelectasis formation during anaesthesia. Acta Anaesthesiol Scand 2011;55:75-81.
8. Neumann P, Rothen HU, Berglund JE, Valtysson J, Magnusson A, Hedenstierna G. Positive end-expiratory pressure prevents atelectasis during general anaesthesia even in the presence of a high inspired oxygen concentration. Acta Anaesthesiol Scand 1999;43:295-301.
9. Tusman G, Bohm SH, Vazquez de Anda GF, do Campo JL, Lachmann B. 'Alveolar recruitment strategy' improves arterial oxygenation during general anaesthesia. Br J Anaesth 1999;82:8-13.
10. Pelosi P, Ravagnan I, Giurati G, Panigada M, Bottino N, Tredici S *et al*. Positive end-expiratory pressure improves respiratory function in obese but not in normal subjects during anesthesia and paralysis. Anesthesiology 1999;91:1221-31.
11. Hayes AH, Breslin DS, Mirakhur RK, Reid JE, O'Hare RA. Frequency of haemoglobin desaturation with the use of succinylcholine during rapid sequence induction of anaesthesia. Acta Anaesthesiol Scand 2001;45:746-9.
12. Naguib M, Samarkandi AH, Abdullah K, Riad W, Alharby SW. Succinylcholine dosage and apnea-induced hemoglobin desaturation in patients. Anesthesiology 2005;102:35-40.
13. El Orbany MI, Joseph NJ, Salem MR, Klowden AJ. The neuromuscular effects and tracheal intubation conditions after small doses of succinylcholine. Anesth Analg 2004;98:1680-5, table.
14. Lee C, Jahr JS, Candiotti KA, Warriner B, Zornow MH, Naguib M. Reversal of profound neuromuscular block by sugammadex administered three minutes after rocuronium: a comparison with spontaneous recovery from succinylcholine. Anesthesiology 2009;110:1020-5.
15. Sorensen MK, Bretlau C, Gätke MR, Sørensen AM, Rasmussen LS. Rapid sequence induction and intubation with rocuronium-sugammadex compared with succinylcholine: a randomized trial. Br J Anaesth 2012;108:682-9.
16. Lee YS. Effects of propofol target-controlled infusion on haemodynamic and respiratory changes with regard to safety. J Int Med Res 2004;32:19-24.
17. Liistro G, Stănescu DC, Veriter C, Rodenstein DO, D'Odemont JP. Upper airway anesthesia induces airflow limitation in awake humans. Am Rev Respir Dis 1992;146:581-5.
18. Rolf N, Cote CJ. Frequency and severity of desaturation events during general anesthesia in children with and without upper respiratory infections. J Clin Anesth 1992;4:200-3.
19. Dupeyrat A, Dubreuil M, Ecoffey C. Preoxygenation in children. Anesth Analg 1994;79:1027.
20. Xue FS, Luo LK, Tong SY, Liao X, Deng XM, An G. Study of the safe threshold of apneic period in children during anesthesia induction. J Clin Anesth 1996;8:568-74.

21. Xue F, Luo L, Tong S, Liao X, Tang G, Deng X. Children's development effecting blood oxygen desaturation following apnea. Chin Med J (Engl) 1995;108:434-7.
22. Dunford JV, Davis DP, Ochs M, Doney M, Hoyt DB. Incidence of transient hypoxia and pulse rate reactivity during paramedic rapid sequence intubation. Ann Emerg Med 2003;42:721-8.
23. Mort TC. Preoxygenation in critically ill patients requiring emergency tracheal intubation. Crit Care Med 2005;33:2672-5.
24. Mort TC. Esophageal intubation with indirect clinical tests during emergency tracheal intubation: a report on patient morbidity. J Clin Anesth 2005;17:255-62.
25. Berry CB, Myles PS. Preoxygenation in healthy volunteers: a graph of oxygen «washin» using end-tidal oxygraphy. Br J Anaesth 1994;72:116-8.
26. McGowan P, Skinner A. Preoxygenation--the importance of a good face mask seal. Br J Anaesth 1995;75:777-8.
27. Nimmagadda U, Salem MR, Joseph NJ, Lopez G, Megally M, Lang DJ *et al*. Efficacy of preoxygenation with tidal volume breathing. Comparison of breathing systems. Anesthesiology 2000;93:693-8.
28. Russell GN, Smith CL, Snowdon SL, Bryson TH. Preoxygenation and the parturient patient. Anaesthesia 1987;42:346-51.
29. Gambee AM, Hertzka RE, Fisher DM. Preoxygenation techniques: comparison of three minutes and four breaths. Anesth Analg 1987;66:468-70.
30. Herriger A, Frascarolo P, Spahn DR, Magnusson L. The effect of positive airway pressure during pre-oxygenation and induction of anaesthesia upon duration of non-hypoxic apnoea. Anaesthesia 2004;59:243-7.
31. McCrory JW, Matthews JN. Comparison of four methods of preoxygenation. Br J Anaesth 1990;64:571-6.
32. Rooney MJ. Pre-oxygenation: a comparison of two techniques using a Bain system. Anaesthesia 1994;49:629-32.
33. Fleureaux O, Estèbe JP, Bléry C, Douet N, Mallédant Y. [Effects of preoxygenation methods on the course of PaO2 and PaCO2 in anesthetic post-induction apnea]. Cah Anesthesiol 1995;43:367-70.
34. Baraka AS, Taha SK, Aouad MT, El-Khatib MF, Kawkabani NI. Preoxygenation: comparison of maximal breathing and tidal volume breathing techniques. Anesthesiology 1999;91:612-6.
35. Choinière A[1], Girard F, Boudreault D, Ruel M, Girard DC. Voluntary hyperventilation before a rapid-sequence induction of anesthesia does not decrease postintubation PaCO2. Anesth Analg 2001;93:1277-80.
36. Tanoubi I, Drolet P, Fortier LP, Donati F. [Inspiratory support versus spontaneous breathing during preoxygenation in healthy subjects. A randomized, double blind, cross-over trial]. Ann Fr Anesth Reanim 2010;29:198-203.
37. Baillard C, Depret F, Levy V, Boubaya M[3], Beloucif S. *et al*. Incidence and prediction of inadequate preoxygenation before induction of anaesthesia. Ann Fr Anesth Reanim 2014;33:e55-e58.
38. Tanoubi I, Drolet P, Donati F. Optimizing preoxygenation in adults. Can J Anaesth 2009;56:449-66.
39. Berthoud MC, Peacock JE, Reilly CS. Effectiveness of preoxygenation in morbidly obese patients. Br J Anaesth 1991;67:464-6.
40. Jense HG, Dubin SA, Silverstein PI, O'Leary-Escolas U. Effect of obesity on safe duration of apnea in anesthetized humans. Anesth Analg 1991;72:89-93.
41. Rapaport S, Joannes-Boyau O, Bazin R, Janvier G. Comparaison de la technique de préoxygénation à huit capacités vitales et à volume courant chez les patientes ayant une obésité morbide. Ann Fr Anesth Reanim 2004;23:1155-9.
42. Cressey DM, Berthoud MC, Reilly CS. Effectiveness of continuous positive airway pressure to enhance pre-oxygenation in morbidly obese women. Anaesthesia 2001;56:680-4.
43. Coussa M, Proietti S, Schnyder P, Frascarolo P, Suter M, Spahn D *et al*. Prevention of atelectasis formation during the induction of general anesthesia in morbidly obese patients. Anesth Analg 2004;98:1491-5, table.
44. Solis A, Baillard C. [Effectiveness of preoxygenation using the head-up position and noninvasive ventilation to reduce hypoxaemia during intubation]. Ann Fr Anesth Reanim 2008;27:490-4.
45. Delay JM, Sebbane M, Jung B, Nocca D, Verzilli D, Pouzeratte Y *et al*. The effectiveness of noninvasive positive pressure ventilation to enhance preoxygenation in morbidly obese patients: a randomized controlled study. Anesth Analg 2008;107:1707-13.
46. Futier E, Constantin JM, Pelosi P, Chanques G, Massone A, Petit A *et al*. Noninvasive ventilation and alveolar recruitment maneuver improve respiratory function during and after intubation of morbidly obese patients: a randomized controlled study. Anesthesiology 2011;114:1354-63.
47. Harbut P, Gozdzik W, Stjernfalt E, Marsk R, Hesselvik JF. Continuous positive airway pressure/pressure support preoxygenation of morbidly obese patients. Acta Anaesthesiol Scand 2014;58:675-80.
48. Dixon BJ, Dixon JB, Carden JR, Burn AJ, Schachter LM, Playfair JM *et al*. Preoxygenation is more effective in the 25 degrees head-up position than in the supine position in severely obese patients: a randomized controlled study. Anesthesiology 2005;102:1110-5.
49. Altermatt FR, Munoz HR, Delfino AE, Cortinez LI. Preoxygenation in the obese patient: effects of position on tolerance to apnoea. Br J Anaesth 2005;95:706-9.
50. Byrne F, Oduro-Dominah A, Kipling R. The effect of pregnancy on pulmonary nitrogen washout. A study of preoxygenation. Anaesthesia 1987;42:148-50.
51. Norris MC, Dewan DM. Preoxygenation for cesarean section: a comparison of two techniques. Anesthesiology 1985;62:827-9.
52. Bernard F, Louvard V, Cressy ML. Préoxygénation avant induction pour césarienne. Ann Fr Anesth Reanim 1994;13:2-5.
53. Chiron B., Laffon M, Ferrandiere M, Pittet JF, Marret H, Mercier C. Standard preoxygenation technique versus two rapid techniques in pregnant patients. Int J Obstet Anest 2005;14:79.
54. Selsy DS, Jones JG. Respiratory function and the safety of anesthaesia. In: Taylor TH, Major E, editors. Hazard and complications of anaesthesia. Second Edition. London, UK: Churchill Livingstone; 1995. p. 213-21.
55. Samain E, Biard M, Farah E, Holtzer S, Delefosse D, Marty J. Monitorage de la fraction expirée d'oxygène lors de la préoxygénation dans la bronchopathie chronique obstructive. Ann Fr Anesth Reanim 2002;21:14-9.
56. Morrison JE, Jr., Collier E, Friesen RH, Logan L. Preoxygenation before laryngoscopy in children: how long is enough? Paediatr Anaesth 1998;8:293-8.
57. Patel R, Lenczyk M, Hannallah RS, McGill WA. Age and the onset of desaturation in apnoeic children. Can J Anaesth 1994;41:771-4.
58. Kinouchi K, Fukumitsu K, Tashiro C, Takauchi Y, Ohashi Y, Nishida T. Duration of apnoea in anaesthetized children required for desaturation of haemoglobin to 95%: comparison of three different breathing gases. Paediatr Anaesth 1995;5:115-9.
59. Baillard C, Fosse JP, Sebbane M, Chanques G, Vincent F, Courouble P *et al*. Noninvasive ventilation improves preoxygenation before intubation of hypoxic patients. Am J Respir Crit Care Med 2006;174:171-7.
60. Fraioli RL, Sheffer LA, Steffenson JL. Pulmonary and cardiovascular effects of apneic oxygenation in man. Anesthesiology 1973;39:588-96.
61. Teller LE, Alexander CM, Frumin MJ, Gross JB. Pharyn-

geal insufflation of oxygen prevents arterial desaturation during apnea. Anesthesiology 1988;69:980-2.
62. Taha SK, Siddik-Sayyid SM, El-Khatib MF, Dagher CM, Hakki MA, Baraka AS. Nasopharyngeal oxygen insufflation following pre-oxygenation using the four deep breath technique. Anaesthesia 2006;61:427-30.
63. Ramachandran SK, Cosnowski A, Shanks A, Turner CR. Apneic oxygenation during prolonged laryngoscopy in obese patients: a randomized, controlled trial of nasal oxygen administration. J Clin Anesth 2010;22:164-8.
64. Ferson DZ, Rosenblatt WH, Johansen MJ, Osborn I, Ovassapian A. Use of the intubating LMA-Fastrach in 254 patients with difficult-to- manage airways. Anesthesiology 2001;95:1175-81.
65. Weiss M, Schwarz U, Dillier C, Fischer J, Gerber AC. Use of the intubating laryngeal mask in children: an evaluation using video-endoscopic monitoring. Eur J Anaesthesiol 2001;18:739-44.
66. Bourgain JL, Desruennes E, Fischler M, Ravussin P. Transtracheal high frequency jet ventilation for endoscopic airway surgery: a multicentre study. Br J Anaesth 2001;87:870-5.
67. Combes X, Jabre P, Margenet A, Merle JC, Leroux B, Dru M *et al*. Unanticipated difficult airway management in the prehospital emergency setting: prospective validation of an algorithm. Anesthesiology 2011;114:105-10.
68. Parmet JL, Colonna-Romano P, Horrow JC, Miller F, Gonzales J, Rosenberg H. The laryngeal mask airway reliably provides rescue ventilation in cases of unanticipated difficult tracheal intubation along with difficult mask ventilation. Anesth Analg 1998;87:661-5.
69. Bourgain JL, Chollet M, Fischler M, Gueret G, Mayne A; membres du conseil du club en anesthésie en ORL. [Guide for the use of jet-ventilation during ENT and oral surgery]. Ann Fr Anesth Reanim 2010;29:720-7.
70. Heier T, Feiner JR, Lin J, Brown R, Caldwell JE. Hemoglobin desaturation after succinylcholine-induced apnea: a study of the recovery of spontaneous ventilation in healthy volunteers. Anesthesiology 2001;94:754-9.
71. Gold MI, Duarte I, Muravchick S. Arterial oxygenation in conscious patients after 5 minutes and after 30 seconds of oxygen breathing. Anesth Analg 1981;60:313-5.
72. Nimmagadda U, Chiravuri SD, Salem MR, Joseph NJ, Wafai Y, Crystal GJ *et al*. Preoxygenation with tidal volume and deep breathing techniques: the impact of duration of breathing and fresh gas flow. Anesth Analg 2001;92:1337-41.
73. Gagnon C, Fortier LP, Donati F. When a leak is unavoidable, preoxygenation is equally ineffective with vital capacity or tidal volume breathing. Can J Anaesth 2006;53:86-91.

Conflicts of interest.—The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

Received on October 28, 2014. - Accepted for publication on June 3, 2015. - Epub ahead of print on June 5, 2015.

Corresponding author: J. Bouroche, Département d'Anesthésie, Gustave Roussy Cancer Campus Grand-Paris, 114 rue Edouard Vaillant, 94800 Villejuif. E-mail: jean-louis.bourgain@gustaveroussy.fr