
Oxygen Administration in the Emergency Department: Choosing the Appropriate Dosage and the Technology

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Abstract

Oxygen (O₂), like any drug, should be administered only when indicated, and at the appropriate dose. The indications for O₂ administration are to relieve global or regional hypoxia and to provide therapeutic hyperoxia. In treating hypoxia, one must first rule out low inspired O₂ concentrations (FIO₂). The causes of hypoxia can then be categorized into hypoventilation, which responds to small increases in FIO₂, and increased venous admixture (“shunt”), which requires maximal FIO₂. FIO₂ up to about 0.4 can be administered via common plastic face masks and Venturi masks. Masks with O₂ reservoirs can increase the FIO₂ to about 0.7 at best. Masks with sequential gas delivery can provide FIO₂s exceeding 0.9 and are best suited to provide therapeutic hyperoxia and treat diseases characterized by excess “shunt”.

MeSH Words: oxygen mask; inspired oxygen concentration; non-rebreathing mask; hypoxia; peak inspiratory flow; oxygen therapy, sequential gas delivery

Introduction

Oxygen (O₂), one of the oldest, most commonly prescribed and most effective drugs in our pharmacopoeia, is paradoxically the most inappropriately administered drug in the hospital: either the wrong dose is prescribed, or the prescribed dose is inadvertently not administered (1). For prescribing O₂, the term analogous to ‘mg/kg by a given rout’, is ‘O₂ flow for a given minute ventilation by a given mask’.

The purpose of this review is to outline the principles for determining the appropriate dose of O₂ to prescribe and the means of reliably administering the prescribed dose.

Indications for O₂ administration

The commonest and unequivocal indication for administration of O₂ is to relieve global hypoxia. Oxygen is also frequently administered in an attempt to minimize hypoxia or ischemia in specific vascular beds such as the heart (angina, myocardial infarction), brain (ischemia, stroke), or placenta (fetal distress). Blood O₂ concentration is only one of the determinants of the O₂ concentration in the tissues. Other important determinants are those controlling regional blood flow, such as perfusion pressure and vascular resistance (2). An example of therapeutic hyperoxia is in the treatment of

carbon monoxide poisoning to accelerate its elimination.

In most cases, global hypoxia is adequately and readily treated by moderate supplementation of inspired O₂ concentrations using commonly available masks. However, great difficulty is often encountered in recruiting the technology to provide high FiO₂ when it is required. Failure to provide an FiO₂ that brings the patient's arterial PO₂ to an "acceptable" level causes the level of care to advance to the ultimate default treatment of endotracheal intubation and mechanical ventilation.

In this review, we will briefly survey the causes and treatment of global hypoxia, then focus on the issue that is generally least well managed in the Emergency Department -- the mechanics of providing the highest FiO₂ to spontaneously breathing patients.

Causes of hypoxia

Hypoxia can result from low FiO₂ (Table 1), low alveolar ventilation (Table 2), or high venous admixture ("shunt") (#3, Table 3). The O₂ prescription must be appropriately matched to the cause of the hypoxia.

Table 1: Causes for low inspired FiO₂

Breathing circuit present?	Cause of hypoxia
No	environmental (e.g. high altitude)
Yes	wrong gas entering circuit (e.g. N ₂ O, N ₂ , or CO ₂ instead of O ₂)
	inadequate concentration of O ₂ in inspired gas
	inadequate O ₂ or air flow into a rebreathing circuit (i.e. O ₂ consumption exceeds total O ₂ entering circuit)

Table 2: Causes for low alveolar ventilation

Type	Cause
Neurological: Central nervous system depression	drugs (sedatives, narcotics, hypnotics)
	Trauma
	tumors (malignant, benign, swelling and others)
	vascular (ischemic, hemorrhagic, permeability and others)
	intrinsic toxins (organ failure e.g. kidney, liver)
	extrinsic toxins (e.g. ingested, inhaled)
Neurological: Peripheral nervous system depression	Spinal cord to neuro-muscular junction. Many etiologies.
Muscular	myopathy, dystrophy, fatigue and others
Musculo-skeletal	deformities, fractures and others
Chest wall	obesity, increased intra-abdominal pressure and others
Pleura	hemo- pneumo- hydro- thorax, fibrosis, tumors and others
Conducting airways	upper and lower airway obstruction
Lungs	emphysema, atelectasis, foreign body, edema, fibrosis, tumors and others

Table 3: Partial list of indications for high FiO₂.

Condition	Type	Etiology and details
1. Respiratory distress		etiology not yet diagnosed
2. Low perfusion states	Global	shock, hypotension
	Regional	brain (stroke)
		heart (myocardial ischemia or infarction)
		limb ischemia (atheromatous occlusion, embolism)
		microcirculatory insufficiency
		placental insufficiency or fetal distress
3. Shunt (Venous admixture)	Pulmonary	Atelectasis
		Pneumonia
		pleural effusion
		aspiration
	Cardiac	right to left shunt (congenital or acquired)
4. Decreased O ₂ carrying capacity of the blood	Anemia	multiple etiologies including acute hemorrhage
	Hemoglobinopathy	carboxyhemoglobin, methemoglobin
5. Therapeutic hyperoxia	Prophylaxis	infection, nausea, vomiting
	Radiotherapy	sensitization of tumors to radiotherapy
	Carbon monoxide poisoning	increased rate of elimination
	Hypermetabolic states	malignant hyperpyrexia
		thyroid storm
		seizures

Table 4: Relationship between O₂ flow, entrainment ratio and mask O₂ concentration of Venturi mask.

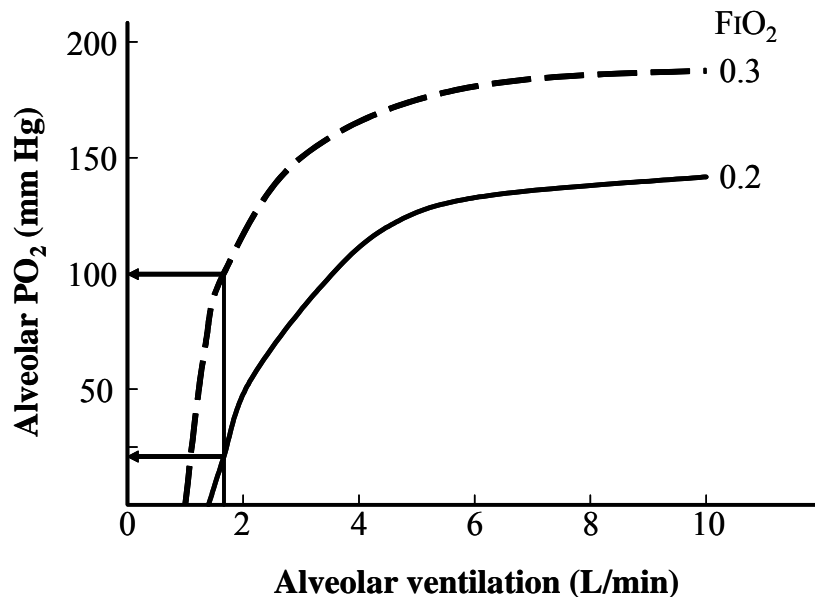
Mask O ₂ concentration (%)	O ₂ flow (L/min)	Entrainment ratio (air:O ₂)	Air flow (L/min)	Total flow (L/min)
24	4	20:1	80	84
28	6	10:1	60	66
40	8	3:1	24	32
60	8	1:1	8	16
100	8	0:1	0	8

Oxygen prescription

Treatment for low FiO₂

The treatment of low FiO₂ requires the rapid identification of the cause and taking appropriate corresponding action. Once a low FiO₂ is excluded as the cause of hypoxia, it is important to determine the cause as either 'hypoventilation' or 'shunt', as the O₂ prescription differs significantly.

Figure 1



a) Variable performance masks

Treatment for, or prophylaxis against, hypoxia due to hypoventilation is usually relegated to the ubiquitous nasal prongs or "standard" green mask, both of which do no more than blow O₂ at the face. As the patient inhales, the O₂ flowing into the mask is diluted by entrained air before entering the mouth and nose (3). Assuming the total flow of O₂ enters the patient's lungs during inhalation, the net FiO₂ is not determined by the particular mask used, but by the peak inspiratory flow (PIF). For example, if the PIF is 50 L/min and the O₂ flow is 8 L/min, the FiO₂ at the PIF will be about 0.34 ((8 + (42 x 0.21)) L O₂ flow/min in a total inspiratory flow of 50 L/min).

Treatment for hypoventilation

Figure 1 illustrates the effect of alveolar ventilation on alveolar PO₂. Note that the very steep part of the slope of the curve at low alveolar ventilations implies that a small increase in FiO₂ (in this case from 0.21 to 0.3) markedly increases alveolar PO₂. Treatment of hypoventilation, therefore, is "FiO₂ responsive" and can be achieved with either of the two types of O₂ masks discussed below.

As the patient's PIF increases, for example, as a result of breathlessness or agitation, the FiO₂ approaches that of room air. As the PIF falls (for example, as a result of sedation or obtundation), FiO₂ approaches 1.0. Variable O₂ delivery devices are effective when treating hypoxia due to hypoventilation because the net FiO₂ rises as respiratory depression gets worse—a sort of self-regulatory protective mechanism.

Variable performance masks, therefore, are good safeguards for respiratory depression -- unless the respiratory depression is thought to be due to a reduction in hypoxic respiratory drive (4). In that case there is a concern about the development of a vicious circle--hyperoxia

resulting in respiratory depression, causing more hyperoxia-- that can end in respiratory arrest. The concern over abolishing hypoxic respiratory drive led to the development of fixed performance masks (3;5).

b) Fixed performance masks

To maintain a fixed FiO₂ as PIF falls, the inhaled O₂ concentration must be independent of the PIF. For this to occur, O₂ must enter the mask at a fixed concentration and at a flow exceeding the PIF. In that case, as PIF falls the FiO₂ will approach the concentration of O₂ being delivered (Figure 2).

Figure 2

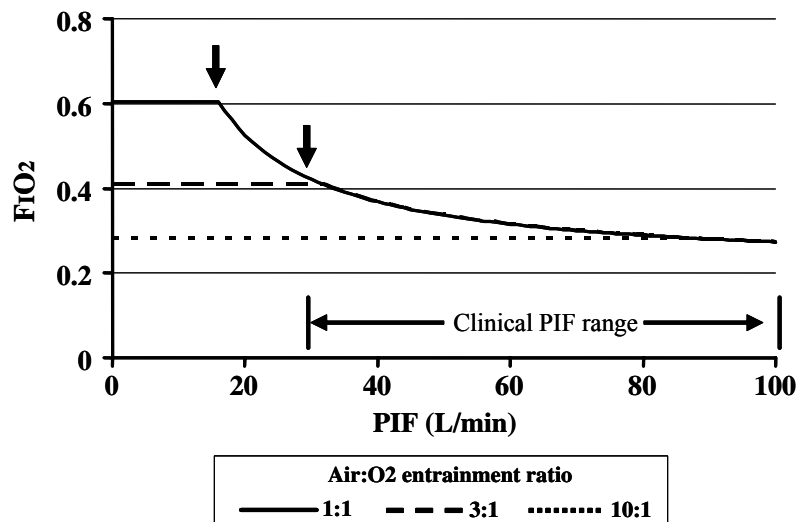
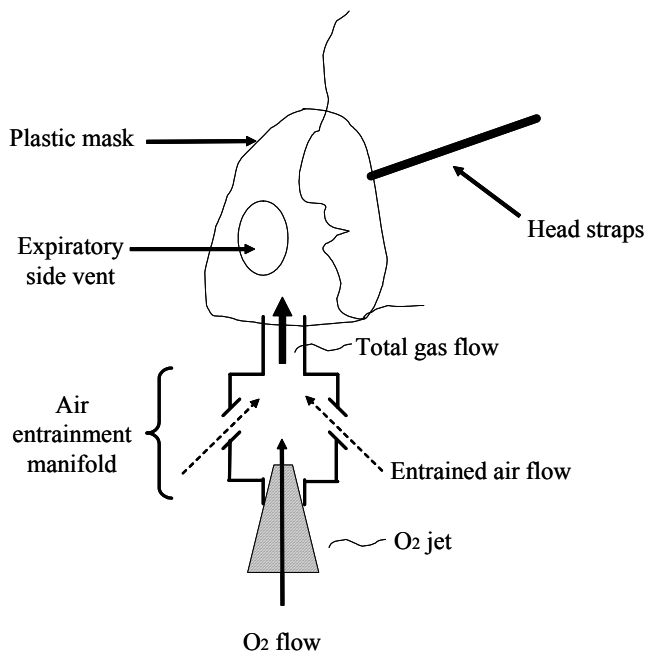


Figure 3



The Venturi mask (Figure 3) was designed precisely for this purpose (3,5). Oxygen and air enter the mask via a manifold. The O₂ emanating from its source at a given flow is forced through a restricted opening into the manifold, thus increasing its velocity as it leaves the nozzle. The high velocity of the O₂ causes a reduction in lateral pressure in the manifold by the Bernoulli Principle, and increases the tendency of outside air to be sucked into the manifold. The opening of the manifold to the outside can then be of different apertures that restrict the entrainment of air to a fixed ratio of the O₂ flow. The resulting mixture then floods the mask that cups the face. The mask contains side vents for elimination of excess gas flow and expired gases and that act as safety relief valves during inspiration if the PIF exceeds the net gas flow.

Manipulation of O₂ flow and aperture size will result in different combinations of total gas flow and O₂ concentrations. At low set O₂ concentrations, the air entrainment ratios are high and vice versa (Table 4). As PIF falls below the net flow into the mask, FiO₂ becomes limited to the concentration of O₂ entering the mask (Figure 2).

Note, that the “fixed performance” label does not apply to Venturi masks at minute ventilations where PIF exceeds net gas flow (6-8). At this point, Venturi masks become “variable performance” devices in which the *FiO₂ is no different than with the other “variable performance” devices* (right of arrows in Figure 2).

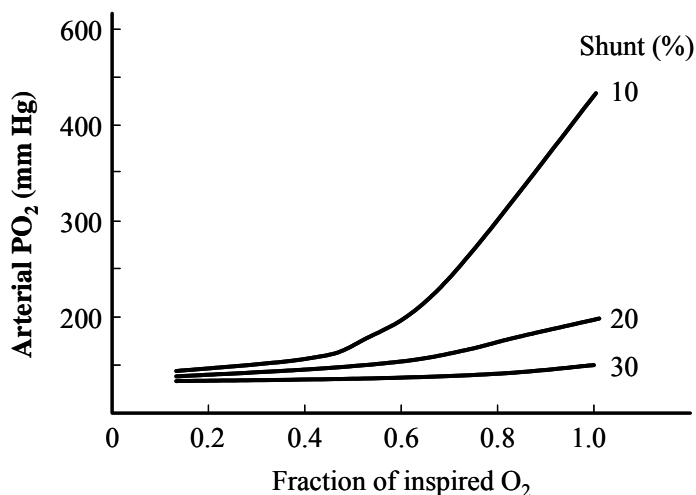
We will now turn our attention to the issues related to providing high FiO₂.

Administration of high FiO₂

The most common conditions requiring high FiO₂—often as near as possible to 1.0—are due to increases in shunt fraction (see #3 of Table 3). In the presence of large ventilation-to-perfusion mismatch, the arterial PO₂ is relatively unresponsive to increases in FiO₂ (Figure 4) (9). In such cases, there is little point in initiating treatment with a variable performance mask with a view to “titrating up” the FiO₂, if required. The highest FiO₂ attainable should be applied initially, and later, if necessary, the FiO₂ can be “titrated down”.

We will now address the advantages and limitations of the available technology for providing high FiO₂.

Figure 4



a) *Variable performance masks*

To attain an FiO₂ of 1.0 using “variable performance” masks (including the Venturi masks when PIF exceeds the net flow), the net flow would have to equal the PIF. This is no trivial matter, as hospital O₂ flow controllers frequently have maximum flows of about 25 L/min. Attempting to meet a PIF of just 50 L/min requires tandem setups from two O₂ sources flowing into a single mask. This is uncomfortable for the patient who is exposed to large volume of absolutely dry gas and a distressing noise level. Even so, tandem set ups may be inadequate as breathless patients often have PIF much higher than 50 L/min. Furthermore, high O₂ flows are very wasteful which may be an important issue when O₂

supplies are limited such as during patient transport or during disasters.

b) *Non-rebreathing mask*

The non-rebreathing O₂ mask is designed to meet the PIF with O₂ from an O₂ reservoir that empties into the mask on inhalation. This reservoir fills with O₂ during exhalation while a low resistance one-way valve prevents exhaled gas from entering the reservoir (Figure 5). However, during inhalation, the reduced pressure in the mask not only draws the O₂ from the reservoir but also draws outside air through the mask’s side vents. The O₂ and entrained air mix in the mask, reducing the net FiO₂. It is this obligatory entrainment of outside air that limits the FiO₂ with the non-rebreathing O₂ masks to approximately 0.6 (10).

Figure 5



Attempts to reduce the entrainment of outside air have not resulted in significant increases in FiO₂. For example, placing a flap covering one of the set of side vents still leaves the other side vent open for air entrainment (11) (Both side vents cannot be closed as one is required as a safety relief valve in case the minute ventilation exceeds the total O₂ flow.) Another way to restrict the flow of entrained air is to attach 'whiskers' (short lengths of corrugated tubing) to the side vents on the mask to retain small volumes of previously expired gas (12). Since, with O₂ supplementation, expired gas usually contains a higher concentration of O₂ than air, there is a greater O₂ concentration in the mask when expired air is entrained. These measures may increase the FiO₂ slightly but may also increase the CO₂ load and work of breathing.

c) *Sequential gas delivery (SGD) mask: Hi-Ox⁸⁰*

Sequential gas delivery (SGD) is a recent concept in O₂ administration (10;13;14). The Hi-Ox⁸⁰ (Viasys Healthcare Yorba Linda CA, USA) is a SGD mask and is similar to the non-rebreathing mask except that there are no side vents (Figure 6). As a result, the O₂ in the reservoir is inhaled without dilution. If the reservoir is depleted prior to the completion of the breath, a one-way valve—which has heretofore remained closed—opens, allowing the balance of the breath to be taken from atmosphere.

Figure 6

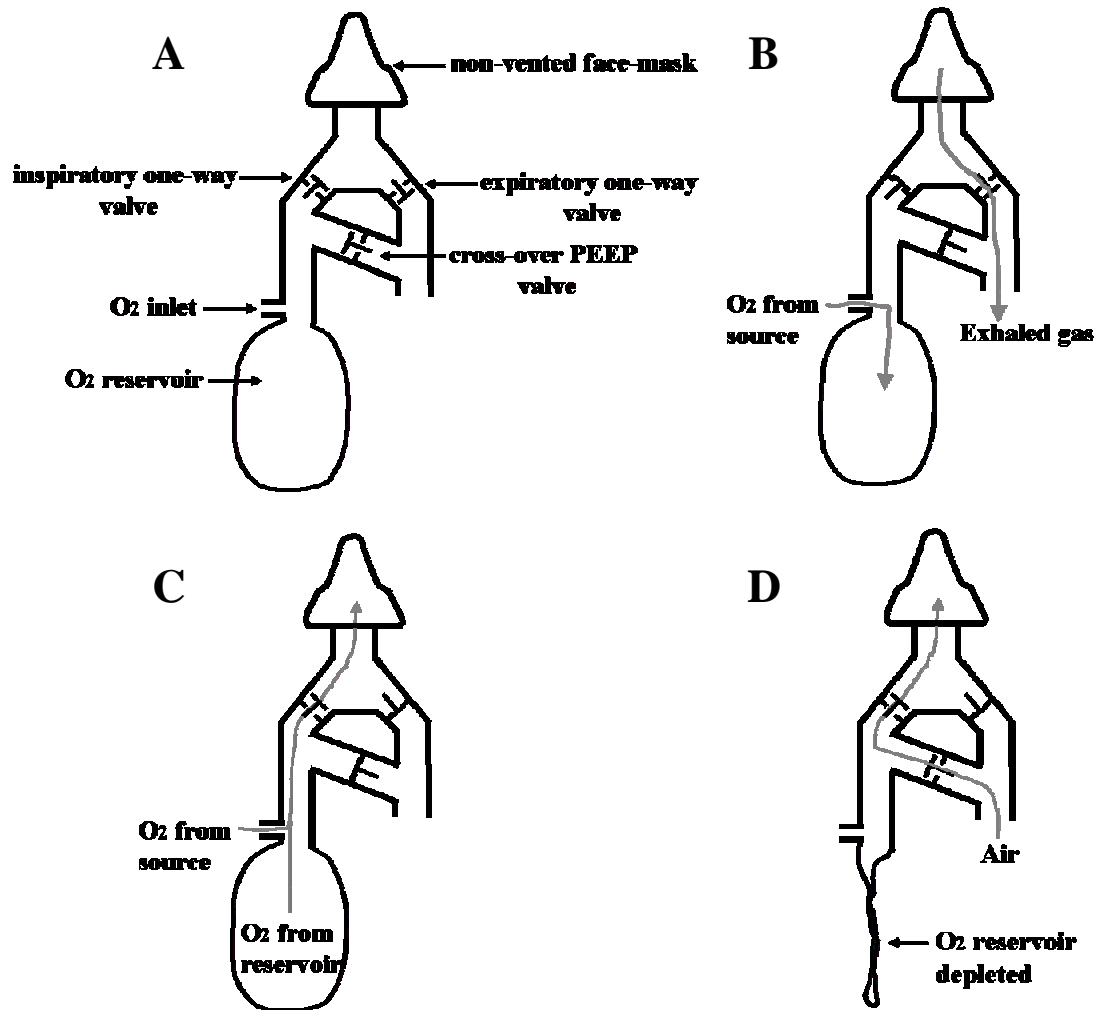
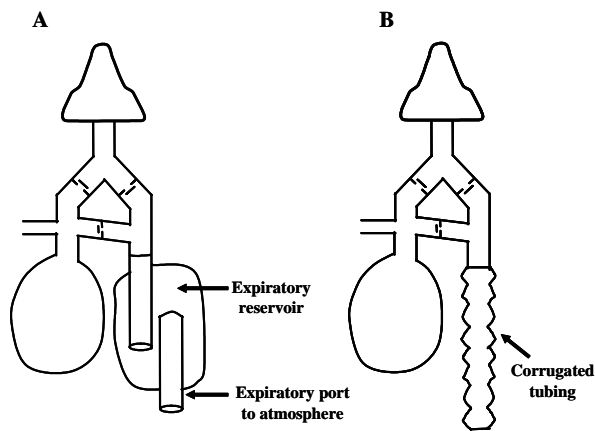


Figure 7

The Hi-Ox⁸⁰ provides a number of advantages that can be exploited. The FiO₂ is no longer determined by the relationship between O₂ flow and PIF, but by O₂ flow and minute ventilation. As opposed to “variable performance” and Venturi masks, there is an element of efficiency built into the system that reduces the dilution effect of the air. If the minute ventilation exceeds O₂ flow, all of the O₂ enters the alveoli undiluted but the air in the balance of the breath is distributed between the anatomical deadspace and the alveoli (10). Only the air that enters the alveoli dilutes the O₂ and reduces the net FiO₂. Assuming that anatomical deadspace is 2 ml/kg (15), SGD would reduce the dilution of the inspired O₂ by 150 ml of air on each breath in a “textbook 75 kg patient”. In a recent study in healthy volunteers, the Hi-Ox⁸⁰ provided an FiO₂ greater than or equal to that provided by the non-rebreathing mask at minute ventilations of up to 30 L/min at only half the O₂ flow (10). This efficiency of the Hi-Ox⁸⁰ can be useful in conserving O₂ when supplies are limited (i.e. during ambulance transport, man-made and natural disasters and combat casualty care).

Since the face mask of the Hi-Ox⁸⁰ contains no vents, all gases enter and leave the mask via the manifold. This may become useful if it is desirable to isolate the patient’s breath by placing a microbacterial filter on its expiratory limb (16).

d) *Hi-Ox⁸⁰ with a gas reservoir on expiratory port*

The efficiency of the Hi-Ox⁸⁰ mask can be significantly increased at high minute ventilations by attaching an open-ended reservoir to its expiratory port (Figure 7). The reservoir can be as simple as a length of corrugated tubing. If exhaled gas instead of air is available to make up the balance of the breath when minute ventilation exceeds O₂ flow, the FiO₂ becomes self regulating at 1.0. This is because with increasing ventilation at constant O₂ extraction, there is less O₂ extracted from each breath, so the O₂ concentration in expired gas approaches the inspired concentration--1.0.

Note that gas retained in the expiratory reservoir is also high in CO₂. However, this gas is only inhaled after all of O₂ in the inspiratory reservoir has been depleted. The O₂ flow therefore need only be equal to the alveolar ventilation required to maintain a given PCO₂. In a patient who is markedly hyperventilating, an O₂ flow of 10-12 L/min would provide an FiO₂ of 1.0 yet maintain a significant safety margin with respect to PCO₂- certainly below 40 mmHg (13). A Hi-Ox⁸⁰ with expiratory reservoir should also provide significant savings in O₂ when high FiO₂ is required and O₂ supplies are limited.

Afterword:

The performance of all abovementioned devices is affected by appropriate mask fitting. This is especially important with the devices intended to

deliver high F_IO₂, such as the non-rebreathing and the Hi-Ox⁸⁰ masks, in which any air leaking around the edges of the mask will dilute the inhaled O₂ and reduce the F_IO₂. Emergency Room staff should consider using adhesive tape (e.g., Tegaderm, 3M Health Care, St Paul, MN,

USA) to improve the seal around the mask when a high F_IO₂ is required.

Appendix:

What is F_IO₂?

Is it simply the highest O₂ concentration measured from a sample port in the mouth or nasopharynx? This cannot be correct because the O₂ concentration varies throughout inspiration, so the highest value doesn't necessarily represent the *net* F_IO₂. The net F_IO₂ is actually a flow-compensated average concentration. To measure this, one would have to simultaneously measure inspiratory flow and O₂ concentration. This is difficult to do in patients wearing O₂ masks. A simpler and more accurate method is to calculate it from end-tidal PO₂ and PCO₂. To make the calculation, each end-tidal partial pressure is divided by barometric pressure minus water vapour pressure to obtain end-tidal FO₂ (F_{ET}O₂) and FCO₂ (F_{ET}CO₂); one then uses the alveolar gas equation to calculate the F_IO₂: (17)

$$F_{I}O_{2} = \frac{(F_{ET}O_{2} \times RQ + F_{A}CO_{2})}{(RQ + F_{A}CO_{2} \times (1 - RQ))}$$

where F_{ET}CO₂ can be substituted for fractional concentration of alveolar CO₂ (F_ACO₂) and the respiratory quotient (RQ) can be assumed to be 0.8 (17).

Figure Legends:

Figure 1: The effect alveolar ventilation on alveolar PO₂ during marked hypoventilation. In this illustration, alveolar PO₂ reduced to dangerously low levels by hypoventilation (lower arrow) is improved to normal levels (upper arrow) by a small increase in the inspired concentration of O₂ from 0.21 to 0.3. Although this figure represents a particular case of oxygen consumption and shunt fraction, it illustrates the sensitivity of alveolar PO₂ to increases in inspired concentrations of O₂ when hypoxia is

due to hypoventilation. (Modified from Nunn (18) with permission of the publisher).

Figure 2: Predicted F_IO₂ as a function of peak inspiratory flow (PIF) for O₂ flow of 8 L/min to a Venturi mask with air:O₂ entrainment of 10:1, 3:1 and 1:1. At PIF less than the net gas flow into the mask, the F_IO₂ is limited by the concentration of O₂ in the net flow entering the mask. When PIF exceeds the net flow entering the mask (indicated by arrows), F_IO₂ becomes a function of PIF. Note that at PIF greater than about 30 L/min, the F_IO₂ is independent of the entrainment ratio and changing from a "40% Venturi" to a "60% Venturi" will not affect the F_IO₂. Note also, that the F_IO₂ for all variable performance masks would follow the heavy line (60% Venturi) and at low PIF would follow the curve to intersect the vertical axis at 1.0.

Figure 3: Schematic of a Venturi mask. Plastic mask with side vents for expiration is connected to a manifold. O₂ is directed into the manifold by an O₂ jet that increases O₂ flow velocity creating negative pressure inside the manifold according to Bernoulli principle. As a result, air is entrained into the manifold through small apertures at a fixed ratio to O₂ flow. Total gas flow into the mask is equal to the sum of O₂ flow and the entrained air flow.

Figure 4: The effect of changes in inspired O₂ concentration on arterial PO₂ in the presence of "shunt". Although this figure represents a particular case of alveolar ventilation, hemoglobin concentration and arterio-venous O₂ difference, it illustrates the insensitivity of arterial PO₂ to increases in inspired concentrations of O₂ when a large shunt fraction is present. (Modified from Nunn (18) with permission of the publisher).

Figure 5: Typical non-rebreathing O₂ mask. Plastic mask with side vents for expiration is connected to a manifold containing O₂ inlet, gas reservoir and a one-way inspiratory valve (drawn, superimposed on photograph). One of the side vents (outlined in black) is covered with a flap that prevents air flow into the mask during inspiration through that vent only. O₂ reservoir ensures that enough O₂ is present to meet peak inspiratory flows.

Figure 6: Schematic and function of the Hi-Ox⁸⁰ mask. A valved manifold is connected to an O₂

reservoir and a non-vented mask (panel A). During expiration, all of exhaled gas is directed down the expiratory limb by a pair of low resistance one-way valves; meanwhile, O₂ from the source collects in the reservoir (panel B). At the start of inhalation, O₂ is drawn from the source and the reservoir (panel C). If minute ventilation exceeds O₂ flow, the reservoir empties creating negative pressure in inspiratory limb. This opens the cross-over valve that has

slightly higher opening pressure than the other two valves. The balance of the breath is made up of air flowing via a cross-over limb (panel D). In this way, O₂ and air enter the lung sequentially: first O₂, then air.

Figure 7: Modification of the Hi-Ox⁸⁰ mask by adding an exhaled gas reservoir (Panel A) and a length of corrugated tubing (Panel B).

References:

1. Oxygen behind the mask [editorial]. *Lancet* 1982; 2(8309):1197-1198.
2. Iscoe S, Fisher JA. Hyperoxia-induced hypocapnia: an underappreciated risk. *Chest* 2005; 128(1):430-433.
3. Campbell EJ. A method of controlled oxygen administration which reduces the risk of carbon-dioxide retention. *Lancet* 1960; 2:12-14.
4. Dunn WF, Nelson SB, Hubmayr RD. Oxygen-induced hypercarbia in obstructive pulmonary disease. *Am Rev Respir Dis* 1991; 144:526-530.
5. Campbell EJ, Gebbie T. Masks and tent for providing controlled oxygen concentration. *Lancet* 1966; 1(7435):468-469.
6. Goldstein RS, Young J, Rebeck AS. Effect of breathing pattern on oxygen concentration received from standard face masks. *Lancet* 1982; 2(8309):1188-1190.
7. Jones JG. The 'fixed' performance venturi: effect of downstream pressure on outflow and FIO₂. *Anaesthesia* 2004; 59:704-709.
8. Jones HA, Turner SL, Hughes JMB. Performance of a large-reservoir oxygen mask (Ventimask). *Lancet* 1984; 1(8392):1427-1431.
9. Lawler PG, Nunn JF. A reassessment of the validity of the iso-shunt graph. *Br J Anaesth* 1984; 56(12):1325-1335.
10. Slessarev M, Somogyi R, Preiss D, Vesely A, Sasano H, Fisher J. Efficiency of O₂ administration: sequential gas delivery vs. 'flow into a cone' methods. *Critical Care Medicine*. In press.
11. Boumphrey SM, Morris EA, Kinsella SM. 100% inspired oxygen from a Hudson mask—a realistic goal? *Resuscitation* 2003; 57(1):69-72.
12. Hnatiuk OW, Moores LK, Thompson JC, Jones MD. Delivery of high concentrations of inspired oxygen via Tusk mask. *Critical Care Medicine* 1998; 26(6):1032-1035.
13. Somogyi RB, Vesely AE, Preiss DP, Prisman E, Volgyesi G, Azami T et al. Precise control of end-tidal PCO₂ using sequential rebreathing circuits. *Anesthesia and Intensive Care* 2005; in press.
14. Somogyi R, Preiss D, Vesely A, Prisman E, Tesler J, Volgyesi G et al. Case Report: Behind the mask. *Respiratory Therapy* 2002.
15. Comroe JH. The lung, clinical physiology and pulmonary function tests. Chicago: Year Book Publishers, 1956.
16. Somogyi R, Vesely AE, Azami T, Preiss D, Fisher J, Correia J et al. Dispersal of respiratory droplets with open vs closed oxygen delivery masks: implications for the transmission of severe acute respiratory syndrome. *Chest* 2004; 125(3):1155-1157.
17. Waldau T, Larsen VH, Bonde J. Evaluation of five oxygen delivery devices in spontaneously breathing subjects by oxygraphy. *Anaesthesia* 1998; 53(3):256-263
18. Nunn JF. *Applied Respiratory Physiology with special reference to anaesthesia*. 4th Edition ed. London: Butterworth-Heinemann, 1993.

Competing Interests: Joseph Fisher and Marat Slessarev are members of the team that develops and researches sequential gas delivery systems. The team has applied for patents on their

technology, some of which have been licensed to Viasys Healthcare of Yorba Linda California, the manufacturers and marketers of Hi-Ox⁸⁰.

This manuscript has been peer reviewed

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