Membrane Oxygenators: A Few Observations

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Abstract

A review of our experience with membrane oxygenators in the clinical setting was conducted in an attempt to identify the advantages and disadvantages of a membrane system. The increased complexity of set up, priming and operation of the membrane oxygenator may be offset by their safety features and their ability to generate reproducible blood gas data.

Introduction

Since there are a number of new membrane oxygenators (M.O.‘s) entering the marketplace, a perfusionist, unacquainted with M.O.‘s, should be interested in the characteristics of the two M.O.‘s enjoying wide use today, namely the Travenol Membrane Oxygenator (TMO) and the Sci-Med Kolobow Lung (SMKL). Historically, there has always been debate over the necessity of using M.O.‘s with most of the clamor coming from the membrane community about cellular trauma and protein denaturation. Meanwhile, the manufacturers of bubble oxygenators (B.O.‘s) calmly go about the business of capturing and maintaining 85% of the market.

Theoretically, M.O.‘s reduce protein denaturation and cellular damage by the elimination of the blood gas interface.1-13 Protein denaturation may result in such pathologic sequelae as: intravascular red cell aggregation, central nervous system dysfunction, alterations in pulmonary compliance, modifications in vascular permeability, secondary changes in microvascular perfusion with reduced aerobic metabolism6,9 and suppression of immunoglobulin.13 Destruction and dysfunction of platelets, hemolysis, and the reduction of and interference with WBC’s are also supposedly reduced.3,6,9,13,14,15

However, studies performed by the proponents of M.O.’s have not been without errors in scientific methodology.16 This, coupled with other studies which either do not substantiate or else refute the significant differences16,17,18,19 claimed by the M.O. proponents, leaves the theoretical issue unresolved. Recognizing this confusion in the scientific literature, the operational differences between M.O.’s and B.O.’s were investigated.

Methods

The literature, our protocols and student evaluation records were reviewed along with the perfusion records of one hundred patients undergoing cardiopulmonary bypass (CPB) in which either a TMO or a SMKL was used, and 100 in which a B.O. was used. The literature, protocols and student evaluation records were used to compare the simplicity/complexity factors and safety characteristics, while the blood gas data and operat-
ing conditions reported in the perfusion records were used to compare the precision of M.O.'s and B.O.'s.

Results

Advantages. It has been reported that collapsible reservoir bags such as those utilized in TMO and SMKL systems prevent massive air embolization. Although we have no clinical data supporting this, our experience in the animal laboratory, training students, leads us to agree with this report.

M.O.’s may be utilized as a closed system, (isolated from the atmosphere) thus allowing for better blood volume control during CPB and especially at the initiation of CPB. The initiation of CPB can be balanced and is therefore easier for the perfusionist and gentler to the patient. This balanced initiation may eliminate the initial decrease in blood pressure seen at the initiation of CPB.

M.O.’s are cleaner. As part of their quality control, they are water flushed prior to packing in order to pressure test each unit. This flushing removes any particulate matter generated during construction. The presence of antifoam in B.O.’s makes this flushing impossible for the antifoam would be washed out also. Furthermore, it has been demonstrated that M.O.’s generate fewer microbubbles than B.O.’s.

Blood gas control is simple and generally more reproducible with M.O.’s. Both the SMKL and the TMO use separate mechanisms to control oxygenation and ventilation. \( \text{PO}_2 \) is controlled by varying the shim pressure in the TMO while in the SMKL, the \( \text{PO}_2 \) is controlled by varying the concentration of oxygen delivered to the oxygenator. Both systems control the \( \text{PCO}_2 \) by varying the minute volume (QG).

The ideal gas flow requirements or estimated optimal ventilation (EOV) necessary to produce adequate \( \text{PCO}_2 \) during normothermic CPB can be estimated by the following formula:

\[
\text{Estimated Optimal Ventilation} = \frac{\text{BSA(M}^2\text{)} \times 80 \text{ (ml/min/M}^2\text{)}}{5.3%}
\]

\* Manufacturer’s Protocols.

This formula is based on the \( \text{O}_2 \) consumption rate (\( \text{VO}_2 \)) of an anesthetized, paralyzed patient on CPB being approximately 100 ml/M\(^2\). For each ml of \( \text{O}_2 \) consumed, 0.8 ml of \( \text{CO}_2 \) is produced. Therefore the numerator of the formula predicts the \( \text{CO}_2 \) production of the patient. The denominator represents the concentration of \( \text{CO}_2 \) required to produce a \( \text{PCO}_2 \) of 40 mm/Hg under normal barometric conditions. This formula works because the \( \text{PCO}_2 \) of the expired gas from an artificial lung corresponds to the arterial \( \text{PCO}_2 \). With both SMKL and TMO the above formula produced \( \text{PCO}_2 \)'s between 35–42 mm/Hg 95% of the time.

When cooling to 28–30°C, a decrease of 50% in the metabolic rate and \( \text{VO}_2 \) can be assumed with a resultant decrease in \( \text{CO}_2 \) production. Therefore the ventilation rate or gas flow may be reduced by approximately 50%. This should be done on the initiation of cooling and will put you in range of a normal \( \text{PCO}_2 \) but may not be entirely adequate. Therefore, when cooling is stopped, a measurement is made and the ventilation rate is adjusted according to the following formula:

\[
\text{New QG} = \frac{\text{Old QG} \times \text{PaCO}_2}{\text{Desired PaCO}_2}
\]

With the above formula, when using both SMKL and TMO, \( \text{PCO}_2 \)'s of 35–42 mmHg were obtained at stable temperatures of 28–30°C 98% of the time. During profound hypothermia the blood gas data obtained were too variable to establish a correlation.

When rewarming with a TMO, the gas flow should be 5L/min at the initiation of warming and increased to 7L/min when the rectal temperature begins to climb. Only pediatric patients cooled to 18–20°C were available for generating rewarming data for the SMKL. These produced variable data but the best \( \text{PCO}_2 \) results were achieved by returning to EOV at the initiation of warming and doubling EOV when the rectal temperature reached 28–30°C. A SMKL tends to limit the removal of \( \text{CO}_2 \) during the rewarming phase so it is best that a patient does not fall into the extreme upper ranges of the oxygenator’s recommended operational capabilities.

A \( \text{PO}_2 \) of between 90–150 mm/Hg was obtained 81% of the time with a TMO at normothermia by setting the shim pressure at the patient’s weight...
in pounds. The same PO₂ was achieved 94% of the time at stable temperatures of 28–30°C with the shim pressure set on the patient’s weight in kilograms. The data gathered during the warming phase suggest a return to the patient’s weight in pounds on the shim pressure. It is important to note that shim pressures in excess of 280 mm/Hg may impede oxygenation by closing off blood paths.

Under normothermic conditions, using the SMKL, a FiO₂ of .6 maintained the PO₂ between 90 and 150 mm/Hg, 95% of the time, and a FiO₂ of .3 at stable temperatures of 28–30°C, maintained the same PO₂, 94% of the time. Once again, most of the SMKL warming data were gathered on pediatric patients cooled to 18°C and these conditions produced such variable results that it was impossible to develop a correlation but the data suggest an FiO₂ of .4 at the initiation of warming with an increase in the FiO₂ to .6 when the rectal temperature reaches 28–30°C.

We have not been able to establish a relationship between ventilatory function and PCO₂ or PO₂ in B.O.’s. For instance, in a series of 100 patients at normothermia the correlation between QG/QB and PCO₂ was very low \( R = -.124 \). In the same series, the correlation between QG/QB and PO₂ was again very low \( R = .271 \).

Disadvantages. The disadvantages of M.O.’s are probably the real reasons why B.O.’s control the marketplace. The main disadvantage is the complexity of the M.O. system. It takes more time to set up and break down. Using students’ times for comparison, B.O.’s take 8 minutes to set up, TMO’s take 11 minutes, and SMKL’s take 15 minutes. None of the smaller SMKL’s nor any of the TMO’s have integral heat exchangers, which adds to their set-up time and their complexity. SMKL’s should be CO₂ flushed and vacuumed prior to priming. Although slightly longer to set up, it takes less time to change a M.O. during CPB. During laboratory disaster drills, students take about 1 minute to change a TMO, 2 minutes to change a SMKL and 3 minutes to change a B.O.

Some perfusionists dislike the two pump system of the TMO because, if not monitored closely, it can allow desaturated blood to be pumped back to the patient. The shunt line on the SMKL can also produce disaster such as exsanguination of the patient if left open when on bypass or transfusing.

Since the M.O.’s reservoir is not opened to air, the arterial line filter (if used) is purged into the cardiotomy reservoir. If the cardiotomy reservoir becomes pressurized, air can be forced backwards into the filter and disaster in the form of air embolization may occur. Routine pre-CPB checks to insure that the cardiotomy reservoir is properly vented, the use of purge lines with one-way valves and venting into the non-filtered side of the cardiotomy reservoir will eliminate that particular catastrophe.

M.O.’s require slightly larger priming volumes than B.O.’s. A B.O. requires around 1700 ml of priming solution; a TMO requires 2000 ml of priming solution; and a SMKL requires 2200 ml of priming solution.

The cost of an M.O. is greater than that of a B.O. An adult B.O. cost slightly over $200.00 and the hardware is approximately $75.00. An adult TMO system cost $365.00 and the hardware costs over $1050.00. An adult SMKL costs $432.00 and the hardware costs $300.00.

Discussion

The most important and probably the only advantage that clearly justifies the extra cost of membrane oxygenators is the reduced risk of massive air embolization. Probably the next most important advantage is the ability to control blood gases.

There should be no debate upon the desirability of controlling the PCO₂ due to effect of PCO₂ on acid base balance and cerebral circulation. However, the need to control PO₂ is less clear, there being no concrete information on oxygen toxicity, or whether high O₂ tensions harm patients at all. But until we know that high PO₂’s do no harm, values as close to normal as possible should be aimed for.

In conclusion, M.O.’s take longer to set up, cost more, are more complex to operate and have the potential for shunting. However, they offer more precise methods for gas control and, most importantly, they protect against massive air embolism.

References


