

Gaseous microemboli in cardiac surgery with cardiopulmonary bypass: the use of veno-arterial shunt as a preventive method

Microembolia gasosa em operação cardíaca com uso de circulação extracorpórea: emprego de shunt venoarterial como método preventivo

Edison Emidio dos Reis¹, Livia Dutra de Menezes², Caio César Lanaro Justo³

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Abstract

Neurological complications are an important cause of morbidity in the postoperative period of cardiac surgery and its incidence reaches up to 75% of patients. An important cause of these events is the formation of microbubbles in the bloodstream during cardiopulmonary bypass. Integrative review was carried out on gaseous microemboli in cardiopulmonary bypass. This study analyzed studies with different methodological approaches, but that address the issue. The result suggests the denitrogenation of blood by hyperoxia dissolved microbubbles in the blood and venoarterial shunt can balance the respiratory parameters changed with hyperoxia.

Descriptors: Extracorporeal circulation. Embolism, air. Cognition disorders. Cardiac surgical procedures.

Resumo

As complicações neurológicas representam importante causa de morbidade no período pós-operatório de cirurgia cardíaca e sua incidência alcança até 75% dos pacientes. Uma importante causa desses eventos é a formação de microbolhas na corrente sanguínea durante a circulação extracorpórea. Realizou-se revisão integrativa sobre microembolia gasosa na circulação extracorpórea. Esse trabalho analisou estudos com abordagens metodológicas diferentes, mas que contemplam o tema. O resultado sugere que a desnitração do sangue causada pela hiperoxia dissolve microbolhas formadas no sangue e o *shunt* venoarterial pode equilibrar os parâmetros respiratórios alterados pela hiperoxia.

Descritores: Circulação extracorpórea. Embolia aérea. Transtornos cognitivos. Procedimentos cirúrgicos cardíacos.

1. Biologist - UNIMONTES-MG, Brazil. Technical adviser. Author
2. Biomedical - Methodist University, São Paulo, SP, Brazil.
3. Biomedical - Methodist University, Physiologist - ABC University, São Paulo, SP, Brazil

Correspondence address: Edison Emidio dos Reis
Rua Desembargador Eliseu Guilherme, 147 – Paraíso – São Paulo, SP,
Brazil – Zip Code: 04004-030
E-mail: edisonreis@hotmail.com

Abbreviations, acronyms & symbols	
Atm	atmosphere
CaO ₂	Oxygen content of arterial blood
CcO ₂	Capillary Oxygen Content
FEO ₂	Fraction of expired oxygen
Hb	hemoglobin
N ₂	Nitrogen
PAO ₂	Alveolar oxygen tension.
PH ₂ O	Water vapor pressure
PN ₂	Arterial Nitrogen Pressure
Pox.O ₂	Oxygen saturation of the hemoglobin of arterial blood
PVC	Polyvinyl chloride
Qs/Q _t	Shunt as percent of total blood flow
SaO ₂	Oxygen saturation of the hemoglobin of arterial blood
SvO ₂	mixed venous oxygen saturation
VO ₂	oxygen demand

INTRODUCTION

Technology has led to a reduction of morbimortality in cardiac surgery [1-2]. However, neurological complications related to it still represent an important cause of morbidity in the postoperative period [3,4]. Cardiopulmonary bypass (CPB) was introduced into cardiac surgery 60 years ago, and since then, it has been reported that certain patients develop some type of neurological repercussion [1,2,5].

From then on, neurological aftereffects and CPB itself have become the focus of much research [6]. It is believed that the incidence of these complications, in differing degrees, affects up to 75% of patients [5-8]. Fortunately, the great majority are sub-clinical. But clinically, significant cases of cerebral air embolization are largely sub-diagnosed, sub-treated, and sub-notified [3,9,10].

Because it is not a technology that maintains principles of normal physiology, its routine use stimulated analysis of the complications associated with it [10], and as a result, it has a large evolution of procedures and materials used in CPB. In spite of all the progress, neurological complications still occur, and are frequently the cause of serious injury [4, 10]. It is known that the post-operative cognitive alterations observed are caused by multiple factors [8], inherent to the patient or inherent to the surgical procedure itself, which, in conjunction may determine a greater or lesser degree of cognitive deficit and morbimortality [10-11].

Cerebral embolism may be the primary cause of encephalic injury or of the aggravation of pre-existing damage [10]. Gaseous microemboli stand out as the principal cause of neurological disturbances, in addition to inducing inflammatory response by activation of complement, and according to Barak [12] and Rodriguez [13], it can also affect clotting.

Gaseous microemboli can be caused by a number of different sources during cardiopulmonary bypass such as the circuit and oxygenators themselves, in addition to the surgical and perfusion techniques used [14]. Gaseous microemboli can be suddenly displaced from areas where they are and released into the circulation [14]. The process of cooling and reheating the blood modifies the physical properties of the dissolved gases (their solubility) and this predisposes the formation of microbubbles in the bloodstream [10,15]. Perfusionist interventions to administer drugs and collect blood samples produce gaseous microbubbles [2,11,16], as well as vacuum assisted drainage [17,18].

Here we address the application of the veno-arterial shunt, despite having been devised in order to decrease the inflammatory response caused by the passage of blood in the oxygenator, especially in the region of the membrane, it can be an alternative to controlling the production of gaseous microemboli during the operation. Is this system capable of preventing or minimizing these gaseous embolic events? That is what will be discussed below.

METHODS

This study raised the hypothesis that hyperoxia can prevent the formation of microbubbles using a veno-arterial shunt in the membrane oxygenators for CPB. Thus, we proposed an integrative review [19] on the subject of gaseous microemboli during CPB. This type of work allows us to analyze studies with different methodological approaches that address the issue. The selected studies in this type of work lead to the building of a body of knowledge necessary for technical-scientific improvement [20]. The bibliographic research was conducted between December 2010 and September 2011 in the following databases:

The Journal of Extracorporeal Technology, www.ctsnet.org, <http://www.scielo.br/>,

www.anesthesiology.com, <http://www.anesthesia-analgesia.org/>, and www.perflin.com, <http://circ.ahajournals.org/>. Publications from the last 20 years including books of relevance to the study, original, experimental and revision articles were also consulted. The keywords used were: shunt, gaseous microemboli, hyperoxia, microbubbles, neurologic injury after cardiopulmonary by-pass, cerebral air embolism, nitrogen microemboli and cognitive dysfunction after cardiac surgery.

After the reading the analysis of 169 selected articles and 3 books on specific chapters relating to the content, we selected 48 articles that addressed the topic and were related to our objective. The articles were selected, accessed and printed electronically from the site of the database and through the acquisition of journals.

LITERATURE REVIEW

Transcranial Doppler

The use of transcranial Doppler (TCD) made it possible to detect the occurrence of microbubbles in the circulation [12]. The TCD allows continuous measurement of blood flow velocity in cerebral arteries and is able to pinpoint echogenic signals of solid and gaseous origin [5,12,13].

The detection of gaseous microemboli (GME) by application of TCD has been described by several authors among different groups of patients for its accuracy. Newer models have the ability to qualify and quantify the occurrence of microbubbles as the EDAC® quantifier (Luna Technologies, USA) [21-23] and Gampt BC200 (GAMPT mbH, Germany) [16], which, according to the authors, has sufficient sensitivity to identify microbubbles with a diameter of 10µm and count up to 1000 microbubbles per second in streams with 0.2 to 6 L/min [21-23].

The physiology of blood gases

CPB is an artificial cardiopulmonary bypass in which the flow of blood, responsible for transporting oxygen is produced by a peristaltic or centrifugal pump, being that the peristaltic generates pulsatile flow hydrodynamically (not physiologically) and the centrifuge generates a continuous flow, where the gaseous exchanges are performed by a membrane oxygenator which mimics the function of the pulmonary dynamics of exchange, namely by diffusion. The diffusion of a gas depends on the pressure gradient between the means of the gas exchange. Thus,

the higher the gas pressure the greater its diffusion between these means through the membranes [1].

Air is a gas mixture consisting of nitrogen, oxygen and carbonic gas and other gases to a lesser extent, as shown in Table 1. The pressure exerted by each gas component of air is defined as partial pressure [1]. The general characteristics of the diffusion of gases allow us to quantify how quickly a gas can diffuse, that is called diffusion coefficient. Oxygen, by its diffusion characteristics in living organisms, has a diffusion coefficient of 1. The diffusion of other gases is measured in relation to oxygen [24]. Table 2 lists the diffusion coefficient of some gaseous components of air.

The concentration of a gas in a solution depends on the solubility coefficient; the gases which are dissolved in water in larger amounts have a greater solubility coefficient. A change in that solubility is an important factor for determining the risk of air microemboli [1,9,15,24,25]. Oxygen and carbon dioxide have high solubility in blood while nitrogen has low solubility and can, therefore, remain in the blood in the form of gas for 48 hours [9].

According to Boyle's and Graham's laws, the speed and solubility of a gas in a liquid is directly proportional to the temperature and the average pressure applied to it and inversely proportional to the square root of its molecular weight, i.e., the greater the molecular weight of the gaseous solute, the lower the diffusion rate, as well as the gas solubility [12, 25,26]. The N₂ has the highest partial pressure, both in the air and in the blood, and has little coefficient of solubility, characteristics that make it the main gas inside the bubbles formed in the blood.

Decompression sickness

Decompression sickness is caused by nitrogen bubbles that expand blood or tissue and can cause gaseous microemboli. It occurs in divers returning from greater depths of immersion without appropriate decompression due to changes of pressure and solubility of nitrogen (N₂) in blood and tissues, causing air embolism. Severe cases are treated with hyperbaric oxygen therapy (HBOT) [27,28].

Table 1. Atmospheric air composition

Atmospheric gases	Concentration	Partial pressure(P)
Nitrogen	78.62%	597 mmHg
Oxygen	20.84%	159 mmHg
Carbonic gas	0.04%	0.3 mmHg
Water vapor	0.50%	3.4 mmHg
Total	100%	760 mmHg

Table 2. Diffusion coefficient of the gases

Gas	Coefficient
Oxygen	1
Carbonic gas	20.3
Carbon Monoxide	0.81
Nitrogen	0.53
Helium	0.95

Table 3. U.S. Navy Treatment Table for Decompression Sickness

Pressure	Time(min)	FIO ₂	PO ₂	PN ₂	Total
3 ATA	20	100%	2280	0	0:20
3 ATA	5	21%	479	1801	0:25
3 ATA	20	100%	2280	0	0:45
3-2 ATA	30	100%	2280-1520	0	1:15
2 ATA	5	21%	319	1201	1:20
2 ATA	20	100%	1520	0	1:40
2 ATA	5	21%	319	1201	1:45
2-1 ATA	30	100%	1520-760	0	2:15

Hyperbaric Oxygen Therapy has many applications, including treatment of decompression sickness, carbon monoxide poisoning and arterial embolism. The treatment is performed in an enclosed chamber where the patient is subjected to pressures ranging up to 3.0 atmospheres absolute pressure (ATA). At these pressures, the concentration of O_2 in the body is increased by up to 1900%, and PaO_2 higher than 2000 mmHg in sessions lasting 20 to 30 minutes and as a consequence the PaN_2 is reduced to minimum values, as shown in Table 3 for Treatment of Decompression Sickness type 1 from the United States Navy.

Murphy et al. [29], between 1970 and 1984, treated 16 patients for embolism, of which 2 cases were for embolism during a CPB. Guy et al. [30] succeeded in treating a patient with HBOT who had suffered large gaseous emboli during a cardiac surgery. Ziser et al. [31] used HBOT to treat cerebral arterial embolism in 17 patients undergoing CPB.

Grist [28], based on the use of HBOT suggested pure O_2 in CPB in order to treat and prevent N_2 microemboli. The analogy made by Grist between the hyperbaric chamber and the oxygenation chamber of the membrane oxygenator shows a resemblance in general characteristics of closed systems where there is the possibility of using pure oxygen and reduce PaN_2 .

The low pressure of the nitrogen in the blood promotes the tendency of the solubilization of this gas in the formed microbubbles.

Hyperoxia

The oxygenation of blood during infusion corresponds to a PO_2 above 100mmHg and below 200mmHg, called normoxia. Its maintenance occurs according to the offer of greater or lesser fraction of inspired oxygen at (FI_{O_2}) in the gas line connected to the oxygenator during CPB. Hyperoxia is defined as a high concentration of oxygen in the blood, with this condition being produced by the oversupply of oxygen.

Hyperoxia can produce reperfusion injury, excessive production of free radicals [32,33] and even exposure to the toxic effects of oxygen [34]. The use of hyperoxia in CPB is considered unnecessary by most perfusionists. Toraman et al. [35] showed that between 35 and 45% FI_{O_2} during CPB is sufficient to allow oxygen extraction safely.

Veno-arterial shunt

Moraes [38] evaluated the use of a diversion of veno-arterial blood. This deviation was done by a bypass connecting the venous line before entering the oxygenator to an arterial line, which allowed the diversion of part of the circulation, so that a fraction of blood flow was not passed through the oxygenation chamber and returned to the normal circuit system after the oxygenator. This author aimed to reduce the volume of blood in contact with the membrane oxygenator, as shown in Figure 1. The objective was to

demonstrate that the shunt could minimize the inflammatory response induced by cpb. The concept was based on the physiology of fetal circulation, where blood circulating in a fetus is at its greatest volume, a mixture of oxygenated and non-oxygenated blood.

In fetal circulation the only oxygenated blood reaches the fetus from the placenta exclusively by umbilical vein to the point of confluence with the inferior vena cava through the *ductus venosus*. The mixing of oxygenated and non-oxygenated blood with $SaO_2 = 62\%$ in the thoracic aorta is sufficient for the fetus because their metabolic needs are reduced [36]. Moraes [37] published a comparative where he noted that there was less bleeding and a lesser need for transfusions in the group using veno-arterial shunts. This concept is applied in the manufacture of oxyshunt® oxygenators (Instrumental Zammi, Duque de Caxias, Rio de Janeiro, Brazil).

By observing this system based on the physiology of pulmonary shunts, which also occurs between arterial and mixed venous blood, we notice an important relationship. The pulmonary shunt is defined as a disturbance in the pulmonary ventilation-perfusion relation, where a fraction of inadequately ventilated pulmonary blood [38,39] follows without sustaining gas exchange, as illustrated in Figure 1. This non ventilated blood removes some oxygen from the oxygenated blood, which results in a slight reduction in arterial PO_2 (PaO_2), hemoglobin oxygen saturation (SaO_2) and, consequently in arterial oxygen content (CaO_2) (Figures 2 and 3).

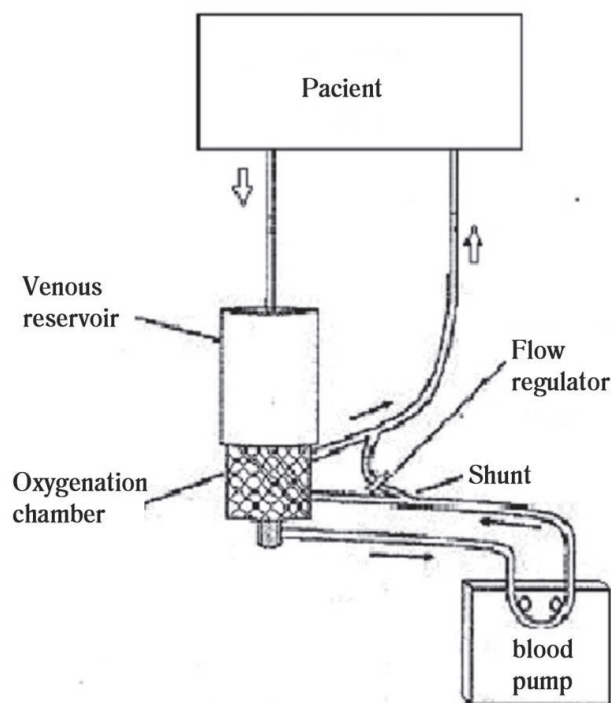


Fig.1 – Schematic representation of the CPB circuit, extracted from the publication

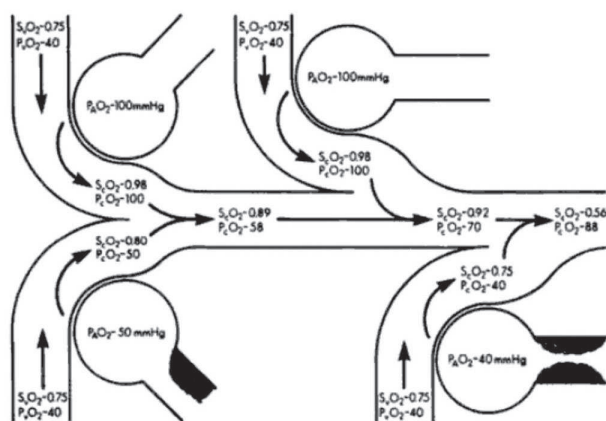


FIGURE 3.6. The greater the ventilation reduction relative to the perfusion, the lower the PaO₂ level.

Fig. 2 - Adapted from *Essentials of oxygenation*, T. Ahrens, 1993

In a normal, healthy individual, the physiological shunt represents 2-4% of the heart debt [1], the larger the fraction of the shunt, the lower the arterial oxygen content (CaO₂)

The pulmonary shunt fraction is measured by the classical shunt equation [38,39].

$$Qs/Qt = (CcO_2 - CaO_2) / (CcO_2 - CvO_2)$$

Where:

Qs: Shunt Flow

Qt: Total blood flow

Qs/Qt: % of blood flow that is diverted to the shunt

CcO₂: Content of the O₂ capillary

CaO₂: Arterial content of O₂

CvO₂: Venous content of O₂

Calculations

$$CcO_2 = (Hb \cdot 1.34 \% S_{cO_2}) + (0.003 \cdot PaO_2)$$

$$CaO_2 = (Hb \cdot 1.34 \% SaO_2) + (0.003 \cdot PaO_2)$$

$$CvO_2 = (Hb \cdot 1.34 \% SvO_2) + (0.003 \cdot PaO_2)$$

CcO₂, CaO₂ and CvO₂ are given in ml of O₂ by 100 ml of blood (ml/100ml).

Applying the equation, considering:

$$PaO_2 = 100 \text{ mmHg} \quad SaO_2 = 100\%$$

$$PvO_2 = 40 \text{ mmHg}$$

$$SvO_2 = 75\%$$

$$Hb = 10 \text{ g/dL}$$

$$FIO_2 = 100\% \text{ (pure oxygen)}$$

Atmospheric Pressure = 760 mmHg, at sea level.

Replacing the CcO₂ with C_{ox}O₂ (blood content in the blood after passing through the membrane) and ScO₂ with SoxO₂ (oxygen saturation after passing through the membrane).

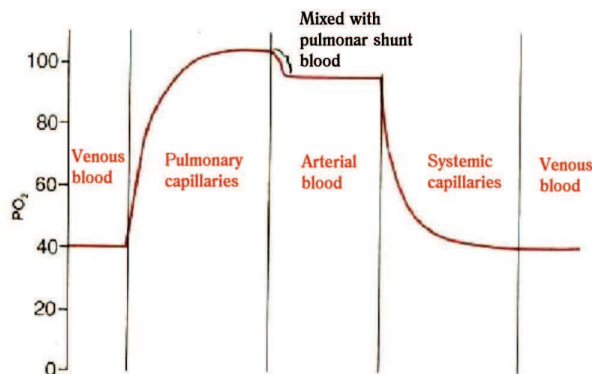


Fig. 3 - Extracted from Guyton, it shows PO₂ variations in pulmonary capillary blood, arterial blood and systemic capillary blood, whereby the effect of venous mixture is observed.

When applying this equation using parameters taken from a cardiopulmonary bypass with an oxygenator, with veno-arterial shunt in use, this relationship is as follows:

We have:

$Qs/Qt = (C_{ox} O_2 - CaO_2) / (C_{ox} O_2 - CvO_2)$, the result is given as a % of total blood flow.

$$Qs/Qt = [(Hb \cdot 1.34 \% S_{ox} O_2) + (0.003 \cdot 760 \text{ mmHg})] - [(Hb \cdot 1.34 \% SaO_2) + (0.003 \cdot PaO_2)] / [(Hb \cdot 1.34 \% S_{ox} O_2) + (0.003 \cdot 760 \text{ mmHg})] - [(Hb \cdot 1.34 \% SvO_2) + (0.003 \cdot PvO_2)] =$$

$$Qs/Qt = [(10 \text{ g/dL} \cdot 1.34 \cdot 1) + (0.003 \cdot 760 \text{ mmHg})] - [(10 \text{ g/dL} \cdot 1.34 \cdot 1) + (0.003 \cdot 100 \text{ mmHg})] / [(10 \text{ g/dL} \cdot 1.34 \cdot 1) + (0.003 \cdot 760 \text{ mmHg})] - [(10 \text{ g/dL} \cdot 1.34 \cdot 0.75) + (0.003 \cdot 40 \text{ mmHg})] =$$

$$Qs/Qt = [15.5] - [13.7] / [15.5] - [10.17] = Qs/Qt = 1,8 / 5,33 =$$

$$Qs/Qt = 0.33 \text{ or } 33\%.$$

Admitting Qt = 5 L/min,

$$Qs = 0.33 \times 5 \text{ L/min} = 1.65 \text{ L/min. Or that is, for } Qt = 5 \text{ L/min, um } Qs = 1.65$$

L / min will be sufficient to control hyperoxia generated with FIO₂ = 100%.

In other words 33% of the perfusion flow passing through the shunt permits an adequate perfusion with the hyperoxia duly controlled.

The idea then occurred to evaluate the prospect of the shunt to control the pure O₂ induced hyperoxia, as this could prevent the formation of microbubbles in CPB and this relationship seems to elucidate the question of microbubbles of nitrogen.

We compared the functional anatomy of the ventilatory unit shown in Figure 4 with the anatomy of the Oxyshunt® Zammi oxygenator (Figure 5).

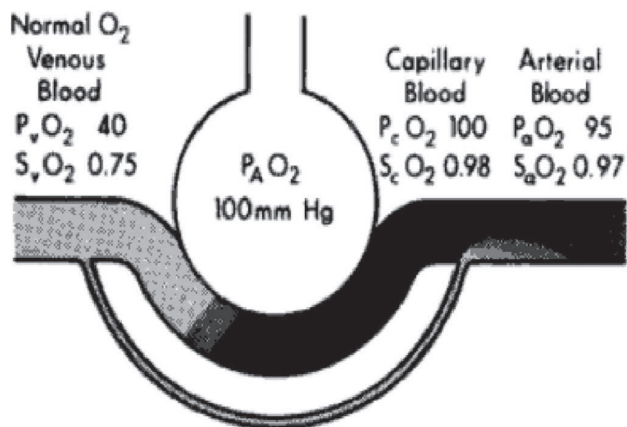


Fig. 4 - Dynamics of the blood as it travels the pulmonary capillary. Adapted from Essentials of oxygenation, T. Ahrens, 1993

The control of venous blood flow (shunt) is made by a clamp mounted on the shunt line, this clamp has an adjustable diameter control line and it is driven by pulse oximetry in the arterial and venous lines, where the targets are 95 and 75% of arterial and venal saturation respectively with a 100% FIO₂ oxygen gas/arterial blood flow of 1:1

Thus, the removal of CO₂ by the membrane behaves in the same manner as in oxygenators that utilize a gas mixer.

An “online” Oximetry is necessary due to the risk of hypoxia produced by an inadvertently kept larger fraction of shunt and by the contrary, hyperoxia.

Both the veno-arterial shunt and the pulmonary shunt permit the reduction of blood passing through the exchange membrane. This diverted blood does not receive oxygen, and as it returns to the arterial line, it mixes with the blood that passed through the membrane. At the moment of the mixture there is a gradient of PaO₂ that is balanced by diffusion in a short segment of arterial line, thus hyperoxia does not occur by the use of pure oxygen avoiding the possible occurrence of reperfusion injury and the harmful effects of excess O₂.

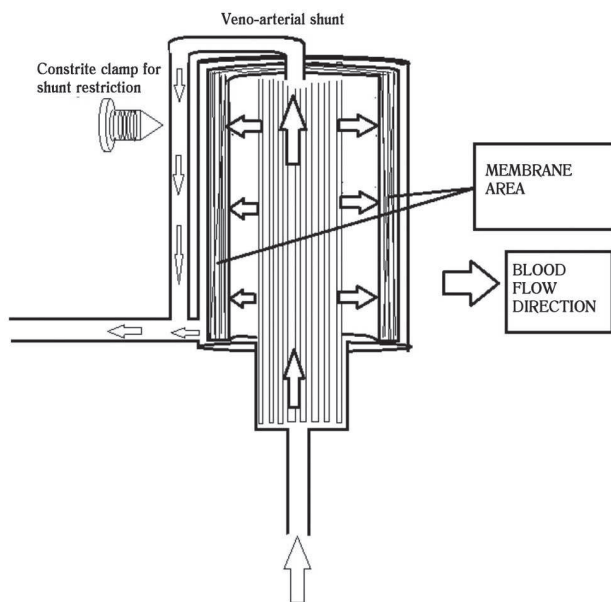


Fig. 5 - Dynamics of the blood in the Oxyshunt oxygenator. Schematic design

The dynamics of blood in the oxygenator follow the principles of hemodynamics, the passage of blood by the membrane area produces high strength, and the tendency of the flow segment is to follow by the segment of least resistance (due to the shunt deviation), which makes necessary a constraint mechanism to control the flow of blood running through the shunt. The deviation causes a smaller portion of blood flow to traverse the membrane so there is a tendency of a lower incidence of hemolysis.

Table 4. Partial pressure of Nitrogen

Partial pressure	Air	Arterial blood	Venous blood	Tissue
PO ₂	158	100	40	40
PCO ₂	0.3	40	46	46
PN ₂	596	573	573	573
PH ₂ O	5.7	47	47	47
Total	760	760	760	706

Nitrogenation

Nitrogen gas is responsible for arterial embolization, this occurs because of its low solubility coupled with its high concentration in blood as well as in tissue (Table 4).

The partial pressure of nitrogen in the blood is 573 mmHg, according to Table 4.

The use of pure oxygen produces denitrogenation of blood, reducing PaN₂ and hence the content of dissolved nitrogen in the body, Tovar et al. [9], and increases the window of oxygen, or that is, with lowered PaN₂, the blood has a lower content of dissolved nitrogen, which allows oxygen to fill this empty space formerly occupied by nitrogen.

With the low pressure of N₂, there is greater tendency for solubilization of the N₂ microbubbles in blood and this still prevents the microbubbles which may be formed in the vascular system. With this concept Berry & Myles [40] produced alveolar denitrogenation with ventilation to a FIO₂ at 100%, and recorded FEO₂ (Fraction expiratory O₂) of 96% after 3 minutes exposure, this experiment shows that this process is rapid and probably happens in the same condition in the membrane oxygenator.

Georgiads et al. [41] subjected 185 patients who were mechanical valve carriers to ventilation with FI_{O_2} to 100% while he monitored microembolic events produced by cavitation valves with TCD. This study showed a significant decrease in the production of microbubbles compared with room air ventilation, suggesting that the principal source of gas embolism in those patients consisted of N_2 bubbles. Rodriguez & Belway [42] developed a flow chart reproduced here, which relates the mechanisms involved in the events that follow up to the cerebral embolism produced by cavitation (Figure 6).

Among the mechanisms mentioned are, complement activation, adhesion of plasma proteins and platelet destruction leading to thrombus formation and vascular injury and embolism from the formation of nitrogen bubbles in the blood.

In the description of Tovan et al.[9], who sees a proportional relationship between volume, area and diameter of the Nitrogen microbubbles formed (Table 5), where gas

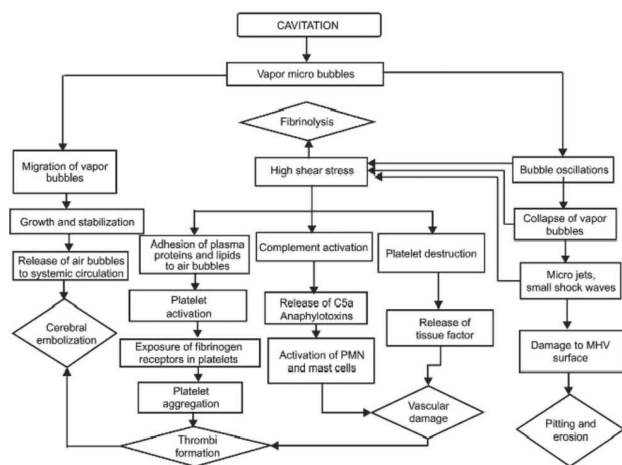


Fig. 6 - The flowchart shows a sequence of events resulting in the formation of cavitation microbubbles that culminates with the final outcome embolism, the same sequence of events can occur during CPB

bubbles enter the bloodstream in dynamic equilibrium with the dissolved gas in plasma, it is noted that these will be enlarged or reduced according to the partial pressure of the gas in solution [11].

However, using the veno-arterial shunt with the flow fraction described above will cause the denitrogenation produced to break this balance and the microbubbles tend strongly to dissolve in the blood.

For the situation of microbubbles formed which passed through the veno-arterial shunt (e.g., bubbles formed in the venous reservoir), when they encounter the arterial blood which passed through the membrane (denitrogenized), there is a strong tendency to dissolve.

Similarly, possible O_2 microbubbles formed in the oxygenator will be dissolved by mixing with the blood passing through the shunt with low PaO_2 , as shown in the flowchart (Figure 7).

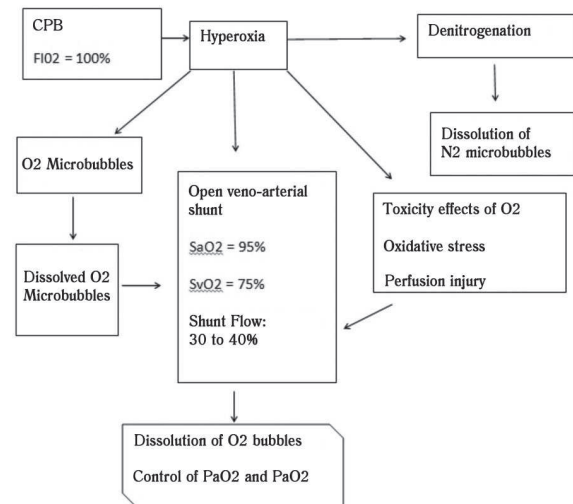


Fig. 7 - The flowchart shows how the shunt venoarterial could control hyperoxia used to produce denitrogenation of blood

Table 5. Relationship between diameter, volume and surface area of the N_2 microbubble

Bubble diameter	Bubble volume	Number of	Volume of bubble x Number of bubbles	Bubble surface	Surface x Number of bubbles
1 cm = 10 mm	0.5 ml	1	0.5 ml	3.14 cm ²	3.14 cm ²
0.1 cm = 1 mm	0.5 µl	1000	0.5 ml	3.14 cm ²	31.4 cm ²
0.01 cm	5x10 ⁴ µl	1000000	0.5 ml	31.4 µm ²	314 cm ²
0.001 cm	5x10 ⁷ µl	1000000000	0.5 ml	314 µm	3140cm ²

RESULTS

The denitrogenation promoted by hyperoxia prevents the formation of microbubbles. The veno-arterial shunt as described by Moraes obeys the physiology of pulmonary shunts and can control hyperoxia used to produce denitrogenation of blood during CPB according to the flowchart in Figure 7.

DISCUSSION

Since the first oxygenator developed by Gibbon to current models, many problems inherent to the CPB were identified, problems related to effectiveness, such as adequate oxygenation, removal of CO₂ [42], and others related to safety such as inflammatory activation trauma and blood microemboli.

This evolution of new knowledge minimized the deleterious effects of CPB and with the addition of accessories such as arterial filters and bubble detectors; it has reduced the morbimortality in cardiac surgery procedures with CPB [38,43-45]. However, the incidence, still frequent of neurological complications remains a challenge because, despite all the advances, neurologic complications still occur and are causes of serious damage [4]. Many studies have been conducted with the accuracy of the TCD, which has made it possible to quantify and qualify the occurrence of microbubbles in both the CPB circuit as well as in cerebral arteries and to measure the resulting GME.

It is known that microbubbles have nitrogen gas as their main component. The removal of N₂ from blood is well documented; the denitrogenation decreases the tension of N₂ pressure, so the bubbles thus formed are dissolved. This occurrence of denitrogenation implies the use of pure O₂ in oxygenation which would lead to hyperoxia and its toxic effects, but that can be avoided in cardiopulmonary bypass procedures with the use of veno-arterial shunts in the oxygenator.

Weitkemper et al. [3] stated that "gaseous microemboli are still an unsolvable problem of the CPB circuit." However, if we consider the information presented here, we believe that this paradigm dissolves with the content presented here.

The veno-arterial shunt that at the time was developed with the goal of reducing inflammatory activation in CPB [38] and was not associated with denitrogenation by the use of pure O₂, which appears to be its main advantage and benefit to the patient. And, moreover, it has proven to be a simple and practical method, provided it is properly controlled, the benefit of which has great importance for those who require the use of CPB in heart and organ management operations, with greater cost savings, and mainly eliminating the complications of air microemboli during cardiopulmonary bypass, as well as an evolution in practice. This work sheds light on this possibility and we

suggest that randomized controlled trials are effected to confirm the benefits of the possibility of damage control and vascular complement activation cited by Rodriguez et al. [13].

CONCLUSION

It was our objective to evaluate whether the use of veno-arterial shunts in membrane oxygenators can prevent the occurrence of microemboli in CPB. We used the integrative review method for this purpose, allowing the freedom to relate publications of different methodologies in order to establish a well founded and logical relationship. It is concluded that literary findings support the hypothesis that the shunt, duly controlled, prevents unchecked gaseous microemboli, facilitating the solubilization of microbubbles of nitrogen arising from the operation according to the description of this method found in the literature, while at the same time, the shunt prevents hyperoxia. However the use of an oximeter line (mainly venous) is indicated to provide adequate control flow of the shunt.

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