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RESEARCH ARTICLES

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Mathematical modelling of oxygenation under veno-venous ECMO configuration using either a femoral or a bicaval drainage

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article

Abstract

Background: The bicaval drainage under veno-venous extracorporeal membrane oxygenation (VV ECMO) was compared in present experimental study to the inferior caval drainage in terms of systemic oxygenation.

Method: Two mathematical models were built to simulate the inferior vena cava-to-right atrium (IVC → RA) route and the bicaval drainage-to-right atrium return (IVC + SVC → RA) route using the following parameters: cardiac output (Q_C), IVC flow/ Q_C ratio, venous oxygen saturation, extracorporeal pump flow (Q_{EC}), and pulmonary shunt (PULM-Shunt) to obtain pulmonary artery oxygen saturation ($S_{PA}O_2$) and systemic blood oxygen saturation (SaO_2).

Results: With the IVC → RA route, $S_{PA}O_2$ and SaO_2 increased linearly with Q_{EC}/Q_C until the threshold of the IVC flow/ Q_C ratio, beyond which the increase in $S_{PA}O_2$ reached a plateau. With the IVC + SVC → RA route, $S_{PA}O_2$ and SaO_2 increased linearly with Q_{EC}/Q_C until 100% with $Q_{EC}/Q_C = 1$. The difference in required Q_{EC}/Q_C between the two routes was all the higher as SaO_2 target or PULM-Shunt were high, and occurred all the earlier as PULM-Shunt were high. The required Q_{EC} between the two routes could differ from 1.0 L/min ($Q_C = 5$ L/min) to 1.5 L/min ($Q_C = 8$ L/min) for SaO_2 target = 90%. Corresponding differences of Q_{EC} for SaO_2 target = 94% were 4.7 L/min and 7.9 L/min, respectively.

Conclusion: Bicaval drainage under ECMO via the IVC + SVC → RA route gave a superior systemic oxygenation performance when both Q_{EC}/Q_C and pulmonary shunt were high. The VV-V ECMO configuration (IVC + SVC → RA route) might be an attractive rescue strategy in case of refractory hypoxaemia under VV ECMO.

Keywords: Bicaval drainage, Oxygenation determinants, Oxygenation performance, Pulmonary shunt, Rescue therapy, Structural recirculation, Superior cava drainage, Superior cava shunt, Triple cannulation, VV-V configuration

Background

Veno-venous extracorporeal membrane oxygenation (VV-ECMO) has become the standard of care in cases of severe acute respiratory distress syndrome (ARDS) and extreme respiratory failure [1–3]. Even though the precise criteria for initiation of

extracorporeal therapy are still debated, its efficacy in cases of refractory hypoxaemia has been largely demonstrated [3, 4]. However, VV-ECMO may fail to restore a satisfactory level of oxygen saturation despite a significant extracorporeal blood flow. Direct determinants of systemic oxygenation are nowadays well known: pulmonary shunt, effective extracorporeal blood flow, cardiac output, and theoretical value of mixed venous oxygen saturation (“SvO₂”) [5, 6]. Thus, effective extracorporeal blood flow, which is directly determined by the extracorporeal settings and recirculation phenomena, is the cornerstone of blood reoxygenation under VV-ECMO. Two recirculation mechanisms have imperatively to be distinguished; the structural recirculation that mainly depends on the chosen extracorporeal route, and on the other hand the direct recirculation that depends on many technical factors (position, orientation, size, length and pattern of cannulas, extracorporeal pump flow, local impedance...) as physiological factors (cardiac output, cardiac rhythm, tricuspid regurgitations, venous impedance, intrathoracic pressure, blood volume status...). [7, 8]

Since a 20 years, the most commonly used extracorporeal route under VV-ECMO is the inferior vena cava-to-right atrium (IVC → RA) route, with two main configurations: femoro-jugular and femoro-femoral [2, 4]. This IVC → RA route is reputed simple and efficient [9]. However, despite extensive use, the IVC → RA route has two main limitations: the structural recirculation and the superior cava shunt (i.e., deoxygenated venous blood from superior vena cava [SVC] directly heading to pulmonary artery) [6–8]. These structural limits in relation to the reinfusion of reoxygenated blood in the superior vena cava (SVC) are inevitable when extracorporeal blood flow is higher than the blood flow into the IVC and cannot be avoided by a simple modification of cannulas position [6]. These two phenomena may explain extracorporeal therapy failures in cases of major pulmonary shunt.

Another extracorporeal route has been proposed to limit the phenomenon of structural recirculation and superior cava shunt: the bicaval drainage-to-right atrium return (IVC⁺SVC → RA) route [10]. The IVC⁺SVC → RA route may be performed using either a single dual-lumen cannula (e.g., Avalon Elite cannula, Getinge, Germany) or three separate single-lumen cannulas (VV-V configuration) [10–12]. Using bicaval drainage and a proximal extracorporeal return, the IVC⁺SVC → RA route should be considered as a serial configuration, which should theoretically reduce structural recirculation. More important, the superior caval shunt, which is the main factor responsible for refractory hypoxaemia under V-V ECMO, would be also reduced; this is not possible with the IVC → RA route [6]. However, despite a strong physiological rationale to limit the superior caval shunt, evidences on the superiority of the IVC⁺SVC → RA route to reoxygenate systemic blood is lacking. Therefore, we wanted to compare the IVC → RA route with the IVC⁺SVC → RA route in terms of blood reoxygenation performance using a recently published ECMO mathematical model [6].

The main objective of present study was to compare the IVC → RA route (V–V configuration) to the IVC⁺SVC → RA route (VV–V configuration) using mathematical modelling (systemic oxygenation, required extracorporeal blood flow) and to identify clinical situations, where bicaval drainage might be a relevant strategy.

Methods

Physiological bases of the two models

Two mathematical models were built on XLSTAT 7.5.2 software (Addinsoft, New York) considering the presence of two central venous systems, the IVC and the SVC [6].

The main abbreviations used in our two models are as follows:

Q_C , cardiac output.

Q_{SVC} , blood flow in the superior vena cava.

Q_{IVC} , blood flow in the inferior vena cava.

k_{IVC} , proportion of cardiac output coming from the inferior vena cava.

Q_{EC} , extracorporeal pump flow.

Q_{Eff} , effective extracorporeal pump flow.

PULM-Shunt, proportion of flow crossing the pulmonary shunt.

" S_{VO_2} ", theoretical value of mixed venous blood oxygen saturation, which is the product of IVC, SVC and coronary sinus blood re-entering the heart. " S_{VO_2} " value could theoretically be assessed in absence of ECMO therapy in the pulmonary artery, but becomes unmeasurable under ECMO therapy.

S_{PAO_2} , blood oxygen saturation in the pulmonary artery.

SaO_2 , arterial blood oxygen saturation.

The first model uses one only site of extracorporeal drainage into the IVC (the IVC \rightarrow RA route), the other uses two extracorporeal drainage sites into the IVC and the SVC (the IVC⁺SVC \rightarrow RA route). These two models are based on blood flows continuity and integrate blood flow and oxygen saturation for each anatomic compartment (Fig. 1). The admixture of blood produced by the ECMO reinfusion in the RA was considered to be homogeneous in these models [12, 13]. To better apprehend structural phenomena induced by these configurations, no direct recirculation was considered in the two models, leading to ideal situations for each of them. The IVC⁺SVC \rightarrow RA route was, moreover, considered as a serial design without any structural recirculation when $Q_{EC} \leq Q_C$. When $Q_{EC}/Q_C > 1$, Q_{Eff} was equal to Q_C and Q_{EC} could only increase due to structural recirculation.

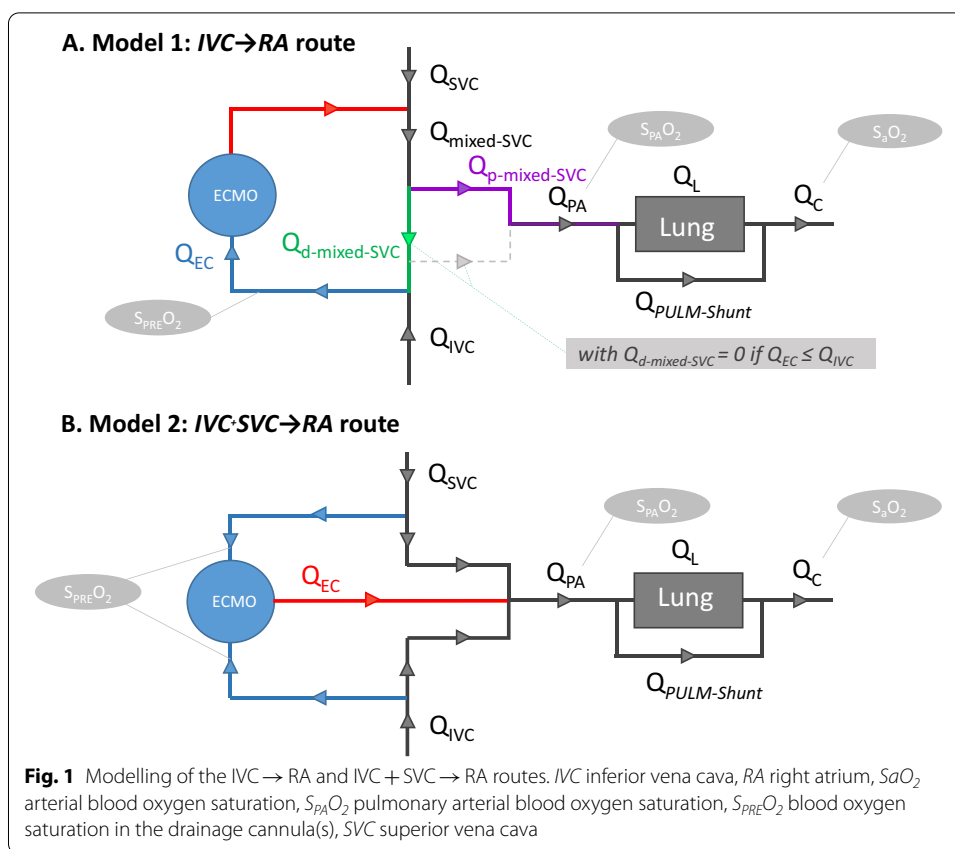
The main equations for the two models are presented in the Additional file 1.

Modelling clinical situations

The settings for four parameters (k_{IVC} , " S_{VO_2} ", Q_C , PULM-Shunt) were determined a priori for the two models to represent different clinical scenarios: k_{IVC} constant and arbitrary defined at 0.67 [14, 15]; " S_{VO_2} " constant and arbitrary defined at 60%; Q_C constant and arbitrary defined at 5 or 8 L/min; PULM-Shunt ranging from 5 to 100% in steps of 5%. For each analysis, once these clinical conditions were defined, Q_{EC} was gradually increased to determine SaO_2 for each route. The Q_{EC} value was expressed by the Q_{EC}/Q_C ratio in steps of 0.1 (ranging from 0 to 2).

Study design

The consequences of PULM-Shunt on SaO_2 were first represented according to the value of S_{PAO_2} to better understand this pathophysiological concept under VV-ECMO, as well



as the S_{PA}O₂ objectives of extracorporeal therapy, knowing that S_{PA}O₂ is the “SvO₂” in the absence of extracorporeal therapy. A specific analysis was also performed for different target levels of SaO₂ (90%, 94%, 98%).

Oxygenation performance of ECMO therapy was also determined for each route according the Q_{EC}/Q_C ratio, which was proven to be a robust reflect of extracorporeal therapy (Q_{EC} component) on oxygenation while integrating physiological conditions and tissue needs (Q_C component). S_{PA}O₂ was thus obtained according to different “SvO₂” levels, whereas SaO₂ was obtained according to PULM-Shunt (“SvO₂” constant at 60%).

Finally, the difference in required Q_{EC} to obtain the SaO₂ targets (90%, 94%, 98%) between the IVC → RA and IVC+SVC → RA routes was calculated according to PULM-Shunt. These results were expressed using the Q_{EC}/Q_C ratio but also as crude values of Q_{EC} (L/min) to better reflect clinically current situations (Q_C = 5 and 8 L/min).

Results

PULM-Shunt and extracorporeal therapy

For a given S_{PA}O₂, SaO₂ was linearly and inversely associated with PULM-Shunt (Fig. 2). At the two extremes: SaO₂ = 100% when PULM-Shunt = 0% and SaO₂ = S_{PA}O₂ when PULM-Shunt = 100%. As expected, the higher the S_{PA}O₂, the higher the SaO₂ was for a given PULM-Shunt, highlighting the direct influence of extracorporeal therapy.

When PULM-Shunt was low, a large range of S_{PA}O₂ values allowed to obtain an SaO₂ target of 90% or more: with a PULM-Shunt = 20%, an S_{PA}O₂ value comprised between 50

and 90% allowed to obtain an SaO₂ target between 90 and 98% (Fig. 2B). Corresponding boundaries values of S_{PA}O₂ for a PULM-Shunt = 40% were 75% and 95%.

In contrast, when PULM-Shunt was high, only a high S_{PA}O₂, close to 90%, allowed to obtain an SaO₂ target of 90% or more: with a PULM-Shunt = 60%, an S_{PA}O₂ value comprised between 83 and 97% allowed to obtain an SaO₂ target between 90 and 98%. Corresponding boundaries values of S_{PA}O₂ for a PULM-Shunt = 80% were 88% and 98% (Fig. 2B).

Oxygenation performance and extracorporeal routes

Using the IVC → RA route, S_{PA}O₂ increased linearly with Q_{EC}/Q_C until the threshold of 0.67. Beyond this threshold, the increase in S_{PA}O₂ rise was strongly reduced and reached a plateau (Fig. 3). SaO₂ showed the same behaviour as S_{PA}O₂, with a value directly dependent on PULM-Shunt. Despite a clinically important Q_{EC}/Q_C, SaO₂ < 94% may occur when PULM-Shunt is significant: Q_{EC}/Q_C = 0.6 with a PULM-Shunt > 35% for example, Q_{EC}/Q_C = 0.8 with a PULM-Shunt > 50%, or and Q_{EC}/Q_C = 1 with a PULM-Shunt > 60%.

In contrast, with the IVC⁺SVC → RA route, S_{PA}O₂ and SaO₂ increased linearly with Q_{EC}/Q_C until it reached 1, corresponding to a value of 100%. With a Q_{EC}/Q_C > 0.86, SaO₂ was ≥ 94% regardless of the importance of PULM-Shunt.

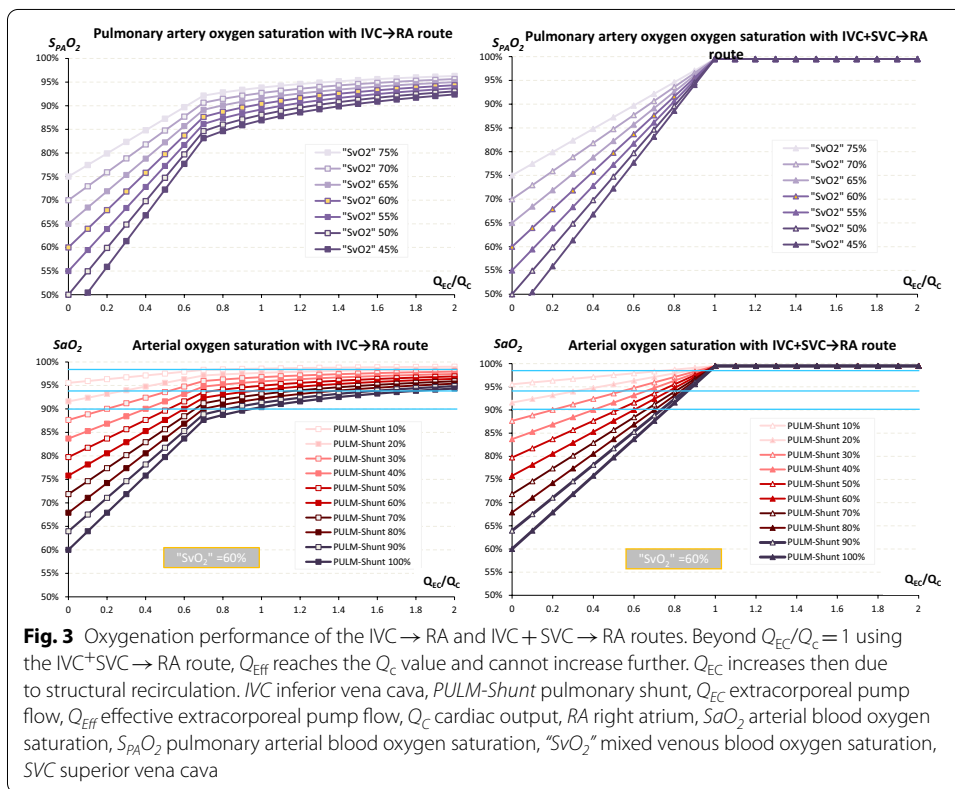
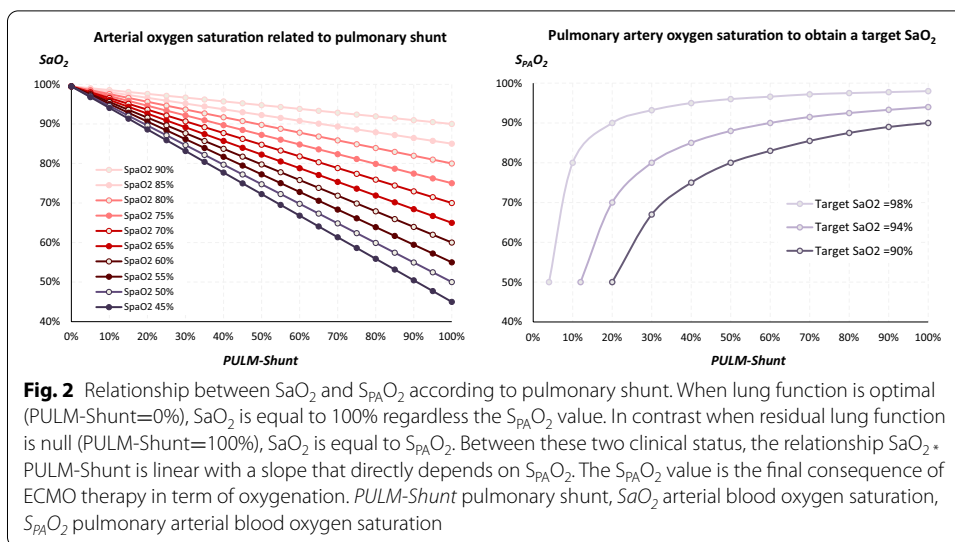
Comparison of the IVC → RA and IVC⁺SVC → RA routes

The higher the SaO₂ target, the more the difference in required Q_{EC} between the two routes was observed for low values of PULM-Shunt: 80% for an SaO₂ target of 90%, 45% for an SaO₂ target of 94%, and 12% for an SaO₂ target of 98%. Similarly, the higher the SaO₂ target, the higher this difference was for a given PULM-Shunt (Fig. 4).

The difference in required Q_{EC} between the two routes for an SaO₂ target of 90% could reach 1.0 L/min when Q_C = 5 L/min, and 1.5 L/min when Q_C = 8 L/min (Fig. 5). For an SaO₂ target of 94%, this difference in required Q_{EC} could reach 4.7 L/min and 7.9 L/min, respectively. A target SaO₂ of 98% could not be obtained with the IVC → RA route when PULM-Shunt was 28% or more.

Discussion

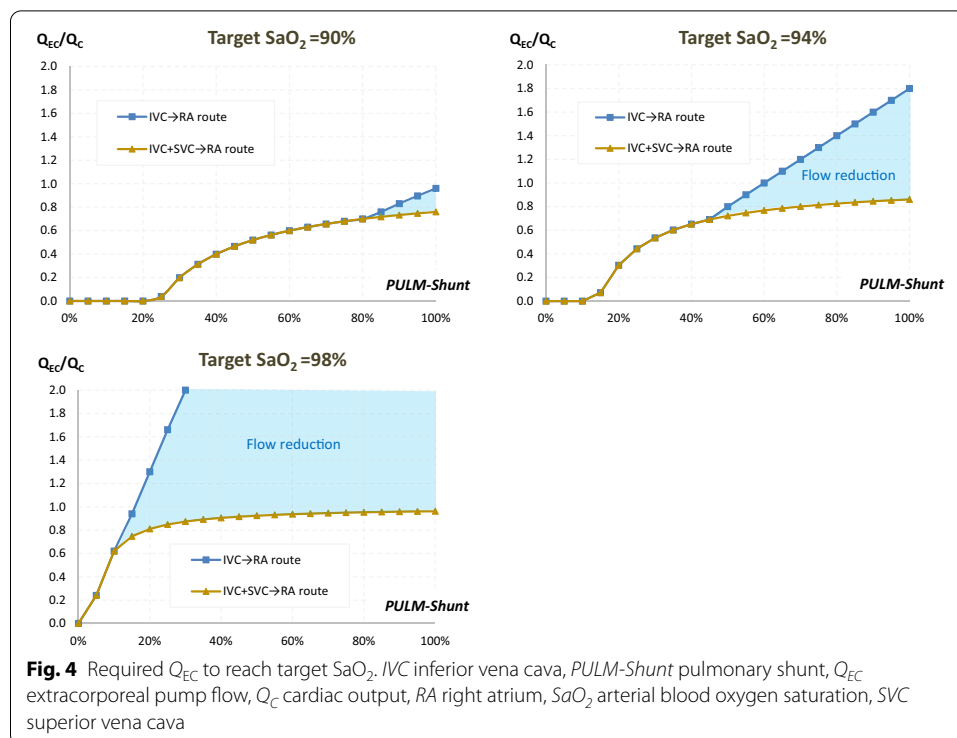
Present study compared the oxygenation performance of the IVC → RA route versus the IVC⁺SVC → RA route using mathematical ECMO models. First, our work has demonstrated that a significant pulmonary shunt imposes a high S_{PA}O₂ to maintain physiological systemic oxygenation. Second, SaO₂ under the IVC → RA route inevitably reaches a plateau despite increasing Q_{EC}, whereas SaO₂ under the IVC⁺SVC → RA route increases linearly with Q_{EC} until 100%. The difference between these two routes occurs indeed in parallel with structural recirculation, corresponding to Q_{EC} > Q_{IVC}. Third, the present study highlights that the higher the SaO₂ target, the earlier is the difference in required Q_{EC}/Q_C between the IVC → RA and IVC⁺SVC → RA routes, and the greater this difference for low pulmonary shunt. Our analysis has also revealed that several SaO₂ levels cannot be obtained under the IVC → RA route when pulmonary shunt was high because of structural recirculation and superior caval shunt. Bicaval drainage could, therefore, be



a part of a rescue therapy when pulmonary shunt induces refractory hypoxaemia under the $IVC \rightarrow RA$ route.

Understanding the pulmonary shunt and clinical implications

The expression of pulmonary shunt as a simple percentage may appear abstract in clinical practice, especially in cases of heterogeneous ARDS or unsystematized alveolar



damage. Definition of pulmonary shunt indeed is not only defined by the volume of aerated lung/alveolar collapse, but rather by the percentage of pulmonary blood circulation that will not be oxygenated through the lung; two theoretical flows into the pulmonary venous return may thus be distinguished: reoxygenated blood ($SO_2 = 100\%$) and deoxygenated blood ($SO_2 = S_{PA}O_2$). This definition includes redistribution of the pulmonary circulation, as well as anatomic and functional pulmonary shunts [17, 18]. In clinical practice without ECMO therapy, $S_{PA}O_2$ and “ SvO_2 ” are logically equal [19]. Pulmonary shunt is simply accessible with SaO_2 , when the value of “ SvO_2 ” is known ($SaO_2 = 100 \times (1 - PULM-Shunt) + “SvO_2” \times PULM-Shunt$).

Under VV-ECMO, the “ SvO_2 ” becomes a theoretical value translating cellular extraction that cannot be measured. Moreover, the relationship between PULM-Shunt and SaO_2 becomes more complex, because it is strongly influenced by the variations of $S_{PA}O_2$ induced by extracorporeal support (Fig. 2). Effects of VV-ECMO on oxygenation may indeed be simply summarized by an increase in $S_{PA}O_2$, which, combined with the PULM-Shunt, directly determines SaO_2 . For these reasons, we focused the first part of our analysis on the inter-relationship between these determinants. Thus, our work demonstrates innovatively that for a low PULM-Shunt, a large range of $S_{PA}O_2$ values will generate a physiological SaO_2 ; in other words, a large range of Q_{EC} values will generate a physiological SaO_2 . In contrast, in the case of significant PULM-Shunt, only a high $S_{PA}O_2$ target obtained by high Q_{EC} will allow an acceptable SaO_2 to be maintained. In this case, a small reduction in $S_{PA}O_2$ may induce a strong decrease in systemic oxygenation, highlighting the importance of extracorporeal rheological conditions in the most critical situations (Fig. 2). The present analysis allows, therefore, better understanding of the interactions between ECMO therapy and the clinical pulmonary situation. In

addition, Fig. 2 shows that a high $S_{PA}O_2$ generated by ECMO could mask significant pulmonary impairment, which will not be visible on SaO_2 . This observation explains why the SaO_2 target under ECMO therapy should not be maximal to better understand the clinical worsening and the status of alveolar recruitment.

Structural recirculation and extracorporeal routes

Direct recirculation under VV-ECMO is well known by clinicians, unlike structural recirculation, which is less understood [7, 20, 21]. However, structural recirculation is inevitable under the $IVC \rightarrow RA$ route when Q_{EC} exceeds Q_{IVC} . [6] The direct consequence is that effective Q_{EC} , extracorporeal blood well oxygenated, becomes lower than the set Q_{EC} . In parallel, the superior cava shunt caused by the admixture of extracorporeal reinfusion in SVC will favour hypoxaemia despite a high value of Q_{EC} . All these mechanisms are intrinsic and inevitable limits of this route that cannot be reduced by any repositioning of cannulas. Thus, refractory hypoxaemia is possible using the $IVC \rightarrow RA$ route when the pulmonary shunt is massive despite optimized ECMO therapy (Fig. 3) [22]. This has been demonstrated by our “ideal” model that does not integrate direct recirculation. Other routes such as to right atrium-to-inferior vena cava were also proposed, but they showed a higher rate of structural recirculation [9, 12, 23]. In contrast, the $IVC^+SVC \rightarrow RA$ route using bicaval extracorporeal drainage behaves like a serial design, without structural recirculation and with a Q_{Eff} close to the Q_{EC} . The direct consequence is that the superior cava shunt may be reduced allowing systemic oxygenation to be maintained in the case of a massive PULM-Shunt (Fig. 3). Three conclusions can thus be drawn from the comparison between $IVC \rightarrow RA$ and $IVC^+SVC \rightarrow RA$ routes. First, the difference between these two routes in terms of oxygenation is mainly observable when PULM-Shunt is severe (Fig. 4). Second, this difference of performance is higher when SaO_2 target and/or PULM-Shunt are high. Third, the $IVC^+SVC \rightarrow RA$ route becomes superior only when structural recirculation occurs under $IVC \rightarrow RA$ route.

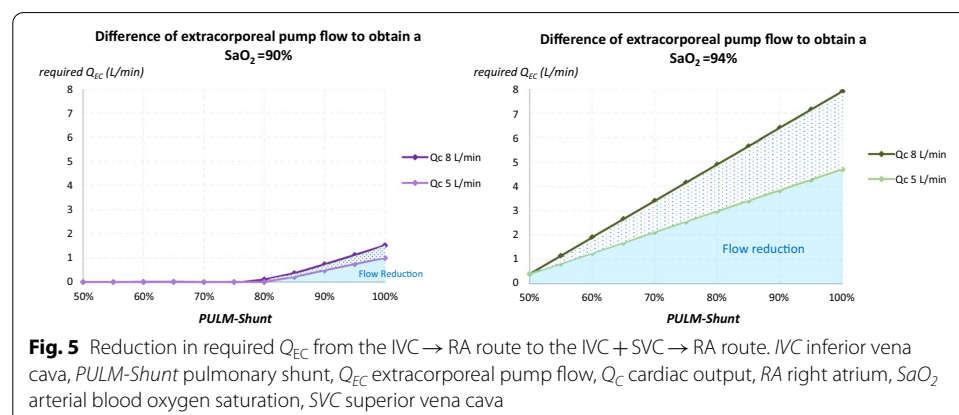
Clinical indications of bicaval drainage

Our study explores, therefore, the clinical situations, where the $IVC^+SVC \rightarrow RA$ route may be superior to the $IVC \rightarrow RA$ route in terms of blood reoxygenation. The performances of these two routes may be considered as comparable in the absence of structural recirculation (i.e., $Q_{EC} < Q_{IVC}$) or when PULM-Shunt is moderate. In the case of refractory hypoxaemia, the first step of management should be an increase in the Q_{EC}/Q_C ratio, either increasing Q_{EC} and/or decreasing Q_C (β -blockers, sedation, etc....) [24, 25]. However, the change of Q_{EC}/Q_C ratio may also increase direct recirculation leading to an uncertain result. Furthermore, ELSO recommendations consider that as SaO_2 around 85% could be an acceptable clinical value [26]. Anyway, in the presence of significant structural recirculation under $IVC \rightarrow RA$ route, the $IVC^+SVC \rightarrow RA$ route appears to be superior in terms of systemic oxygenation. These assumptions are only valuable if direct recirculation phenomenon does not increase with the change of route. Our analysis supports thus that, in the case of refractory hypoxaemia, the $IVC^+SVC \rightarrow RA$ route should be used when other optimization measures have failed. It is nevertheless important to understand that its added value depends on the target SaO_2 . Differences are particularly visible for high target of SaO_2 (Fig. 4); the reduction in Q_{EC} using bicaval drainage may

rapidly reach 1–2 L/min, which may make a real difference in clinical practice (Fig. 5). Moreover, several SaO_2 targets will not be attainable with the IVC → RA route, because required Q_{EC} is too high.

Technical considerations for the VV-V configuration

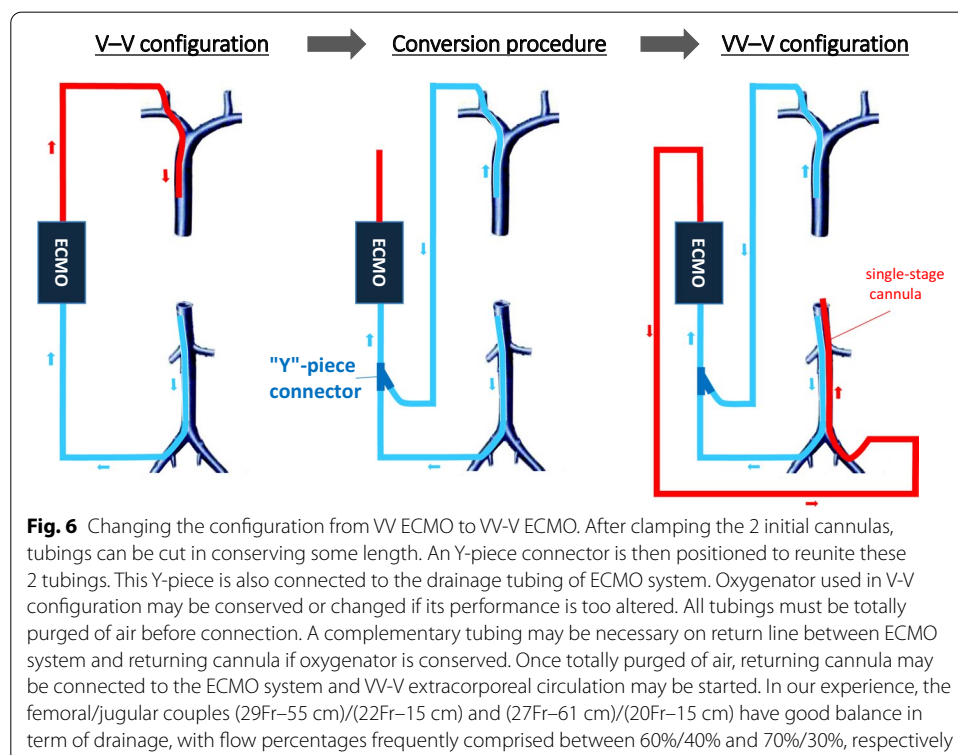
Bicaval drainage can be realized as soon as implantation of ECMO using a dual-lumen cannula via the SVC and positioned across the RA [27]. However, a bicaval dual-lumen cannula imposes large diameters (≥ 27 Fr) to obtain an acceptable Q_{EC} and its position is reputed instable. Furthermore, direct recirculation may widely increase in cases of moderate shifting [11]. Finally, this kind of device may hardly be proposed as second step when clinical situation remains critical, because a change of configuration at this phase would be too risky. A second option could be to use a multi-staged interrupted drainage cannula upon ECMO implantation [28]. However, this configuration requires transatrial cannulation, which may be technically challenging during its placement. Furthermore, as dual-lumen cannula, repartition between superior and inferior drainage is not known and probably strongly varies with several factors (i.e., Q_{EC} , local impedance, drainage pressure, cannula size...). Finally, a cannula change in case of critical situation does not appear without risk. A third option is the use of a triple cannulation, the VV-V ECMO configuration: two drainage cannulas (IVC and SVC) and one reinfusion cannula (RA) [10]. VV-V ECMO allows a choice of optimal and sufficient diameters for each cannula and a better precision and stability of their positioning. Superior/inferior drainage ratio may be adjusted by cannulas mobilization and external compression of the tubing to favour one or the other of the drainages to limit direct recirculation. Moreover, this attractive configuration may be proposed as a stepwise procedure if the IVC → RA route has failed (Fig. 5). For example, conversion from the femoro-jugular V-V configuration to a VV-V configuration is easy, keeping the two cannulas previously placed as drainage cannulas and repositioning a third cannula for the return of extracorporeal blood into the RA (Fig. 6). An accurate positioning in the RA and a single-stage design are, however, indispensable to limit direct recirculation. In addition to the reduction in the superior cava shunt and optimization of the Q_{EFP} , this configuration would also allow improvement in the impedance of venous blood drainage and maximal pump flow [10]. We strongly believe that this sequenced strategy of extracorporeal respiratory support



is the most appropriate way to adapt to the clinical needs of patients and their delayed worsening; a gradual response when necessary! However, prospective studies must be done to prove the safety and efficacy of this kind of strategy.

Potential limitations of the present study

First, direct recirculation was not considered in our analysis. A bad positioning of a cannula may indeed significantly increase recirculation phenomena, especially under the IVC⁺SVC → RA route or when Q_{EC} is high. Furthermore, the heart cycle and tricuspid valve closure may reinforce these phenomena. Thus, behaviour of VV-V configuration, more at risk of direct recirculation and more dependent from cannula positioning, could be less efficient in term of oxygenation than the ideal model used in our works. However, it seems to us essential to understand the differences in structural recirculation according to different extracorporeal routes to identify the limitations and disadvantages of each ECMO configuration. Second, our IVC⁺SVC → RA model was built as previously detailed without any structural recirculation when $Q_{EC} \leq Q_C$. This simulates obviously an ideal situation that in clinical conditions depends on many parameters, such as position of each cannulas, theirs patterns, their diameters, local impedances, and thoracic or abdominal pressures. In clinical practice, a structural recirculation may exist under IVC⁺SVC → RA route, which logically alters performance of this configuration. Third, our model did not consider the influence of dissolved oxygen in the reoxygenated blood. A strong increase in the partial pressure of oxygen (e.g., oxygen fraction crossing the oxygenator close to 100%) may increase the mass of oxygen transferred into the extracorporeal circulation by



increasing the mass of dissolved oxygen. However, we assume that this analysis bias exists similarly for each route and has a modest influence on the SaO_2 [29, 30]. Anyway, to better understand structural recirculation phenomena, it appears more relevant to apprehend extracorporeal oxygenation determinants without mechanisms of solubility of gases. Fourth, pulmonary shunt may vary during the respiratory cycle or by a change in the airway pressure level by modifying West's lung zones [31]. Many clinical factors influencing the pulmonary shunt could indeed be integrated into the model. However, we believe that this simplified approach offers a global understanding of the clinical situation and balance between lung and extracorporeal oxygenation, which is the central question during VV-ECMO management.

Conclusion

Our mathematical modelling has allowed us to compare the differences between the $\text{IVC} \rightarrow \text{RA}$ and $\text{IVC}^+\text{SVC} \rightarrow \text{RA}$ routes in terms of oxygenation performance. The usual $\text{IVC} \rightarrow \text{RA}$ route generates inevitably a structural recirculation and a superior cava shunt, which may lead to refractory hypoxaemia in extreme cases. The $\text{IVC}^+\text{SVC} \rightarrow \text{RA}$ route would not suffer from these structural limitations. The present study suggests, therefore, that the VV-V ECMO configuration might be an attractive therapeutic option when VV-ECMO does not allow sufficient oxygenation, provided that the configuration change is not associated with a significant increase of direct recirculation.

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s40635-022-00434-x>.

Additional file 1. Mathematical Basis of Model 1: the $\text{IVC} \rightarrow \text{RA}$ Route. Mathematical basis of Model 2: the $\text{IVC}^+\text{SVC} \rightarrow \text{RA}$ Route.

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None.

Authors' contributions

J.C. prepared the draft of the study and conducted the modelling. E.C., G.D., P.D., C.M. and H.W. performed figures and annexes. J.C. wrote the article, which was revised by T.L. and X.C. All authors are physicians that regularly implement and manage ECMO therapy in their daily practice. All authors read and approved the final manuscript.

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Availability of data and materials

The data sets used and analyzed are during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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References

1. Papazian L, Aubron C, Brochard L et al (2019) Formal guidelines: management of acute respiratory distress syndrome. *Ann Intensive Care* 13:69
2. Schmidt M, Pham T, Arcadipane A et al (2019) Mechanical ventilation management during extracorporeal membrane oxygenation for acute respiratory distress syndrome. An international multicenter prospective cohort. *Am J Respir Crit Care Med* 200:1002–1012
3. Peek G, Mugford M, Tiruvoipati R, et al., CESAR trial collaboration (2009) Efficacy and economic assessment of conventional ventilatory support versus extracorporeal membrane oxygenation for severe adult respiratory failure (CESAR): a multicentre randomised controlled trial. *Lancet* 374: 1351–1363.
4. Combes A, Hajage D, Capellier G et al (2018) EOLIA Trial Group, REVA, and ECMONet: extracorporeal membrane oxygenation for severe acute respiratory distress syndrome. *N Engl J Med* 378:1965–1975
5. Schmidt M, Tachon G, Devilliers C et al (2013) Blood oxygenation and decarboxylation determinants during venovenous ECMO for respiratory failure in adults. *Intensive Care Med* 39:838–846
6. Charbit J, Deras P, Courvalin E et al (2021) Structural recirculation and refractory hypoxemia under femoro-jugular venovenous extracorporeal membrane oxygenation. *Artif Organs* 45:893–902
7. Abrams D, Bacchetta M, Brodie D (2015) Recirculation in venovenous extracorporeal membrane oxygenation. *ASAIO J* 61:115–121
8. Togo K, Takewa Y, Katagiri N et al (2015) Impact of bypass flow rate and catheter position in veno-venous extracorporeal membrane oxygenation on gas exchange in vivo. *J Artif Organs* 18:128–135
9. Rich P, Awad S, Crotti S, Hirschl R, Bartlett R, Schreiner R (1998) A prospective comparison of atrio-femoral and femoro-atrial flow in adult venovenous extracorporeal life support. *J Thorac Cardiovasc Surg* 116:628–632
10. Ichiba S, Peek G, Sosnowski A, Brennan K, Firmin R (2000) Modifying a venovenous extracorporeal membrane oxygenation circuit to reduce recirculation. *Ann Thorac Surg* 69:298–299
11. Van Heijst A, van der Staak F, de Haan A et al (2001) Recirculation in double lumen catheter veno-venous extracorporeal membrane oxygenation measured by an ultrasound dilution technique. *ASAIO J* 47:372–376
12. Broman M, Frenckner B, Bjällmark A, Broomé M (2015) Recirculation during veno-venous extra-corporeal membrane oxygenation—a simulation study. *Int J Artif Organs* 38:23–30
13. Messai E, Bouguerra A, Harmelin G, Di Lascio G, Cianchi G, Bonacchi M (2013) A new formula for determining arterial oxygen saturation during venovenous extracorporeal oxygenation. *Intensive Care Med* 39:327–334
14. Coleridge J, Hemingway A (1958) Partition of the venous return to the heart. *J Physiol* 142:366–381
15. Mohiaddin R, Wann S, Underwood R, Firmin D, Rees S, Longmore D (1990) Vena caval flow: assessment with cine MR velocity mapping. *Radiology* 177:537–541
16. Murray J, Matthay M, Luce J, Flick M (1988) An expanded definition of the adult respiratory distress syndrome. *Am Rev Respir Dis* 138:720–723
17. Cruz J, Metting P (1987) Understanding the meaning of the shunt fraction calculation. *J Clin Monit* 3:124–134
18. Berggren S (1942) The oxygen deficit of arterial blood caused by nonventilating parts of the lung. *Acta Physiol Scand* 4:1–92
19. Reinhart K, Kuhn H, Hartog C, Bredle D (2004) Continuous central venous and pulmonary artery oxygen saturation monitoring in the critically ill. *Intensive Care Med* 30:1572–1578
20. Frenckner B, Broman M, Broomé M (2018) Position of draining venous cannula in extracorporeal membrane oxygenation for respiratory and respiratory/circulatory support in adult patients. *Crit Care* 22:163
21. Xie A, Yan T, Forrest P (2016) Recirculation in venovenous extracorporeal membrane oxygenation. *J Crit Care* 36:107–110
22. Nunes L, Mendes P, Hirota A et al (2014) ECMO Group: severe hypoxemia during veno-venous extracorporeal membrane oxygenation: exploring the limits of extracorporeal respiratory support. *Clinics* 69:173–178
23. Kolla S, Awad S, Rich P, Schreiner R, Hirschl R, Bartlett R (1997) Extracorporeal life support for 100 adult patients with severe respiratory failure. *Ann Surg* 226:544–564
24. Montisci A, Maj G, Zangrillo A, Winterton D, Pappalardo F (2015) Management of refractory hypoxemia during venovenous extracorporeal membrane oxygenation for ARDS. *ASAIO J* 61:227–236
25. Guarracino F, Zangrillo A, Ruggeri L et al (2012) β -Blockers to optimize peripheral oxygenation during extracorporeal membrane oxygenation: a case series. *J Cardiothorac Vasc Anesth* 26:58–63
26. Tonna J, Abrams D, Brodie D, Greenwood J, Rubio MS, Jose A, Usman A, Fan E (2021) Management of adult patients supported with venovenous extracorporeal membrane oxygenation (VV ECMO): guideline from the extracorporeal life support organization (ELSO). *ASAIO J* 67:601–610
27. Badu B, Cain M, Durham L (2020) A dual-lumen percutaneous cannula for managing refractory right ventricular failure. *ASAIO J* 66:915–921
28. Broman L, Wittberg L, Westlund C (2019) Pressure and flow properties of cannulae for extracorporeal membrane oxygenation II: drainage (venous) cannulae. *Perfusion* 34:65–73
29. Hills B (1972) Diffusion and convection in lungs and gills. *Respir Physiol* 14:105–114
30. Butler J, Tsuda A (2011) Transport of gases between the environment and alveoli—theoretical foundations. *Compr Physiol* 1:1301–1316
31. West J, Dollery C, Naimark A (1964) Distribution of blood flow in isolated lung; relation to vascular and alveolar pressures. *J Appl Physiol* 19:713–724

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