



## The ECMO Book, First Edition

**Jeffrey DellaVolpe. Elsevier, Philadelphia, PA, USA; 2023. CAD 137.99, 248 pages.  
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The use of extracorporeal membrane oxygenation (ECMO) in patients with severe cardiac or respiratory failure (or both) has expanded dramatically over the past decades. Despite the simplistic mechanism of taking blood out of body and pumping back after oxygenation and carbon dioxide (CO<sub>2</sub>) removal, many ECMO programs struggle to respond appropriately because detailed understanding of ECMO's complexities frequently is lacking. *The ECMO Book*, First Edition (2023), by Jeffrey DellaVolpe from the institute for ECMO support (San Antonio, TX, USA) and published by Elsevier, is an approachable, easy-to-read book that provides a systematic framework for understanding and approaching the care of patients who will require ECMO support.

This book has four sections: cardiorespiratory physiology of shock and oxygen transport, fundamentals of ECMO, physiology of ECMO, and management of ECMO. The chapters are well-written, concise, and use simple language. All the chapters provide clinical pearls supported by easy-to-understand examples and highlight the key points. Significant value is added to the book chapters with numerous coloured illustrations and up-to-date references.

I was captivated by the usefulness of sections 2, 3, and 4, which present easy-to-understand comprehensive reviews of ECMO configurations, physiology, and management. The toxicity of medical and ECMO management is splendidly explained along with critical timing for ECMO initiation. The differences between venovenous (VV) ECMO and peripheral and central venoarterial (VA) ECMO are impressively presented with

particular attention to recirculation, effects on the right and left ventricle, need and methods of decompression of the left ventricle, and Harlequin syndrome physiology. The pharmacology of anticoagulation and dosing and monitoring strategies are very informative for naive learners.

The value of this book could have been enhanced by adding a chapter on tips for troubleshooting. Another reservation is that a few respiratory physiology models reflect concepts that are not contemporary. For example, in Chapter 4 (p. 40), the concept of gravity-dependent blood flow distribution to dependent zones of lungs is outdated as experiments in Space Shuttles under zero and enhanced gravity and in standing horses have shown that gravity is not an important factor in loop circulation and that blood weight-induced vascular distention does not necessarily signify increased blood flow.<sup>1–3</sup> Similarly, in Chapter 11, some readers might not be able to easily follow the logic of the description of the rate-limiting step of oxygen delivery and CO<sub>2</sub> washout. A simplistic approach would be that the rate-limiting step for CO<sub>2</sub> removal is primarily determined by its low partial pressure (PP) in blood, which determines the fraction of CO<sub>2</sub> in millilitres that can be transported by the sweep rate in the case of ECMO or alveolar minute ventilation (MV<sub>a</sub>) in the case of the lungs. For example, under stable conditions, assuming a respiratory quotient of 0.8, CO<sub>2</sub> production would be 200 mL·min<sup>-1</sup> for 250 mL·min<sup>-1</sup> of oxygen consumption. If the P<sub>PCO<sub>2</sub></sub> in blood leaving ECMO/lungs is 40 mm Hg (about 5% of atmospheric pressure), it will require a sweep rate or MV<sub>a</sub> of 4 L·min<sup>-1</sup> (5% of 4 L is 200 mL; Dalton law of partial pressures and % volumes, 0% CO<sub>2</sub> in inflow gas). At this sweep rate or MV<sub>a</sub> of 4 L·min<sup>-1</sup>, even using air, 800 mL·min<sup>-1</sup> of O<sub>2</sub> will be delivered to the ECMO membrane or functional residual capacity of the lungs—which is 550 mL in excess than the required 250 mL·min<sup>-1</sup> (assuming 20% O<sub>2</sub> in air with a P<sub>PO<sub>2</sub></sub> of 150 mm Hg).

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Hence, gas returning from the ECMO circuit or the lungs will still contain 17% O<sub>2</sub> (550 mL in 4 L·min<sup>-1</sup> flow). For ECMO blood flow, a minimal flow of 0.4 L·min<sup>-1</sup> will be required to carry 200 mL of CO<sub>2</sub> (assuming a venous blood CO<sub>2</sub> content of 50 mL·100 mL<sup>-1</sup>) and 1.25 L·min<sup>-1</sup> of flow will be required to carry 250 mL of O<sub>2</sub> (assuming an arterial blood O<sub>2</sub> content of 20 mL·100 mL<sup>-1</sup>). An additional 1.25 L·min<sup>-1</sup> of blood flow will be required to maintain an O<sub>2</sub> partial pressure in venous blood of ≥ 25 mm Hg to maintain an O<sub>2</sub> diffusion gradient at the capillaries. This O<sub>2</sub> content of 250 mL cannot be extracted by tissues and will keep recirculating. Similarly, an additional 400 mL·min<sup>-1</sup> of blood flow will be required to maintain the PPCO<sub>2</sub> at 40 mm Hg at the ECMO membrane and this CO<sub>2</sub> content will keep recirculating.<sup>4,6</sup> Factors including the effects of water condensation on the ECMO membrane, the surface area of the membrane, the fraction of CO<sub>2</sub> washed out by ventilated lungs and ECMO, the blood flow rate, and the effect of hemoglobin concentration can be added to this basic model. Ideally, this chapter would have clarified that more CO<sub>2</sub> diffuses across wet membranes than O<sub>2</sub> does because CO<sub>2</sub> forms H<sub>2</sub>CO<sub>3</sub>, whereas more O<sub>2</sub> diffuses across dry membranes than CO<sub>2</sub> does (Graham's law). Finally, three major selectable regulating variables are blood flow rate, sweep flow rate and membrane area for CO<sub>2</sub> washout.<sup>6</sup>

Further to the above, I detected the following printing errors :

- Chapter 2 (p. 28): Fig. 2.20 shows vasopressin reducing vasoconstriction and increasing inotropy, which contradicts the text.
- Chapter 4 (p. 41, 42, 52): Figs 4.7, 4.8, and 5.8 show iso-shunt lines deviated leftward from the stranded iso-shunt diagram, giving the impression that 100% oxygen becomes ineffective in increasing P<sub>a</sub>O<sub>2</sub> in blood only when shunt is ≥ 50%, whereas in routine clinical practice 100% oxygen becomes clinically ineffective once the shunt magnitude is > 30%.<sup>7</sup>
- Chapter 4 (p. 44–45): The discussion in the context of mechanical ventilation omits the concept of plateau pressure, and the equation for compliance mentions peak inspiratory pressure in place of plateau pressure without specifying ventilation mode.
- Chapter 18 (p. 185): In the equation “H<sub>2</sub>O + CO<sub>2</sub>,” it should correctly read “H<sub>2</sub>CO<sub>3</sub>” instead of “H + CO + .”
- Chapter 19 (p. 192): Fig. 19.1, showing positive airway pressure sway above positive end-expiratory pressure instead of negative swing during spontaneous inspiratory phase, may be confusing to readers. On page 193, open chest with sternotomy for central ECMO should be included as an indication for paralysis and controlled ventilation.
- Chapter 20 (p. 204): The text on intrinsic cascade (factor IX activating factor XI, which activates factor X) is contradictory to Figs 20.6 and 20.13 on pages 205 and 212.

In summary, *The ECMO Book* will provide ECMO care team members with a relevant and practical understanding of ECMO physiology as well as valuable management tips. I highly recommend that this book be included in any library accessed by those who provide ECMO care.

**Disclosures** None.

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