Severe Amlodipine Toxicity treated with Veno-Arterial Extracorporeal Membrane Oxygenation

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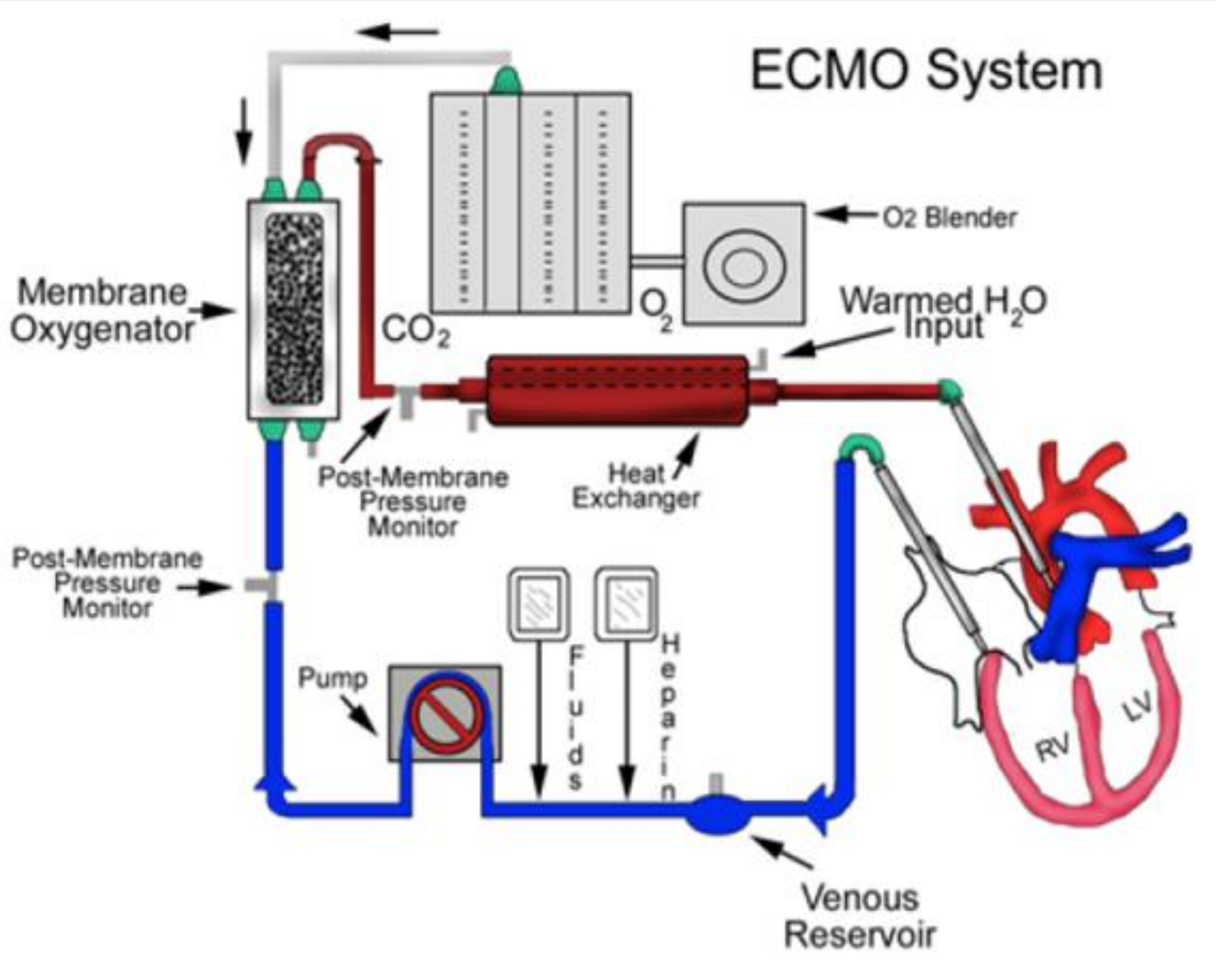
Ted Hoenke, Nathan D. DeBruine MD, Kevin J. Boluyt, Bryan S. Judge MD, Kristin Sternhagen RN
Michigan State University College of Human Medicine, Department of Emergency Medicine, Grand Rapids, Michigan

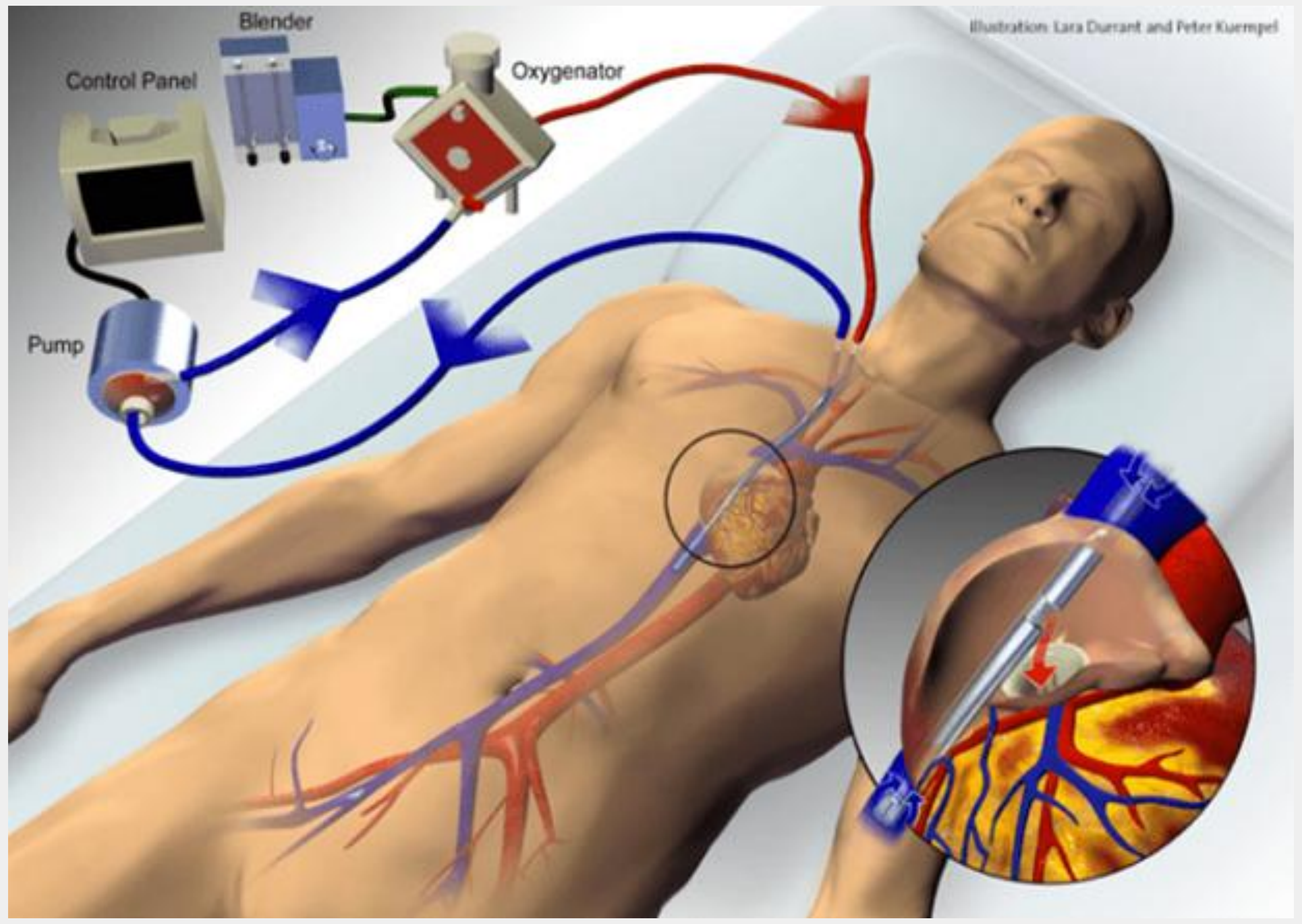
Introduction

- Amlodipine is a dihydropyridine calcium-channel blocker most often used in the treatment of essential hypertension.
- Amlodipine toxicity primarily causes cardiovascular effects through L-type calcium channel blockade both in the vasculature and myocardium.
- A 22-year-old male presented with severe amlodipine toxicity after ingesting 595 mg of amlodipine.
- This case was refractory to multiple medical therapies.
- Venoarterial extracorporeal membrane oxygenation (VA-ECMO) and continuous renal replacement therapy (CRRT) were successfully used in this patient to restore oxygen perfusion and allow intrinsic drug metabolism and elimination.

Patient Description

- A 22-year-old man (75 kg) with a history of hypertension and human immunodeficiency virus presented to a rural emergency department following an intentional ingestion.
- He ingested 119 tablets of Amlodipine, dosed at 5 mg per tablet, for a total of approximately 8 mg/kg at 595mg.
- The patient was initially hemodynamically stable with a Glasgow Coma Scale score of 15, but subsequently developed shock requiring norepinephrine, vasopressin, and dobutamine.
- Calcium chloride infusion was initiated, and insulin was provided per euglycemic protocol.
- An electrocardiogram showed normal sinus rhythm with a first-degree heart block.
- The patient's creatinine and lactic acid levels began to rise. With progressing vasodilatory shock, he was transferred to the ICU of a tertiary care center.





Intervention

- Despite resuscitation attempts with multiple medical therapies, the patient continued to decompensate with lactic acid of 17.0 mmol/L and bicarbonate of 5.8 mmol/L approximately 28 hours after ingestion.
- An echocardiogram revealed an ejection fraction of 70%.
- Continuous renal replacement therapy was initiated for management of refractory metabolic acidosis.
- Because of worsening acidosis and refractory vasodilatory shock, the patient was placed on VA-ECMO (Figures).
- With VA-ECMO the patient's hemodynamic parameters improved.
- The patient was decannulated from VA-ECMO on hospital day 4, with vasopressors weaned off completely by hospital day 5.
- On hospital day 6, the patient was extubated and weaned to room air.
- His hospital stay was prolonged due to the development of pneumonia caused by Serratia marcescens.
- On hospital day 22, the patient had a normal mental status and was discharged to a medical psychiatric facility.

Conclusions

- This case underscores the importance and efficacy of quickly providing aggressive therapies for patients with life-threatening amlodipine toxicity.
- VA-ECMO should be considered early in cases of severe amlodipine toxicity, particularly with refractory vasoplegia despite prompt medical resuscitation.