Prediction of hemodynamic impact of the venoarterial extracorporeal membrane oxygenation (ECMO)

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Abstract—The venoarterial extracorporeal membrane oxygenation (ECMO) has been the last resort and extremely useful in cardiogenic shock with respiratory failure. However, how the ECMO interacts with the native hemodynamic condition remains poorly understood. We developed a circulatory equilibrium framework where we represented cardiac performance by the cardiac output (CO) curve, while venous returning function by the venous return surface (VRS) as functions of right and left atrial pressure (PRA and PLA, respectively). Incorporating the impact of ECMO into this framework enabled us to predict the total CO (=LV CO + ECMO flow), P LA, and P RA reasonably well. The proposed framework is capable of predicting the impact of ECMO on hemodynamics, and is useful in safety management of patients on ECMO.

I. Background

The venoarterial extracorporeal membrane oxygenation (ECMO) has been the last resort and extremely useful in cardiogenic shock with respiratory failure. However, how the ECMO interacts with the native hemodynamic condition remains poorly understood. That is why we sometimes deal with unexpected pulmonary edema in clinical settings[1]. The aim of this study is to quantitatively predict the hemodynamic impact of ECMO and avoid evitable complications.

II. Theoretical analysis

We developed a circulatory equilibrium framework where we represented cardiac performance by the cardiac output (CO) curve, while venous returning function by the venous return surface (VRS) as functions of right and left atrial pressure (PRA and PLA, respectively)[2]. The intersection between the CO curve and VRS defines the circulatory equilibrium. Incorporating the ECMO into this framework indicates that the ECMO shifts the VRS downward by k·COECM (k: constant, COECM: ECMO flow). In contrast, the ECMO increases arterial pressure independent of left ventricular (LV) CO, thereby shifts the LV CO curve (COLV) downward by COECM(1-EFE) (EFE: effective ejection fraction). Equilibrating the downward-shifted COLV curve and VRS gives COLV, PRA, and PLA.

III. Methods and results

In 9 anesthetized dogs, we isolated the carotid sinuses and vagotomized to abolish baroreflex and then created myocardial infarction. We changed COECM stepwise at 3 levels (control±20%) and derived the native CO curve. We altered COECM from 0 to 200% of baseline CO and predicted total CO (=COLV+COECM), P LA and P RA. The predicted total CO, P LA, and P RA matched reasonably well with those measured as shown in Fig.1. ECMO decreased P LA, while increased P LA when LV function was severely compromised. In Fig.2, numerical analysis indicated that the high COECM and poor LV/RV function are prerequisite to elevate P LA.

IV. Conclusion

The proposed framework is capable of predicting the impact of ECMO on hemodynamics, and is useful in safety management of patients on ECMO. ECMO could worsen pulmonary edema in patients with severely compromised cardiac function. We can cope with coincident pulmonary edema beforehand using the proposed framework.

REFERENCES
