## P#16

Abstract Title:	Does Driving Pressure Matter During ECLS For Trauma With Single Lung Ventilation?
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Objective:	<ol> <li>Describe the effect of low-flow CO2 removal on driving pressure after chest trauma.</li> </ol>
Abstract:	Introduction: Increased ventilator driving pressure has been shown to be significantly associated with increased mortality in patients with acute respiratory distress syndrome (ARDS). Driving pressure has also been shown to be the ventilator variable significantly associated with mortality risk. Extracorporeal Life Support (ECLS) has an increasing role in critical care and had been proposed as a treatment modality for trauma-induced ARDS. Veno-venous (VV) ECLS for trauma permits lung rest and reduction in mechanical ventilator settings. We previously demonstrated that VV ECLS, specifically extracorporeal CO2 removal (ECCO2R) at low, dialysis like flows, enabled a 50% reduction in MV settings in uninjured mechanically ventilated anesthetized swine over 72 hours of intensive care unit (ICU) care. Recently we reconfirmed the above lung protective capabilities of ECCO2R in a model of acute respiratory distress syndrome (ARDS) due to severe smoke inhalation and 40% total body surface area burns. In this study we undertook on assessment of the distribution of driving pressure during VV ECLS for support of animals with ARDS due to trauma, hemorrhage and damage control surgery with right sided pulmonary hilum clamping.
	<b>Methods</b> : Sixteen female Yorkshire swine ( $54.5 \pm 13.6$ kg) were anesthetized, sedated, and mechanically ventilated. All animals received right sided pulmonary contusion via modified captive bolt stunner, right sided tube thoracostomy to 25 cm H2O suction, followed by manual hemorrhage of 12 mL/kg/min to a mean arterial pressure of 40 mmHg. Animals then received a 30-minute hemorrhagic shock period. At the end of the shock period, animals received right sided thoracotomy and hilum clamping, resulting in a functional pneumonectomy. Following hilum clamping, animals received fluid resuscitation of lactated Ringers solution (three times the shed blood volume), followed by return of shed

blood. Animals were then randomized to two groups: Standard of Care (SOC, n=8), or VV ECLS (ECLS, n=8). SOC animals received continued fluid resuscitation and, if needed, vasopressor support. ECLS animals received left jugular cannulation with a 23 Fr dual-lumen catheter (Avalon Elite, Maquet/Getinge Group), followed by ECLS support via the Cardiohelp ECLS system (Maquet/Getinge Group). The study continued for 24 hours after completion of blood transfusion. Driving pressure was calculated as  $\Delta P= PPLAT-PEEP$  at baseline, after contusion (pCont), after hemorrhage (pHem), after shock (pShock), after pneumonectomy (pPneumo), after transfusion (pTrans), and after ECLS initiation (ECLS animals) or 1 hour after transfusion (SOC animals) (pECLS / 1hr). Data was collected every three hours until study completion. Statistics were performed by SAS, Cary, NC. Data is reported as mean  $\pm$  SEM, significance accepted at p < 0.05.

**Results**: Five of eight SOC animals survived to study end (38% mortality at 24 h). Seven of eight ECLS animals survived to study end (13% mortality).  $\Delta P$  at baseline was 14.88  $\pm$  1.11 cm H2O vs. 14.83  $\pm$  1.17 for SOC vs. ECLS, respectively. Throughout the study, there was trend towards higher ΔP in the ECLS group which was significant only at the end of the study whereas for SOC increases were documented at pHem, 6, 15, 21, 24 hrs. At study end,  $\Delta P$  was 22.0  $\pm$  6 in the SOC group vs. 35.75  $\pm$  5.31 in the ECLS group. This difference in  $\Delta P$  did not reach significance between groups (Figure 1). In the ECLS group, ECLS blood flow ranged from 1.25  $\pm$  0.11 Lpm at initiation to 1.82  $\pm$  0.2 Lpm at end of study. There was no correlation between ELCS blood flow and  $\Delta P$ . There was a strong correlation of  $\Delta P$  to peak inspiratory pressure (r2 0.923, p < 0.0001), with weaker yet significant correlations to dynamic compliance (r2 -0.660, p < 0.0001) and airway resistance (r2 0.714, p < 0.0001). Weak correlation was also seen with respiratory rate (r2 0.600, p < 0.0001). There was no significance between minute ventilation required between groups throughout the study, although ECLS animals received on average 60% lower minute ventilation than SOC animals after initiation of ECLS therapy. Minute ventilation was very weakly correlated with  $\Delta P$  (r2 0.395, p < 0.0001). Tidal volume/kg started at 10 mL/kg for all animals and was reduced to 7.41 ± 1.01 in SOC animals and  $5.5 \pm 0.49$  in ECLS animals by the end of study.

**Conclusions**: Animals treated with VV ECLS showed lower mortality. Despite larger reductions in delivered minute volume and reductions in tidal volume/kg in the animals that received VV ECLS therapy we did not observe a group difference in driving pressures in this study. Changes in driving pressure while utilizing ECLS after trauma may be related to other factors not such as fluid status, single lung ventilation and the opening of the thoracic cavity with hilum clamping. Next, we will carry

out this work in an extended model of 72-hour model of prolonged field care culminating in simulated aeromedical evacuation at altitude.

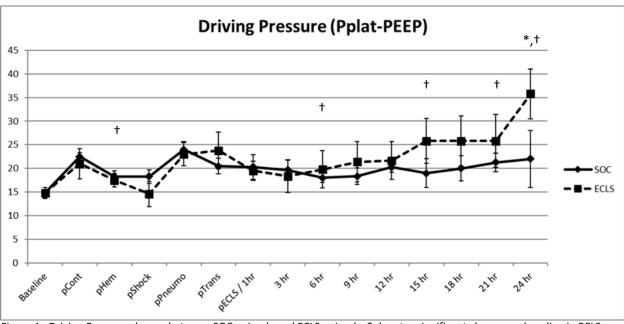


Figure 1. Driving Pressure change between SOC animals and ECLS animals. \* denotes significant change vs. baseline in ECLS animals, † denotes significant change vs. baseline in SOC animals. Both groups significance at p<0.05.