P029
3-DIMENSIONAL CARDIOGENIC SHOCK SIMULATOR PHASE I: SUCCESSFUL DEVELOPMENT OF A LARGE ANIMAL CARDIOGENIC SHOCK MODEL

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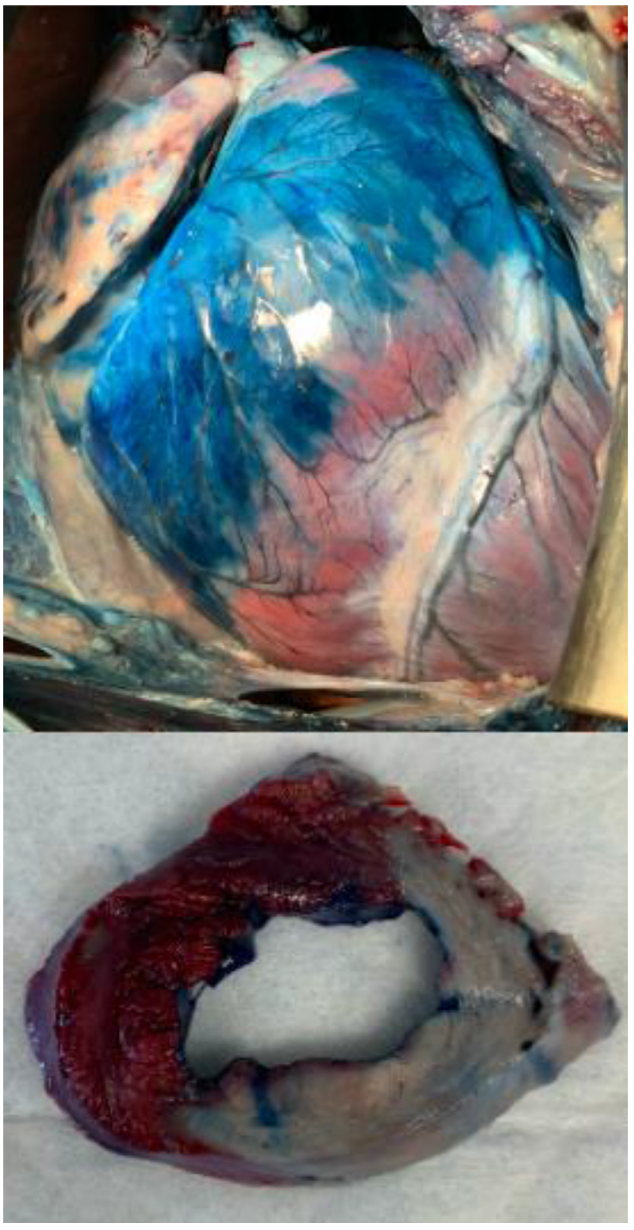
BACKGROUND: Post-myocardial infarction (Post-MI) cardiogenic shock (CS) is a serious life-threatening condition that requires complex coordinated care. Despite this, early mortality remains as high as 50%, likely owing to still inadequate understanding of pathophysiology and the potential benefits of percutaneous ventricular assist devices (pVADs). Although all pVADs increase circulatory flow, they vary in their ability to help restore cardiac pump function. Additionally, shortcomings in terms of our understanding of the optimal timing of such devices in STEMI and shock algorithms likely contributes to the high mortality. Currently, preclinical pVAD evaluation is limited by the need for animal research where the induction of CS is often lethal before devices can be tested. We therefore plan to develop a high-fidelity simulator to facilitate device evaluation and help train clinical teams. The first phase to this program requires the elaboration of a stable animal post-MI CS model that can generate the physiologic data necessary for simulator validation.

METHODS AND RESULTS: Using 10 anesthetized 65-75kg female pigs, we sought to induce a large anterior infarct by percutaneous balloon occlusion of the left anterior descending artery with a provision for ethanol injection if CS was not observed within 2 hours of balloon occlusion. Continuous physiologic monitoring was ensured in order to record right and left heart and arterial pressures, mixed venous oxygen saturation, cardiac output, and serum lactate levels. Indices of myocardial contractility and relaxation were calculated at regular intervals. At the end of the experiment, Evans Blue was injected into the coronary circulation in order to determine the size of the myocardium at risk. The explanted heart was then stained with TTC mitochondrial stain and fixed with formaldehyde to determine the size of the infarct. A rate of survival of the anterior infarct of ≥50% and a rate of induction of CS in at least 50% of the survivors was considered necessary for the viability of the program. We successfully induced myocardial infarction in all 10 pigs and 5 pigs survived to develop cardiogenic shock.

CONCLUSION: While challenging, we successfully demonstrated that a large animal model of cardiogenic shock is possible and stable enough to generate the physiologic data necessary for the development of a CS simulator. The second phase will consist of the collection of detailed physiological data in a CS state with and without different pVAD support.



P030
CLINICAL CHARACTERISTICS AND OUTCOMES OF COVID-19 PATIENTS WITH MYOCARDIAL INJURY: ONE-YEAR EXPERIENCE IN VANCOUVER, CANADA

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BACKGROUND: The coronavirus 2019 (COVID-19) pandemic has caused unparalleled public health crisis worldwide. Myocardial injury in acutely ill hospitalized COVID-19 patients was associated with higher mortality and worse clinical outcomes. However, Canadian data on COVID-19 and

myocardial injury is not available, and this is paramount to inform quality of care and direct efficient resource allocation.

METHODS AND RESULTS: We conducted a retrospective study of patients with COVID-19 with myocardial injury admitted to two quaternary hospitals in British Columbia; Vancouver General Hospital between March 2020 – February, 2021 and St. Paul's Hospital between April – December, 2020. Myocardial injury was defined as troponin elevation greater than 99% upper limit of normal (normal troponin-I < 0.05 ng/L, troponin-T < 9 ng/L in female, < 14 ng/L in male) on admission or during hospitalization. Baseline demographics, laboratory results, and in-hospital outcomes were collected. The study was approved by our institutional research board. We included 494 COVID-19 patients in the study. The mean age was 63.4 years, and 58.9% were men. The prevalence of myocardial injury was 37.2%. Sixty-five patients (13.2%) died during hospitalization, of which 49 (9.9%) had myocardial injury. Patients with myocardial injury were more likely to require mechanical ventilation (31.1% vs. 12.1%, p < 0.001) and inotropic support (2.7% vs. 0.0%, p < 0.01). They had higher mortality (26.8% vs. 5.2%, p < 0.001), shock (11.5% vs. 1.0%, p < 0.00), cardiac arrest (15.3% vs. 2.9%, p < 0.001), heart failure (7.1% vs. 0.3%, p < 0.001), stroke (2.7% vs. 0.3%, p=0.019), and significantly longer length of hospitalization (23.7 vs. 13.6 days, p < 0.001) than patients without myocardial injury. Four myocardial injury patients were diagnosed with ST-elevation or non-ST elevation MI.

CONCLUSION: Myocardial injury was frequent amongst hospitalized COVID-19 patients in British Columbia, and was associated with worse clinical outcomes and increased length of hospital stay.

P031
LACTATE CLEARANCE PREDICTS MORTALITY IN CARDIOGENIC SHOCK: A SYSTEMATIC REVIEW AND META-ANALYSIS

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BACKGROUND: Despite identification of prognostic markers of cardiogenic shock (CS), few markers demonstrate suitability as therapeutic targets that can be used to guide therapeutic decisions. Lactate clearance is a potential treatment target, but its role in guiding CS management has not been defined. In this systematic review and meta-analysis, we compared lactate clearance in CS survivors versus non-survivors.

METHODS AND RESULTS: In December 2020 we performed a systematic search of studies evaluating survival and blood lactate levels in patients with CS. We reviewed the mean difference in lactate clearance at 8-, 12-, and 24-hours between survivors and non-survivors. The results were pooled and a meta-analysis was performed using a random effects model comparing the mean difference in lactate clearance between survivors and non-survivors. We screened 3,150 titles and abstracts and reviewed the full text of 123 studies. Eleven studies were selected for inclusion with a total of 1,585 patients. The time-to-mortality rates included ICU mortality (N = 1), in-hospital mortality (N = 4), and 30-day mortality (N = 6). The median sample size was 87 (interquartile range [IQR] 43-139) and the median mean patient age was 56.2 years (IQR 54-57.5). Lactate levels were evaluated at 8 hours in 7 studies and at 24 hours in 9 studies. In studies that monitored lactate levels at 8 hours, the median percentage lactate clearance was 21.9% (IQR 14.6-42.1%) and 0.6% (IQR -3.7-14.6%) in survivors and non-survivors, respectively. When pooled, the mean difference in percentage lactate clearance at 8-hours was 17.3% (95% CI 11.6-23.1%; P < 0.001). Similarly, the mean percentage lactate clearance at 24-hours ranged between 50-80% (median 60.7%, IQR 58.1-76.3%) in survivors, and between, nh69.7-70% (median 40.3%, IQR 30.2-55.8%) in non-survivors. When pooled, the mean difference in 24-hour lactate clearance between survivors and non-survivors was 27.9% (95% CI 14.1-41.7%; P < 0.001). There was no statistically significant evidence of heterogeneity between studies that reported 8-hour lactate levels (I2 = 0%); however, significant statistical heterogeneity was noted between studies reporting 24-hour lactate levels (I2 = 78%).

CONCLUSION: Lactate clearance in patients with CS is a useful prognosticator of mortality. The absence of a relative reduction in lactate within the first 8 hours of treatment is associated with an increased risk of death. Prospective studies with management protocols designed to target lactate clearance are needed to verify this assumption.