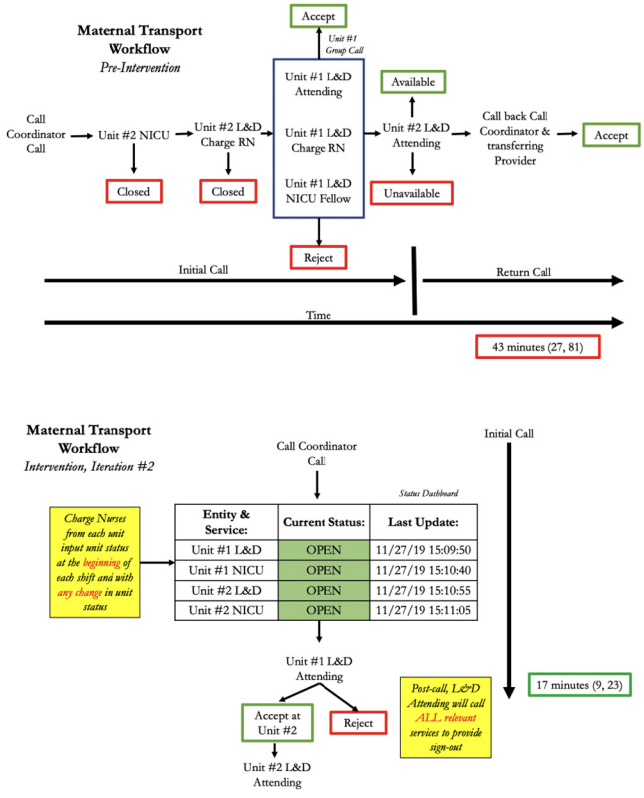
the receiving unit verbalized transfer approval. A new workflow and intervention were designed and implemented, including the creation of an online-dashboard reflecting the real-time status of each unit needed for maternal and/or neonatal acceptance. Following a 3-month period of execution, post-analysis was performed.

RESULTS: 59 maternal transports occurring between May - Aug '19 were included in initial analysis. Median time from the first call with the transporting hospital until verbal 'acceptance' was 43 minutes (IQR 27-80). Following 3-months of intervention deployment and utilization, post-intervention analysis was performed: 81 maternal transports occurred with the median time of "acceptance" decreasing to 17 minutes (IQR 9-23), representing a 26-minute reduction (60%) from baseline to post-intervention.

CONCLUSION: A real-time, synchronous dashboard can coordinate disparate unit's response to incoming maternal transports resulting in optimized acceptance times. Although clinical outcomes are currently lacking, decreasing maternal transport time to an appropriate care facility should greatly improve both maternal and neonatal outcomes.



656 Fetal hypoxia induced by partial maternal aortic occlusion is tolerated in a lamb model

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OBJECTIVE: Hemorrhage is a leading cause of maternal mortality. Resuscitative endovascular balloon occlusion of the aorta (REBOA) has been used for hemorrhage control in obstetrical patients, however the fetal effects are unknown. Preliminary data from a pregnant ewe model showed that fetal physiologic changes from partial aortic occlusion (pREBOA) first occur at mean placental arterial pressure (pMAP) 40mmHg, however data are lacking on fetal neurological outcomes. We aimed to further evaluate the effects of decreased pMAP on the fetus in a sheep model.

STUDY DESIGN: A REBOA catheter was placed in the infrarenal aorta of four gravid term ewes. pMAP was approximated by maternal femoral MAP. The REBOA balloon was inflated to decrease pMAP to 40mmHg, then decreased in 5mmHg increments to a fetal SaO2 of 15%. Fetal hemodynamics, SaO2, pH, and lactate were monitored via right fetal carotid arterial lines. At fetal SaO2 15%, the REBOA was deflated followed by a recovery period. pREBOA was then repeated twice as part of a separate study. Data from the first round are presented here. Post-mortem fetal brain magnetic resonance imaging (MRI) was performed to evaluate for ischemia.

RESULTS: Mean baseline pMAP was 57.5±6.2mmHg and it was decreased to 40mmHg (n=1), 30mmHg (n=2), and 25mmHg (n=1) prior to recovery. Fetal SaO2 was significantly lower than baseline (58.5±7.4%) at each decreased pMAP ($p<0.0001$, Fig 1A). Fetal heart rate did not significantly increase until pMAP 25mmHg (baseline 133.5±8.8 vs. 156±14.1, $p=0.001$, Fig 1B). Fetal pH and lactate did not significantly change until pMAP 30mmHg (pH 7.22±0.02 vs. 7.14±0.1, $p=0.01$; lactate 1.9±0.4 vs. 3.5±2.8, $p=0.004$). After 3 rounds of hypoxia, there was no evidence of ischemia on fetal brain MRIs (Fig 2).

CONCLUSION: Fetal SaO2, heart rate, pH, and lactate did not change significantly until a relatively low pMAP. After three rounds of progressive hypoxia from pREBOA, there was no evidence of fetal brain ischemia on MRI. Future studies in a survival model are needed to evaluate neurologic function after maternal pREBOA.

Figure 1: Fetal heart rate at each decreasing placental MAP (A); correlation of maternal placental MAP and fetal SaO_2 (B)

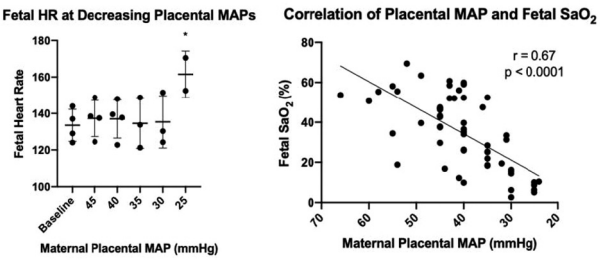

Figure 2: Representative fetal brain MRIs showing no evidence of ischemia. A) T1-weighted, B) T2-weighted, and C) Diffusion-weighted imaging (DWI).