ABSTRACT	TITLE	AUTHOR(S)	INSTITUTION	
#8	Clamp, Crash, Recover: Anesthetic Management of Porcine Orthotopic Liver Allotransplantation	Justin Silvey (1) Lisa Halliday (2) Maria Jimena (3)	University of Illinois Chicago	Abstract

ASR-26-8
Clamp, Crash, Recover: Anesthetic Management of Porcine Orthotopic Liver Allotransplantation

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Introduction:
Swine have been used extensively to study transplant immunology and rejection. Although information exists regarding transplant physiology and graft survival, there is a gap in the literature regarding the clinical management of these patients. This presentation will discuss the intraoperative management of a swine orthotopic liver allotransplant survival model performed without venovenous bypass.

Methods:
The transplant recipients were premedicated with 10 mg/kg ketamine and 0.2 mg/kg medetomidine IM. Intravenous catheters were placed in the lateral ear veins and external jugular vein. A percutaneous femoral artery catheter was used to monitor invasive blood pressure. Propofol was given IV to effect for intubation. Anesthesia was maintained with 1% isoflurane, mechanical ventilation, and lidocaine/ketamine or hydromorphone CRI. Preoperative gastrointestinal protectants consisted of 0.2 mg/kg ondansetron IV, 1 mg/kg maropitant IV, and 1 mg/kg pantoprazole IV. Regional analgesia consisted of a transversus abdominus plane block and a splash block using 1 mg/kg bupivacaine mixed with 0.03 mg/kg buprenorphine. Intraoperative interventions for hemodynamic support consisted of physiologic fluids (Plasma-Lyte), vasopressors (vasopressin, norepinephrine, and phenylephrine), and whole blood acquired from the donor.

Results:
Orthotopic liver transplantation has numerous intraoperative challenges that are unique compared to other major survival surgeries. This procedure is divided into three surgical phases: approach/dissection, anhepatic, and reperfusion/recovery. The anhepatic phase poses immediate life-threatening complications arising from severe hypotension, while the reperfusion/recovery phase can cause metabolic derangements.

Immediately prior to the anhepatic phase, vasopressors are used to increase mean arterial pressure (MAP) to 120 mmHg to mitigate the severe hypotension that follows vascular clamping. Vasopressin (0.3-0.4 U/kg/hr) is the primary agent used to create this hypertensive state. Norepinephrine (3-60 ug/kg/hr) serves as an adjunct with 10 ug IV boluses as needed.
The anhepatic phase is initiated once the portal vein, cranial vena cava, caudal vena cava, and hepatic artery are clamped. During the anhepatic phase, efforts are directed towards maintaining a MAP greater than 50 mmHg. Pressor use is continued, fluids are delivered at maximum rates utilizing pressure bags, and whole blood transfusion is initiated. Vasopressin and norepinephrine rates typically reach maximal dosing, with intermittent 10 ug norepinephrine and 100 ug

phenylephrine boluses used to rescue the MAP. At the end of the anhepatic phase, metabolic acidosis and reperfusion injury are mitigated with 1,000 mg calcium gluconate and 100 mEq bicarbonate. Risk of hyperacute rejection is reduced with 250 mg methylprednisolone IV. Arterial blood gas samples are drawn and analyzed every 30 minutes while the patient is weaned from vasopressors and mechanical ventilation.

Conclusions:
Survivability in a porcine orthotopic hepatic allotransplantation model is heavily dependent on aggressive hemodynamic support and minimization of anhepatic time. If the anhepatic phase is less than 20 minutes and MAP is maintained above 45 mmHg, the prognosis is markedly improved. Vasopressin and norepinephrine provide the most effective hemodynamic support during vascular clamping. However, aggressive fluid resuscitation, whole blood transfusion, and correction of metabolic derangements are also critical for a successful recovery.