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"Ischemia-Reperfusion Injury after CPR in Porcine Model of Severe Hemorrhagic Shock"

The current American Heart Association guidelines for pulselessness tell health care providers to start cardiopulmonary resuscitation (CPR) regardless of the underlying cause of the pulselessness. However, previous studies in animals have shown that CPR is not beneficial and may be harmful in traumatic pulseless electrical activity (TPEA). TPEA occurs when a trauma victim's heart beats, but there is no central pulse due to loss of blood from hemorrhage. In these cases, since the heart continues to contract in an organized way, it makes sense that external chest compressions would not be productive in improving tissue perfusion.

While investigating the effects of CPR on perfusion in TPEA victims, our lab showed that in a porcine model of severe hemorrhagic shock, external chest compressions did not improve brain and skin perfusion; it also supported existing evidence that chest compressions lower diastolic blood pressure and do not raise overall mean arterial pressure. Because the myocardium is supplied with blood during diastole, we hypothesize that a lower diastolic pressure may contribute to worse ischemia-reperfusion injury on the heart in CPR-treated TPEA victims compared to non-CPR treated victims. The objective of the present study is to measure the extent of ischemia-reperfusion injury in CPR-treated vs non-CPR-treated TPEA victims using plasma and cardiac tissue samples from the preliminary experiment.

Ischemia-reperfusion injury is the phenomenon in which after a period of insufficient blood flow to an organ (ischemia), the return of blood to the area (reperfusion) results in worse tissue damage than the ischemia itself. Syndecan-1 is a glycoprotein of the endothelium glycocalyx that is cleaved and released into the blood in ischemia-reperfusion injury. Thus, Syndecan-1 levels can be used as a biomarker of ischemia-reperfusion injury.

In the present study, the extent of ischemia-reperfusion injury on the heart in CPR vs. non-CPR-treated TPEA swine was investigated by measuring Syndecan-1 levels in the cardiac tissue samples. The CPR group showed higher levels of Syndecan-1 in the cardiac tissue; however, these results did not achieve statistical significance, although they were very close to being significant. A higher amount of Syndecan-1 in the cardiac tissue of the CPR group has two possible, opposing explanations: Less Syndecan-1 was cleaved from the cardiac blood vessels and thus less ischemia-reperfusion injury occurred, or there was worse body-wide ischemia-reperfusion injury and thus more cleaved Syndecan-1 was present systemically in the blood of the heart compared to the non-CPR group. To clarify the results, Syndecan-1 levels will need to be measured in the plasma samples. If there is a higher level of Syndecan-1 in the plasma of the CPR group, that will support worse ischemia-reperfusion injury in CPR-treated TPEA swine.