Hypertonic saline does not restore VO_2/DO_2 abnormality during vasomotor shock induced by IL-1β.

Yasuyuki Kuwagata, Hisashi Sugimoto, Takeshi Shimazu.

Department of Acute Critical Medicine (D-8), Osaka University Medical School

Summary

We have shown that interleukin-1β (IL-1β) induced a vasomotor shock and impaired oxygen consumption (VO_2)/oxygen delivery (DO_2) relation by increasing the slope of the supply-independent line in rabbits. In the present study, we investigated the effect of hypertonic saline (HS) on the vasomotor shock induced by IL-1β. Experiment 1: six rabbits were randomly divided into two groups (n = 3, each) and given intravenously either 4 ml/kg of HS (2,400 mOsm/L NaCl) or 4 ml/kg of normal saline following i.v. administration of 10 μg/kg of IL-1β. All rabbits developed circulatory shock in resoponse to IL-1β. The HS administration temporarily increased cardiac output, but had no effect on the decreased mean arterial pressure. Experiment 2: six rabbits were randomly divided into two groups (n = 3, each) and given 10 μg/kg of IL-1β follwed by 4 ml/kg of HS (IL-1+HS) or vehicle alone followed by 4 ml/kg of normal saline (Ctrl) intravenously. After baseline measurements, all rabbits were subjected to stepwise cardiac tamponade to reduce DO_2 down to 5 ml/kg/min by inflating a balloon placed into the pericardial sac. The VO_2/DO_2 relation was analyzed by the dual-line method. The IL-1+HS group showed significantly greater slope of the supply-independent line than Ctrl (IL-1+HS: $y = 0.12x + 6.2$, Ctrl: $y = 0.06x + 8.5$) during the stepwise decrease in DO_2. These results indicate that the intravenous HS administration may increase DO_2 to the tissue, but does not restore the VO_2/DO_2 abnormality during the vasomotor shock.