THE EFFECT OF ATP ADMINISTRATION IN IRREVERSIBLE SHOCK. Samir M. Talaat *, Walter H. Massion and John A. Schilling. Depts. of Surgery & Anesthesiology, Univ. of Okla. Medical Center, Oklahoma City, Okla.

Studies of myocardial metabolism during hemorrhagic shock have shown that rapid dephosphorylation of high energy phosphate will occur as a result of myocardial hypoxia. The loss of ATP and the destruction of coenzymes which require ATP for resynthesis may contribute to the irreversibility of shock after restoration of blood volume. Three groups of dogs were heparinized and connected to a reservoir via the femoral artery. Mean arterial pressure was maintained at 35 mm Hg by adjusting the reservoir level. The shed blood was retransfused after 5 hours of shock or after spontaneous uptake of 20% of the shed volume. In the control group blood loss averaged 47.2 ml/kg; the survival rate was 22%. The second group received 6.2 mg/kg ATP-Na with the retransfused blood and an equal amount IM 2 hours after retransfusion. Blood loss in this group was 49.8 ml/kg; 55% of the animals survived. The third group received 6 mg/kg ATP-Na IM one hour before the experiment and an equal amount in the retransfused blood. Average blood loss was 52.5 ml/kg and the survival rate was 80%. A fourth splenectomized group is currently under study. The results of this study indicate that administration of adenosine triphosphate preceeding hemorrhage may prevent or reduce the incidence of the irreversible phase of shock in this standard preparation.

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BRONCHOCONSTRICTION IN EXPERIMENTAL AUTOLOGOUS PULMONARY EMBOLISM. G. Tanabe*, V. Rege*, D. Thomas* and M. Stein. Beth Israel Hospital and Harvard Medical School, Boston, Massachusetts.

Fresh autologous peripheral venous thrombi were released to the lungs of 26 anesthetized spontaneously breathing dogs. Embolization produced significant increases in total lung resistance, lung elastance, respiratory rate and arterial-alveolar (a-A) CO_2 tension difference which were unrelated to changes in lung volume. Intravenous heparin (5000 units) administered 30 minutes before release of preformed thrombi completely prevented increases in lung resistance and elastance although post embolic tachypnea and increases in the a-A CO_2 tension difference still occurred. Acute bronchoconstriction produced by intravenous histamine, acetylcholine and serotonin was not effected by the prior injection of heparin. When l - methyl lysergic acid butanolamide (MLA), an antiserotonin agent, was administered intravenously during embolization, the increases in lung resistance and elastance were completely prevented. Examination of lung emboli immediately after release revealed that significant amounts of thrombin could be eluted. The following conclusions can be made: (l) The effects of heparin and MLA demonstrate that neither the physical presence of emboli in the lung nor the reduced alveolar CO_2 tension are responsible for post embolic bronchoconstriction; (2) The failure of heparin to prevent the bronchoconstriction due to intravenously injected serotonin indicates that heparin does not block this action of serotonin directly; (3) Thrombin on fresh pulmonary emboli induces the release of serotonin from blood platelets with subsequent bronchoconstriction; and (4) Heparin prevents the release of serotonin by its anti-thrombin action.