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burn patients (average burn 20% TBSA) to that of normal volunteers by flow cytometry using specific monoclonal antibodies. Our preliminary data suggested that L-Selectin, Sialyl Lewis-X and CD18 expression of PMN were depressed in burn patients after the first week. On the other hand, CD11b expression increased significantly within 24 hrs after injury but returned to normal levels after seven days. Expression of CD11a and CD11c were not altered by burn injury. In contrast to PMN L-Selectin expression, monocyte L-Selectin levels returned to near normal after 7 days. Cell surface expression of monocyte Sialyl Lewis-X and CD11/CD18 followed the same pattern as PMN. These results suggest that the depression of selectin expression in PMN and monocytes may be responsible for the observed diminution in leukocyte-endothelial cell interactions in burn injury.

34 ENDOGENOUS OPIOID PEPTIDES INTENSIFY HYPOTENSION FOLLOWING HEMORRHAGIC SHOCK IN RAT BRAIN. L. Fan and J.S. Hua, Div. of Neurosurgery, Univ. of Pennsylvania, Philadelphia, PA 19104 and Dept. of Physiology, Beijing Med. Univ., Beijing 100083, P.R. China.

Endogenous opioid peptides in brain have been shown to contribute to the hypotension observed during hemorrhagic shock. In the present study, we investigated effects of modulation opioid receptor subtypes on hemorrhagic hypotension in rat brain. Male Wistar rats were anesthetized with pentobarbital. Hemorrhage was induced by bleeding rat through a arterial catheter, and mean arterial pressure (MAP) was maintained at 45 mmHg for 30 min without resuscitation. Immediately following hemorrhage, the following compounds were intracerebroventricularly (iev) administered (n=8 per group): 8-4-8 peptide receptor antagonist, CI1174-646 (2 μg); κ-opioid receptor antagonist, nor-BNI (10 μg); met-enkephalin antiserum (1:2400, 10 μl); leu-enkephalin antiserum (1:1500, 10 μl); dynorphin antiserum (1:15000, 5 μl) and normal saline (10 μl). MAP and heart rate recovery following hemorrhagic shock were recorded for 60 min. An accelerated recovery of MAP following hemorrhagic shock was found after administration of CI1174-864 (p<0.05), nor-BNI (p>0.01), met-enkephalin antiserum (p>0.01) and dynorphin antiserum (p>0.01) but not leu-enkephalin antiserum when compared with saline control animals. Following hemorrhagic shock, no significant change of heart rate was observed in any of the treatment groups when compared with saline. Our results suggest that endogenous met-enkephalin and dynorphin mediate hypotension by activating δ- and κ-opioid receptors in brain following hemorrhagic shock.


With expanded research interest in the development of hypertonic saline/dextran solutions for the treatment of hemorrhagic hypotension, concerns have arisen regarding adverse effects on hemostasis. We have previously reported that infusion of 4 ml/kg 7.5% NaCl/6% Dextran-70 (HSD) in hemorrhaged animals did not adversely affect clotting times or platelet aggregation, nor did it interfere with typing and cross-matching of red blood cells (RBC). To present the blood picture following hemorrhage and HSD infusion more completely, the present study reports complete blood count (CBC) analysis and RBC morphology in rabbits and pigs infused with HSD. Blood was obtained from both euverolnic and hemorrhaged rabbits (8ml/kg) and pigs (17ml/kg) at times up to 7 d after infusion of 4ml/kg HSD. CBCs and platelet counts were determined, and qualitative RBC morphology was evaluated. In both species changes in hematocrit, hemoglobin, RBC, and platelet counts reflected the extent of hemorrhage and the subsequent plasma volume expansion induced by HSD. Infusion of HSD did not significantly affect mean corpuscular volume, mean corpuscular hemoglobin or mean corpuscular hemoglobin concentration; findings consistent with the observed lack of significant morphological changes in RBC size, shape and staining intensity. Interestingly, a transient increase in white blood cell counts was seen, and at 2 to 4 hr after HSD infusion, a marked increase in segmented neutrophils was observed. In general, no anemia was detected, and the data support other observations that HSD infusion should have minimal effects on hemostasis.