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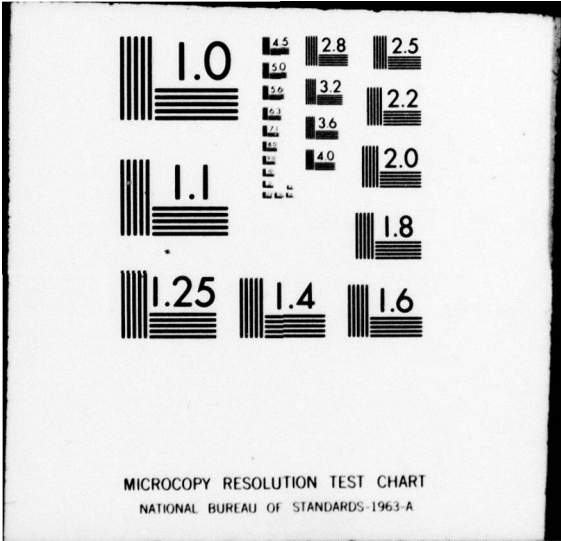
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MYOCARDIAL EFFECTS OF SHOCK

Final Report

July 1973

by

Lester Williams, Jr., M.D.

Supported by

US Army Medical Research and Development Command  
Fort Detrick, Frederick, Maryland 21701

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Boston University School of Medicine  
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Principal Investigator: Lester F. Williams, Jr., M.D.

Background material for the original proposal indicated that one of the pathogenic considerations in shock was a specific humoral factor responsible for myocardial depression (1,2,3,4,5,6). This myocardial depression factor (MDH) was found to originate in ischemic gut; consequently the project focused upon the relative relationship between ischemic intestine and myocardial depressant activity. The factor was never biologically identified in our studies but it was characterized as a small molecular weight (MW 800-1000) peptide that was found in portal vein blood prior to its appearance in systemic blood although it could be measured by bioassay in both locations (7,8). We also identified that this agent had no specific vaso-active properties. Because of difficulty in quantitating the degree of intestinal ischemia, its physiological significance and the production of the systemic effect, a series of studies were conducted. These documented: (a) significant impairment in bidirectional flow of fluid and electrolyte abnormalities in the presence of intestinal ischemia (9,10); (b) although autoregulation and autoregulatory escape could be well documented even in conditions of low pressure and after drug manipulation, these normal physiologic phenomena did not appear to be maintained when intestinal ischemia was extensive (11); (c) by the use of the tetrazolium assay for describing the degree of disruption and monitoring of mucosal and serosal pH, one could predict the degree of intestinal ischemia that had been induced by interruption of the blood supply and thus potentially the viability of the bowel (12). An additional therapeutic approach utilizing cortical steroids was studied experimentally (13). As a result of these studies, including the clinical data provided by several reviews (14,15,16,17,18,19,20,21,22,23,24,25,26,27,28,29,30), the diagnosis and treatment of non-occlusive mesenteric ischemia has been drastically altered from that which had been utilized during the early parts of the investigative research.

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