infection is increased after x-radiation and after cortisone administration are, however, quite different. When an animal is exposed to moderate doses of x-radiation severe injury to the bone marrow and lymphoid tissue occurs. All of the formed blood elements are markedly depressed for 2-3 weeks. The functional capacity of the reticuloendothelial system is disturbed and capillary permeability is increased. The inflammatory response is suppressed due to scarcity of cells. Although cortisone treatment does induce atrophy of lymphoid tissue, it does not impair the function of either bone marrow or reticuloendothelial system. In fact, the number of neutrophils is usually increased during cortisone therapy. The inflammatory response is suppressed largely because of decreased capillary permeability. The increased susceptibility to infection following cortisone therapy appears to be almost secondary to the suppression of the protective inflammatory response(10,11, 12).

Summary. Peritoneal exudates were induced in female rabbits and collected either 1, 5 or 30 days after total body exposure to 400 r and 1 or 5 days after 500 r. Exudate cells from unirradiated rabbits served as controls. Phagocytin was extracted from the exudate cells (heterophils) and tested for bactericidal activity against *Escherichia coli, Salmonella enteritidis, Klebsiella pneumoniae* and *Pseudomonas aeruginosa.* Phagocytin from cells collected 1 day after exposure to 400 or 500 r and 5 days after exposure to 500 r showed a decreased activity against all the test microorganisms. Phagocytin from cells collected 5 days after exposure to 400 r showed only a slight change in activity from controls. A marked increase in phagocytin activity was observed in extracts of cells collected 30 days after exposure to 400 r.

The authors are indebted to Dr. W. S. Moos, Dr. J. W. Yusken and Mr. R. Haas, Radiology Dept., College of Medicine, Univ. of Illinois, for the dosimetry and to Miss Mabel Counelis for irradiating the rabbits.

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Received March 2, 1962. P.S.E.B.M., 1962, v110.

Effect of Postganglionic Mesenteric Sympathectomy in Irreversible Hemorrhagic Shock.* (27473)

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Recent investigation has implicated the gastro-intestinal tract as one of the prime organs responsible for irreversibility of experimental shock. The most striking autopsy finding of irreversible endotoxic or hemorrhagic shock is the diffuse hemorrhagic necrosis of the bowel. Irreversibility and bowel necrosis can be prevented by pretreatment of animals

* Supported in part by a research grant from USPHS (Arthritis and Metab. Dis.).

with adrenergic blocking agents(1-4), or bypass perfusion of the mesenteric circulation (5). Furthermore, survival time can be increased by excising the major portion of the gut prior to induction of shock(6). Lillehei postulated that the shock state is associated with severe vasoconstriction in the splanchnic bed leading to necrosis of the bowel mucosa. The attendant increase in bowel wall permeability brings about lethal changes in plasma composition. If this hypothesis were correct, elimination of vasoconstriction in the splanchnic bed should afford protection from irreversible shock by preventing bowel necrosis.

To test this thesis experimental shock was induced in dogs following abdominal postganglionic sympathectomy, a procedure which selectively deprives splanchnic blood vessels of sympathetic impulses(7,8).

Materials and methods. Thirty adult mongrel dogs (10-15 kg) were premedicated with 1.5 mg/kg of morphine sulphate one hour prior to the experiment. The femoral artery and vein were cannulated with a T shaped glass cannula under sterile conditions using local procaine anesthesia. Fine's method of graded hemorrhage was used to induce and maintain shock(9). The blood pressure was continuously monitored. After heparin (1 mg/kg) was injected into the femoral vein, the animals were bled from the femoral artery into a reservoir placed 40 cm above the level of the right heart thereby maintaining an arterial pressure of about 30 mm Hg. Autopsies were performed on all animals whether they died or were sacrificed. Dogs alive 72 hours after onset of the experiment were considered permanent survivors.

Ten control animals (Group I) were shocked as described. Ten animals (Group II) were treated one hour prior to onset of the hemorrhage with 0.5 mg/kg of dibenzyline. Ten animals (Group III) were subjected to abdominal postganglionic sympathectomy under nembutal anesthesia. The celiac, superior and inferior ganglia were excised and the corresponding arteries were stripped of their adventitia. Seven to 14 days after operation the animals were subjected to hemorrhagic shock in a manner identical to the other 2 groups.

TABLE I. Effect of Dibenzyline Pre-Treatment and Mesenteric Sympathectomy on Hemorrhagic Shock.

Group	Survivals	MBV	MBT	TUT
Controls 10	0	48	53	121
Dibenzyline 10	6	40 $p<.05$*	105 $p=.05$*	219 $p<.025$*
Sympathectomized 10	2	40 $p<.05$*	48 —	170 $p=.01$*

MBV (avg maximal bleeding vol in cc/kg).
MBT (avg maximal bleeding time in min.)
TUT (avg total uptake time in min.)

* Compared to controls.

Results and discussion. The results are expressed in terms of survival, maximal bleeding volume, maximal bleeding time and total uptake time (Table I). Maximal bleeding volume (MBV) is defined as the maximal amount of blood shed by the animal into the reservoir. Maximal bleeding time (MBT) is the interval between onset of the hemorrhage and attainment of MBV. Total uptake time (TUT) represents the period between the time MBV was reached and retransfusion was instituted.

Retransfusion was begun when 40% of the MBV was taken up by the animal. In the event that the animal did not take up 40% of the MBV, retransfusion was started 6 hours after onset of the hemorrhage.

Our results with control dogs and dibenzyline group are similar to those of others (10). There were no survivors in the control group, but 6 of 10 survived with dibenzyline pre-treatment. In the animals that did not survive, the intestines demonstrated the usual hemorrhagic necrosis. In the dibenzyline treated animals, MBV was reduced and MBT and TUT prolonged. Since these animals have bled less (MBV reduced), there must be a higher residual volume of blood which maintains better tissue perfusion.

The sympathectomized dogs resembled the dibenzyline animals in that the MBV was reduced and the TUT prolonged. Although the procedure may have been beneficial to the organism as measured by the moderate protection (2 of 10 animals survived), the results are not significant. The MBT was

identical with the control series. This may indicate that even though the mesenteric vascular bed did not participate in the generalized vasoconstrictive reaction the rest of the vascular system constricts rapidly, and the animals bleed out the available blood at a normal rate of speed. There appeared to be little protection against the bowel changes since in the 8 animals that died there was the usual hemorrhagic intestinal necrosis.

In evaluation of this regional sympathectomy several problems inherent in the procedure itself should be considered. The splanchnic bed is only one segment of the vascular tree participating in vasoconstrictor activity during shock. As such it would be unreasonable to expect from its denervation the same hemodynamic effect as is obtained from chemically induced total sympathetic blockade. The trauma of the denervation procedure and the subsequent diarrhea, which these animals almost invariably exhibit, may have weakened the animal's resistance to the stress of shock. Furthermore, an increased sensitivity of end organs to circulating epinephrine and norepinephrine may have obviated some of the protective effects of the operation. Lastly, while sympathectomy produces vasodilation, it also causes spasm of the intestinal musculature which may compress its blood supply and limit further its beneficial effect.

Nevertheless, this postganglionic mesenteric sympathectomy produced hemodynamic changes in hemorrhagic shock which approached those produced by dibenzyline pre-treatment and yet did not afford the same overall beneficial result. It would appear that dibenzyline protects from hemorrhagic shock by acting on organs other than the intestine.

Summary. A postganglionic mesenteric sympathectomy was performed in a series of dogs who subsequently were subjected to irreversible hemorrhagic shock. Hemodynamic changes resulted which mimicked those produced in a series of dibenzyline pretreated animals but offered no significant improvement in survival when compared to a control series.

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Received April 5, 1962. P.S.E.B.M., 1962, v110.

Metabolic and Serum Lipid Effects of Δ^1-Testololactone.* (27474)

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In atherosclerosis research and cancer chemotherapy the search continues for steroidal derivatives in which the characteristic endocrine effects of the parent compound are lacking or markedly attenuated. Testololactone is reported to be anabolic but not androgenic in the rat(1). Δ^1-Testololactone is reported to be lacking in androgenic effect in women, although it appears to be as useful as testosterone propionate in the treatment of metastatic breast cancer(2). The present investi-

* This work was supported in part by grants from Nat. Heart Inst., U. S. P. H. S., Oklahoma State Heart Assn., Ayerst Laboratories, New York, and Squibb Inst. for Medical Research, New Brunswick, N. J.