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ENDOGENOUS ENDOTOXEMIA DURING HEMORRHAGIC SHOCK IN THE SUBHUMAN PRIMATE

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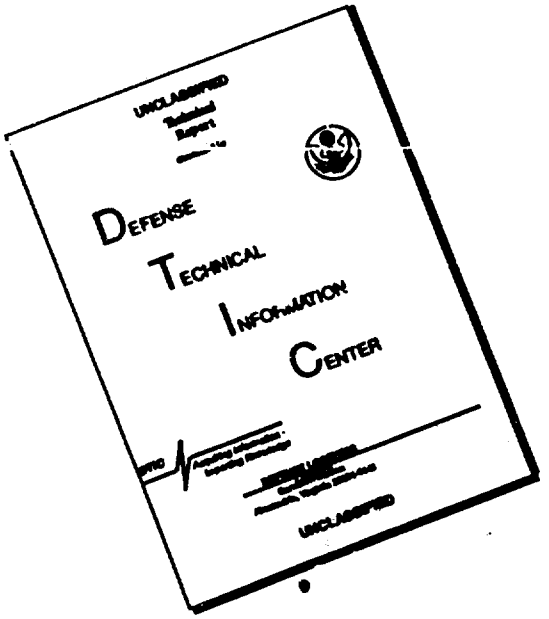

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13. ABSTRACT

Ischemic bowel damage resulting in endogenous endotoxemia is not a common feature of hemorrhagic shock in baboons. The observed sporadic endotoxemia occurs no more frequently during shock than in the baseline period, and is not related to the duration or degree of severity of hemorrhagic shock in this subhuman primate species.

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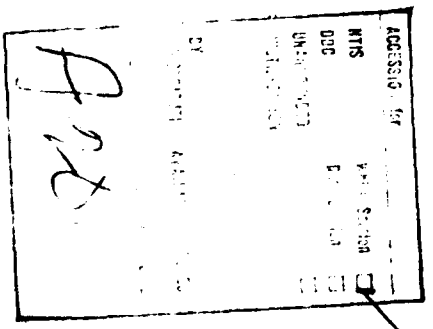
2. Endotoxin						
3. Portal Circulation						
4. Isochemic Bowel						
5. Portal Vein						
6. Limulus lysate method						
7. Endotoxemiz						
8. Ischemic Bowel Damage						
9. Splanchnic Viscera						



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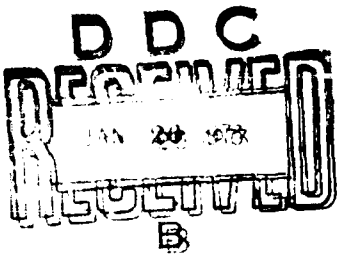

ENDOGENOUS ENDOTOXEMIA DURING HEMORRHAGIC SHOCK IN THE SUBHUMAN PRIMATE

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EXPERIMENTS were carried out to test the hypothesis that, during hemorrhagic shock, endotoxin enters the portal circulation from ischemic bowel and is then associated with irreversibility of the hemorrhagic shock state. Since this theory has evolved mainly from canine experimental data, we used baboons in order to determine the validity of the theory in a species phylogenetically closer to man.

METHODS

After placement of sampling catheters in the portal vein, right atrium, and aorta, 14 awake, restrained baboons were subjected to 1 hr of hemorrhagic shock at a mean arterial pressure (MAP) of 60 torr followed by a second hour at 40-torr MAP. Six animals were resuscitated with lactated Ringer's solution in a volume three times the total blood loss followed by reinfusion of their shed blood. Eight animals were maintained hypotensive until death. Serial blood samples from all three sites were analyzed for endotoxin by the Limulus lysate method (1). Comparisons of incidence of endotoxemia according to sampling site and period of shock were made by Chi-square analysis.

RESULTS

The assay was 100% effective in detecting endotoxin at a concentration of 10^{-2} mg/ml, 94% effective at 10^{-4} mg/ml, and 39% effective at 10^{-6} mg/ml. There were no false-positive results.

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Table 1

	BASELINE*	EARLY SHOCK* (MAP > 40 TORR)	LATE SHOCK* (MAP < 40 TORR)
Resuscitated group	5/36	6/36	6/36
Non-resuscitated group	5/48	3/48	6/108

* Values expressed as samples positive for endotoxin/total number of samples.

Endotoxemia was found infrequently (Table 1), with no greater incidence ($P > .6$) in portal venous samples than in systemic blood, so these data were pooled for further analysis. Furthermore, endotoxemia was no more frequent ($P > .6$) late in shock than it was in early shock or during the baseline period. Autopsy showed only mild submucosal edema of the bowel in the resuscitated animals, with no splanchnic or other organ abnormalities in the nonresuscitated group.

COMMENT

Several possible explanations can be considered for the inconsistent and infrequent finding of endotoxemia in this study. Endotoxin could have been present at concentrations below the limit of sensitivity of the detection method. However, while it is not known what level of endotoxin can be damaging during a low flow state, endotoxin concentrations which are lethal in themselves in a baboon would have been detected with 100% certainty by our assay method. With no signs of ischemic bowel damage, it is more likely that the mesenteric blood flow and the integrity of the splanchnic viscera are not selectively compromised by hemorrhagic shock in this subhuman primate species.

CONCLUSION

Ischemic bowel damage resulting in endogenous endotoxemia is not a common feature of hemorrhagic shock in baboons. The observed sporadic endotoxemia occurs no more frequently during shock than in the baseline period, and is not related to the duration or degree of severity of hemorrhagic shock in this subhuman primate species.

REFERENCE

1. Levin J, Tomasulo PA, Oser RS: Detection of endotoxin in human blood and demonstration of an inhibitor. J Lab Clin Med 75:903–908, 1970