Occlusion of Superior Mesenteric Artery and Effect of Corticosteroid in Dogs

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ABSTRACT: In twenty dogs the effects of superior mesenteric artery occlusion (SMAO) for two hours on several conditions likely to be responsible for the lethality of animals or men following release of SMAO were evaluated with and without corticosteroid administration. Six of ten dogs without steroid treatment died from irreversible shock within 24 hours. However, ten steroid treated dogs maintained an adequate blood pressure and survived until sacrifice 24 hours after SMAO. Release of lysosomal hydrolases, metabolic acidosis, hyperkalemia or plasma loss seemed not to have exerted important effects on the fate of animals, because their differences between the groups were not significant. Hence the mechanism underlying the beneficial effects of corticosteroid administered should be sought elsewhere.

KEY WORDS: superior mesenteric artery, SMAO shock, lysosomal enzymes, metabolic acidosis, hyperkalemia, plasma loss, endotoxin, corticosteroid.

INTRODUCTION

The role of intestine in the development of irreversible shock has been extensively discussed since Lillehei and Washington 21 described an intestinal factor in irreversible hemorrhagic shock in dogs. Several investigations were carried out to find toxic substances likely to be released from the ischemic bowels following occlusion of the superior mesenteric artery (SMAO). Vasoactive substances liberated were described by Selkurt 31, by Baez and his associates 4 and by Kobold and Thal 20. Recently an interest has been increasingly focused on the release of lysosomal hydrolases from the ischemic intestine 1, 7. These enzymes are known to have a potent proteolytic property at an acid pH and are considered to affect organisms in a noxious way finally inducing cellular autolysis 11. On the other hand, Fine and his associates 9, 26, 33 ascribed the most important factor to be endotoxin absorbed from the intestine with a reduced blood flow. However, Janoff and his associates 16, Altura and his associates 3 and Carter and Einheber 10 could not substantiate the claim. As another causes of lethality in SMAO shock a loss of plasma into the gut followed by hemoconcentration and hypovolemia was proposed by Marston 23, 24. However, this seems to be unlikely in the light of recent studies by Kangwalklai and his associates 17 and by Tjong and his associates 34. Others indicated systemic acidosis and hyperkalemia to be a main pathology in SMAO shock 5, 25, 29, 34. Thus, although numerous explanations have been offered for the etiology of SMAO shock, no consensus of opinions...
Fig. 1. Changes in mean arterial blood pressure (MABP), pulse rates and central venous pressure (CVP) after SMAO (Mean±SE).