EFFECT OF RAISED PORTAL VENOUS PRESSURE ON BLOOD PRESSURE, URINE OUTPUT AND BODY FLUIDS

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Summary : Acute rise of portal venous pressure resulted in a fall of blood pressure and subsequent anuria which could be either due to the state of shock or may be reflex in origin. There also occurred an increase in extra-cellular fluid volume with an initial increase in both plasma volume and interstitial fluid volume. Subsequently the extra-cellular fluid volume remained at a high steady value whereas the plasma volume increased at the expense of interstitial fluid volume which decreased. It is further postulated that the rise of extra-cellular fluid volume is due to the shift of fluid from intra-cellular compartment to the extra-cellular compartment, as a defence mechanism to combat the state of shock.

Key words : portal venous pressure, blood pressure, plasma volume, interstitial fluid volume, urine output, extra-cellular fluid volume

INTRODUCTION

The effect of acute changes in portal pressure on urine output has been reported by Liang (4). He observed that a mild increase in portal pressure produces a reflex diuretic response which changes to an anti-diuretic response at higher portal pressures (15 cm H_2O). He explained these effects in terms of changes in renal haemodynamics.

The above response of the kidney is likely to be associated with a shift between the various fluid compartments of the body. In view of the paucity of information on this hypothesis, the present study was undertaken in an attempt to investigate this problem.

MATERIAL AND METHODS

Nineteen healthy mongrel dogs of either sex were used. Chloralose was used as an anaesthetic agent (100 *mg/kg*, body wt.). Blood pressure was recorded by an arterial cannula inserted in the femoral artery. Portal pressure was recorded by the water manometer and was raised by pulling the portal vein by curved glass hook. Urine output from both kidneys was recorded. The extracellular fluid volume was estimated by the

method described by Bowler (1) and plasma volume by that described by Crooke and Morris (2).

The various parameters were estimated before manipulation as also 30, 60 and 90 min after raising portal venous pressure.

RESULTS AND DISCUSSION

The results demonstrate the effect of acute rise of portal venous pressure on blood pressure, and plasma volume, extra-cellular and interstitial fluid volumes and urine flow.

Increase in portal venous pressure resulted in fall of blood pressure which was statistically significant at all three intervals after manipulation (Table I). The observations are in accordance with those of Elman and Cole (3).

TABLE I : Pre-manipulation levels of blood pressure, plasma volume, extra-cellular fluid volume (ECFV), interstitial fluid volume and urine output, and those after raising portal venous pressure. The results are expressed as mean±S.E. The number of animals N=19 in each case.

	Portal pressure (CMH_2O)	*Blood pressure* mm Hg	*Plasma volume* ml	*ECFV* ml	*Interstitial fluid volume* ml	*Urine output* drops
Initial	2.84±0.21	115.79±2.01	457.24±21.92	1878.30±205.06	1421.06±183.14	2.40±0.19
30 min	13.26±0.26	98.21±2.81	467.95±19.53	1979.80±255.29	1511.85±235.76	0.4±0.004
After rise	P<0.01	P<0.01	P<0.05	P<0.05	P<0.05	P<0.01
60 min	13.26±0.26	88.95±2.96	503.11±16.90	1979.60±255.27	1476.49±238.37	0
After rise	P<0.01	P<0.01	P<0.05	P<0.05	P<0.05	
90 min	13.26±0.28	85.79±2.88	529.00±20.90	1979.70±255.26	1450.70±234.36	0
After rise	P<0.01	P<0.01	P<0.05	P<0.05	P<0.05	

The plasma volume increased steadily after manipulation. The extra-cellular fluid volume also showed an increase in the first 30 minutes after manipulation. Later, however, it remained constant. The difference between extra-cellular fluid volume and plasma volume was taken as a measure of interstitial fluid volume, which increased to a maximum 30 min after manipulation, gradually decreasing thereafter.

An increase in the extra-cellular fluid volume and plasma volume 30 min after raising portal venous pressure suggests a movement of fluid into both spaces. The increase

in interstitial fluid volume occurred only after 30 min and later decreased, whereas the plasma volume showed a steady increase. This suggests that the fluid came into the interstitial space soon after raising the portal venous pressure and then gradually shifted into the vascular system. The source of this fluid could be the intra-cellular compartment. Such a fluid movement from the intra-cellular to the extra-cellular space, most likely served an important physiological function of restoring the falling blood pressure and hence combating the state of shock resulting from acute portal hypertension.

The observations on urine output differed from those of Liang (4), who observed diuresis followed later by an antidiuretic response at a higher portal venous pressure (15 cm H_2O). No diuretic response was observed in the present study, probably as the effect of mild increase in portal venous pressure was not noted. The anuria which resulted may have been reflex in origin, as also postulated by Liang (4), or it may be subsequent to the state of shock and falling blood pressure. The additive effect of the two may explain the quick onset of anuria in the present study.

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