# HEMODYNAMIC ANALYSIS OF THE HYPOTENSIVE ACTION OF PORTOCAVAL TRANSPOSITION OF THE VESSELS IN VASORENAL HYPERTENSION

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The effect of portocaval transposition (PCT) on the general hemodynamics was studied in intact dogs and in dogs with experimental vasorenal hypertension. In the experimental dogs the arterial pressure fell after PCT chiefly on account of a decrease in the peripheral vascular resistance to the blood flow. In healthy dogs PCT caused no substantial changes in the hemodynamics.

KEY WORDS: vasorenal hypertension; portocaval transposition; hemodynamics.

Changes in the portal and caval blood flow, so that blood from the inferior vena cava together with blood from the kidneys and adrenals is directed into the liver, whereas blood from the portal vein is directed into the inferior vena cava to bypass the liver (portocaval transposition – PCT), had a hypotensive action in dogs with experimental vasorenal hypertension [5, 7].

The object of this investigation was to study the effect of PCT on the hemodynamics in intact dogs and in dogs with experimental vasorenal hypertension.

## EXPERIMENTAL METHOD

Experiments were carried out on 11 anesthetized mongrel dogs preliminarily accustomed to the experimental situation. In the experimental group of animals (five dogs) vasorenal hypertension was first produced by constriction of both renal arteries, and on the ninth to 12th day of arterial hypertension, the PCT operation was performed (Fig. 1). The animals of the control group (six dogs) underwent the PCT operation only. This operation was performed under endotracheal ether–oxygen anesthesia combined with muscle relaxants [1]. The arterial pressure was measured by a tacho-oscillographic method [2]. The stroke volume of the heart was calculated by a physical method [3] based on the ECG, kinetocardiogram, phonocardiogram, and sphygmogram, recorded on a Mingograph-34 apparatus. The circulating blood volume was determined by the dye-dilution method using T-1824 as the indicator.

## EXPERIMENTAL RESULTS AND DISCUSSION

Analysis of the hemodynamic data in Table 1 shows that constriction of the renal arteries caused a significant increase in the blood pressure. The peripheral vascular resistance was substantially increased. The minute volume of the circulation was unchanged and the circulating blood volume was very slightly reduced.

After the PCT operation on the dogs with vasorenal hypertension the arterial pressure fell significantly and this was accompanied by a decrease in the peripheral vascular resistance. On the seventh day

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TABLE 1. Some Hemodynamic Indices after Portocaval Transposition in Healthy Dogs and in Dogs with Vasorenal Hypertension (M ± m)

| Index studied | Control | | | Experiment | | | |
|---|---|---|---|---|---|---|---|
| | initial data | 7th day | 30th day | initial data | hypertension | 7th day | 30th day |
| Mean arterial pressure (in mm Hg) | 102±3,1 | 99±3,0 | 101±2,5 | 107±1,7 | 132±2,0 | 114±3,4 | 109±4,1 |
| Cardiac index (in liters/min/$m^2$) | 3,98±0,15 | 4,0±0,05 | 3,84±0,08 | 3,76±0,13 | 3,71±0,29 | 4,54±0,09 | 3,98±0,12 |
| Heart rate (in beats/min) | 111±3,9 | 116±1,8 | 107±2,1 | 105±3,7 | 104±6,9 | 139±4,0 | 113±3,7 |
| Stroke index (in ml/$m^2$) | 35,9±0,17 | 35,2±0,19 | 35,8±0,14 | 35,7±0,2 | 35,0±0,25 | 32,6±0,28 | 35,2±0,10 |
| Specific peripheral resistance of vessels (in dynes · ($sec^{-1}$) $cm^{-5}/m^2$) | 2080±96 | 1934±57 | 2121±70 | 2320±61 | 2986±205 | 2020±107 | 2219±128 |
| Circulating blood volume (in ml/kg) | | | | 109±4,5 | 100±3,7 | 117±3,25 | 119±4,1 |

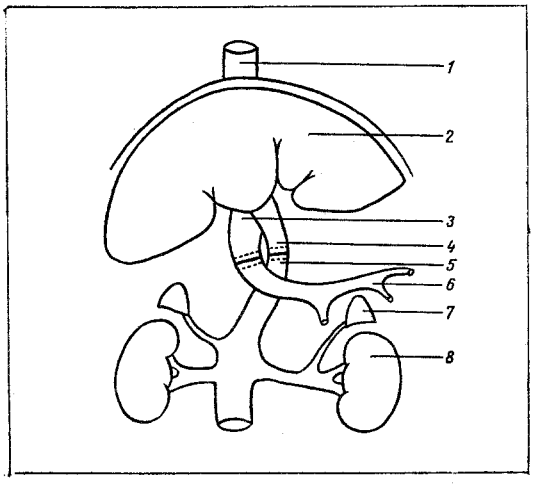


Fig. 1. Scheme of operation for portocaval transposition of the vessels: 1) supradiaphragmatic portion of inferior vena cava; 2) liver; 3) subhepatic portion of inferior vena cava; 4) proximal portion of portal vein; 5) inferior vena cava; 6) portal vein; 7) adrenal; 8) kidney.

after PCT the minute volume of the circulation of these dogs was significantly increased because of a marked increase in their heart rate and a small increase in the circulating blood volume.

In the healthy dogs PCT caused no significant changes in the hemodynamics (Table 1).

The hypotensive action of PCT in vasorenal hypertension can thus be explained chiefly by a lowering of the peripheral vascular resistance to the blood flow. According to some workers [4, 6], portalization of the blood flowing from the kidneys and adrenals leads to metabolic conversion of renin, angiotensin, and aldosterone in the liver, to the abolition of secondary hyperaldosteronism, and to a decrease in sodium retention. Consequently the Na/K ratio falls in the vessel wall and the sensitivity of the latter to vasoconstrictor influences is reduced.

The causes of the absence of any corresponding hemodynamic responses to PCT in healthy dogs are not sufficiently clear. It can be suggested that under normal conditions the metabolic activity of the enzyme systems of the liver is not as high as under pathological conditions, when the increased concentration of hormones in the blood stream facilitates induction of the metabolic systems of the liver.

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