# Changes in Caval Blood Flow and Right Atrial Pressure in Response to Catecholamines

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The type and magnitude of changes in blood flow in the anterior (cranial) and posterior (caudal) caval veins and shifts in the mean right atrial pressure induced by catecholamines (epinephrine and norepinephrine) were studied in acute experiments on cats. It was found that irrespective of right atrial pressure shifts, the increase in the blood flow in the anterior vena cava was more pronounced than in the posterior vena cava and was determined by blood redistribution due to more pronounced increase in vascular resistance in the abdominal aorta basin.

**Key Words:** *blood flow; anterior vena cava; posterior vena cava; right atrial pressure; catecholamines*

Instant blood flow values in caval veins depend on suction power of the right atrium during diastole [3,4]. It is believed that during this period (which coincides with the period of blood ejection from ventricles) the atrioventricular septum is shifted towards the heart apex, which leads to a decrease in the right atrial pressure (RAP) and increase in venous blood flow [6]. During atrial systole, the atrial pressure sharply increases and venous flow to the heart is discontinued [5,6]. We previously showed that after treatment with pressor agents RAP rapidly increases to the initial level, while venous return normalizes slower [2]. The increase in venous return was not paralleled by RAP changes (RAP either increases or decreases). These data attested to the absence of direct relationships between the mean RAP and venous return to the heart [2]. Here we evaluated the type and magnitude of changes in RAP and blood flow in the anterior and posterior caval veins (BAVC and BPVC, respectively) after treatment with catecholamines in different doses and studied the mechanisms of these changes.

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## MATERIALS AND METHODS

The study was carried out on 14 open-chest artificially ventilated cats (3.5-5.0 kg) under Nembutal narcosis (35-40 mg/kg intramuscularly). Arterial pressure (AP) was recorded in the left femoral artery with a transducer made on the base of a 6MDKh1B superminiature manotrone [2]. RAP was measured with a low-pressure transducer basis on a 6MD11S monotrone [2] using a catheter inserted into the right atrial cavity through the auricle. The mean RAP was measured with an integrator for the maximum and minimum pressure. BAVC and BPVC were measured with cuff transducers connected to a T-230 two-channel ultrasonic flowmeter (Transonic), cardiac output (CO) was measured in the ascending aorta using cuff transducer of an MFV2100 electromagnetic flowmeter (Nihon Kohden). Heart rate (HR) was measured with a tachometer by *R-R* intervals recorded in standard lead II. Bolus intravenous injection (into the left femoral artery) of epinephrine or norepinephrine in doses of 2.5 and 5.0 μg/kg served as the pressor stimuli. Hemodynamic parameters were recorded with an H-338-8P ink-writing device.

The results were statistically processed using Student's *t* test, original and standard (Axum 5.0, Math Soft Inc.) software.

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## RESULTS

The initial AP was 94±8 mm Hg, RAP 4.3±0.4 mm Hg, BAVC and BPVC 51±5 and 124±14 ml/min, respectively, venous return 175±16 ml/min, CO 172±18 ml/min, and HR 192±7 bpm. Intravenous injection of epinephrine reduced RAP in some of animals (group 1) and increased it in others (group 2, Table 1). The type of RAP shifts did not depend on HR (this parameter increased in both groups, Table 1).

Similar changes in RAP were observed after injection of norepinephrine (Table 2), which, in contrast to epinephrine, stimulates primarily vascular α-adrenoreceptors [5,6]. The increase in HR in response to norepinephrine was less pronounced than after injection of epinephrine (Tables 1 and 2). In groups 1 and 2 the maximum shifts in RAP were observed 12-16 sec after intravenous injection of catecholamines. The maximum shifts in the venous blood flow were observed after 20-22 sec (vena cava anterior) and 38-40 sec (vena cava posterior), that is, did not coincide with changes in RAP (as in our previous study) [2]. Correlation analysis also revealed no direct relationship between RAP and BAVC and BPVC shifts induced by catecholamines (linear correlation coefficient ranged from 0.44 to 0.05). Since shifts in RAP appeared, peaked, and leveled before the changes in venous blood flow, we analyzed the BAVC/BPVC ratio during the maximum shift of RAP.

Epinephrine in a dose of 2.5 μg/kg increased BAVC in group 1 more markedly than in group 2 (Table 1), though the differences were insignificant. The shifts in BPVC were virtually the same in both groups (Table 1). After injection of 5.0 μg/kg epinephrine the BAVC increase was more pronounced in group 2, while the increase in BPVC was more pronounced in group 1 (Table 1). Epinephrine in both doses more markedly increased BAVC than BPVC and these shifts did not depend on shifts in RAP.

The more pronounced increase in BAVC after injection of epinephrine can be explained by peculiar changes in vascular resistance in the brachiocephalic artery and thoracic aorta basins and, hence, in blood supply to the anterior and posterior caval vein regions [1]. It should be noted that the lower capacity of the anterior vena cava region in comparison with the posterior vena cava region is associated with lower inertia of this basin, which also could manifest in more rapid shifts in volume blood flow velocity in these vessels [6].

**TABLE 1.** Changes (%) in Systemic Hemodynamics Parameters and Caval Blood Flow during Maximum RAP Shifts (*M*±*m*)

| Parameter | Epinephrine dose, μg/kg | | | |
|---|---|---|---|---|
| | 2.5 | | 5.0 | |
| | group 1 (*n*=8) | group 2 (*n*=5) | group 1 (*n*=4) | group 2 (*n*=7) |
| RAP | -19±5 | 15±4 | -25±6 | 26±7 |
| AP | 24±6 | 19±5 | 41±13 | 41±16 |
| HR | 17±3 | 14±2 | 12±4 | 18±3 |
| CO | 20±3 | 18±5 | 32±9 | 16±3 |
| BAVC | 48±8 | 34±8 | 43±14 | 58±7 |
| BPVC | 5.5±3.0 | 6.8±3.0 | 28±7 | 5±2 |

**TABLE 2.** Changes (%) in Systemic Hemodynamics Parameters and Caval Blood Flow during Maximum RAP Shifts in Response to Norepinephrine (*M*±*m*)

| Parameter | Norepinephrine dose, μg/kg | | | |
|---|---|---|---|---|
| | 2.5 | | 5.0 | |
| | group 1 (*n*=10) | group 2 (*n*=4) | group 1 (*n*=8) | group 2 (*n*=6) |
| RAP | -24±5 | 12±2 | -25±5 | 18±4 |
| AP | 50±13 | 42±8 | 89±18 | 51±12 |
| HR | 4±2 | 4±2 | 9±3 | 5±2 |
| CO | 17±4 | 15±6 | 20±5 | 18±4 |
| BAVC | 50±7 | 26±6 | 64±7 | 34±5 |
| BPVC | 15±4 | 12±4 | 25±7 | 20±6 |

For elucidation of the mechanisms of these changes, we studied BAVC and BPVC shifts during maximum RAP changes in response to norepinephrine. Similarly to experiments with epinephrine, the increase in BAVC was more pronounced than changes in BPVC in both groups (Table 2). In group 1 BPVC increased more markedly than in group 2, while BPVC increased to the same extent in both groups (Table 2).

Thus, norepinephrine induced more pronounced shifts in AP compared to epinephrine in both groups, while changes in CO were similar (Tables 1 and 2). We concluded that the greater increase in BAVC during RAP decrease is determined by increased vascular resistance in the abdominal aorta basin and redistribution of the blood flow to the anterior vena cava region, rather than by increased pressure gradient for venous blood flow [1]. This is confirmed by more pronounced increase in BAVC during RAP increase (Table 2).

Results of experiments with higher dose of norepinephrine also confirmed this assumption. The maximum shifts in RAP in groups 1 and 2 were virtually the same as in response to norepinephrine in a dose of 2.5 μg/kg, but the increase in BAVC in response to 5.0 μg/kg of norepinephrine was more pronounced than after a dose of 2.5 μg/kg (Table 2). The fact that norepinephrine in dose of 5.0 μg/kg more markedly increased BPVC than 2.5 μg/kg epinephrine confirms the assumption on blood redistribution (Table 2). Since CO shifts in response to 5.0 μg/kg norepinephrine and 2.5 μg/kg epinephrine were similar, the increase in BAVC is primarily determined by blood redistribution to the anterior vena cava basin due to greater increase in vascular resistance of the abdominal aorta basin. One more proof is more pronounced increase in AP after injection of epinephrine in a dose of 5.0 μg/kg compared to the effect of 2.5 μg/kg norepinephrine in both groups (Table 2).

Hence, the increase in BAVC during the maximum RAP shifts after injection of catecholamines is more pronounced than the increase in BPVC (irrespective of RAP shifts), and therefore venous return to the heart during this period is determined by the BAVC/BPVC ratio and does not depend on the mean RAP. Since changes in CO after catecholamine injection were similar in animals with decreased and increased RAP, the more pronounced increase in BAVC is determined by higher vascular resistance in the brachiocephalic artery basin compared to abdominal aorta basin. This is confirmed by the direct relationship between BAVC and systemic AP. The shifts in the mean RAP after injection of catecholamines did not depend on changes in AP, CO, and HR, that is, opposite shifts of RAP were not determined by changes in systemic hemodynamics.

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