Relationships between Venous Return and Blood Pressure in Caval Veins and Right Atrium During Pressor Stimulation

B. I. Tkachenko, V. I. Evlakhov, and I. Z. Poyasov

Translated from *Byulleten' Eksperimental'noi Biologii I Meditsiny*, Vol. 132, No. 10, pp. 368-371, October, 2001
Original article submitted August 2, 2001

The dynamics of changes in blood flow and pressure in the superior and inferior vena cava, total venous return, and right atrial pressure in response to pressor stimuli were studied in acute experiments on cats. It was shown that blood flow and pressure in caval veins changed synchronously and unidirectionally, while shifts in the right atrial pressure did not depend on direction and magnitude of changes in caval flow and pressure and total venous return. Our results suggest that right atrial pressure does not play a role in the regulation of venous return.

Key Words: *venous return; right atrial pressure; blood flow in the superior vena cava; blood flow in the inferior vena cava*; *pressor stimuli*

There is evidence that venous return (VR) depends on central venous pressure (measured in the right atrium) [4]. At the same time, opposite shifts in right atrial pressure (RAP) in response to increased VR during physical exercise were observed [6]. It was recently shown that changes in VR in response to pressor stimuli were not accompanied by simultaneous and co-directed changes in RAP, and that RAP rapidly approached the initial level irrespective of VR [1]. Blood pressure in the caval veins and right atrium is often considered to be equal [4]. Our aim was to study the relationship between blood flow and pressure in the caval veins and between VR and RAP under the effect of various pressor stimuli.

MATERIALS AND METHODS

Experiments were performed on 14 artificially ventilated open-chest cats weighing 3.5-5.0 kg anesthetized with Nembutal (35-40 mg/kg, intramuscularly). Blood pressure (BP) in the left femoral artery was measured with a transducer made on the basis of an ultraminiature 6MDKh1B mechanotron [1]. RAP and pressure in the posterior vena cava near the right atrium were measured with low-pressure transducers based on 6MD11S mechanotrons [1] and connected to two catheters inserted into the right atrium through the auricle and into caval vein, respectively. The same type of transducers was used for pressure recording in the superior vena cava via a catheter introduced through the right external jugular vein. The mean caval and right atrial pressures were determined from their maximum and minimum values using an integrator. Blood flows in the superior (cranial) and inferior (caudal) caval veins were measured with a T-130 Transonic ultrasonic and an MVF-2100 Nihon Kohden electromagnetic cuff gage flowmeters, respectively, and VR was calculated as the sum of these flows. ECG was recorded in the second standard lead, and the heart rate was calculated from *R—R* intervals using a tachometer. A bolus injection epinephrine (2.5 and 5.0 μg/kg) or 20 ml physiological saline (for 5 sec) into left femoral vein were used as pressor stimuli. The test parameters (AP, RAP, pressure and flow in the su-

Department of Physiology of Visceral Systems, Institute of Experimental Medicine, Russian Academy of Medical Sciences, St. Petersburg. ***Address for correspondence:*** viespbru@mail.ru. Evlakhov V. I.

0007-4888/01/1324-0926$25.00

perior and inferior caval veins) were recorded with an N-338-8P ink-pen recorder. The data were statistically analyzed using Student's *t* test (MathSoft Inc. and Axum 5.0 and originally designed software).

RESULTS

Intravenous injection of 2.5 μg/kg epinephrine increased AP by 22±6%, HR by 16±3% ($p<0.05$), and blood flows in the superior and inferior caval veins by 68±12% and 18±7%, respectively ($p<0.05$) compared to the baseline. Blood pressure in the superior and inferior caval veins increased by 69±18% and 18±3%, respectively ($p<0.05$, Fig. 1). These changes in caval pressures and flows were synchronous and their values and direction were similar (Fig. 1).

Injection of 2.5 μg/kg epinephrine induced opposite shifts in RAP: in some animals (group I) RAP increased by 8±3% ($p<0.05$), while in others (group II) it decreased by 13±6%, ($p<0.05$) compared to the baseline. Initial VR in these groups were similar (242±28 and 210±25 ml/min). Epinephrine increased VR in these groups by 28±7% and 29±4%, respectively (Fig. 2). Hence, VR shifts did not determine the direction and magnitude of RAP response to pressor stimuli.

Thus, changes in the blood flow and pressure in the caval veins in response to 2.5 μg/kg epinephrine are synchronous and co-directed, while the RAP responses did not correlate with changes in caval flow and VR. Moreover, no correlation between RAP and caval pressure was revealed.

Intravenous injection of epinephrine in a dose of 5 μg/kg induced similar responses: similar changes in VR in both groups and opposite shifts in RAP at the peak of response (Table 1).

Opposite changes in RAP in response to epinephrine could be due to different initial level of RAP in these two groups, which can result from different afterload conditions in the right heart, in particular, blood pressure in the pulmonary artery [2]. Atrial pressure depends not only on diastolic filling rate and duration of the diastole, but also on residual ventricular volumes, end-diastolic pressure, phasic changes in ventricular pressure, elasticity and contractility of the myocardium [2,3,5]. These factors are probably responsible for independence of RAP dynamics on changes in VR and caval pressure observed in our experiments.

TABLE 1. Changes in Venous Return and Right atrial Pressure Induced by Epinephrine (5 μg/kg, *M*±*m*)

Parameters		Group 1	Group 2
RAP	baseline, mm Hg	5.9±1.1	6.9±0.9
	Δ, %	23.9±3.8*	-22.5±6.8*
VR	baseline, ml/min	195±17	188±25
	Δ, %	34±7*	44±12*

Note. Here and in Table 2: *$p<0.05$ compared to the baseline.

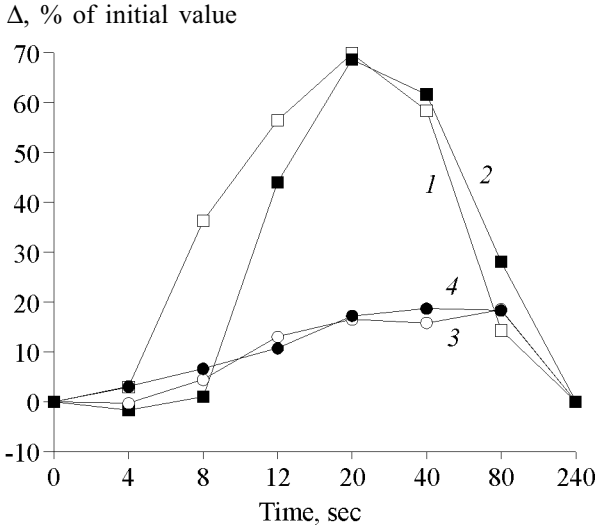

Fig. 1. Changes in blood flow and pressure in the superior and inferior caval veins induced by epinephrine (2.5 μg/kg). *1*) pressure in superior vena cava, *2*) blood flow in superior vena cava, *3*) pressure in inferior vena cava, *4*) blood flow in inferior vena cava.

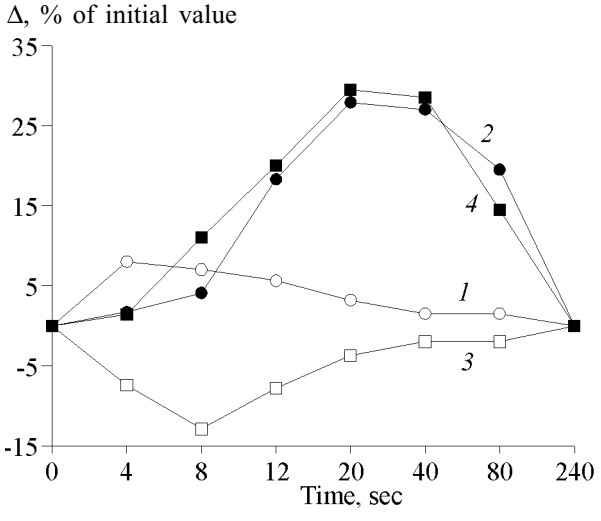

Fig. 2. Dynamics of right atrial pressure (*1*, *3*) and venous return (*2*, *4*) in cats with positive (*1*, *2*) and negative (*3*, *4*) changes in right atrial pressure in response to 2.5 μg/kg epinephrine.

Intravenous bolus injection of 20 ml physiological saline increased RAP, VR, and blood flow and pressure in both caval veins (Table 2). Blood flow in the superior and inferior caval veins considerably increased 20 sec postinjection and these changes were accompanied by co-directed shifts in venous pressures (Table 2). Forty seconds after injection of physiological saline, RAP returned to the baseline, while blood flow and pressure in the caval veins and VR remained significantly increased ($p<0.05$, Table 2). Thus, injection of physiological saline induced co-

TABLE 2. Changes in Blood Flow (ml/min) and Pressure (mm Hg) in Caval Veins, Venous Return (ml/min), and Right Atrial Pressure (mm Hg) Induced by Infusion of 20 ml Physiological Saline (*M±m*)

Parameters	Baseline, abs.	Δ, %	
		20 sec	40 sec
Blood flow in the superior vena cava	44±5	152±31*	118±26*
Pressure in the superior vena cava	6.4±0.6	48±8*	34±6*
Blood flow in the inferior vena cava	155±22	52±14*	52±14*
Pressure in the inferior vena cava	9.9±0.5	23±4*	17±5*
Total VR	199±24	55±19*	49±16*
RAP	6.2±0.7	19±3*	4±5*

directed and synchronous shifts in VR and blood flow and pressure in the caval veins. As in previous experiments, RAP returned to the baseline 40 sec after injection of physiological saline [1], and its dynamics did not correlate with shifts in VR and blood flow and pressure in the caval veins.

Thus, blood flow and pressure in both caval veins and total VR underwent synchronous and co-directed shifts in response to pressor stimuli applied to the cardiovascular system, while the dynamics of RAP differed from VR changes in time and direction. These findings point to different dynamics of pressure changes in caval veins and right atrium, suggesting that RAP is not a leading factor, which determines the amplitude and direction of the shifts in VR in response to pressor stimulation.

The work was supported by Russian Foundation of Fundamental Researches (grant No. 00-04-49342).

REFERENCES

1. B. I. Tkachenko, V. I. Evlakhov, and I. Z. Poyasov, *Byull. Eksp. Biol. Med.*, **131,** No. 5, 501-503 (2001).
2. A. Boussuges, Ch. Pinet, P. Ambrosi, *Am. J. Crit. Care*, **162**, No. 2, 670-675 (2000).
3. E. Braunwald and Ch. J. Frahm, *Circulation*, **24**, No. 3, 633-642 (1961).
4. A. C. Guyton, *Am. J. Physiol.*, **259**, No. 3, Pt. 2, R865-R877 (1990).
5. B. Pump, N. Christensen, and R. Videbak, *Ibid.*, **273**, No. 6, Pt. 2, H2632-H2638 (1997).
6. D. D. Sheriff, R. A. Augustyniak, and D. S. O'Leary, *Ibid.*, **275**, No. 3, Pt. 2, H767-H775 (1998).