Relationship between Venous Return and Right-Atrial Pressure

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The dynamics and amplitude of changes in venous return and right atrial pressure (central venous pressure) in response to pressor stimuli were studied in acute experiments on cats. The increase in venous return was accompanied by either increase, or decrease in the central venous pressure. Thus, shifts in systemic venous return were not accompanied by simultaneous and co-directed changes in the central venous pressure. These findings suggest the absence of a direct relationship between these parameters.

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

Blood flow via caval veins and right atrial pressure (or central venous pressure, CVP) determine total venous return (VR) to the heart [2,5,6]. It was demonstrated that increasing CVP to 7 mm Hg reduces VR to zero [2,5]. A conclusion was made that VR is directly proportional to the difference between the mean filling pressure in vessels and blood pressure in the right atrium [2]. However, these experiments were carried out on animals with blocked nervous regulation of the cardiovascular system and could not explain the causal relationships between VR and CVP. Moreover, the cardiovascular system belongs to non-linear non-equilibrium open systems [4], and linear approximation is applicable for the analysis of its behavior only within a narrow range of variations of hemodynamic parameters [3].

Our aim was to study the relationship between CVP changes and VR under the effect of pressor stimuli in intact cardiovascular system.

MATERIALS AND METHODS

The study was performed on 13 open-chest artificially ventilated cats weighing 3.5-5.0 kg anesthetized with nembutal (35-40 mg/kg, intramuscularly). Blood pressure (BP) was measured with a transducer constructed on the basis of an ultraminiature 6MDKh1B mechanotron [1]. Right atrial pressure was measured with a low-pressure transducer via a catheter introduced into the right atrium through the auricle and connected to a low-pressure manometer (6MD11S mechanotron) [1]. The mean CVP was calculated by systolic and diastolic atrial pressures using an integrator. Blood flows in the superior (cranial) vena cava was measured with a T-130 Transonic ultrasonic and in the inferior (caudal) vena cava with an MVF-2100 Nihon Kohden electromagnetic cuff flowmeters. VR was calculated as the sum of these flows calculated on-line with a computer. Heart rate was determined from *R—R* intervals on standard lead II ECG with a tachometer. A bolus injection of physiological saline into the left femoral vein (20 ml/5 sec) and intravenous injection of epinephrine in doses of 2.5 and 5.0 μg/kg were used as pressor stimuli.

Test parameters (BP, CVP, and blood flows in the superior and inferior caval veins) were recorded with a N-338-8P recorder. The data were statistically analyzed by Student's *t* test using originally designed and standard Axum 5.0 and MathSoft Inc. software.

RESULTS

Baseline mean hemodynamic parameters in cats were: BP=105±7 mm Hg, CVP=7.1±0.9 mm Hg, VR=

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229.4±12.7 ml/min, blood flow=57.5±4.2 ml/min in the superior vena cava and 171.9±13.3 ml/min in the inferior vena cava, and HR=152±6 beats/min.

During the first 5 sec of intravenous bolus injection of physiological saline VR sharply increased ($p<0.05$), which led to an increase in CVP ($p<0.05$, Fig. 1, *a*). The increase in VR in response to injection of physiological saline was due to increased blood flow in the inferior vena cava (by 106±22%, $p<0.05$) against the background of decreased superior vena cava flow (by 14.2%, $p<0.05$). Simultaneous decrease in HR probably resulted from enhanced vagal inhibitory influences (by 18±4%, $p<0.05$), which in turn were a response to extension of the right atrium [6].

Normalization of VR took about 4 min, while CVP returned to baseline 40 sec after bolus injection of physiological saline (Fig. 1, *a*). At this moment, superior and inferior vena cava flows remained above the baseline by 77±25% and 40±12%, respectively ($p<0.05$), *i.e.*, the increase in blood flow was more pronounced in vena cava superior. Thus, changes in CVP and VR were co-directed only during the first 5 sec after bolus injection. Then, the right atrial pressure returned to normal, while VR remained above the baseline during the subsequent 4 minutes.

Intravenous injection of 2.5 μg/kg epinephrine increased BP by 24±7%, HR by 20±3%, and VR by 29±4% ($p<0.05$). At the same time, CVP decreased ($p<0.05$, Fig. 1, *b*). The increase in VR resulted from increased flows in the superior and inferior caval veins (by 48±13 and 24±8%, respectively, $p<0.05$). In other words, similar to bolus injection of physiological saline, the relative flow shifts in the superior vena cava induced by epinephrine were more pronounced.

Thus, intravenous injection of epinephrine increased VR, but decreased CVP, *i.e.* the relationship between VR and CVP differed dramatically from that observed during bolus injection of physiological saline. It is likely that the decrease in CVP in response to epinephrine injection was a result of a decreased end-systolic ventricular volume due to enhanced myocardial contractility. However, special experiments are

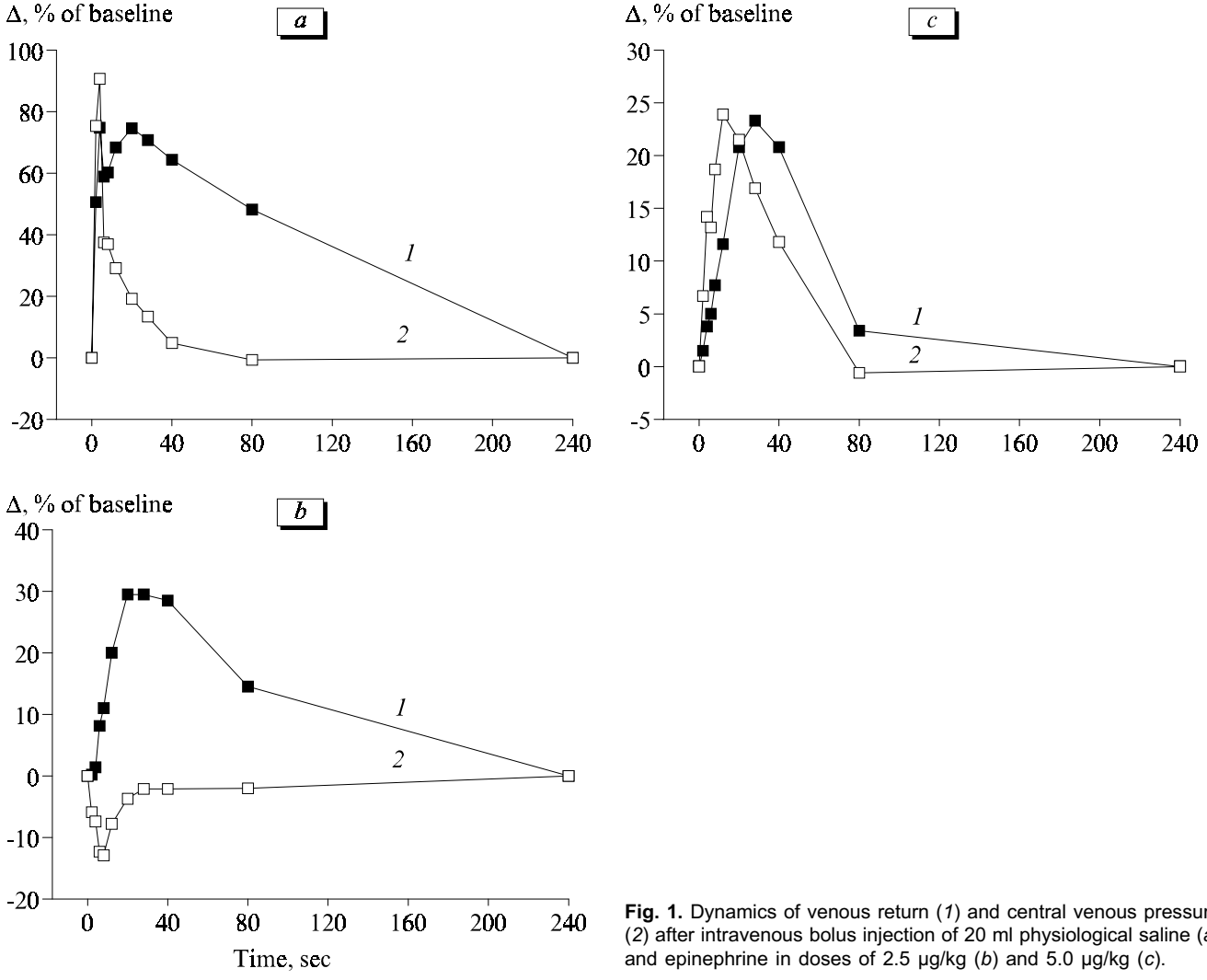

Fig. 1. Dynamics of venous return (*1*) and central venous pressure (*2*) after intravenous bolus injection of 20 ml physiological saline (*a*) and epinephrine in doses of 2.5 μg/kg (*b*) and 5.0 μg/kg (*c*).

necessary to verify this assumption. It should be also noted, that CVP returned to the baseline 20 sec after epinephrine injection (2.5 μg/kg), while VR remained increased during 4 min (similar dynamics of these parameters was observed after bolus injection of physiological saline). Thus, the dynamics of VR and CVP was different in both cases.

Taking into account published data on a linear correlation between CVP and VR [2,5], one can assume that increasing the dose of norepinephrine will produce a more pronounced increase in VR and decrease in CVP. Injection of 5.0 μg/kg epinephrine increased BP by 40±8% and HR by 17±2% ($p<0.05$), but VR increased only by 23±4% ($p<0.05$), similar to the effect of 2.5 μg/kg epinephrine. This increase VR was determined by increased superior and inferior vena cava flows (by 65±12 and 20±3%, respectively, $p<0.05$). In contrast to a lower dose of epinephrine, injection of 5.0 μg/kg epinephrine did not decrease, but even increased CVP by 24±7% ($p<0.05$, Fig. 1, *c*). This increase in CVP in response to administration of 5.0 μg/kg epinephrine can result from the increase in the end-systolic ventricular volume due to increased afterload associated with sharp BP rise. CVP changed more rapidly and returned to baseline earlier than VR (Fig. 1, *c*). In other words, they had different dynamics.

Hence, changes in CVP and VR caused by pressor stimuli were oppositely directed and were characterized by different dynamics. These findings suggest that the increase in VR in intact organism is not necessarily accompanied by simultaneous and co-directed changes in CVP, which suggests the absence of a direct relationships between these parameters. The right atrial pressure is finally determined by the proportion between caval flows, changes in HR, and contractility of the myocardium.

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