PHYSIOLOGY

Independence of Changes in Right Atrial Pressure and Central Venous Pressure

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

Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 134, No. 10, pp. 371-373, October, 2002
Original article submitted May 30, 2002

Dynamics of changes in right-atrial pressure and venous pressure measured in the inferior and superior vena cava at their orifices (central venous pressure) after bolus injection of 20 ml physiological saline or epinephrine (5.0 μg/kg) was studied in acute experiments on cats. The initial pressure in the right atrium was equal to that in caval veins. Pressor stimuli either increased or decreased the right atrial pressure, but always increased blood pressure in the caval veins. Moreover, right atrial pressure returned to the initial level more rapidly compared to that in caval veins. Our results suggest that the dynamics of the right-atrial pressure does not reflect the shifts in the central venous pressure.

Key Words: *right-atrial pressure*; *central venous pressure*; *pressor stimuli*

It is commonly accepted that the right-atrial pressure (RAP) adequately reflects the central venous pressure, *i.e.* venous pressure at the orifices of the caval vein. In light of this changes in the central venous pressure are routinely estimated by RAP [4]. However, when intrathoracic or intraabdominal pressures increase or decrease, the blood pressure measured in the inferior and superior vena cava differs from RAP despite a strong correlation between RAP and inferior vena cava pressure under conditions of quiet breathing [2,3,5]. For methodical restrictions, clinical studies also can not elucidate the actual relationship between shifts in RAP and central venous pressure during circulatory responses to various stimuli under natural conditions.

We recently reported that after pressor stimulation RAP recovers more rapidly than changes in venous return [1]. Our aim was to study the dynamics of changes in RAP and venous pressures at the vena cava orifice during pressor stimulation.

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

MATERIALS AND METHODS

The study was performed on 14 open-chest cats weighing 3.5-5.0 kg. The animals were anesthetized with Nembutal (35-40 mg/kg, intramuscularly) and artificially ventilated. Pressure in the superior vena cava (PSVC) near the right atrium was measured with a low-pressure 6MD11S mechanotron transducer [1] via a catheter inserted through the right external jugular vein. Pressure in the inferior vena cava (PIVC) and RAP were recorded with similar transducers connected to catheters introduced into the inferior vena cava and into the right atrium throught the auricle. The mean PSVC, PIVC, and RAP were determined from the peak-and-valley magnitudes using an integrator. Arterial blood pressure was recorded in the left femoral artery with a 6MDKh1B ultraminiature mechanotron transducer [1].

Bolus injection of 20 ml physiological saline into the left femoral vein over 5 sec and injection of 5.0 μg/kg epinephrine were used as pressor stimuli. The right-atrial, venous, and arterial pressures were recorded on an N-338-8P ink-pen recorder.

0007-4888/02/1344-0318$27.00

The data were statistically analyzed by Student's *t* test using standard and original software (Axum 5.0 and MathSoft Inc.).

RESULTS

The baseline pressures were: arterial blood pressure 95±9 mm Hg, RAP 5.9±1.1 mm Hg, PSVC 6.4±0.6 mm Hg, and PIVC 6.9±0.8 mm Hg. Thus, the initial RAP and central venous pressure did not differ significantly.

Intravenous bolus injection of 20 ml physiological saline increased RAP and pressures in both caval veins (Fig. 1). On the 4th sec postinjection RAP, PSVC, and PIVC significantly increased compared to baseline values (Fig. 1). At 40 sec postinjection RAP returned to the initial level, while PSVC and PIVC surpassed the baseline even 80 sec postinjection (Fig. 1) and returned to baseline values only after 4-5 min. Hence, RAP changes more rapidly than PSVC and PIVC and does not adequately reflect the central venous pressure. The dynamics of PSVC and PIVC after injection of physiological saline was similar.

Intravenous injection of 5.0 μg/kg epinephrine increased arterial blood pressure by 51±10% ($p<0.05$). RAP decreased in some animals ($n=8$, group 1) and increased in others ($n=6$, group 2). The maximum decrease in group 1 animals was 23±6% ($p<0.05$), while the maximum increase was 24±7% ($p<0.05$, Fig. 2, *a*). It should be noted that vena cava pressures increased in all animals irrespective of changes in RAP (Fig. 2, *b*). The dynamics of pressure shifts in both caval veins was similar, but differed from RAP changes. It was previously demonstrated that different RAP responses to epinephrine [1] did not depend on its initial level, blood flow in caval veins, and HR changes, but can result from different right-heart afterload, in particular blood pressure in the pulmonary artery [6].

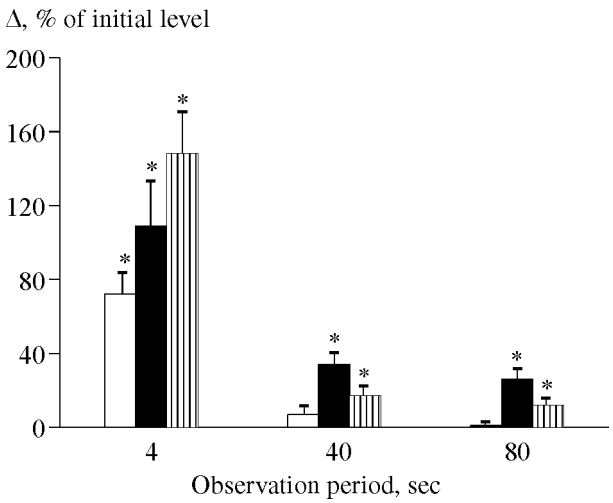

Fig. 1. Changes in right-atrial pressure (light columns) and pressures in the superior (dark columns) and inferior vena cava (dashed columns) after bolus intravenous injection of 20 ml physiological saline. *$p<0.05$, compared to the initial level.

Thus, our findings indicate that pressure changes in the inferior and superior vena cava in response to intravenous injection of epinephrine (5.0 μg/kg) were synchronous and codirected, while changes in RAP were not always parallel to the shifts in the central venous pressure.

The amplitudes of these responses were also different. Published data suggest that atrial pressure depends not only on diastolic filling rate and duration of diastole [3,6], but also on residual volume, end-dia-

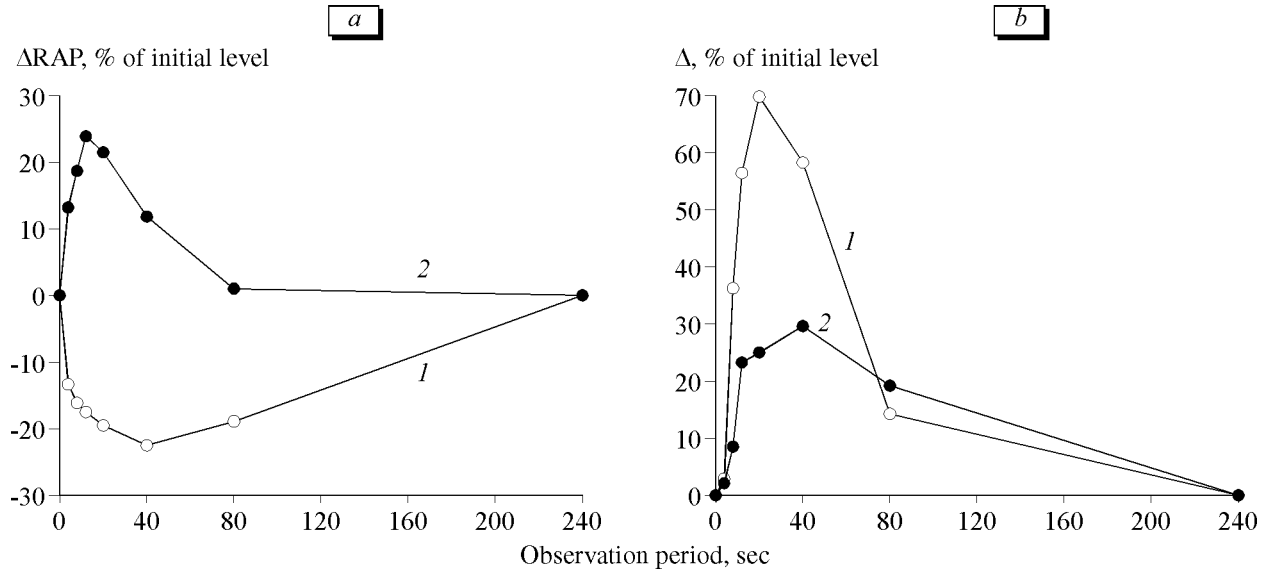

Fig. 2. Changes in right-atrial pressure (, *a*) and vena caval pressures (*b*) after bolus intravenous injection of 5.0 μg/kg epinephrine. *a*: groups with decrease (*1*) and increase (*2*) in RAP; *b*: venous pressure in the superior (*1*) and inferior (*2*) vena cava.

stolic pressure, phasic changes in ventricular pressure, and elasticity and contractility of the myocardium [2]. At the same time, blood pressure in the caval veins depends not only on the cardiac function, but also on circulating volume, venous tone and compliance, and venous blood flow rate. This probably explains independence of changes in RAP and central venous pressure observed after pressor stimulation.

In summary, our experiments demonstrated that RAP is equal to PSVC and PIVC under normal conditions, but after pressor stimulation shifts in RAP can differ in amplitude and direction from changes in the central venous pressure. These findings contradict the current views that the central venous pressure can be evaluated by RAP.

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*., **132**, No. 10, 368-371 (2001).
2. A. Alzeer, S. Arora, and Z. Ansari, *Can. J. Anaest.*, **45**, No. 8, 798-801 (1998).
3. H. I. Chait, M. A. Kuhn, and V. C. Baum, *Crit. Care Med*., **22**, No. 2, 219-224 (1994).
4. A. C. Guyton, Am. J. Physiol., **259**, No. 3, Pt. 2, R865-R877 (1990).
5. E. Nahum, O. Dagan, J. Sulkes, and T. Schoenfeld, *Intensive Care Med.*, **22**, No. 6, 571-574 (1996).
6. D. D. Sheriff, R. A. Augustyniak, and D. S. O'Leary, *Am. J. Physiol.*, **275**, No. 3, Pt. 2, H767-H775 (1998).