PHYSIOLOGY

Dynamic Relations between Right and Left Atrial Pressure Shifts Induced by Catecholamines

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

Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 139, No. 1, pp. 4-7, January, 2005
Original article submitted January 25, 2004

Acute experiments on cats showed that injection of catecholamines induced unidirectional shifts in right and left atrial pressure in 70% cases (these shifts were positive in one half of cats and negative in the other half). In 30% cases, the left and right atrial pressures changed in opposite direction: right atrial pressure decreased, while left atrial pressure increased (19%), or vice versa (11%). The pressure changes in the left atrium had greater amplitude and longer duration compared to those in the right atrium.

Key Words: *right atrial pressure*; *left atrial pressure*; *venous return*; *catecholamines*

Measurements of left and right atrial pressures are frequently used in physiological and clinical studies to assess systemic hemodynamics and cardiac function [1,4,6]. It is commonly accepted that shifts in the right and left atrial pressures induced by catecholamines are normally unidirectional [1,5,7]. However, asymmetry of left and right atrial pressure shifts was previously documented in humans during Valsalva's maneuver, antiorthostatic maneuver, and other functional tests [6]. In rats, the pattern of atrial pressure changes induced by catecholamines was not always definite [3]. In fact, scanty published data do not provide clear picture of the kinetics of atrial pressure shifts induced by catecholamines.

Our aim was to compare the direction, amplitude, and time course of changes in the left and right atrial pressures induced by intravenous injection of catecholamines in cats.

MATERIALS AND METHODS

The study was performed on 13 artificially ventilated open-chest cats weighing 3.5-5.0 kg anesthetized with nembutal (35-40 mg/kg, intramuscularly). Blood pressure (BP) was measured in the left femoral artery using an 6MDKh1B ultraminiature mechanotron transducer [2]. The right and left atrial pressures (RAP and LAP, respectively) were measured with low-pressure transducers based on 6MD11S mechanotrons [2] using catheters introduced into the atrial chambers via the corresponding auricles. The mean values of RAP and LAP were calculated from their systolic and diastolic levels using an integrator. Blood flows in the superior (cranial) and inferior (caudal) caval veins were measured with a T-230 Transonic dual-channel ultrasonic cuff gage flowmeter. Venous return was calculated as the sum of flows in both caval veins. Cardiac output was measured in the ascending aorta with a MFV-2100 Nikhon Kohden electromagnetic cuff gage flowmeter. ECG was recorded in the second standard lead, and heart rate was calculated from *RR* intervals by a tachometer. Catecholamines (adrenaline or norepinephrine) in doses of 2.5-5.0 μg/kg were administered into the left femoral vein (bolus infusion). These doses of catecholamines increased BP by 40-50% of the baseline level.

All test parameters were recorded with an N-338-8P ink-pen recorder and processed on an IBM Pentium computer. The data were statistically analyzed with

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

0007-4888/05/1391-0001

originally designed and standard Axum 5.0 and Math. Soft. Inc. software using Student's *t* test.

RESULTS

Baseline mean values of hemodynamic parameters in cats were: BP 83±8 mm Hg, RAP 5.4±0.4 mm Hg, LAP 8.0±0.5 mm Hg, venous return 234±22 ml/min, cardiac output 239±26 ml/min, and heart rate 173±4 bpm.

Intravenous injection of norepinephrine and adrenaline induced different shifts in the left and right atrial pressure: in 70% cats these changes were unidirectional. In one half of the cats (group 1), norepinephrine decreased LAP by 11±2% and RAP by 21±4% (peak values, $p>0.05$, Fig 1, *a*), while in the other half (group 2) these parameters increased by 17±4% and 37±9%, respectively ($p>0.05$, Fig 1, *b*). Adrenaline injection also induced co-directional pressure changes in both atria (negative in group 1 and positive in group 2). In group 1, the maximum shifts in LAP (-25±9%) and RAP (-23±6%) practically coincided (Fig 2, *a*). In group 2, peak increases in RAP and LAP were 17±3% and 23±2%, respectively ($p>0.05$, Fig 2, *b*).

Different shifts in RAP in response to injection of catecholamines were demonstrated previously [2]. Here we revealed a similar character of changes in LAP. Thus, catecholamines can induced either positive or negative shifts in the left and right atrial pressures. In 70% cases, pressure changes in the atria had the same sign.

When both atrial pressures shifted in a similar direction in response to catecholamines, the changes in LAP had greater amplitude than those in RAP. This can be due to higher baseline level of LAP in cats, compared to RAP. The latter probably reflects the differences in the end-diastolic pressures and afterload in the left and right ventricles [1,4,5]. This hypothesis, however, does not agree with the fact that, in group 1 cats, injection of adrenaline resulted in nearly equal pressure changes in both atria. Thus, intrinsic mechanisms responsible for predominance of LAP shifts in the control and similarity of changes in LAP and RAP when they decreased in response to adrenaline still remain unclear and require special studies.

In 30% cats, catecholamines induced asymmetric pressure shifts in the right and left atria. In 19% of experiments, RAP decreased (by 7-22%), while LAP increased (by 9-16%). By contrast, in the rest 11% cats, RAP increased (by 9-15%) and LAP decreased (by 9-28%). Thus, the direction and magnitude of atrial pressure shifts induced by catecholamines were different, which contradicts traditional point of view on their parallel character [1,4,5].

In 70% cats of both groups, catecholamine-induced changes in BP, venous return, and cardiac output differed insignificantly irrespective of atrial pressure shifts. BP increased by 40-50%, venous return and cardiac output increased by 30-40%, and heart rate increased by 10-15%. Opposite shifts in LAP and RAP were not related to changes in these parameters of systemic hemodynamic. It was previously established that the decrease in RAP after catecholamine injection was accompanied by a significant increase in venous return and right ventricular contractility [2]. If, under similar conditions less pronounced increases in venous return and right ventricular contractility were observed, RAP tended to grow [2]. By analogy, one can suppose that the character of left atrial pressures shifts depends on the relationship between the left heart inflow and the left ventricular contractility.

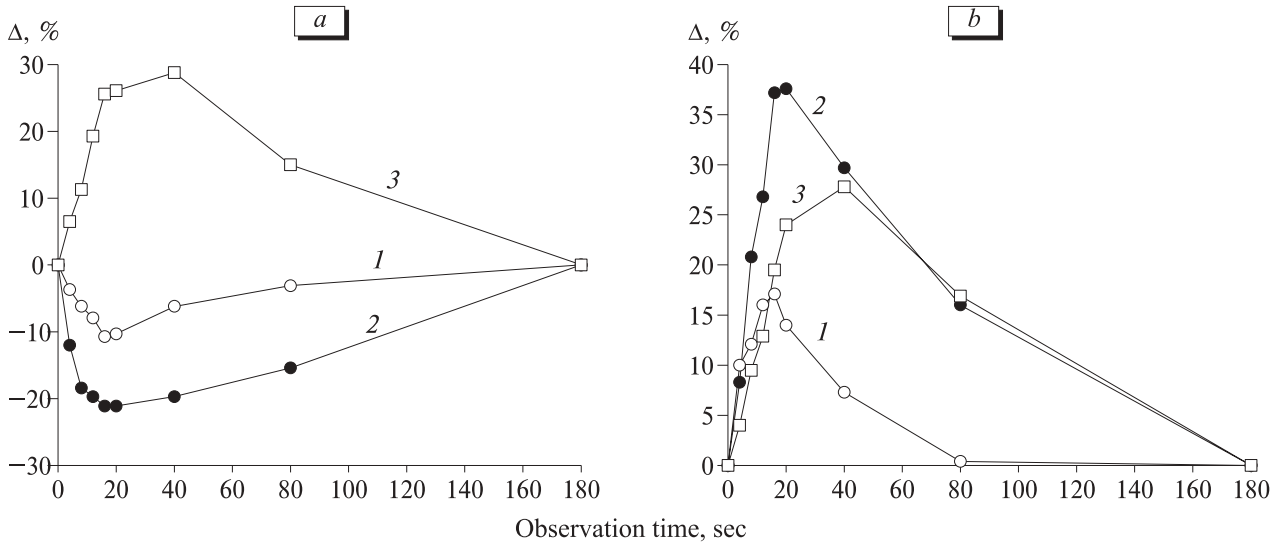

Fig. 1. Changes in in right (*1*) and left (*2*) atrial pressures and venous return (*3*) in response to bolus injection of norepinephrine (2.5 μg/kg, intravenously). Here and on Fig. 2: groups of cats with negative (*a*) and positive (*b*) atrial pressure shifts.

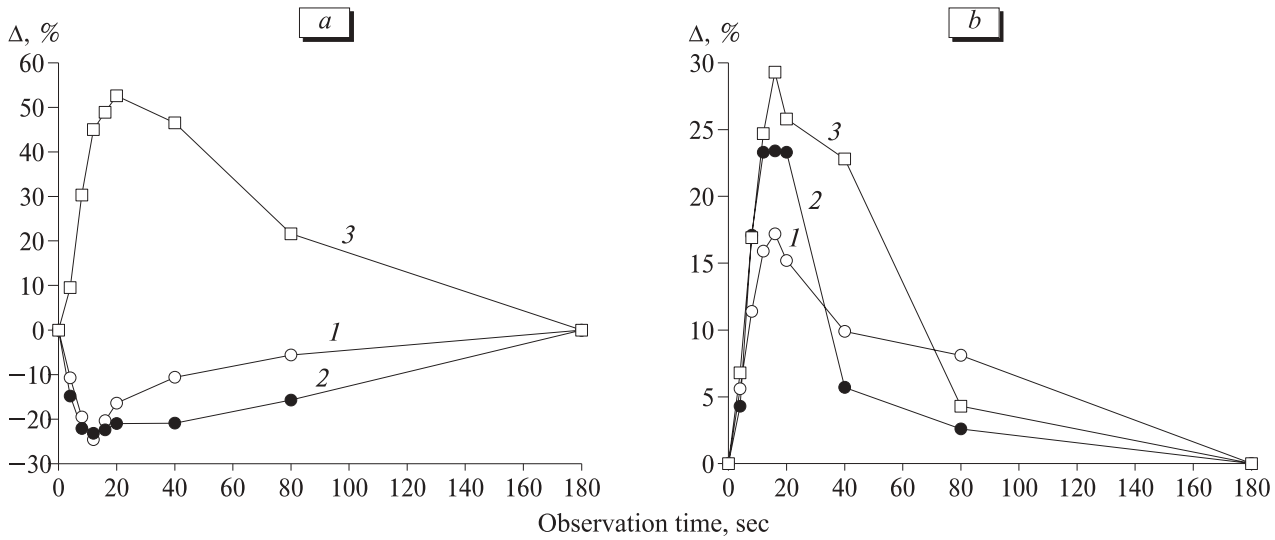

Fig. 2. Changes in right (*1*) and left (*2*) atrial pressures and venous return (*3*) in response to bolus injection of adrenaline (2.5 μg/kg, intravenously).

In case of opposite atrial pressure shifts (30% cats), catecholamines induced variable increases in BP (12-138%) and caused imbalance between shifts in venous return (39-70%) and cardiac output (51-140%). The factors responsible for this imbalance after catecholamine injection and the role of this imbalance in the realization of asymmetric changes in RAP and LAP is a matter of further investigations.

The dynamics of changes in RAP, LAP, and venous return was analyzed in 70% experiments (Fig 1, 2). The peak changes in both atrial pressures were observed at nearly similar time (16 sec), while the recovery of LAP lasted longer than that of RAP. Thus, RAP typically returned to its baseline level at 60 sec, while LAP recovered completely only 3 min after injection (Fig 1, 2). However, in group 2 animals LAP more rapidly returned to normal after adrenaline injection than RAP (Fig 2, *b*). The dynamics of LAP changes was similar to that of venous return, this fact requires special investigation. In 30% cats with opposite atrial pressure shifts the peak of RAP changes was observed much earlier than that of LAP changes (12 and 20-40 sec, respectively). In both 30% experiments and previous 70% experiments LAP shifts characterized by a slower kinetics compared to RAP shifts and were similar by their dynamics to the venous return changes. We can hypothesize that longer kinetics of LAP responses to catecholamines is defined by specific conditions of left ventricular afterload (total peripheral vascular resistance) [4]. The fact that the duration of LAP responses was similar to that of BP changes (about 3 min) confirms indirectly this assumption.

Thus, injection of catecholamines induced co-directed pressure changes in both atria in 70% cats. In one half of these cats LAP and RAP decreased, while in the other half they increased. In other 30% cats asymmetrical shifts in LAP and in RAP were observed. In most of them RAP decreased, while LAP increased, and in the rest vice versa. LAP shifts had greater amplitude and longer duration, compared to RAP shifts. The dynamics of LAP shifts was similar to that of changes in venous return.

The work was supported by Russian Foundation of Fundamental Researches (grant SS-2330.2003.4).

REFERENCES

1. R. Rashmer, *Cardiovascular Dynamics*, Moscow, 1981.
2. B. I. Tkachenko, V. I. Evlakhov, and I. Z. Poyasov, *Ros. Fiziol. Zhurn*., **89**, No. 5, 533-542 (2003).
3. W. Barth, A. Deten, M. Bauer, *J. Mol. Cell Cardiol.*, **32**, No. 2, 273-284 (2000).
4. M. H. Drazner, M. A. Hamilton, G. Fonarow, *et al.*, *J. Heart Lung Transplant*., **18**, No. 11, 1126-1132 (1999).
5. A. L. Golden, J. M. Bright, G. B. Daniel, *et al.*, *Am. J. Vet. Res.*, **59**, No. 4, 509-513 (1998).
6. S. Gorgulu, A. Eksik, M. Eren, *et al.*, *Int. J. Cardiol.*, **92**, No. 2, 241-245 (2003).
7. M. C. Mehta, A. C. Jain, M. D. Billie, *Clin. Cardiol.*, **24**, No. 9, 620-626 (2001).