PHYSIOLOGY

Changes in Inferior Vena Cava Blood Flow at Increased Negative Intrathoracic Pressure

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An increase in the negative intrathoracic pressure had practically no effect on the mean blood flow in the posterior vena cava and femoral vein in cats and healthy volunteers, because instantaneous (phasic) values of the venous blood flow increased during inspiration, while during expiration they decreased to a lower extent than during natural respiration. These phasic changes in the venous blood flow were due to changes not only in intrathoracic, but also in transdiaphragmatic pressure.

Key Words: intrathoracic pressure; venous return; postcaval blood flow; femoral vein blood flow

It is widely accepted that negative intrathoracic pressure (NITP) plays an important role in venous blood return to the heart [3-6]. Taking into account close correlation between venous return and cardiac output [1], it can be assumed that cardiac output should increase with increasing the depth of respiration and, hence, venous return. However, our previous study [2] showed that deep breathing in animals and human subjects were usually not accompanied by considerable changes in cardiac output and blood flow in the abdominal aorta and carotid artery compared to quiet breathing. The increase in the carotid blood flow observed in 42% animals closely correlated with tachycardia induced by increased NITP. In human subjects, carotid blood flow tended to decrease despite tachycardia accompanying deep breathing. These contradictions necessitated more accurate assessment of the relationships between venous return and NITP. In this study we investigated changes in blood flow in the inferior vena cava in anesthetized animals in horizontal position and in awake humans deeply breathing in vertical position.

MATERIALS AND METHODS

The study consisted of 2 experimental series: series I was carried out on 14 naturally ventilated cats (3.5-5 kg) under nembutal anesthesia (25-30 mg/kg), the series II involved 6 young male volunteers.

To increase the depth of respiration and NITP, the animals were exposed to a 2-min test with increased inspiration resistance, during which they inhaled from an evacuated reservoir (6.5±0.5 mm Hg) and exhaled through a special valve to the atmosphere. Blood pressure in the left femoral artery and intrathoracic pressure (in the thoracic part of the esophagus) were measured with Statham P23XL probes, the volume blood flow in the abdominal part of the inferior vena cava (IVC) was determined with a Transonic ultrasound cuff flowmeter. All indices were recorded on a Recor polygraph (Siemens). In humans, blood flow in the left femoral artery was measured with a Philips sonograph. The subjects were asked to perform 6 deep slow inspirations and expirations. The data were analyzed statistically using Student's t test.

RESULTS

In animals, the mean IVC blood flow during quiet breathing was 50.0±7.4 ml/min and NITP during in-

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spiration was -11.8±2.6 mm Hg (Table 1). Inspiratory resistive load induced deep respiration and increased NITP (after 1.5 min) without affecting the mean IVC blood flow (Table 1) and blood pressure.

Thus, the increase in NITP was not accompanied by an increase in the mean blood flow in IVC and seemed to have no effect on venous return to the heart.

The analysis of phasic (instantaneous) changes in the IVC blood flow revealed that the inspiration-related increase in blood flow during deep breathing with elevated NITP was more pronounced than during quiet breathing (the difference was insignificant), whereas during expiration this parameter dropped below the normal (Table 1). Hence, deep breathing was characterized by a significantly higher amplitude of inspiration/expiration oscillations in the IVC blood flow compared to quiet breathing. It can be concluded that despite the amplitude of inspiration/expiration phasic changes significantly increases with increasing NITP, the mean IVC blood flow and instantaneous values

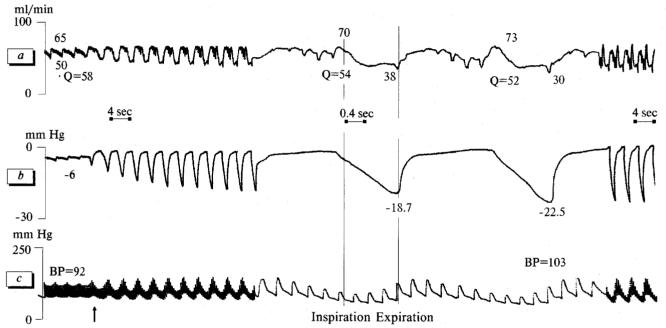


Fig. 1. Effect of increased intrathoracic pressure on blood flow in the vena cava. Original recordings. *a)* blood flow in vena cava; *b)* intrathoracic pressure; *c)* blood pressure. The start of inspiratory resistive load is indicated by an arrow. Numbers over and under the curves correspondto instantaneous blood flow and peak intrathoracic pressure, respectively. Q and BP: mean blood flow and blood pressure, respectively. 4 and 0.4 sec: time calibrations.

TABLE 1. Blood Flow in the Abdominal Part of Vena Cava during Breathing with Inspiratory Resistive Load in Animals with Intact and Open Abdominal Cavity $(M\pm m)$

	Closed abdominal cavity			Open abdominal cavity		
Index	normal breathing	breathing inspiratory resistive load	% of changes	normal breathing	breathing inspiratory resistive load	% of changes
Blood flow, ml/min during expiration	44.5±9.2	34.5±8.7	-23±5	47.0±6.6	40.5±7.4	-14±3
during inspiration	55.5±6.4	64.0±3.9	15±4	57.5±6.0	54.0±7.7	-6±2
Mean blood flow in the abdominal part of the caudal vein, ml/min	50.0±7.4	49.3±5.7	-1.6±3.0	52.3±6.0	47.3±7.1	-9±3
Amplitude of inspiration/expiration blood flow, ml/min	11.0±2.8	29.5±7.3*	168±22	10.5±2.4	14.5±5.3	38±9
Intrathoracic pressure at inspiration peak, mm Hg	-11.8±2.6	-37.6±4.5	219±22	-9.5±1.4	-14.5±2.3	53±11

Note. *p<0.05 compared to normal.

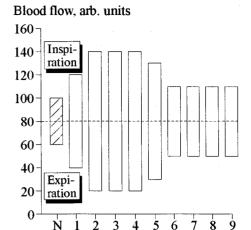


Fig. 2. Changes in instantaneous blood flow values in human femoral vein during normal (N) and deep breathing. Dotted line indicates the mean blood flow.

during inspiration and expiration remain unaffected. Therefore, blood-drawing effect of NITP during inspiration is nullified during expiration.

The dynamics of changes in the IVC blood flow and NITP indicates that respiration-related shifts in instantaneous venous blood flow values do not coincide with changes in NITP (Fig. 1). Venous blood flow peaked (70 ml/min) at the start of inspiration, when NITP was the lowest, and was minimum (54 ml/min) at the start of expiration, *i.e.* when NITP was high. Therefore, there was no temporal correlation between IVC blood flow and NITP.

Inspiratory resistive load induced enhanced contractions of abdominal muscles. To evaluate the effect of transdiaphragmatic pressure on venous blood flow, inspiratory resistive load was applied before and after opening of the abdominal cavity. The mean IVC blood flow in animals with opened abdominal cavity tended to be lower but did not differ significantly from that during normal breathing (Table 1). This decline resulted from a decrease in instantaneous blood flow during both the expiration and inspiration, which was not

observed in intact animals (Table 1). Thus, opening of the abdominal cavity reducing the transdiaphragmatic pressure did not increase, but even decreased the instantaneous and mean blood flow values under conditions of deep breathing.

The observations performed in human volunteers confirmed the regularity revealed in animal experiments: deep breathing did not affect the mean blood flow in the femoral vein, while the increase in blood flow during inspiration and its decrease during expiration were more pronounced than during normal breathing. Like in animals, the amplitude of inspiration/expiration blood flow oscillations in the femoral vein was significantly higher than during normal breathing (Fig. 2). It should be stressed that in humans all these changes disappeared after 4 deep inspirations. The mechanism of this phenomenon is still to be examined.

Thus, both humans and animals reveal no significant changes in the mean IVC blood flow under conditions of deep breathing with increased NITP. This is explained by diverse changes in instantaneous blood flow during inspiration (increase) and expiration (decrease). These phasic oscillations in the venous blood flow are due to changes not only in intrathoracic, but also in transdiaphragmatic pressure. These data suggest that NITP has no effect on venous return to the heart.

REFERENCES

- 1. B. I. Tkachenko, Ros. Fiziol. Zh., 85, No. 9, 1255-1266 (1999).
- B. I. Tkachenko, V. I. Evlakhov, and I. Z. Poyasov, Byull. Eksp. Biol. Med., 129, No. 2, 129-132 (2000).
- F. L. Abel and J. A. Waldhausen, Am. Heart J., 78, No. 2, 266-275 (1967).
- 4. G. A. Brecher and G. Jr. Mixter, Am. J. Physiol., 172, No. 2, 457-461 (1953).
- G. Harms, T. J. Wetter, S. R. McClaran, and D. F. Pegelow, J. Appl. Physiol., 85, No. 2, 609-618 (1998).
- J. Raine, A. N. Redington, and A. Benatar, Eur. J. Pediatr., 152, No. 7, 595-598 (1993).