PHYSIOLOGY

Arterial Blood Flow during Deep Breathing

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

Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 129, No. 2, pp. 129-132, February, 2000
Original article submitted July 28, 1999

An increase in the negative intrathoracic pressure during deep breathing did not change blood flow rate in the carotid artery in 100% humans and 57.2% cats, while in 42.8% cats this parameter strongly correlated with acceleration of the heart rate. Similar relations were found for the ascending and abdominal aorta.

Key words: *intrathoracic pressure; arterial blood flow; cardiac output; carotid blood flow*

The interactions between cardiovascular and respiratory systems manifest itself in modulation of blood flow and pressure in major arteries by respiration [1, 5]. Blood flow rate and pressure in arteries during inspiration are usually lower by 5-10% compared with expiration. The deeper inspiration, the more significant is the decrease [2,3]. Arterial blood pressure and flow rate fluctuate due to different factors: the negative intrathoracic pressure (NIP), changes in capacitance and resistance of pulmonary vessels, cardiac preload and afterload, and others [4]. It is proved that arterial blood flow varies synchronously with respiratory cycle. However, circulatory shifts induced by long-term (compared to respiratory cycle) changes in respiration pattern were not studied. At the same time, deep breathing is normally associated with various changes in the functional state (physical exercise, hyperthermia, *etc.*) as well as with pulmonary and cardiovascular pathologies, and can lead to changes in arterial blood flow. Our aim was to study the influence of deep respiration on flow rates in arterial part of the cardiovascular system.

MATERIALS AND METHODS

Acute experiments were performed on 28 spontaneously breathing cats (3.5-5.0 kg) anesthetized with Nembutal (25-30 mg/kg, intramuscularly). Deep respiration and an increase in NIP was produced by applying a negative pressure load during inspiration. The animal inspired air from a reservoir with 16.5±0.5 mm Hg negative pressure for 2 min, and expired freely through a special valve. Blood pressure in the left femoral artery was measured with a Statham P23XL transducer. Intrathoracic pressure was measured in the thoracic part of the esophagus. Blood flow in the carotid artery (CA) was measured with an Transonic ultrasonic flowmeter (cuff gauge). Blood flow in the ascending and abdominal aorta (cardiac output) was measured with a Nihon Kohden MVF-2100 electromagnetic flowmeter (cuff gauge). Heart rate (HR) was calculated from *RR* intervals on the ECG recorded in standard lead II. Blood gases were determined with a Radiometer gas analyzer. Blood pressure, flow rate in CA and in the ascending and abdominal aorta, and intrathoracic pressure signals were recorded on a Recor polygraph (Siemens).

In 6 young volunteers, flow rate in the left CA was measured in vertical position with a Philips sonograph ultrasonic detector. Subjects were asked to perform 6 slow deep inspirations and expirations during 30 sec. The data were statistically analyzed by Student's *t* test.

RESULTS

In cats, inspiratory load increased NIP by 196±8% (from -5.6±0.9 to -16.6±1.8 mm Hg). The instant blood

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

0007-4888/00/0002-109$25.00

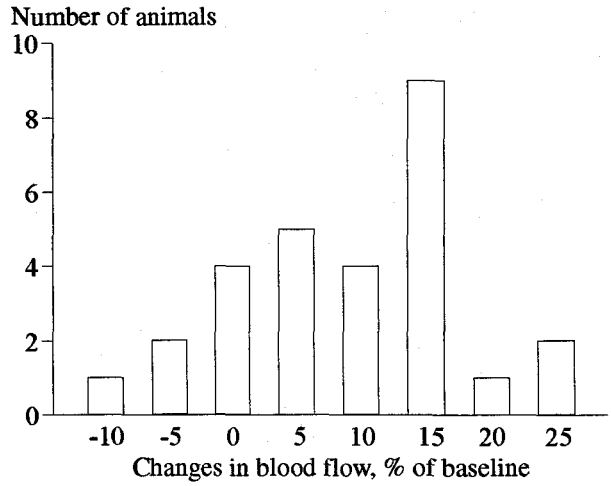

Fig. 1. Distribution histogram of animals according to changes in carotid blood flow under conditions of inspiratory load.

flow rates varied with the respiration cycle, being lower during inspiration and higher during expiration. The amplitude of these phasic (inspiration-expiration) modulations increased with the depth of respiration, the maximum increase being 10%. At the same time, blood flow increased only by 6.0±1.7% ($p<0.05$) compared with its baseline value (34±4 ml/min). The blood flow in CA increased in response to the inspiratory load in 42.8% of animals and did not increased in 57.2% (Fig. 1). Changes in flow rate varied, while the rise of NIP was practically the same in all animals. The blood flow started to increase 20 sec after the onset of the load and reached its maximum after 90 sec. Simultaneously, blood pressure and HR increased by 14±5% (from 125±4 mm Hg) and 12±4% (from 192±8 beats/min), respectively. A strong correlation was found between changes in blood flow in CA and HR (r= 0.82±0.07, $p<0.05$). It can be hypothesized that in our experiments the rise in the mean blood flow was due to the increase in HR in response to the inspiratory load.

In this case tachycardia can result from enhanced sympathetic influences on the heart due to activation of carotid chemoreceptors in response to shifts in arterial blood gases. A long latency (20 sec) of CA blood flow rise confirms this assumption. To check this hypothesis, we determined O_2 and CO_2 tensions in the arterial and venous blood (Table 1). Inspiratory load slightly decreased Po_2 and increased Pco_2 in arterial and venous blood. However, these changes were insignificant. Moreover, there was no correlation between Po_2 and HR (r=0.34±0.3). Thus, the changes in blood gases can not be considered as a basic mechanism for the tachycardic response to increased inspiratory resistive load, although may contribute to it. The rise in HR can also be explained by reduced parasympathetic influences on the heart due to increased NIP, but this is the matter of other studies.

Forced respiration indistinctly changed blood flow in the abdominal and ascending aorta (cardiac output). Cardiac output increased significantly only in two cats (by 7% and 9%), while HR in these animals increased by 6% and 11%, respectively. In one cat cardiac output decreased, and in 3 cats it remained unchanged. Only in 1 of 4 cats, blood flow in the abdominal aorta increased significantly (8%), which was accompanied with tachycardia (7%). In other 3 cats, neither blood flow, nor HR increased.

Thus, the increase in NIP not necessarily leads to changes in cardiac output and blood flow in the abdominal aorta or in CA. If any, these changes can be either positive or negative. However, when these indices increase, HR also increases.

Under conditions of inspiratory load, pulmonary hemodynamics can be disturbed dramatically due to increased pulmonary vascular resistance [5]. Thus, the alternative (bidirectional) shifts in arterial blood flow in response to the inspiratory load, or their absence can be determined by peculiarities of experimental model [2]. Moreover, lung extension at the same intrathoracic pressure can vary in different animals due to different lung compliance (elastic or non-elastic) and bronchial resistance, which produced different

TABLE 1. Arterial and Venous Blood Gases under Conditions of Inspiratory Load in Cats ($M±m$)

Parameters		Respiration		
		normal	with inspiratory load	
			abs.	Δ, % of baseline
Arterial blood	Po_2, mm Hg	92.5±4.3	85.9±4.1	-7±3
	Pco_2, mm Hg	32.2±3.8	34.0±3.7	6±2
	pH	7.42±0.01	7.41±0.03	-0.15±0.12
Venous blood	Po_2, mm Hg	62.0±1.6	57.7±4.3	-7±3
	Pco_2, mm Hg	48.1±4.6	51.6±7.6	7±4
	pH	7.33±0.05	7.31±0.04	-0.27±0.15

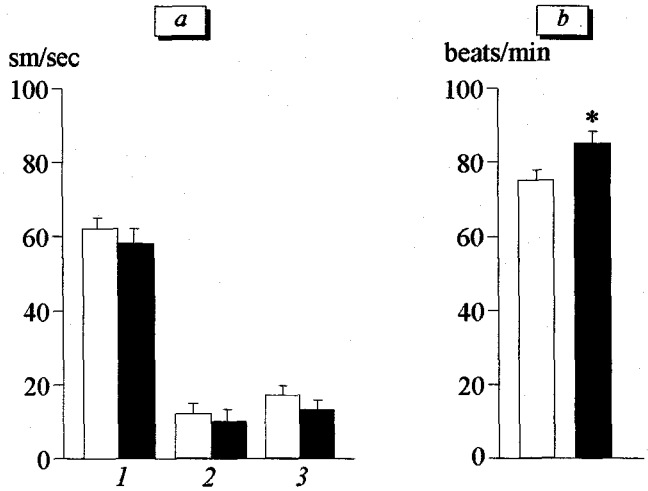

Fig. 2. Blood flow (*a*) in the carotid artery and heart rate (*b*) in humans during normal (light bars) and deep breathing (dark bars). Systolic blood flow (*1*); diastolic blood flow (*2*); mean flow rate (*3*); *$p<0.05$ compared with heart rate during normal respiration.

changes in the tidal volume and pulmonary vascular capacitance and resistance. In this connection, experimental studies on human subjects, who could voluntarily control respiration without changing the respiratory resistance are of special importance.

Observations on humans demonstrated that during both quiet and forced respiration, the blood flow in CA changed synchronously with the respiration cycle. Instant flow rates were lower during inspiration and higher during expiration. The decrease in systolic, diastolic, and mean flow rates in CA observed during forced respiration (30 sec after beginning) was insignificant (Fig. 2). HR increased by 13±4% (from 75±2 to 85±3 beats/min, $p<0.05$) during forced respiration (Fig. 2, *b*). Similar tachycardia was observed in all subjects.

Thus, in humans, the blood flow in CA virtually did not change during deep breathing, but HR increased. In animals, this tachycardia strongly correlated with rise of CA blood flow, while in humans it had a tendency to decrease because deep breathing without inspiratory load resulted in a pronounced increase in the lung volume and, probably, pulmonary vascular capacitance. Under these conditions venous return to the right atrium increased, and blood can be deposed in the lungs. In this case the blood flow in CA decreased. In animals, deep breathing was associated with increased inspiratory resistance. Therefore, tidal volume not necessarily increased. In addition, this can lead to increased resistance of pulmonary vessels, which impedes blood deposition in the lungs. For more accurate interpretation the relationships between NIP and venous return should be calculated.

REFERENCES

1. A. Gaiton, *Cardiac Minute Volume and Its Regulation* [in Russian], Moscow (1969).
2. R. Berne and M. N. Levy, *Cardiovascular Physiology*, 3rd Ed., St. Louis (1977).
3. H. Bjurstedt, G. Rosenhamer, C. M. Hesser, and B. Lindborg, *J. Appl. Physiol.*, **48**, No. 6, 977-981 (1980).
4. J. A. Innes, S. C. De Cort, W. Kox, and A. Guz, *J. Physiol. (London)*, **460**, 487-502 (1993).
5 L. B. Rowell, *Human Cardiovascular Control*, Oxford (1993).