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Phrenic Artery Blood Flow under Loaded Respiration

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Acute experiments on anesthetized cats with additional resistive loads to inspiration and/or expiration revealed adequate changes in vascular bed resistance and blood flow rate in the phrenic artery. The increase in the phrenic artery blood flow during airflow resistive loading by 30-40 mm H_2O was similar to the changes previously found for inspiration of gas mixtures containing 5% O_2 and 5% CO_2. Minute ventilation increased 2 times under hypoxia and 3.5 times under hypercapnia. This index did not change under additional loading.

Key words: *phrenic artery; blood flow; loaded respiration; ultrasound*

Additional resistive loading to respiration is coupled with increased respiratory muscle work that needs an extra oxygen supply [1,2,12,13]. This may be achieved by increasing either blood flow in the respiratory muscles or oxygen extraction from the blood (i.e., rise in O_2 arteriovenous difference) in the muscles [12]. Circulation in respiratory muscles under inspiratory loading has been studied previously. It was less explored, however, how the circulation in respiration muscles is affected by additional expiratory loading.

In present work we compared the phrenic artery blood flows during inspiratory and expiratory as well as coupled inspiratory-expiratory resistive loading.

MATERIALS AND METHODS

Acute experiments were performed under Nembutal anesthesia (40-50 mg/kg, intraperitoneally) on 14 male and female cats weighing 2.2-3.8 kg. Linear and volume blood flow rates were measured in the left phrenic artery (PA) using miniature band ultrasonic transducers (inner diameter range 0.3-0.5 mm, working frequency 26.8 mHz). The transducers were calibrated to measure volume flow rate. Linear velocities were determined from the Doppler frequency changes [7]. Arterial pressure (AP) was registrated in the femoral artery by a semiconductor manometer [6]. The signals of AP and PA blood flow were fed to an analogue minicomputer to obtain PA vascular bed resistance in the real time. Experiments were performed on spontaneously breathing animals with closed abdominal cavity. The method of PA blood flow recording was described previously [7]. In order to detect the chest excursions the lower part of the chest was wrapped with an elastic bandage with a strain gauge fixed on it. Minute ventilation (MV) was measured. Additional loads to respiration were made with two water valves. Blood flow rate and other above-mentioned parameters were studied under inspiratory and expiratory as well as coupled inspiratory-expiratory resistive loading with 30-60 mm H_2O. The loading test lasted from 3 to 60 min.

RESULTS

Additional resistive loading when breathing with air does not produce statistically significant changes in MV, although respiratory rate is decreased [2,3,5]. At the same time, the respiratory muscle work increases approximately 2-fold [3]. There is evidence that MV is decreased during loading [4].

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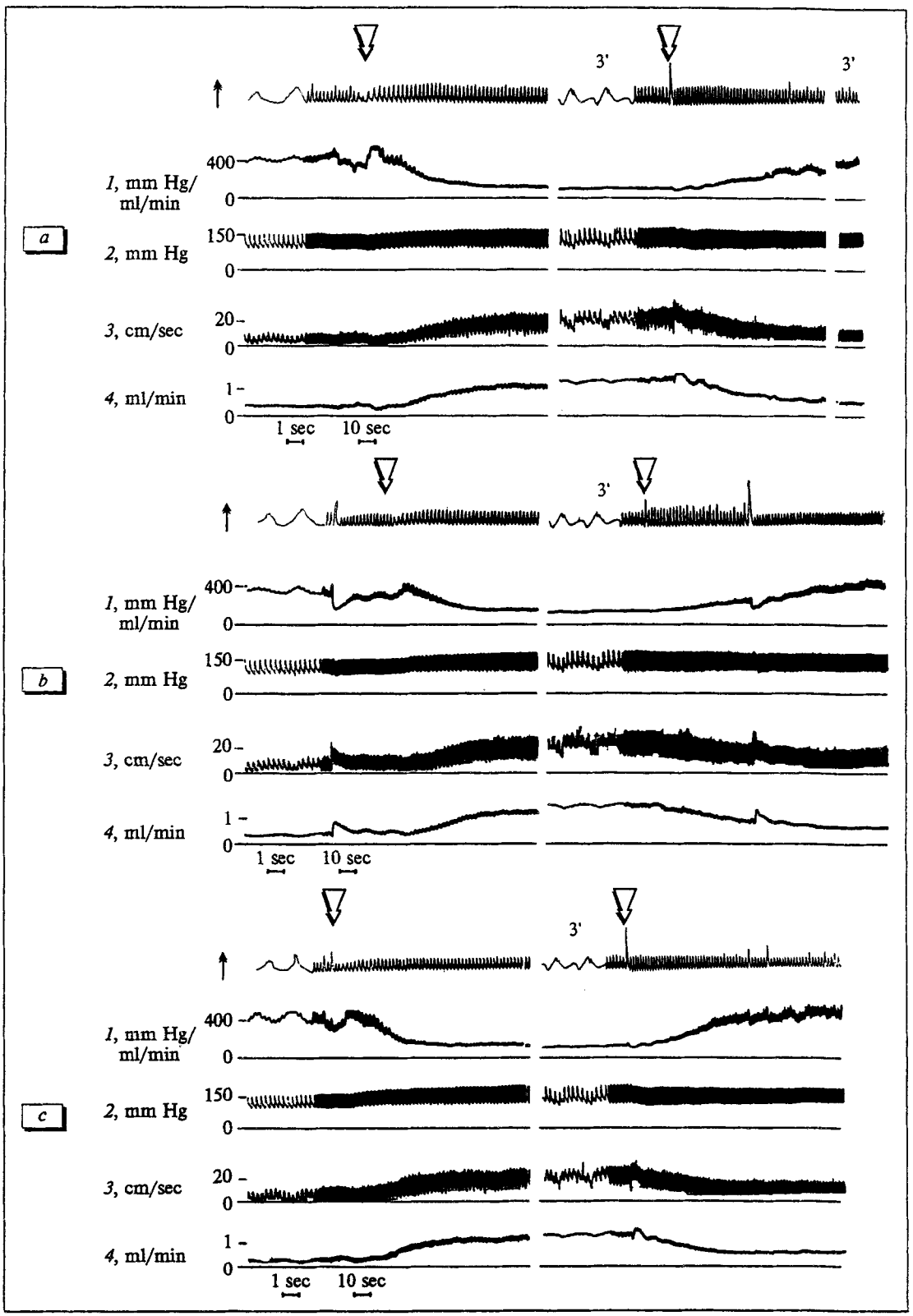

Fig. 1. Changes in the phrenic artery blood flow due to inspiratory (*a*), expiratory (*b*) and inspiratory-expiratory (*c*) resistive loading by 30 mm H_2O. The records are: vascular bed resistance (*1*); arterial pressure registrated in the femoral artery (*2*); linear (*3*) and volume (*4*) flow rates in the phrenic artery; Up-arrows — inspiration; thin lines — zero levels; Down arrows — the start and the end points of respiration with a load; the figures at the tops of the records — the time (min) after stimulus application or removal.

In our experiments the initial breathing rate ranged from 20 to 44 cycles/min (predominantly 24-28 cycles/min). While loading, it was decreased by 2-8 cycles/min in 9 experiments, remained unchanged in 4 experiments; and increased by 4 cycles/min in a single experiment. Almost in all experiments, the amplitude of respiratory movements increased, while MV remained unchanged or within 10% from initial level.

The initial AP values in different cats ranged from 125/75 mm Hg to 200/120 mm Hg. When additional loading was applied during inspiration or expiration, systolic AP increased by 5-30 mm Hg. Diastolic pressure did not change or increased to a lesser degree than systolic AP; in some experiments it decreased by 10-20 mm Hg. As a result, pulse pressure increased during loading by 30-40 mm Hg (range 15-50 mm Hg).

Volume blood flow rate was 0.43 ml/min (varied from 0.1 to 1 ml/min from animal to animal, for most of them (n=8) being within 0.25-0.4 ml/min). These values are very similar to those (about 3 ml/min) obtained on dogs weighing 8-10 times as much as cats [11,14]. They are also comparable to the values obtained on cats [7].

Linear flow rate was 6.1 cm/sec (3-14 cm/sec). The PA vascular bed resistance was 437 mm Hg/ml/min (200-750 mm Hg/ml/min).

Absolute values of blood flow did not depend on animal weight.

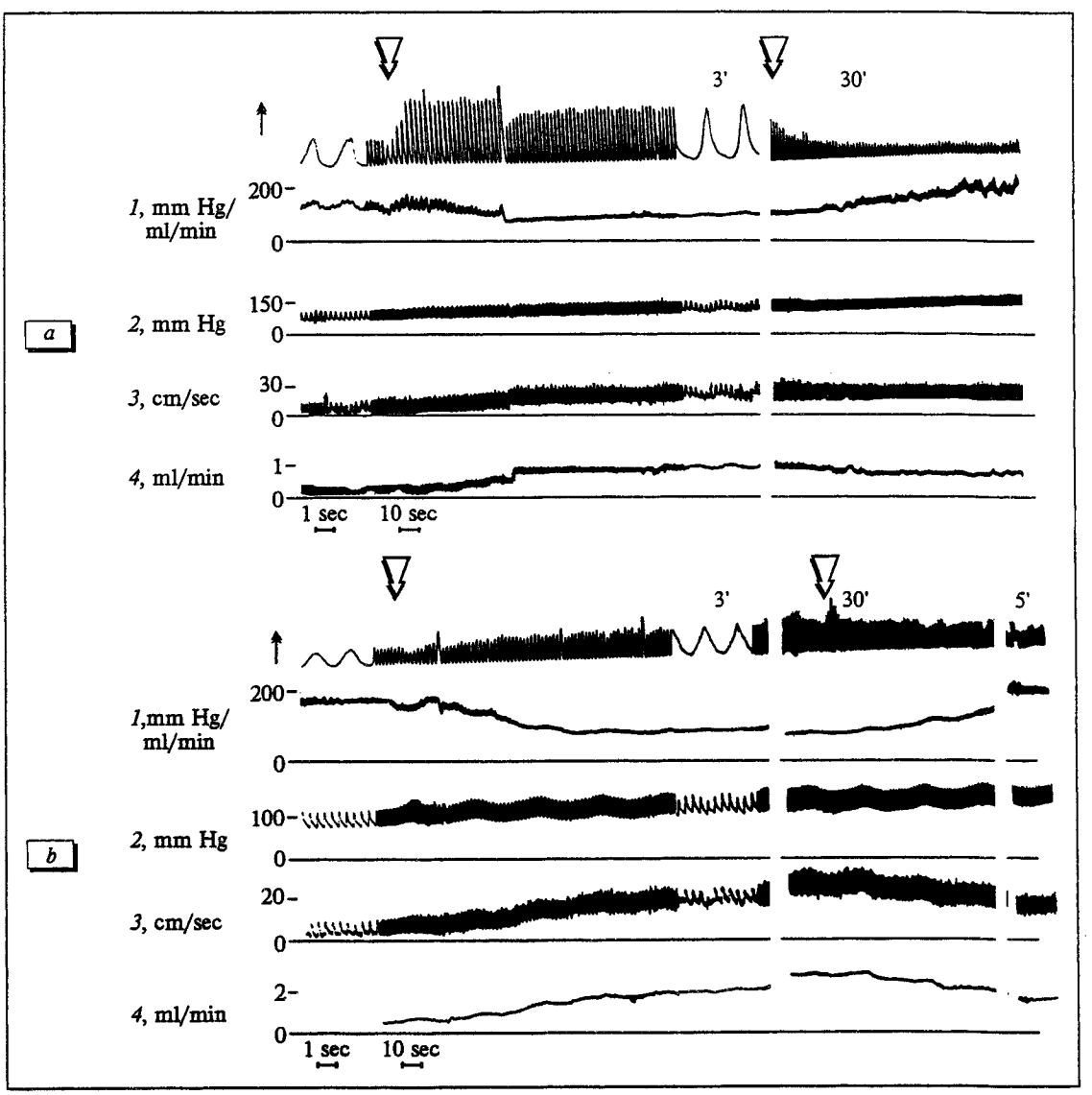

Fig. 2. Changes in the phrenic artery blood flow due to prolong inspiratory-expiratory resistive loading by 50 mm H_2O. Two types of reactions: (a) and (b). For details see Fig. 1.

Large animals might have low values, while small ones — high values of volume and linear rates of PA blood flow.

An additional inspiration loading with 30-40 mm H_2O decreased the PA vascular bed resistance by 40% (21-60%) compared with initial level and increased the volume blood flow (Fig. 1, *a*). The volume flow rate grows in the mean up to 239% (160-400%, in different experiments) compared with the initial level). The linear flow rate increased to 209% (160-320%) of the initial level. These changes in the PA vascular bed resistance and in volume blood flow are comparable to those observed during respiration with gas mixtures containing 5% O_2 and 5% CO_2, when MV grew up substantially. No changes in MV were observed during respiratory loading.

Expiratory loading produced similar changes in the PA vascular bed resistance as inspiratory loading (Fig. 1, *b*). There is evidence that resistive loading during expiration is more difficult to overcome than that during inspiration [5]. The load that a narcotized cat is able to overcome during expiration does not exceed 15-20 mm H_2O [4].

In our experiments cats easily overcame as much loads during expiration as 30-40 mm Hg. It is assumed that adaptation to additional loading is achieved not by increased activity of expiratory muscles, but is due to a more intense inspiration resulting in larger lung expansion and, therefore, in greater elastic return, providing expiration with greater force [10]. This is in agreement with our results showing that PA blood flow changes are very similar under either inspiration or expiration loading. Meanwhile, enhanced activation of both inspiratory and expiratory neurones was observed under expiration loading [4].

With both inspiration and expiration loading, no summation of changes in the blood flow occurred: the PA vascular bed resistance as well as the PA blood flow grew up to the same values as if inspiration or expiration were loaded separately (Fig. 1, *c*).

Because of variations in the initial level of blood flow, the percentage of the blood flow increase was widely dispersed. The absolute volume flow rates regardless of their initial levels achieved much the same values: 1.44 ml/min, ranging within 1.0-1.6 ml/min. These values seem to be equal to energy demands of the diaphragm for a given resistive load. It should be noted that under a higher resistive loading (50-60 mm Hg), the absolute values of the volume flow rate were quite similar to those mentioned above.

The diaphragm has a multiple blood supply. Each half of the diaphragm is supplied via the PA, internal thoracic artery, and 8-12 intercostal arteries [9,14]. However, a strong correlation was shown between changes in the PA blood flow in dogs and the diaphragm blood supply as a whole, as measured with the use of microspheres [11]. This implies that the relationships found for changes in the PA vascular bed resistance and blood flow under loaded respiration adequately describe diaphragmatic hemodynamics as a whole.

Previously, we showed that blood flow in the PA alters synchronously with breathing: during inspiration (diaphragm contraction), the linear and volume flow rates decrease, during expiration they increase. Blood flow in the PA during inspiration was equal to 79% (62.5-90%) of that during expiration [7]. During forced breathing (under either hypoxia or hypercapnia) these proportions were the same, while the total blood flow increased. By resistive loading, variations of blood flow in accordance with respiration phases were more significant: blood flow in the PA during inspiration was equal to 67% (50-83%) compared with expiration.

Our experiments demonstrate that a prolong inspiratory-expiratory resistive loading (up to 60 min) results in two basic reactions: some animals responded by a decrease in the PA vascular bed resistance, the blood flow growing during the first 2-3 min of loading and then remaining at this level till the end of loading. After removal of the load, the PA vascular bed resistance and blood flow returned to their initial levels during a longer period than after a short-lasting respiration loading (after 5-10 min, Fig. 2, *a*). In other animals, the PA vascular bed resistance decreased, and the blood flow was growing during the entire loading period. When the load was removed, all parameters gradually restored (Fig. 2, *b*).

In some experiments we observed a drop in the blood flow after 20-30 min of loaded respiration. This was followed by a notable reduction in the amplitude of breathing movements. This shows indirectly that limited blood supply may account for the weakening of breathing movements.

Thus, resistive inspiration and/or expiration produces similar changes in the PA vascular bed resistance and in the PA blood flow. The increases in the phrenic artery blood flow during airflow resistive loading by 30-40 mm H_2O were much the same as the changes previously found for inspiration of gas mixtures containing 5% O_2 and 5% CO_2. Minute ventilation increased 2 times under hypoxia and 3.5 times under hypercapnia [8]. Under these conditions respiratory muscles performed a dynamic work. Under additional respiration loading, MV did not change, although making a force against resistive load, which needed adequate energy expenses resulting in an appropriate excess of blood supply to the diaphragm.

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