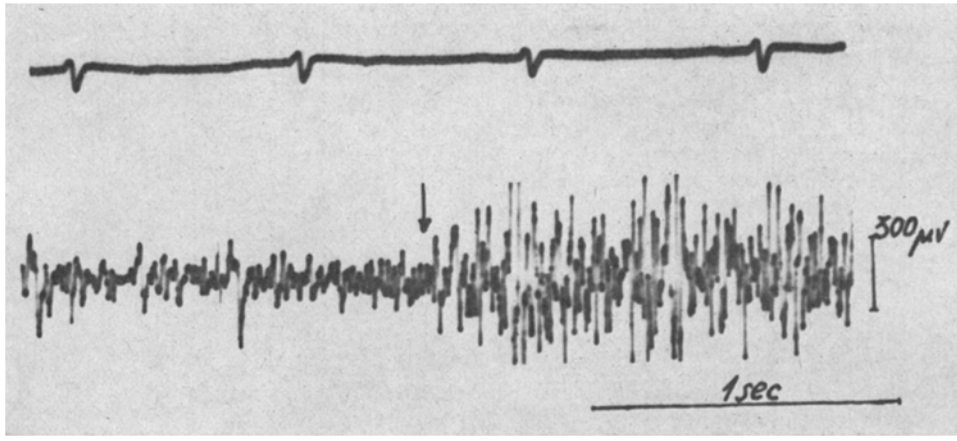

Fig. 1. Top record. While standing. There is no electrical activity from the intercostal (except ECG). Bottom record. While leaning forward. Tonic electrical activity from the same muscles. Rotation of the head to the left (row) results in increase the tonus of the muscles of that side.

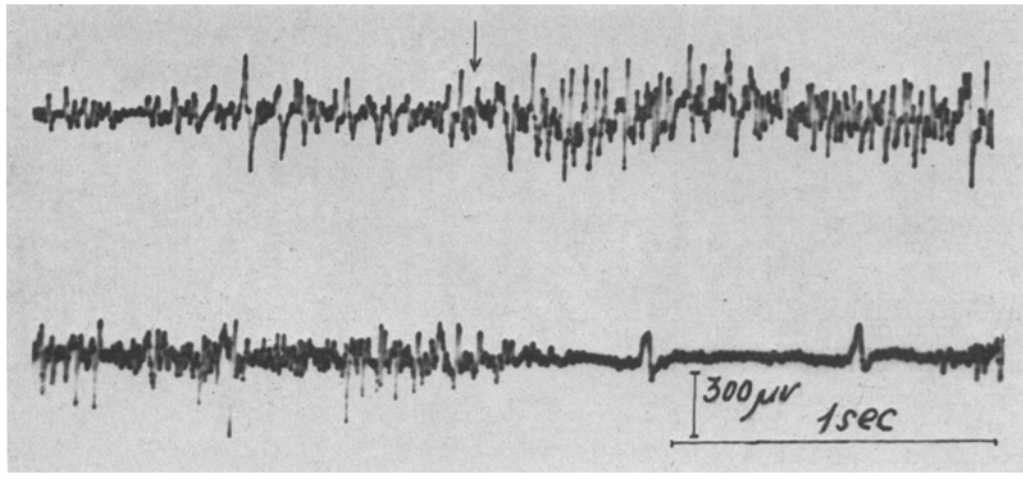

Fig. 2. Top record. While leaning forward. Tonic electrical activity from intercostal. Backward tilting of the head (row) increases the tonus of the muscles. Bottom record. Forward tilting of the head (row) decreases the tonus.

Thus the MAGNUS reflex may be demonstrated in normal adults, not only in the limb musculature but also in the chest musculature if the muscles were first hypertonic.

ВЫВОД. Рефлексы Магнуса могут быть вызваны в мышцах грудной клетки человека, но для этого надо, чтобы эти мышцы находились в состоянии повышенного тонуса.

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Do the Respiratory Muscles and Lung Receptors Influence the Respiratory Sensitivity to CO_2?

According to CAMPBELL[1], breathlessness in man may be prevented by paralysis of respiratory muscles, and according to GUZ[2], by the vagal blockade. In connection with these observations it is of interest to know whether afferent impulses from the respiratory muscles and from the lungs change respiratory sensitivity to CO_2. Both the old and recently published results are contradictory[3–11].

Our investigations which are presented in this paper permit the following conclusion. Neither the impulses from the respiratory muscles nor the impulses from the lungs influence the respiratory sensitivity to CO_2. In fact the respiratory sensitivity to CO_2 depends on Pa CO_2. If the experimental operation increases Pa CO_2 in the animal, the respiratory sensitivity to CO_2 is decreased; if, on the contrary, the experimental operation in the animal decreases Pa CO_2, the respiratory sensitivity to CO_2 is increased.

The experiments were performed on 10 adult cats anaesthetized with 30–40 mg/kg nembutal. The animals were subjected to passive overventilation lowering the Pa CO_2 till the appearance of hypocapnic apnoe. Expired volumes were measured by connecting the tracheal

Table I

Spinalization	Minute volume (l/min) Before	After	Frequency (resp./min) Before	After	Spontaneous breathing: pHa Before	After	Spontaneous breathing: Pa CO_2 Before	After	At the beginning of apnoe: pHa Before	After	At the beginning of apnoe: Pa CO_2 Before	After	At the beginning of respiration after apnoe: pHa Before	After	At the beginning of respiration after apnoe: Pa CO_2 Before	After	Duration of hypocapnic apnoe (sec) Before	After
Mean	0,540	0,360	24	14	7,244	7,216	34,2	37,4	7,360	7,346	23,4	27,8	7,242	7,196	34,0	44,0	47	80
SD	0,043	0,033	4,52	2,23	0,002	0,001	3,27	1,8	0,001	0,002	1,14	1,64	0,001	0,002	1,35	3,93	15,4	13,7
P		$< 0{,}01$		$< 0{,}01$		$< 0{,}02$		$< 0{,}01$		$> 0{,}2$		$< 0{,}01$		$< 0{,}02$		$< 0{,}01$		$< 0{,}01$

pHa, pH arterial blood; Pa CO_2, partial pressure of carbon dioxide in arterial blood; Mean, mean values; SD, standart deviations; *P*, significance of differences from values for normal animals; *n*, number of animals.

Table II

Vagotomy	Minute volume (l/min) Before	After	Frequency (resp./min) Before	After	Spontaneous breathing: pHa Before	After	Spontaneous breathing: Pa CO_2 Before	After	At the beginning of apnoe: pHa Before	After	At the beginning of apnoe: Pa CO_2 Before	After	At the beginning of respiration after apnoe: pHa Before	After	At the beginning of respiration after apnoe: Pa CO_2 Before	After	Duration of hypocapnic apnoe (sec) Before	After
Mean	0,460	0,460	22	12	7,276	7,332	35,2	29,6	7,418	7,452	26,2	19,9	7,314	7,330	37,1	30,4	45	26
SD	0,031	0,035	4,0	2,5	0,008	0,017	2,33	2,04	0,002	0,003	1,35	1,25	0,02	0,02	2,25	0,96	10,2	8,5
P		$> 0{,}5$		$< 0{,}01$		$< 0{,}01$		$< 0{,}01$		$< 0{,}05$		$< 0{,}01$		$< 0{,}02$		$< 0{,}01$		$< 0{,}01$

See the glossary of abbreviations in Table I.

cannula through a non-return valve to a bellow spirometer. Electromyograms were recorded by bipolar steel-wire electrodes affixed to diaphragm. Blood samples were obtained from femoral arteries. Blood pH and Pa CO_2 measurements were made in micro Astrup.

In the first group of experiments 5 animals were spinalized at the level C_8-Th_1. As can be seen (Table I), the respiration after apnoe in spinalized cats appears at Pa CO_2 = 44 mm Hg instead of 34 mm Hg as before spinalization. Thus the respiratory sensitivity to CO_2 after spinalization decreases. But the decrease does not depend on the ablation of afferent respiratory muscle impulses – it depends on the increase of Pa CO_2 following the spinalization.

This conclusion is confirmed by the experiments of SANT'AMBROGIO et al.[11]. According to these authors, in rabbits after bilateral phrenicotomy the $\dot{V}_E$[12] decreases, Pa CO_2 correspondingly increases and the respiratory sensitivity to CO_2 decreases. In cats after bilateral phrenicotomy the extradiaphragmatic inspiratory muscles compensate the $\dot{V}_E$. No significant change in arterial pH and Pa CO_2 were recorded, and correspondingly the sensitivity of the responses to carbon dioxide had not changed.

In the second group of experiments, double vagotomy in the mid-cervical region were performed in 5 cats. As it is seen (Table II) before vagotomy the respiration after apnoe begins at Pa CO_2 = 37.1 mm Hg, after vagotomy – at 30.4 mm Hg. Thus the respiratory sensitivity to CO_2 after vagotomy increases. But again, it does not depend on the ablation of afferent vagal impulses. The increase of the respiratory sensitivity to CO_2 depends on the decrease of Pa CO_2 following the vagotomy.

This conclusion is confirmed by the analysis of the data of other authors[4–7,9], using anaesthetized cats and dogs and reported that vagotomy increases the respiratory sensitivity to CO_2. Vagotomy in these animals increases the alveolar ventilation and decreases Pa CO_2. On the contrary, workers[8,10] using anaesthetized rabbits have reported that vagotomy decreases the respiratory sensitivity to CO_2. Vagotomy in rabbits decreases alveolar ventilation and increases Pa CO_2. The latter also depends on the weakness of the respiratory muscles of the rabbits.

Thus neither the impulses from the respiratory muscles nor the impulses from the lungs influence the respiratory sensitivity to CO_2. There must be some other mechanisms to explain the effect of muscle paralysis and vagal blockade in breathlessness.

ВЫВОДЫ. Ни импульсация с дыхательных мышц, ни импульсация с лёгких не оказывает влияния на чувствительность животного к CO_2. В действительности чувствительность к CO_2 обусловливается изменением напряжения CO_2 крови, вызываемым самим оперативным вмещательством. Если паралич дыхательных мышц или ваготомия вызывают изменения дыхания, ведущие к повышению Pa CO_2, чувствительность животного к CO_2 снижается. Если, наоборот, эти вмешательства вызывают снижение Pa CO_2, чувствительность животного к CO_2 повышается.

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[12] V_E = expired gas volume per unit time.

Der Einfluss der γ-Globuline auf die Viskosität

Viskositätswirksam für die Plasmaviskosität sind die Gesamtprotein-, die Fibrinogen-, die α_1-, α_2-, β- und γ-Globulinkonzentration, für die Vollblutviskosität der Hämatokrit, die Erythrocytenaggregationstendenz und die Plasmaviskosität[1,2]. Erhöhte und pathologische γ-Globuline sind für die Hyperviskosität beim Myelom und anderen Makroglobulinaemien verantwortlich[3,4]. Untersuchungen über das Viskositätsverhalten der γ-Globuline zu unterschiedlichen Fibrinogen- und Albuminkonzentrationen wurden bisher nicht durchgeführt.

Material und Methodik. Die Untersuchungen wurden mit γ-Globulin (γ-Globulin aus Humanserum, lyophil, rein, 97%, Serva, Heidelberg), Fibrinogen (Forschungs-Fibrinogen-Kabi, Deutsche Kabi GmbH, München 15) und Albumin (Human-Albumin, trocken, «reinst», Behringwerke AG, Marburg-Lahn) derart durchgeführt, dass zu konstanten Ausgangskonzentrationen (0,5 g/l, 1,0 g/l, 2,0 g/l, 3,0 g/l) der einen Lösung, z. B. von 0,5 g/l γ-Globulinlösung, deren Ausgangsviskosität gemessen wurde, «variable» Konzentrationen von 0,5 g/l, 2,0 g/l, 4,0 g/l, 6,0 g/l und 7,5 g/l der anderen Lösung z. B. Albumin gegeben und die Viskosität gemessen wurde.

Die Viskositätsmessungen wurden mit dem Harkness-Viskosimeter[5] bei konstanter Temperatur (25 °C), einem Schergrad von 660 sec^{-1}, einem stets gleichbleibenden Druck von 75 mmg Hg und einer kinematischen Viskosität von 2×10^{-2} cm^2 pro sec mittels elektrischer Zeitmessung mit einer Genauigkeit von 0,02 sec als Durchschnittswert von 5 Einzelmessungen durchgeführt.

Die Angabe der Viskosität erfolgt in sec als Differenz von η Lösung – η 0,9% NaCl-Lösung.

Ergebnisse. Die Ergebnisse zeigen die Figuren 1–6. Die Viskosität einer Lösung aus γ-Globulinen und Fibrinogen

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