

## Feature of Functional Condition of Respiratory Muscles of Patients with Bronchial Asthma in Prevention of Complications of Chronic Respiratory Insufficiency

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### Abstract

We study and assess the functional state of the respiratory muscles in patients with mild and moderate bronchial asthma. We also analyse the following indicators: echocardiography, biochemical blood tests, coagulogram, external respiration function. This study allows us to evaluate the prognostic value as a predictor of the adverse course of bronchial asthma and can serve as the basis for developing indications for the differentiated complex use of treatment methods, including electromyostimulation in respiratory muscle dysfunction, which help to relieve the manifestations of respiratory muscle fatigue syndrome, will improve respiratory functions and increase quality of life for patients and expand rehabilitation programs.

**Keywords:** Respiratory Muscles; Bronchial Asthma; Chronic Respiratory

### Introduction

Bronchial asthma is a serious social and medical problem. Bronchial asthma (AD) is currently the most common respiratory disease in the world. Asthma is a heterogeneous chronic inflammatory disease of the respiratory tract that affects about 300 million people worldwide. About 5 - 10% of all asthmatics suffer from severe or uncontrolled asthma, associated with increased mortality and hospitalization, reduced quality of life and increased health care costs.

The skeletal muscle dysfunction lies among the systemic complications of the pathology of the respiratory system, which progresses in parallel with a decrease in the functional reserve of the lungs and an increase in shortness of breath, leading to even greater depletion of a patient and a decrease in exercise tolerance [9]. Weakness and atrophy of skeletal and respiratory muscles is one of the most common systemic effects in pulmonary patients [2,4,5,7]. One of the main ones is hyperinflation of the lungs, leading to flattening of the diaphragm. A clinical assessment of the functional status of respiratory muscles (RM) is usually associated with elucidating their "contribution" to the development of respiratory failure (DN), determining the compensatory reserves of various muscle groups and ways to correct contractile function. RM have great reserve capabilities, however, with excessive loads their dysfunction develops [6,10]. Dysfunction of the respiratory muscles is a condition in which there is a decrease in the strength and speed of its contractions as a result of a significant increase in the volume of work performed, insufficient energy supply, as well as the initial non-physiological state of the muscles [3]. Chronic inflammation causes the development of bronchial hyperreactivity, which leads to repeated episodes of wheezing, shortness of

breath, chest tightness and coughing, especially at night or in the early morning. These episodes are associated with widespread variable airway obstruction in the lungs, which is often reversible spontaneously or under the influence of treatment. An increase in respiratory resistance during bronchial obstruction leads to an increase in the load on the respiratory muscles, their hyperfunction, and asynchronous ineffective work [2,11]. The progression of the disease is accompanied by a deterioration in energy supply while increasing energy consumption, the development of fatigue of the respiratory muscles, which in turn exacerbates ventilation disorders [1,2,8]. That is why early diagnosis and treatment of RM dysfunction is an urgent task in clinical practice.

**Objective of the Study**

To identify the functional state of respiratory muscles, an echocardiogram, respiratory function, and biochemical blood parameters in patients with bronchial asthma (BA).

**Materials and Methods**

We observe 28 patients with mild to moderate BA and 20 healthy individuals. The age of the patients is  $41.6 \pm 7.5$  with a disease duration of  $12.3 \pm 3.5$  years, who received standard therapy according to indications (GINA-2006). Clinical and instrumental examination included: echocardiography (EchoCg), spirometry, blood lipid spectrum, coagulation, enzyme activity (ALT and AST, total bilirubin). To assess the functional state of the respiratory muscles and diaphragm, electroneuromyography (ENMG) was performed on a Myograph Synopsis Neurotech Russian apparatus. The following modes of operation of the apparatus were used: muscle response (M-response), latency (L-lat). The following were determined: the amplitude (mV) of the M-response of the phrenic and long pectoral nerves: laterally (ms) of the nerves of Phrenicus D and S (region of the sternoclavicular - mastoid muscle) and N. Thoracicus longus D and S (Erb point). The ENMG was recorded using surface electrodes at the muscle attachment sites with calm breathing. Two stockings were applied: one on the phrenic nerve, the second on the diaphragm. Two 4x4 cm plate electrodes are placed on the lateral surfaces of the neck in the middle third of the sternocleidomastoid muscle and the procedure of electrical stimulation of the phrenic nerves is performed. The examination was carried out lying on your back or sitting in a chair. The degree of violation of the M-response of the muscles at rest was revealed and consequently, the weakness and degree of fatigue obtained after measuring the response to the stimulus of the diaphragm and other respiratory muscles. Diagnosis of the severity of fatigue of the diaphragm was carried out according to the discriminant equation  $f = 17.3 \times \text{MOS50 (l/s)}$  (Perelman Yu M., *et al.* 1998). With discriminant function less than 65.1, diaphragm fatigue was diagnosed. The study of the function of external respiration was carried out using a Shiller desktop computer spirometer, and a peak flow meter. The following FVD indicators were calculated: FVC (l); FEV 1.0L; FEV 1.0 / FVC -%; FEF 25-75%; PEF l/s; MEF 75%; MEF 50%; MEF 25%. The peak expiratory flow rate (PSV) was measured according to the generally accepted technique [8]. The coagulogram was carried out on a Huma Clot Junior apparatus (Human. Germany). The first group included patients with mild asthma (10), patients with moderate asthma amounted to group 2 (18).

**Results and Discussion**

Table 1 presents the obtained ENMG data in both BA groups by severity in a comparative aspect with the control group.

	Phrenicus Lateralis. мс		Phrenicus amplitude. мВ		N. Thoracicus longus. lateralis. мс		N. Thoracicus longus. amplitude. мВ	
	D	S	D	S	D	S	D	S
Norm	7,4 ± 2,4	0,8 ± 0,9	7,4 ± 2,3	0,8 ± 1,2	3,9 ± 3,3	0,8 ± 0,7	3,9 ± 2,6	0,8 ± 1,0
Severe BA	4,1 ± 0,76	4,9 ± 0,98	1,3 ± 0,68	0,9 ± 0,28	4,9 ± 0,37	6,5 ± 0,78	0,9 ± 0,60	0,5 ± 0,26
Moderate BA	2,5 ± 0,8	2,4 ± 0,37	0,89 ± 0,65	0,34 ± 0,02	5,4 ± 0,42	8,35 ± 0,80	0,36 ± 0,66	0,08 ± 0,54
Control	5,33 ± 1,7	1,5 ± 2,4	0,45 ± 1,9	0,59 ± 1,8	3,9 ± 2,9	0,9 ± 4,3	4,05 ± 3,9	0,95 ± 3,7

**Table 1**

We can see from table 1, that in healthy and in the control group, muscle responses from the phrenic and pectoral nerves were within normal limits. This indicates the absence of disturbances from the peripheral neuromuscular apparatus and chest muscles. In patients with AD, a tendency towards a decrease in the excitability of Phrenicus Lateralis, Phrenicus amplitude on the left and an increase in Thoracicus longus lateralis on the right with unexpressed muscular-dystrophic changes were mainly revealed.

The discriminant indicator of diaphragm fatigue in patients with BA with moderate severity averaged  $42.5 \pm 4.29$ . and  $58.8 \pm 3.0$  in patients with mild asthma. As the disease progresses, the compensatory mechanisms are depleted and respiratory muscle failure develops. The severity of hypotrophy of the diaphragm and the degree of restriction of its mobility depend on the duration of the disease, as well as the conditions of blood supply.

Studies of the cardiovascular system according to echocardiography (EchoCg) were performed on the day of the ENMG examination (Table 2).

	Aorta	Aortic valve opening	Leftatrium	FDS LV	FSSLV	FDV LV	EF	SV LV	IS
Norm	Up to 3.5 cm	1,6 - 2,2 cm	Up to 4 cm	4,5 - 5,4 cm	2,8 - 4,0 cm	70 - 147 ml	Not less than 55%	45 - 88 ml	Up to 1.1 cm
Mild BA	$3,2 \pm 0,09$	$1,8 \pm 0,07$	$3,1 \pm 0,19$	$4,9 \pm 4,01$	$3,4 \pm 0,12$	$109,6 \pm 8,09$	$55,8 \pm 1,96$	$66,0 \pm 4,0$	$1,1 \pm 0,08$
Moderate BA	$3,4 \pm 0,07$	$2,2 \pm 0,05$	$3,1 \pm 0,2$	$5,4 \pm 3,29$	$4,0 \pm 0,13$	$141,0 \pm 7,09$	$50,2 \pm 2,0$	$71,6 \pm 3,06$	$1,1 \pm 0,04$

**Table 2:** Echocardiograms of the examined patients

Note: FDS LV: Final Diastolic Size of the Left Ventricle; FSS LV: Final Systolic Size of the Left Ventricle; FDV LV: Final Diastolic Volume of the Left Ventricle; EF: Ejection Fraction; SV LV: Stroke Volume of the Left Ventricle; IS: Interventricular Septum.

According to echocardiography, 5 patients showed signs of wall sclerosis and a decrease in overall contractility in 6 patients.

In the examined BA patients, 13 showed signs of liver compaction, 6 had chronic cholecystitis, and 4 had fatty hepatosis. In this regard, we conducted biochemical studies in the examined patients, presented in table 3 and 4.

	HDLP mmol/l	LDLP mmol/l	Prothrombin time sec.	Quick test %	APTT sec.	Fibrinogen Mg/dl	Thrombin time sec.	MHO
Norm	>1,0 - 1,2	< 3,7	10 - 14	75 - 130	26 - 36	200 - 400	< 30	< 2
Group 1	$1,4 \pm 0,08$	$3,33 \pm 0,22$	$13,0 \pm 0,35$	$84,2 \pm 7,39$	$33,0 \pm 1,46$	$265,3 \pm 10,07$	$11,1 \pm 0,27$	$1,1 \pm 0,03$
Group 2	$1,38 \pm 0,06$	$3,33 \pm 0,5$	$13,4 \pm 0,31$	$88,5 \pm 4,97$	$32,9 \pm 1,87$	$265,3 \pm 9,91$	$11,8 \pm 0,45$	$1,01 \pm 0,05$

**Table 3:** The content of lipoproteins and indicators of coagulation in patients.

Note: HDLP: High Density Lipoproteins; LDLP: Low Density Lipoproteins, Activated Partial Thromboplastin Time, International Normalized Relations.

Biochemical parameters are presented in table 4.

BA	Glucose	ALT	AST	Bilirubin	Urea	Creatinine	Cholesterol	Triglycerides
Group 1	3,6 ± 2,0	16,0 ± 3,2	12,0 ± 1,8	10,7 ± 1,06	5,0 ± 0,7	75,0 ± 3,3	4,9 ± 0,51	1,59 ± 0,91
Group 2	5,3 ± 0,21	22,1 ± 4,9	13,3 ± 1,17	8,0 ± 1,04	6,1 ± 0,31	70,7 ± 4,0	5,2 ± 0,36	2,4 ± 0,50

**Table 4:** Blood biochemical parameters.

As we can see from the table 3 and 4, the average biochemical parameters of blood in patients were within normal limits.

When studying the indicators of external respiration, a sharp violation of the mixed type in 10 patients and a moderate violation of the obstructive type in 2 examined patients were revealed (Table 5).

In the study of HPF, the majority of the examined patients showed moderate restrictive disorders with a decrease in bronchial patency of the bronchi, as evidenced by FEV 1.0 which was moderately reduced ( $p < 0.05$ ). A moderate decrease was noted - MEF 50% to 38.7 ± 9.13% (Table 5).

	FVC % due	FEV1,0% due	FEV1,0/ FVC%	FEF25-75%	PEF	MEF 75%	MEF 50%	MEF 25%
Group 1 Mild	67,2 ± 2,7	63,4 ± 4,7	99,0 ± 6,0	45,5 ± 6,9	66,0 ± 1,9	42,0 ± 4,8	40,2 ± 8,3	50,3 ± 7,7
Group 2 Moderate	39,6 ± 5,96	40,5 ± 8,67	97,7 ± 4,57	25,0 ± 10,27	33,3 ± 6,73	21,6 ± 7,47	38,3 ± 9,13	39,9 ± 9,66

**Table 5**

**Example:** Patient M, 46 years old, was admitted to the pulmonology department of the RSNPMTs T and MR with a diagnosis of moderate asthma, phase of exacerbation chronic rhinitis.

The diagnosis has been established since 2005. In the anamnesis: since 1999 - chronic rhinitis and since 2007 - bronchial asthma. BA exacerbation 2 - 3 times a year for 1.5 - 2 months, provoked by colds. This exacerbation is 2 weeks.

Upon receipt of a complaint of coughing, more at night, difficult to separate viscous mucous sputum, suffocation every 8 - 10 hours, nasal congestion. On examination: a state of moderate severity, asthenic physique, the skin is pale and moist to the touch. Periodic inspiratory retraction of the abdominal muscles.

The chest is barrel-shaped. Percussion - box sound over the entire surface of the lungs. Breathing hard, scattered dry wheezing. Heart sounds are muffled. Pulse 80 beats per minute, rhythmic. BELL 130/80 mmHg. The liver and spleen are not palpable. No peripheral edema.

In an X-ray examination of the chest, the lungs are without infiltrative changes, the contours are structural. Pulmonary pattern is not changed. The diaphragm is located normally. The sinuses are free. The heart across is not expanded. On the ECG - sinus rhythm, 72 beats/minutes. The vertical direction of the electric axis.

Complete blood count: erythrocytes - 4.1 T/l, Hb 134.0 g/l, white blood cells - 8.7 G/l, segmented - 53%, monocytes - 6%, lymphocytes - 27%, eosinophils - 6%, ESR - 6 mm/h. Sputum analysis: viscous, whitish, microscopically - eosinophils ++, alveolar cells. Exami-

nation of the function of external respiration: BH - 18 per minute FVC - 41%, FEV1.0 - 39%, FEV1.0/FVC - 89%, FEF 25-75-26%, PEF-30, MEF 75-46%, MEF 50 - 49%, MEF 25 - 30%. Diaphragm ENMG parameters: M-response with Phrenicus Lateralis- 0.89 mV, M-response with Phrenicus amplitude- 0.34 mV, M response with Thoracicus longus Lateralis- 8.35 ms. Thoracicus longus amplitude- 0.45 mV.

Discriminate indicator of fatigue of the diaphragm = 42.5. When evaluating ENMG data, a decrease in the M-response from the diaphragm, a moderate increase in latency in the long pectoral nerves, as well as a decrease in the discriminant indicator indicate the patient has fatigue of the respiratory muscles.

## Conclusion

Thus, in patients with asthma we find: a moderate decrease in the functional activity of respiratory muscles. The research results will allow us to evaluate the prognostic value as predictors of an unfavorable course of diseases and can serve as a basis for developing indications for a differentiated complex use of treatment methods, including electromyostimulation in case of respiratory muscle dysfunction, which contribute to the removal of symptoms of respiratory muscle fatigue syndrome, which will improve respiratory functions and increase the quality of life of patients and expand rehabilitation programs in patients with AD.

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