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# Opinion

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# The Pathogenesis Of Pain Syndrome In Acute Pneumonia And The Purpose of Its Elimination

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### Introduction

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Acute pneumonia [AP] is the common name of this nosology, which currently has a wide variety of designations. However, regardless of the terminology reflecting the modern tendency to emphasize the features of etiology, the main unifying feature of this disease was and remains the inflammatory process of lung tissue. The development of an acute inflammatory process in the tissues of the body is constantly accompanied by five characteristic signs, which were described by Celsus and Galen at the beginning of the first millennium AD and over the past period have received their confirmation and universal recognition.

The occurrence of classical signs of inflammation is an inevitable accompaniment of inflammatory transformation of body tissues and manifests itself with the onset of this process, regardless of its localization. Pain is a constant sign of inflammation, however, biological phenomena may have exceptions to the general rules, understanding the essence of which makes it possible to understand the features of the pathogenesis of this disease. The latter circumstance is important for the specific correction of the complex of medical care for such patients. One of the most striking examples of such exceptions among inflammatory processes is AP, the development of which, as a rule, proceeds without pain syndrome. As is known, the lung tissue is devoid of pain receptors and in the case of this disease, pain occurs only when the inflammatory process reaches the pleural leaf [1]. Such pain appears during a certain period of the disease and has its own distinctive feature to intensify during respiratory excursions.

Such a classic sign of inflammation as pain is predominantly a signaling one. Convincing evidence that it is pain, and not any other factors, that causes autonomous adaptive reactions of the body has

not yet been presented. The pain signal primarily forces the patient to spare the affected area and observe protective measures in relation to the affected area. From these positions, it is impossible to imagine the positive value of pain in the initial period of AP, even theoretically. If each development of a focus of inflammation in the lung tissue was immediately accompanied by a pain syndrome, then in addition to the negative effect on respiratory function, such a factor cannot bring real benefits to the body. Since in this context we are not talking about peripheral localizations of inflammation, the initial period of development of which does not significantly affect the general condition of the body, but about the defeat of one of its vital systems, nature initially took care that in the case of AP the patient's body was automatically protected from the sudden occurrence of conditions incompatible with life. Therefore, instead of pain in the initial period of AP, another, more important and necessary reaction appears at this moment.

Tissue changes in the focus of inflammation cause irritation of receptors in this area. Lung tissue, devoid of pain receptors, is at the same time one of the sensitive reflexogenic zones, which is equipped, in particular, with baroreceptors, which occupy an important place in the regulation of general blood circulation. It is these receptors that respond to the inevitable increase in blood pressure in the pulmonary vessels, causing the so-called unloading reflex, which was first described almost a century ago [2]. The main essence of this autonomous adaptive reaction, which automatically begins to act when a relative excess of venous return and the first signs of an imbalance between the two circulatory circles appear, is blood retention at the periphery. The last factor of blood circulation restructuring has a direct effect on peripheral microcirculation, the manifestation of signs of which completely depends on the individual rate of development of the process and current changes. With the rapid development of inflammatory edema and infiltration of lung tissue, the body does not have time to smoothly make the necessary adaptation, and the launched mechanism of unloading the vessels of the small circle loses its original clarity and coherence. In such cases, the mechanism of initial changes in blood flow is complemented by generalized reflex spasms of small pulmonary vessels and a tendency to decrease systemic blood pressure [3-5]. The clinical picture in such observations fully meets the criteria of pulmonogenic shock [3], which currently continues to be unreasonably considered as septic [6].

If by the time of the development of the situation described above, the inflammatory process in the lung reaches the outer layers of the organ parenchyma, leading to inflammatory changes in the pleural membrane, then the patient's severe condition is complemented by the appearance of pain syndrome. The appearance of pain makes it difficult for the patient to breathe, as it increases during respiratory excursions. This reduces the massaging effect of breathing on the pulmonary vessels, reinforcing the already existing shifts in blood circulation. Increased pain during breathing is caused by friction of inflamed areas of the pleura, and the spontaneous disappearance of pain or their significant decrease indicates the appearance of pleural effusion, which only excludes their contact and is not a sign of positive dynamics.

The described syndrome in AP is manifested by pain in the side or chest on the side of the lesion and often creates an additional problem in the differential diagnosis of other possible causes of its occurrence. In most cases, this manifestation of the disease, especially in elderly patients, raises suspicions of heart problems. More often in children and less often in adults, pain syndrome with AP can simulate a catastrophe in the abdominal cavity. The use of painkillers in patients with pain with AP does not bring them complete relief, since this is symptomatic, not pathogenetic help. In addition, if the question of the intra-abdominal cause of pain remains open, then analgesic therapy with medications is contraindicated due to the risk of even greater difficulties in making a correct diagnosis.

In previous years, in the Soviet Union, the Ministry of Health issued an order prescribing cervical novocaine vagosympathetic blockade [CVSB] for the purpose of differential diagnosis of the cause of abdominal syndrome in AP. This type of blockade was developed and proposed by the famous surgeon A.V. Vishnevsky and was successfully used during World War II in the Soviet Army as one of the methods of first aid to the wounded in pleuropulmonary shock [7]. In peacetime, this technique gradually began to lose its position in emergency medicine, despite the simplicity of its implementation compared to other types of blockades. Probably, this circumstance was related to the previous experience of its use, which was obtained primarily in traumatic chest injuries. Outside of the now-defunct state [USSR], this type of blockade remained virtually unknown.

The experience of performing CVSB has shown that it not only has an anti-shock effect, improving the well-being and condition of the wounded, but also eliminates pain syndrome. At the same time, if the cause of the pain syndrome was in the abdominal cavity, then the implementation of this blockade did not bring an analgesic effect. The latter circumstance led to the use of CVSB as a differential diagnostic procedure to clarify the diagnosis between abdominal syndrome and acute intra-abdominal pain. For example, in childhood, the most common situations are when a patient with AP requires urgent exclusion of acute appendicitis. The mechanism of action of this blockade requires a more detailed study, but initially the meaning of its application was based on the interruption of nerve impulses of autonomic innervation at the level of the cervical mediastinum. Below is a brief description of the CVSB execution technique.

The patient lies on his back with a small roller under the shoulder blades. The head leans back and turns in the direction opposite to the blockade. The patient's arm is slightly lowered along the body from the side of the procedure. The doctor places the index finger of the left hand just above the intersection point of the external jugular vein with the posterior edge of the sternocleidomastoid muscle. By pressing on this point, the doctor shifts the muscles and blood vessels medially and probes the anterior surface of the cervical vertebrae. The needle is inserted near the tip of the finger slightly upwards at an angle of about 15-30 degrees to the surface of the skin and is directed to the anterior surface of the cervical vertebrae. When the tip of the needle rests against the anterior surface of the vertebra, the needle is slightly pulled out, a trial aspiration is performed to make sure that there is no puncture of the vessel, and then an anesthetic solution is injected. The dose of 0.25% novocaine solution in children ranges from 5 to 15 ml, depending on age, in adult patients-up to 30-60 ml. The anesthetic solution blocks the vagus and sympathetic nerves. Instead of novocaine, you can use any anesthetic that is used to perform other types of blockades. Gorner's syndrome [ptosis, myosis, enophthalmos], which usually appears within 2-5 minutes, is a confirmation of the correct execution of the blockade and the beginning of its action.

Subjective impressions about the effectiveness of CVSB in patients with abdominal syndrome of AP were confirmed by us using objective testing, the results of which exceeded expectations. By recording comparative rheopulmonograms, evidence was obtained of an improvement in cardiac and respiratory functions with normalization of the ratio of ventilation and perfusion [3]. Very indicative in such observations was the fact that these changes occurred literally a few minutes after a properly performed blockade and required only further continuation of pathogenetic support. Such results are especially indicative compared to the principles of intensive use of various drugs and intravenous infusions, which currently continue to be used in wide practice without much success [6]. In addition to the main antishock effect, this technique allows to eliminate the pain pseudoabdominal syndrome of AP without requiring additional diagnostic methods.

Thus, a brief analysis of information about the features of the pathogenesis of AP, with special emphasis on the results of objective studies, allows us to note that pain syndrome in the inflammatory process in the lung can occur already in the initial period of the disease. This variant of AP development is typical for the so-called croup forms of the disease according to the old classifications, when the process quickly reaches the parietal pleura. Pain syndrome in this category of patients quite naturally attracts the main attention of attending physicians, as it requires urgent differential diagnosis with acute extrapulmonary causes. Using CVSB for this purpose not only speeds up the clarification of the correct diagnosis, but also brings much greater benefits as pathogenetically sound first aid to such patients.

Localization of the inflammatory process in AP is the cause of the unique pathogenesis of the disease, when a special pulmonogenic form of shock with rapid development occurs. The mechanisms of development of this shock reaction are of pathogenetic rather than infectious origin, and the principles of their emergency elimination are diametrically opposed to generally accepted approaches to the treatment of septic conditions [6]. The latter circumstance is convincingly confirmed when comparing the results of various methods of treatment of AP [3]. For pain syndrome, CVSB is the method of choice among the first aid methods for this category of patients. In such cases, the implementation of this type of blockade allows for the simultaneous differential diagnosis of the cause of pain and anti-shock assistance to the patient.

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#### **Conflict of Interest**

No conflict of interest.

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