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## Letter to the Editor

# Modern View of Acute Pneumonia and Forgotten Achievements of Medical Science

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## Letter to the Editor

Acute pneumonia is known to medicine for more than two and a half millennium since the time of Hippocrates. All known old classic postulate that pneumonia is not contagious infection. And today, patients with acute pneumonia are not subjected to isolation and other precautions. Moreover, it is well known that the etiology of pneumonia is quite diverse. The causative agents of pneumonia are very numerous, are not a constant feature of the disease and, as a rule, belong to the representatives of symbiotic nonspecific micro flora.

However, despite such a long history of fame, modern ideas about the nature of acute pneumonia look very primitive and paradoxical, especially against the background of outstanding achievements of medical science in many areas related to pulmonology. The concept of causes and mechanisms of development of any disease determines both the nature of medical care for these patients and the final results of treatment. Acute pneumonia is no exception to this rule. Modern ideas about the nature of acute pneumonia dictate the direction and specificity of the necessary assistance to these patients. And the paradox of modern ideas about the nature of pneumonia is as follows.

Today, all efforts in the treatment of this group of patients are aimed primarily at the suppression of the microbial factor. Considering the current recommendations for the treatment of acute pneumonia, we can assume that we are talking about dangerous and severe mono infection.

Another important argument for this assessment is vaccination against pneumonia. This pneumococcal vaccination has a specific focus and does not cover many other etiological options. Vaccination has forced to forget about many hazardous infections and is guaranteed protection from being infected by them. However, a mass campaign of vaccination against pneumonia did not lead to the desired revolutionary results. Moreover, contrary to expectations, against the background of pneumococcal vaccination of the population of developed countries, a statistically significant increase in purulent complications of acute pneumonia was registered [1-3]. This unexpected result of vaccination does not fit into the modern understanding of the nature of acute pneumonia as an infectious process with a clear predominance of one of the pathogens. Therefore, experts and scientific analysts were not ready for a reasoned explanation of this unpleasant "surprise".

Currently, the initial treatment of acute pneumonia is defined as "antibiotics alone". The use of the same antibiotics in inflammatory diseases of different localization and pathogenesis is generally accepted. Thus, treatment (especially initial) of completely different diseases is equivalent. It should be noted that today in developed countries, the majority of patients with acute pneumonia are still treated on an outpatient basis with the help of "antibiotics alone". However, the etiology of pneumonia in such cases remains unknown.

The problem begins to manifest itself in those observations where the initial treatment failed and the patient needs hospitalization. As a rule, such situation is explained by the presence of super-aggressive micro flora without presenting convincing evidence. At the same time, all additional treatments for patients with acute pneumonia, which begin to be used in the case of hospitalization, are not strictly specific. The use of such methods is based only on subjective assumptions about their beneficial effect on patients with acute pneumonia, but their actual impact on the dynamics of the inflammatory process in the lungs, has not passed due objective testing.

For example: why patients with acute pneumonia in the early hours of the disease should receive intravenous fluids? What catastrophic fluid loss can occur during this period of illness? What really lies behind the so-called term "intoxication"? But we know that the first barrier to which the injectable intravenous solutions reach is the area of inflammation in the lung, don't we? In other words, this procedure increases blood flow to the area of progressive inflammatory edema and infiltration of tissues.

Another example: Why a small focus of acute inflammation in the lung can cause severe violations of gas exchange and we must give for patients the insufflation of oxygen? Why atelectasis of the lobe and even the entire lung usually does not give such heavy changes of respiration? These known differences suggest that the volume of functioning pulmonary parenchyma is not the main cause of gas exchange shifts and there is another unaccounted mechanism of these disorders.

Such issues require, first of all, a review of views on the nature and mechanisms of acute pneumonia. In this regard, it is necessary to recall the following well-known axioms and facts of medical science.

1. Acute pneumonia is not contagious specific disease and its etiology is represented by non-specific bacteria that are usually found among the symbionts of healthy people. Patients with acute pneumonia do not require isolation or other epidemiological measures.
2. The body's response to any stimulus, including the initiation of inflammation, is highly individual and unique.
3. The basis for the inflammatory transformation of the body tissue is a vascular reaction with a specific stage sequence.
4. Small and large circles of blood circulation have not only a direct anatomical connection, but also an inverse functional interdependence.
5. The vessels of the lesser circulation are highly sensitive reflexogenic zone.
6. Among the nonspecific forms of inflammation, acute pneumonia is the only process occurring in the system of lesser circulation.
7. Any acute inflammation is accompanied by five classical signs, which were described several centuries ago by Celsus and Galen (heat, pain, redness, swelling, and loss of function). Depending on the localization of the process, the fifth sign (loss of function) is the most important as it determines the clinical manifestation and severity of the disease.

The information mentioned above is well known to everyone since the time of the University bench. Taking this information into account is important not only to understand the mechanisms of the disease, but also to explain many manifestations of acute pneumonia, which still cause difficulties in their interpretation. At the same time, it remains a mystery that such important materials remain unclaimed in justifying and determining the complex of care for patients with acute pneumonia.

The above-mentioned classical facts of medical science formed the basis of the new doctrine of acute pneumonia. Additional studies were conducted to clarify certain elements of the pathogenesis of the disease. All additional analyses were based on representative material and subjected to statistical processing. This work has been done and tested in a clinical setting in the years 1976-1984 in Novokuznetsk State Institute for postgraduate doctors (USSR, Russia). Following private studies were additionally performed.

1. Experimental model of AP (4 series of experiments, 44 animals) obtaining a model of pleural complications (certificate for invention No 1631574, A1, 1 November 1990, USSR).

2. X-ray examination 56 lung anatomical preparations with different forms of the AP, taken from the dead patients.

3. Record comparative rheopulmonography before and after performing medical procedures (36 patients).

4. Analysis of the observation and treatment of 994 children with AP and its various destructive and pleural complications.

The main result of this work was the creation of a new doctrine of acute pneumonia and on its basis the revision of the principles of medical care for these patients. The revised recommendations for treatment were applied in 101 patients in the initial period of aggressive forms of AP, as well as in 102 patients who at the time of hospitalization already had effusion in the pleural cavity. The received results allow speaking about possibility of the guaranteed prevention of suppurative and destructive complications of the disease.

The summary of the work and its parts were published only in Russian [4]. Unfortunately, the conclusions and recommendations of the research have not received proper dissemination and application among Russian-speaking professionals. The past years have shown that the problem of prevention of purulent and destructive complications of acute pneumonia has not only remained unresolved, but also aggravated by the appearance of additional causes.

Against the background of a gradual decrease in the effectiveness of antibiotics and the constant replenishment of the group of antibiotic-resistant strains of microorganisms, the treatment strategy of OP has not undergone any radical changes. Over the past two decades, there has been an increase in the number of purulent complications of OP. The expected triumph of vaccination against pneumonia has not taken place.

However, despite the changes and dynamics among the causative agents of acute pneumonia, the nature and mechanisms of inflammation in the lung remain dependent on the General biological laws. This rule is an integral characteristic of each nosological form. Therefore, successfully tested strategic approaches to the treatment of acute pneumonia should be of interest to specialists in various fields that come into contact with this problem. To this end, the results of previous studies and clinical trials were translated into English and published last year as a separate book [5].

The details of the studies that are described in this book, combined with the classical principles of biology give an idea of the unique mechanisms of development of acute pneumonia and existing methods of influence on them in the direction of stimulation and inhibition. The submissions supported by objective testing, statistical processing, and results of clinical approbation of the Contents of the book gives a realistic idea about the possibility of guaranteed prevention of purulent-destructive complications of the disease and is the basis for further work in this direction.

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