

The Specifics of Intensive Therapy for Acute Nonspecific Inflammation of The Lung Tissue

Igor Klepikov*

MD, Professor, Renton, WA, USA

Submission: September 27, 2023; **Published:** October 04, 2023

***Corresponding author:** Igor Klepikov, MD, Professor, Renton, WA, USA

Keywords: Acute pneumonia; Inflammation; Lung tissue; Antibiotics; Microorganisms

Abbreviations: ANSIL: Acute Nonspecific Inflammation in the Lungs; AP: Acute Pneumonia

Opinion

Acute nonspecific inflammation in the lungs (ANSIL), known as acute pneumonia (AP), is one of the oldest nosologies known to medicine throughout its foreseeable history. At present, when medical science and practice have achieved unprecedented success in many areas and directions, the logic of such a reality will allow us to assume that such a long-known disease as AP should not pose a problem for providing effective care to this category of patients. However, the real situation in this section of medicine refutes the logic of assumptions and is a reflection of one of the main paradoxes of modern medicine. AP has now become one of the leading problems even in the conditions of the most developed health systems, demonstrating a steady trend towards a gradual increase in the number of complicated forms of disease and mortality rates [1-3].

To understand the reasons for the inconsistencies between the use of, on the one hand, increasingly improving methods of AP treatment, but, on the other hand, their gradually and fatally decreasing effectiveness, it is necessary to look at a number of events that have been observed in this section of medicine over the past decades, but at the same time it is fundamentally important to analyze the origins of the formation of modern dominant ideas about the essence of ANSIL from the standpoint of the classical foundations of medical science. It is a critical look and a radical revision of those ideas and dogmas that today determine the strategy for solving the ANSIL problem that will allow us to understand the origins of existing misconceptions and begin to eliminate them.

The main feature of AP, reflecting the essence of this disease, is the inflammatory process of the lung tissue. The general

essence of the manifestations of inflammatory processes was brilliantly presented in the description of their classical signs, the interpretation of which was first proposed by Celsus and Galen (heat, pain, redness, swelling, and loss of function) and received further confirmation and recognition. However, despite the commonality of the main signs of an acute inflammatory process, they acquire specific nuances depending on the localization of the lesion, even with the participation of one type of pathogen. That's why, for example, pneumococcal pneumonia will be radically different from pneumococcal meningitis or pneumococcal otitis media, right? Similar examples of the participation of similar pathogens in the development of inflammatory processes of various localization serve as a convincing example that the etiology of ANSIL does not have a decisive influence on the features of the manifestations of such diseases, and the main importance in the formation of the specifics of the disease continues to have a classic sign of inflammation, known as loss of function of the affected organ.

Despite the logic of classical interpretations of the manifestation of inflammatory processes, which has been verified and clarified by centuries of experience in observing various categories of patients with inflammatory processes, the currently dominant system of views on the essence of the problem of AP considers non-specific pathogens of the process as its leading and determining factors. This point of view was the result of many years of formation of professional ideas under the influence of an excessively hypertrophied perception of antibacterial therapy as an exceptional means to help cope with this disease.

Inconsistencies between the main therapeutic use of antibiotics and the hypertrophied role that they began to play in the treatment

of this category of patients were observed throughout their wide practical application. The ability of these drugs to selectively affect certain types of microorganisms was known even before their introduction into medical practice, as well as the absence of their direct effect on the mechanisms of the inflammatory process. However, the first results of using this therapy in practice created the illusion of getting a “pill for pneumonia”. The captivating simplicity of providing such assistance in the early years of the era of antibiotics formed an entire industry for the preservation and maintenance of this therapy, the effectiveness of which began to decline relatively quickly. The concentration of efforts of pharmacists and practitioners in this direction allowed for a long period to limit the treatment of many patients with AP with the formula “antibiotics alone”, giving preference to etiotropic agents and leaving aside pathogenetic methods of treatment.

The powerful psychological influence of antimicrobials on professional ideas about the essence of ANSIL manifestations is reflected in a number of distortions that are currently presented as undoubted characteristics of these diseases when discussing the search for effective means to combat them. For example, the lack of evidence of transmission of the AP disease itself from a patient to a healthy person does not prevent classifying these processes as infectious with the definition of anti-epidemic measures as an important therapeutic section. Or, for example, how to consider the assessment by modern medicine of the emergence and transformation into a serious health problem of such consequences of the use of antibiotics as strains of microorganisms resistant to them? One of the discoverers of penicillin, A Fleming [4], warned about the danger of this side effect of a new type of therapy even before its widespread introduction into practice, and EP Abraham and E Chain [5], already in the process of their work on isolating this drug for industrial release, provided evidence of the rapid development of microorganisms resistant to it. Such preliminary warnings throughout the entire period of antibiotic use did not lead to the timely development and implementation of special antimicrobial treatment programs that could reduce the severity of such consequences.

The relatively short history of the use of antibiotics is characterized by various attempts at early recognition of AP pathogens and the desire to apply the most optimal selection of drugs to achieve maximum sterilizing effect. This period is characterized by the appearance of new, more active drugs, which made it possible to achieve short-term success, after which the effectiveness of treatment began to fall again. However, in general, the combination of research and innovation represented a long marathon of competition between pharmacology and the microflora surrounding us. During this long-term rivalry, not only the number of variants of antibiotic-resistant strains grew, when many of them became common finds among symbionts of healthy people, but also the potential role of viruses in the etiology of these processes began to grow.

How can we assess today the fact that from the moment of the first information about the inevitable formation of resistant forms of microorganisms as a result of the use of antibiotics to the announcement of these consequences as one of the global health problems only in 2021 [6], when this problem has already reached a critical point, so much time has passed during which the race to destroy pathogens was conducted nonspecific inflammation, but no rational programs have been undertaken to reduce these consequences? Of course, such a belated recognition of this catastrophe should at least cause confusion about the long-term indifference to these side effects. However, this event takes an unexpected turn in the light of plans to reduce the burden of these side effects, which are proposed by WHO experts declaring resistant microflora a global health problem [6]. The prospect of such proposals for the future really impresses with its principles, which are based on the continuation of the development of even more advanced (!?) antimicrobial drugs. In other words, we are talking about the continuation of the competition that led to this problem. In this case, as they say, comments are unnecessary, since the essence of such proposals is a reflection of the depth of the negative didactic influence of antibiotics on the nature of professional representations.

The modern understanding of the leading causes of the occurrence and development of AP is directly related to the formation of the principles of intensive treatment of this group of patients. Conceptual ideas about this category of diseases today determine the direction of the search in solving this whole problem. If microorganisms, as representatives of the nature around us, did not show their biological qualities and did not have the ability to adapt to external influences that pose a danger to their existence, then there would be no obstacles to the use of antibiotics. And today, effective treatment of inflammatory processes based on the principle of “only antibiotics” would continue. However, nature throughout the entire period of this type of therapy sends us absolutely unambiguous signals that the imbalance of the habitually existing proportions in the microcosm around us requires, at a minimum, the formation of new ratios.

The gradual decrease in the antimicrobial effect, which was observed during the entire period of antibiotic use, required not only the development and release of new drugs, but also the use of additional means of assistance. In the latter case, the choice of auxiliary techniques was determined by the leading role of the causative agent of the process, which was assigned to him under the impression of the initial effects of antimicrobial therapy. The false nature of such approaches to the choice of intensive care methods was constantly emphasized by the futility of attempts to obtain differential diagnostic criteria for various forms of AP, depending on their etiology. However, as recent events show, previous failures to identify such differences have not affected the overall trend of research conducted today. If in previous years attempts were made to separate this disease by microbial

factor, at the moment it has proved impossible to do so even with differential diagnosis between bacterial forms of community acquired pneumonia and COVID-19 pneumonia [7-9].

If we do not take into account the specifics of auxiliary ventilation, then the main set of methods of intensive treatment of patients with AP is based today on the same principles of choice and methods of implementation as emergency methods for inflammatory processes of all other localizations. Dissonance in carrying out equivalent methods of therapeutic effects on fundamentally different mechanisms of inflammatory processes within the pulmonary blood flow and in the peripheral circulation area has existed for many years, but there are no signs of eliminating these inconsistencies yet.

It is difficult to imagine that a modern graduate of a medical university would not be guided by the fundamental differences in blood flow in the small circle of blood circulation from the systemic circulation, which allow maintaining a balance between two completely incomparable halves of the circulatory system in terms of blood flow parameters. At the same time, the automatic preservation of the vital ratio between the two circulatory circles is provided by the regulating mechanisms of the small circle. This information, combined with the unavoidable fact that inflammatory transformation in lung tissue always begins with a vascular reaction and invariably involves mechanisms of regulation of general blood flow in the process, serves as the basis for considering the pathogenesis of AP as a unique form of manifestation of inflammatory diseases.

Scientifically based information that allows us to understand the principle of functional unity of the entire circulatory system in the presence of cardinal differences between its two halves remains unclaimed in everyday practice. Contrary to the existing differences between the indicators of blood pressure in each circulatory circle [10,11], at present the severity of disorders of the general blood flow in patients with AP is usually determined, as with all other diseases, by indicators of systemic circulation [12-14], deviations of which in the case of the development of primary focus in the zone of the small circle are initially adaptive. And again, it is surprising that no one even expresses doubts about the distortion of this approach to diagnosis. The continuation of this strategic line is the widespread use of intravenous infusions in patients with AP, who, first of all, reach the zone of increasing edema in the lung tissue, overloading its vessels with additional venous return [15].

This appeal is not intended to describe a consistent picture of the pathogenesis of AP, which has already been presented by the author of these lines in numerous publications both in the form of a description of individual fragments and in the form of a general variant of development. We are also not talking about comparing the disadvantages and advantages of individual methods of emergency care for patients with AP, which were used by the

author in the course of work using the registration of objective criteria and allowed to prove during clinical trials the need for a radical revision of the doctrine of the disease. Such materials are freely available, including the summary results of this work [16]. Therefore, the repeated presentation of such information is unlikely to have a more tangible impact on changing the general trend of inconsistency between the dominant ideas about the concept of the origin and development of this disease in relation to the fundamental provisions of medical science. A sufficiently long period of time that has passed since the completion of this work has shown that this dissonance, due to the hypertrophied perception of the causative agent of the process as its main cause and excessive hopes for the success of etiotropic drugs, not only persists, but also continues to deepen.

For example, the very first summing up of the campaign launched for largescale pneumococcal vaccination of the population of developed countries against pneumonia should have given rise to a critical assessment of the principles of such an approach to solving the problem [17,18]. In this case, we are talking not only about the implementation of the main goal of such a comprehensive program, which is preventive, not curative. Moreover, in the presence of various variants of pathogens of nonspecific forms of inflammation of the lung tissue, only one of these representatives was selected. The results showed that a slight decrease in the incidence of AP was accompanied by a parallel significant increase in the number of complicated forms of the disease. The latter circumstance indicated that in those situations when the disease really took place and had an aggressive nature of its development, the necessary intensive therapy was ineffective.

There was also a continued increase in complicated forms of AP due to an increase in the number of septic conditions, including septic shock, for the diagnosis of which in recent years evidence of the presence of pathogens in the general bloodstream was no longer required [12,13]. The "simplified" process of diagnosing septic complications in nonspecific inflammation began to be carried out according to the same scheme, regardless of the localization of the primary focus in different circulatory circles, which brought AP into the category of the main causes of septic conditions, in the group of which negative bacteriological confirmations were strikingly predominant [19].

The growth of viral forms of lung inflammation did not affect the system of views on the causes of the severity of the condition of these patients, which automatically began to be explained by the development of viral variants of sepsis and shock [20-22]. At the same time, no objective criteria have been presented to confirm the viral nature of such complications, except for an analogy with the previously existing concept of bacterial forms of the disease, and the use of ineffective intensive support measures continues with the same bleak statistics of results.

In this regard, it should be noted that, despite the general trend of research and efforts to neutralize the alleged leading role of the pathogen in the development of inflammatory processes of the lungs, recently it is possible to get acquainted with new evidence of the important role of primary blood flow disorders in the pulmonary vessels in the pathogenesis of these diseases. For example, computed tomography examination of blood flow disorders in the small circle of circulation in patients with COVID-19 pneumonia reveals generalized spasm of small vessels of the lungs, which may explain the cause of subsequent disorders of the general circulation [23,24]. However, the authors confine themselves only to stating the detected abnormalities of blood flow, also suggesting as one of the causes of such disorder's widespread thrombosis of such vessels, incompatible with the preservation of vital functions.

Thus, even a brief analysis of the situation that has developed in solving the ANSIL problem clearly indicates the need for a radical revision of views on the doctrine of these diseases, which is becoming more and more obvious every year. If we take into account that such a revision concerns not only changes in learned and habitual stereotypes, but also requires a lot of work to change curricula and regulatory documents, then the complexity of such a step is beyond doubt. However, on the other hand, there is no doubt that the inevitability of such a radical correction has existed for many years, and without this stage of work we must abandon hopes of achieving long-term success in the treatment of this pathology. Only the transformation of the AP doctrine in accordance with the inevitable violation's characteristic of this localization of inflammatory processes will make it possible to understand the principles of intensive measures in this category of patients and ultimately move the solution of this urgent problem from the dead point.

References

1. Montull B, Menendez R, Torres A, Reyes S, Mendez R, et al. (2016) Predictors of severe sepsis among patients hospitalized for community-acquired pneumonia. *PLoS One* 11: e0145929.
2. Cavallazzi R, Furmanek S, Arnold FW, Leslie AB, Richard GW, et al. (2020) The burden of community-acquired pneumonia requiring admission to ICU in the United States. *Chest* 158(3): 1008-1016.
3. Cilloniz C, Torres A, Niederman MS (2021) Management of pneumonia in critically ill patients. *BMJ* 375: e065871.
4. Fleming A (1945) "The Nobel Prize in Physiology or Medicine 1945 - Penicillin: Nobel Lecture".
5. Abraham EP, Chain E (1940) "An enzyme from bacteria able to destroy penicillin". *Nature* 146(3713): 837.
6. (2021) Antimicrobial resistance. WHO.
7. C Heneghan, A Plueddemann, KR Mahtani (2020) Differentiating viral from bacterial pneumonia. The Centre for Evidence-Based Medicine. Evidence Service to support the COVID-19 response.
8. Kamat IS, Ramachandran V, Eswaran H, Guffey D, Musher DM (2020) Procalcitonin to Distinguish Viral from Bacterial Pneumonia: A Systematic Review and Meta-analysis. *Clin Infect Dis* 70(3): 538-542.
9. Kim D, Quinn J, Pinsky B, Nigam HS, Ian B (2020) Rates of co-infection between SARS-CoV-2 and other respiratory pathogens. *JAMA* 323: 2085-2086.
10. Olivia Vynn (2001) *Cardiology secrets*. Chapter 41, p. 210. Adair Edition: 2, illustrated Published by Elsevier Health Sciences.
11. "Normal Hemodynamic Parameters - Adult". Edwards Lifesciences LLC.
12. Singer M, Deutschman CS, Seymour CW, M Shankar-Hari, Djillali A, et al. (2016) The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). *JAMA* 315(8): 801-810.
13. Ilg, A, Moskowitz, A, Konanki, V, Parth VP, Maureen C, et al. (2019) Performance of the CURB-65 score in predicting critical care interventions in patients admitted with community-acquired pneumonia. *Ann Emerg Med* 74(1): 60-68.
14. Jungian C, Bing L, Houwei D, Hailong L, Cunrong C, et al. (2021) Performance of CURB-65, PSI, and APACHE-II for predicting COVID-19 pneumonia severity and mortality. *European Journal of Inflammation* 19.
15. Klepikov I (2017) "The Effect of Intravenous Infusion on the Dynamics of Acute Pneumonia". *EC Pulmonology and Respiratory Medicine* 4(1): 15-20.
16. I Klepikov (2022) *The Didactics of Acute Lung Inflammation*. Cambridge Scholars Publishing Pp: 320.
17. ST Li, DJ Tancredi (2010) Empyema Hospitalizations Increased in US Children Despite Pneumococcal Conjugate Vaccine. *Pediatrics* 125(1): 26-33.
18. RE Strachan, TL Snelling, A Jaffe (2013) Increased paediatric hospitalizations for empyema in Australia after introduction of the 7-valent pneumococcal conjugate vaccine. *Bulletin of the World Health Organization* 91(3): 167-173.
19. A Ceccato, A Torres (2018) Sepsis and community-acquired pneumonia. *Ann Res Hosp* 2: 7.
20. Lin GL, McGinley JP, Drysdale SB, Andrew JP (2018) Epidemiology and immune pathogenesis of viral sepsis. *Front Immunol* 9: 2147.
21. Weiss SL, Peters MJ, Alhassani W, Michael SDA, Heidi RF, et al. (2020) Surviving sepsis campaign international guidelines for the management of septic shock and sepsis-associated organ dysfunction in children. *Pediatr Crit Care Med* 21(2): e52-e106.
22. LJ Schlapbach, N Kissoon, A Alhassani, Maha HA, Ron D, et al. (2020) World Sepsis Day: a global agenda to target a leading cause of morbidity and mortality. *Am J Physiol Lung Cell Mol Physiol* 319(3): L518-L522.
23. Thillai M, Patvardhan C, Swietlik EM, McLellan T, Jan DB, et al. (2021) Functional respiratory imaging identifies redistribution of pulmonary blood flow in patients with COVID-19. *Thorax* 76(2): 182-184.
24. Dierckx W, De Backer W, Lins M, De Meyer Y, Ides K, et al. (2022) CT derived measurements of pulmonary blood volume in small vessels and the Page 9 of 10 For Review Only need for supplemental oxygen in COVID-19 patients. *J Appl Physiol* 133(6): 1295-1299.



This work is licensed under Creative Commons Attribution 4.0 License
DOI: [10.19080/IJOPRS.2023.06.555697](https://doi.org/10.19080/IJOPRS.2023.06.555697)

**Your next submission with Juniper Publishers
will reach you the below assets**

- Quality Editorial service
- Swift Peer Review
- Reprints availability
- E-prints Service
- Manuscript Podcast for convenient understanding
- Global attainment for your research
- Manuscript accessibility in different formats
(Pdf, E-pub, Full Text, Audio)
- Unceasing customer service

Track the below URL for one-step submission
<https://juniperpublishers.com/online-submission.php>