



Why is First Aid Ineffective for Acute Pneumonia?

Igor Klepikov*

Professor, Retired, Renton, WA, USA

*Corresponding Author: Igor Klepikov, Professor, Retired, Renton, WA, USA.

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Abstract

First and emergency aid in acute diseases is the driving force of further treatment, determining its success. According to established traditions, in which the main role in the treatment of acute pneumonia is given to antibiotics, the latter are considered a universal remedy, which is also a method of first aid. Distortions of views on the nature of the disease and established stereotypes of providing assistance require waiting for the results of such treatment for 48-72 hours, which is unacceptable in conditions of rapidly progressing inflammatory changes. Additional means of helping such patients do not have a pathogenetic basis and are not able to change the situation. An analysis of the causes and ways out of this impasse is presented.

Keywords: Acute Pneumonia; Antibiotics; First Aid; Infusion Therapy; Etiology; Pathogenesis

First aid (FA) for any acute disease is extremely important, and the fate of the patient largely depends on its compliance with the nature of the observed pathology and its effectiveness. At the same time, the main importance during the first contacts with the patient is given to methods that allow for the rapid elimination of the most acute manifestations of the established disease and the prevention of further spread of the process. Analysis of modern results of first aid to patients with acute pneumonia (AP) from such fundamentally specific positions allows one to seriously doubt its effectiveness, one only has to look at the results and statistics of this disease.

For example, AP, as one of the oldest nosologies known to medicine, still remains in the group of diseases that are considered global problems [1]. The number of patients with OP who need hospitalization in intensive care units (ICU) has been characterized in recent years by a gradual and stable increase [2], an increase in the number of complicated forms of disease and mortality [3,4].

Only 58% of patients treated with this disease in the intensive care unit are referred there immediately after diagnosis due to the more threatening onset of the process [5]. At the same time, 25% of those who started treatment in general departments are sent to the intensive care unit in the first 2 days due to the ineffectiveness of the treatment started and the deterioration of their condition [5,6].

However, this is only a small part of the problem under discussion. AP, especially in the last couple of decades, is inextricably linked with the problem of sepsis (SS). This is due to the fact that more than half of the cases of generalized infection are a consequence of AP [7,8]. If we take into account the fact that SS has become one of the most serious problems of global health, and the negative dynamics of its statistics causes deep concern among specialists, then such native integration of two serious problems cannot be limited to just a banal expression of concern. According to the World Health Organization (WHO), in the last few years alone, the total number of SS cases has increased from 30 to 49

million per year, and fatal outcomes - from 6 to 11 million per year [9,10]. Hospital mortality in SS, which in recent years reached 20% in Europe and North America, that is, in the most advanced health care systems, [11,12], has increased to 40% [13]. At the same time, in the USA, SS is the main cause of hospital mortality [14].

Although there is currently a tendency to consider SS as an independent nosology and many publications have stopped referring to the sources of septic complications, it is easy to understand that in the above WHO statistics, SS in most patients is a continuation of inadequately treated AP. Such statistical manipulations are one of the reflections of existing professional misconceptions, primarily in solving the problem of AP. SS in this chain and sequence of manifestations of the observed process is a secondary link, acting as a complication of the main process. After all, SS does not occur suddenly without apparent reasons, right? In order to understand the reasons for the discrepancies that have arisen, the most optimal way is a consistent analysis of the assimilation of medical information at the university. Judging by the materials presented in the educational literature, clinical disciplines setting out the principles of treating AP ignore fundamental information, presenting the disease as a result of the action of certain pathogens. It is interesting to note that over the years the list of pathogens has been revised, but without reasonable explanations.

The study of medicine begins with knowledge of anatomy, physiology and other basic disciplines, without mastering the materials of which it is impossible to even imagine a modern doctor. Let us limit the analysis of such information to only those materials that are directly related and important to the topic under discussion. Studying the respiratory and circulatory organs, each student of a medical university learns not only about their inseparable anatomical and functional connection, but also about the incomparable features of the two circulatory circles. Fundamental differences concern both the morphological structure of the vessels and their functional parameters. Blood pressure in the pulmonary vessels is approximately 6-8 times lower than on the periphery [15-18], but the body needs to preserve and maintain these proportions, which are of vital importance. Disproportions between the two halves of the circulatory system are incompatible with their synchronous operation and life support.

Small changes in the parameters of pulmonary blood flow can lead to far-reaching consequences. For example, it has been established that an increase in pressure in the pulmonary vessels by only 5 mm Hg is accompanied by interstitial edema of the pulmonary tissue, and by 10 mm Hg already leads to pulmonary edema [18]. Despite such unexpected changes that can occur in various situations, the body comes out of such states on its own and does not require any volitional efforts from us, which in this case are of no importance. To eliminate such deviations, our body is equipped with an autonomous defense system that automatically triggers as soon as the first signs of disturbances appear. One of the most important mechanisms of such protection is the so-called unloading reflex, which was discovered almost a century ago [19]. This reflex is due to the presence of baroreceptors in the pulmonary vessels, which react to the slightest pressure shifts, leading to blood retention in the periphery and unloading of the pulmonary circulation.

The next educational section, important for discussion and understanding of the problem raised here, concerns the phenomenon of non-specific inflammation and inflammatory tissue transformation. Modern interpretations of such processes focus on their description at the cellular and molecular level. This trend is supported by the search for features of micromechanisms depending on the action of various pathogens and factors of the response defense. The results of such studies are of purely scientific importance, since practicing clinicians receive only a virtual representation of all these phenomena at an invisible level, which cannot help in monitoring patients. Attempts to implement such results in practice, especially when it comes to acute and severe diseases, are based on incorrect theoretical assumptions, so in this case, when solving the problem of AP, they a priori cannot bring an urgent improvement in the integrative indicators of the disease. It is the integrative manifestations of the process and the same proportionate actions that can bring the desired result.

Integrative processes of classical inflammation begin with the primary reaction of the vessels, with local plethora in the inflammation zone, slowing of blood flow and a sharp increase in the permeability of the vascular walls [20]. These changes are accompanied by the obligatory appearance of 5 classical signs of inflammation, described about two thousand years ago by Celsus and Galen and having received convincing practical confirmation.

The last sign «loss of function» is of the greatest importance, which forms the specificity of clinical manifestations depending on the localization. All these described signs are of an integrative nature, as are the measured parameters of functional disorders, which makes it easier for clinicians to navigate the condition of patients.

A well-known feature of pneumonia is the development of the process without pain syndrome due to the absence of pain receptors and the appearance of pain only after the involvement of the pleural sheets [21]. Nature has provided a more important and effective factor, replacing pain receptors in the lung tissue with baroreceptors of its vessels. In this regard, the pain signal in patients with AP would not have the same significance as it has in other localizations of inflammation. At the same time, the reflex of unloading from baroreceptors allows avoiding sudden critical situations. Nevertheless, the individual rate of development of the inflammatory reaction determines the depth of the onset of restructuring. With aggressive development of the disease, compensatory changes in the systemic blood flow create a typical picture of shock, which, however, is not septic in nature. This form of shock is of pulmonogenic origin and can be quickly eliminated with the timely use of pathogenetic methods. All this was described and proven by objective tests and clinical results 40 years ago [22].

The above information gives an idea that inflammation disrupts the specific function of the affected organ, which is caused by the inflammatory of specific structures, and not by the etiology of non-specific processes. That is why persistent attempts to find differential diagnostic differences of AP depending on the pathogen using various methods have invariably failed. Over this long period, a huge amount of money and effort has been spent, the tactics for achieving the set goal have changed, but the results have remained unchanged. It is surprising that medicine has ceased to attach due importance to its own experience and does not seek to find out the reasons for failures and revise the principles of solving the set problems. The modern professional view of this problem completely ignores the basic information, the most important provisions of which were noted above. The abundance of articles, reports and the results of discussions at the highest expert forums indicate that the intensive search for methods of etiological diagnostics and etiotropic treatment continues. In this closed space, modern medicine continues a hopeless search for a way out

of the impasse into which it has imperceptibly led itself. However, there is still no sign of a critical and radical analysis of the profound changes in general conditions that have occurred during this time under the influence of antibiotics.

In this regard, it is impossible to find a reasoned explanation for the fact that modern medicine, having achieved colossal success in many areas, continues to primitively consider antibiotics to be the main and primary means of treating inflammatory processes. The ineffectiveness of this established stereotype is most clearly demonstrated by the example of patients with AP. As is known, since the appearance of antibiotics in clinical practice, these drugs are capable of specifically suppressing individual types of microorganisms, but do not have a direct effect on the mechanisms of the process itself, the individual nature of which determines the entire specificity and severity of the disease. The spectacular and impressive results of the first uses of penicillin were due to the virginity of the microflora surrounding us, not yet familiar with this type of aggression in such a concentrated form. The introduction of antibiotics at that time led to the rapid suppression of an important factor of inflammation, and the body eliminated the changes that had arisen without any particular problems.

However, the initial triumph of antibiotics was short-lived, as the etiology of AP began to demonstrate its inconstancy, which served as an incentive for the increased production of new drugs [23], laying the foundation for the future ideology of maintaining the effect of this therapy. Concentration of attention on early diagnostics of the causative agent of AP and targeted etiotropic therapy continue to determine the basis for solving the problem under discussion. If we take into account the fact that antibiotics have been widely used in clinical practice for more than 80 years, then there is no explanation for the fact that none of the specialists over this long period have tried to reconsider the true place of these drugs in the treatment of AP, where they, possessing only selective antimicrobial activity, today can play at best an auxiliary role. At the same time, we must forget about the primary conditions in which antibiotic therapy was started. The etiology of pneumonia has completely changed by now and its future prospects are difficult to predict.

Can we continue to assert the crucial role of antibiotics in the treatment of AP if the viral forms of this disease continue to grow

rapidly worldwide? As the events of the SARS-CoV-2 pandemic have shown, memorized formulas and beliefs about the indispensable role of antibiotics in the treatment of this group of patients have completely destroyed professional approaches to providing care for COVID-19 pneumonia. Coronavirus inflammation of the lung tissue during the pandemic was treated with antibiotics in 70-80 percent or more, although bacterial coinfection was observed in only a few percent [24-26]. In addition to the increase in viral forms of the disease in recent years, in 60 percent or more of cases, the causative agent of AP remains unidentified, despite the improvement of diagnostic methods [27-29]. Despite widespread attempts to find ways of optimal targeted therapy, wide expert forums of specialists have begun to recognize the irrelevance of determining the etiology of AP, recommending an empirical choice of drugs [30].

However, information about the constant and dynamic change in the etiology of AP is a fact that arose with the beginning of the clinical use of antibiotics, continues and will be valid as long as antibiotics are in demand. This fact should not only be recognized, but, most importantly, it is necessary to conduct a comprehensive analysis and make adequate strategic conclusions. So far, in this discussion, the entire problem is considered as microbial resistance and ways to overcome it. In this regard, reasonable concerns are caused by the plans and prospects proposed by WHO experts, who, on the one hand, declare microflora resistance a global catastrophe, and on the other hand, support and approve the creation of a new generation of antimicrobial drugs [31].

From my point of view, the gloom and hopelessness of this direction lies in the fact that, without assessing and not fully understanding the consequences of the previous experience of antibiotic therapy, specialists want to start a new development of these drugs. Without fully realizing what serious consequences the previous experience "gave" us, making plans to revive the effect of this therapy at the molecular, nanotechnological and even genetic level seems extremely ill-considered. Having received by now horrific consequences that medicine cannot explain reasonably and comprehensively, let alone correct the medical situation, it is not worth checking how nature will react to the new aggression. The consequences may be most unexpected, but there is no doubt that this direction does not promise reliable prospects.

The above information was presented as the most important prerequisites known to medicine, which should predetermine the formation of the existing principles of primary treatment of such patients. However, the traditions and paradigms of modern care for AP were formed in the era of antibiotics and, despite the constant change in the etiology of the disease, a decrease in the effectiveness of drugs and a decrease in the justification for their use, were increasingly focused on the leading role of antibiotics in the treatment of this category of patients. Today, antibiotics, as never before, continue to be recognized as the main means of providing not only general, but also emergency care for AP. Over the past decades, many studies have been conducted on the results of AP treatment depending on the timing of the start of antimicrobial therapy immediately after diagnosis. No one has been able to provide convincing data on the effect of the time of antibiotic administration on the results, but it is very important to note that, despite the abundance of such materials, the persistent use of this idea, which has long since lost its meaning, to find solutions continues to this day.

Antibiotics, continuing to form the therapeutic basis of AP, simultaneously play the role of first aid in this disease. Until now, the rule prevailing in practical medicine is that the prescription of antibiotics implies waiting for their therapeutic effect within 48-72 hours (!?) [30,31]. This time is considered a very important period for changing the nature of further etiotropic treatment, since the modern ideology of the disease is based on the leading role of the pathogen in the development process, and its suppression is the goal of therapeutic directions. However, no one focuses on the fact that in recent years, the number of patients with AP has been growing, in whom the lack of effect after the prescription of antibiotics forces the use of additional measures of assistance. Such support is especially urgently needed in aggressive forms of the development of the process, but the methods used do not correspond to the pathogenesis of AP, which explains the reason for the ineffectiveness of first aid and the need to transfer 25% of patients from the general department to the intensive care unit in the first two days [5,6].

The modern ideology of the discussed problem has literally turned the provision of auxiliary care to this category of patients «upside down». Guided by the principles of eliminating the

consequences of the pathogen's aggression, regardless of the localization of the primary focus, today in all cases of inflammatory processes standard general therapeutic approaches to the selection of additional means of assistance are used. In the discussions and recommendations of specialists, there are no indications of the danger of such procedures in AP as intravenous infusions. Meanwhile, focusing on the features of the pathogenesis of AP, it is easy to understand the fundamental difference in the mechanisms of pulmonary inflammation compared to inflammatory processes of any other localization, but in the systemic circulatory pool. A comprehensive and complex analysis of the observed phenomena during infusions in patients with AP, including the results of experimental data, allows us to understand the reasons for the ineffectiveness of modern therapeutic efforts and to be convinced of the danger of infusion therapy in AP, especially in the initial period of the disease [22]. Therefore, today the agenda should not be the quality of the therapeutic agents used, but the principles of priority directions.

If we take into account the inevitability of lung function impairment as a result of organ tissue damage by the inflammatory process, then on the basis of what scientific arguments are antibiotics still considered first aid for this disease? As the abundance of materials on this topic shows, the picture and severity of the disease do not depend on the type of pathogen, and no one has managed to either prove this fact or conduct differential diagnostics depending on this factor. In addition, we would like to understand the logic of such an interpretation, when the effect of emergency care should be expected within 2-3 days [30,31]? Not only does such care have a delayed effect, but it also has a specificity of action, which does not guarantee its success. As long-term practice shows, super-aggressive forms of inflammation are capable of demonstrating significant expansion in this short period of time of a couple of days. In recent years, such forms of the disease have become more common, which is largely due to a change in the etiology of the disease and the growing allergization of the population [32]. In any case, considering the use in emergency situations of a technique that can only have an indirect effect and only with an adequate choice of drug, in modern conditions and circumstances seems at least frivolous.

What is the first and emergency aid for patients with AP today? Antibiotics, the effect of which requires an unacceptably long wait? Or intensive infusion therapy, aimed against protective

and adaptive factors and stimulating the processes of edema and infiltration in the area of inflammation, which it reaches first? Or maybe oxygen supply, which usually does not affect the general condition of the patient? The real situation shows that the arsenal of official medicine currently does not have a coordinated and scientifically substantiated first aid that can immediately alleviate the condition of patients with AP. In order for the first therapeutic efforts to bring inevitable satisfaction to both the patient and the doctor, it is necessary to try to reduce the influence of the causes that cause functional disorders in the lungs, right? After all, striving today to fight the pathogen by any means, the medical strategy remains very far from the stated goal.

The above brief mention of the mechanisms of development of the inflammatory process in the lungs shows that this disease is primarily a circulatory, and not a respiratory catastrophe, as is commonly believed. Therefore, the methods of increasing oxygenation used today, various methods of improving ventilation of the lungs do not bring the desired result. At the same time, as our experience has shown, confirmed by objective tests and subsequent results, such methods as, for example, cupping therapy or cervical vagosympathetic block, bring rapid relief to the patient as a result of equalizing the proportions between ventilation and blood flow in the lungs [22]. An even greater clinical effect was observed after the use of short-term cold wraps or cold baths, but in this case we were unable to confirm the observed effect using objective tests. Nevertheless, such procedures have an undoubted positive pathogenetic effect at the initial stages of the process, allowing to sharply reduce the intensity of its development. One would hope that some researcher will be able to objectively prove the therapeutic benefits of such cooling procedures, which are currently widely used only in health and sports complexes.

The use of first aid for AP should be as early as possible, being only the first stage in the treatment of this severe category of patients. Further therapy should support and continue the achieved pathogenetic effect, focusing on the mechanisms of pathogenesis of the disease, and not on its etiology. The use of etiotropic therapy should be considered as an auxiliary, and not the main treatment. We used such principles in the most aggressive forms of AP and the results showed that timely initiation of such treatment allows for rapid arrest and resolution of the process, avoiding possible complications [22].

Thus, the analysis of the quality and compliance of first aid with the nature of the AP allows us to note a deep deformation of professional views on the discussed problem, which arose under the influence of an incorrect interpretation of the role of antibiotics in the treatment of inflammatory processes. The simple principle of suppressing the causative agent of the process can at best eliminate only the «tip of the iceberg», leaving the entire array of the problem. At the same time, the incorrect use of additional methods of providing assistance can significantly aggravate the changes that have occurred. The reason for such discrepancies and the growing trend of negative results is the discrepancy between the general concept of the disease and the classical materials of medical science. At present, a radical revision of views on this problem is the most important step in its solution.

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Conflict of Interest

The author states that he has no conflict of interest.

Bibliography

1. World Health Organization. The top 10 causes of death (2020).
2. Laporte L., *et al.* "Ten-year trends in intensive care admissions for respiratory infections in the elderly". *Annals of Intensive Care* 8 (2018): 84.
3. Kristin R Wise., *et al.* "Increased Long-Term Mortality in Patients Admitted to the Intensive Care Unit with Health-Care Associated Pneumonia". *medRxiv* (2021): 2021.12.23.21267010.
4. Lakbar I., *et al.* "Association between mortality and highly antimicrobial-resistant bacteria in intensive care unit-acquired pneumonia". *Scientific Report* 11 (2021): 16497.
5. Boëlle PY., *et al.* "Trajectories of Hospitalization in COVID-19 Patients: An Observational Study in France". *Journal of Clinical Medicine* 9 (2020): 3148.
6. Cavallazzi R., *et al.* "The burden of community-acquired pneumonia requiring admission to ICU in the United States". *Chest* 158 (2020): 1008-1016.
7. Cilloniz C., *et al.* "Pure viral sepsis secondary to community-acquired pneumonia in adults: risk and prognostic factors". *Journal of Infectious Disease* 220 (2019): 1166-1171.
8. Lin CK., *et al.* "Serum vascular endothelial growth factor affects tissue fluid accumulation and is associated with deteriorating tissue perfusion and oxygenation in severe sepsis: a prospective observational study". *European Journal of Medical Research* 28 (2023): 155.
9. Burkhart M. "Improving Sepsis Bundle Compliance in the Emergency Department". The Eleanor Mann School of Nursing Student Works (2001).
10. WHO. Sepsis (2024).
11. Fleischmann C., *et al.* "Assessment of global incidence and mortality of hospital-treated sepsis. Current estimates and limitations". *American Journal of Respiratory and Critical Care Medicine* 193 (2016): 253-272.
12. Rudd KE., *et al.* "Global, regional, and national sepsis incidence and mortality, 1990-2017: analysis for the Global Burden of Disease Study". *Lancet* 395 (2020): 200-211.
13. Vincent JL., *et al.* "Frequency and mortality of septic shock in Europe and North America: a systematic review and meta-analysis". *Critical Care* 23 (2019): 196.
14. Rhee C., *et al.* "Prevalence, underlying causes, and preventability of sepsis-associated mortality in US acute care hospitals". *JAMA Network Open* (2019): 2e187571.
15. "What Is Pulmonary Hypertension?". From Diseases and Conditions Index (DCI). National Heart, Lung, and Blood Institute. September (2008).
16. Colledge NR., *et al.* "Davidson's Principles and Practice of Medicine (21st ed.)". Edinburgh: Churchill Livingstone/Elsevier (2010).
17. "Normal Hemodynamic Parameters - Adult". Edwards Lifesciences LLC. Archived from the original on 2010-11-10.
18. Olivia Vynn. "Cardiology secrets". Chapter 41, p. 210. Adair Edition: 2, illustrated Published by Elsevier Health Sciences, (2001).
19. Schwegk H. "Der Lungenentlastungsreflex". *Pflügers Archges Physiol* 236 (1935): 206-219.
20. Inflammation.

21. Chandrasoma P and Taylor CR. "Part A. "General Pathology", Section II. ""The Host Response to Injury", Chapter 3. "The Acute Inflammatory Response", sub-section "Cardinal Clinical Signs". Concise Pathology (3rd ed.). McGraw-Hill. OCLC 150148447 (2005).
22. I Klepikov. "Myths, Legends and Real Facts About Acute Lung Inflammation". Cambridge Scholars Publishing. (2024): 338.
23. Aminov RI. "A brief history of the antibiotic era: lessons learned and challenges for the future". *Frontiers in Microbiology* 1 (2010): 134.
24. BD Huttner, *et al.* "COVID-19: don't neglect antimicrobial stewardship principles!" *Clinical Microbiology and Infection* 26.7 (2020): P808-810.
25. B Beovic, *et al.* "Antibiotic use in patients with COVID-19: a 'snapshot' Infectious Diseases International Research Initiative (ID-IRI) survey". *Journal of Antimicrobial Chemotherapy* 85 (2020): 326 (2020).
26. Rawson TM, *et al.* "Bacterial and fungal co-infection in individuals with coronavirus: A rapid review to support COVID-19 antimicrobial prescribing [published online ahead of print, 2020 May 2]". *Clinical Infectious Disease* (2020): ciaa 530.
27. C Cillóniz, *et al.* "Community-acquired pneumonia in outpatients: aetiology and outcomes". *European Respiratory Journal* 40.4 (2012): 931-938.
28. Jain S, *et al.* "CDC EPIC Study Team Community-acquired pneumonia requiring hospitalization among U.S. adults". *The New England Journal of Medicine* 373 (2015): 415-427.
29. C Castillo. "2020 IDCA/ATS Community-Acquired Pneumonia Guideline: more micro, less macrolide, no HCAP". 15th Annual NW Regional Hospital Medicine Conference (2020).
30. JP Metlay, *et al.* "Diagnosis and Treatment of Adults with Community-acquired Pneumonia. An Official Clinical Practice Guideline of the American Thoracic Society and Infectious Diseases Society of America". *American Journal of Respiratory and Critical Care Medicine* 200.7 (2019): e45-e67.
31. Martin-Loeches I, *et al.* "ERS/ESICM/ESCMID/ALAT guidelines for the management of severe community-acquired pneumonia". *Intensive Care Medicine* 49 (2023): 615-632.
32. Allergy.