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ALTERATIONS OF BLOOD PRESSURE IN SCHOOL-AGE CHILDREN WITH ACUTE PNEUMONIA

Summary.

Acute inflammatory diseases of the respiratory tract remain a leading cause of morbidity, hospitalization, and mortality among children worldwide. Among these, acute community-acquired pneumonia (CAP) is particularly significant, accounting for approximately 16% of global deaths in children under five years of age. Pediatric presentations of pneumonia differ markedly from those observed in adults; however, the presence of cardiovascular comorbidities can complicate the clinical course across all age groups. It is well established that respiratory pathologies may predispose patients to cardiovascular complications, which are associated with a substantially increased risk of adverse outcomes.

Objective. The present study aimed to evaluate changes in blood pressure (BP) in children with CAP during the acute phase and early recovery.

Materials and methods of the study. Thirty children aged 8-12 years (mean age 11.0 ± 0.2 years) diagnosed with CAP were enrolled in the study. A comparison group consisted of 30 age- and sex-matched healthy children. In the CAP group, BP measurements were taken twice: on the day of hospital admission and during the early recovery phase. Heart rate and oxygen saturation were also assessed.

The study was conducted in accordance with the principles of the World Medical Association's Declaration of Helsinki «Ethical Principles for Medical Research Involving Human Subjects». Informed consent to participate was obtained from all those included in the study (parents of children or their guardians), which emphasizes the absence of invasive interventions. The study protocol was discussed and approved at a meeting of the Biomedical Ethics Committee of Bukovinian State Medical University.

Descriptive statistics analysis was done using program Statistica (v.6, Statsoft). Quantitative data were presented as mean and standard error of mean ($M \pm SE$), in comparisons p -values were two-tailed and $p < 0.05$ was considered statistically significant. For assessment and explanation data AI program «Copilot» (Microsoft) was used.

The work was carried out as part of the planned scientific research of the Department of Pediatrics, Neonatology, and Perinatal Medicine of Bukovinian State Medical University: «Chronobiological and adaptation aspects and features of autonomic regulation in pathological conditions in children of different age groups» (registration number 0122U002245, duration 2022-2026).

Results of the study. According to the general condition all cases of CAP classified as mild or moderate, and no severe or complicated forms were identified. During the acute phase, signs of a systemic inflammatory response were present, including fever, tachycardia, and tachypnea, as well as manifestations of mild hypoxemia, which persisted into the early recovery period. BP indices (systolic, diastolic, and pulse pressure) were lower in both periods compared with healthy children, likely due to vasodilation and alterations in autonomic regulation associated with inflammation. At disease onset, a reduction in stroke volume and cardiac output was observed, followed by an increase during treatment along with a gradual decrease in heart rate. These findings are consistent with previous studies and indicate that the hemodynamic and oxygenation changes in children with acute pneumonia are functional and reversible in nature.

Conclusions. In CAP cases physiological burden on both cardiovascular and respiratory systems could be present. Recovery leads to partial normalization, but some parameters – like SaO_2 and cardiac output, may take longer time to fully return to baseline. CAP as systemic illness affects multiple organ systems and recovery is gradual rather than immediate. In managing children with CAP must be avoided routine aggressive fluid loading with signs of cardiac compromise.

Keywords: Acute Community-Acquired Pneumonia; Children; Blood Pressure; Heart Rate; Cardiac Output.

Introduction

Acute respiratory diseases in children remain a leading global cause of high morbidity. Between them community-acquired pneumonia (CAP) is a common infection frequently affecting children and provoked significant level of hospitalization and mortality [1-3]. The estimated annual incidence of pneumonia in Europe is 11 to 16 cases/1000 in those 7 to 14 years of age. Clinical presentations and agents of pneumonia in children and infants are different compared with adults [4].

Acute pneumonia in school-age children is accompanied by significant changes in systemic hemodynamics, including alterations of arterial blood pressure (BP) that reflect the combined effects of inflammation, hypoxia, and age-related cardiovascular physiology [3, 5]. School-age

children possess more mature autonomic and myocardial regulation than infants, yet they remain highly sensitive to disturbances in oxygen delivery and systemic inflammation. These developmental characteristics shape the specific pattern of BP responses observed in CAP [4, 6].

During the acute phase of CAP, the inflammatory process initiates a cascade of metabolic, autonomic and endothelial disturbances that collectively influence systemic BP regulation [7, 8]. Pro-inflammatory cytokines impair endothelial function, alter nitric oxide – dependent vascular tone, and increase capillary permeability. In parallel, fever, tachypnea, and elevated metabolic demands accelerate oxygen consumption [5, 8]. These changes may contribute to transient reductions in systemic vascular resistance and fluctuations of arterial pressure [9].

It is well known that patients with cardiovascular comorbidities had a significantly higher risk of the severe outcomes, aligning with findings showing they are more susceptible to poor outcomes in respiratory infections [3, 10]. BP, heart rate (HR) and cardiac output, are the principal hemodynamic processes parameters, which could be affected in pneumonia cases. On the other hand, cardiovascular disorders worsen the course of acute respiratory diseases, especially pneumonia [11, 12]. Episodes of diseases often cause increased heart rates leading to an elevated cardiac workload in right heart chambers, right ventricle myocardial diastolic dysfunction and even potentially HR in severe cases [4, 7, 13].

In the pathogenesis of CAP several factors contribute to hemodynamic alterations, including the degree of arterial narrowing and vasoconstriction, fluctuations in blood volume, and increases in HR and BP [5, 6]. Patients recovering from pneumonia who exhibit cardiovascular changes may experience delayed return to their pre-infection health status [7]. Numerous clinical studies have demonstrated an elevated risk of cardiovascular events following acute respiratory infections, including CAP [8, 9]. Additional factors related to autonomic dysregulation, such as increased sympathetic tone, promote vasoconstriction via α -adrenergic receptors, further reducing blood flow [9, 10, 11].

During CAP, a combination of vasomotor, hormonal, and metabolic disturbances may lead to mitochondrial dysfunction, which in turn exacerbates systemic dysfunction syndromes. The inflammatory response not only impairs endothelial function but also disrupts autonomic control of microcirculation, compounding the hemodynamic instability [11]. Sympathetic overactivity, characterized by heightened α -adrenergic receptor stimulation, contributes to peripheral vasoconstriction and diminished perfusion [9].

In general, systemic arterial BP in CAP often decreases or remains unstable, particularly in severe cases. This instability is primarily driven by hypoxia, systemic intoxication, and compromised cardiac function. Tachycardia – commonly present during fever and respiratory distress – initially helps maintain cardiac output, but persistent elevation in heart rate reduces diastolic filling time, leading to diminished stroke volume [12, 13]. Inflammatory mediators may additionally depress myocardial contractility. Elevated pulmonary pressures secondary to hypoxic vasoconstriction can impose right ventricular strain, limiting left ventricular filling through ventricular interdependence. Consequently, systemic cardiac output is reduced, further contributing to a decline in arterial blood pressure [14-16].

Mechanisms by which pneumonia affects systemic blood pressure include hypoxia and intoxication [17]. Inflammation of pulmonary tissue reduces blood oxygenation and insufficient oxygen saturation leads to decreased vascular tone and reduced peripheral resistance, contributing to a drop in arterial pressure [18-19]. From the other side reduced cardiac output could be present – elevated pressure in the pulmonary circulation causes right ventricular overload and limits left ventricular

filling [20]. Fever and increased respiratory water loss further aggravate this reduction in effective circulating volume [21]. As a result, the volume of blood entering the systemic circulation decreases, leading to lowering of arterial BP. The pro-inflammatory state induced by infectious agents contributes to endothelial dysfunction, which can significantly affect BP and microcirculatory regulation – both of which are critical for maintaining adequate tissue perfusion [22, 23]. Although children typically exhibit stronger compensatory mechanisms than infants or toddlers, CAP can still lead to measurable decreases or fluctuations in BP, especially in the presence of significant respiratory compromise or systemic intoxication [15, 24]. Understanding the mechanisms behind BP alterations in this age group is essential for early recognition of hemodynamic instability and timely therapeutic intervention [25, 26].

The aim of the study was to present study aimed to assess BP changes in children with CAP during acute and early recovery phases of the disease.

Materials and methods

In this study 30 children with CAP in age 8-12 years ($11,0 \pm 0,2$ years) were enrolled. The comparison group – 30 age- and sex-matched healthy children. Children from both groups did not have a significant difference in anthropometric measurements – height and weight. The criteria for inclusion in the main group presents of confirmed CAP diagnosis. The exclusion criteria for both groups were any kidney, heart, nervous system or other chronic diseases. In main group BP measurements were done twice – on the day of admission and during early recovery on the 5-7th day. BP and HR were measured by oscillometric device (Omron HEM-7121, Omron, Japan) with an appropriately sized cuff. Systolic (SBP), diastolic (DBP) and pulse BP (PPB) were fixed. Systolic and minute blood volume were also calculated, as the most important indicators of the contractile function of the heart and cardiac output. Determination of systolic and minute volumes was calculated by an indirect method using Star's formula [27]. The HR and oxygen saturation (SaO_2) were also assessed by pulse oximetry device (CMS50D Contec Medical System, PRC).

The study was conducted in accordance with the principles of the World Medical Association's Declaration of Helsinki «Ethical Principles for Medical Research Involving Human Subjects». Informed consent to participate was obtained from all those included in the study (parents of children or their guardians), which emphasizes the absence of invasive interventions. The study protocol was discussed and approved at a meeting of the Biomedical Ethics Committee of Bukovinian State Medical University.

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Research results and discussion

According to our data, all cases of CAP classified as mild or moderate, and no severe or complicated forms were identified in the study cohort. The clinical picture during the acute phase was characterized by a systemic inflammatory response, with the majority of children presenting with elevated body temperature, tachycardia, and a significantly increased respiratory rate (Table 1). These findings correspond well with established clinical guidelines, which describe fever, tachycardia,

and tachypnea as early diagnostic markers of moderate pediatric pneumonia [3].

In addition, in beginning of disease some proportion of the patients with moderate CAP demonstrated outward signs of respiratory effort, including nasal flaring, mild intercostal retractions, and occasional use of accessory respiratory muscles, indicating compensatory mechanisms to maintain adequate ventilation. SaO_2 measurements confirmed the presence of mild hypoxemia in the acute period. Although oxygen saturation improved progressively with treatment, it did not fully normalize during early recovery, a trend also noted in previous observational studies [3, 28]. This delayed normalization of oxygenation may reflect ongoing reparative processes in the lung parenchyma and residual ventilation – perfusion mismatch, which tends to resolve more slowly than clinical symptoms. The persistence of slightly reduced SaO_2 emphasizes the importance of monitoring oxygenation even after the child's general condition has visibly improved.

Table 1

Blood pressure and related disease parameters in children with CAP

	Parameter (M \pm m)	CAP group		Healthy group
		Admission (Day 1-2)	Early Recovery (Day 4-7)	
1	Systolic BP, mmHg	104.5 \pm 9.2	98.7 \pm 8.5*	111.2 \pm 6.5
2	Diastolic BP, mmHg	68.3 \pm 7.1	64.1 \pm 6.5*	72.5 \pm 4.8
3	Pulse Pressure, mmHg	36.2 \pm 4.3	34.6 \pm 4.0*	38.7 \pm 3.8
4	Heart Rate, bpm	126.8 \pm 13.7*	102.4 \pm 11.2*	86.4 \pm 9.8
5	Stroke Volume, ml	59.9 \pm 6.6*	71.6 \pm 8.4	68.5 \pm 5.8
6	Cardiac Output, l	5.05 \pm 1.5	7.31 \pm 0.8*	5.91 \pm 0.6
7	Temperature, °C	38.7 \pm 0.6*	36.8 \pm 0.3*	36.4 \pm 0.3
8	SaO_2 (%)	93.2 \pm 3.9*	96.3 \pm 1.7*	98.6 \pm 0.2

* – Statistical difference with healthy group and ** – admission with recovery, ($p < 0.05$)

BP was altered during illness: SBP was 104.5 ± 9.2 mmHg at admission and decreased to 98.7 ± 8.5 mmHg in early recovery; the healthy group mean SBP was 111.2 ± 6.5 mmHg. The same dynamic was with DBP but PBP – they were lower than in healthy group but without significant difference between them. Stroke volume had peculiar dynamic with lower level at admission (59.9 ± 6.6 ml) and increased in early recovery (71.6 ± 8.4 ml) even more than in healthy control (68.5 ± 5.8 ml). Cardiac output had nearly same dynamic.

The mean SBP in both periods in patient was lower possibly due to vasodilation, altered autonomic regulation, or fluid shifts associated with systemic inflammation [4, 21]. These hemodynamic changes were accompanied by a mild narrowing of pulse pressure, which may reflect a transient reduction in stroke volume or increased peripheral vascular resistance. Treatment of pneumonia in patients was successful and produces stepwise improvements in vital signs as inflammation resolves, oxygenation improves, and the child becomes hemodynamically stable. As treatment progressed, BP values demonstrated a gradual upward trend toward age-appropriate norms, supporting the hypothesis that these changes are reversible and primarily functional rather than structural.

Tachycardia is common in infection and fever improves with recovery but remains elevated. SBP lower during illness and early recovery, likely due to systemic inflammation or reduced vascular tone. DBP had similar trend as systolic BP, indicating overall hypotension

during CAP. Narrowed pulse pressure may reflect reduced stroke volume or increased peripheral resistance. Stroke volume initially reduced, likely due to impaired cardiac function or dehydration; improves with treatment. Cardiac output increased during recovery due to compensatory mechanisms including rise stroke volume and decrease of HR.

Our findings of hemodynamic and oxygenation are consistent with prior pediatric CAP literature. As reported elsewhere, fever and systemic infection were associated with tachycardia that declined with clinical recovery. Elmeazawy R. et al. (2025) show that heart rate sometimes remained above baseline despite symptomatic improvement [11]. Investigators from Japan Akita University – Watanabe K. et al (2020) show the same reaction as in our research, when stroke volume was reduced on admission and improved with treatment, while cardiac output rose during recovery as stroke volume increased and heart rate decreased [21]. Hypoxemia were common on presentation and improved but was not fully normalized in early recovery [28]. These observations align with previously published cohorts and physiologic studies [15].

Conclusions. Our data clearly illustrates the physiological burden of CAP on both cardiovascular and respiratory systems. Recovery leads to partial normalization, but some parameters – like SaO_2 and

cardiac output, may take longer time to fully return to baseline. It's show how systemic illness like CAP affects multiple organ systems and how recovery is gradual rather than immediate. In managing children with CAP must be avoided routine aggressive fluid loading with signs of cardiac compromise.

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Prospects for further research. It may be rational to study blood pressure changes in other respiratory pathologies, such as acute bronchitis and bronchiolitis.

Conflict of interest. The authors report the absence of any conflict of interest.

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ЗМІНИ АРТЕРІАЛЬНОГО ТИСКУ У ДІТЕЙ ШКІЛЬНОГО ВІКУ З ГОСТРОЮ ПНЕВМОНІЄЮ

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Резюме.

Гострі запальні захворювання дихальних шляхів залишаються основною причиною захворюваності, госпіталізації та смертності дітей у всьому світі. Серед них є значущою гостра позалікарняна пневмонія (ГПП), на яку припадає приблизно 16% світових смертей серед дітей віком до п'яти років. Педіатричні прояви пневмонії помітно відрізняються від тих, що спостерігаються у дорослих, однак наявність серцево-судинних супутніх захворювань може ускладнити клінічний перебіг у всіх вікових групах. Достовірно встановлено, що респіраторна патологія викликає серцево-судинні зміни, які пов'язані зі значно підвищеним ризиком несприятливих наслідків.

Мета дослідження. Оцінити зміни артеріального тиску у дітей з гострою позалікарняною пневмонією під час гострої фази та раннього одужання.

Матеріали та методи. У дослідження було включено 30 дітей віком 8-12 років (середній вік $11,0 \pm 0,2$ року) з діагнозом ГПП. Групу порівняння склали 30 здорових дітей однакового віку та статі. У групі хворих вимірювання АТ проводили двічі: у день госпіталізації та на ранньому етапі відновлення. Також оцінювали частоту серцевих скорочень і насилення крові киснем. Дослідження проводилося з урахуванням принципів Гельсінської декларації Всесвітньої медичної асоціації «Етичні принципи медичних дослідень за участю людини в якості об'єкта дослідження». У всіх осіб включених в дослідження було отримано інформовану згоду на участь (батьків дітей або їх опікунів), яка акцентувала увагу на відсутності інвазійних утручань. Протокол дослідження обговорено та затверджено на засіданні комісії з біоетики Буковинського державного медичного університету, рішенням якої воно було схвалено. Аналіз описової статистики проводили за допомогою програми Statistica (v.6, Statsoft). Кількісні дані подано як середнє значення та стандартна похибка середнього ($M \pm SE$); при порівнянні використовували двосторонні *p*-значення, і $p < 0,05$ вважалося статистично значущим. Для оцінки та пояснення даних використовувалася програма штучного інтелекту «Copilot» (Microsoft). Робота виконана в рамках планової науково-дослідної роботи кафедри педіатрії, неонатології та перинатальної медицини Буковинського державного медичного університету: «Хронобіологічні й адаптаційні аспекти та особливості вегетативної регуляції при патологічних станах у дітей різних вікових груп» (№ державної реєстрації 0122U002245, термін виконання 2022-2026 рр.).

Результати дослідження. Відповідно до загального стану всі випадки пневмонії були класифіковані як легкі або середньої тяжкості; тяжких або ускладнених форм не виявлено. У гострій фазі спостерігалися ознаки системної запальної відповіді, зокрема підвищення температури тіла, тахікардія та тахіпnoe, а також прояви легкої гіпоксемії, які зберігалися і в ранній період одужання. Показники АТ (системолічного, діастолічного та пульсового) в обидва періоди були нижчими, ніж у здорових дітей, що, ймовірно, пов'язано з вазодилатацією та змінами вегетативної регуляції на тлі запалення. На початку захворювання відмічалося зниження ударного об'єму та серцевого викиду, які згодом змінювалися зростанням цих показників у процесі лікування разом із поступовим зниженням частоти серцевих скорочень. Отримані результати узгоджуються з даними попередніх досліджень і свідчать про функціональний та зворотний характер гемодинамічних і оксигенатічних змін у дітей із гострою пневмонією.

Висновки. У випадках ГПП зростає фізіологічне навантаження як на серцево-судинну, так і на дихальну системи. Відновлення призводить до часткової нормалізації, але для деяких параметрів, таких як SpO_2 і серцевий викид, може знадобитися більше часу, щоб повністю повернутися до вихідного рівня. ГПП як системне захворювання вражає декілька систем органів, і одужання є поступовим, а не миттєвим. При лікуванні дітей з ГПП слід уникати агресивного навантаження рідинною у осіб з ознаками порушення серцевої діяльності.

Ключові слова: гостра позалікарняна пневмонія; діти; артеріальний тиск; ЧСС; серцевий викид.

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