Review

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Necrotizing enterocolitis in newborns: description, diagnosis, treatment, suggestions (literature review)

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ABSTRACT. This review presents current data on the diagnosis and intensive care of necrotizing enterocolitis in neonates from the perspective of an anesthesiologist-intensivist. The disease is considered a multifactorial process, most common in preterm infants. The main risk factors are described, including early antibiotic use, formula feeding, and hemodynamic disturbances. Criteria for patient transfer to the intensive care unit are systematized based on multiple organ dysfunction scores and ultrasound findings. The principles of fluid management, inotropic and respiratory support, antibiotic therapy, as well as approaches to parenteral nutrition and coagulopathy correction, are detailed. Special attention is paid to indications for surgical treatment, resection techniques, and peritoneal drainage. Practical suggestions for optimizing the management of this patient population are formulated, including the use of dalargin, prolonged epidural analgesia, and low-molecular-weight heparins.

KEYWORDS: *necrotizing enterocolitis, neonates, intensive care*

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Некротизирующий энтероколит новорожденных: описание, диагностика, лечение, предложения (обзор литературы)

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РЕЗЮМЕ. В обзорной статье представлены современные данные о диагностике и интенсивной терапии некротизирующего энтероколита у новорожденных с позиции анестезиолога-реаниматолога. Заболевание рассматривается как многофакторный процесс, чаще встречающийся у недоношенных детей. Описаны основные факторы риска, включая раннее применение антибиотиков, искусственное вскармливание и гемодинамические нарушения. Систематизированы критерии перевода пациентов в отделение реанимации на основе шкал полиорганной дисфункции и ультразвуковых данных. Детально изложены принципы инфузионной, инотропной, респираторной и антибактериальной терапии, а также подходы к парентеральному питанию и коррекции коагулопатий. Отдельное внимание уделено показаниям к хирургическому лечению, тактике резекции и дренирования брюшной полости. Сформулированы практические предложения по оптимизации ведения данной категории пациентов, включая применение даларгина, продленной эпидуральной аналгезии и низкомолекулярных гепаринов.

КЛЮЧЕВЫЕ СЛОВА: *некротизирующий энтероколит, новорожденные, интенсивная терапия*

Источник финансирования. Авторы заявляют об отсутствии внешнего финансирования при проведении исследования.

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DEFINITION

Necrotizing enterocolitis (NEC) is a common complication of neonatal pathology; however, in extremely preterm infants, it can be regarded as a distinct clinical entity [1, 2]. It is among preterm infants that NEC occurs most consistently. For instance, NEC is reported in 7–11% of very-low-birth-weight preterm infants in the USA, in 5.1% of preterm infants born at less than 33 weeks of gestation in Canada, in 5.8% of those with a gestational age below 27 weeks in Sweden, and in 3–4% of preterm infants under 32 weeks of gestation in Switzerland [3–5]. Mortality in infants with stage I–II NEC ranges from 20% to 30%, while in surgically treated patients it reaches 50–60% [3, 6, 7].

NEC lacks a clearly defined etiology, and there is no consensus among researchers on the fundamental nature of this disease [8–11]. A broad group of conditions of both iatrogenic and non-iatrogenic origin is recognized as predisposing factors. These include birth weight below 1500 g; formula feeding or the use of cow's and goat's milk; mechanical and osmotic stressors, such as oral and enteral administration of medications and hyperosmolar formulas; diseases and conditions associated with intrapulmonary or extrapulmonary right-to-left blood shunting — for instance, respiratory distress syndrome, pneumonia, patent ductus arteriosus, pulmonary embolism, and cyanotic congenital heart defects — and intestinal capillary hypoperfusion due to low cardiac output, vasoplegic shock, or diastolic dysfunction, the latter most often caused by hypocalcemia [12–20]. It is also necessary to take into account the spasmogenic effects of opiates (morphine, fentanyl) — with the exception of trimeperidine, which acts as a spasmolytic — as well as vasoactive agents that increase oxygen demand (such as catecholamines, methylxanthines (aminophylline), corticosteroids, sildenafil, and ibuprofen) and mechanical ventilation with high positive end-expiratory pressure (PEEP). Each day of antibiotic therapy (ABT) has been shown to increase the risk of NEC by 20%, with an odds ratio (OR) of 1.2 (95% confidence interval [CI], 1–1.5); when the duration of ABT exceeds 10 days, the OR reaches 3.0 (95% CI, 1.5–3.3). A well-known adverse effect of protected semi-synthetic penicillins, and to a lesser extent cephalosporins, is the development of pseudomembranous colitis, i.e., the provocation of NEC [21, 22]. Factors that reliably reduce the risk of NEC include feeding with native human milk (both maternal and donor milk) and probiotics containing Lactobacillus and Bifidobacterium species [13, 18, 23]. Lactoferrin reduces the risk of both NEC and late-onset neonatal sepsis [2, 3, 24]. Early pharmacological closure of the ductus arteriosus with ibuprofen is traditionally regarded as beneficial; at the same time, however, the negative impact of this drug on cerebral, pulmonary, and intestinal blood flow is emphasized, and data on the long-term consequences of its use remain lacking [3, 16, 17, 25–27].

The clinical picture consists of a combination of systemic toxic symptoms (lethargy, somnolence, poikilothermia, episodes of apnea, and a tendency toward bradycardia and arterial hypotension) and gastrointestinal manifestations, including abdominal distension (70–98%), increased gastric residual volume (>70%), regurgitation, vomiting (>70%), gastrointestinal bleeding (22–59%), and diarrhea (4–26%) [28–30].

The stages of NEC are defined by Bell's classification as modified by Walsh and Kliegman [31], supplemented by the ultrasound (US) findings presented below [32–36].

Stage Ia: suspected NEC. The skin has a mottled appearance; brief episodes of apnea and temperature instability are noted. The abdomen is distended and tender to palpation. Stools are frothy and odorless. Cessation of enteral feeding leads to clinical improvement. US reveals increased intestinal echogenicity.

Stage Ib: suspected NEC. The overall clinical picture is similar to that of stage Ia. Blood may be present in the stool. Radiographic signs include intestinal distension with gas, bowel wall edema, irregularity of intestinal loops, and moderate hepatosplenomegaly. US demonstrates hyperemia, increased perfusion (on Doppler imaging), bowel wall thickening, and increased intestinal echogenicity.

Stage IIa: definite NEC (negative dynamics). Lethargy, hypothermia, muscular hypotonia, and centralization of circulation (shock) develop. Apneic episodes become more frequent and prolonged. Bradycardia is observed. Vomiting is bile-stained, and blood is present in the stool. Intra-abdominal pressure rises: the abdomen is enlarged, the skin is tense ("shiny"), and palpation is painful. Hyperemia of the abdominal wall (Besenreiser's sign) is noted. Hepatosplenomegaly is pronounced. Radiographic signs include a progressive increase in intestinal gas; paresis with fluid levels;

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worsening bowel wall edema; moderate pneumatosis; and separation of intestinal loops in the presence of free fluid in the abdominal cavity. In addition to the previously described signs, US may reveal free gas in the portal area of the liver. Thinning of the bowel wall and decreased perfusion indicate pre-perforation. In the absence of adequate intensive care, this stage progresses within several hours to stage IIb and subsequently to stage III.

Stage IIb: NEC with physiological deterioration. Bradycardia, heart failure, metabolic acidosis, thrombocytopenia, jaundice, oliguria, somnolence or stupor, and muscular atonia develop. Edema of the abdominal wall is markedly pronounced. Any touch is painful. The bowel contains frothy stool, but defecation is absent. Radiographic signs include an increase in the amount of free fluid in the abdominal cavity with separation of intestinal loops, pronounced pneumatosis, and obliteration of the preperitoneal fat line. Ultrasound (US) findings are identical. Stage IIb is rapidly progressive and requires emergency stabilization of vital functions.

Stage IIIa: advanced NEC without perforation. Peritonitis develops as a result of increased intestinal wall permeability and loss of the intestinal barrier (glycocalyx); the abdomen is extremely distended and markedly tense, with visible contours of intestinal loops, ecchymoses, and severe hyperemia of the abdominal wall. The patient's condition is critical: sepsis, vasoplegic and cardiogenic shock, and multiple organ dysfunction syndrome (MODS) are present.

Stage IIIb: advanced NEC with perforation. Pneumoperitoneum and gangrene are observed. Radiographic signs include significant hepatosplenomegaly, a reduced bladder size, a marked increase in the amount of free fluid and pneumatosis of the intestine and portal vein area, with a corresponding decrease in the amount of free gas.

DIAGNOSIS

The objective physical signs of NEC have been described above. It should be emphasized that, when NEC is suspected, palpation of the abdomen during physical examination must be avoided. Regular surgical consultations are mandatory. Other investigations include measurement of abdominal circumference every 2 hours; ultrasound (US); a complete blood count, with particular attention paid to thrombocytosis, leukocytosis, and the neutrophil-to-lymphocyte ratio (not exceeding 5.5); serum electrolytes; acute-phase proteins — C-reactive protein, fibrinogen, and transthyretin (a dynamic decrease in which indicates depletion of energy reserves required to counteract inflammation); creatinine; urea; blood gas analysis and acid – base status parameters; bacteriological studies; and a coagulation profile [37–42].

TREATMENT (NON-SURGICAL)

Intensive care includes nil per os, gastric decompression, total parenteral nutrition, antibiotic therapy, inotropic and respiratory support, correction of fluid and electrolyte balance, management of coagulopathies and thrombocytopenia, as well as infrequently used and rarely employed measures, such as the administration of lactulose and dalargin.

ELABORATION AND DISCUSSION OF THE ABOVE POINTS

1. Cessation of feeding produces a clear positive effect when NEC is suspected (stage Ia) [2, 3, 43]. As the process progresses, the beneficial role of fasting is reduced to the prevention of abdominal distension and the intra-abdominal hypertension caused by it. However, prolonged fasting (exceeding 24 hours) in a preterm newborn is a risk factor for intestinal atrophy, since the small intestine receives 50% of its nutrition from its lumen, and the large intestine receives 80% [44–46]. Atrophy of the intestinal wall leads to degradation of the glycocalyx and facilitates the translocation of intestinal flora in the setting of an immature biocenosis, i.e., it provokes septic complications [1, 24]. For this reason, some researchers, in the absence of gas in the portal area of the liver, permit trophic feeding starting with a semi-elemental formula, with subsequent transition to maternal or donor milk, up to and including the development of stage IIa [41, 46, 47]. In the parenteral nutrition program, it is essential to adhere to the following conditions. Total energy supply should not exceed 50 kcal/kg per day and should be achieved gradually (over 7–9 days), with a lipid-to-glucose ratio of 1:1.5 (40% of energy supply provided by third-generation lipid emulsions — the agent of choice being SMOFlipid — and 60% by glucose). Amino acid mixtures (Aminoven Infant, Aminoplasma-Ped) are dosed according to the principle of 100 kcal per 1 g of amino nitrogen or 6.25 g of amino acids,

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while for newborns of very low and extremely low birth weight who require increased protein supplementation, the energy supply may be reduced to 80 kcal per 1 g of amino nitrogen [48–50]. Prevention of cholestasis requires the maintenance of gastrointestinal motility: perianal mechanical stimulation; the administration of hypertonic microenemas in consultation with the surgeon; and, as a priority, continuous epidural anesthesia (the technique of choice being caudal anesthesia with 0.2% ropivacaine) [51, 52]. Gastric decompression is continuous: the tube is not clamped, it is looped, and the free end is positioned above the zero reference point (the manubrium of the sternum). The administration of probiotics containing Lactobacillus and Bifidobacterium species is always indicated.

2. Antibiotic therapy. Despite the fact that NEC begins as an aseptic process, reduced microcirculation destroys the intestinal barrier (glycocalyx), leading to the appearance of microcroses; degradation of the glycocalyx facilitates the translocation of intestinal flora; and microbial colonization of the intestine outpaces the development of primary immune responses, which dictates the need for early initiation of antibiotic therapy in stages I–II. No clear, objectively substantiated recommendations exist regarding the choice of antibiotics [3]. Traditionally, antibiotic therapy in newborns is often initiated with protected semi-synthetic penicillins as monotherapy or in combination with an aminoglycoside. However, residual clavulanic acid, sulbactam, avibactam, and cilastatin carry a risk of inducing pseudomembranous colitis and disinhibiting clostridial intestinal flora, and Clostridium difficile is recognized as an etiological factor in toxic megacolon. For this reason, a combination of an aminoglycoside with a third- or fourth-generation cephalosporin appears more justified, with metronidazole added when additional anti-anaerobic activity is required. As an alternative, carbapenem monotherapy (meropenem or doripenem) is recommended. In stage I, the course of antibiotic therapy should be short (ideally 72 hours), whereas in stages II–III it should last 10–14 days. Probiotics are not administered during antibiotic therapy. The issue of combined antibiotic therapy remains without evidence-based solutions. Two, three, and sometimes up to five agents are frequently prescribed based on the susceptibility (more precisely, the minimal inhibitory concentration) of microorganisms isolated from non-sterile sites, without consideration of pharmacological and bacteriological antagonism [53, 54].

3. Inotropic support. While the goal of inotropic support in the setting of NEC is the restoration of capillary perfusion in the gastrointestinal tract, the primary task to be accomplished in achieving this goal is an increase in cardiac stroke volume (a direct inotropic effect; if its efficacy is insufficient, a vasoconstrictor effect is additionally recruited) [55–59]. In doing so, it is important not to raise peripheral vascular resistance above a certain critical

Table 1. Effects of vasoactive drugs

Таблица 1. Эффекты вазоактивных медикаментов

Препараты (рецепторы) Drugs (receptors)	СИ CI	ЧСС HR	САД MAP	ОПСС TVR	ЛСС PVR	Потребность миокарда в O_2 Myocardial O_2 demand
Добутамин (α-1; β-1,2) Dobutamine (α-1; β-1,2)	++	++	+	–	–	++
Допамин (α-1; β-1,2; D) Dopamine (α-1; β-1,2; D)	+	+	++	++	+	+
Адреналин (α-1; β-1,2) Epinephrine (α-1; β-1,2)	++	++	++	+++	+++	+
Норадреналин (α-1; β-1) Norepinephrine (α-1; β-1)	+/–	–	++	+++	+++	++
Милринон (α-1) Milrinone (α-1)	+	+/–	+/–	–	–	+/–

The table was compiled by the authors

Таблица составлена авторами

Note: PVR — pulmonary vascular resistance; TVR — total vascular resistance; MAP — mean arterial pressure; CI — cardiac index; HR — heart rate.

Примечание: ЛСС — легочное сосудистое сопротивление; ОПСС — общее периферическое сопротивление сосудов; САД — среднее артериальное давление; СИ — сердечный индекс; ЧСС — частота сердечных сокращений.

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value, which, unfortunately, has not been precisely defined. Table 1 presents the qualitative characteristics of catecholamines and milrinone.

At the initiation of inotropic support, the oxygen extraction index (O2ER) should be quantitatively determined [60, 61]:

$$O_2ER = (SpO_2 - SvO_2)/SpO_2,$$

where SpO_2 is the oxygen saturation of hemoglobin in the pulsatile blood flow (which constitutes 97–99% of SaO_2) [62], and SvO_2 is the oxygen saturation of hemoglobin in venous blood.

The normal range of this index is from 0.22 to 0.35. Values ≤0.2 indicate an excessive oxygen supply or, less commonly, hypometabolism that reduces the efficacy of vasopressors, i.e., they reflect the need to reduce or discontinue oxygen supplementation; values above 0.4 invariably reflect hypercatabolism and may require deepening of sedation and an increase in the fraction of inspired oxygen (FiO_2). In the absence of milrinone, the agent of choice is dobutamine, which does not increase systemic vascular resistance (SVR; also referred to as total vascular resistance, TVR, in Table 1), improves perfusion of the pulmonary circulation and overall capillary perfusion, at an initial dose of 5 μg/kg/min, with possible escalation to 10–15 μg/kg/min [45, 63]. Dopamine at doses of 5 to 10 μg/kg/min acts similarly to dobutamine but increases mean arterial pressure (MAP) by raising SVR (enhancing myocardial contractility to a lesser extent), whereas dobutamine increases cardiac output with virtually no effect on SVR. The target parameter for assessing the efficacy of vasoactive agents is mean arterial pressure, which in full-term newborns should be no lower than 40 mmHg and no higher than 55 mmHg. In extremely preterm infants, the lower acceptable limit of MAP is 35 mmHg and the upper limit is 50 mmHg. If MAP remains unstable under the action of dobutamine or dopamine, the regimen is modified [1, 2, 44, 56]. Two options are possible, based on the physiological effects of vasoactive medications:

1) epinephrine from 0.05 to 1–2 μg/kg/min (does not require combination with other agents);
2) norepinephrine 0.2–2 μg/kg/min + dobutamine.

During inotropic and vasopressor support, one should aim for the gradual but earliest possible discontinuation of catecholamines.

4. Respiratory support. NEC, even at stage I, requires respiratory support, since increasing intra-abdominal pressure restricts tidal volumes and promotes atelectasis of the lower lobes of the lungs, compensatory increasing the work of breathing. The reduction in the respiratory surface area decreases the ventilation-perfusion (V/Q) ratio to <0.8, thereby increasing the shunt of deoxygenated blood into the systemic circulation. Furthermore, NEC rarely begins to develop against a background of full health but rather emerges as a complication of a wide variety of pathologies in the neonatal period. The principle that, in any neonatal pathology, the hemodynamic, external respiratory, and digestive systems are universally affected has been confirmed by the work of N.N. Volodin [64] and A.G. Antonov [65].

Non-invasive respiratory support, most commonly in the form of continuous positive airway pressure, may delay the need to transition the newborn to volume-controlled mechanical ventilation but may also serve as a factor in increasing intragastric and subsequently intra-abdominal pressure. When the latter rises above 10 mmHg, the conditions for adequate renal perfusion and capillary perfusion in the celiac trunk region deteriorate [45, 63, 66]. For this reason, early transition of patients with NEC to invasive respiratory support is considered advisable [67, 68].

High-frequency oscillatory ventilation (HFOV), which is gaining popularity, holds promise for lesions of the pulmonary parenchyma and interstitium, since active expiration guarantees the absence of auto-PEEP and reduces the risk of dysatelectasis, barotrauma, and volutrauma [30]. However, in the presence of elevated intra-abdominal pressure and reduced splanchnic blood flow, there is no physiological rationale or sufficient evidence for the superiority of HFOV over the more conventional convection (volume-controlled) ventilation [69, 70]. In particular, the effect of high oscillation frequency (≥5 Hz) on perfusion blood flow in the celiac trunk region has not been studied.

Among the specific aspects of volume-controlled ventilation in newborns with NEC, the need to overcome hypoventilation of the segments of the lower third of the lungs resulting from intra-abdominal hypertension should be noted. In these segments, the ventilation-perfusion (V/Q) ratio is substantially lower than 0.8, and during a rapid inspiration, a significant proportion of alveoli and bronchioles do not participate in gas exchange, thereby increasing the

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capacity of the functional dead space [44, 45, 67, 71]. By the term "rapid inspiration," we denote the inspiratory-to-expiratory time ratio (Ti:Te) routinely used in most neonatal intensive care units, i.e., a ratio of 1:3–1:4 [72, 73]. To counteract this effect, elevated PEEP and FiO_2 are routinely employed, which exert opposing influences on CO_2 exchange. A high FiO_2, by stimulating the Haldane effect, enhances the "washout" of CO_2 from erythrocytes in venous blood, whereas PEEP impedes its alveolar elimination [67, 68, 74]. As a result, both hypercapnia at normal or reduced respiratory rates and hypocapnia at elevated respiratory rates are frequently recorded. The PEEP level also affects the perfusion of pulmonary capillaries, the perfusion pressure in which does not exceed 5–7 mmHg (6.7–9.4 mbar) in extremely preterm infants, while an FiO_2 above 0.6 promotes cerebral vasodilation, increasing the risk of hemorrhages and periventricular leukomalacia. The Ti:Te ratio, as shown in Table 2, depends on the respiratory rate per minute [67, 68, 73]. At a fixed inspiratory time (usually 0.3±0.05 s), a respiratory rate of 40 breaths per minute yields a Ti:Te of 1:4, at 50 per minute — 1:3, at 65 per minute—1:2, and only at 80 per minute does it correspond to the physiological ratio for quiet breathing — 1:1.5 [44].

In view of the above, the initial ventilation parameters required to establish and maintain an "open lung" in a newborn with NEC (especially those weighing less than 1500 g) may be presented as follows:

- tidal volume (Vt) not exceeding 4–5 mL/kg (in the absence of signs of acute respiratory distress syndrome [ARDS], it may be gradually increased to 6–7 mL/kg);
- circuit flow (flow) 3–4 L/min (up to 5–6 L/min when a heat and moisture exchange filter is used);
- respiratory rate (RR) 45–50 breaths/min;
- inspiratory time 0.67–0.6 s (Ti:Te—1:1), with gradual reduction (as SpO_2 normalizes) to 0.53–0.48 s (Ti:Te—1:1.5);
- peak inspiratory pressure (PIP) 18–22 mbar;
- PEEP 3–5 mbar;
- FiO_2 at the minimum level required to achieve SpO_2 of 88–94%, preferably not exceeding 0.4;
- maintain the following parameters within the specified ranges: O_2ER 0.22–0.4; SpO_2 88–94%; arterial oxygen tension (PaO_2) 57–75 mmHg; venous oxygen tension (PvO_2) 35–45 mmHg; venous carbon dioxide tension ($PvCO_2$) 35–50 mmHg.

5. Correction of fluid and electrolyte balance. In the early neonatal period, by analogy with the general principles of intensive care, infusion therapy is considered a priority anti-shock measure. However, no physiologically substantiated volumes or rates of infusion solution administration

Table 2. Dependence of inhalation time on respiratory rate and the ratio of “inhalation time : exhalation time”

Таблица 2. Зависимость времени вдоха от частоты дыханий и соотношения «время вдоха : время выдоха»

Частота дыхания в минуту Respiratory rate in minute	Ti + Te (с) Ti + Te (s)	Соотношение Ti:Te Ti:Te ratio							
		1,2:1	1:1	1:1,25	1:1,5	1:2	1:2,5	1:3	1:4
20	3	1,64	1,5	1,33	1,2	1	0,86	0,75	0,6
25	2,4	1,31	1,2	1,1	0,96	0,8	0,69	0,6	0,48
30	2	1,1	1	0,89	0,8	0,67	0,57	0,5	0,4
35	1,7	0,93	0,85	0,76	0,68	0,57	0,49	0,43	0,34
40	1,5	0,82	0,75	0,67	0,6	0,5	0,43	0,38	0,3
45	1,33	0,73	0,67	0,59	0,53	0,44	0,38	0,33	0,27
50	1,2	0,65	0,6	0,53	0,48	0,4	0,34	0,3	0,24
55	1,1	0,6	0,55	0,49	0,44	0,37	0,31	0,28	0,22
60	1	0,55	0,5	0,44	0,4	0,33	0,29	0,25	0,2
65	0,92	0,5	0,46	0,41	0,37	0,31	0,26	0,23	0,18
70	0,86	0,47	0,43	0,38	0,34	0,29	0,25	0,22	0,17
80	0,75	0,41	0,38	0,33	0,3	0,25	0,21	0,19	0,15

The table was compiled by the authors

Таблица составлена авторами

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exist for the neonatal period. For example, the algorithm for resuscitation in the delivery room of a newborn with an Apgar score of 0 prescribes, after successful resuscitation, the intravenous administration of isotonic sodium chloride solution at 10 mL/kg over 10 minutes (i.e., at a rate of 60 mL/kg per hour!) [75]. In the absence of intravascular volume loss and with restored pump function of the cardiac chambers, such an emergency rehydration regimen is regarded as a risk factor for pulmonary circulatory overload and acute kidney injury [76–79]. Moreover, the absence of the newborn's first breath implies the absence of evacuation of fetal extravascular lung water, i.e., hyperhydration of the pulmonary interstitium. The combination of a high total body water content in the newborn, a large relative amount of extracellular water (40% of body weight), and rapid recirculation (at least 25 circulating blood volumes per day) gives rise to lability of the extracellular compartment. It follows that the danger of hypervolemia of the pulmonary circulation due to rapid infusion combined with pulmonary interstitial edema exceeds the danger of dehydration and hypovolemia [76, 77, 80, 81]. Hypovolemia in newborns, especially preterm infants, is, as a rule, a consequence not of blood loss but of capillary plasma leakage, and massive infusion is highly likely to exacerbate this leakage. In preterm newborns, already at the onset of NEC development, the high rate of water recirculation and the relative weakness of the left cardiac chambers create a risk of volume overload of the pulmonary circulation, intestinal edema, and acute kidney injury [82–84]. Thus, the principle of judicious restriction of infusion volumes and rates in newborns at risk of NEC, and even more so in those with an established process, appears logical.

The infusion therapy program is as follows.

1. Emergency rehydration: 10 mL/kg per hour until a mean arterial pressure of 40 mmHg is achieved (35 mmHg in newborns with a body weight less than 1500 g) and capillary refill time is ≤3 s, but for no longer than 2 hours.

2. Maintenance rehydration: the physiological requirement on the first day of life is no more than 60 mL/kg, with a gradual increase in daily volumes to 135 mL/kg (for infants with a birth weight of at least 1500 g) or to 150 mL/kg (for preterm infants with a birth weight less than 1500 g) over 12–14 days. Perspiration losses in open systems cannot be precisely determined (ranging from 1 to 2.5 mL/kg per hour); in an incubator with a relative humidity above 75%, perspiration is negligible [70].

3. Composition: isotonic Sterofundin (Plasmafusol) in a volume that supplements the deficit of oral or enteral feeding. If intravenous administration of medications is necessary, they are accounted for in the total rehydration volume.

4. Monitoring: determination of body weight at least twice daily with recording of the mean value (during the first 5–7 days, a weight loss of 1% of birth weight per day indicates a normal rate of rehydration; from the eighth day onward, a weight gain in the same order should be observed) and a urine output rate of no less than 0.5 mL/kg per hour up to two days of life, and no less than 1 mL/kg per hour thereafter. Additional monitoring criteria include the absence of signs of right heart overload on electrocardiography/echocardiography and the absence of signs of interstitial pulmonary edema, pneumatosis, and progressive thickening of the intestinal walls on ultrasound [85–88]. Along with monitoring of water balance, it is necessary to monitor the levels of major ions in the blood plasma. Heart rate variability analysis is rarely used but provides highly valuable information, reflecting the balance of the autonomic nervous system [55, 78, 89].

6. Management of coagulopathies and correction of thrombocytopenia. In the neonatal period, factors that create a risk of both thrombosis and embolism and paradoxical bleeding are combined. The most important of these are: tissue factor (thromboplastin); antithrombin III deficiency and an insufficient rate of synthesis of its cofactor, heparin; insufficient functional intensity of the cytochrome P450 system and the absence of an intestinal biocenosis, both of which are necessary for the activation of phytomenadione; a high hematocrit; and biliary system overload with a deficiency of glucuronic acid, which is engaged in the detoxification of bilirubin [90]. Microthrombosis of the capillaries of the mesentery and intestinal villi is an important factor in the progression of NEC. The generally accepted class of drugs for the prevention of thrombotic complications is low-molecular-weight heparins (LMWHs). Subcutaneous administration of these agents is unacceptable in the neonatal period; therefore, the only method of LMWH administration in newborns is continuous intravenous infusion. These drugs should preferably be used prophylactically in all newborns with manifestations of infectious processes or at risk of infectious lesions,

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harnessing the following effects. These include blockade of TNFα expression, damping of platelet adhesive function, and an increase in the optical density of lipoproteins, which collectively reduce the activity of the inflammatory cytokine cascade and the risk of cholestasis. The daily dose of nadroparin or enoxaparin (anti-Xa to anti-IIa activity ratio of 4:1) may be 150–200 IU (anti-Xa)/kg or 2000 IU/m^2; however, for preterm infants, weight-based doses are safer. For dalteparin, the dose is lower (125–150 IU/kg), since the anti-Xa to anti-IIa activity ratio for this agent is 2:1. Monitoring is performed 2–4 times per week (thrombocytosis, international normalized ratio [INR], D-dimer, bleeding time). Thrombocytopenia does not necessarily correlate with a reduction in the activity of vascular-platelet hemostasis, since platelets can break down into microvesicles whose adhesive properties exceed the activity of intact platelets [90, 91]. The administration of platelet concentrate is indicated only in cases of overt bleeding against a background of profound thrombocytopenia. The classical regimen for arresting hemorrhage in hemorrhagic disease of the newborn — transfusion of fresh citrated blood and administration of phytomenadione (konakion) — is also relevant for NEC. In cases of massive bleeding, tranexamic acid (20–100 mg/kg with a maintenance infusion of 10 mg/kg per hour), prothrombin complex concentrate (25 IU/kg), and Coagil-VII (90 μg/kg with a maintenance infusion of 20 μg/kg per hour for up to 12 hours) are additionally employed. These agents are not used prophylactically.

7. Infrequently used and rarely employed measures. The use of lactulose is theoretically promising as a choleretic agent and a factor that enhances the activity of lactic acid flora and acidifies the contents of the large intestine. This reduces the toxicity of ammonia (i.e., promotes its migration into the intestinal lumen and conversion to ammonium) and diminishes the activity of opportunistic flora, including anaerobic species. The hyperosmolarity of lactulose syrup (concentration 66.7%) must be taken into account; therefore, the daily dose (depending on the child's body weight, from 3 to 5 mL) should be divided into four administrations via a nasogastric tube, with each portion diluted 5- to 10-fold. This means the preparation can be used during trophic feeding but not in fasting patients.

The use of dalargin, a Russian-developed drug created as a stress protector for the prevention and treatment of stress ulcers of the gastrointestinal tract, is highly effective. Dalargin is a synthetic analogue of leu-enkephalin and is used in general anesthesia regimens as an opioid adjuvant, allowing a 30% reduction in opioid consumption [92, 93]; its use in the neonatal period has been described in rare publications [94–96]. Other proven properties of dalargin include antioxidant and antihypoxant effects, and it prolongs the half-life of surfactant [93]. The estimated dose is 0.2–0.4 mg/kg per day, administered as a continuous infusion over 2–4 days.

SURGICAL TREATMENT

When there is doubt as to the necessity of laparotomy, laparoscopy is performed. In the presence or with a high probability of peritonitis, a palliative intervention — laparocentesis and drainage of the abdominal cavity — is indicated. Depending on the nature of the exudate obtained, the indication for extending the surgical intervention is determined. After revision of the abdominal organs, economical (local) resection of the affected segment of the intestine with the creation of a double enterostomy or colostomy (or multiple stomas) is most expedient. The rationale for this tactic lies in preserving intestinal functional activity sufficient for growth and development [97]. Staged closure of the intestinal stomas is generally considered feasible 1–5 months after the initial operation. In the presence of isolated perforations (1–2 ulcers), suturing of the ulcerative defects with a two-layer suture is performed. Resections of necrotized intestinal segments are completed by the formation of primary anastomoses. A combined surgical tactic (laparostomy in combination with intestinal stomas) is used in the presence of multiple, mosaically distributed subserosal necroses; the use of an isolated laparostomy is also justified. The placement of a polyvinyl chloride micro-irrigator, 0.2 to 0.4 cm in diameter, through a separate skin puncture is indicated. This allows for the administration of an antibiotic and a 0.5% metronidazole solution into the area of the local purulent-inflammatory focus. This method of passive sanitation is used for 3–4 days after surgery [98, 99].

SUGGESTIONS BASED ON THE ABOVE

1. Newborns with suspected NEC (stage Ia) should be managed in the intensive care unit (ICU) when the sum of scores on the following scales increases by 2 points per day from a baseline of

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Table 3. Quantitative assessment of indications for transfer of a newborn with suspected necrotizing enterocolitis to the pediatric intensive care unit

Таблица 3. Количественная оценка показаний для перевода новорожденного с подозрением на некротический энтероколит в педиатрическое отделение реанимации и интенсивной терапии

Признаки **Signs**	**Баллы** **Points**		
	0	**1**	**2**
Кожные покровы Skin	Розовые Pink	Мраморность Mottling	Цианоз Cyanosis
Ритм дыхания, частота дыхательных движений в минуту Respiratory rate, respiratory rate per minute	40–60	61–80	>80 или апноэ >80 or apnea
Время наполнения капилляров, с Capillary refill time, s	1–3	4	4
Гепатоспленомегалия Hepatosplenomegaly	Нет No	–	Да Yes
Перистальтика желудочно-кишечного тракта Gastrointestinal peristalsis	Живая Brisk	Спорадическая Sporadic	Усиленная или отсутствует Increased or absent
Прирост окружности живота, см Abdominal circumference increase, cm	0–1	2	>2
Метеоризм Flatulence	Нет No	Умеренный Moderate	Выраженный Severe
Застой в желудке Gastric congestion	Нет No	–	Да Yes
Кал Stool	Обычный Normal	Жидкий Liquid	Пенистый или отсутствует Foamy or absent
Кровотечение из желудочно-кишечного тракта Gastrointestinal bleeding	Нет No	Скрытая кровь Occult blood	Явное Obvious
Рвота (срыгивание) Vomition (regurgitation)	Нет No	1–2 раза за сутки 1–2 times per day	>2 раз за сутки >2 times per day
Данные ультразвукового исследования Ultrasound data	Норма Normal	Повышенная эхогенность кишечника Increased intestinal echogenicity	Пневматоз, утолщение стенки кишки, билиопневматоз Pneumatosis, intestinal wall thickening, biliopneumatosis

The table was compiled by the authors

Таблица составлена авторами

Note: 0–2 points — no translation required; 3 points — translation subject to agreement with the head of the intensive care unit; 4 points — translation required.

Примечание: 0–2 балла — перевод не требуется; 3 балла — перевод по согласованию с заведующим отделением реанимации и интенсивной терапии; 4 балла — перевод обязателен.

0 points [100–105]. These scales include NEOMOD (Neonatal Multiple Organ Dysfunction Score, 1999), pSOFA (pediatric Sequential Organ Failure Assessment, 2017), and aSOFA (adaptive Sequential Organ Failure Assessment, 2007). Table 3 is proposed for the formalization of indications.

2. The following should be mandatory components of monitoring and intensive care:

- daily ultrasound of the abdominal cavity;
- continuous epidural anesthesia (alternatively, caudal anesthesia) with ropivacaine;
- the use of dalargin as a stress protector;
- prophylaxis of thromboembolic complications with low-molecular-weight heparin fractions.

3. Stratification and standardization of antibiotic therapy regimens are necessary:

- monotherapy or combination therapy with two (at most, three) agents;
- if any component of a combination is deemed ineffective, the entire combination should be replaced;

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- a judicious approach to microorganisms contaminating non-sterile body regions or isolated from non-sterile sites as potential pathogens;
- the development of and adherence to a protocol for probiotic administration.

4. If prolonged analgosedation is required, the routinely used spasmogenic opioid (fentanyl) should be replaced with an agent possessing spasmolytic properties (trimeperidine) or, by decision of the medical committee, with dexmedetomidine. The prescribing information for dexmedetomidine does not contain a formal contraindication for use in children of any age.

5. Discontinue the use of agents that remain the subject of debate in the literature — sildenafil, cholecalciferol, vikasol, and octreotide.

ADDITIONAL INFORMATION

Authors' contribution. A.N. Shmakov — article concept development, article writing; K.V. Budarova — article visualization, editing, and approval.

All authors made a substantial contribution to the conception of the study, acquisition, analysis, interpretation of data for the work, drafting and revising the article, final approval of the version to be published and agree to be accountable for all aspects of the study.

Competing interests. The authors declare that they have no competing interests.

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Все авторы внесли существенный вклад в разработку концепции, проведение исследования и подготовку статьи, прочли и одобрили финальную версию перед публикацией.

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