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ETIOPATHOGENETIC FEATURES OF THE DEVELOPMENT OF SYNDIALYSIS HYPOTENSION IN PATIENTS RECEIVING HEMODIALYSIS

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Resume

Studies indicate that in the context of the use of modern technologies of renal replacement therapy, the classical concepts of risk factors for the development of syndialysis hypotension require revision. The review presents studies on the pathogenetic factors of syndialysis hypotension and clinical guidelines for the prevention of syndialysis hypotension.

Key words: syndialysis arterial hypotension, chronic renal failure, hydration, body composition monitor, water status, hemodialysis.
Relevance

CKD is a concept of kidneys of various nature. The reasons for the isolation of this concept are based on the unity of the main pathogenetic mechanisms of the progression of the pathological process in the kidneys. There is a close pathophysiological relationship between CKD and CVD; there is a commonality of many risk factors for the development and progression of diseases. The introduction of the concept of CKD into health care practice should be considered as an important strategic approach to reducing CV and overall mortality, and increasing the life expectancy of the population.

CKD screening should take an important place in the structure of prevention of both renal and cardiac pathology, and improving nephroprotection measures will also lead to a decrease in CV mortality. The main cause of death in patients with chronic renal failure receiving treatment with programmed hemodialysis is cardiovascular diseases and associated complications [1]. One of the problems of programmed hemodialysis is syndialysis hypotension, which complicates hemodialysis in 10-50% of cases [2], and is a prognostically unfavorable factor that determines the increased mortality of patients on hemodialysis [3]. Numerous large-scale studies (ACCOMPLISH, ADVANCE, ALTITUDE, CARRESS-HF, ONTARGET, ROADMAP) have confirmed that CKD has a fairly high prevalence (10-15% of the population) and occupies one of the leading places in the overall structure of mortality and morbidity in the population. Along with such diseases as ischemic heart disease (CHD), hypertension, diabetes mellitus [4], about 14 million people in Russia suffer
from CKD. Kidney disease as a direct cause of death is ranked 11th, and as a cause of disability - 17th place [5]. The kidneys are involved in the pathological process in a wide range of diseases - diabetes mellitus, arterial hypertension, ischemic heart disease and others [6]. At the same time, renal dysfunction is a strong independent risk factor for the unfavorable course and outcome of patients with cardiovascular pathology, which is explained by the commonality of their pathophysiological mechanisms. The peculiarity and danger of CKD is that patients do not present any specific complaints for a long time and do not go to a nephrologist for renal pathology, they are observed for a long time by other specialists, which leads to late diagnosis and ineffectiveness of conservative nephroprotective therapy. Analysis of the results of large population studies (HOPE, PREVEND, LIFE), the work of Butler J, allowed us to identify an independent direct relationship between a decrease in the estimated glomerular filtration rate (eGFR), albuminuria and cardiovascular morbidity, in particular, arterial hypertension, coronary heart disease, chronic heart disease. insufficiency [7]. About 40-80% of pre-dialysis CKD patients have CVD. The risk factors for the development of CKD largely coincide with the risk factors for CVD, which means that the main directions of nephro- and cardioprotection also coincide: normalization of blood pressure, decrease or reverse development of albuminuria, correction of carbohydrate metabolism disorders, correction of anemia, dyslipidemia and calcium-phosphorus metabolism, a low-salt diet, the fight against obesity and smoking, as well as the prevention of an acute decline in kidney function and the fight against hyperkalemia. Hemodialysis is a high-tech procedure involving the use of multicomponent equipment, extracorporeal circulation, correction of the water-electrolyte balance, changes in the acid-base state and osmolar balance. In this regard, hemodialysis is accompanied by various complications. Syndialytic hypotension is the result of an inadequate response of the cardiovascular system to a decrease in circulating blood volume, which occurs during the removal of a large volume of fluid from the body in a short period of time. During a typical dialysis procedure, the volume of ultrafiltrate removed is often equal to or greater than the volume of circulating plasma. Despite the large volume of
ultrafiltrate, the plasma volume is usually reduced by only 10 - 20%. This ability to maintain plasma volume during ultrafiltration is provided by the mobilization of fluid from the interstitial to the intravascular space. The replenishment of the intravascular volume is influenced by both the individual characteristics of the patient himself and the factors associated with drug therapy received by this patient, which affect the distribution of water between the sectors of the body [8,9,10]. Patients on maintenance hemodialysis develop both structural and functional cardiovascular disorders. Despite improvements in dialysis technology, cardiovascular mortality remains high in this population. The pathophysiological mechanisms of these changes are complex and poorly understood. It has been suggested that several unconventional risk factors associated with uremia, especially long-term uremic conditions, may affect the cardiovascular system. In chronic kidney disease, many cardiovascular changes occur, including left ventricular hypertrophy, myocardial fibrosis, microvascular disease, accelerated atherosclerosis, and arteriosclerosis. These structural and functional changes in chronic dialysis patients make them more susceptible to myocardial ischemia. By itself, hemodialysis can adversely affect the cardiovascular system due to non-physiological fluid removal, which leads to hemodynamic instability and the occurrence of systemic inflammation. In the last decade, there has been a growing understanding that pathophysiological mechanisms cause cardiovascular dysfunction in chronic dialysis patients [8,9].

Syndialysis hypotension is one of the most common complications. The development of syndialysis hypotension is a polyetiological pathological process with a complex pathogenesis. Among the reasons for the development of IDH, first of all, it is advisable to single out those that are directly related to the hemodialysis procedure. The blood pressure level may depend on the temperature of the dialysate [8,9]. During a hemodialysis procedure using a dialysis solution of standard temperature (37–37.5 °C), the activity of the sympathetic nervous system increases (due to a decrease in circulating blood volume), which leads to vasoconstriction of skin vessels and an increase in body temperature [10,12]. Subsequently, peripheral vasodilation occurs,
which increases the risk of IDH development [13, 14]. The use of dialysate with a temperature below standard leads to an increase in myocardial contractility, an increase in peripheral vascular resistance [13, 15], and a 7.1-fold decrease in the number of IDH episodes [16]. The frequency of episodes of syndialysis hypotension in patients receiving treatment with programmed hemodialysis is interrelated with body mass index, pre-dialysis and post-dialysis blood pressure, the ratio of interdialysis weight gain and ultrafiltration volume to dry weight, and echocardiographic parameters [16,17]. The absolute values of intradialysis blood pressure correlate with the duration of hemodialysis treatment, serum albumin level and interdialysis weight gain, and the indicators characterizing the relative decrease in blood pressure during hemodialysis correlate with the serum concentration of electrolytes (sodium, potassium and calcium) [17]. The amount of interstitial fluid available for replenishing intravascular volume is influenced by the patient's established "dry" body weight. A small volume of interstitial fluid at any volume of ultrafiltration is more likely to be accompanied by hemodynamic instability. This explains the development of hypotension during the hemodialysis procedure in patients whose "dry" weight is set below the true values. On the contrary, an increase in the amount of interstitial fluid will expand the boundaries of the volume available for filling the intravascular space, thereby reducing the likelihood of developing hypotension. In most patients, at present, "dry" weight is adjusted to the minimum amount of interstitial fluid, since chronic volume overload has long-term adverse effects on the cardiovascular system [18]. Determination of "dry" body mass in most cases is carried out empirically, by trial and error. "Lean" weight is defined as body weight below which unacceptable symptoms develop: seizures, nausea and vomiting, or hypotension. In many patients, "dry" weight is very variable and can fluctuate during the addition of intercurrent pathology (for example: diarrhea, infectious diseases), with a change in the blood hematocrit (use of erythropoietin) [19]. The use of dialysate with a high sodium content (more than 140 mmol / L) is an effective way to ensure adequate replenishment of the vascular bed and is rightfully one of the most effective and easily tolerated by patients methods of treatment of
episodes of hypotension. The essence of the technique for profiling sodium in a dialysate solution is to change the sodium concentration during the hemodialysis procedure. The most common form of profiling is to set an initially high sodium concentration, followed by a progressive decrease towards the end of the procedure to an isotonic or even hypotonic level. This method of sodium regulation allows, due to the diffusion of sodium from the dialysate at the very beginning of the procedure, to prevent a rapid decrease in plasma osmolarity (as a result of the removal of urea and other low molecular weight substances). For the remainder of the procedure, when the decrease in osmolarity associated with urea removal is not so pronounced, the sodium in the dialysate should be set at a lower level than the initial level, because a higher sodium concentration in the dialysis fluid leads to thirst, an increase in interdialysis increments and the development of arterial hypertension in the interdialysis period.

Ultrafiltration profiling consists in a deliberate increase in the ultrafiltration level at the beginning of the hemodialysis procedure, when the volume of interstitial fluid available for replenishing the vascular bed is maximum, followed by a gradual decrease in the ultrafiltration level in parallel with the expected decrease in the volume of interstitial fluid. In recent studies, it is recommended to combine UF and dialysate sodium profiling for greater efficiency. Devices that control intradialysis changes in circulating blood volume are promoted as a tool for minimizing syndialysis hypotension. Such devices are based on the concept of the likelihood of developing hypotension, which always occurs when approximately the same volume of blood is removed [20]. Unfortunately, most studies have not found a relationship between changes in the BCC in a single individual and the incidence of hypotension. A prospective randomized trial evaluated the effect of blood volume monitoring during hemodialysis on the incidence of vascular access-related and non-vascular hospitalizations and mortality compared with standard dialysis. The lack of a visible advantage of using blood volume monitoring compared to standard dialysis can partly be explained by the peculiarity of this method of underestimating the true value of the volume to be removed. The method is based on measuring the degree of hemoconcentration that occurs as a result of
ultrafiltration; the degree of mixing of plasma and erythrocytes in the entire circulatory bed is assumed to be the same. Recent studies suggest that this assumption is incorrect. The average hematocrit of the whole body is slightly lower than in arterial or venous blood as a result of an active decrease in hematocrit in the microvasculature, known as the Faraeus phenomenon. During the ultrafiltration process, compensatory mobilization from the microvasculature to the central blood vessels with a low hematocrit probably occurs. Such hemodilution minimizes the degree of hemoconcentration, which potentially leads to an underestimation of the total volume of the ultrafiltrate. [17,18,19].

Blood volume monitoring has been shown to be more effective when incorporated into a biofeedback system, in which dialysate conductance and ultrafiltration levels are continuously adjusted based on incoming data on changes in circulating blood volume. The system has been designed to deliver blood volume reductions on an individual, pre-set schedule to avoid acute and sudden volume reductions that quickly lead to hypotension. It was found that this technique can reduce the frequency of episodes of hypotension and provide more stable blood pressure, both during and after the hemodialysis procedure. In dialysis patients with a tendency to hypertension, this technique can improve blood pressure indicators by optimizing the volemic status, as well as by reducing the frequency of episodes of hypotension compared to the standard procedure [21]. The use of biofeedback systems during the HD procedure, in which the level of ultrafiltration is corrected according to changes in blood pressure measured every 5 minutes, can also reduce the frequency of syndialytic hypotension. This method uses a fuzzy logical blood pressure control system that corrects the ultrafiltration level in accordance with momentary changes in blood pressure in the context of the already available data on the dynamics of the patient's blood pressure during previous procedures [22]. In patients with recurrent episodes of syndialytic hypotension, an inadequate response of the sympathetic nervous system to the removal of circulating blood volume is usually found. Compared with patients with stable hemodynamics in the plasma of patients with intradialysis hypotension, the concentration of chromogranin A (a protein released together with catecholamines)
increases to a lesser extent after dialysis, which correlates the development of hypotension with a decrease in the activity of the sympathetic nervous system. The exact mechanism leading to dysfunction of the autonomic nervous system in uremia is unknown, but chronic hyperkalemic depolarization of nerve fibers plays a role [23]. With a decrease in the tone of the sympathetic nervous system, various vasoconstrictors are most often used to increase the general peripheral resistance. Systematic review of the literature S. Prakash et al. indicates that preference is given to the appointment of 2.5 to 10 mg of midodrine (selective α1 adrenergic agonist), which has proven to be effective in the treatment of some patients with frequent episodes of syndialytic hypotension. This drug preserves effective cerebral circulation to a greater extent in patients with orthostatic hypotension after dialysis, which is an additional beneficial effect. Recent reports have reported the effectiveness of vasopressin in maintaining blood pressure during the removal of circulating blood volume. Previous studies have found that plasma vasopressin levels increase insignificantly during the ultrafiltration process, despite the expected baroreceptor response. In a randomized, double-blind, placebo-controlled study, continuous administration of subpressor doses of vasopressin provided better hemodynamic stability, even when the ultrafiltration volume was increased by 0.5 kg above the initial level (Table 1). In recent years, some success has been achieved in the prevention and correction of syndialysis hypotension. Probably, to minimize this complication, the greatest hopes can be placed on technologies that can correct dialysate composition and ultrafiltration level throughout the HD procedure, based on real-time changes in parameters reflecting vascular replacement. Carrying out the HD procedure in this mode is possible thanks to regulatory systems based on minute-by-minute changes in the parameters of the cardiovascular system in response to ultrafiltration. In practice, the setting of parameters before each procedure is based on the assumption that the reaction of the cardiovascular system will be the same as during the previous procedures. In most cases, this assumption is incorrect due to the dynamic nature of the factors affecting vascular replenishment [24].
### Table 1. Treatment of hemodynamic instability in patients receiving hemodialysis.

<table>
<thead>
<tr>
<th>1. To exclude causes not related to dialysis (myocardial ischemia, noncardiogenic edema, infections)</th>
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<tr>
<td>2. Individual selection of hemodialysis mode:</td>
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<tr>
<td>- precise definition of &quot;dry&quot; body weight</td>
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<tr>
<td>- optimization of dialysate parameters</td>
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<td>- sodium concentration in dialysate is more than 140 mmol /</td>
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<tr>
<td>-bicarbonate dialysis</td>
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<tr>
<td>- avoid low magnesium dialysate</td>
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<tr>
<td>- avoid low calcium dialysate</td>
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<tr>
<td>3. Optimization of the ultrafiltration process:</td>
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<tr>
<td>- profiling of ultrafiltration without or in conjunction with profiling by sodium</td>
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<tr>
<td>- sequential UV and isovolemic dialysis</td>
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<td>-low dialysate temperature</td>
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<td>4. Avoid eating on dialysis</td>
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<td>5. Avoid taking antihypertensive drugs on the day of dialysis</td>
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<td>Intravenous drugs:</td>
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<tr>
<td>- midodrin</td>
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<td>-vasopressin</td>
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<td>- adenosine antagonists</td>
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Syndialysis hypotension (SDH) against the background of high UV rates remains a frequent and potentially dangerous complication of the HD procedure, which worsens the long-term prognosis of HD patients mainly due to an increase in cardiovascular morbidity and mortality. A new method for the prevention and correction of hypotension using ASKD allows a timely decrease in the rate of UV, preventing the development of episodes of hypotension, reducing its frequency and improving the achievement of target BP values for both pre- and post-dialysis BP values, as well as
its syndialysis variations [24]. Simultaneous sodium profiling and ultrafiltration improves hemodynamic stability during hemodialysis [25]. Most often, the principle of so-called "mirror" profiling is used, when an increase in the sodium level in the dialysate corresponds to a high rate of ultrafiltration and vice versa [26]. Hemodynamic stability with mirror-like profiling is achieved through increased sodium concentration in dialysate to increase intravascular volume replenishment during periods of high ultrafiltration rate and decreased dialysate sodium concentration during periods of slow ultrafiltration rate when intravascular volume replenishment is less intense [27]. Thus, simultaneous profiling slows down the rate of decrease in blood volume and reduces the risk of developing subdural hematoma [28]. However, according to the research results of A.G. Strokov and V.A. Terekhova [29], profiling the concentration of sodium ion in dialysate and the rate of ultrafiltration does not significantly affect the dynamics of the relative blood volume during hemodialysis and, accordingly, the number of episodes of subdural hematoma. The development of SDH is a polyetiological pathological process with a complex pathogenesis. Among the reasons for the development of subdural hematoma, first of all, it is advisable to single out those that are directly related to the hemodialysis procedure. The level of intradialysis blood pressure may depend on the temperature of the dialysate [29]. During hemodialysis with standard temperature dialysate solution (37–37.5 °C), the activity of the sympathetic nervous system increases (due to a decrease in circulating blood volume), which leads to vasoconstriction of skin vessels and an increase in body temperature [29,30]. There are a variety of risk factors for the development of IDH: age over 55 years, female sex, diabetes mellitus, heart disease, nitrate therapy, hyperphosphatemia, low predialysis blood pressure (<100 mm Hg) [30]. Rare causes of hypotension on dialysis include: cardiac amyloidosis, pericardial tamponade, myocardial infarction, aortic dissection, internal or external bleeding (gastrointestinal bleeding, adrenal hemorrhage), sepsis, hemolysis, air embolism, pulmonary embolism, reaction to pulmonary embolism solution, pneumothorax, compression of the inferior vena cava (polycystic kidney disease), exudative or constrictive pericarditis, valvular dysfunction (acute mitral regurgitation), cardiogenic shock. some of these reasons are potentially
modifiable, which further enhances the importance of predicting and preventing SDH risk. The pathogenesis of subdural hematoma is a complex and insufficiently studied process in which, under the influence of causes associated with the patient or with the hemodialysis procedure, a decrease in plasma volume and a violation of cardiovascular regulatory mechanisms occur. SDH occurs as a result of changes in factors responsible for maintaining hemodynamic stability: slow movement of fluid from the extravascular space into the vessels (refilling), a decrease in systemic vascular resistance and cardiac output. In HD patients, the indicators of role and physical functioning were significantly reduced, which is associated with the psychological and physical dependence of patients on medical personnel and equipment, and the performance of activities associated with daily physical activity was reduced. A detailed analysis of individual QOL parameters in different types of RRT revealed significant differences in burden scales and the effect of kidney disease on daily activities, social and emotional functioning. Patients on PD rated themselves higher according to these indicators, which is due to less dependence on medical centers [31].

The main mechanism of SDH is a rapid decrease in blood volume due to UV and a decrease in extracellular osmolarity during dialysis [18]. The patient's predisposition to subdural hematoma is largely determined by the rate of movement of fluid from the interstitial space into the vessels and the presence of capillary permeability. The essence of the law of transcapillary exchange of Starling is that the movement of fluid and its accumulation in the intervascular space occurs due to the difference in the absolute gradient of hydrostatic and oncotic pressure, with increased hydrostatic pressure inside the capillaries [32,33]. The rate of replenishment of the vascular space decreases under the influence of Starling forces - hypoalbuminemia, right ventricular heart failure, increased capillary membrane permeability, increased hydrostatic pressure in the capillaries. Widespread use of calcium antagonists from the group of dihydropyridines (nifedipine, amlodipine) leads to dilatation of the precapillary sphincter, thus increasing intracapillary pressure and decreasing the rate of fluid movement between the inside and extravascular spaces. Osmolar factors are another reason for the slow movement of
fluid. During hemodialysis, the osmolarity of plasma and extracellular excess fluid decreases as a result of diffusion of osmotic active substances into the dialysate solution. A rapid decrease in osmolar plasma leads to a decrease in the rate of intravascular replacement and the development of hypotension. This phenomenon is observed when the sodium concentration in the dialysate is less than that in the plasma by 4 mmol/L or more. This causes a drop in osmolar plasma, and water can move into the extravascular (interstitial and intracellular) space until a new equilibrium is established. At this time, the patient experiences a rapid decrease in plasma volume and develops hypotension. The likelihood of hypotension in this situation can be reduced if UV is not performed in the first hour of hemodialysis [32]. One of the main factors in maintaining hemodynamic stability during hemodialysis is the regulation of cardiac output (CO). During hemodialysis, to maintain hemodynamic stability, a decrease in blood volume occurs. Cardiopulmonary receptors in the atria and main pulmonary veins and arterial baroreceptors in the aortic arch and carotid sinus respond to a decrease in blood volume. This leads to suppression of the activity of the sympathetic nervous system, a subsequent increase in peripheral vascular resistance and molasses velocity, which prevents a decrease in blood pressure; therefore, any component of these compensatory mechanisms can be involved in the pathogenesis of SDH, weakening of the ability of the heart to increase CO, vascular pathology as a result of a decrease in the elasticity of the venous wall or decreased responsiveness to vasoconstrictor stimulation or autonomic neuropathy as a result of cardiopulmonary receptor pathology [32,33,34]. The structure of the venous wall determines the amount of venous return. With reduced elasticity of the venous walls, a slight decrease in blood volume leads to a pronounced decrease in venous pressure and arterial hypotension [33,34,35]. In patients with reduced elasticity of the venous vessels, fluid replacement from the interstitial space is difficult due to the disturbance of the capillary starling equilibrium. The elasticity of the venous vessels is especially reduced in dialysis patients with arterial hypertension, which contributes to the structural pathology of the venous wall [33].
High rates of ultrafiltration (more than 13 ml / kg / hour) have a negative effect on the level of blood pressure during the hemodialysis procedure in patients with chronic kidney disease, increasing the prevalence of intradialysis hypotension. Its prevalence was 26.69%, with a negative correlation between the rate of ultrafiltration and systolic blood pressure [36.37]. Predialysis, intra-dialysis and post-dialysis hypotension negatively affect the survival rate of patients with chronic kidney disease on programmed hemodialysis, increasing the risk of death in patients with pre-dialysis diastolic blood pressure by 55% and 69% in quartiles of 45-77.8 mm Hg. Art. and 77.8-83.7 mm Hg. Art. respectively. Reduction of post-dialysis systolic blood pressure to 125.9 mm Hg. Art. and more associated with a 59% increased risk of death, a decrease in post-dialysis blood pressure to 88.1 mm Hg. Art. and more revealed a significant increase in the risk of death by 35%. The risk of death is increased in all patients with chronic kidney disease with syndialysis hypotension, namely: with mild (> 10 mm Hg. Art.) 1.3 times (31%); at moderate (> 20 mm Hg) 2.4 times (by 240%); in severe (> 30 mm Hg) 2.49 times (249%). [37.38]. The use of a computer algorithm for controlling the ultrafiltration rate contributes to the optimization and personalization of the hemodialysis program, allowing to reduce the number of episodes of syndialysis hypotension and to reduce the frequency of interventions with hypertonic solutions aimed at stabilizing blood pressure. The elimination of errors in the work of medical personnel and constant monitoring of the controlled parameters of the patient during the period of the hemodialysis procedure reduces the risk of complications by 20.33%. Carrying out the hemodialysis procedure requires constant monitoring of the blood pressure level for the prevention and timely correction of episodes of intradialysis hypotension due to their negative impact on the survival rate of patients with chronic kidney disease [39].

Hemodialysis procedures for patients with chronic kidney disease entered into programmed hemodialysis should be carried out at an ultrafiltration rate of no higher than 13 ml / kg / h and avoid a decrease in intradialysis blood pressure by more than
20-30 mm Hg. Art. In patients with chronic kidney disease, prone to episodes of intradialysis hypotension, large interdialysis fluid additions, with the need for significant amounts of ultrafiltration, it is optimal to carry out hemodialysis on devices with a computer algorithm for controlling the ultrafiltration rate [40].

**Conclusion**

Thus, the issue of modern analysis of methods for the prevention of SDH remains an urgent topic. Modern methods for the prevention of SDH using cold dialysis fluid (35–36 °C), bicarbonate buffer for hemodialysis, maintaining the sodium content in the dialysis fluid at 138–144 mmol / L, potassium - 2-3 mmol / L, calcium 1.50 mmol / l, magnesium - 0.75 mmol / l and continuous profiled ultrafiltration, using special monitors to control blood volume during the hemodialysis procedure, as well as the use of objective methods for assessing dry weight, remains a relevant scientific direction requiring deep scientific research.

**LIST OF REFERENCES:**


4. Shilo, V. Association of predialysis and intradialytic blood pressure changes with 5- year survival rates in multicenter prospective cohort study of ESRD patients on maintenance hemodialysis (HD) / V. Shilo, I. Drachev.


10. Chuprasov VB. Programmed hemodialysis. Folio, SPb., 2001; 256


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