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PERSONIFICATION OF ANTIHYPERTENSIVE THERAPY IN ISCHEMIC CEREBRAL STROKE

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Abstract.

The purpose of the study is to optimize monitoring and personalize antihypertensive therapy in patients with severe ischemic cerebral stroke (ICS).

We examined 37 patients with ICS, average age 74,1±1,3 years, who received treatment in intensive care wards of the stroke department with general neurology beds of the Municipal Non-Profit Enterprise "City Hospital № 9" of the Zaporizhzhia City Council. There were 16 men (43,2%), average age 71,9±2,1 years; women – 21 (56,8%), average age 75,8±1.6 years.

Personification of antihypertensive therapy for severe ICS was carried out based on the etiology of hypertensive hemodynamic disorders: hyperkinetic type of arterial hypertension (Cardiac index ≥ 3,80 L×min^{-1}×m^{-2}) or hypokinetic type of arterial hypertension (Cardiac index ≤ 2,98 L×min^{-1}×m^{-2}).

In patients with severe ICS and hyperkinetic type of arterial hypertension, initial hemodynamic parameters were characterized by Mean arterial pressure (MAP) of 111,4 ± 1,4 mm Hg; Heart rate (HR) of 107,2±1,6 min; Cardiac index (CI) 6,74±0,27 L×min^{-1}×m^{-2}; the Total peripheral vascular resistance (TPVR) is 674±36 dyn×sec^{-1}×cm^{-5}. For the purpose of antihypertensive correction of the hyperkinetic type of arterial hypertension (CI ≥ 3,80 L×min^{-1}×m^{-2}), a solution of Magnesium Sulfate was used intravenously at a dose of 2500-5000 mg×day-1 in combination with Bisoprolol 5-10 mg×day^{-1} orally. This made it possible to stabilize hemodynamic parameters by the end of intensive therapy within the limits of eukinetic values: MAP 95,2 ± 1,5 mm Hg (p < 0,05); HR 81,9 ± 1,5 min (p < 0,05); CI 3,60±0,15 L×min^{-1}×m^{-2} (p < 0,05); TPVR is 1079±58 dyn×sec^{-1}×cm^{-5} (p < 0,05).

In patients with severe ICS and hypokinetic type of arterial hypertension, initial hemodynamic parameters were characterized by MAP of 117,7 ± 2,8 mm Hg; HR of 76,7 ± 1,5 min; CI 2,74±0,18 L×min^{-1}×m^{-2}; TPVR is 1754±123 dyn×sec^{-1}×cm^{-5}. For the purpose of antihypertensive correction of the hypokinetic type of arterial hypertension (CI ≤ 2,98 L×min^{-1}×m^{-2}), a solution of Ebrantil was used intravenously as a bolus of 1,25-2,5 mg with a further infusion of 5-40 mg×hour^{-1}. This made it possible to stabilize hemodynamic parameters by the end of intensive therapy within the limits of eukinetic values: MAP 92,7 ± 1,7 mm Hg (p < 0,05); HR 81,4 ± 0,9 min (p < 0,05); CI 3,65±0,16 L×min^{-1}×m^{-2} (p < 0,05); TPVR is 1036±46 dyn×sec^{-1}×cm^{-5} (p < 0,05).

Key words. Ischemic cerebral stroke, hyperkinetic type of arterial hypertension, hypokinetic type of arterial hypertension, antihypertensive personalized therapy.

Introduction.

Brain stroke is a leading cause of death and disability worldwide. In Ukraine, according to official statistics, about 100 thousand cerebral strokes occur every year (more than a third of them in people of working age), 30-40% of patients with cerebral stroke die within the first 30 days and up to 50% within the first year from the onset diseases, 20-40% of surviving patients become dependent on outside help (12,5% of primary disability), and only about 10% return to a full life [1].

Moreover, ischemic cerebral stroke accounts for approximately 76% of all cerebral strokes [2].

The modern concept of intensive care for patients with intracranial hypertension requires timely receipt of the results of special instrumental studies characterizing the dynamics of the development of the pathological process. In most cases, treatment is determined only by clinical signs such as hypertension [3] or if there is evidence of other organ damage such as acute heart failure, pulmonary edema, aortic aneurysm dissection caused by hypertension [4].

As part of cerebral autoregulation, blood pressure typically increases during the acute phase of ischemic cerebral stroke, maximizing perfusion in ischemic brain regions. However, severe arterial hypertension can lead to hemorrhagic transformation of the infarction, hypertensive encephalopathy, as well as cardiopulmonary and renal complications [5].

Therefore, the tactics of antihypertensive therapy in patients with ischemic cerebral stroke in the acute period has its own characteristics [5,6].

Currently, it is recommended to correct arterial hypertension only for systolic blood pressure values ≥ 220 mm Hg. and diastolic blood pressure ≥ 120 mm Hg. Correction of arterial hypertension in the first 24 hours after the development of ischemic cerebral stroke should not exceed 15% of the initial blood pressure value.

If intravenous thrombolysis is indicated, it is recommended to reduce systolic blood pressure to ≤ 185 mmHg before initiating it. and diastolic blood pressure to values ≤ 110 mmHg. After completion of the procedure, monitor blood pressure numbers in the range of systolic blood pressure ≤ 180 mm Hg. and diastolic blood pressure ≤ 105 mm Hg. If it is impossible to ensure the specified blood pressure values, it is recommended to refuse thrombolysis due to the risk of hemorrhage [8].

Under physiological conditions, the constancy of cerebral blood flow is ensured by cerebral autoregulation through dilatation or constriction of cerebral arterioles. However, this mechanism of cerebral autoregulation is effective only when the mean arterial pressure is in the range of 50-150 mmHg. Pressure above this level causes hyperemia and swelling of the brain, which is accompanied by an increase in intracranial pressure. Therefore, control and correction of arterial hypertension takes a predominant place in the complex of intensive care for ischemic cerebral stroke [8].

The purpose of the study is to optimize monitoring and personalize antihypertensive therapy in patients with ischemic cerebral stroke.

Materials and Methods.

We examined 37 patients with ischemic cerebral stroke, average age 74,1±1,3 years, who received treatment in intensive care wards of the stroke department with general neurology beds of the Municipal Non-Profit Enterprise "City Hospital № 9" of the Zaporizhzhia City Council. There were 16 men (43,2%), average age 71,9±2,1 year; women – 21 (56,8%), average age 75,8±1,6 years.

Depending on the etiology of hypertensive hemodynamic disorders (hyperkinetic type of arterial hypertension or hypokinetic type of arterial hypertension), patients with ischemic cerebral stroke were divided into 2 groups according to the cardiac index values [9].

Group 1 consisted of 21 patients (average age 74,1±1,6 years) with ischemic cerebral stroke whose hemodynamic parameters were characterized by the hyperkinetic type of arterial hypertension, and cardiac index values corresponded to ≥ 3,80 $L \times min^{-1} \times m^{-2}$.

There were 8 men (38,1%), average age 74,1±2,9 years; women – 13 (61,9%), average age 74,1±1,9 years.

In the intensive care complex, in order to personalize the antihypertensive component, a solution of Magnesium Sulfate was used intravenously at a dose of 2500-5000 mg×day-1 in combination with Bisoprolol 5-10 $mg \times day^{-1}$ orally.

Group 2 consisted of 16 patients (average age 74,1±2,3 years) with ischemic cerebral stroke whose hemodynamic parameters were characterized by a hypokinetic type of arterial hypertension, and cardiac index values corresponded to ≤ 2,98 $L \times min^{-1} \times m^{-2}$.

There were 8 men (50,0%), average age 69,6±2,9 years; women – 8 (50,0%), average age 78,6±2,7 years.

In the intensive care complex, in order to personalize the antihypertensive component, a solution of Ebrantil was used intravenously as a bolus of 1,25-2,5 mg with a further infusion of 5-40 $mg \times hour^{-1}$.

The diagnosis was established in accordance with the existing criteria for clinical neurological examination and neuroimaging methods using a computed tomograph "Neu Viz 16 Classic Multi slice CT (China)".

The severity of ischemic cerebral stroke was assessed based on the severity of neurological symptoms as determined by the National Institutes of Health Stroke Scale [10].

Inclusion criteria:

Patients with ischemic stroke without cardiac arrhythmia.

Exclusion criteria:

Patients with ischemic stroke with cardiac arrhythmia.

Written consent to conduct the study was obtained from each patient or his relative (in the absence of productive contact with the patient), in accordance with the recommendations of ethical committees on biomedical research, Ukrainian legislation on health protection and the 2000 Declaration of Helsinki, European Society Directive 86/609 on participation people in biomedical research.

Indicators of systemic hemodynamics and the level of blood oxygen saturation were determined using a bedside patient monitor "Vismo PVM-2703" (Nihon Kohden Corporation, Japan):

- systolic blood pressure, mm Hg.
- diastolic blood pressure, mm Hg.
- mean arterial pressure, mm Hg.
- heart rate, in min.
- minute blood volume, $L \times min^{-1}$.
- cardiac index, L×min-1×m^{-2}.
- arterial blood oxygen saturation, %.

Total peripheral vascular resistance (TPVR) was calculated using formula 1 [11]:

$TPVR = MAP \times 79980 / MBV$, $dyn \times sec^{-1} \times cm^{-5}$ (1)

where MAP – mean arterial pressure, mm Hg.

MBV – minute blood volume, $L \times min^{-1}$.

The oxygen delivery index (IDO_2) was calculated using formula 2 [12]:

$IDO_2 = CI \times (1{,}34 \times Hb \times SaO_2) = CI \times CaO_2$, $ml \times min^{-1} \times m^{-2}$ (2)

where CI – cardiac index, $L \times min^{-1} \times m^{-2}$.

Hb – blood hemoglobin concentration, $g \times l^{-1}$.

SaO_2 – arterial blood oxygen saturation, %.

CaO_2 – oxygen content in arterial blood, $ml \times L^{-1}$.

Statistical processing of the study results was carried out using descriptive statistics methods using the Microsoft Excel 2010 software package. The reliability of the values was assessed according to Student's t-test for group 1 (n = 21) and for group 2 (n = 16). The results obtained were considered significant at a significance level of $p < 0{,}05$ ($t \geq 2{,}08$) for group 1 (n = 21) and $p < 0{,}05$ ($t \geq 2{,}12$) for group 2 (n = 16).

Results and Discussion.

Changes in neurological status indicators in patients with ischemic cerebral stroke (ICS) and hyperkinetic type of arterial hypertension are presented in Table 1.

As can be seen from Table 1, in patients with ICS and hyperkinetic type of arterial hypertension, the level of consciousness determined by the Glasgow Coma Scale (GCS) at the beginning of intensive therapy (IT) was characterized by deep stupor. However, by 48 hours of IT, the severity of neurological symptoms decreased by 9%, and after 72 hours and by the End of IT by an average of 14%.

The severity of neurological symptoms according to the National Institutes of Health Stroke Scale (NIHSS) at all stages of treatment corresponded to severe ICS. At the same time, by 72 hours there was a decrease in neurological deficit by 16%, and by the End of IT by 18%.

Changes in hemodynamic parameters in patients with ischemic stroke and hyperkinetic type of arterial hypertension are presented in Table 2.

As can be seen from Table 2, in patients with ICS and hyperkinetic type of arterial hypertension, a decrease in systolic blood pressure (BP syst.) was achieved starting from 4,5 hours of IT by 16%, then within 12-24 hours by 20%, by 48 hours at 22%, from 72 hours until the End of IT at 24%.

Diastolic blood pressure (BP diast.) tended to decrease statistically by 6% only by 72 hours from the start of treatment.

Table 1. *Characteristics of neurological status indicators in patients with ischemic cerebral stroke and hyperkinetic type (CI ≥ 3,80 L×min^{-1}×m^{-2}) arterial hypertension (M±m).*

Indicators	Intensive therapy (IT)						
	Start of IT	4,5 hours	12 hours	24 hours	48 hours	72 hours	End of IT
GCS, points	12,0 ±0,4	12,1 ±0,4	12,2 ±0,5	12,9 ±0,4	13,2 ±0,4*	13,9 ±0,3*	14,0 ±0,3*
NIHSS, points	16,7 ±0,7	16,2 ±0,7	16,0 ±0,7	15,1 ±0,8	14,8 ±0,7	14,0 ±0,7*	13,7 ±0,7*

*Note: *-p <0,05 compared to baseline values (n = 21)*

Table 2. *Characteristics of hemodynamic parameters in patients with ischemic cerebral stroke and hyperkinetic type (CI ≥ 3,80 L×min^{-1}×m^{-2}) arterial hypertension (M±m).*

Indicators	Intensive therapy (IT)						
	Start of IT	4,5 hours	12 hours	24 hours	48 hours	72 hours	End of IT
BP syst., mm Hg	174,3 ±2,6	146,7 ±3,2*	138,6 ±3,2*	139,0±2,8*	136,2±2,0*	132,9±2,0*	133,3 ±1,9*
BP diast., mm Hg	80,0 ±1,5	78,1 ±2,0	78,6 ±1,4	77,6 ±1,4	77,1 ±1,0	75,2 ±1,5*	76,2 ±1,6
MAP, mm Hg	111,4 ±1,4	101,0 ±2,2*	98,6 ±1,8*	98,1 ±1,4*	96,8 ±1,1*	94,4 ±1,4*	95,2 ±1,5*
HR, min.	107,2 ±1,6	100,6 ±1,6*	92,6 ±1,4*	87,7 ±1,0*	84,4 ±0,9*	83,2 ±1,0*	81,9 ±1,5*
SV, mL	123,32± 4,80	96,35 ±3,96*	85,23 ±3,28*	89,73 ±4,01*	86,66 ±3,15*	85,48 ±3,02*	86,16 ±3,39*
MVBC, L×min^{-1}	13,22 ±0,50	9,69 ±0,40*	7,89 ±0,33*	7,87 ±0,38*	7,31 ±0,28*	7,12 ±0,24*	7,06 ±0,30*
CI, L×min^{-1}×m^{-2}	6,74 ±0,27	4,94 ±0,23*	4,03 ±0,17*	4,01 ±0,21*	3,73 ±0,20*	3,63 ±0,17*	3,60 ±0,15*
TPVR, dyn×sec^{-1}×cm^{-5}	674 ±36	833 ±50*	999 ±55*	997 ±64*	1059 ±42*	1062 ±51*	1079 ±58*

*Note: *-p <0,05 compared to baseline values (n = 21)*

Table 3. *Characteristics of indicators of oxygen transport status in patients with ischemic cerebral stroke and hyperkinetic type (CI ≥ 3,80 L×min^{-1}×m^{-2}) arterial hypertension (M±m).*

Indicators	Intensive therapy (IT)						
	Start of IT	4,5 hours	12 hours	24 hours	48 hours	72 hours	End of IT
SaO_2, %	93,2 ±0,6	96,2 ±0,3*	96,8 ±0,2*	97,3 ±0,1*	97,5 ±0,1*	97,8 ±0,1*	98,1 ±0,1*
IDO_2, ml×min^{-1}×m^{-2}	1130 ±51	825 ±40*	698 ±39*	696 ±39*	663 ±38*	654 ±36*	640 ±40*

*Note: *-p <0,05 compared to baseline values (n = 21)*

Table 4. *Characteristics of neurological status indicators in patients with ischemic cerebral stroke and hypokinetic type (CI ≤ 2,98 L×min^{-1}×m^{-2}) arterial hypertension (M±m).*

Indicators	Intensive therapy (IT)						
	Start of IT	4,5 hours	12 hours	24 hours	48 hours	72 hours	End of IT
GCS, points	12,1 ±0,5	12,4 ±0,5	12,8 ±0,5	13,1 ±0,5	13,4 ±0,5	13,6 ±0,5*	13,8 ±0,5*
NIHSS, points	16,6 ±0,5	16,1 ±0,5	15,4 ±0,5	15,1 ±0,6	14,7 ±0,6*	13,7 ±0,8*	13,6 ±0,8*

*Note: *-p <0,05 compared to baseline values (n = 21)*

Table 5. *Characteristics of hemodynamic parameters in patients with ischemic cerebral stroke and hypokinetic type (CI ≤ 2,98 L×min^{-1}×m^{-2}) arterial hypertension (M±m).*

Indicators	Intensive therapy (IT)						
	Start of IT	4,5 hours	12 hours	24 hours	48 hours	72 hours	End of IT
BP syst., mm Hg	161,9 ±3,6	138,8 ±2,6*	136,9 ±3,0*	133,8 ±2,9*	137,5 ±2,8*	135,6 ±2,7*	130,6 ±2,5*
BP diast., mm Hg	95,6 ±2,7	78,1 ±1,0*	77,5 ±1,4*	75,6 ±1,3*	78,1 ±2,5*	77,5 ±1,9*	73,8 ±1,5*
MAP, mm Hg	117,7 ±2,8	98,3 ±1,1*	97,3 ±1,6*	95,0 ±1,6*	97,9 ±2,5*	96,9 ±2,0*	92,7 ±1,7*
HR, min.	76,7 ±1,5	78,9 ±1,3	79,6 ±1,4	82,4 ±1,5*	87,3 ±0,5*	83,9 ±0,5*	81,4 ±0,9*
SV, mL	69,99 ±3,47	86,33 ±3,67*	86,52 ±4,83*	87,58 ±3,58*	84,80 ±4,18*	81,65 ±3,36*	87,96 ±3,01*
MVBC, L×min^{-1}	5,37 ±0,29	6,81 ±0,32*	6,89 ±0,43*	7,22 ±0,34*	7,40 ±0,37*	6,85 ±0,30*	7,16 ±0,26*
CI, L×min^{-1}×m^{-2}	2,74 ±0,18	3,48 ±0,13*	3,51 ±0,24*	3,68 ±0,18*	3,78 ±0,23*	3,50 ±0,18*	3,65 ±0,16*
TPVR, dyn×sec^{-1}×cm^{-5}	1754 ±123	1155 ±52*	1130 ±60*	1053 ±49*	1058 ±97*	1131 ±77*	1036 ±46*

*Note: *-p <0,05 compared to baseline values (n = 21)*

Table 6. *Characteristics of indicators of oxygen transport status in patients with ischemic cerebral stroke and hypokinetic type (CI ≤ 2,98 L×min^{-1}×m^{-2}) arterial hypertension (M±m).*

Indicators	Intensive therapy (IT)						
	Start of IT	4,5 hours	12 hours	24 hours	48 hours	72 hours	End of IT
SaO_2, %	94,0 ±0,6	96,6 ±0,2*	97,1 ±0,2*	97,5 ±0,1*	97,7 ±0,2*	97,9 ±0,1*	98,3 ±0,1*
IDO_2, ml×min^{-1}×m^{-2}	471 ±35	614 ±44*	631 ±47*	664 ±38*	688 ±57*	641 ±48*	657 ±54*

*Note: *-p <0,05 compared to baseline values (n = 21)*

Mean arterial pressure (MAP) decreased by 9% at 4,5 hours of IT, by 11% at 12 hours, by 12% at 24 hours, by 13% at 48 hours, and by 15% from 72 hours until the End of IT.

Heart rate (HR) decreased by 6% by 4,5 hours of IT, by 14% by 12 hours, by 18% by 24 hours, by 21% by 48 hours, by 22% by 72 hours, and by 24% by the End of IT.

The value of stroke volume (SV) decreased by 22% by 4,5 hours of IT, by 31% by 12 hours, by 27% by 24 hours, and by an average of 30% from 48 hours until the End of IT.

The values of minute volume of blood circulation (MVBC) and cardiac index (CI) decreased equally by 27% by 4,5 hours of IT, by an average of 40% within 12-24 hours, by 45% by 48 hours, by 46% by 72 hours, by 47% by the End IT.

The values of total peripheral vascular resistance (TPVR) increased by 19% by 4,5 hours of IT, by an average of 33% within 12-24 hours, by 36% by 48 hours, by 37% by 72 hours, and by 38% by the End of IT.

Changes in indicators of oxygen transport status in patients with ischemic stroke and hyperkinetic type of arterial hypertension are presented in Table 3.

As can be seen from Table 3, in patients with IS and hyperkinetic type of arterial hypertension, a statistically significant increase in blood oxygen saturation (SaO_2) was noted from 4,5 hours of intensive therapy by 3%, then within 12-48 hours by an average of 4%, from 72 hours until the End of IT by an average of 5%.

A decrease in the oxygen delivery index (IDO_2) was observed by 4,5 hours of IT by 27%, within 12-24 hours by an average of 38%, by 48 hours by 41%, by 72 hours by 42% and by the End of IT by 43%.

Changes in neurological status indicators in patients with ICS and hypokinetic type of arterial hypertension are presented in Table 4.

As can be seen from Table 4, in patients with ICS and hypokinetic type of arterial hypertension, the level of consciousness determined by the GCS at the Start of IT was characterized by deep stupor. However, by the 72 hours of IT, the severity of neurological symptoms decreased by 11% and by 12% by the End of IT.

The severity of neurological symptoms according to the NIHSS scale at all stages of treatment corresponded to severe ICS. At the same time, by 48 hours of IT, there was a decrease in neurological deficit by 11%, by 72 hours by 17%, and by the End of IT by 18%.

Changes in hemodynamic parameters in patients with ICS and hypokinetic type of arterial hypertension are presented in Table 5.

As can be seen from Table 5, in patients with ICS and hypokinetic type of arterial hypertension, a decrease in BP syst. was achieved starting from 4,5 hours of IT by 14%, then within 12-48 hours by an average of 15%, to 72 hours by 16% and by the End of IT by 19%.

A decrease in BP diast. was observed by 4,5 hours of intensive therapy by 18%, by 12 hours by 19%, by 24 hours by 21% and by the End of IT by 23%.

A decrease in MAP was observed by 16% by 4,5 hours of intensive therapy, finally stabilized by 17% by 48 hours, then by 18% by 72 hours and by 21% by the End of IT.

A statistically significant increase in HRe was observed by 24 hours of IT by 7%, a maximum increase by 48 hours by 12% and by the End of IT by 6%.

The value of SV increased by 19% by 4,5 hours of IT, and by 20% by 24 hours and by the End of IT.

The values of MVBC and CI increased equally by 21% by 4,5 hours of IT, then by 22% by 12 hours and 72 hours, and by 25% by the End of IT.

By 4,5 hours of IT, there was a decrease in TPVR by 34%, within 24-48 hours by 40% and by the End of IT by 41%.

Changes in indicators of oxygen transport status in patients with ICS and hypokinetic type of arterial hypertension are presented in Table 6.

As can be seen from Table 6, in patients with ICS and hypokinetic type of arterial hypertension, a statistically significant increase in SaO_2 was noted from 4,5 hours of IT by 3%, and subsequently for 24 hours until the End of IT on average by 4%.

An increase in the IDO_2 was observed by 4,5 hours of IT by 23%, by 12 hours by 25%, by 24 hours by 29%, by 48 hours by 32% and by the End of IT by 28%.

Conclusion.

1. Personification of antihypertensive therapy for severe ICS was carried out based on the etiology of hypertensive hemodynamic disorders: hyperkinetic type of arterial hypertension (CI ≥ 3,80 $L\times min^{-1}\times m^{-2}$) or hypokinetic type of arterial hypertension (CI ≤ 2,98 $L\times min^{-1}\times m^{-2}$).

2. In patients with severe ICS and hyperkinetic type of arterial hypertension, initial hemodynamic parameters were characterized by MAP of 111,4 ± 1,4 mm Hg; HR of 107,2±1,6 min; CI 6,74±0,27 $L\times min^{-1}\times m^{-2}$; the TPVR is 674±36 $dyn\times sec^{-1}\times cm^{-5}$.

For the purpose of antihypertensive correction of the hyperkinetic type of arterial hypertension (CI ≥ 3,80 $L\times min^{-1}\times m^{-2}$), a solution of Magnesium Sulfate was used intravenously at a dose of 2500-5000 mg×day-1 in combination with Bisoprolol 5-10 $mg\times day^{-1}$ orally.

This made it possible to stabilize hemodynamic parameters by the end of intensive therapy within the limits of eukinetic values: MAP 95,2 ± 1,5 mm Hg ($p < 0,05$); HR 81,9 ± 1,5 min ($p < 0,05$); CI 3,60±0,15 $L\times min^{-1}\times m^{-2}$ ($p < 0,05$); TPVR is 1079±58 $dyn\times sec^{-1}\times cm^{-5}$ ($p < 0,05$).

3. In patients with severe ICS and hypokinetic type of arterial hypertension, initial hemodynamic parameters were characterized by MAP of 117,7 ± 2,8 mm Hg; HR of 76,7 ± 1,5 min; CI 2,74±0,18 $L\times min^{-1}\times m^{-2}$; TPVR is 1754±123 $dyn\times sec^{-1}\times cm^{-5}$.

For the purpose of antihypertensive correction of the hypokinetic type of arterial hypertension (CI ≤ 2,98 $L\times min^{-1}\times m^{-2}$), a solution of Ebrantil was used intravenously as a bolus of 1,25-2,5 mg with a further infusion of 5-40 $mg\times hour^{-1}$.

This made it possible to stabilize hemodynamic parameters by the end of intensive therapy within the limits of eukinetic values: MAP 92,7 ± 1,7 mm Hg ($p < 0,05$); HR 81,4 ± 0,9 min ($p < 0,05$); CI 3,65±0,16 $L\times min^{-1}\times m^{-2}$ ($p < 0,05$); TPVR is 1036±46 $dyn\times sec^{-1}\times cm^{-5}$ ($p < 0,05$).

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