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Impact of Intra-Extracranial Hemodynamics on Cerebral Ischemia by Arterial Hypertension (Part 2)

Alexander G. Kruglov, PhD, ScD, Georgiy Y. Gebel, PhD, ScD†, Alexander Y. Vasilyev, PhD, ScD*

Moscow State University of Medicine and Dentistry, Moscow, Russian Federation

Abstract

The association between hemodynamic and biochemical parameters of cerebral blood flow have been studied in man, using mathematical methods of statistics. The values have been obtained through catheterization using a probe jammed at the level of the bulb of the superior jugular vein. Relationships with central hemodynamic parameters have been evaluated, including the right atrium, the right ventricle, and the left ventricle, as well as with pressure and biochemical values of the arterial bed. Data have been acquired in patients with stable arterial hypertension. Analysis of all relationship between hemodynamic and biochemical parameters has shown that the uniform hemodynamic zone: Sin.P. – SJV – SEV – the right atrium, normally participates in regulation of gaseous exchange in the human brain depending on the minimum pressure on the way of outflow from the brain. In stable arterial hypertension, this type of regulation is lost. On the basis of the results of this study, it has been concluded that blood viscosity is normally a primary controlled parameter of homeostasis. In stable arterial hypertension, homeostatic control of factors determining rheological and thrombogenic properties of blood, as well as participating in the development of brain ischemic conditions is lost. This increases risk of disturbances in central hemodynamics. IJBM 2012; 2(2):96-101.

Key words: *blood viscosity, brain gaseous exchange, central hemodynamics, correlation of hemodynamics and biochemistry, homeostatic control.*

For introduction and methods, refer to Part 1 of the Article.

Abbreviations

aorta – Ao,
brain sigmoid sinus – Sin.P.,
superior jugular vein – SJV,
superior vena cava – SVC,
systolic pressure – SP,
diastolic pressure – DP,
pulse pressure – PP,
medium pressure – med.P.,
maximum value – Max.,
minimum value – Min.,
mean value – M,
confidence interval ratio – m,
a-v oxygen content difference-O_2 – $AVDO_2$,
cardiac minute output – MVC,
stroke volume – IVC,
right atrium – Au.d.,
right ventricle – V.d.,
left ventricle – V.S.,
final diastolic pressure of the right ventricle – F.d.p.,
stable arterial hypertension – SAH(H),
catalase in erythrocytes – Ker,
catalase in plasma – Kpl,
norm-N.

All data of blood pressure are in mmHg.

*Corresponding author: Prof. Alexander Y. Vasilyev, PhD, ScD, Head of Radiology Department, Moscow State University of Medicine and Dentistry, 15 Aviaconstructor Mil str., housing 1, apt. 70, 109431, Moscow, Russian Federation.
Tel: 7-495-6110177 E-mail: auv62@mail.ru

Results and discussion

Table 1
Pressure levels in the right atrium and the right ventricle.

		Au.d					V.d.	
		A	**X**	**V**	**Y**	**Med.**	**F.d.p.**	**Med.**
N	**M**	5.7	2.4	4.6	3.4	4.1	5.1	14.4
	m	0.9	0.7	0.8	0.2	1.1	1.2	1.8
	Max	8.8	5.3	6.5	6.0	8.4	10	19.8
	Min	3.0	0	1.6	0	1.2	0	8.8
H	**M**	7.4	4.0	6.4	4.7	6.1	3.4	13.9
	m	2.7	2.1	2.8	2.0	2.9	2.6	4.4
	Max	11.8	9.0	14.1	9.0	14.6	8.4	28
	Min	1.8	0	1.8	0	1.6	0	6.6

Note the increase in all values of Au.d. pressure in SAH compared with the norm based on Ms ($p < 0.01$), being outside the normal zone.

Table 2
Pressure levels in the aorta and the left ventricle.

		MVC	IVC	Ao				V.S.	
				SP	**DP**	**PP**	**Med.P**	**SP**	**PP**
N	**M**	10.9	128.4	114	71	40.3	92.6	108	100.5
	m	1.3	22.4	4.5	3.8	3.5	5.1	6.4	7.1
	Max	18.7	186	147	87	65	115	130	130
	Min	5.5	80	90	50	21	70	90	85
H	**M**	11.1	147	180	101	79	137	180	168
	m	3.1	38.9	31	21	22	23	34	28
	Max	21.7	246	270	155	144	210	260	248
	Min	5.3	50	150	78	40	96	150	138

The given data are common. They are presented for full description of the material acquired by us.

Table 3
Relationship between pressure levels of Sin.P. and pressure levels of the aortal chamber and the left ventricle.

		Ao				MVC	IVC	SPVS	PPVS
		SP	**DP**	**PP**	**Med.P**				
N	**SP**				+	-		-	-
	DP		+		+	-		-	-
	PP		-						
	Med.P				+	-	-	-	-
H	**SP**			+					
	D.P.								
	PP	+	+	+	+		-	+	
	Med.P	-	-		-				

Note that active process of V.S. (which was assessed based on Ao and V.S. SP and Ao. PP) does not correlate with PP of Sin.P. in N, which reflects absence of interdependent relationship between them. SP of the V.S. is associated negatively with systolic, diastolic, and med.P. of Sin.P. Thus, formation of Sin.P. does not normally depend on Ao. PP, being simultaneously related to the pressure levels of the arterial part of the vascular bed. In SAH, relationship between Sin.P. and pressure in the aortal chamber is lost, while V.S. active-phase relationship with pulse Sin.P. occurs (with a process creating Sin.P. gradient). Thus, levels of Sin.P. in N are correlated with the phase of blood ejection from the V.S.: blood ejection from the V.S. in Ao.; achievement of maximum pressure in V.S. – Ao. system, with open aortic valves; creation of maximum pressure for the cranium, in general; creation of wave process in Ao., the nearest point of application of which is the system of extra- and intracranial vessels.

Table 4
Relationship between Sin.P. and the levels of central venous pressure.

		Au.d.					V.D.	
		Med.P	**A**	**X**	**V**	**Y**	**F.D.P.**	**Med.P**
N	**SP**	+	-	+				+
	DP	+		+				+
	PP			-			+	
	Med.P	+		+		+	-	+
H	**SP**		+			+		
	DP		+			+		
	PP					+		
	Med.P					+	+	+

Note the almost complete change of relationship in SAH when compared with N, with establishment of positive correlations with Y-wave of the Au.d. for all levels of Sin.P.

Table 5
Relationship between SJV pressure and the levels of central venous pressure.

		Au.d.					V.D.	
		Med.P	**A**	**X**	**V**	**Y**	**F.D.P.**	**Med.P**
N	**SP**	+	-		-	+		
	DP				-		-	+
	PP		-		-	-		+
	Med.P						-	
H	**SP**		+	+		+		+
	DP		+	+		+		
	PP			+				+
	Med.P		+	+		+		

In case of substantial change of correlations, it should be noted that in SAH, as opposed to N, relations to X – the Au.d. collapse appear, that is common for all levels of SJV pressure.

In case of SAH, relationship between Au.d. pressure and the values of total and plasma viscosity (F-n, Ht), vasoactive MV regulators (Kpl., Ker., pH), and final brain metabolites (HbO2, AVDO2) is lost. The above-mentioned data on the relationships between Sin.P., SJV, the Au.d. (the uniform hemodynamic chamber with negative pressure gradient) allow to consider this zone as one of the gaseous exchange regulators (possibly exchange, in general) of the brain, where the main regulator is the minimum pressure on the way of outflow from the brain in the Au.d.. In SAH, this regulation channel is lost.

We state that SAH causes loss or inversion of relationships between pressure levels of the zone regulating gaseous exchange in the brain depending on the minimum pressure and physical and chemical values of the blood inflowing to the brain: total and plasma viscosity (F-n, Ht), acid-base balance vasoactive substances, and values of plasma and associated gases (pCo2, HbO2).

Table 6
Relationship between pressure levels in the right atrium and biochemistry of blood outflowing from the brain.

			K pl	K er	Na pl	Na er	pH	pCO_2	SB	pO_2	HBO_2	HB	Ht	F-n	P-n	$AVDO_2$
N	Au.d.	A	-				-				-			-		+
		X	-	-						-	-	-	+	-		-
		V	-	+	-		+		+	-	-		+	+		+
		Y	-	+	-	-	+			-	-	-	+	-		+
H	Au.d.	A														
		X			+			+								
		V								+						
		Y														

Table 7
Relationship between pressure levels of the right atrium and biochemistry of blood inflowing to the brain.

		K pl	K er	Na pl	Na er	pH	pCO_2	SB	pO_2	HBO_2	HB	Ht	F-n	P-n
N	A	-			-		-					+	-	
	X	-			-					+		+	+	
	V	-		-	-	+					+	+		
	Y	-		-	-	+	-			+		+	-	
H	A								-				+	
	X			+	-	-			-					
	V			+					-					
	Y	+	+	+					-				+	

Table 8
Relationships between arterial pressure levels and biochemistry of blood inflowing to the brain.

		K pl	K er	Na pl	Na er	pH	pCO_2	SB	pO_2	HBO_2	HB	Ht	F-n	P-n
N	SP												+	
	DP						-	-					+	
	PP													
	Med.P						-	-	+					
H	SP									+	-			
	DP						+	+			-			
	PP													
	Med.d						+	+						

Summarizing data in Tables 8 and 9, it is necessary to note that SAH (with substantial increase of all Ao pressure values) causes loss or inversion of relationships between Ao pressure levels and all values of physical and chemical properties of both, inflowing and outflowing blood (with the values of plasma viscosity, membrane permeability, gaseous pressure, and acid-base balance), while maintaining sufficient MV as well as levels of AVDO2 and Sin.P. equal to those in N.

We start our discussion from the phase of blood ejection from the V.S. According to [3], the V.S. forms a rotating blood flow, the peculiarity of which is that the flow particles rotate about the external (variable) axis and move forward along this axis. Rotation and emergent flow

Table 9
Relationships between arterial pressure levels and biochemistry of blood outflowing from the brain.

		K pl	K er	Na pl	Na er	pH	pCO_2	SB	pO_2	HBO_2	HB	Ht	F-n	P-n
N	SP													
	DP			+									+	
	PP												-	
	Med.P			+				-						
H	SP			-							-			
	DP			-		+				+				
	PP				-						-		+	
	Med.P	-	-	-		+				+				

turbulence increase thermodynamic (energy and information) power of the flow. Sphenoid wave structure of the aortal flow, i.e. pulse aortal pressure in healthy man (N) in supine position reaches all points of the body before closure of aortal valves [4]. This phase also includes ejection of venous blood from the cancellous tissue of the cardiac muscle through the coronary sinus to the Au.d. during the so-called uniform beat of the ventricular block [2]. Negative correlation of processes occurring during this phase with the levels of Sin.P. can be explained only by existence of the structure changing its lumen under impact of the wave process between the cranial cavity and extracranial vessels through which the wave process is taking place. We think that this structure is presented by carotid artery traps (CAT), located in the cavernous venous sinus. It is the CATs that are conductors of heart wave structures, being informative characteristics of different control levels [1], interfering in venous sinuses of the brain. Our data prove the negative association of the ejection phase and Sin.P. levels, which can be explained only by the presence of the CAT system. In SAH, this association is positive, which proves changing of Sin.P. is parallel to changes in aortal pressure having increased energy and information level. This is possible only through change of functional relationships in the system: “carotid artery trap—cavernous sinus”, connected with the sigmoid sinus (point of catheter impaction and initial data acquisition) through the lower petrosal sinus. Note that the CAT sympathetic nerve plexuses participate in innervation of the gaze, playing a significant role in implementation of goal-directed behavior.

A modest increase in Sin.P. in patients with SAH, under conditions of linear dependence of Sin.P. on AP, may show that in order to maintain Sin.P. at the required level, it is necessary to increase AP and perfusion grade. This is possible only in case of loss of extracranial regulation (damage of carotid artery trap) and associated damage of intracranial vascular bed with luminal narrowing and vessel wall stiffness.

Aortal maximum phase during splitting of Ao and V.S. is considered to be the final stage of the high-energy phase and the initial stage of diastolic evolution. Activity of CAT, the narrowing of which is caused by the wave process of the previous phase, takes off pressure, starting with the value equal to the medium arterial pressure. All levels exceeding this value normally remain in reserve to respond in acute stress situations (minimum adaptive regulation: weakening of CAT response to the wave process is enough). It is the medium AP which is correlated with Sin.P. This positive correlation is preserved up to the level of diastolic AP showing that the uniform hydrodynamic system: Ao – vessels at entry into the cranium – Sin.P. – reaches the phase of diastolic evolution (from medium to diastolic). This explains the correlation between diastolic evolution of the intracranial venous section and diastolic evolution of the arterial bed. After achievement of Mins. in these vascular areas and minimum grade of Ao-Sin.P., the high-energy process described above occurs once again. Summing up, we can say that during one cardiac cycle, correlation between relationships of Ao and Sin.P. pressure levels changes from positive to negative through zero. Difference for SAH is the dependence of Sin.P. only on the high-energy phase of inversion of the regulation mark; absence of correlation at all stages of diastolic evolution; absence of pressure level reserve for emergency adaptation. Thus, in patients with SAH, despite an increase in energy consumption for regulation of MV and exchange, the effectiveness of support of brain functioning rapidly decreases.

Very important is the fact of correlation between fibrinogen and arterial blood flow hemodynamics. We consider that the initiator of these relationships is fibrinogen, based on our results and other investigations [5]. Changes in concentration of F-n in N are correlated with changes in hemodynamics, preventing negative manifestations of fibrinosis. This alteration occurs both, in the arterial and venous beds. Although changes in pressure are differently directed in Ao, Sin.P., and SJV, their effect is synergic and directed at the prevention of ischemic disturbances.

In patients with SAH, Ao pressure is not correlated with F-n. Subsequently, it leads to development of changes facilitating thrombogenesis not accompanied by hemodynamic correction, where increase of venous pressure is capable of aggravating thrombogenesis.

On the analyzed hemodynamic way of outflow from the brain, the Au.d. is always adjoined, and the V,d, adjoins in phasic manner. We have allocated two periods for the Au.d.-V.d. system:

1. This occurs with detached Au.d. and V.d.

and closed valve, during diastolic processes in the Au.d. muscle against the background of V.d. systolic and transient processes. This period is characterized by "X-collapse" and "V-wave". In N, this phase is related to the values of the final brain exchange products, AVDO2. In SAH, only relations with pO2 remain, and relations with F-n, Ht, Ht, HbO2, electrolytes, and AVDO2 are lost.

2. The phase of the uniform Au.d. and V.d. chamber, after opening of the tricuspid valve including "Y-collapse", "A-wave" of the Au.d., as well as the final DP of the V.d.

Closure of the tricuspid valve takes place after passing of "A-wave" of the Au.d., at the transition from final DP to the level of med.P. of the V.d. which is formed with the help of the tricuspid valve sagging into the cavity of the Au.d. (a process inverse to the mechanism of "X-collapse"). Thus, the Au.d. pressure levels impacting Sin.P. pressure on the valveless hemodynamic way, SJV – SEV – Sin.P., are formed with the help of dynamics of the tricuspid valve. "Y-collapse" is accompanied by the diastolic evolution of the Au.d. and V.d. muscles. Specifically, the levels of venous pressure transmitted toward Sin.P. are formed not only by pressure of the venous blood, but also with participation of the myocardium diastolic evolution of this section and sucking diastolic function of the V.d. "A-wave" of the Au.d., and final DP of the V.d. are formed though muscle activity of the Au.d. during continued evolution in the myocardium of the V.d.. The wave process generated by the Au.d. during this phase spreads in the SJV – SEV – Sin.P. system against the blood flow (as opposed to those generated by the V.S. and spreading along with the blood flow). In patients with SAH, correlations with F-n, Ht, Hb, pH, electrolytes, and AVDO2 are lost in this period. This means that control of correlation between thrombogenic factors and pressure at outflow from the brain existing in N are lost.

Conclusions

Acquired and processed with the help of mathematical methods of statistics, described in parts 1 and 2, the results demonstrate that in the blood's rheological properties, viscosity is the primary controlled parameter of homeostasis in a healthy man. We also consider that uniform hydrodynamic formation with negative pressure grade of Sin.P. – SJV – SEV – the Au.d. is one of the regulators of gaseous exchange in the brain, where the main regulator is the minimum pressure on the way of outflow from the brain in the Au.d.. In case of SAH, homeostatic control of factors determining rheological and thrombogenic blood properties, and participating in formation of ischemic conditions in the human brain is lost. Also lost, is the regulation channel of gaseous exchange in the brain depending on minimum pressure on the way of outflow from the brain.

References

1. Baevsky RM, Bondarchuk VI, Chernyshov MK. Temporal organization of cardiac rhythm in terms of evolution. In the book: Problems of comparative cardiology (Syktyvkar), 1979. p 4.
2. Gebel GY, Kruglov AG, Utkin VN, Golostenova LM, Dasaev AN, Ignatov NG. Total gaseous pressure in blood of the vessels of outflow from the cardiac ventricular block in human. 6th All-Russian scientific forum "Keys to diagnostics and treatment of cardiovascular diseases". Moscow, IRC, January 20 -23, 2004. p 116-122.
3. Kuzmina NB, Drogaicev AD. Formation of blood flow in the cavity of the left ventricle. In book: Cardiac physiology. Series: Guidance in physiology, 1980. p 212-214.
4. Lightfoot A. Transport phenomena in live systems. Biomedical aspects of momentum and mass transport. Moscow: Mir, 1977. p 520.
5. Levtov VA, Regirer SA, Shadrina NH. Hemorheology. Moscow: Medcine, 1982. p 270.