

The Role of Integrated Gas Compounds in Regulation of Gas Homeostasis in the Norm

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Abstract

In practically healthy people on the background of self-breathing, we used catheterization to obtain blood samples from Ao, PT, SC, VJI, SS, VH and VR. We believe that the standard tests of blood gases by volume (pO_2 and pCO_2) and their A-V gradients, quantitatively determined, are insufficient to fully assess the hypoxic states both in the whole organism and in individual organs. To estimate gas homeokinesis, we performed integral gas tests, including an additive criterion of blood gases—pressure in mmHg: 1) the summary pressure of the plasma gases, PS; 2) Gas functional, the gradient between the total indices of arterial and vein gases (Gradient D); and 3) the exchange gradient, Gradient DP [(arterial pO_2 – venous ($pO_2 + pCO_2$))]. Each test indicator at all studied points was determined in mmHg. Correlation analyses were carried out between the parameters of all tests.

We found that the processes forming PS limit the amplitude of the PS deviation under changing parameters of the constituent components (pO_2 and pCO_2) due to acts of mutual replacement between them, as well as the influence of integral gas complexes under shifts in pO_2 , pCO_2 , A-V SO_2 . Unlike the generally accepted tests that record quantitative differences between the points studied, the integral gas tests allow us to identify vectors and mechanisms of adaptive changes in gas homeostasis, to perform a qualitative comparison of the functioning of the studied organs by gas-dynamic tests in the norm and in pathology. (**International Journal of Biomedicine. 2017;7(3):185-191.**)

Key Words: integrated gas compounds • blood gases • gas homeokinesis • gas functional

Abbreviations

Ao, aorta; **A-V**, arteriovenous; **BP**, blood pressure; **DP**, diastolic pressure; **Er**, erythrocyte; **MP**, mean pressure; **PT**, pulmonary trunk; **PI**, plasma; **PP**, pulse pressure; **PS**, summary gas pressure; **P-n**, total protein; **PSv** - PS of venous blood; **RV**, right ventricle; **SS**, sigmoid sinus; **SP**, systolic pressure; **SC**, coronary sinus; **SO₂**, oxygen saturation; **VJI**, internal jugular vein; **VH**, right hepatic vein; **VR**, right renal vein.

The aim of the study was to evaluate the distribution of the PS values in the investigated points of the human body and their participation in the regulation of gas homeostasis in the norm.

Methods and Results

With the patient in the supine position, we used catheterization to obtain blood samples from the outflow vessels of the three-chamber block of ventricles (Ao, PT, SC), VJI, VH, and VR.⁽¹⁾ To investigate general and organ gas exchange, in normal practice, we used data for temporal

dynamics arterial and venous blood and their A-V gradients. In our opinion, the separately taken values of pO_2 and pCO_2 are changed quantitatively (in absolute values) and are inadequate for evaluating polyorganic hypoxic states, since the use of the general average values does not reveal qualitative changes that characterize and explain the so-called mutual replacements of pO_2 and pCO_2 at varying ratios of the levels of pO_2 and pCO_2 (the biophysical equivalent of the Haldane effect) in the investigated areas of the human body.

To estimate the gas homeostasis, along with the generally accepted methods we used tests that take into account the additive trait for pO_2 and pCO_2 —a pressure in mmHg:

- 1) $PS = pO_2 + pCO_2$ (at each investigated point)
- 2) Gas functional (Gradient D, GD) between PSs in arteries and veins ($GD = (pO_2 + pCO_2)_A - (pO_2 + pCO_2)_V$).
- 3) Exchange gradient (Gradient DP, GDP) [(arterial pO_2 –

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venous ($pO_2 + pCO_2$)]. GDP between Ao pO_2 (which provides metabolic processes involving O_2 , including the binding of O_2 to Hb), and PSv (which is formed as a result of metabolism, including Hb- O_2 dissociation) that change conjugately and unidirectionally (+).

These tests can be changed quantitatively (absolute values) and qualitatively (by sign). Unlike gas volume tests, gradient DP is less dependent on the factors affecting O_2 transport in the vascular bed, Hb concentration, or stroke and minute heart volumes. GDP helps to identify the qualitative differences in organ metabolism that are not determined by other methods. The complementary relationship between two test groups makes possible their joint application.

The following values and correlations between the proposed tests were calculated for Ao-TP (heart-lung block); Ao-CS (myocardium block); Ao-VJI (brain block); Ao-VH (liver block); and Ao-VR (kidney block). As a result, we obtained information on the distribution of integrated gas indicators at various points of the human cardiovascular system.⁽²⁻⁵⁾ The vector and amplitude of the distribution of the indices studied were determined by the relationships with the mean hemodynamic pressure of each organ (Pmed, defined as the initial value of the perfusion pressure, which provides the metabolism). We present cases with the following parameters: Ao pCO_2 – 30-45 mmHg, Ao pO_2 – 70-100 mmHg, HR – 75.6±1.2 bpm, Hb – 13.1±1.4 g/l, Ht – 45.0±0.1%. The remaining data are presented in Table 2.

The quantitative, numerical results of gas homeostasis, which we have studied for a long time, were presented earlier in our works.⁽⁶⁻³⁴⁾ The results of the study of the qualitative relationships of the gas homeostasis in the norm are given in Table 1, which shows statistically significant ($P < 0.05$) values (+, 0, -) of correlations for the studied parameters, without indicating their magnitudes.

It should be noted that the correlation analysis showed the absence of reliable links for all proposed tests with Hb in all investigated points (in contrast to volume-additive tests), gas functionals with pCO_2 and pO_2 at all the points studied, and Ao PS with pCO_2 at all points.

Reliable correlations for PS levels in venous points with pO_2 (positive) and pCO_2 (positive) in Ao blood were revealed, which indicates a combined unidirectional dynamics of these processes. We believe that this is why PSv can remain constant at a significant amplitude of shifts in pO_2 , pCO_2 and HbO_2 (%), due to acts of mutual replacement of pO_2 and pCO_2 . Reflection of the general dependence on Ao pO_2 is positively correlated with PS levels at the venous points. We believe that this is a reason for the stability of PSv with significant shifts in the parameters of blood gases and HbO_2 . Thus, SC PS is positively correlated with VR pO_2 , and gradient DP is positively correlated with Ao pO_2 .

We estimated the distribution of PS in the outflow vessels of the three-chambered block of heart ventricles (Ao, TP, SC), including studying the reproducibility of this distribution in different states of the human body. According to data of the norm in the blood of Ao, TR, SC, VJI, VH, VR, we determined pO_2 , pCO_2 in mmHg, their A-V gradients, PS, as well as special gradients: GD and GDP.

Thus, the following correlations between the proposed tests were calculated: Ao-TP (heart-lung block); AO - CS (myocardium block); Ao-VJI (brain block); Ao-VH (liver block), and Ao-VR (kidney block).

To determine the vector and amplitude of the distribution of the investigated indices, their relationships with Pmed were analyzed in the investigated areas. [bulbus Ao (86.5±1.7), SC (5.3±0.4), TP (14.0±0.22), SS (9.4±1.2), VJI (6.7±1.3), RA (4.1±0.72), LA (8.1±0.8); HR (75.6±1.2 bpm), Hb (13.1±1.4 g/l), Ht (45.0±0.1%)].

All data were obtained under self-breathing. The data presented were investigated in the same order as blood ejection from the three-chamber block of ventricles into the outflow vessels (Ao, TP, SC) during united systole.⁽¹⁴⁾

When analyzing the baseline data, we established the stable changes in PS, regardless of the sampling point of blood, conjugate to changes and equivalent to the magnitude of the changes of each gas by its total value (both an increase and a decrease). It is also noted that PS can vary in the following directions: 1) less than one of them, but more than the other; and 2) less than the value of each of them, minimizing PS deviations and compensating the shifts of individual parameters.

PS is greater in those areas where Pmed is also greater (Table 2): the maximum in Ao (for $pO_2 > pCO_2$); the minimum in SC (for $pO_2 < pCO_2$); intermediate in TP ($pO_2 = pCO_2$). PS values in the outflow vessels are close, or correspond, to the values of systemic BP:

- Ao PS (for $pO_2 > pCO_2$) is within SBP: maximum = 145 mmHg [pO_2 (100 mmHg) + pCO_2 (45 mmHg); minimum = 100 mmHg [pO_2 (70 mmHg) + pCO_2 (30 mmHg)].

- TP PS (with variable ratios of pO_2 and pCO_2) is mathematically close to the average BP in the norm (PS = 84.8±0.5 mmHg, BP=86.5±5.1 mmHg.)

- PS SC (for $pO_2 < pCO_2$) is mathematically close (within the statistical error) to DBP (PS=71.3±0.6mmHg, DBP=71±0.38 mmHg)

The values of these parameters are formed in the chambers of the three-chambered block of ventricles at the end-diastolic pressure in the right and left ventricles and the average pressure in SC. All pressure values (PS and systemic BP) are created conjugately between each other and other parameters of the cardiocycle on a single basis (the impulse of a quantum generator): contraction of the myocardium of the three-chambered block of ventricles.

The data presented demonstrate the dynamics of the regulation of gas homeostasis in the norm in the investigated areas of the body:

SC:

- A) Levels of pO_2 and PS are minimal in comparison with other investigated points.

- B) Gradients with respect to pO_2 , pCO_2 , and gas functionals are maximal in comparison with the other investigated points.

- C) SC PS is less than arterial blood pO_2 , and gradient DP for the heart is positive (20.66±0.44 mm.Hg).^(2,10,36,37) PS of SC blood, outflowing from the spongy (venous) chamber of three-chambered block of ventricles, is minimal (71.0±0.6 mmHg) and close to the systolic pressure in the aorto-pulmonary block with placental and beginning pulmonary gas exchange (open

Botalli's duct), when it functions as a single chamber.⁽¹⁶⁾

SC PO₂ corresponds to the values of the hypoxic limit found in the venous blood of other points: VJI of 20mmHg is a critical level, accompanied by loss of consciousness. Figuratively speaking, the blood flow from the spongy (venous) chamber of three-chambered block of ventricles includes the criteria of the hypoxic limit of the human body.⁽²⁷⁾

TP:

PS in mixed venous blood, the flow of which is created at different ratios of pO₂ and pCO₂ flowing from RV (84.8± 0.5 mmHg), is lower than in Ao blood and close to arterial blood pO₂ (91.7±0.5 mmHg) and to Pmed in Ao bulb (86.5±1.7 mmHg).

This venous flow is created by the mixing of blood flowing from the spongy chamber of three-chambered block of ventricles, mixing with the flow of venous blood from the lower and upper hollow veins. A measure of the adequacy of the combined and unidirectional shifts of TP PS and Ao pO₂ is Gradient DP (91.91±0.5 mm.Hg), varying both in magnitude and in sign (+, 0, -) within the values obtained for the kidneys and the heart.^(11,15,16) TP PS is mathematically close (within the margin of error) to PS in VJI and VH, with a common proximity to Ao Pmed.

Ao:

PS of the blood flowing from VS, at pO₂ greater than pCO₂, is maximal (128.3±0.6 mmHg) and close to SBP in aortic bulb (107.6±2.6 mmHg); it is created by the mixing of the blood of RV and Thebesian veins of LA and LV, having ability to direct and reverse the flow of blood (two-way communications of the left heart chambers with the myocardium by pressure).

VJI:

PS (84.89±0.54 mmHg) is mathematically (within the margin of error) close to the following parameters of arterial and venous blood:

- a) Average Ao pO₂ (85.72±0.65 mmHg); the measure of adequacy of unidirectional shifts of Ao pO₂ and VJI PS is Gradient DP (0.83±0.57 mmHg);
- b) Average Ao Pmed (86.5±1.7 mmHg);
- c) TS PS (84.8±0.5 mmHg).

It should be noted that the blood of VJI is formed by mixing the venous streams of two zones:

- 1) Tissue zone (removal of brain metabolites) with low pO₂
- 2) Cerebrospinal fluid (choroidal plexus) with high pO₂

We consider a significant fact to be almost complete coincidence (within the margin of error) of quantitative indicators of pCO₂ levels in the blood of VJI and SC, while noting the difference in pO₂ levels. We assume that the minimum value of pO₂ (23.77±0.3 mmHg) among the recorded values during catheterization is the individual hypoxic limit of the organism.

VH:

PS (88.51±0.54 mmHg) is mathematically close to IVC PS, Ao Pmed, and Ao pO₂.^(20,29,35) A measure of the adequacy of the unidirectional shifts of Ao pO₂ and VH PS is Gradient DP (1.93±0.54 mmHg). VH Pmed is mathematically close to the pressure in the liver parenchyma (8.0±0.7 mmHg) and PV (8.42±0.76 mmHg) with a minimum of gradients between these three points.

VR:

Under superperfusion and the mixing of the blood inside the kidney (A-V shunts), including juxtglomerular blood fractions with differences in pO₂ and pCO₂ in countercurrent exchange (analogue of the mixing process), there are formed 1) the maximal pO₂ and VR PS levels in comparison with those for brain block, heart-lungs block, myocardium block, and liver block, wherein the ratio VR pO₂>VR pCO₂ is similar to that in Ao blood; 2) the values of Gradient D for pO₂, SO₂ and PS for the studied organs and blocks (heart-lungs, splanchnic pool, brain) are minimal; 3) VR PS is greater than Ao pO₂, therefore, Gradient DP for the kidneys is negative (-11.9±0.57).^(20,25,27,28,31,32) We believe that the upper limit of the norm for oxygenation (pO₂) of venous blood is VR pO₂. In the blood of TP, VJI and VH, the blood oxygenation levels can vary between the minimum limit (SC pO₂) and the maximum limit (VR pO₂). The values of gas parameters beyond the maximum and minimum values can be a sign of decompensation of gas homeostasis.

A detailed analysis of all the qualitative and quantitative indicators (Table 1 and 2) was presented by us in earlier works.^(15,16, 17-24, 34)

The interaction of specific proteins with different affinities for O₂ is a unifying factor in the gas exchange of the heart, brain and kidneys.

1) In the heart, myoglobin (Mb) has an affinity to O₂ 6 times greater than Hb, with a shorter dissociation time and association of Mb and O₂, with the possibility of forced dissociation of HbO₂ at SO₂ of 50% and lower.

2) In adults, the mixing of blood flows with differences in pO₂ and SO₂ is a common characteristic in the formation of the blood composition in VJI and VR, as in the fetus—the mixing of blood of v.umbilicalis with its own venous blood (intravascular gas exchange).

The dissociation and association of HbO₂ in different sections of the S-shaped curve depends on temperature, pH, electrolytes, etc. in different ways, as well as the Hb affinity for O₂. When the flows are mixed, the processes proceed without the participation of membranes, with the creation of new levels of pO₂ and SO₂. Blood with low SO₂ carries out forced dissociation of HbO₂, as in the transmembrane interaction of Hb and Mb in the heart, as well as in arterial blood and VR blood in the counterflow system of the kidney. In other words, the described mechanism allows us to consider the venous system of the organs represented as an internal oxygenator, ensuring cooperative multi-organ regulation of homeostasis as a whole.

The proposed integral tests reveal qualitative differences in the functioning of the organ-blood systems, which are not detected by the generally accepted tests:

a) In the myocardium block, when Ao blood periodically enters the exchange zone, where the minimum levels of pO₂, HbO₂(%), SC PS are created, Gradient DP is maintained positive, and maximum gas gradients are created, including Gas functional.

b) In the microcirculatory bed of the kidney block (with participation of countercurrent metabolism), the maximum (venous) levels of pO₂, HbO₂(%), VR PS and the minimal Gas gradients, including Gas functional, are created during 1 cardiocycle.

Table 1. Integrated gas indicators at various points of the human body

Variable	PS Ao	PS PT	PS VJI	PS VH	PS SC	PS VR	GD	GDP
Ao	K pl			+				
	K er	+		+				
	Na pl	-	+	-	+			
	Na er	-	-		+			
	pH	-	-	-	+			
	pCO ₂		+	+	+			
	SB	-	-	-	-			
	pO ₂	+	+	+	+	+	+	
	HbO ₂	+	+	+				
	Hb							
pS Ao		+	+					
PT	K pl	+						
	K er		+		+			
	Na pl	+						
	Na er	-		+	+			
	pH		+		+			
	pCO ₂		+		+			
	SB	-	+					
	pO ₂	+	+	+	+			
	HbO ₂	+	+	+	-			
	Hb							
pS PT	+		+	+				
VJI	K pl		+	+				
	K er	-	-	-	+			
	Na pl	-	-	-				
	Na er		-		-			
	pH	-		-				
	pCO ₂		+	+	+			
	SB	-	-	-	-			
	pO ₂	+	+	+	+			
	HbO ₂	+		+	-			
	Hb							
PS VJI	+	+		-				

Table 1. (Continued)

Variable	PS AO	PS PT	PS VJI	PS VH	PS SC	PS VR	GD	GDP
VH	K pl	+			+			
	K er	-	+	-	+			
	Na pl	-	-	-				
	Na er		-		-			
	pH	-		-				
	pCO ₂		-	+	+			
	SB	-						
	pO ₂	+	+	+	+			
	HbO ₂	+		+	-			
	Hb							
pS VH		+	-					
SC	pCO ₂				+	+		
	pO ₂							
	Hb							
VR	pO ₂				+			
	pCO ₂					+		
	Hb							
Ao	sp	-	+	-		x	x	- -
	dp	-	-	-		x	x	-
	pp		+			x	x	
	mp	-	-	-		x	x	-
PT	sp	-	-	-		x	x	- -
	dp	-	-	-	+	x	x	-
	pp	-	-	-		x	x	- -
	mp	-	-	-		x	x	- -
VJI	sp	-	-	-	-	x	x	-
	dp	-	-	-	-	x	x	
	pp	-	-	-	-	x	x	-
	mp	-	-	-	-	x	x	
VH	sp		-		-	x	x	-
	dp	-	-	-		x	x	-
	pp	+	-	+	-	x	x	- +
	mp	-	-		-	x	x	-
PS PT		+		+	+	x	x	
PS VH			+	-		x	x	
GD		+	+				x	x
GDP		+	+				x	x

Table 2. Blood gas parameters in the points studied

Variable	pO ₂	PCO ₂	PS	pH	SO ₂	Pmed
SC	23.77±0.30	47.50±0.34	71.3±0.6	7.345±0.005	38.02±0.70	5.3 ±0.4
TP	42.96±0.40	41.86±0.30	84.80±0.50	7.372±0.003	76.80±0.42	14.0±0.2
Ao	91.70±0.54	36.62±0.21	128.3±0.6	7.396±0.003	96.74±0.07	86.5 ±1.7
VJ	37.95±0.57	47.95±0.24	84.89±0.54	7.353±0.003	68.21±0.74	6.7±1.3
VH	43.63±0.49	44.88±0.34	88.51±0.54	7.360±0.004	76.76±0.62	8.38±1.38
VR	59.4±1.20	41.35±0.24	100.7±0.80	7.375±0.003	87.75±0.46	8.97±1.74
D(Ao -V)	DpO ₂	DPCO ₂	DPS	DpH	DSO ₂	
D(Ao - SC)	67.93±0.43	-10.9±0.26	57.00±0.42	0.051±0.002	58.70±0.73	
D(Ao - TP)	43.77±0.47	-4.31±0.21	39.46±0.45	0.023±0.001	19.48±0.40	
D(Ao - VJI)	46.72±1.86	-8.79±0.97	37.93±2.27	0.030±0.006	24.35±2.53	
D(Ao -VH)	46.82±0.55	-5.51±0.25	41.31±0.60	0.025±0.002	20.00±0.60	
D(Ao - VR)	29.40±0.54	-2.88±0.16	26.51±0.50	0.013±0.001	8.76±0.40	

In this case, $VR PS > Ao pO_2$; as a result, Gradient DP is negative and multidirectional with respect to the remaining gradients of the gas parameters in the heart-lung block, brain block and liver block with minimization of deviations from zero (destruction of differences between $Ao pO_2$ and the venous PS), where the common mechanism is the formation (after mixing) of flows with different levels of pO_2 and $HbO_2(\%)$ in a zone of values between the maximum level of Gradient D (A-SC) created in the myocardium block and the minimum level of Gradient D (A-VR) created in the kidney block. (4,16-24,34-37) Normally, the possible deviations in Gradient DP of organs do not reach DP value in the myocardium block (with the + sign) or in the kidney block (with the sign -).

We consider the venous system as a special organ which, in addition to other functions, participates in the overall metabolism by mixing blood flows with differences in pO_2 , pCO_2 and $HbO_2\%$, having the function of maintaining the gradients by integral gas parameters between different exchange zones. An important condition for the norm of gas exchange as a whole is the minimization of deviations from the average PS value.⁽²⁶⁾ Minimization of differences between organs by PS occurs while maintaining the differences between them, according to pO_2 , pCO_2 , pH, SO_2 and their ratios in the arterial and venous systems, being a reflection of polyorganic interactions.

Maintaining multidirectional deviations in Gradient DP from the zero in the kidney block and myocardium block indicates their conjugated functioning, the derivatives of which are gas-hemodynamic complexes that limit the amplitude of oscillations of the gas parameters in the brain, liver, and heart-lung blocks.

The stability of the homeostasis of the liquid medium of the body in interaction with the external gaseous environment is maintained through the interrelation of metabolic processes in the studied blocks, in which our tests, relatively independent from factors affecting O_2 transport (Hb, stroke and minute heart volumes), allow us not only to detect changes, but also to create mechanisms influencing organ functioning, both in the normal and pathology states.

We believe that PS in the liquid media of the human body that depends on the combined processes (tissue, plasma, erythrocyte, external respiration, hemodynamics, etc.), as a derivative from the total pressure of gases in gas media (alveolar air, atmospheric air), is an evolutionary-deterministic basis for the formation of all types of pressure, including hemodynamic pressure. Tests for PS are an integral part of the analysis of a single cooperative system of the regulation of metabolism and other functions of the human body by pressure (arterial, venous, intracardiac, tissue, liquor, oncotic, amniotic, etc.).

Conclusion

Summary gas pressure (PS) is a mechanism to maintain the stability of intrasystemic metabolic processes and gas homeostasis as a whole. The processes forming PS limit the amplitude of the PS deviation under changing the parameters of the constituent components (pO_2 and pCO_2) due to acts of

“mutual replacement” between them, as well as the influence of integral gas complexes under shifts in pO_2 , pCO_2 , A-V SO_2 . Processes that minimize the fluctuations in the sum of gases, in contrast to the volume tests ($O_2v\%$, $CO_2v\%$, $HbO_2\%$), are relatively independent from factors affecting the O_2 transport (Hb, stroke and minute heart volumes, SO_2 dissociation regulation systems). This makes it possible to consider them as significant factors for stabilizing the homeostatic control of the gas-hemodynamic environment of the human body. GDP, as a measure of the adequacy of the combined changes in arterial pO_2 and venous PS, is relevant for multi-organ monitoring of metabolic processes and inter-system relations in dynamics: norm - compensation - subcompensation – decompensation. Unlike the generally accepted tests that record quantitative differences between the points studied, the integral gas tests allow us to identify vectors and mechanisms of adaptive changes in gas homeostasis, to perform a qualitative comparison of the functioning of the studied organs by gas-dynamic tests in the norm and in pathology.

Competing interests

The authors declare that they have no competing interests.

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