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GASES OF ALVEOLAR AIR AND ARTERIAL BLOOD DURING DUST DISEASES OF THE RESPIRATORY ORGANS

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Wright-Patterson Air Force Base, Ohio

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U. S. BOARD ON GEOGRAPHIC NAMES TRANSLITERATION SYSTEM

Block	Italic	Transliteration	Block	Italic	Transliteration
А а	*А а*	A, a	Р р	*Р р*	R, r
Б б	*Б б*	B, b	С с	*С с*	S, s
В в	*В в*	V, v	Т т	*Т т*	T, t
Г г	*Г г*	G, g	У у	*У у*	U, u
Д д	*Д д*	D, d	Ф ф	*Ф ф*	F, f
Е е	*Е е*	Ye, ye; E, e*	Х х	*Х х*	Kh, kh
Ж ж	*Ж ж*	Zh, zh	Ц ц	*Ц ц*	Ts, ts
З з	*З з*	Z, z	Ч ч	*Ч ч*	Ch, ch
И и	*И и*	I, i	Ш ш	*Ш ш*	Sh, sh
Й й	*Й й*	Y, y	Щ щ	*Щ щ*	Shch, shch
К к	*К к*	K, k	Ъ ъ	*Ъ ъ*	"
Л л	*Л л*	L, l	Ы ы	*Ы ы*	Y, y
М м	*М м*	M, m	Ь ь	*Ь ь*	'
Н н	*Н н*	N, n	Э э	*Э э*	E, e
О о	*О о*	O, o	Ю ю	*Ю ю*	Yu, yu
П п	*П п*	P, p	Я я	*Я я*	Ya, ya

* ye initially, after vowels, and after ъ, ь; e elsewhere. When written as ё in Russian, transliterate as yё or ё. The use of diacritical marks is preferred, but such marks may be omitted when expediency dictates.

FOLLOWING ARE THE CORRESPONDING RUSSIAN AND ENGLISH DESIGNATIONS OF THE TRIGONOMETRIC FUNCTIONS

Russian	English
sin	sin
cos	cos
tg	tan
ctg	cot
sec	sec
cosec	csc
sh	sinh
ch	cosh
th	tanh
cth	coth
sch	sech
csch	csch
arc sin	$\sin^{-1}$
arc cos	$\cos^{-1}$
arc tg	$\tan^{-1}$
arc ctg	$\cot^{-1}$
arc sec	$\sec^{-1}$
arc cosec	$\csc^{-1}$
arc sh	$\sinh^{-1}$
arc ch	$\cosh^{-1}$
arc th	$\tanh^{-1}$
arc cth	$\coth^{-1}$
arc sch	sech^{-1}
arc csch	csch^{-1}
rot	curl
lg	log

GREEK ALPHABET

Alpha	Α	α	[illegible]		Nu	Ν	ν	
Beta	Β	β			Xi	Ξ	ξ	
Gamma	Γ	γ			Omicron	Ο	ο	
Delta	Δ	δ			Pi	Π	π	
Epsilon	Ε	ε	[illegible]		Rho	Ρ	ρ	[illegible]
Zeta	Ζ	ζ			Sigma	Σ	σ	ς
Eta	Η	η			Tau	Τ	τ	
Theta	Θ	θ	[illegible]		Upsilon	Υ	υ	
Iota	Ι	ι			Phi	Φ	φ	ϕ
Kappa	Κ	ϰ	κ	[illegible]	Chi	Χ	χ	
Lambda	Λ	λ			Psi	Ψ	ψ	
Mu	Μ	μ			Omega	Ω	ω	

GASES OF ALVEOLAR AIR AND ARTERIAL BLOOD DURING DUST DISEASES OF THE RESPIRATORY ORGANS

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Among the many criteria which are set as the basis of the clinical evaluation of the functional state of the external respiration system during dust-induced bronxo-pulmonary diseases, the diagnostic significance of the partial pressure of gases in alveolar air and their tension in arterial blood remains the least studied. First of all, this is explained by the procedural difficulties connected with the complexity of determining the tension of CO_2, and especially O_2, and also obtaining the "ideal" alveolar air. However, if the Holden procedure (with different variations and supplements) is all that remains up to now to obtain the latter, the concept of the tension of gases in arterial blood can be obtained using indirect methods to determine them.

In those investigations conducted by us to calculate the tension of CO_2 in arterial blood ($PaCO_2$) we used the Collier principle, according to which, after a certain time after circular respiration in a closed system with 7-8% CO_2, its concentration becomes equal to the CO_2 content in the mixed venous blood. Using a low-inertia gas analyzer CO_2 (GUM-2) made it possible for us to judge the equalization of the carbon dioxide concentrations in the mixed venous blood and the closed system most accurately - by means of the appearance of a characteristic plateau (Fig. 1). Having calculated the CO_2 tension at the moment of the plateau formation and considering the venous-arterial difference of CO_2 which is distinguished by exceptional constancy and is equal to 6 mm Hg (Campbell and Howell; Stein and Colp; N. N. Kanayev), the tensions of this gas in the arterial blood were obtained. The tension of oxygen in the arterial blood (PaO_2) is calculated from the Rahn and Fenn nomograph proceeding from the results of determining $PaCO_2$ and the oxygen saturation of the arterial blood. The latter was determined oxyhemometrically (in a vessel manner), the blood was taken from a finger of a hand preheated at 45° for 15 min.

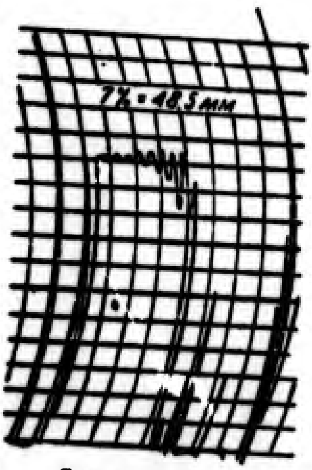

Figure 1. Capnogram of circular respiration for determining the PCO_2 of mixed venous blood and the calculation of $PaCO_2$.

Sampling an analysis of the alveolar air were conducted according to Holden's classical procedure. Investigations were conducted with 10 persons without signs of pulmonary and cardiovascular pathology and with 43 patients with dust bronxopulmonary diseases, caused by continuous work in contact with silicon-bearing dust (core molding, cast fettling, grinding, electric welding, etc.). The age of both the healthy persons and the examined patients was approximately the same - from 32 to 60 years.

On the basis of the results of the clinical-roentgenological examination of 33 patients, pneumoconiosis is established (stages I in 16 people and II in 17 people). In 24 of them pneumoconiosis accompanied the clinically pronounced phenomena of bronchitis, frequently with an asthmatic component. In 10 patients without roentgenological signs of pneumofibrosis, but with a distinct picture of bronxo-pulmonary disease, chronic dust bronchitis is diagnosed. In accordance with the clinical data and the results of the investigation of the external respiration function, based on the criteria developed by us (A. A. Penknovich et al.), pulmonary deficiency of the I degree was diagnosed in 13 patients, in 12 - II, and in 12 - III degree. In 6 people the disease lasted without a breakdown of the external respiration function.

Figure 2 gives individual data which reflect the PaO_2, the partial pressure of oxygen in alveolar air (PAO_2), and the size of the alveolar-arterial gradient of the oxygen in healthy and sick people with a different flow pattern of the dust bronxo-pulmonary pathology. The mathematical-statistical processing of these data showed that PAO_2, equal to 107.4±2.64 mm Hg in healthy people, descends even in patients with pneumoconioses, which occurs without the clinically pronounced phenomena of bronchitis (99.9±1.70 mm Hg; P/0.01). An even larger reduction in PAO_2 is revealed in patients whose dust bronxo-pulmonary pathology is accompanied by a distinct bronchial component. In this case, the PAO_2 in patients with pneumoconiosis in conjunction

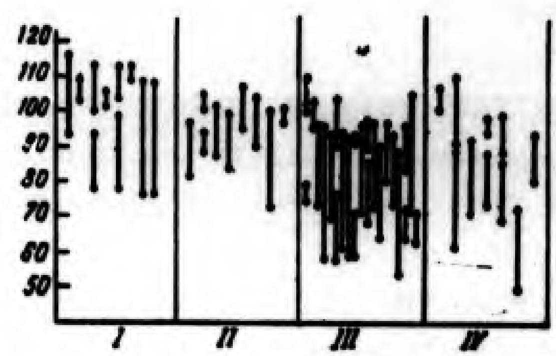

Figure 2. PAO_2 [•], PaO_2 [o] and the size of the alveolar-arterial gradient of the oxygen in healthy persons in patients with a different flow pattern of dust bronxo-pulmonary pathology. Along the ordinates - gradient in mm Hg; along the abscissas: I - healthy, II - patients with pneumoconiosis without bronchitis, III - patients with pneumoconiosis with bronchitis, IV - patients with chronic dust bronchitis.

with chronic bronchitis (91.6±1.94 mm Hg) proved to be less, not only compared with the healthy, but also with the sick people with a "pure" form of fibrosis ($P<0.05$).

The differences in the amount of PaO_2 were somewhat less pronounced. So, with "pure" forms of pneumoconiosis the PaO_2 was virtually the same as in healthy people (respectively, 88.8±3.28 and 91.9±3.17 mm Hg). Only in patients with chronic dust bronchitis and especially with pneumoconiosis with a bronchial component the amount of PaO_2 was statistically reliably lower (respectively, 77.6±5.19 and 72.1±2.46 mm Hg), than in the persons of the control group ($P<0.02$), while in patients with pneumoconiosis with bronchitis it is less than in those with "pure" pneumoconiosis ($P<0.01$).

The unidirectional nature of PAO_2 and PaO_2 changes during the development of dust bronxo-pulmonary pathology is the reason for the fact that even during serious diseases a considerable increase in the alveolar-arterial gradient of the oxygen is by no means always noted. In any case, we did not succeed in revealing a statistically reliable increase in this gradient in patients with pneumoconiosis in conjunction with chronic bronchitis compared with its size in healthy persons.

From the data given in Fig. 3, it is seen that the most significant increase in the alveolar-arterial gradient of the oxygen occurs in patients with pulmonary deficiency of the III degree. Its average size in patients of this group (22.8± ±3.37 mm Hg) was statistically reliably more compared with persons without breathing deficiency, in whom this gradient was equal to 13.5±3.11 mm Hg, and also compared with patients with pulmonary deficiency of the I degree (14.4±1.70 mm Hg).

In light of current concepts about the reasons for the increase in the alveolar-arterial gradient of the oxygen

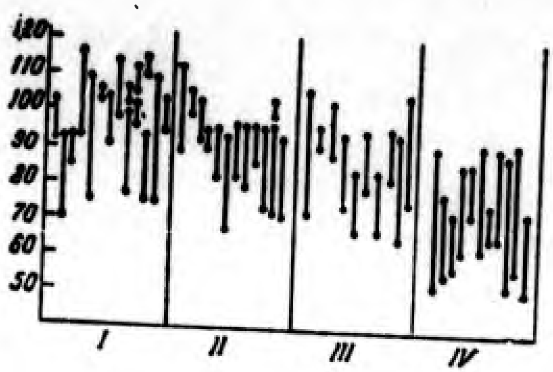

Figure 3. PAO_2 [•], PaO_2 [o] and the size of the alveolar-arterial gradient of the oxygen in persons with a different manifestation of pulmonary deficiency. Along the ordinates - gradient in mm Hg; along the abscissas: I - person without pulmonary deficiency, II - patients with pulmonary deficiency of the I degree, III - patients with pulmonary deficiency of the II degree, IV - patients with pulmonary deficiency of the III degree.

(L. L. Shik) the data obtained by us make it possible to assume that only with pronounced pulmonary deficiency in patients with dust bronxo-pulmonary diseases do prerequisites appear for a change in the relationship between the partial pressure of oxygen in the alveolar air and its tension in the arterial blood. In other words, if the relative PaO_2 reduction in patients with the II, and especially the I, degree of pulmonary deficiency, is the main consequence which develops in them as a result of ventilation disorders of the alveolar hypoxia, then during the III degree of the breathing deficiency with factors which facilitate the onset of hypoxemia, they obviously stop the breakdown of the relationship between ventilation and blood flow, and also the circulatory and diffusion breakdowns in a small circle of blood circulation.

Examining the questions of the relationship between the partial pressure of the gases in alveolar air and their tension in arterial blood, it should be noted that recently attempts were made to theoretically substantiate and experimentally demonstrate the presence of an arterial-alveolar CO_2 gradient (K. A. Mayanskaya et al.; Koziorowski and Radwan), which they consider increases in proportion to the development of the breathing deficiency. However, as shown in our determinations $PaCC_2$, adopted from the Collier method, and $PACO_2$, from Holden, the existing difference in the size of these indicators in the majority of persons without pulmonary deficiency does not differ from this difference in patients with pulmonary deficiency of the III degree. If, the arterial-alveolar CO_2 gradient in the persons without breathing deficiency comprised on the average of 5.4±0.65 mm Hg, then in patients with pulmonary deficiency of the I degree it was equal to 5.8±0.95 mm, II degree - 5.4±0.74 mm and III degree - 1.7±1.87 mm Hg.

Thus, without denying the presence of the pressure of the CO_2 gradient in arterial blood and alveolar air, based on the data we obtained, we cannot agree with the fact that the gradient increases in patients with pulmonary pathology.

Conclusions. 1. The most pronounced reduction in the partial pressure of the oxygen in alveolar air and its tension in the arterial blood in patients with dust diseases of the respiratory organs is observed when the clinically pronounced phenomena of chronic bronchitis are present. A close connection of the reduction in these indicators with the degree of pulmonary deficiency in the patients is established.

2. A significant increase in the alveolar-arterial gradient of the oxygen is noted, as a rule, during pulmonary deficiency of the III degree; this indicates that the arterial hypoxemia in these patients is developed not only because of ventilation breakdowns, but also because of a charge in the relationship between ventilation and blood flow in the lungs, "bypassing," and the reduction in the diffusion ability of the lungs.

3. The presence of the arterial-alveolar CO_2 gradient was revealed, whose size, however, does not undergo substantial changes depending on the manifestation of the breathing deficiency.

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