CARBON MONOXIDE VALUES IN BLOOD IN KARATE PRACTITIONERS AND THEIR POSSIBLE PREDICTIVE SIGNIFICANCE

Ján **PIVOVARNÍK**[1ABCDEF], Jakub **PIVOVARNÍK**[2DEF]

[1]*University of Prešov, Faculty of Sport, Slovakia*

[2]*Charles University, Second Faculty of Medicine, Czech Republic*

Keywords:
- carboxyhemoglobin,
- bicarbonate,
- oxygen.

Abstract:

The paper focuses on predictive possibilities of carbon monoxide blood levels of karate practitioners, in connection with other elements of the buffering mechanism, as a possible diagnostic element which can be used to simply indicate the possibilities of a performance or top athlete. The aim is to point out its different concentration in venous blood during dual exercise of relatively the same length, volume, and intensity of training. Specifically there are different values, probably in the complex of utilization of other buffering mechanisms, the removal of metabolites (catabolite waste), and thus affecting current sports performance. The results show substantial significance of COHb at level of $p<0,05$ – CHT (closed hands training) training (input – output).

INTRODUCTION

Carbon monoxide (CO) is a colorless, non-flammable gas and its concentration in atmospheric air is very low, approximately 0,03%, and is heavier than air. The accumulation of CO in the body leads to fatigue, loss of consciousness to unconsciousness (narcotic effect). This is also closely related to carbon dioxide, since CO_2 dissolved in blood and other body fluids by mutual interaction forms Carbonic acid (H_2CO_3), bicarbonate (bicarbonate ion - HCO_3^-), and a proton (H^+), thereby participating in acid-base balance (ABB). When its levels in the organism raise, respiratory acidosis (RAC) occurs, when it decreases, respiratory alkalosis occurs (RAL), its increased or decreased levels can compensate for metabolic disorders (e.g. hyperventilation during metabolic acidosis – MAC). Factors that affect the diffusion of O_2 also affect the diffusion of CO, which is extremely fast bound by haemoglobin [Lékařský slovník 2019].

Physiologically, CO_2 generated in cells dissolves in intracellular fluid – water, and diffuses through the extracellular fluid into venous blood. Transport is carried out by erythrocytes and haemoglobin. CO_2 is 20x more soluble in blood than O_2, so there is more of it in the solution, then, CO_2 dissolved in plasma diffuses into erythrocytes until the concentration gradient is equalized. In arterial blood, the pCO_2 is 5,33 kPa, in tissues CO_2 diffuses from the cells into the blood, and pCO_2 thereby increases to 6,27 kPa in venous blood. The CO_2, that rapidly diffuses into erythrocytes is rapidly converted to HCO_3^-.

Approximately 70% of the HCO_3^- formed in erythrocytes diffuses into plasma (in exchange for Cl^- anion, to maintain electroneutrality). In pulmonary capillaries, due to decreasing pCO_2, all of these reactions take place in the opposite direction, with HCO_3^- diffusing from the plasma back to the erythrocytes, receiving H^+ and generating CO_2 and H_2O. The content of CO_2 in mixed venous blood, both freely dissolved and chemically bound, is 23-24 $mmol.l^{-1}$, in arterial blood 22-23 $mmol.l^{-1}$. The relationship between HCO_3^- and dissolved CO_2 varies according to blood pH (at pH 7,4, the plasma ratio of HCO_3^-/CO_2 is 20:1). When these values are compared, the normal pH in erythrocytes is 7,2, which corresponds to a 12:1 ratio. The HCO_3^-/CO_2 system is the main mechanism for maintaining acid-base balance in the blood. If arterial blood plasma pH drops below 7,35, this indicates acidosis. When the change is primarily in pCO_2, it is primarily a respiratory disorder, primary change in HCO_3^- concentration is manifestation of a metabolic disorder. Subsequent metabolic acidosis (MAC) can be caused by a variety of causes, hyperventilation occurs during it (chemoreceptor irritation in medulla oblongata), its purpose being to exhale a relative excess of CO_2 and adjust the buffer system to normal. During normal metabolic processes, CO_2 is continuously produced at 15-20 mol/day. At rest, CO_2 output is approximately 260ml or 11,7 $mmol.l^{-1}$. With a cardiac output of 5 l/min, 52ml (2,3 mmol) of $CO_2.l^{-1}$ passes from the tissue into the blood, and from there, along the concentration gradient to pulmonary alveoli, where pCO_2 partial pressure is lower, and CO_2 leaves the body in exhaled air. This volume is several times higher during physical exertion. There is more than 100x more CO_2 in the exhaled air than in inhaled air (0,035 vs 0,0003 volume % CO_2), so one does not receive any CO_2 from the air. In a calm state, one breathes out approximately 250 ml CO_2 $.min^{-1}$ [Silbernagl & Despopoulos 1984].

This is closely related to carbon monoxide (CO). Colorless, odourless, and tasteless gas, occurs mainly where imperfect combustion takes place. It may also occur in poorly ventilated rooms in which CO is released from fire from damaged furnaces or heating equipment. CO has approximately 300x higher affinity for Hb than O_2. Poisoning by this gas is most often manifested by hyperaemia and hypoxia, due to impaired transport of O_2 to tissues. After binding of CO to Hb, carboxyhemoglobin (COHb) is formed, with consequent restriction of O_2 transport to tissues and removal of CO_2 from tissues, which leads to acidosis. In addition to hypoxia, CO is directly toxic to the myocardium and brain. Direct binding to myoglobin in the myocardium reduces cardiac function. CO is excluded from the body by exhalation. In spontaneous breathing, elimination half-time of CO is 4 hours, and is accelerated by inhaling O_2 enriched with CO_2, which decreases this half-time to approximately 40 minutes. Normal COHb values: up to 1% COHb – people living in clean air, up to 5% COHb - people living in cities, up to 10 - 15% COHb – strong smokers. Clinically, poisoning occurs at a concentration of COHb above 10-15%; followed by 20 - 40% COHb – fatigue, headache, nausea, vomiting, visual disturbances; 40-60% COHb – tachypnoe, tachycardia, ataxia, syncope, cramps; 60 - 80% COHb – coma and death [Dzurik, Trnovec 1997, Šašinka 2003, Denshaw-Burke, Savior 2009].

Carbon monoxide (CO) is a serious health hazard. Carbon monoxide binds to haemoglobin, myoglobin, some oxidoreductive enzymes, and other proteins. These bonds are reversible, but very strong: they bind haemoglobin approximately 220-300 times easier than oxygen and convert it into carboxyhemoglobin (or carbonylhemoglobin, hereinafter COHb), which is unable to carry oxygen [Dobiáš 2007]. CO binds to haemoglobin about 200 to 300x stronger than O_2, as mentioned, and by occupying bonds for oxygen it decreases its binding capacity. Carbon monoxide causes affinity growth and shifts the dissociation curve to the left, causing poorer oxygen release to the tissues [Ward, Linden 2010]. It binds to the heart muscle, brain tissue and liver in greater amounts than to other organs, causing death by suffocation at higher concentrations. From the reversible bond to haemoglobin (and other

substances) carbon monoxide can be displaced by oxygen according to the ratio of their partial pressures [Henry, Satran, Lindgren et al. 2006, Onal, Celik, Aslanlar et al. 2016].

Aim of the work

The aim of the work is to analyze the problem of occurrence of CO in the blood of practitioners during specifically focused CHK and OHK trainings, and subsequently to identify possible occurrence levels with their predictive possibilities in performance and top athletes.

Material and Method

The intentional research sample consisted of 7 master-degree performance karate practitioners (n = 7/6 men and 1 woman), age = 29,14±8,48 years, body height = 177,14±9,48cm, body weight input CHK = 77,63±10,49kg, body weight input OHK = 78,71±10,45 kg). Training unit (TU) CHK is focused on specific relaxation and concentration, TU OHK is more loose and rather represents sport karate with more tension phase. In TU, identical principles were followed as far as possible.

We investigated physiological, biochemical, and other parameters at input and output. Dynamic venous blood was collected, at input from the left forearm, at output from the right forearm, capillary blood was separated from this blood. Laboratory data of biochemical character were determined in the specialized biochemical laboratory Synlab Slovakia a.s. Principle of determination of inorganic elements in blood – potentiometry, indirect ISE. The measurement principle of acid-base balance is potentiometry. Comparison of results at inputs and outputs of CHK and OHK by means of Mann-Whitney non-parametric U-test, which cannot be used to assume a normal probability distribution of the mentioned trait, where the significance level was monitored at levels of $p<0,01$, $p<0,05$. To ascertain the significance of effect of stimulus in CHK and OHK, we used Vilcoxon non-parametric test of difference at input and output at the level of $p<0,01$, $p<0,05$. Dependent variables in their distribution of values on the Gaussian distribution curve were also observed (skewness, kurtosis). More details in [Pivovarník 2018].

RESULTS

The measured results are included in the following tables.

Table 1 Changes of COHb (%) in probands

Proband	**input CHK**	**output CHK**	**input OHK**	**output OHK**
JP	0,9	0,5 ↓	1	0,9 ↓
PB	0,9	0,7 ↓	0,7	0,9 ↑
JV	*2,6*	1,4 ↓	*2,5*	1,5 ↓
LK	*3,1*	1,5 ↓	1,5	0,9 ↓
JK	1,4	0,9 ↓	1,1	0,9 ↓
DK	*2,6*	*1,8* ↓	*2,9*	*2,0* ↓
PP	0,8	0,8 =	0,7	1,1 ↑

Caption: norm venous blood 0,5 – 1,5 %

Table 2 Changes of COHb (%), statistical values

	mean	**median**	**St. deviation**	**skewness**	**kurtosis**
Input CHK	1,76	1,4	0,91	0,28	1,32
Output CHK	1,09 ↓	0,9 ↓	0,45	0,29	1,62
Input OHK	1,49	1,1	0,81	0,72	1,91
Output OHK	1,17 ↓	0,9 ↓	0,4	1,21	2,95

Table 3a Results of Vilcoxon test OHK

PairofVariables	performance=OHK WilcoxonMatchedPairs Test (statistpivovarnik) Markedtests are significantat p <,05000			
	Valid N	T	Z	p-value
COHb & COHb 2	7	6,500000	1,267731	0,204895

Table 3b Results of Vilcoxon test CHK

PairofVariables	performance=CHK WilcoxonMatchedPairs Test (statistpivovarnik) Markedtests are significantat p <,05000			
	Valid N	T	Z	p-value
COHb & COHb 2	6	0,00	2,201398	0,027709

Level of significance of COHb on the level of p<0,028 – CHK training (input/COHb – output/COHb 2).

Table 4 Changes of HCO_3^- ($mmol.l^{-1}$), statistical values

	mean	**median**	**St. deviation**	**skewness**	**kurtosis**
Input CHK	27,87	27,9	1,06	1,04	3,1
Output CHK	28,76 ↑	29,0	1,99	-1,51	4,04
Input OHK	29,36	29,1	2,26	-0,44	2,17
Output OHK	23,8 ↓	24,4	3,91	-0,38	1,76

Caption: norm 22 – 26 $mmol.l^{-1}$

Table 5 Changes of pO_2 (kPa), statistical values

	mean	**median**	**St. Deviation**	**skewness**	**kurtosis**
Input CHK	3,91	3,91	0,46	0,17	2,90
Output CHK	2,95 ↓	2,75	0,49	0,23	1,59
Input OHK	3,88	3,59	0,77	0,89	2,59
Output OHK	6,24 ↑	5,25	2,31	0,95	2,92

Caption: norm 8,00 – 14,00 kPa

Table 6 Interpretation of ABB disorders based on pCO_2 and HCO_3^-, according to Droste,Planta (1992)

Proband	**Input CHK**	**output CHK**	**input OHK**	**output OHK**
JP	RAC	MAL and RAC	MAL and RAC	NORMAL
PB	RAC	MAL and RAC	MAL and RAC	RAC to NORMAL
JV	MAL and RAC	MAL and RAC	MAL and RAC	NORMAL
LK	RAC (almc NORMAL)	RAC (almost NORMAL)	MAL and RAC	RAC to NORMAL
JK	NORMAL	RAC(borderline MAL)	RAC	MAC and RAL
DK	RAC	RAC	RAC	NORMAL
PP(žena)	RAC	NORMAL	RAC	MAC and RAL

Caption: RAC – respiratory acidosis, RAL – respiratory alkalosis, MAC – metabolic acidosis, MAL – metabolic alkalosis

Table 7 Difference of pCO_2 pressure on input and output of TU (kPa)

	mean	**median**	**St.deviation**	**skewness**	**kurtosis**
Difference CHK	0,14	0,11	0,51	-0,64	2,69
Difference OHK	-1,71	-1,73	0,57	0,43	2,19

Table 8 Difference between cations and anions in blood sample before and after training unit. Monitored cations – Na^+, K^+, Ca^{2+}, monitored anions – Cl^-, HCO_3^- , data in $mmol.l^{-1}$

	mean	**median**	**St.deviation**	**skewness**	**kurtosis**
Difference catio CHK	2,29	2	1,58	0,04	2,14
Difference catio OHK	3,14 ↑	3	0,83	1,21	4,04
Difference anion CHI	1,57	1	1,92	0,13	1,37
Difference anion OHI	-2,43 ↓	-1	2,38	-1,71	4,35

SUMMARY

Level of significance of COHb on the level of standardized level of $p<0,05$ – CHK training (input – output), shows us this training unit as more advantageous within our focus, with a more favourable buffering system, which may be a diagnostic predictor (Table 1, 2, 3a, 3b). In Table 4, we can see the work of the main buffer system HCO_3^-, which increases in TU CHK. The pO_2 pressure (Table 5) is significantly reduced in TU CHK, compared to TU OHK, while the values in both are below the norm. We would expect breathing by CHK system to be more advantageous, where some specific regulation takes place, but in concurrence with tension, and regulation of mental nature and psychic concentration, a particular mix of respiratory and metabolic acidosis, respectively alkalosis, takes place (Table 6). It even seems to be preferable to breath in an unregulated way of OHK, where we reach the normal state more often. We can see it hinted, that RAC is likely to be the result of a combination of specific breathing and muscle relaxation, which helps to some liquidity of CO_2 in the body, and it consequently manifests also on adjustment of CO (COHb) level. This is reflected in the statistical significance of COHb change at $p<0,05$ in TU CHK. This suggests that not only breathing, but in particular some specific movement and adjustment of muscle tone to relaxation may be a decisive factor, therefore a certain mental setting.

This is subsequently confirmed by the values of change of pCO_2 in TU input and output (Table 7). An ionogram of cation and anion differences indicates the opposite trend in the monitored TU CHK and OHK, where we also follow the opposite trends of change (Table 8).

From this it can be concluded that not only does specific breathing affect the possibilities of the body of performance and top athletes, but so does also mental setting with influence on relaxation of the body on a conscious and unconscious level.

CONCLUSIONS

By using specific breathing in karate in connection with a certain mental setting of the organism, limited muscle and tendon tension, this specific activity manifests itself in the monitored variables. In our particular case, we focused on CO in the body, where it was confirmed that it is possible to monitor carbon monoxide in venous blood, and its level, respectively change, may indicate the technical level or physical condition possibilities of the karate practitioner. The problem is that nowadays the traditional qualitative methods of the training process are disappearing and we focus more on the quantitative side. There are few specialists in such focus, as the sporting period of athletes is relatively short, and so the training does not involve the adoption of such original methods. During OHK sports training the statistical significance was not confirmed, in the CHK system training the statistical significance was confirmed. Importantly, however, there has been a clear demonstration of different involvement of the buffer system of the organism of karate athletes, which also leads to different results of the monitored variables, with further possibility to use them for prediction.

REFERENCES

1. Denshaw-Burke M., Savior D. (2009), *Methemoglobinemia*. Medical College of Pensylvania, Oct. 4
2. Droste C., Planta M. (1992), *Memorix vademecum lékaře*. Scientia Medica, spol.s r.o., Praha [in Czech].
3. Dobiáš V. (2007), *Intoxikácia oxidom uhoľnatým a oxidom uhličitým*. Via practica. 9, pp. 422–423 [in Slovak].
4. Dzurik R., Trnovec T. (1997), *Štandardné terapeutické postupy*. Osveta, Martin [in:] Roche Slovensko s.r.o.: OMNI S - Návod k obsluze, 2003 [in Czech].
5. Henry C.R., Satran D., Lindgren B., et al. (2006), *Myocardial injury and long-term mortality following moderate to severe carbon monoxide poisoning*. JAMA, 295, 4, pp. 398-402.
6. Lékařský slovník. (2019), Available from URL: http://lekarske.slovniky.cz/lexikon-pojem/oxid-uhlicity-co2 [date of access: 03.09.2019] [in Czech].
7. Onal O., Celik D., Aslanlar E., et al. (2016), *Myocardial Infarction in a Young Patient After Acute Exposure to Carbon Monoxide*. Anesth Pain Med.6;3.
8. Pivovarník J. (2018), *Dual concentration in karate and its possible use in sport diagnostics*. LAP LAMBERT Academic Publishing.
9. Silbernagl S., Despopoulos A. (1984). *Atlas fyziologie člověka*. Avicenum, Praha [in Czech].
10. Šašinka M., et al. (2003), *Vademecum medici*. Osveta, Martin [in Slovak].
11. Ward J.P.T., Linden R.W.A. (2010), *Základy fyziologie*. Galén, Praha [in Czech].