

Red blood cell (RBC) arteriovenous gap as a marker of hemotransfusion in massive and fulminant hemorrhage with the formation of Multiple Organ Dysfunction Syndrome (MODS). Case report

Ilie Vasiliev^{1,7} ORCID: <https://orcid.org/0000-0002-8962-2927> , **Irina Vasilieva**^{1,4,5,6}
ORCID: <https://orcid.org/0000-0001-7019-4443> , **Maria Vasilieva**^{1,2,3,6,7} ORCID:
<https://orcid.org/0000-0003-4588-2716>

¹State University of Medicine and Pharmacy” Nicolae Testemițanu”.

²”Nicolae Testemițanu” University Clinic of Primary Medical Assistance of State University of Medicine and Pharmacy.

³Laboratory of Neurology and Medical Genetics Republic of Moldova.

⁴Department of Laboratory Medicine. State University of Medicine and Pharmacy” Nicolae Testemițanu”

⁵”Timofei Moșneaga” Republican Clinical Hospital, Republic of Moldova.

⁶Institute of Emergency Medicine. Republic of Moldova.

⁷World Academy of Medical Sciences, Netherlands, Republic of Moldova.

Scientific adviser: Professor Dr Ilie Vasiliev, MD, Academy Professor World Academy of Medical Sciences. First Superior Executor Vice - President of the World Academy of Medical Sciences. Chairman of the World Council of the Academy WAMS. WAMS' International Medical Research Council (IMREC). The Chairman of the WAMS National Council of Moldova. Netherlands, Republic of Moldova.

<https://wams.online/about-us/>

https://wams.online/our_team/dr-ilie-vasiliev-md/

Keywords: Biomarker; Arteriovenous AV Gap blood RBC - marker; venous (V) blood RBC; arterial (A) blood RBC; Multiple organ dysfunction syndrome; Acute Respiratory Distress Syndrome; Microcirculatory Mitochondrial Distress Syndrome; pO₂ (AV gap); Electro-Ion Membrane Distress Syndrome; Recruitment of microcirculatory – mitochondrial control strategy.

Goal of the study: Arteriovenous Gap (AV Gap) as a universal test of specific prognostic markers of the development of MODS in extremely in massive, fulminant hemorrhage, hemorrhagic shock caused by polytrauma and of volume of blood loss more than 3 liters. To prove the medico-legal indication of blood transfusion and the cause of the difference in arteriovenous erythrocyte (Hb (RBC AV gap) with RBC venous blood (V), first analysis (VHct 36%; VtHb 12.3 g/dL), which does not suggest blood transfusion, however, arterial blood (A) third analysis, data (AHct < 12%; AtHb reduced g/dL) justify red blood cell transfusion. Thus, the hypothesis of successful resuscitation of cardiac arrest by Academician V. Negovschi by intra-arterial administration under pressure of RBC, here we have proven it as an axiom.

Summary: The intensive care unit (ICU) needs fast and effective methods of diagnosis, resuscitation and restoration of lost functions or maintenance of dying functions, tenatogenesis in clinical death and Multiple organ dysfunction syndrome (MODS) [1,2]. Contemporary assessment of the molecular pathological physiology of the critically ill patient with MODS requires urgent determination of the level of disorders of the

respiratory and cardiovascular systems. In this direction, the determination of blood gases and acid-base homeostasis will be of significant importance. In scientific practical medical literature, disorders of the respiratory and cardiovascular systems are designated Acute Respiratory Distress Syndrome (ARDS) and Microcirculatory Mitochondrial Distress Syndrome (MMDS) [3]. Confirmation of these serious conditions requires targeted treatment [4].

Introduction: The critical - terminal states install the centralization of the macro - circulation and trigger MODS, blocking the microcirculation at the level of the cellular capillary metabolic space. With MODS, with an increase in $\uparrow p\text{CO}_2$, caused by pulmonary / extrapulmonary ARDS and confirmed by the fall in the oxygenation index $\downarrow \text{PaO}_2 / \text{FiO}_2 \downarrow 300$ in the context of the Berlin 2012 classification, violations of pathologies of gas exchange are also taken into account: 1) Lung gas exchange: a) acute respiratory failure - \downarrow end-tidal CO_2 fraction ($\text{FetCO}_2 \downarrow$), $\text{SaO}_2 \downarrow$, $\text{PaO}_2 \downarrow$, $\text{FiO}_2 \downarrow$; b) parenchymal (endothelial-epithelial disorder to alveolar and vascular tissue) - $\text{FetCO}_2 \downarrow$ / or normal, $\text{SaO}_2 \downarrow$, $\text{PaO}_2 \downarrow$; 2) transportation of gas in the blood (minute volume) \downarrow , $\text{Hb} \downarrow$, $\text{SvO}_2 \downarrow$, $\text{PvO}_2 \downarrow$, avSO_2 , avPO_2 ; 3) Gas exchange in tissues - $\text{SvO}_2 \uparrow$, $\text{BE} \uparrow$, $\text{PvO}_2 \uparrow$, $\text{avSO}_2 \downarrow$, $\text{avPO}_2 \downarrow$; lactate / pyruvate \uparrow [5,6]. Pyruvates (salts of pyruvic acid) are a product of the anaerobic metabolism of glucose, known as glycolysis. Pyruvic acid supplies energy to cells via the citric acid cycle (also known as the Krebs cycle) in the presence of oxygen (aerobic respiration), and in the absence of oxygen it is fermented by lactate dehydrogenase (LDH) to form lactate (lactic acid) [7]. With sufficient oxygen supply, pyruvate is further metabolised in the mitochondria to H_2O and CO_2 . If the difference $p\text{CO}_2$ (AV gap) > 6 mmHg., is of extrapulmonary origin, then this is considered as a marker of tissue hypoxia, allowing the development of MMDS to be assessed.

Material and methods:

We presented a described marker of tissue hypoxia, with a difference $p\text{CO}_2$ (AV gap) > 6 mmHg, at the International Conference on Biotechnology, Biomarkers, Systems Biology (2019). Amsterdam, Netherlands. The Republic of Moldova had a special honor, since the Session Chair of the Conference was Professor DR Ilie Vasiliev represented by the World Academy of Medical Sciences, Netherlands, in collaboration with Japan, where the Session Co-Chair was Brilliant Professor DR Hitoshi Sohma (Sapporo Medical University Center for Medical Education, Japan) [8]. CO_2 is formed in mitochondria, and in cases of undamaged lungs and when there is an arterio-venous difference $p\text{CO}_2$ AV gap $p\text{CO}_2 > 6$ mm Hg, MMDS is established. The difference in $p\text{CO}_2$ between arterial $p\text{ACO}_2$ and venous $p\text{VCO}_2$ indicates the balance between CO_2 elimination through the lungs and its production by tissues. This arteriovenous difference $p\text{CO}_2$ ($\text{PaCO}_2 - \text{PvCO}_2$) is determined by the difference $p\text{CO}_2$ gap or $\Delta p\text{CO}_2$ and is considered a marker of tissue hypoxia, if difference $p\text{CO}_2$ (AV gap) > 6 mmHg. Using the ratio of $p\text{CO}_2$ (AV gap) to $p\text{O}_2$ (AV gap) according to the formula $[\text{pCO}_2 \text{ (AV gap)} / \text{pO}_2 \text{ (AV gap)}]$ more informatively reflects the ejection cardiac fraction of output between Oxygen delivery (DO_2) and VO_2 ($\text{VO}_2 \text{ max}$ is the maximum (max) velocity, V of oxygen, O_2) consumption, which is important for adequate O_2 extraction by cells, which mechanism is seriously disrupted in critical conditions. The $[\text{pCO}_2 \text{ (AV gap)} / \text{pO}_2 \text{ (AV gap)}]$ ratio can replace the respiratory quotient (RQ), which is the ratio of $\text{VCO}_2 / \text{VO}_2$ (carbon dioxide production/oxygen consumption).

The [pCO₂ (AV gap) / pO₂ (AV gap)] ratio has been identified as a mortality biomarker for patients with severe ARDS.

Possible calculations of RBC, pCO₂ (AV gap) / pO₂ (AV gap) which are tangential with the Oxygen transport and gas exchange parameters described below.

Volumetric concentration of oxygen in arterial blood (CaO₂) = (0.0139 x SaO₂, % x Hb, g%) + (0.0031 x PaO₂, mm Hg) Norm 18-21 vol%

Volume concentration of oxygen in mixed venous blood (CvO₂) = (0.0139 x SvO₂, % x Hb, g%) + (0.0031 x PvO₂, mmHg) Norm 14.5 - 15.5 vol%

Volumetric concentration of oxygen in capillary blood (Ccap O₂) = (1.39 x Hb, g%) x (0.0031 x PaO₂ mmHg) (sO₂ in capillary blood is taken to be 100%)

Arteriovenous difference in volume concentration of oxygen (Ca-vO₂) Norm 3.5-5.5 vol%

Oxygen consumption = (cardiac output, ml/min) x Ca-vO₂, vol%/100. Norm 200 - 250 ml/min

Oxygen delivery to tissues = (cardiac output, ml/min) x CaO₂, vol%/100. Norm 1100-1300 ml/min

Oxygen extraction = (oxygen consumption) / (oxygen delivery to tissues). Norm 0.22 - 0.32

Fick equation: (cardiac output) = (oxygen consumption, ml/min) / [hemoglobin, g% x 0,00139 x (Sa-vO₂, vol%)] Normal 3-7 l/min

Respiratory quotient = (CO₂ release) / (O₂ consumption) Norm 0.7 - 1.0

Shunt fraction (QS/QT - the ratio of shunt blood flow to total pulmonary blood flow) = CcapO₂ / (CcapO₂ - CvO₂), sO₂ in capillary blood is taken equal to 100% Norm < 5%

Results: We have covered the successful treatment of ARDS and MMDS in different countries [3-5, 9-14]. The result was due to decentralization, anti-shock therapy, detoxification and analgesia in the Recruitment of microcirculatory – mitochondrial control strategy, supplemented by Multiple Organ Support Therapy - Extracorporeal Life Support Organization (MOST-ELSO) (Extracorporeal membrane oxygenation (ECMO) and Extracorporeal carbon dioxide removal (ECCO2R), etc.) and in combination with antibacterial / antifungal [15]/ antiviral treatment and surgical correction [16], counteracts the MMDS mitochondrial collapse, and regression of MODS [5,17]. Respiratory, cardiac and cerebral support, has justified itself not only in critical-terminal obstetrics, but also in oncological MODS, with massive injuries and bleeding [2,18,19] and even with infection coronavirus SARS-Cov2 / COVID19 and other viral infections [20-22], where Maria and Irina Vasilieva syndrome (Electro-Ion Membrane Distress Syndrome induces Chronic Fatigue Syndrome/Myalgic Encephalomyelitis, CFS/ME) is significant [23]. The absence of a continuous decrease in the pCO₂ marker of tissue hypoxia AV gap <5 mmHg proves the suspension of the continuation of cell necrosis/apoptosis, hypo- (a) energy and confirms mitochondrial euenergetic metabolic remodeling with the elimination of mitochondrial hypo (an) energy, active lysosomal clearance (mitophagy), thus supporting the presence of euenergetic mitochondria with the normalization of mitochondrial Ca ++ - channel uniporter and cyclosporine-sensitive mitochondrial pore (mitochondrial permeability pore transition), with beneficially productively inactivate Reactive oxygen species (ROS) and Reactive nitrogen species (RNS) [24-28].

1. Venous blood acid-base balance (first analysis)

| <u>Test</u> | <u>Valoare</u> | <u>Unitati</u> |
|-------------------|----------------|----------------|
| pH | 6.983 | |
| pCO ₂ | 69.2 | mmHg |
| pO ₂ | 36.9 | mmHg |
| SO ₂ | 51 | % |
| Hct | 36 | % |
| tHb | 12.3 | g/dL |
| Na | 143.3 | mmol/L |
| K | 4.22 | mmol/L |
| Cl | 111.3 | mmol/L |
| iCa | 1.21 | mmol/L |
| iMg | 0.75 | mmol/L |
| Glu | 10.6 | mmol/L |
| Lac | 8.2 | mmol/L |
| O ₂ Hb | 49.3 | % |
| COHb | 2.5 | % |
| MetHb | 0.5 | % |
| HHb | >40.0 | % |
| tBil | 191.5 | umol/L |
| HbF | 0.9 | % |
| TCO ₂ | 18.7 | mmol/L |

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Calculat

| <u>Test</u> | <u>Valoare</u> | <u>Unitati</u> |
|-----------------------------------|----------------|----------------|
| BE-ecf | -15.2 | mmol/L |
| BE-b | -14.9 | mmol/L |
| SBC | 12.5 | mmol/L |
| HCO ₃ | 16.57 | mmol/L |
| O ₂ Cap | 17.1 | mL/dL |
| O ₂ Cl | 8.8 | mL/dL |
| A | 63.3 | mmHg |
| A-aDO ₂ | 26.4 | mmHg |
| a/A | 0.6 | |
| P ₅₀ | 23.5 | mmHg |
| RI | 0.7 | |
| pO ₂ /FIO ₂ | 176.8 | |
| nCa | 1.01 | mmol/L |
| nMg | 0.61 | mmol/L |
| Gap(K) | 19.7 | mmol/L |
| nCa/nMg | 1.7 | mol/mol |
| CcO ₂ | 16.5 | mL/dL |
| ePV | 5.203 | dL/g |
| MCHC | 34.4 | g/dL |

2. Venous blood acid-base balance (second analysis)

| <u>Test</u> | <u>Valoare</u> | <u>Unitați</u> | |
|-----------------------------------|----------------|----------------|-----|
| pH | 7.131 | | |
| pCO ₂ | 66.6 | mmHg | |
| pO ₂ | 31.4 | mmHg | |
| SO ₂ | 41 | % | |
| Hct | 34 | % | |
| tHb | 11.5 | g/dL | |
| Na | 141.3 | mmol/L | |
| K | 4.80 | mmol/L | |
| Cl | 111.5 | mmol/L | |
| iCa | 1.22 | mmol/L | |
| iMg | 0.64 | mmol/L | |
| Glu | 7.8 | mmol/L | |
| Lac | 6.2 | mmol/L | |
| O ₂ Hb | 40.2 | % | |
| COHb | 1.8 | % | |
| MetHb | 0.3 | % | |
| HHb | >40.0 | % | 111 |
| tBil | 186.6 | umol/L | |
| HbF | 0.5 | % | |
| TCO ₂ | 24.4 | mmol/L | |
| ----- | | | |
| Calculat | | | |
| <u>Test</u> | <u>Valoare</u> | <u>Unitați</u> | |
| BE-ecf | -7.0 | mmol/L | |
| BE-b | -6.8 | mmol/L | |
| SBC | 17.9 | mmol/L | |
| HCO ₃ ⁻ | 22.39 | mmol/L | |
| O ₂ Cap | 16.0 | mL/dL | |
| O ₂ Ct | 6.7 | mL/dL | |
| A | 66.2 | mmHg | |
| A-aDO ₂ | 34.8 | mmHg | |
| a/A | 0.5 | | |
| P ₅₀ | 26.8 | mmHg | |
| RI | 1.1 | | |
| pO ₂ /FIO ₂ | 150.2 | | |
| nCa | 1.07 | mmol/L | |
| nMg | 0.54 | mmol/L | |
| Gap(K) | 12.3 | mmol/L | |
| nCa/nMg | 2.0 | mol/mol | |
| CcO ₂ | 15.6 | mL/dL | |
| CaO ₂ | 6.5 | mL/dL | |
| ePV | 5.739 | dL/g | |
| ΔPV | 10.3 | % | |
| MCHC | 34.2 | g/dL | |

3. Arterial blood acid-base balance (third analysis)

| <u>Test</u> | <u>Valoare</u> | <u>Unitați</u> |
|-------------------|----------------|----------------|
| pH | 7.432 | |
| pCO ₂ | 19.5 | mmHg |
| pO ₂ | 187.5 | mmHg |
| SO ₂ | Suspect | % |
| Hct | <12 | % |
| tHb | Scăzut | g/dL |
| Na | 129.2 | mmol/L |
| K | 3.43 | mmol/L |
| Cl | 121.3 | mmol/L |
| iCa | 0.28 | mmol/L |
| iMg | 0.17 | mmol/L |
| Glu | 6.4 | mmol/L |
| Lac | 2.2 | mmol/L |
| O ₂ Hb | Suspect | % |
| COHb | Suspect | % |
| MetHb | Suspect | % |
| HHb | Suspect | % |
| tBil | Suspect | umol/L |
| HbF | Suspect | % |
| TCO ₂ | 13.7 | mmol/L |

| Calculat | | |
|-----------------------------------|----------------|----------------|
| <u>Test</u> | <u>Valoare</u> | <u>Unitați</u> |
| BE-ecf | -11.4 | mmol/L |
| BE-b | -8.0 | mmol/L |
| SBC | 18.1 | mmol/L |
| HCO ₃ | 13.11 | mmol/L |
| A | 122.7 | mmHg |
| a/A | 1.5 | |
| pO ₂ /FIO ₂ | 897.3 | |
| nCa | 0.29 | mmol/L |
| nMg | 0.17 | mmol/L |
| nCa/nMg | 1.7 | mol/mol |
| SO ₂ | 100 | % |

4. Venous blood acid-base balance (fourth analysis)

| <u>Test</u> | <u>Valoare</u> | <u>Unitati</u> |
|-------------------|----------------|----------------|
| pH | 7.464 | |
| pCO ₂ | 29.7 | mmHg |
| pO ₂ | 73.6 | mmHg |
| SO ₂ | 98 | % |
| Hct | 24 | % |
| tHb | 9.2 | g/dL |
| Na | 138.3 | mmol/L |
| K | 4.57 | mmol/L |
| Cl | 112.9 | mmol/L |
| iCa | 1.10 | mmol/L |
| iMg | 0.52 | mmol/L |
| Glu | 7.5 | mmol/L |
| Lac | 1.8 | mmol/L |
| O ₂ Hb | 94.8 | % |
| COHb | 2.7 | % |
| MetHb | 0.3 | % |
| HHb | 2.2 | % |
| tBil | 101.3 | umol/L |
| HbF | 0.1 | % |
| TCO ₂ | 22.4 | mmol/L |

| <u>Calculat</u> | | |
|-----------------------------------|----------------|----------------|
| <u>Test</u> | <u>Valoare</u> | <u>Unitati</u> |
| BE-ecf | -2.4 | mmol/L |
| BE-b | -1.0 | mmol/L |
| SBC | 23.6 | mmol/L |
| HCO ₃ | 21.53 | mmol/L |
| O ₂ Cap | 12.4 | mL/dL |
| O ₂ Ct | 12.7 | mL/dL |
| A | 110.1 | mmHg |
| A-aDO ₂ | 36.5 | mmHg |
| a/A | 0.7 | |
| RI | 0.5 | |
| pO ₂ /FIO ₂ | 352.3 | |
| nCa | 1.14 | mmol/L |
| nMg | 0.55 | mmol/L |
| Gap(K) | 8.5 | mmol/L |
| nCa/nMg | 2.1 | mol/mol |
| CcO ₂ | 12.5 | mL/dL |
| ePV | 8.261 | dL/g |
| MCHC | 38.5 | g/dL |

Translation from romanian to english: scazut → reduced; calculat → calculating; valoare → value; unitati → unity; suspect → suspect

Case report. In the context of arteriovenous differences, we will present a clinical case where you can properly familiarize yourself with the pCO₂ difference (AV gap), as a marker, which confirms MMDS. According to the attached acid-base homeostasis and blood gases. The use of Recruitment of microcirculatory – mitochondrial control strategy reduced pCO₂ (AV gap) 49.7 (mmHg)→ 10.2 mmHg, and therefore, fatal acidosis (pH 6.9 → 7.4; pCO₂ 69→ 29 (mmHg); pO₂ 36→73 (mmHg); SO₂ 51→ 98 (%); BE-ecf - 15.2 → - 2.4 (mmol/l); BE-b (-14,9) → (- 1) mmoli/l; AV(K) 19.7 → 8.5 (mmol/l). Oxygenation index from 176.8 → 352.3. MMDS, according to arteriovenous difference PCO₂ (AV gap) or Δ PCO₂ = PaCO₂ – PvCO₂ = 69.2 -19.5 = 49.7 (mmHg) confirmed, since pCO₂ (AV gap) > 49.7 (mmHg), and after Recruitment of microcirculatory – mitochondrial control strategy it decreased to 10.2 mmHg. PCO₂ (AV gap), since, Δ PCO₂ = PaCO₂ – PvCO₂ = 29.7 - 19.5 = 10.2 mmHg. In this pathological biochemical context, we were able to reduce lactate from (V) 8,2 mmol/l → (V) 6,2 mmol/l → (A) 2,2 mmol/l → (V) 1,8 mmol/l by Recruitment of microcirculatory – mitochondrial control strategy. It should be noted that elevated lactate levels are a marker of cerebral hypoxia, confirming decreased cerebral blood flow or ischemia and the above-described MMDS or decreased metabolism with a concomitant decrease in brain pyruvate levels. Lactic acid levels are considered high if they range from 2 to 4 mmol/l and pH is below 7.25, and are regarded as hyperlactatemia or lactic acidosis. An increase in lactate to a concentration exceeding 5 mmol/l and a decrease in pH below 7.25 can confidently indicate lactic acidosis. However, and other a difficult situation arose when the visual blood loss of more than 3 liters did not correspond to the data of venous red blood (venous VHct 36%; VtHb 12.3 g/dL). For the protocol implementation of red blood cell transfusion, the basis was the arteriovenous difference in red blood (arterial blood AHct < 12%; AtHb low g/dL) against (VHct 36%; VtHb 12.3 g/dL). After the red blood cell transfusion → VHct 24%; VtHb 9.2 g/dL. Thus, it was possible to prove the indications for blood transfusion, based on the Arteriovenous Gap of Red Blood Cell (RBC) marker. Have not encountered such a method in the medical literature as an indication for hemotransfusion in difficult situations. But we decided it exclusively in the interests of the patient.

Conclusion:

Detectable Systemic Perfusion Pressure (SPP) in capillary resistance pressure (CPR) in ICU is the gold standard not limited in time compared to the definition of the Hemodynamic Support Algorithm used by the ARDS Network in the “Prospective, Randomized, Multi-Center Trial of (Fluid Conservative” vs. (Fluid Liberal) Management of Acute Lung Injury and ARDS to determine, Pulmonary artery wedge pressure, Cardiac index, Pulmonary diastolic pressure. Volemic resuscitation, where instead of permissive hypoperfusion provided by protocols, was guided by permissive SPP, with prevention of dilutional coagulopathy with limitation of fluid use. Of the fluids, intravenous Ringer was preferred. We draw attention to the fact that the permissive SPP solved disputes about large or low liquid volume Eliminating, thus, the problems of hyper or hypo hydration. Using arteriovenous gap markers pCO₂, K⁺, pO₂, Lactate, pRBC, confirming MMDS, blocking microcirculation with sequestration of its

contents, and the transition of metabolism to anaerobic glycolysis with an increase in lactate accelerating brain death and tenatogenesis. Using Recruitment of microcirculatory – mitochondrial control strategy suspended the development of MMDS, evidenced improved by acid-base homeostasis, after recruitment of microcirculation. Also, in 2007, we noted the successful stabilization of the “metabolic stage” of Coagulation Resuscitation, while we are still far from the methods of “no fluid resuscitation” or “low-volume resuscitation” [2]. The decisive one in the dispute, in this case, was SPP [2,5,12,29] based on AV gap markers: pCO₂, pK⁺, pO₂, pLactate, pRBC.

The microcirculatory-mitochondrial recruitment contributes to the reduction of the tissue hypoxia marker pCO₂ at the A-V difference, rejects necrosis / apoptosis, cellular hypo- (an) energetic and allows mitochondrial eu-energetic metabolic remodeling by eliminating hypo - (an)- energetic mitochondria carried out by lysosomal clearance (mitophagy), thus demonstrating eu-energetic mitochondria with the normalization of the uniporter-Ca ++ mitochondrial pore transition and mitochondrial permeability, which productively inactivates toxic forms of oxygen and nitrogen.

With some elementary technologies and simple maneuvers to balance multiple changes, without the need for sophisticated technologies, it is possible to reduce Thus the mortality of the critically - patient [4-6, 30-33]. With the simultaneous rapid development of technocratization and robotization of medicine, mathematical-geometric models of research in medicine and artificial intelligence , where throughout the main thing will remain private intellectual property, as Saint Exupery spoke about [34-49].

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Professor Anatolie Vișnevschi Head of the Department of Laboratory Medicine of the "Nicolae Testemițanu" State University of Medicine and Pharmacy. Member of the

Faculty of the World Academy of Medical Sciences, WAMS (Faculty of the Academy), Head of the Laboratory Medicine Department of WAMS. President of the WAMS International Council of Laboratory Medicine. Member of the General Council of the World Academy of Medical Sciences (World Council of Medical Sciences). Member of the WAMS Committee of the Republic of Moldova (WAMS Moldovan Council) https://wams.online/our_team/dr-anatolie-visnevschy-md/ <https://wams.online/about-us/>

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