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Role of pCO_2 (AV gap) of Multi Organ Dysfunction Syndrome

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Abstract

Recruitment of Microcirculatory-Mitochondrial (RMM) reduces Microcirculatory-Mitochondrial Distress Syndrome (MMDs), and Syndrome of Multi-Organ Dysfunction (MODs), by accelerated speed of delivery and return of blood flow which directly leads to a decrease in tissue hypoxia marker pCO_2 (AV gap) and respectively with ↓ many other Endogenous Toxic Substances (ETS).

In cases of pulmonary damage with ↑ pCO_2 & ↓ Oxygenation Index PaO_2/FiO_2 ↓ 300 the development of Acute Respiratory Distress Syndrome (ARDs), MMDs are also aggravated at ↑ with pCO_2 AV gap. RMM also needs additional support of Multiple Organ Therapies-Multi-Organ Supportive Therapy (MOST), Alveolar Recruitment, Extracorporeal Life Support Organization (ELSO), Modeling of the Index of Extravascular Lung Fluid, EVLWI, Th_4- Th_5 Thoracic Epidural Block, Active detoxification methods.

The absence of decreasing of the pCO_2 tissue hypoxia marker at the pCO_2 AV gap ↓ 5.0 mmHg, after RMM proves the mitochondrial eu-energetic metabolic remodeling with the elimination of the hypo(an)ergic mitochondria performed by lysosomal clearance (mitophagy) makes the predominance eu-ergic mitochondria with the normalization of mitochondrial Ca^{++}-uniporter-channel and mitochondrial permeability pore transition which productively inactivate the toxic forms of oxygen and nitrogen.

Keywords: Microcirculatory-Mitochondrial Distress Syndrome; Microcirculatory-Mitochondrial Recruitment; Multiple Organ Dysfunction Syndrome; Marker of Tissue Hypoxia pCO_2 (AV gap); Acute Respiratory Distress Syndrome; Multi Organ Supportive Therapy; Mitochondral Remodeling; Mitochondrial Ca^{++} Uniporter Channel; Toxic forms of Oxygen and Nitrogen; Nascular Compliance ΔVP

Introduction

Case studies

Obstetric mortality was absent for more than 35 years of emergency anesthesiology and resuscitation in Moldova, Central Asia and Russia and on air ambulance with critical [1] situations in obstetrics, Massive Hemorrhagic Shock; Disseminated Intravascular Coagulation Syndrome (DICs); HELLP; Eclampsia; Rupture of Cerebral Aneurysm [2]; Coma; Swelling (anasarca); SIRs; CARs; Septic Shock; MODs; Pulmonary Embolism; Urgent Cesarean in former medical rural health facilities under general anesthesia and respiratory support with replacement of blood transfusions of newborns during hyper-bilirubin group conflict through the catheterized umbilical vein otherwise jugular or subclavian successful generic resolution of a pregnant woman with 3rd defects of cardiac valves in the mother-failure and stenosis: aortic, tricuspid and mitral.

RMM had the goal of optimizing vascular compliance ΔVP of microcirculatory perfusion of space: capillars ↔ cell ↔ mitochondria, with accelerated venous return and elimination of CO_2 and other ETS with Energy-Resuscitation of Mitochondrial Collapse (MC) [3,4].

In shock cells, the mitochondria become targets and in a vicious circle further destabilizes the respiratory coefficient, Oxygen-glucose index as an indicator of Energy Metabolism. Since in Mitochondria whose electric potential is~180 mB (electric generator) is associated with oxidative metabolism and generates an electrochemical potential $\Delta\mu H^+$, capable of providing potentially-dependent processes and above all ATP synthesis. Intact channels Ca^{++}-uniporter, interrupt, automatism irregular of bathmotropic networks P-pacemaker myocardium (mitochondria and their energy are entering as a non-

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invasive mitochondrial automatic switch and protect the heart from damage), what is the direct inotropic effect of using ATP or indirectly from cardiac glycosides which increases intracellular Ca^{++} or Ca^{++} ion desensitizers. MMDs are due to accumulation in the tissue space of the pCO_2 AV gap>6 mmHg, which is accepted as a valid marker of tissue hypoxia, provoked by a decrease in perfusion pressure, SPP increasing with a low ejection fraction of the heart. As a result, cell anabolism and respiratory coefficient decrease, and enzyme activity is blocked, with the deterioration of the extraction rate and O_2 by the cells. Disorders of venous return and accumulation of cellular catabolites, further aggravate MC. At the same time, the equilibria are disturbed: iso-osmotic; oncotic; ionic; electric; tonic, acid-base; and also hemostasis rheology, immune-nutritional energy with disorder of microcirculatory compliance ΔVP and aggravation of secondary→hyp oxia→ischemia→acidosis→cell necrosis. MODs is established, in which the detoxification ability of systems/cells/subsystems is inactivated, and the accumulation of ETS increases cell death. Programmed Cell Death (Apoptosis) increases the effect on the mitochondrial potential of the Ca^{++}- Uniporter Channel which opens the damaged pores of the mitochondrial permeability pore transition, and loses the ability to inactivate toxic forms of oxygen and nitrogen [5].

At the same time the immune compromise contributes to the Generalization of Local Infection (LIRs) through SIRs-a cascade of Pro-Inflammatory Cytokines (IL-1, 6, 8, TNF, IFNγ) or anti-inflammatory CARs cytokines (IL 4,10,13) predominating that provoke PICs persistent inflammation immuno-suppression and catabolism syndrome.

Materials and Methods

Detoxification and adequate analgesia [6-8] enhance the strategic management of RMM to decentralize macro-circulation and restore blood flow in the microcirculatory-mitochondrial space to ensure cellular metabolism. Improving the delivery of oxygen and nutrients into the cell, and the elimination of CO_2 and other catabolites from the cell, is carried out by RMM by stabilizing the vascular microcirculatory compliance ΔVP maintained by the systemic SPP (the norm is ~70 mmHg) equal to the difference of the Mean Arterial Pressure MAP (~90 mmHg) Capillary Resistance Pressure CRP (~20 mmHg). MAP defines the monitor, and in its absence is determined according to the formulas. According to Vasilieva study [9] a tear can be used as a diagnostic test for various diseases, and CRP in the practice of the doctor on duty can be compared with intraocular pressure by the Kalmakov method with the exception of oculist glaucoma consultation of which is important and for examining the fundus in these patients.

Calculated examples in patients with hypertension who have high Blood Pressure (BP) numbers, SPP will be higher and the extracellular fluid will shift in the intracellular space and vice versa with low blood pressure the SPP ↓ 70 mmHg will decrease and the liquid on the contrary will go from the extracellular sector to the vascular one. From which it follows that Modifying Vascular Compliance ΔVP can also enhance detoxification. Successful RMM requires the comparison of the MAP with the locally-regional characteristics of the blood circulation by functional organs.

Constancy of ΔVP compliance of the brain is ensured according to the Monroe Kelly Doctrine a balance between cerebral blood flow cerebrospinal fluid and mass of the brain. Cerebral perfusion pressure not less than 100 mmHg, designed to provide a metabolic rate in gray matter at 75 mL/100 g/min, in white 30 mL/100 g/min, and an average of 55 mL/100 g/min. In situations of falling blood flow up to 25 mL/100 g/min, there is a diffuse decrease in the electrical neural activity of the cerebral cortex. And when the blood flow ~15 mL/100 g/min there is a slowdown/disappearance of the bioelectric nervous activity of the cerebral cortex <10 mL/100 g/min irreversible, hypoxic and ischemic cerebral lesions are observed stopped for 8-10 seconds-the consciousness is lost.

SPP modeling is possible due to maneuvering: 1) cardiovascular compliance ΔVP, heart pump and BP: a) Ino-vasoconstrictive effects to maintain the cardiac fraction with ↑ systolic BP, ↑ diastolic BP and ↑ Total Peripheral Vascular Resistance (TPVR) and b) Ino-vasodilation to maintain the cardiac fraction ↑ systolic BP, but with ↓ TPVR, thanks to vasodilation with ↓ diastolic BP 2) Effective Circulating Blood Volume ECBV [10] by means of volemic resuscitation, oncotic pressure, correction of anemia, reologic-resuscitation, equilibration of processes of coagulation- anticoagulation-fibrinolysis (thrombus mass, leukomass, fresh frozen plasma, rFVIIa, aprotinin, antithrombotic, thrombolytic, heparin, including with low molecular masses of 3000-9000) oncotic pressure (proteins, albumin) osmotic pressure and colloid osmotic pharmacological removal of excess fluid by sequestering, translocation and reduction of venous return-spinal/epidural block, ganglio-H- blokators, diuretics 3) CRP is modified by actions points 1 and 2 and pharmacological selective actions at the α1-2, β1-2-3, γ-D1-5, α, β, C, Dopamine adrenoreceptor levels-localized pre, post-synaptic and H cholino-receptors (ganglio-H-blockers). Modification by points 1,2 and 3 modulates the macrocirculation into microcirculation, where blood from the AV shunt-anastomosis is recruited directly into the paralyzed metabolic capillary, thus activating it and to reduce the capillary leakage syndrome where 5% albumin plays a special role [11-15].

Results and Discussion

Pharmacologically taking into account M. Berenbaum drug interactions (zero-additional, supraadititive-potentiation, synergism, infraaditive mistakenly perceived as antagonistic) the function of the heart pump and blood vessels, blood pressure are modified a) Cardiac-inotropic, chronotropic, dromotropic, bathmotropic and lusitropic b) Vascular support BP-with the help of vasoconstrictor and vasodilator agents. Reduce bradycardia and ↑ BP → β and - α - dopamine dependent adrenomimetics and with tachycardia and ↓ BP → α-vasopressors (norepinephrine) in which the adrenomimetics vasoconstrictive effect prevails ↑ BP and causes compensatory bradycardia. Are successfully used at NO-dependent hypotension and Moldovan preparations [16] derivatives of isothiourea, isoturon and difeture (raviten) with vasoconstrictive myotropic action have a hypertensive effect by blocking nitroxide synthetase an enzyme responsible for the synthesis of NO endothelial relaxing factor. In bradycardia with a low cardiac output and high compensatory hypertension due to ↑ TPVR → Dobutamine is optimal with an inotropic β1-mimetic effect which ↑ systolic BP inotropic way, but due to simultaneous vasodilation preload is optimized which improves venous return and reduces pulmonary hypertension preventing pulmonary edema as a result post-load is also improved creating an almost perfect SPP. Close to dobutamine there is levosimendan is another ino-myo vasorelaxant (ino-cardioprotector, vasodilator) but unlike dobutamine acts as a calcium desensitizer by increasing the sensitivity of contractile proteins to existing Ca^{++} ions, since Ca^{++} enters the cell through the mitochondrial Ca^{++}-uniporter channel, and thereby increases the cardiac contraction force-inotropes,without increasing myocardial consumption in O_2. As well for these purposes, cardio-inotropic effect is used, with vasodilation of pulmonary and peripheral arterial vessels, without ↑ Heart Rhythm (HR) but with ↓ post- and preload, phosphodiesterase IIIa inhibitors

(ino-myo vasorelaxant) which are superior to combining the use of dobutamine with selective β-blockers (carvedilol). In the presence of a normal HR hyper- and normovolemia (↑ ECBV, ↑ BP) with an increased pre and post load, selectively justified themselves nitrates and α-β-adreno blockers (Labetalol) which by establishing peripheral vasodilation with in arterioles and venules anti-hypertensive effects have a beneficial effect on the cardiac output fraction and HR. In this direction proved that the combined use of α-vazopressor on the background of the H-cholinergic ganglion blockade, ganglion blockade without hypotension is established at which a different pharmacological effect occurs a new not present in their isolated application since post-capillary venules increase their susceptibility to the vasopressor and thereby support the macrocirculation while decentralizing it, which optimizes vascular SPP ΔVP in which the microcirculation improves mainly increased susceptibility to adrenomimetic-pre, postpsynaptic etc. metabolic capillary sphincters close the shunting through capillary anastomoses.

Optimization of SPP by RMM at MMDs simultaneously reducing pCO_2 (AV gap) also reduces anion gap & urinary anion gap.

When optimizing SPP besides autonomous brain pressure and other loco-regional for example pressure and in the pulmonary perfusion system are taken into account since an increased pressure in the left atrium causes pulmonary hypertension due to a spasm of the pulmonary arteries resulting in a decrease in Minute Blood Volume (MBV) Kitaev's reflex as well as in response to an obstruction in non-ventilated lung areas pressure in the pulmonary arteries also increases at which reflex pulmonary hypertension occurs as described by van Euler-Lildzhestaander. These cases are solved by RMM when optimizing Vascular Compliance ΔVP SPP and reducing pulmonary hypertension, at the level of CRP by maintaining autonomous perfusion pressure (pulmonary artery wedge pressure ~12 mmHg) which is important for the ratio of ventilation and perfusion-VA/Q. The corresponding modification of vascular compliance ΔVP SPP extends and to autonomous abdominal pressure during Abdominal compartment syndrome with simultaneous surgical correction (Figure 1).

With MODs, with an increase in ↑ pCO_2, caused by pulmonary/extrapulmonary Acute Respiratory Distress Syndrome, ARDs [17] and confirmed by the fall in the oxygenation index ↓ PaO_2/FiO_2 ↓ 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 $FetCO_2$ ↓, SaO_2 ↓, PaO_2 ↓, FiO_2 ↓, b) Parenchymal (endothelial-epithelial damage to alveolar and vascular tissue) $FetCO_2$ ↓ / or normal, SaO_2 ↓, PaO_2 ↓. 2) Transportation of gas in the blood (minute volume) ↓, Hb ↓, SvO_2 ↓, PvO_2 ↓, $avSO_2$, $avPO_2$. 3) Gas exchange in tissues SvO_2 ↑, BE ↑, PvO_2 ↑, $avSO_2$ ↓, $avPO_2$ ↓ lactate/pyruvate ↑.

At the same time the pressure/volume loop of the trachea is also considered which are presented in 4 types (cucumber, pod, pear, tomato) which means that the more the loop surface is expanded the more the respiratory pattern as well as the definition of the dynamic (Cdyn) and statistical (Cst) compliance confirming damage to the respiratory organs aggravating MK MMDs and RMM in such cases are supplemented with MOST therapy in the EXTRACORPOREAL Life Support Organization (ELSO) with active detoxification methods: 1) Alveolar recruitment with respiratory support in special modes of ventilation mainly APRV with permissive hypercapnia at normal pH, 2) Recruitment of Microcirculatory-Mitochondrial RMM, 3) MOST Extracorporeal Life Support Organization (ELSO) extracorporeal oxygenation ECMO and CO_2 elimination by type $ECCO_2R$ [18], 4) Active detoxification methods intra and extracorporeal electrochemical ultraviolet (laser) photomodulation of auto blood ultra diafiltration continuous intermittent filtering, hemodialysis, bioimmunoactivation and biodetoxification through the use of extracorporeal bio xenoperfusion (myelo-timo-spleen) enterosorption, volnerosorption, plasma sorption, plasma exchange, lympho sorption, liquoro sorption, peritoneal dialysis, oxygenation of the liver through the bougienized umbilical vein, hypothermia and others [2,9,19-22], 5) Modeling of the index of extravascular pulmonary fluid EVLWI. If EVLWI is

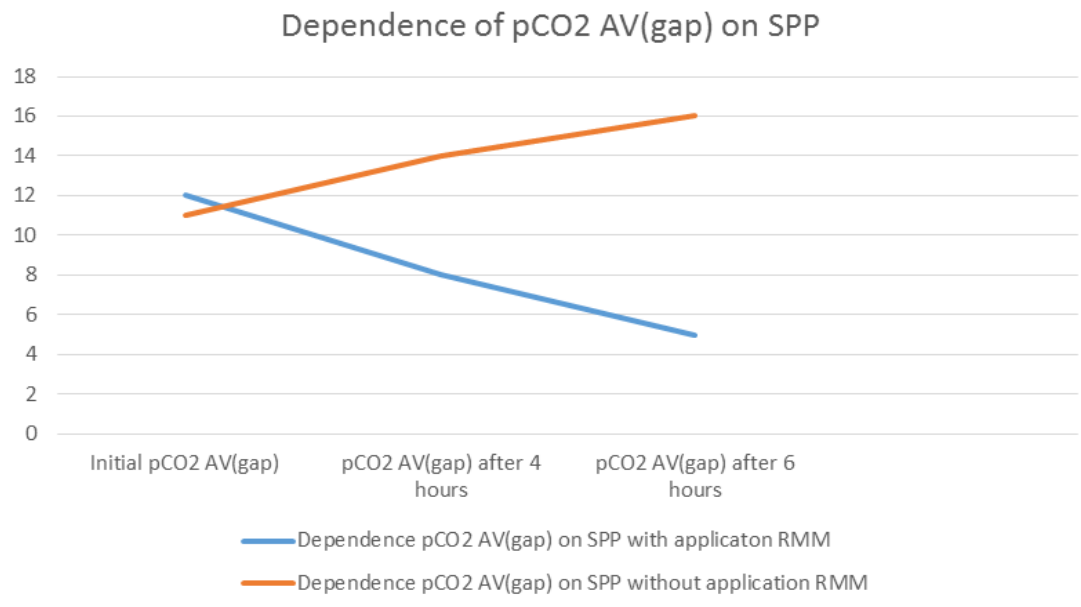

Figure 1: Dependence of pCO_2 AV (gap) on SPP with and without RMM application.

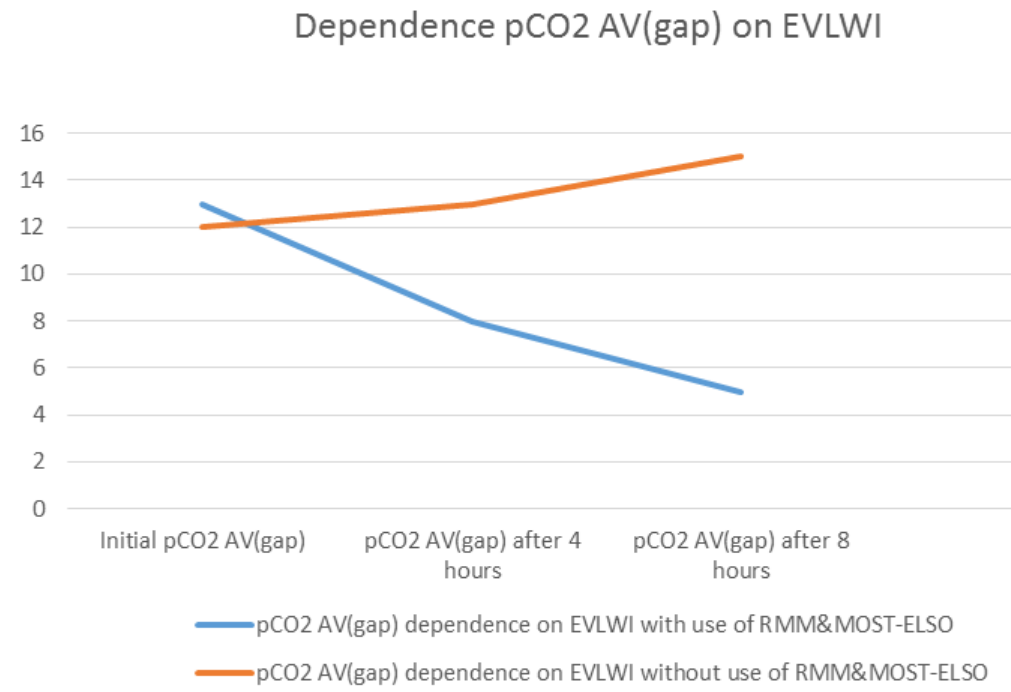

Figure 2: Dependence pCO_2 AV (gap) on EVLWI with or without use of RMM & MOST-ELSO.

<10 mL/kg this indicates alveolar atelectasis which requires volemic resuscitation, bronchoscopy, alveolar recruitment and surfactant therapy. In situations where EVLWI is>10 mL/kg, which is a threat to pulmonary edema which requires a reduction in volemic resustation and the inclusion of diuretics ultrafiltration and MOST-ELSO, inotropic therapy and invasive monitoring, 6) Th_4-Th_5 thoracic epidural block. The level of catheterization of the epidural space should be Th_4-Th_5 (thoracic epidural block) in hyper-eu-kinetic patients especially with hypertension and hypervolemia (EVLWI>10 mL/kg), but without hypocoagulation coagulopathy. Epidural analgesia at the level of the chest is favorable because it expands spastic coronary arterioles (cardio-coronary dilatation) increases the delivery of O_2 to the myocardium reduces myocardial oxygen consumption, reduces the risk of myocardial infarction and ischemia, improves lung function and contributes to the functioning of lung gas exchange; reduces pulmonary hypertension, accelerates intestinal motility, promotes bowel movement and conforms to the multimodal analgesia protocol. Permanent infusion of local anesthetics (0.2% Ropivacaine/0.125% Marcaine) in combination with opioid analgesics (fentanyl 2-4 μg/mL). After bolus 4 mL follows be a constant infusion of 5.0-7.0 mL/h according to clinical indications. The patient-controlled Fepidural anesthesia is 0.2% Ropivacaine/0.125% Marcaine in combination with opioid analgesics (Fentanyl 2-4 μg/mL). For one bolus of 4 mL of the mixture is followed by a constant infusion of 3.5-6.0 mL/h with a lockout interval of 20-30 minutes. The dose should be 1.0 to 2.0 mL, so the patient can enter it himself. In the case persistent of pain, a multimodal anesthesia protocol follows. Paracetamol significantly increased the effect of analgesia, which could be compared with opioid analgesia or nonsteroidal anti-inflammatory drugs (Figure 2).

Thus decentralization, anti-shock therapy, detoxification and analgesia in the RMM control strategy, supplemented by MOST-ELSO (ECMO and CO_2 elimination $ECCO_2R$, etc.) and in combination with antibacterial/antiviral treatment and surgical correction counteracts the MMDs mitochondrial collapse and regression MODs.

Conclusion

1. In tissue hypoxia, an increase in the valid pCO_2 marker of AV gap>6 mmHg with the exclusion of acute respiratory distress syndrome confirms microcirculatory-mitochondrial distress syndrome as a mitochondrial collapse explaining the slowing/stopping of venous CO_2 return from the periphery to the center due to the disturbance and perfusion of the SPP, which is responsible for equilibrium macro circulation-microcirculation.

2. Recruitment of microcirculatory-mitochondrial restoring systemic perfusion pressure thereby decentralizes the macro circulation and improves the microcirculatory space at the level of the capillary ↔ cell ↔ mitochondria in the metabolic space and suspends the functioning of the direct anastomosis AV through the establishment of microcirculation through the metabolic capillary thus remodeling the cell energy metabolism rate.

3. Recruitment of microcirculatory-mitochondrial drains into the macro circulation accumulated in the microcirculatory space catabolites and endogenous toxic substances, with impaired venous return creating translocation macro circulatory hipertoxemia which argues the effectiveness of treatment of the microcirculatory mitochondrial distress syndrome.

4. The absence of a continuous decrease in the pCO_2 marker of tissue hypoxia AV gap <5 mmHg proves the suspension of the continuation of cell necrosis/apoptosis, hypo (a) energy and confirms mitochondrial eu-energetic metabolic remodeling with elimination of mitochondrial hypo (an) energy, active lysosomal clearance (mitophagy) thus supporting the presence of eu-energetic mitochondria with the normalization of mitochondrial Ca^{++}-channel uniporter and cyclosporine-sensitive

mitochondrial pore (mitochondrial permeability pore transition) with beneficial productively inactivate toxic forms of oxygen and nitrogen.

5. Systemic perfusion pressure can be modelled by modifying a heart pump, effective circulating blood volume, and a capillary resistance pressure.

6. Reduction of the pCO_2 A-V gap and suspending the development of syndrome of multi-organ dysfunction is achieved by applying the recruitment of microcirculatory-mitochondrial in the complex associated Multi-organ Supportive Therapy Extracorporeal Life Support Organization (ECMO and CO_2 elimination $ECCO_2R$ etc.)

7. Optimization of systemic perfusion pressure by recruitment of microcirculatory-mitochondrial at microcirculatory mitochondrial distress syndrome, simultaneously reducing pCO_2 (AV gap) also reduces anion gap & urinary anion gap.

8. The effectiveness of recruitment of microcirculatory mitochondrial, as a strategic management, is also approved by a clinical examination of the patient warming and restoring skin tone and turgor regression of white spot syndrome with a slight pressure on the nail stabilization of homeostasis.

9. Despite the fact that we have not observed obstetric mortality due to the use of applying the recruitment of microcirculatory mitochondrial associated Multi Organ Supportive Therapy Extracorporeal life support organization for more than 35 years with sufficiently multiple critical obstetric cases we recognize that the ideal therapy for achieving regression of the microcirculatory mitochondrial distress and of the syndrome of multi-organ dysfunction with resuscitation of "no-fluid resuscitation" or "low-volume resuscitation" we are still very far away.

Author's Contribution

The distinguished G.Litarczek Patriarch of the scientific Anesthesia - Therapy Care of Romania (born in Boston, USA), he died on 14 March 2019. Academician name G.Litarczek.

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