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The recruitment of microcirculatory-mitochondrial of critical obstetric situations in the complex multi-organ support therapy reduces pCO_2 (AV gap) and the development of the syndrome of acute multi-organ dysfunction

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Abstract

A retrospective analysis of the 35-year absence of maternal mortality in critical obstetrics, in different countries, was due to the timely decentralization of macro-circulation, detoxification and analgesia. Macro-circulation was decentralized once the systemic perfusion pressure has been established; which is the difference between the mean blood pressure and the pressure of the capillary resistance, and what contribute to by decreasing the tissue hypoxia marker pCO_2 (pCO2 AV gap >6 mm Hg) due to micro-circulatory-mitochondrial recruitment, through improved microcirculation at the level of the capillary-cell metabolic area: metabolic capillary ↔ cells ↔ mitochondria; with ameliorate of the venous return compliance and reduction (pCO2 AV gap <6 mm Hg), and respectively, diminishes of the microcirculatory-mitochondrial distress syndrome (MMDs), and stopping expansion syndrome of acute multi-organ dysfunction. In cases of development of respiratory-pulmonary pCO_2 ↑ (ARDs), confirmed ↓ PaO_2/FiO_2 ↓300 to Acute Respiratory Distress Syndrome (Berlin definition, 2012), thus also aggravates the MMDs (pCO_2 AV gap >6 mmHg), mitochondrial collapse and the recruitment of the microcirculatory-mitochondrial is supplemented with multi-organ support therapy (MOST), including detoxification: alveolar recruitment through respiratory support in specific ventilation modes, predominantly APRV, with permissive hypercapnia at a normal pH; MOST-extracorporeal with technical support. Extracorporeal life support organization-ELSO; modelling of extra-vascular pulmonary fluid index EVLWI; Th4-Th5 thoracic epidural block; active detoxification methods. The absence of decreasing of the pCO_2 tissue hypoxia marker at the pCO2 AV gap ↓ 5.0 mm Hg after microcirculatory- mitochondrial recruitment, rejects the necrosis/apoptosis, hypo- (an) ergic cell and proves the mitochondrial eu-energetic metabolic remodelling with the elimination of the hypo-(an) ergic mitochondria performed by liposomal clearance (mitophagy), thus demonstrating eu-ergic mitochondria with the normalization of mitochondrial uniporter-Ca++ and mitochondrial permeability pore transition, which productively inactivate the toxic forms of oxygen and nitrogen.

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