**Pneumolysis and “Silent Hypoxemia”**

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Patients suffering from COVID-19 can present severe lung compromise that can evolve to life threatening hypoxia. However, they do not become aware that they are suffering hypoxia referred to as “silent hypoxemia” or “happy hypoxemia”. Then suddenly they present dyspnea (shortness of breath) and tachycardia. They hyperventilate and start to feel gradual suffocation and can drop dead even in the streets as evidenced around the world. The mechanisms involved are not fully understood. Initially, past-experience lead to the implementation of standardized protocols assuming this disease would be the same as SARS-CoV, the previous Coronavirus. Impulsive use of ventilators in extreme cases ended up in over 88% fatality.

We compare medical and physiological high altitude acute and chronic hypoxia experience, obtained during 50 years of existence of the High Altitude Pulmonary and Pathology Institute, with COVID-19 hypoxemia. Application of the Tolerance to Hypoxia formula = Hb/PaCO2 +3.01 to COVID-19, enlightens the critical hypoxemia. *Pneumolysis* is defined as progressive alveolar-capillary destruction resulting from CoV-2 attack to pneumocytes. The adequate interpretation of the histopathological lung biopsy photomicrographs reveals these alterations. The three theoretical clinical stages of progressive hypoxemia (silent hypoxemia, gasping and death zone) are described. At sea level, in COVID-19, the *silent hypoxemia* SpO2 <= 90% (comparable to a normal SPO2 at3,500m) is suddenly replaced by critical hypoxemia due to progressive *pneumolysis* + inflammation + over expressed immunity + HAPE-type edema resulting in pulmonary shunting. Treatment based on improvement of the Tolerance to Hypoxia (Hb factor), inflammation reduction & antibiotics is proposed.