Case Discussion In Medicine

Happy hypoxia

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Key words : Silent Hypoxia, COVID-19, Lung compliance, Pulse Oximeter.

Case 1 : 28 yr male came to Fever Clinic with h/o fever for 7 days. He also have h/o cough for same duration. Fever was remittent and moderate in grade without chill and rigor. Cough was mostly dry with little expectoration. No h/o haemoptysis . Patient was not dyspneic. While patient was in OPD waiting area suddenly became dyspneic . Physician rushed to patient . Patient was in gasping condition. BP-100/60 mmHg. P/R – 130/min, R/R – 46/min . Chest – diffuse crepititon and rhonchi .Saturation (SPO2) 56% . He was admitted at CCU.Investigations : ABG – type 1 respiratory failure. Xray Chest PA view – B/L infiltrates. HRCT thorax – ground Glass Opacities. COVID 19 nasopharyngeal swab RT-PCR was positive.

Case 2 : Patient 46 yr female came to OPD with fever for 5 days with cough and sputum for same duration. Patient was examined . BP-136/86 mm Hg. P/R – 100/min , R/R – 18/min. saturation not seen by resident . Chest bilateral VBS , no crepitation, no rhonchi. Patient was discharged with oral medicines and advised to come to OPD after 2 days with relevant investigations . After 2 hr patient came back with acute breathlessness . Patient was in gasping situation. R/R- 60/min, BP 110/60 mmhg , saturation (SPO2) 60% . Patient was intubated and admitted in CCU. Again in this patient COVID-19 nasopharyngeal swab RT-PCR was positive .

Issues :

(1) Why apparently clinically stable patient become gasping ?

(2) Is it unique in COVID-19 patients ?

(3) Is it preventable ?

Entity is named as "Silent Hypoxia " or "happy Hypoxia" . It is severe hypoxemia without dyspnea and poorly responsive to supplemental O2. It is not new entity, seen in different physiological and pathological situations, but in COVID era we are relooking to this entity as lack of awareness of this entity leading to confusion in decision making in COVID patients attending in OPD and emergency and leading to more catastrophic result.

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Editor's Comment :

- **Silent Hypoxia common in COVID 19 patients**
- **Can falsely give sense of wellbeing in patient and in physician**
- **Overlooking this entity can result in late prersentation to health care facilities**
- **Routine use of Pulse-oximeter can detect silent hypoxia in early stage of disease.**

Patients with COVID-19 may present to hospitals and emergency with an atypical form of ARDS (acute respiratory distress syndrome)[1].

The COVID-19 pneumonia and these disease spectrum is a specific disease with some peculiar phenotypes. The main characteristic features is the dissociation between hypoxemia severity and the maintenance of good respiratory mechanics. The median respiratory system compliance is around 50 ml/cmH_2O[2].

Hypoxia Physiology :

Hypoxia is a condition in which the body or a region of the body is deprived of adequate oxygen at tissue level. Normal arterial oxygen is approximately 75-100 mm Hg and normal pulse oximeter reading ranges from 94-99%

Hypoxemia

Generally occur in two ways

(1) Ventilation – perfusion mismatch

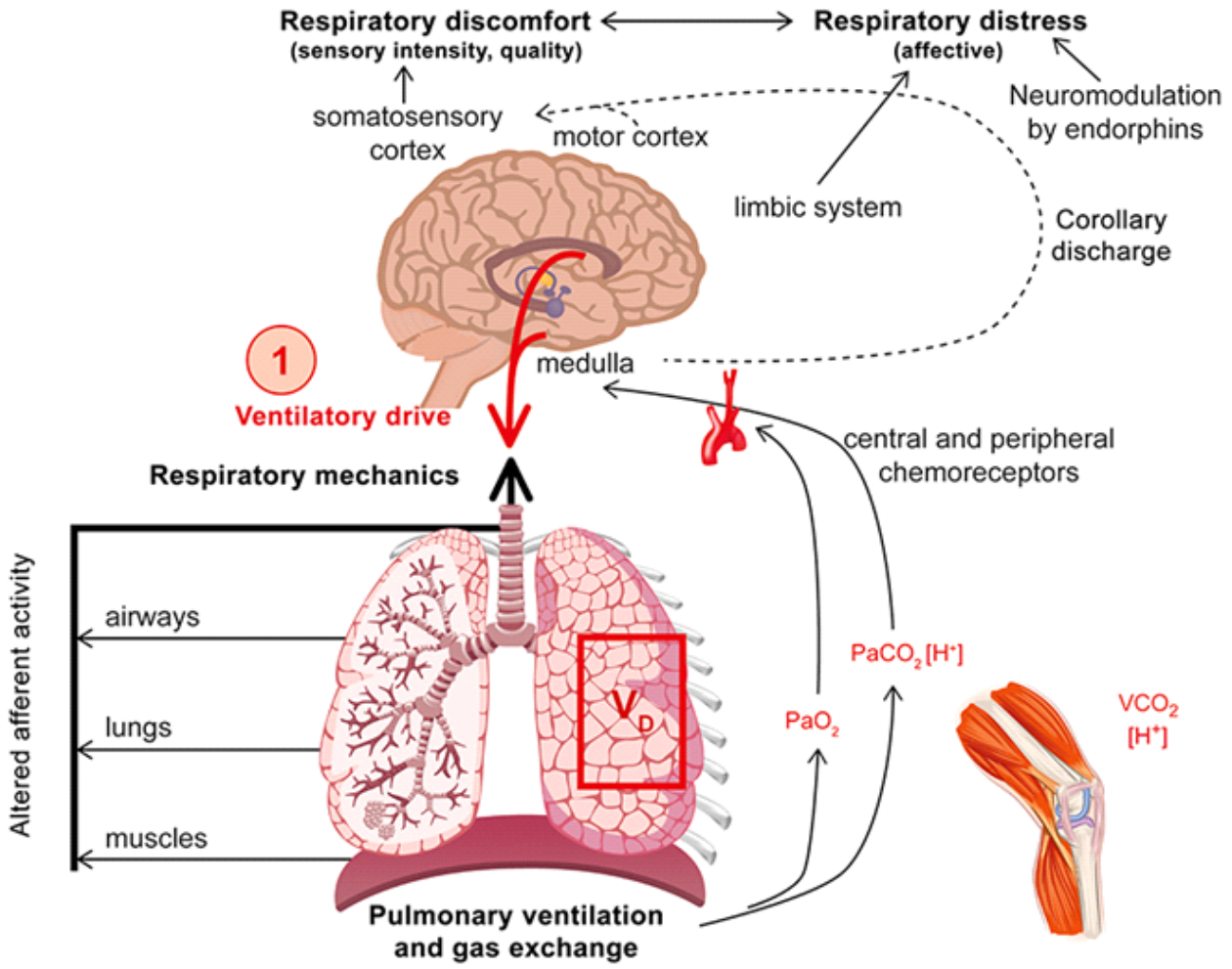
(2) Right to left shunt – either intracardiac or intrapulmonary

CO2 Clearance

It depends on how much gas enter and leave lung and remove CO2 in process.

$$CO2\ level_{(in\ blood)} = \frac{CO2\ production\ in\ body}{Respiratory\ rate \times (Tidal\ volume - dead\ Space)}$$

So body can remove CO2 either by increasing tidal volume i.e depth or by increasing rate.



Respiratory Chemotactic Centre :

Body have two Chemosensor . Central (primary) chemoreceptor located in Brainstem and stimulated by body's level of CO2. Peripheral Chemoreceptor (secondary) located in carotid bulb of Internal carotid artery and stimulated by level of O2. Peripheral chemoreceptors are sensitive to changes in mostly O2, but less to CO2 and pH. Central chemoreceptor's are sensitive to changes in pCO2 and pH.[3] Body primarily response to level of CO2 in blood. If there is Hypercapnia respiratory depth or rate will be increased and CO2 will be eliminated. If there is hypocapnia; respiratory chemosensors will not be activated.

Work of Breathing :

Dyspnea related to work of breathing. Patient becomes dyspneic if work of breathing increases. Work of breathing strongly related to drive to clear CO2 from body. Airhunger caused by primarily hypercapnia /acidosis, whereas hypocapnia/alkalosis decrease ventilator drive. Only when Po2 falls below 60mm Hg then hypoxia becomes stimulus for ventilator drive.

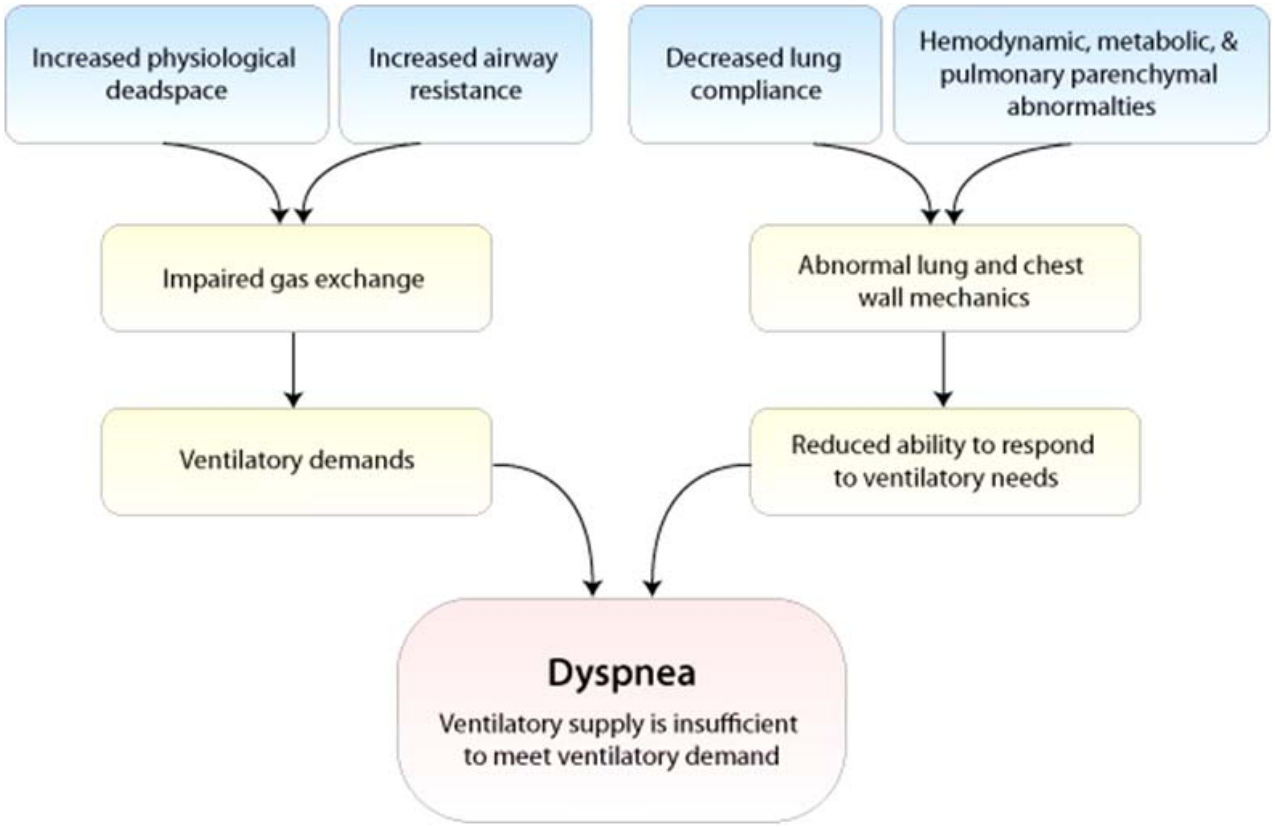
Work of breathing related to a) Tidal Volume – Dead Space b) Lung Compliance c) airway resistance.

If tidal volume decreases body will compensate hypoxia and will eliminate CO2 by increasing rate of respiration, thus work of breathing will increase. If dead Space increases Tidal Volume – Dead space will be low , and CO2 will accumulate , thus body will compensate by increasing Tidal volume that is increasing depth of respiration (as depth of respiration not related to work of breathing so patient will not be dyspneic). If depth of respiration not adequate then rate of respiration will increase and person will be dyspneic.

Silent Hypoxia :

It is a remarkable discrepancy in a patient with gross hypoxaemia yet without proportionate sign of respiratory distress due to well preserved lung compliance but compromised gas exchange.

Refractory hypoxemia with a normal work of breathing can occur :- if there is a shunt (right to left) and if there is



not excessive dead space or if lung compliance and resistance are normal. Lungs are reasonably normal.

It is a form of hypoxia, difficult to detect as patient appear less in distress. Unless Po2 become significantly low person not become symptomatic. This is a common entity in aviation medicine. In COVID era we are giving a relook on it.

Generally, lower lobe consolidation will cause shunt of deoxygenated blood through the collapsed lung, thereby causing hypoxia with a normal work of breathing; causing happy hypoxia.

Experiment in hypobaric chamber revealed that hypocapnic hypoxia is not usually accompanied by airhunger, paradoxically have feeling of well being. Physiology of hypocapnic hypoxia has been extensively studied in aviation medicine.[4]

While climbing in high altitude in a short time (environment of Low pressure oxygen) Po2 frequently low in blood .But body compensate initially by increasing depth of respiration, thus eliminating CO2. As depth is increased not rate person does not become dyspnic. Air hunger does not occur much but suddenly person suffers from blackout when Po2 becomes dangerously low. This is a example of Silent Hypoxia.

What happens in COVID-19 patient —

(A) Corona virus attach more to Pneumocytes of lung alveoli as it has more ACE2 receptor. Pneumocyte1 responsible for forming lining of alveoli and Pneumocyte2 for production of surfactant, responsible for compliance of lung . In mild to moderate stage of Corona infection alveoli are inflamed and filled with exudates,so there is impairment of gas exchange.[5] At this stage Lung compliance is normal. Diffusion capacity of CO2 is 20 times more than O2. But CO2 diffuses out but O2 can not mix properly with blood, so hypoxia occurs without hypercapnia. As CO2 is responsible for work of breathing ,in spite of hypoxia , breathlessness does not occur . This is called Silent Hypoxia and unawareness of this entity will create confusion among physicians regarding admission of COVID patients. In late stage due to fall of surfactant lung compliance decreases, so work of breathing increases and so patient become dyspneic.

(B) In other scenario, mainly in lobar pneumonia due to covid, the consolidated lung tissue may causes hypoxia and as the remaining lung tissue was normal, allowing the patients to clear CO2;

(C) Another cause may be dysfunctional hypoxemic vasoconstrictionwhich explanation for these severe hypoxemia in compliant lungs due to a loss of lung

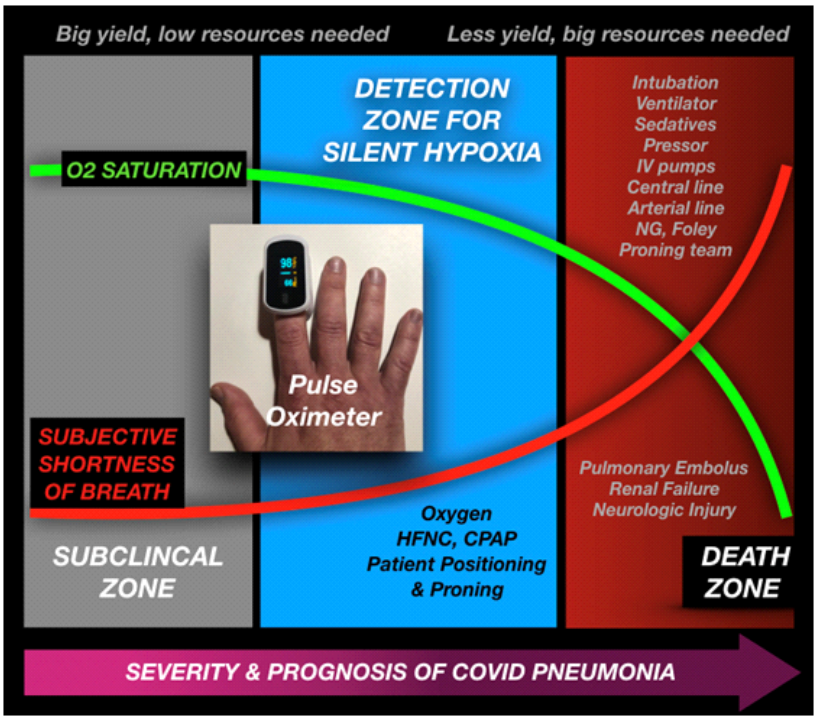
perfusion regulation and also hypoxic vasoconstriction.[6]

(D) Intrapulmonary shunting.[7]

Conclusion :

(1) Understanding of this entity and application of Pulse oximeter in Suspected or confirmed Covid patient in OPD or emergency will helphysicians in early diagnosis of hypoxic patients and brought in appropriate management protocol. Identification of silent hypoxia in Fever patients during pandemic period give a clue to investigate for COVId-19 and admission in health care facilities rather than discharging with oral medications . This knowledge can limit mortality.

(2) In patient with hypocapnic hypoxia an increase in PaCO2 will lead to right shift of oxyhemoglobin dissociation curve resulting in abrupt fall of saturation and resulting circulatory collapse.



(3) In community mild Fever and cough often ignored by individuals.During door to door surveillance in red zone or containment area use of Pulse oximeter by community health workers can identify hypoxia in apparently clinically healthy persons with fever and cough and identification of hypoxia can be clue to COVID-19 infection and can be brought to health care facilities and early investigation and treatment can be initiated before becoming gasping .

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