alveolar Po_2 ($PA O_2$) is calculated by following formula called alveolar gas equation: $\mathbf{PA O_2 = FI O_2 \times (P_B - PH_2O) - Pa CO_2/R}$ where FI O_2 is fractional concentration of inspired O_2 (= 0.21), P_B is Barometric pressure (about 760 mm Hg at sea level), PH_2O is water vapour (47 mm Hg when fully saturated at 37° C) and R is respiratory quotient (ratio of CO_2 production to O_2 consumption i.e. N = 0.8). After substituting values with patient breathing at sea level, equation becomes $\mathbf{PA O_2 = 150 - 1.25 \times Pa CO_2}$. The alveolar – arterial O_2 difference is then calculated by subtracting measured PaO_2 from calculated $PA O_2$. In healthy young person breathing room air the (A-a) gradient normally is less than 15 mm Hg (this value increases with age and goes up to 30 mm Hg in elderly).

PREVENTION AND MONITORING OF OXYGEN TOXICITY

Because the treatment is purely symptomatic, prevention and monitoring for early recognition of toxicity is of prime importance. The point of importance is that sudden stoppage of O_2 at the onset of toxicity, may sometimes aggravate the symptoms – "the oxygen off effect"[27]. Monitoring of pulmonary toxicity is based on reduction (usually 10%) in the vital capacity of the patient[27,35]. Other indicators of monitoring include reduced lung compliance and diffusing capacity for carbon monoxide. To predict pulmonary damage after prolonged O_2 therapy, unit of pulmonary toxicity dosage (UPTD) is calculated. One minute of 100% oxygen at 1 atmosphere is taken to produce 1 UPTD. A UPTD of 1425 will produce a 100% reduction in vital capacity[25]. Electroencephalogram has no value in the monitoring of CNS toxicity[27]. Exogenous antioxidants especially vitamin C and E may be used prophylactically in high risk infants. The recommended dose of vitamin E is 100 mg/kg/day for 4-6 weeks[37]. Adrenalectomy, hypophysectomy and the hypothyroid state are associated with reduced severity of toxicity as is the use of alpha adrenergic blockers[27]. Supplementation of dietary trace elements may be helpful in deficient states.

CONCLUSION

The beneficial effects of oxygen therapy have been extensively investigated in patients with COPD with hypoxaemia[39,40]. The ability to provide supplemental oxygen is a powerful tool in the management of critically ill patients with many disorders. Its injudicious use may lead to toxicity of CNS, lungs, eyes and other tissues. But hypoxia must not be left untreated in view of toxicity since hypoxia is common and damage caused is severe and rapid in comparison to oxygen toxicity which is uncommon. The patient education is also an important aspect. The patient should clearly understand the oxygen prescription, the safety precautions to follow when using oxygen and expected benefit of oxygen therapy.

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