The administration of supplemental oxygen is an essential element of appropriate management for a wide range of clinical conditions; crossing different medical and surgical specialities. The present review summarizes the role of supportive oxygen therapy in various clinical conditions encountered in our day-to-day practice in the speciality of oral and maxillofacial surgery; including major trauma, shock, sepsis; perioperative and postoperative considerations and in patients with various other medical comorbidities. Regular and judicious use of oxygen as a drug is thus recommended in our day-to-day practice in oral and maxillofacial surgery to reduce the morbidity and improve the prognosis of patients.

Key words: Hypoxia, oral surgery, oxygen therapy

Oxygen therapy: definition

Oxygen therapy is the administration of oxygen at concentrations greater than that in ambient air (20.9%) with the intent of treating or preventing the symptoms

Historical review: Evolution of oxygen as a drug

Oxygen is an atmospheric gas essential for survival of all living things; denoted by letter O. The presence of “air” is vital for survival of human was recognized in the ancient Greek as well as in Vedic Hindu literature more than 2000 years ago.[1,2] It was only in the 18th century that gas was isolated by Joseph Priestley and its importance in respiratory physiology by Antoine Lavoiser.[3] The problems of oxygen deficiency as well as need and indications for oxygen therapy were subsequently recognized. Soon oxygen came to be known as “cure all” medicine; used for conditions varying from cholera, arthritis, anemia, and syphilis to glaucoma, epilepsy, diabetes, and cancers. It was around the second decade of 20th century and later that oxygen therapy was adopted for indications based on firm scientific foundations.[4]
and manifestations of hypoxia, which includes agitation, personality change, headache, nausea, increase in pulse, increase frequency or sob, cyanosis [Table 1].

**Clinical Goals of Oxygen Therapy**

- Treat hypoxemia
- Decrease work of breathing
- Decrease myocardial work

**Indications**

Oxygen therapy is indicated in the wide spectrum of clinical conditions for its definitive, supplementary or palliative role. In this review, the supplementary role of oxygen is highlighted; the rationale for which is prevention of cellular hypoxia cause by hypoxemia (low PaO₂) and thus prevention of potentially irreversible damage to vital organs.

Therefore, the most common reasons for oxygen therapy to be initiated are

- Acute hypoxemia- shock, asthma, pneumonia, heart failure.
- Ischemia – myocardial infarction but only if associated with hypoxemia
- Abnormality in quality or type of haemoglobin – acute blood loss in trauma

**Contraindications**

There is no absolute contraindications; but should be administered with caution in patients suffering from paraquate poisoning (BNF 2005) and with acid inhalation or previous bleomycin lung injury.

**How to assess the need for oxygen therapy**

*Patients' need for oxygen therapy based on specific clinical condition*

Examples include post-operative patients, shock, trauma, acute myocardial infarction or cyanide poisoning.

*Lab measures to document hypoxia*

It includes both invasive and non-invasive methods such as hemoglobin saturation, arterial blood gas analysis, pulse oximetry.

**Pulse Oximetry**

This is a good bedside monitor if its limitations are recognized. It is a continuous and non-invasive monitor.

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**Table 1: Manifestations associated with hypoxia**

| Finding       | Mild to moderate | Severe                      |
|---------------|------------------|-----------------------------|
| Respiratory   |                  |                             |
| Tachypnea     |                  | Tachypnea                   |
| Dyspnea       |                  | Dyspnea                     |
| Paleness      |                  | Cyanosis                    |
| Cardiovascular|                  |                             |
| Tachycardia   |                  | Tachycardia; eventual       |
| Mild hypertension |             | bradycardia, arrhythmia    |
| Peripheral    |                  | Hypertension and eventual   |
| Vasodeconstriction |             | hypotension                |
| Neurologic    |                  |                             |
| Restlessness  |                  | Somnolence, confusion       |
| Disorientation|                  | Distressed appearance, blurred |
| Headaches, lassitude |     | vision, tunnel vision, loss of |
|               |                  | coordination               |
|               |                  | Impaired judgement, slow   |
|               |                  | reaction time, manic depressive activity, coma |

Its principal limitation is that in patients who are receiving supplemental oxygen, it will not reliably detect hypoventilation. Hypoventilation must, in clinical environment, usually be confirmed by measurement of the PaCO₂ by arterial blood gas analysis.

**Arterial Blood Gases**

This is the ‘gold standard’ monitor of ventilation. Arterial blood gases are needed to obtain accurate data, in particular, evidence of hypoventilation (raised PaCO₂) as a reason for hypoxaemia. Arterial blood gases may also give an indication of the metabolic effects of clinically important hypoxaemia. However, a blood gas is a painful, invasive, and intermittent procedure that is time consuming in the setting of a busy ward.

**What is normal oxygen saturation levels?**

In individuals less than 70 years of age, normal oxygen saturation at rest is 96–98% when awake,[6] while in those aged 70 and above, greater than 94% when awake is considered normal. However, patients of all ages may have transient dips of saturation to 84% during sleep.[6]

**Oxygen Delivering Systems**

Broadly divided into two:-

1. **High flow system**

The gas flow of a device that is adequate to meet all inspiratory requirements. By providing the complete insp. volume, the high flow system delivers it’s FiO₂ very accurately. High flow systems can delivery both high and low concentrations of O₂. It includes: venturi mask, venturi type nebulizers (Fail > .50 FiO₂), high flow blender system. Gas injection nebulizer (GIN) -works for all FiO₂s.

2. **Low flow system**

It is the one through which O₂ is delivered to supplement
the patient’s tidal volume. It includes: cannula, simple mask, reservoir or non-rebreather, (highest FiO₂).

**Oxygen Delivery Equipments**

**Nasal cannulae**
They are suitable for most patients with both type I and type II respiratory failure. At a flow rate of up to 2–6 l/min gives approx 24–50% FiO₂. Its advantages include comfortable and easily tolerated by patients vs simple mask, low cost product and it is non-rebreathing.

**Simple face mask**
It is a variable performance device, delivering variable oxygen concentration between 35–60% at flow rate of 5–10 l/min. It is commonly used for patients with type I failure.

**Venture/ Fixed performance mask**
It aims to deliver constant oxygen concentration within and between breaths. They are available with color coded nozzles for desired concentration of 24%– blue; 28%– white, 35%– yellow, 40%– red; 60%– green. 24-40% venture masks operate accurately. 60% venture gives approximately 50% FiO₂.

**High concentration reservoir mask**
It is a non-rebreathing reservoir mask, delivers oxygen concentration between 60–80% or above. It is effective for short term treatment in critical illness, trauma patients, post cardiac, or respiratory arrest.

**Tracheostomy mask for patients with tracheostomy or laryngectomy**
These are variable performance masks designed for neck breathing patients, fits comfortably over tracheostomy or tracheotomy. It has an exhalation port on front of neck.

**Pocket mask**
It is used to provide mouth-to-mouth ventilation when performing artificial ventilation in emergency situations. Its advantages include elimination of direct contact with patient’s nose, mouth and secretions; use of one way valve at ventilation port prevents exposure to patient’s exhaled air and supplemental oxygen can be administered through the oxygen inlet. Always make sure to make a good seal around the mask.

**Considerations in Various Clinical Conditions**

**Emergency oxygen in trauma patient**
It is crucial to provide optimal oxygen therapy while the acutely breathless patient is being transferred to hospital, assessed in the emergency department, and treated for their disease. The main concern is to give sufficient oxygen to support their needs.[7]

Low intravascular volume either due to acute blood loss as in trauma can result in poor oxygen transport and tissue hypoxia. These states are therefore potential indications for oxygen administration along with definitive management of underlying cause.

**Guidelines for emergency therapy prepared by North West Oxygen Group**[7] are divided into three groups:
1. Pre-hospital stage;
2. Emergency department assessment stage;
3. Pre-admission stage.

**Pre-hospital stage**
In this situation, the diagnosis for hypoxia is often unclear and the risk of hypoxia is much greater than the risk of Hypercapnia for most patients. So, these patients should be given high concentration oxygen to maintain oxygen saturation above 90% until arrival at an emergency department. This can be achieved in most cases by the use of approximately 40%–60% oxygen via a medium concentration mask at flow rate of 4–10 l/min. Use a reservoir (non-rebreathing) mask if the patient is severely hypoxic and in all major trauma cases.

**Emergency department assessment stage**
Although history taking and clinical examination may clarify the diagnosis, oxygen at 40%–60% should be continued until blood gas results are available unless the patient is drowsy or is known to have had previous episodes of Hypercapnic respiratory failure. In these circumstances, a lower FiO₂ may be required such as 2–4 l/min via a medium concentration mask (equivalent to approximately 28%–40% oxygen) or preferably by the use of controlled oxygen at 24% or 28% via a Venturi mask titrated upwards or downwards to maintain an oxygen saturation of 90%–92% pending the results of blood gas estimations.

**Pre-admission stage and early admission stage**
Patients with asthma, left ventricular failure, pneumonia, pneumothorax, trauma, etc, should be treated appropriately for their condition using 40%–60% oxygen via a medium concentration mask (4–10 l/min) for milder cases or a reservoir mask for hypoxic patients and for all major trauma cases.

**Surgical considerations**
Abnormalities of gas exchange in surgical patients are present in not only those with lung disease but also others, both during the surgical procedure and in the post-operative period. Pulmonary complications
especially infection, atelectasis, and thromboembolism are frequent in postoperative patients. This is especially so in patients who are smokers, obese, aged (over 60 years), and/or with a pre-existing lung disease. Hypoxemia with or without CO\textsubscript{2} retention may occur because of the problems of ventilation-perfusion relationship and/or hypoventilation. Oxygen, along with other forms of treatment is required in all such patients with complications.

**Anaesthetic factors**
Gas exchange abnormalities in the post-operative period occur early or late. Early postoperative hypoxemia is due to anesthetic factors such as alveolar hypoventilation (above), ventilation/perfusion mismatching, decreased cardiac output, and increased oxygen consumption due to shivering (induced by volatile agents or recovery from intra-operative hypothermia). Also, ‘diffusion hypoxia’ may transiently contribute to early hypoxemia as a result of the very soluble nitrous oxide diffusing out of the circulation into the alveoli when anesthesia is terminated, reducing the concentration of oxygen in the alveolar gas.

The later onset gas exchange problems are due to alterations in functional residual capacity (FRC) and factors that affect the patient’s ability to inspire deeply or cause the patient to be immobilised in bed.

It is widely appreciated that extreme hypoxemia can produce severe and permanent brain injury and/or cardiac arrest. However, it is perhaps less readily appreciated that mild to moderate hypoxemia can also contribute to postoperative morbidity, and in combination with other factors, to increased likelihood of mortality as a result of wide ranging pathophysiological disturbances. Hypoxemia has been shown to be deleterious in terms of resistance to infection, wound healing and anastomotic integrity. Decreased cognitive function with moderate levels of hypoxemia has also been demonstrated. Delirium, which is troublesome in itself, can lead patients to remove nasogastric tubes, surgical drains and intra-vascular devices. Hypoxemia can be an important factor, and oxygen therapy very beneficial. Supplemental oxygen has been shown to decrease the incidence of postoperative nausea and vomiting.

**How much and for how long?**
As a guideline, young, fit healthy patients having peripheral surgery should receive oxygen for about 30 minutes in recovery to allow resolution of the effects of diffusion hypoxia, and until they are awake and comfortable and protecting their airway. There is no need to administer high dose oxygen, 4 L/minute being adequate.

A patient having major surgery should receive at least 72 hours of oxygen at concentrations of 28–60%.

**Oral and maxillofacial space infections**
Various space infections of oral and maxillofacial region including parapharyngeal spaces, lateral pharyngeal spaces, and Ludwig’s angina can lead to hypoxia mainly by airway obstruction. Therefore, the correction of obstruction is of main concern, rather than the supplementary oxygen.

**Patients with other medical comorbidities**

**Acute exacerbations of chronic obstructive pulmonary disease**
Patients of chronic obstructive pulmonary disease (COPD) often have chronic hypoxaemia with or without CO\textsubscript{2} retention. Oxygen in this situation is required until the exacerbation is settled. While a high FiO\textsubscript{2} of up to 100% can be initially administered in case hypoxemia is severe, it is soon tapered to around 50–60% FiO\textsubscript{2}. The goal of supplemental oxygen is to maintain a PaO\textsubscript{2} of 55–60 mmHg, which corresponds to SpO\textsubscript{2} of about 90%. Higher concentrations of oxygen blunt the hypoxic ventilatory drive, which may precipitate hypoventilation and CO\textsubscript{2} retention. It is better to use a regulated flow device such as a venti mask, which guarantees oxygen delivery to a reasonable extent. Once the patient is stabilized, one can shift to nasal prongs — a device that is more comfortable and acceptable to the patient.

**Acute severe bronchial asthma**
Patients with acute severe asthma or status asthmaticus have severe airway obstruction and inflammation. They are generally hypoxemic. Arterial blood sample is immediately obtained and oxygen is started via nasal cannula or preferably via a face mask at flow rate of 4-6 L/min to achieve FiO\textsubscript{2} of 35 to 40%. Higher flow is unlikely to improve oxygenation. Flow rate is adjusted to maintain a PaO\textsubscript{2} of about 80 mmHg or near normal value. Concurrent bronchial hygiene and administration of intravenous fluids, bronchodilators and corticosteroids should alleviate the problems in most of the situations. Administration of sedatives and tranquilizers must be avoided. Sedatives may precipitate CO\textsubscript{2} retention not only in patients with COPD, but also asthma. Assisted ventilation is required in case there is persistence of hypoxemia and/or precipitation of hypercapnia.

**Complications of Long Term Oxygen Therapy**

**Cytotoxic damage**
Oxygen administration can cause structural damage to the lungs. Both proliferative and fibrotic changes of
oxygen toxicity have been shown at autopsy on COPD patients treated with long term oxygen. But there is no significant effect of these changes on clinical course or survival of these patients. Most of the structural damage attributable to hyperoxia results from high FiO₂ administration in acute conditions.[22]

Depression of ventilation
With prolonged oxygen therapy there is increase in blood oxygen level, which suppresses peripheral chemoreceptors; depresses ventilator drive and increase in PCO₂ high blood oxygen level may also disrupt the ventilation: perfusion balance (V/Q) and cause an increase in dead space to tidal volume ratio and increase in PCO₂. Therefore, oxygen therapy may accentuate hypoventilation in patients with COPD.[23] This may include hypercapnia a carbon dioxide narcosis. Prehospital hyperoxia from excessive oxygen administration in COPD patients is shown to be dangerous.[24]

Absorption atelactasis
An FiO₂ >0.50 presents a significant risk of absorption atelactasis. N₂ is most plentiful gas in both the alveoli and blood. Breathing high level of O₂ depletes body N₂ levels. As blood N₂ level decreases, total pressure of venous gases rapidly decreases. Under these conditions, gases within any body cavity rapidly diffuse into venous blood leading to absorption atelactasis. Risk of absorption atelactasis is greatest in patients breathing at low tidal volumes as a result of sedation, surgical pain or central nervous system (CNS) dysfunction.

Conclusion
This review summarizes the significant role of supplemental oxygen in management of various clinical conditions encountered in our day to day practice of oral and maxillofacial surgeons. With the acknowledgement of oxygen as a drug, it should be prescribed cautiously and in critical cases should be tailored to individual needs. As healthcare professionals, we should not prescribe merely on verbal orders, instead should be prescribed with written orders with proper dosage, flow rate, and duration keeping in mind the complications that can arise on prolonged therapy.

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