Oxygen in clinical Practice: Comprehensive Review of Therapeutic, Technical, and Logistical Aspects

Dr. Bhola Nath Maurya*, Anshal Anany**

Abstract: Oxygen is indispensable across the entire healthcare spectrum, from acute resuscitation and intensive care to chronic management of respiratory diseases. This review article, provides a comprehensive overview covering the sources, production, storage, delivery systems, clinical applications, safety concerns, and regulatory aspects of medical oxygen. By integrating current knowledge and practice, this review aims to equip healthcare professionals and policymakers with a clear understanding of the multifaceted role of oxygen in modern medicine, highlighting recent innovations, pandemic-driven demands, and future trends.

Introduction:

Oxygen is fundamental to human survival, and its therapeutic use is a cornerstone of modern medicine. Since its discovery in the 18th century and subsequent medical use in the 19th century, medical oxygen has evolved from a niche treatment to a ubiquitous component of patient care. It plays a crucial role in resuscitation, anaesthesiology, intensive care, neonatology, and the treatment of chronic lung diseases. Despite its widespread use, knowledge of oxygen production, storage, control, and administration is often limited. The COVID-19 pandemic has focused global attention on the oxygen supply chain, revealing significant vulnerabilities and promoted innovation throughout the world. From industrial gas plants supplying hospitals to portable oxygen concentrators used at home, the medical oxygen infrastructure is vast and complex. This review strives to explore therapeutic oxygen therapy, spanning from elementary knowledge to advanced clinical information. This structured approach will provide readers with a comprehensive understanding of the technical, clinical, and logistical aspects of therapeutic oxygenation.

Key Words- Oxygen, FiO_2, Carbon dioxide, hypoxemia, hypoxia, mask

Definitions[3,5]

1. **Anoxia** refers to the complete absence of oxygen supply to an organ's tissues

* Assistant Professor Dept. of Sangyaharan, FOA, IMS, BHU, Varanasi, 221005, Email- bnmaurya@bhu.ac.in

** Lakhanpur, Bhullanpur, Varanasi, 221108, Mail id anshalanany@gmail.com

despite adequate blood flow to that tissue.

2. **Hypoxemia** is characterized by an abnormally low partial pressure of oxygen in arterial blood (typically below 80 mmHg). It indicates insufficient oxygen content in arterial blood and can lead to tissue hypoxia due to inadequate oxygen delivery to the body.
3. **Hypoxia** describes a state where oxygen availability at the tissue or cellular level is reduced, even though blood flow to the tissue remains sufficient.
4. **Asphyxia** is a condition characterized by insufficient oxygen or an accumulation of carbon dioxide in the body, leading to unconsciousness and frequently death. It typically results from impaired breathing or inadequate oxygen delivery.

Conflict of interest-N/A **Ethical clearance-N/A**

Abbreviation FiO_2 - fraction of inspired oxygen

ICU – incentive care unit

PiO_2 - partial pressure of inspired oxygen

SpO_2 - peripheral capillary oxygen saturation

CO_2- corbon dioxide

RBC- red blood cells

PaO_2 - arterial oxygen partial pressure

IHD - Ischemic heart disease

CHF - congestive heart failure

V/Q - ventilation-perfusion

ABG – arterial blood gas

2,3-DPG- 2,3- Diphosphoglyceric acid

PO_2/PaO_2 in mmHg - partial Pressure of O2

NRB - non-rebreathing Bag (mask)

FRC - functional residual capacity

ASD- aerial sepal defect

COPD - chronic obstructive pulmonary disease

PICU- paediatric intensive care units

CPAP- continues positive airway pressure

RD - retinal detachment

CNS - Central Nervous System

Physical properties

Oxygen is colourless, tasteless, odourless, has a specific gravity of 1.105 and molecular weight of 32. At atmospheric pressure, it liquefies at -183°C, but at the pressure of 50 atmospheres the liquefaction temperature increases to-119°C. Oxygen supports combustion but it itself is not flammable.

Manufacturing process of oxygen[5]

Industrial synthesis of oxygen is through fractional distillation of liquefied air. Prior to the liquefaction process, carbon dioxide is eliminated. Oxygen and nitrogen are then separated based on their distinct boiling points as oxygen boils at - 183°C while nitrogen at -195°C.

Storage of Oxygen

Oxygen is stored in cylinders at a pressure of 137 bar (approximately 2000 psi or 135.2 atmospheres) at a temperature of 15°C. These cylinders are typically identified by their black body with white stripes at the shoulder

(1bar = 0.987 atm. 1 atm =1.01325 bar)

Supply of oxygen in ward & ICU

Many hospitals utilize piped oxygen systems, which are supplied either by a manifold of oxygen cylinders or from bulk liquid oxygen sources to ensure an uninterrupted supply. The pipeline pressure is maintained at approximately 4 bar (60 psi), which is equivalent to the pressure found downstream of the pressure-reducing valves on gas cylinders connected to Anaesthesia machines.

Domestic production of oxygen

Oxygen concentrators generate oxygen from ambient air by removing nitrogen through selective adsorption onto aluminosilicate materials such as zeolites. These devices are particularly useful for bedside oxygen delivery in hospitals, for long-term home therapy, in resource-limited settings, and during military surgical operations. The oxygen produced may contain trace amounts of inert gases, such as argon, which are physiologically harmless.

Utilisation of oxygen by a cell[4]

Oxygen is consumed and simultaneously carbon dioxide is generated within the mitochondria during cellular respiration. The partial pressure of inspired oxygen (PiO_2)

is approximately 21 kPa (160 mmHg), and it depends on the barometric pressure (P^B) and the fractional concentration of oxygen in inspired air (FiO_2), typically 0.21 (or 21% of atmospheric air). As the inspired air passes through the upper respiratory track, it becomes saturated with water vapor, effectively diluting the oxygen content. Upon reaching the alveoli, further dilution occurs due to the influx of carbon dioxide and the extraction of oxygen into the bloodstream.

Oxygen transport[2]

Each 100 ml of blood contains about 20 ml of oxygen. 97% of oxygen is carried by the RBC, while the rest 3% is carried by plasma. Oxygen is poorly soluble in blood [0.023 ml /100 ml blood]. Each molecule of Hb can carry four molecules of oxygen. The final part of the diffusion pathway for oxygen is from the haemoglobin to the mitochondria where the pO_2 is only 0.5-3 kPa (4-23 mmHg).

Oxygen storage in human body[1]

The total oxygen reserve in the human body is approximately 1500 ml, out of which around 750 ml is bound to haemoglobin, 500 ml is present within the lungs, and approximately 250 mL is stored in myoglobin. However, the functionally available oxygen in the event of apnea is less than 1000 ml. Normal oxygen consumption rate of approximately 250 ml per minute(approximately 3-4 ml/kg).This reserve would sustain approximately 4 minutes of apnea before critical arterial hypoxemia develops.

Clinical significance of Preoxygenation[2,4,5]

Complete preoxygenation (3-5 minutes of preoxygenation) with 100% oxygen can increase body's total stored oxygen to approximately 4500 mL, thereby extending the duration of tolerated apnea by at least fourfold. However, during cardiac arrest, pulmonary oxygen reserves become inaccessible due to the absence of circulation, and since the brain lacks intrinsic oxygen stores, loss of consciousness typically occurs within 10 seconds.

Acute respiratory management involves the use of both ambient and positive pressure therapies delivered through various airway devices such as face masks, oropharyngeal and nasopharyngeal airways, and endotracheal tubes.

Physiological Responses to Hypoxemia & Hypoxia

1. Elevated heart rate (tachycardia)
2. Increased respiratory rate (tachypnoea)

Goal of Oxygen Therapy

To enhance the alveolar oxygen concentration (PAO_2) & improve oxygen delivery to the cellular level.

Clinical Significance[1]

Hypoxemia can develop to varying degrees in any patient during the early postoperative recovery phase due to several potential mechanisms. Therefore, it is recommended that all patients receive supplemental oxygen for at least the first 10 minutes following the cessation of general Anaesthesia. Oxygen therapy is especially effective in correcting hypoxemia caused by hypoventilation, as arterial oxygen partial pressure (PaO_2) can be significantly increased by administering a modest rise in the fraction of inspired oxygen (FiO_2), typically above 30% ($FiO_2 \geq 0.3$) or delivering more than 4L/min of oxygen.

Known oxygen concentrations can be delivered using a tightly fitting mask connected to metered air and oxygen flows, typically through an Anaesthetic breathing system or a CPAP device. For young children, oxygen administration may be provided via an oxygen tent, headbox, or O_2 hood.

Humidification of Oxygen[3]

The tracheobronchial mucosa is more susceptible to dehydration than the nasal mucosa, which can lead to impaired mucociliary function and mucosal inflammation. Normally, alveolar air is fully saturated with water vapor at body temperature, containing approximately 44 mg/L of water vapor and exerting a partial pressure of 47 mmHg.

Devices for Oxygen therapy

The performance of oxygen face masks is primarily influenced by their internal volume, the flow rate of the supplied gas, and the presence of side holes. Without a continuous gas supply, a face mask increases the dead space, with the extent of this increase being proportional to the mask's volume. When oxygen is delivered, the fraction of inspired oxygen (FiO_2) rises; however, the exact FiO_2 achieved depends on the interplay between the oxygen flow rate and the patient's breathing pattern.

Effective FiO_2 range and Oxygen delivery systems

For prolonged oxygen therapy, the effective fraction of inspired oxygen (FiO_2) typically ranges between 0.25 and 0.50. Most oxygen delivery systems function

similarly to non-rebreathing systems, allowing carbon dioxide to be fully exhaled while the patient inhales fresh gas.

Oxygen delivery systems are generally classified into two types:
(a) High-flow or fixed-performance systems, such as Venturi masks, which delivers a precise FiO_2.
(b) Low-flow or variable-performance systems, where the FiO_2 varies depending on the patient's breathing pattern.

Indications for Prolonged Oxygen Therapy[5]

- Hypotension
- Ischemic heart disease (IHD)
- Reduced cardiac output, such as in congestive heart failure (CHF)
- Severe anemia
- Obesity, due to decreased respiratory reserve and increased oxygen demand
- Shivering, which can increase oxygen consumption up to fourfold
- Hypothermia, where vasoconstriction limits oxygen delivery to tissues
- Hyperthermia, which elevates oxygen demand
- Pulmonary oedema, causing ventilation-perfusion (V/Q) mismatch
- Airway obstruction, such as in sleep apnea syndrome
- Following major surgery, where Anaesthetic agents and pain reduce respiratory reserve and depth of respiration
- During the post-Anaesthesia care period
- Throughout the postoperative recovery phase

[2]Relationship between SpO_2 and PaO_2

SpO_2 (peripheral oxygen saturation) represents the percentage of haemoglobin saturated with oxygen as measured by pulse oximetry, while PaO_2 (arterial oxygen partial pressure) reflects the actual oxygen pressure dissolved in arterial blood measured by blood gas analysis (ABG).

1. The relationship between SpO_2 and PaO_2 is described by the oxygen-hemoglobin dissociation curve, which is sigmoidal in shape.
2. At higher PaO_2 levels (above ~80 mmHg), SpO_2 remains relatively stable and close to 95–100%.

3. Small changes in PaO_2 within this upper range cause minimal change in SpO_2.
4. At lower PaO_2 values (below ~60 mmHg), SpO_2 decreases sharply, indicating significant drops in oxygen saturation with small decreases in PaO_2.
5. This steep part of the curve means tissue oxygen delivery can be critically compromised even with small PaO_2 reductions.
6. Factors such as pH, temperature, CO_2 levels, and 2,3-DPG can shift the curve, affecting the SpO_2-PaO_2 relationship.

SpO_2 in %	**PO_2/PaO_2 in mmHg (partial Pressure of O2)**
100 %	97-500mmHg
97%	95-110mmHg
95%	85mmHg
90%	60 mmHg
85%	50mmHg
50%	26 mmHg

Table no. 01

Relationship between Oxygen Flow Rate and FiO_2 for nasal cannula or catheter[4]

The fraction of inspired oxygen (FiO_2) delivered to a patient depends on the oxygen flow rate and the delivery device used.

At low flow rates (1–6 L/min) via nasal cannula, FiO_2 increases by approximately 3–4% for each additional liter per minute. For example:

Flow rates (L/min) via nasal cannula	**FiO_2**
1	0.24
2	0.28
3	0.32
4	0.36
5	0.40
6	0.44

Table no. 02

Effect of Oxygen Flow Rates Greater Than 6 L/min on FiO_2 by nasal cannula or catheter

When oxygen flow exceeds 6 L/min, there is minimal further increase in the fraction of inspired oxygen (FiO_2). This plateau occurs because the nasopharyngeal space acts as a reservoir, becoming saturated with nearly 100% oxygen at flows of around 6 L/min. As a result, increasing the flow beyond this point does not significantly raise FiO_2 since the patient is already inhaling oxygen-rich gas from the reservoir.

Several factors affect fraction of inspired oxygen (FiO_2) delivered via a nasal cannula or catheter is

1. The patient's breathing pattern (tidal volume, respiratory rate)
2. Whether they are breathing through the nose or mouth
3. The fit and positioning of the cannula

Relationship between Oxygen Flow Rate and FiO_2:

Simple Face Mask: Delivers higher FiO_2 than nasal cannula. Minimum flow 5L/min (to flush out CO_2)

O_2 Flow Rate (L/min)	Estimated FiO_2
5	0.40
6	0.44
7	0.48
8	0.52
9	0.55
10	~0.60

Table no.03

FiO_2 range: **0.4-0.6**, depending on mask fitting and breathing pattern.

Venturi Mask (Air-entrainment Mask)

1. Precise FiO_2 control, independent of patient's breathing.
2. Color-coded adapters set fixed FiO_2.

Adopter setting	O_2 Flow rate (L/min)	Delivered FiO_2
Blue	2-4	0.24
White	4-6	0.28
Orange	6-8	0.31
Yellow	8–10	0.35
Red	10–12	0.40
Green	12–15	0.60

Table no. 04

Summary Table

Device Type	Flow Rate (L/min)	Approx. FiO_2
Simple Face Mask	5–10	0.4-0.6
Non-Rebreather Mask	10–15	0.6-0.9
Venturi Mask	Varies by adapter	0.24-0.6 (precise)

Table no. 05

1. Beyond 6 L/min, increasing flow produces minimal additional rise in FiO_2 because the nasopharyngeal reservoir is saturated with oxygen.
2. With face masks or high-flow systems, FiO_2 can be more accurately controlled and increased independently of flow rates due to reduced entrainment of room air.
3. FiO_2 delivered by low-flow devices can vary depending on the patient's breathing pattern because room air mixes with the supplied oxygen during inspiration.

Relationship between Oxygen Flow Rate and FiO_2 for Masks with Reservoir Bags (NRB)

A mask with reservoir bag typically refers to a non-rebreather mask (NRB), a high-concentration oxygen delivery system used for patients in acute respiratory distress or hypoxia.

Key Features:

1. Has a reservoir bag that stores 100% oxygen.

2. Includes one-way valves
3. Prevents exhaled air from re-entering the reservoir.
4. Minimizes mixing with room air.
5. Requires a tight seal to function effectively.
6. Delivers high FiO_2, almost pure oxygen.
7. To ensure if reservoir bag remains inflated then FiO_2 approaches 0.9-1 with good mask seal and flow ≥15 L/min.
8. Minimum flow rate: 10 L/min (to keep the bag partially inflated at all times).
9. Target FiO_2: up to 0.95-1 (realistically 0.85-0.95 due to some room air entrainment).
10. The exact FiO_2 depends on the patient's tidal volume and mask seal.
11. Mask seal and patient's ventilation pattern significantly affect actual FiO_2.
12. Not for long-term use; risk of CO_2 retention if not monitored.

O_2 Flow Rate (L/min) with NRB	**Estimated FiO_2 (%)**
10	**~0.6–0.7**
12	**~0.7–0.85**
15	**~0.85–0.95 (up to 1)**

Table no. 06

Oxygen Therapy in the Recovery Ward[3]

The majority of patients emerging from anesthesia require only a moderate increase in the fraction of inspired oxygen (FiO_2) to counteract mild hypoventilation, diffusion hypoxia, and slight ventilation-perfusion (V/Q) mismatches. An FiO_2 of approximately (0.3) is generally sufficient. This level of oxygenation can typically be achieved by delivering oxygen at a flow rate of around 4 L/min using variable performance oxygen delivery devices.

Oxygen Therapy in Patients with Increased Shunt and Chronic Respiratory Conditions

Patients exhibiting increased intrapulmonary shunting, such as those with acute respiratory distress syndrome (ARDS), pulmonary edema, or pulmonary consolidation,

often require administration of high inspired oxygen concentrations (high FiO_2) to maintain adequate oxygenation.

Conversely, individuals with chronic bronchitis or chronic obstructive pulmonary disease (COPD) may develop chronic hypercapnia, where their ventilatory drive is primarily driven by hypoxemia. In these patients, raising PaO_2 above the hypoxic drive threshold can lead to ventilatory depression. Therefore, oxygen therapy should begin with a fixed-performance device delivering approximately 24% oxygen. The patient's clinical status and arterial carbon dioxide levels ($PaCO_2$) should be carefully monitored. If the patient remains stable and $PaCO_2$ rises by no more than 1–1.5 kPa, oxygen concentration can be cautiously increased to 28%, and if necessary, to higher levels to improve PaO_2.

Functional Classification of Causes of Hypoxemia

1. **Reduced Inspired Oxygen Concentration (Low FiO_2)-** Occurs primarily at high altitudes where the ambient oxygen concentration is decreased.
2. **Ventilation-Perfusion (V/Q) Mismatch-** The most common cause of hypoxemia, especially in the postoperative period, resulting from uneven distribution of ventilation and blood flow.
3. **Diffusion Deficits-** Caused by conditions such as interstitial edema or fluid overload that impairs oxygen transfer across the alveolar-capillary membrane.
4. **Shunting** - Blood bypasses ventilated alveoli, as seen in conditions like atrial septal defect (ASD), leading to hypoxemia unresponsive to oxygen therapy.
5. **Hypoventilation** - Reduced ventilation due to causes such as obstructive sleep apnea syndrome, which may be managed with CPAP therapy.
6. **Diffusion Hypoxia** - Occurs after administration of nitrous oxide (N_2O) anesthesia due to rapid diffusion of N_2O out of the blood into the alveoli, diluting alveolar oxygen.

Diffusion Hypoxia[4]

Nitrous oxide is approximately 40 times more soluble in blood than nitrogen. When nitrous oxide administration is stopped at the end of anesthesia, it rapidly diffuses from the blood into the alveoli in larger volumes than nitrogen diffuses out, resulting in a dilution of alveolar gases. This dilution reduces the partial pressure of oxygen (PaO_2), impairing arterial oxygenation if the patient breathes room air. Additionally, the

decreased alveolar carbon dioxide ($PaCO_2$) can lead to hypoventilation. To prevent diffusion hypoxia, supplemental oxygen is administered for at least 10 minutes following the cessation of nitrous oxide anesthesia.

Pulmonary Changes after Abdominal Surgery

In otherwise healthy patients with previously normal lung function, oxygenation is often impaired for at least 48 hours following abdominal surgery. The severity of this impairment correlates with the surgical site: it tends to be mild after lower abdominal procedures, more pronounced following large upper abdominal incisions, and most severe after thoracoabdominal surgeries. Under these conditions, the postoperative decrease in arterial oxygen tension (PaO_2) compared to preoperative values can reach up to 4 kPa (To convert kPa to mmHg, multiply by 7.50062).

Impairment of Oxygenation in the Postoperative Period

Postoperative impairment of oxygenation is primarily related to a reduction in functional residual capacity (FRC). Following induction of anesthesia, there is an immediate and significant decrease in FRC. Further reductions occur due to wound pain, which induces spasm of the expiratory muscles, and abdominal distension, which causes diaphragmatic splinting. The extent of FRC reduction is influenced by the site of the surgical incision, with the most pronounced decreases observed after thoracic or upper abdominal surgeries. Additionally, the supine position contributes to further reduction in FRC.

In most patients, these pulmonary abnormalities gradually resolve, with functional parameters returning toward normal by the 5th to 6th postoperative day.

Adverse Effects of Oxygen Therapy-

Fire Hazard

Oxygen itself is not flammable but significantly supports the combustion of fuels. Increasing the oxygen concentration from the normal atmospheric level of 21% to 100% progressively accelerates the rate and intensity of combustion.

Cardiovascular Depression

An elevated arterial oxygen tension (PaO_2) induces direct vasoconstriction affecting the peripheral vasculature as well as cerebral, coronary, hepatic, and renal circulations. Additionally, exposure to hyperbaric oxygen pressures can lead to direct myocardial depression.

Hyperbaric Oxygen Therapy and Wound Healing

The application of hyperbaric oxygen at chronic & non healing wound care has been shown to promote improved and accelerated healing due to enhanced oxygen delivery to hypoxic tissues of wounds.

Absorption Atelectasis

Due to the high solubility of oxygen in blood, administering 100% oxygen as the inspired gas can result in absorption atelectasis. This occurs in lung units distal to sites of airway closure, where oxygen is rapidly absorbed into the bloodstream, leading to alveolar collapse.

Reducing CO_2 Narcosis in COPD

In patients with chronic bronchitis and chronic obstructive pulmonary disease (COPD) who retain elevated levels of carbon dioxide (CO_2), there is often a diminished sensitivity of central chemoreceptors to CO_2. Consequently, these patients rely more heavily on peripheral chemoreceptors, which respond primarily to low oxygen levels, to drive ventilation. Administering high concentrations of oxygen (high FiO_2) can suppress this peripheral chemoreceptor-driven respiratory drive, potentially leading to hypoventilation and subsequent ventilatory failure.

Clinical Note

In COPD patients, oxygen therapy should aim for a controlled, low FiO_2 to prevent suppression of the hypoxic respiratory drive and reduce the risk of CO_2 narcosis.

Pulmonary Oxygen Toxicity (Lorrain-Smith Effect)

Chronic exposure to high concentrations of inspired oxygen (high FiO_2) can lead to pulmonary oxygen toxicity, known as the Lorrain-Smith effect.

Pathophysiology[4]

Prolonged exposure results in the formation of hyaline membranes and thickening of interlobular and alveolar septa due to interstitial edema and fibroplastic proliferation. These structural changes impair the synthesis of pulmonary surfactant, predisposing the lungs to alveolar collapse (atelectasis) and pulmonary edema.

Oxygen-induced lung injury typically begins after approximately 30 hours of exposure to a partial pressure of inspired oxygen (PiO_2) around 100 kPa (equivalent to

FiO_2 of 1.0 at sea level).

Central Nervous System (CNS) Oxygen Toxicity

CNS oxygen toxicity typically occurs during exposure to hyperbaric oxygen (FiO_2 = 1.0 at pressures >1 atmosphere). One of the hallmark manifestations is the onset of generalized tonic-clonic (grand mal-like) seizures, often without warning. These convulsions are thought to result from oxygen-induced neuronal hyperexcitability and oxidative stress.

Retrolental Fibroplasia (RLP)/Retinopathy of Prematurity (ROP)
Retrolental fibroplasia, now more commonly referred to as retinopathy ofprematurity (ROP), is a condition primarily affecting premature infants exposed to hyperoxia in neonatal or pediatric intensive care units (PICUs). It results from oxygen-induced retinal vasoconstriction, leading to obliteration of immature retinal vessels. This is followed by abnormal neovascularization at the sites of injury, forming a proliferative retinopathy.

The leakage of intravascular fluid from these fragile new vessels can cause vitreoretinal adhesions and may progress to retinal detachment (RD), potentially resulting in permanent vision loss.

Importantly, the risk is not solely determined by the FiO_2 delivered but by the overall oxygen exposure and fluctuations in arterial oxygen tension (PaO_2).

Depressed Haematopoiesis

Prolonged exposure to high concentrations of inspired oxygen (high FiO_2) has been associated with suppression of hematopoiesis, particularly erythropoiesis, leading to a reduction in red blood cell production and the development of oxygen-induced anemia. This effect is believed to result from decreased erythropoietin synthesis in response to sustained hyperoxia.

Conclusion

Medical oxygen stands as a cornerstone of patient care, intricately woven into every level of the healthcare continuum—from lifesaving interventions in emergencies and surgery to sustaining those with chronic respiratory disorders. This review has illuminated the entire journey of oxygen: tracing its production and storage, describing its delivery and therapeutic uses, and detailing the physiological, safety, and regulatory facets that ensure effective clinical practice. Recent global events, particularly the

COVID-19 pandemic, have spotlighted the strengths and vulnerabilities of oxygen supply systems, driving innovations and raising awareness of its indispensable nature. As we look to the future, continued advances in oxygen technology, delivery methods, and safety protocols will further refine its use, ensuring that healthcare systems are equipped to meet both routine and extraordinary challenges. Understanding the complexities and best practices surrounding medical oxygen empowers practitioners and policymakers alike to deliver safer, smarter, and more resilient care to all who depend on this vital resource

Source of funding – No source of funding was used for this review article.

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