HYPOXEMIA DURING BRONCHOSCOPY PROCEDURE: WHAT WE NEED TO UNDERSTAND AND HOW TO ANTICIPATE IT

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ABSTRACT

By inserting a Diagnostic Sign (bronchoscope) into the airway, bronchoscopy is an invasive medical treatment that allows tracheobronchial viewing in order to evaluate pathological abnormalities that develop in the lung. The importance of bronchoscopy is growing as lung disease cases become more complex and the demand for minimally invasive diagnostic methods rises. Use of bronchoscopy as a diagnostic and therapeutic tool are its two primary indications. With a low morbidity rate (0.1–2.5%) and a very low fatality rate (0.05%), bronchoscopy is a surgery that is generally considered to be safe. The risk of consequences from bronchoscopy still exists, one of which being hypoxemia. One of the most frequent side effects brought on by several variables is hypoxemia. Due to the use of sedation during bronchoscopy, hypoxemia frequently happens. This situation is often transient and is reversible. The operator must take into account the possibility of hypoxemia while doing the process in order to know when to stop. Hypoxemia should be anticipated, therefore monitoring and oxygen supplementation should be priorities during the process.

Keywords: Hypoxemia, Complications, Bronchoscopy

ABSTRAK

Bronkoskopi adalah suatu prosedur medis yang invasif yang memberikan visualisasi trakeobronkial dengan menempatkan Diagnostic Sign (Bronkoskop) ke dalam saluran napas sehingga dapat menilai kelainan patologis yang terjadi di paru. Seiring dengan semakin kompleksnya kasus penyakit paru dan kebutuhan prosedur diagnostik yang minimal, maka kontribusi bronkoskopi semakin penting. Dua indikasi utama penggunaan bronkoskopi yaitu sebagai modalitas diagnostik dan juga terapeutik. Bronkoskopi termasuk salah satu prosedur yang relatif aman dengan angka morbiditas yang rendah (0,1-2,5%) dan angka mortalitas yang sangat rendah (<0,05%). Meskipun demikian, tindakan bronkoskopi juga tetap berpotensi menimbulkan komplikasi dan salah satunya adalah hipoksemia. Hipoksemia adalah salah satu komplikasi yang paling besar kemungkinan terjadi yang disebabkan oleh beberapa faktor. Hipoksemia sering terjadi selama bronkoskopi, akibat penggunaan sedasi. Kondisi ini biasanya bersifat sementara dan dapat normal kembali. Potensi terjadinya hipoksemia pada saat prosedur juga memerlukan pertimbangan dari operator untuk menghentikan prosedur. Pentingnya antisipasi terhadap potensi hipoksemia, maka pemantauan dan suplementasi oksigen perlu menjadi perhatian selama prosedur berjalan.

Kata Kunci: Hipoksemia, Komplikasi, Bronkoskopi

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INTRODUCTION

In order to evaluate the tracheobronchial bifurcation, which is crucial for the diagnosis and treatment of lung disorders, bronchoscopy is an invasive operation.[1] The primary indication for therapeutic bronchoscopy has changed from the removal of airway foreign bodies as a result of the global lung cancer epidemic to many cases of airway obstruction by malignant tumors and benign stenosis.[2,3] As part of staging lung cancer patients, bronchoscopy is essential for mediastinal lymph node evaluation. Less than 1% of patients experience bronchoscopy complications, such as respiratory distress, hemorrhage, pneumothorax, infection, and cardiac arrhythmias. Even when patients get oxygen supplementation during bronchoscopy, hypoxemia is nevertheless a typical side effect, especially if sedation is used.[4–7]

In bronchoscopic operations, hypoxemia is defined as a percutaneous oxygen saturation (SpO2) reduction of more than 5% from baseline values or a sustained SpO2 below 90% for more than one minute.[5] An major bronchoscopy complication with a frequency of 2.5-69% is hypoxemia.[8] If hypoxemia lasts more than a minute, the operator should think about discontinuing the treatment. Typically, this drop in SpO2 is temporary and returns to normal rapidly.[5] During bronchoscopy, routine oxygen saturation monitoring is advised by the British Thoracic Society (BTS).[6,8]

Hypoxemia during bronchoscopy is something to be aware of as it can lead to fatal complications. Age, the length of anesthetic, the use of breathing-depressing medications, the type of action, and the patient's position during bronchoscopy are all risk variables that have been studied in relation to hypoxemia-related problems during bronchoscopy. Since bronchoscopy is now frequently used and more lung cancer patients are having diagnostic bronchoscopy procedures, it is important to identify the patient's risk factors for hypoxemia before the procedure so that any complications can be managed promptly and effectively to prevent fatal ones.[6]

MECHANISM OF HYPOXEMIA

Four factors can lead to hypoxemia: hypoventilation, right-to-left shunt, poor diffusion, and ventilation-perfusion (V/Q) mismatch. Changes in the alveolar-arterial oxygen gradient (A-a gradient) and the reaction to oxygen treatment can be used to identify these four hypoxemia processes. The formula for the A-a gradient is as follows: A-a gradient = PAO2 - PaO2. The A-a gradient is the difference between the oxygen pressure in the alveolus (PAO2) and the oxygen pressure in the arterial circulation (PaO2). When there is damage to the alveolus capillary membrane, the A-a gradient value will widen because it describes the integrity of the alveolus capillary membrane and the efficiency of gas exchange. With aging, the PaO2 will decrease because of an increase in V/Q mismatch, increasing the A-a gradient value, which is now 10 mmHg in young people.[9,10]

Ventilation-perfusion mismatch

The most frequent cause of hypoxemia is a V/Q mismatch, which has a normal value of 0.8. The V/Q ratio, perfusion, and ventilation are not uniformly distributed across the lung. Regional variation of the V/Q ratio is caused by gravity and fluctuating subatmospheric intra pleural pressure. Because the drop in perfusion is greater than the decrease in ventilation at the apex of the lung, ventilation and perfusion are higher at the basal and lower at the apex of the lung, although the V/Q ratio is lower at the basal and higher at the latter. Due to a decrease in PAO2 and consequent decrease in PaO2, hypoxemia only happens at low V/Q ratios.[9]

When hypoxemia develops, particularly in chronic hypoxemia, the body will restrict perfusion to the portion of the lung that has experienced a decrease in ventilation through a distinct hypoxic pulmonary vasoconstriction (HPV) mechanism in pulmonary blood vessels. In the pulmonary vasculature, hypoxia will lead to the closure of potassium (K^+) channels, which will result

in intracellular K^+ ion buildup and cell depolarization. Pulmonary vascular vasoconstriction, which is $Ca2^+$ mediated vasoconstriction, results from calcium (Ca^{2+}) channels opening during depolarization. This vasoconstriction will direct blood to the ventilated area of the lung and decrease perfusion to that area. Maintaining congruence between ventilation and perfusion is the major objective of this compensatory system.[9]

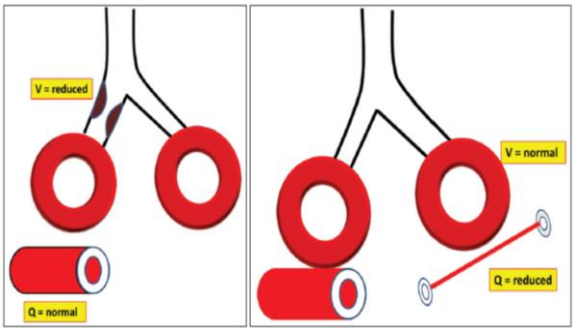

Figure 1. Mismatch mechanism for V/Q

When ventilation is increased in excess of perfusion, as in individuals with pulmonary embolism who may experience a loss of space-like effect, a high V/Q ratio results. Although a high V/Q ratio has little impact on blood oxygenation, it can nonetheless result in hypoxemia if total ventilation does not rise in response. In areas of the lung with low V/Q, a compensatory increase in ventilation may result in the V/Q ratio returning to normal. Figure 1 depicts the V/Q mismatch mechanism. Hypoxemia and a widening A-a gradient are features of V/Q mismatch, both of which can be treated with oxygen therapy. Asthma, COPD, bronchiectasis, cystic fibrosis, intersitial lung disorders (ILDs), and pulmonary hypertension are common causes of hypoxemia via the V/Q mismatch mechanism.[9]

Shunt

As seen in Figure 2, a shunt is a situation where blood from the right side of the heart enters the left side without exchanging gases. When blood from the bronchial veins is transported to the pulmonary veins, there is typically a shunt with a modest fraction (2–3%) of carbon monoxide (CO) present. Because there is no ventilation, a shunt is a severe instance of V/Q mismatch. Shunts stand out from other hypoxemia-causing mechanisms due to their poor sensitivity to therapeutic oxygen supply. The inability of oxygen to raise PAO2 in the unventilated lung is the cause of oxygen therapy's failure to elevate PaO2.[9]

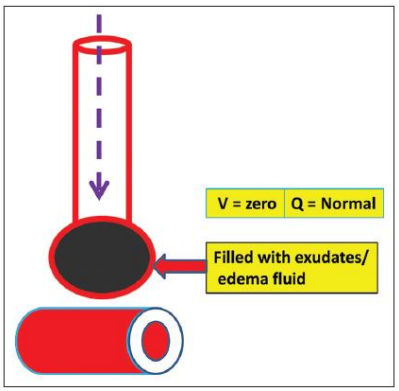

Figure 2. Shunt

In shunts, hypercapnia is uncommon and won't happen until the shunt fraction exceeds 50%. Because of the high pressure of carbon dioxide in the arterial blood (PaCO2) leaving the shunt region, hypercapnia does not happen as a result of chemoreceptor stimulation of the respiratory center. The shunt fraction can be roughly calculated using PaO2/FiO2. If PaO2/FiO2 is less than 200, the shunt fraction value is greater than 20%, and if PaO2/FiO2 is greater than 200, the shunt fraction value is lower than 20%. A widening A-a gradient, a poor response to oxygen administration, and normal PaCO2 levels are all signs of hypoxemia brought on by shunts. Pneumonia, pulmonary edema, acute respiratory distress syndrome (ARDS), alveolus collapse, and pulmonary artery-vein connection are among the conditions that can result in shunts.[9]

Diffusion Disorder

When oxygen transport through capillary membranes is compromised, diffusion problems develop. Reduced lung surface area for diffusion, inflammation and fibrosis of the alveolocapillary membrane, low oxygen levels in the alveoli, and extremely rapid capillary transit times can all contribute to impaired diffusion. Since both oxygen and carbon dioxide are carried across the alveolocapillary membrane, impaired diffusion may potentially result in both hypoxemia and hypercapnia; nevertheless, hypercapnia is uncommon since carbon dioxide (CO2) is 20 times more soluble in water than oxygen. Another explanation would be the removal of CO2 through hypoxemia-mediated ventilatory stimulation.[9] One crucial feature of diffusion problems is that the resulting hypoxemia will worsen during activity. Exercise will result in shorter capillary transit times because of increased CO and lower venous dissolved oxygen concentrations because of increased tissue oxygen use. A widening of the A-a gradient, a generally normal PaCO2, and a satisfactory response to oxygen therapy are further features of hypoxemia brought on by diffusion problems. Emphysema and ILDs are significant contributors to diffusion disorder development.[9]

Hypoventilation

Because sufficient ventilation is required to eliminate CO2, a high PaCO2 concentration is a sign of hypoventilation. Additionally necessary for oxygenation is ventilation, and hypoventilation will lead to decreased PAO2 and a corresponding decline in PaO2. Because the alveolocapillary membrane is intact in this state, hypoventilation also has a normal A-a gradient. Long-term hypoventilation can broaden the A-a gradient and lead to atelectaxis in some lung regions. In healthy lungs, hypoventilation does not significantly increase blood oxygen levels, although severe hypoxemia can occur when lung disease is present.[9]

With oxygen therapy, hypoxemia caused by hypoventilation is quickly and readily treated. Even with hypoventilation and hypercapnia, hypoxemia can be treated with oxygen therapy. The next sign of hypoventilation is the simultaneous movement of PaO2 and PaCO2 in opposing directions. If they do not rise to the same level, hypoventilation can be ruled out as a possible cause of hypoxemia. The respiratory pump, including the respiratory center in the brainstem, the spinal cord, the neuromuscular junction, the respiratory muscles, and abnormalities in the chest wall, all malfunction, resulting in hypoventilation.[9] In table 1, the causes of hypoventilation are listed. Figure 3 depicts the mechanism of hypoxemia as well as its many traits.

Table 1. Causes of Hypoventilation

Central nervous disorder	–	Drug overdose: Opioids, benzodiazepines, alcohol.
	–	Hemorrhage or infarction of the brainstem.
	–	Primary alveolar hypoventilation
Spinal cord level	–	*Amyotrophic lateral sclerosis*
	–	Injury to the cervical spinal cord
Innervation of respiratory muscles	–	*Guillain-Barre Syndrome*
Neuromuscular junction	–	Myasthenia gravis
	–	*Lambert-Eaton Syndrome*
Respiratory muscles	–	Myopathy
Chest wall defect	–	*Kyphoscoliosis*
	–	*Thoracoplasty*
	–	Fibrotorax

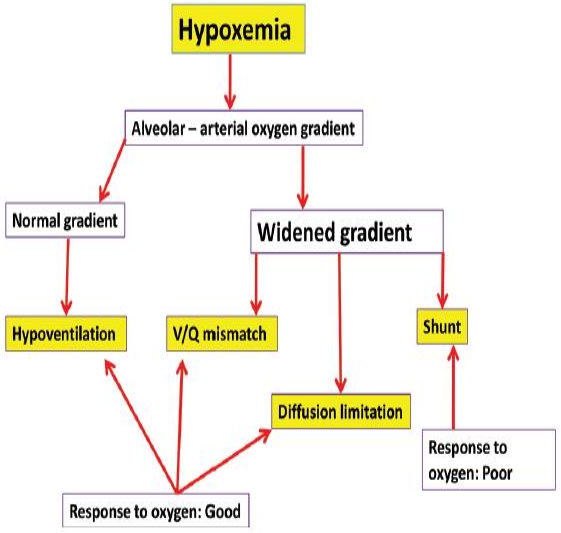

Figure 3. Mechanisms and many aspects of Hypoxemia

IMPACT OF HYPOXEMIA

Cyanosis, tachycardia, and a decline in mental state are symptoms of hypoxemia, but for the majority of patients, a low oxygen saturation test using pulse oximetry is the initial way to diagnose the condition. Little physiological change results from mild hypoxia. When PaO2 is 60 mmHg and pH is normal, arterial oxygen saturation stays about 90%. Only modest mental status impairment, decreased visual acuity, and slight hyperventilation are observed as anomalies. Several organ systems will suffer damage if PaO2 suddenly falls below 40–50 mmHg.[10]

Hypoxemia most frequently causes symptoms of headache and reduced consciousness in the central nervous system. Seizures, retinal bleeding, and lifelong brain damage are among potential effects of severe acute hypoxemia. Catecholamine release can cause tachycardia and mild hypertension, but patients with severe hypoxemia may also have bradycardia, hypotension, and even cardiac collapse. Proteinuria and salt retention may be the results of decreased renal function. Alveolar hypoxia and hypoxia-induced pulmonary vascular vasoconstriction are related, and this can lead to pulmonary hypertension.[10]

Tissue hypoxia can be harmful and result in hypoxemia. Blood oxygen capacity, oxygen affinity to hemoglobin, cardiac output, and blood flow distribution are other parameters that affect oxygen delivery to tissues in addition to arterial oxygen tension. The sensitivity of various tissues to hypoxemia varies greatly. The myocardial and central nervous system are at great risk. Within 4-6 seconds of stopping blood supply to the cerebral cortex, function is lost, followed by loss of consciousness and irreversible alterations within 10-20 seconds.[10]

If tissue PO2 falls below a specific level, aerobic oxidation will stop, and anaerobic glycolysis will take place, producing a lot of lactic acid. The precise PO2 levels at which this happens are unknown and probably vary in various tissues. Around 1-3 mmHg is the crucial range for intracellular PO2 in mitochondria. Despite being a rather ineffective process for turning glucose into energy, anaerobic glycolysis is crucial for preserving tissue viability in cases of respiratory failure. Metabolic acidosis is brought on by the significant amounts of lactic acid that are produced and discharged into the circulation. When tissue oxygenation has increased, lactic acid can either be converted back into glucose or used to produce energy.[10]

HYPOXEMIA DURING BRONCHOSCOPY

Term and Epidemiology

A drop in the partial pressure of oxygen in the blood indicates the presence of hypoxemia. By measuring arterial oxygen pressure (PaO2), arterial oxygen content (CaO2), arterial oxygen saturation (SaO2), and the PaO2/FiO2 ratio, hypoxemia can be detected.[11] One of the most frequent problems during flexible bronchoscopy operations is hypoxemia, which may typically be avoided by careful observation and, if necessary, oxygen administration through a nasal

cannula.[11] During bronchoscopy, the PaO2 typically falls to 20 mmHg, with a range of 4-38 mmHg.[12,13] A fall in SpO2 of more than 4% from the basal value or a drop to less than 90% for longer than a minute during bronchoscopy are both considered hypoxemia in bronchoscopy.[4,14]

One of the serious side effects of bronchoscopy, hypoxemia happens in 2.5–69% of patients and necessitates prompt oxygen supplementation.[8] Without oxygen support, 35% of bronchoscopy patients Despite being common, hypoxemia during bronchoscopy should still be of concern due to the more serious consequences that may occur. An accurate noninvasive way to determine hypoxemia is to use pulse oximetry to monitor the percutaneous oxygen saturation (SpO2) of patients during bronchoscopy.[15] Upper airway obstruction, Bronchoalveolar Lavage (BAL), suction, hypoventilation, excessive sedation, insufficient sedation, inadequate oxygen supplementation, hemorrhage, bronchospasm, laryngospasm, and pneumothorax secondary to TBLB or other interventional treatments are common causes of hypoxemia during flexible bronchoscopy.[16,17]

The imbalance between ventilation and perfusion (V/Q) and hypoventilation brought on by sedation are the two processes causing hypoxemia during bronchoscopy.[18] The most frequent cause of hypoxemia is a V/Q imbalance. Hypoxemia results from a drop in the arterial oxygen concentration following a decrease in the alveolar oxygen concentration due to a low V/Q ratio. The respiratory center in the brainstem, the spinal cord, the respiratory muscle innervation, the neuromuscular junction, the respiratory muscles, and the development of the chest wall are the main respiratory pump components that malfunction and lead to hypoventilation. A high arterial partial pressure of carbon dioxide (PaCO2) is a sign of hypoventilation since sufficient ventilation is needed to eliminate carbon dioxide (CO). Hypoventilation will result in a decrease in the partial pressure of oxygen (PAO2) in the alveoli, which in turn will result in a fall in PaO2. Ventilation is also necessary for oxygenation.[9]

When the patient is first put to sleep, there is typically a large drop in SpO2 that gets worse as the bronchoscope goes through the vocal cords.[15] Studies on the factors that increase the risk of hypoxemia during bronchoscopy have yielded conflicting findings. Older age, low Forced Expiratory Volume $_1$ (FEV1) values, the endobronchial ultrasound (EBUS) procedure, duration of sedation, and dose of midazolam utilized were linked with the incidence of hypoxemia during the procedure, according to a study by Choi et al. on 2520 patients undergoing bronchoscopy with sedation. Age > 60 years (odds ratio [OR], 1.32), low FEV1 values (OR, 0.99), and sedation lasting longer than 40 minutes (OR, 1.33) were identified as important risk variables by multivariate analysis.[5]

Bronchoscopy Risk Factors for Hypoxemia

Age

While this difference was not statistically significant, Silvestri et al. assessed the use of fospropofol disodium for sedation in patients undergoing flexible bronchoscopy and found that older individuals were more likely to experience hypoxemia than younger patients.[19] According to Shinagawa et al., age and the prevalence of hypoxemia during bronchoscopy are related. 55% of patients older than 80 years old and 27% of patients younger than 80 years old experienced desaturation with SpO2 values 90% for 10 seconds.[17] Age was not a factor in systemic desaturation during bronchoscopy, according to research done by Vasko et al. In their study, Putra et al. also discovered that there exhibited hypoxemia; however, with nasal oxygen supplementation of 4 liters/minute, only 3% of midazolam- and 30% of propofol-treated patients experienced hypoxemia.10 In a separate study by Putra et al. at the Jakarta Friendship National Respiratory Referral Hospital, 40 patients (20.5%) out of 195 who underwent bronchoscopy were found to have hypoxemia.[6]

was no correlation between age and the prevalence of hypoxemia during bronchoscopy.[6,8]

Function of the Lungs

One of the variables that can forecast the occurrence of hypoxemia during bronchoscopy is FEV1 value. A study by Vasko et al. confirmed the findings of the study by Choi et al. and revealed that low FEV1 values were a major predictor of desaturation. The probability of systemic desaturation during bronchoscopy decreased by 50% for every 100 ml increase in FEV1, although Forced Vital Capacity (FVC) had no appreciable effect on systemic desaturation.[8] Those with VEP1 1 liter had a higher risk of desaturation during bronchoscopy than those with FEV1 > 1 liter, according to Shinagawa et al.'s study.[17]

Reflex bronchoconstriction due to mechanical stimulation of subepithelial receptors by the bronchoscope, stimulation by large doses of local anesthetic drugs, partial airway obstruction by the bronchoscope, suction action and local anesthetic solutions and rinses in the alveoli are factors that cause V/Q imbalance. Decreased FEV1 values indicate airflow resistance, so bronchoscopy in patients with low FEV1 values may increase the risk of V/Q imbalance leading to hypoxemia.[12,16,20]

Disease of the Lungs

The underlying reason causing hypoxemia during bronchoscopy may be lung disease, with V/Q imbalance serving as the risk factor. The incidence of hypoxemia did not differ between COPD patients and non-COPD patients, according to a case-control study by Grendelmeier et al. on flexible bronchoscopy with sedation in patients with chronic obstructive pulmonary disease (COPD), but the risk of hypoxemia increased in patients with severe and very severe COPD (GOLD III and IV).[21] In a research by Checani et al. The kind of auxiliary procedures carried out for both diagnostic and therapeutic objectives affects hypoxemia in bronchoscopy. Tidal volume and functional residual capacity are decreased during bronchoscopy suction, which may raise the risk of hypoxemia. After Transbronchial Lung Biopsy (TBLB), excessive bleeding can potentially lead to a on 26 COPD patients with hypercarbia who underwent bronchoscopy, it was discovered that 26% of patients experienced oxygen desaturation, and 62% of patients developed bronchospasm.[13,22] According to research by Choi et al. desaturation occurred in 25% of COPD patients having bronchoscopies.[5]

Due to the high incidence of lung cancer in people with idiopathic pulmonary fibrosis, bronchoscopies are frequently performed on patients with pulmonary fibrosis. Due to the difficulties in detecting this condition, pulmonary fibrosis has not previously been identified as a significant risk factor for desaturation during bronchoscopy. According to a research by Shinagawa et al. 3 out of every 4 patients with pulmonary fibrosis who had bronchoscopy suffered bouts of desaturation at some point during the procedure, with an odds ratio of 2.76 (p=0.039) among patients who had been diagnosed with pulmonary fibrosis based on results from thoracic computed tomography (CT) scans. Patients with lung cancer are frequently diagnosed with pleural effusion. Shinagawa et al. discovered that desaturation occurred during bronchoscopy in 79 out of 328 patients (24%) with pleural effusion.[17]

Variety of Action

When a flexible bronchoscope (Figure 1) is inserted into the trachea, the cross-sectional area of the trachea is reduced by 10-15%, increasing airflow resistance. Both rigid and flexible bronchoscopes carry the risk of hypoxemia during the surgery.[20] Rigid bronchoscopes (Figure 2) have an advantage in airway control due to their greater diameter and open slit along the distal trunk to allow for more ventilation.[3,4] Anesthesiologists have alternatives for managing the airway during rigid bronchoscopy under general anesthesia, including apneic oxygenation, spontaneous ventilation, controlled mechanical ventilation, high frequency ventilation, and manual jet ventilation.[23]

V/Q imbalance and hypoxemia.[12,16,24] The risk of hypoxemia after bronchoscopy and BAL is increased, especially if a lot of rinse fluid is utilized.[2,18,25] Desaturation was observed in 216 of 821 patients (26.3%) who underwent endobronchial ultrasound-guided transbronchial needle aspiration (EBUS-TBNA), according to a research by Choi et al.[5]

Sedation Types

Although rigid or flexible bronchoscopy can be carried out under local or general anesthesia, general anesthesia is more frequently used for patient comfort and procedure safety. According to a study by Putra et al., patients having bronchoscopy while under local anesthesia and conscious sedation had a higher prevalence of hypoxemia (21.4%). Patients having bronchoscopy while under local anesthesia and conscious sedation are more likely to experience hypoxemia due to the bronchoconstrictive effects of lidocaine combined with the respiratory depression effects of midazolam, fentanyl, and propofol supported by insufficient airway patency.[6]

In a research by Choi et al., 565 (22.4%) of the 2520 adult patients who underwent bronchoscopy operations while sedated experienced hypoxemia.[5] In flexible bronchoscopy, benzodiazepines and narcotics, such as midazolam and fentanyl, are frequently used. Flumazenil or naloxone can be used to treat secondary hypoventilation caused by sedation caused by opioids, benzodiazepines, and hypnotics.[16,20]

Treatment of Hypoxemia During Bronchoscopy

Most desaturations that happen during bronchoscopy are temporary and don't need treatment. In contrast to hypoventilation, where the alveolar-arterial (A-a) gradient is often normal, V/Q imbalance is characterized by a wider A-a gradient. Both of these methods can cause hypoxemia, and both respond well to oxygen therapy. To lower the risk of cardiac arrhythmias both during flexible bronchoscopy and during the recovery phase, oxygen supplementation is advised to attain an arterial oxygen saturation of at least 90%. The majority of cardiac arrhythmia problems following bronchoscopy operations are associated with hypoxemia, namely PaO2 60 mmHg.[13,16,18] Depending on FEV1 or peak expiratory flow (PEF), the percentage of patients who need additional oxygen during bronchoscopy ranges from 5 to 32%.[15]

The St Vincent's stepwise technique was created by Chhajed et al. to treat hypoxemia during bronchoscopy. Throughout the procedure, a qualified assistant operator should monitor the patient's vital signs such SpO2, ECG, and blood pressure. For oral flexible bronchoscopy, all patients received oxygen supplementation through a nasal cannula at a rate of 4 liters/minute. In the event of desaturation, the first course of action was to increase oxygen supplementation to 6 liters/minute while elevating the jaw.[16]

With a 6 liter/minute nasal cannula, oxygen saturation that falls below 90% is treated with oxygen supplementation delivered through insertion of a nasopharyngeal tube and bronchoscopy drawn up to the trachea. Supplemental oxygen is administered when the SpO2 readings remain below 90% using a 7 French oxygen catheter inserted through the nose either just above the larynx or in the proximal trachea. If the SpO2 stays below 90%, the bronchoscope should be removed, sedative countermeasures administered, and bag and mask ventilation carried out until the target spontaneous ventilation is attained.[16]

Although oxygen supplementation should continue in the recovery room after flexible bronchoscopy, hypoxemia may resolve at some point. In 30% of patients, however, oxygen saturation may linger below pre-procedure levels for three hours. Some patients experience hypoxemia prior to flexible bronchoscopy, which results in the need for higher flow oxygen supply following the procedure and might even necessitate non-rebreathing hoods. The American Thoracic Society advises against flexible

bronchoscopy and BAL in patients with hypoxemia that cannot be treated with oxygen supplementation to a minimum value of PaO2 75 mmHg or SaO2 >90%.[16,24,26]

Prognosis

While hypoxemia frequently occurs during bronchoscopy, it is usually brief and only becomes problematic when it persists for more than a minute. The likelihood of consequences from hypoxemia is influenced by baseline SaO2 concentrations, lung function, comorbidities, sedation, and sampling techniques. The increased cardiac workload brought on by hypoxemia during bronchoscopy is typically accompanied with an increase in heart rate of about 40% over basal values, a rise in blood pressure of about 30% above basal values, and an increase in cardiac index of around 17-32% above basal values. Premature contractions, bigemini, and trigemini are common ventricular arrhythmias that typically manifest when the bronchoscope is inserted via the vocal cords and are linked to low oxygen saturation.[15]

The brain is the organ most susceptible to hypoxia, particularly during desaturation events. In a study, Vasko et al. evaluated cerebral and systemic saturation during a diagnostic bronchoscopy procedure. Despite a drop in systemic saturation, this study did not discover a decrease in cerebral tissue saturation during bronchoscopy. This is believed to be the case because control of brain tissue metabolic rate counteracts short-term systemic desaturation, maintaining brain tissue oxygenation during a brief drop in systemic saturation. Bronchoscopy-related hypoxemia is caused by a V/Q imbalance and hypoventilation, which allows for a good response to oxygen supplementation and the avoidance of subsequent difficulties.[8,9]

CONCLUSION

One of the consequences that frequently happens during bronchoscopy is hypoxemia. Oxygen therapy can be used to treat transient hypoxemia. Age, low FEV1 values, patients with lung illness, kind of action, and sedation are risk factors that can affect hypoxemia during bronchoscopy. Bronchoscopy-related hypoxemia that cannot be treated can result in catastrophic complications like cardiac arrhythmia, coronary vascular abnormalities, neurological problems, acute respiratory problems, and death. To prevent more serious problems, oxygen therapy with a goal arterial oxygen saturation >90% is the optimum approach.

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