Original Article

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Evaluation of Postoperative Hypoxemia by Pulse Oximetry

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Abstract

Present study was carried out in randomly choosen 150 patients of ASA grade-I and grade-II having neither respiratory nor cardiovascular diseases, no obecity.Study included children, adult as well as middle age group posted for various surgical operations under general anaesthesia.The aim of study was to detect early up to one hour postoperative hypoxemia by continuous monitoring of oxygen saturation by pulse oximetry and role of oxygen therapy to treat the hypoxemia. In our study base line hypoxemia was taken at SaO_2 92%.All patients were premedicated with i.v atropine/ glycopyrollate, i.v diazepam, i.v fortwin and was induced with i.v thiopentone sodium or ketamine hydrochloride and endotracheal intubation was done under effect of suxamethonium and anaesthesia was maintained on oxygen + nitrous oxide + i.v norcuron+intermittent halothane for few patients with controlled ventilation.At the end of surgery all patients were extubated after reversal with i.v neostigmine and i.v atropine/i.v glycopyrollate and observed on operation table for first 15. min without supplementation of oxygen to detect the incidence of early postoperative hypoxemia. In first 15 min . Out of 150 patients 53(35.33%) patients became hypoxemic and were treated with supplementation of 4lit/min 100% oxygen through face mask for 15 min.on operation table, then shifted in recovery room which was very near to operation theatre. All patients those who received oxygen as well as not received were observed in recovery room up to one hour. Out of 53 patients only 3 patients became hypoxemic in second episode in between 16 to 30 min.and were treated as above then not a single patient became hypoxemic.

Keywords: Hypoxemia; Oxygen; Pulse Oxemetry; Postoperative.

Introduction

Hypoxemia is one of the most feared critical incidence during anaesthesia as well as in the postoperative period early as well as late. Postoperative hypoxemia can be defined as a state of reduced oxygenation following surgery and anaesthesia, if SaO_2 is less than 92%. At such levels of oxygenation, tissue hypoxemia may occur.

Postoperative hypoxemia was identified as early as in 1930 as a frequent sequelae, Churchill et al in 1927 noted reduction in vital capacity following surgery, segmental atelectasis and an increase in the alveolar arterial oxygen tension gradient due to disturbances in relationship of ventilation to perfusion i.e. regional under-ventilation and airway closure as a cause of postoperative hypoxemia. Tissue hypoxemia is a major cause of morbidity and is ultimately the cause of death in most of the patients. Hypoxemia in a early postoperative period can be difficult to diagnose or assess clinically. Cyanosis is a misleading hard to detect and impossible to quantify.

Tachycardia is not a reliable or specific indicator of hypoxemia. The rate and depth of the breathing is also of not of much useful. Hence application of pulse oximeter technology may be expected to yield increased accuracy in assessing arterial oxygenation compared to clinical assessment. Invasive procedures like analysis of arterial blood gases and transcutaneous oxygen tension measurements, although provide continuous and reliable information but they require local site preparation and air tight probe

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mantling, in addition to limitations and its complications being a invasive procedure. Clinical utility of continuous noninvasive measurement of arterial oxygen saturation by pulse oximetry in operating room was discovered in 1980 by William New. The oximeter detects arterials than the capillary haemoglobin saturation on a beat to beat variation. This method being a fairly rapid and accurate was a one of the vital tool in our study.

In the present study, oxygen saturation was measured preoperatively, intrapperatively as well as in the early postoperative period upto one hour from the conclusion of anaesthesia. The aim of the present randomized study was to determine if and to what extent monitoring with a pulse oximeter in all age group patients receiving general anaesthesia, reduced the incidence, severity and duration of hypoxemia. Preoperatively, intraoperatively in operating room and postoperatively in recovery room.

Material and Methods

Present study was carried out in randomly choosen 150 patients of ASA - grade - I & II having neither respiratory nor cardiovascular disease or no obesity. Study included children, adult as well as middle age group posted for various surgical operations under general anaesthesia.

On operation table SaO_2 of all patients were recorded as base line preoperatively by pulse oximeter. After that all patients were premedicated with I. V. atropine 0.02 mg/kg or I.V. glycopyrolate 0.004 mg/kg I.V. fortwin 0.5 mg/kg and I.V. diazepam 0.2 mg/kg. Patients were induced with I. V. thiopentone sodium 5-7 mg/kg or I. V. ketamine hydrochloride 2 mg/kg and intubated under effect of suxamethonium 2 mg/kg. anaesthesia was maintained on 50% O_2 + 50% N_2O + I. V. norcuron with controlled ventilation and few patients received 0.5 to 1% halothane intermittently.At the conclusion of surgical procedures all patients were reversed by giving I. V. atropine 0.03 mg/kg or I. V. glycopyrollate 0.004 mg/kg and I. V. neostigmine 0.04 mg/kg. In our study after extubation no oxygen was given to any patient. The early postoperative period was labeled and fixed "since extubation upto one hour".

First 15 min. of early postoperative period, the patient was closely observed on the operation table itself for various clinical parameters and the arterial oxygen saturation readings were noted for study purposes at "O" hours, at 15 min., 30 min., 45 min. and at 60 min. Whenever SaO_2 went of to 92% or below, we gave 100% oxygen 4 lit/min through face mask upto 15 min. on operation table itself. Then all patients who received as well as did not received oxygen therapy were transferred to recovery room which was very near to operation theatre. Any patient who had shown second episode of hypoxemia was given once again 4 lit. of oxygen/min for another 15 minutes in recovery room. During arterial oxygen saturation monitoring, pulse rate as well as blood pressure were recorded.

Adequacy of ventilation was assessed clinically by observing the expiratory air flow and volume from the nose or mouth and the movement of the thorax with or without auscultation with stethoscope

Observations and Results

Present study was carried out in 150 patients of ASA [Gr-I and Gr-II] receiving general anaesthesia with or without halothane for various surgical procedures. Arterial oxygen saturation of each patient was monitored from preoperative to one hour of early postoperative period through peroperative and it was corelated with various clinical vital parameters and all the findings were noted and all the observations were as per follows :

Table 1: Showing details of patients study

Age in years	No. of patients	Incidence of hypoxemia			
		Male	Female	Total	Percentage (%)
00-10	38	09	06	15	39.47
11-20	26	05	05	10	38.46
21-30	40	06	06	12	30.00
31-40	28	07	02	09	32.14
41-50	18	02	05	07	38.39
Total	150	29	24	53	-

Table 1 shows that, the youngest one was 1.5 years and oldest was of 50 years. The incidence of hypoxemia between the age groups of 0-10 years, 11-20 years, 21-30 years, 31-40 years and 41-50 years was 39.47%, 38.46%, 30.00%, 32.14% and 38.39% respectively.

In our random study of 150 patients we found

postoperative hypoxemia in 53 (35.33%) patients. We observed that the incidence of postoperative hypoxemia was more in extremes of age. (Paediatric and old patients)

Table 2 shows that, the hypoxemia was detected in 43 (33.86%) patients induced with I. V. thiopentone sodium and in 10 (43.48%) patients induced with I.V. ketamine hydrochloride. We observed that, incidence of hypoxemia was more in patients induced with I.V. ketamine hydrochloride in relation to thiopentone sodium.

Table 3 shows that, the arterial oxygen saturation at "O" hours just after the extubation was between 98-100% in 150 patients, and from 1-15 min. SaO_2 was 92 to 84% in 53 patients out of them (53 patients) only 3 patients landed once again into hypoxemia (second episode) during next 16 to 30 min. After that not a single patient landed into postoperative hypoxemia during 31 to 60 min.

Table 4 Show that, 58 patients underwent for abdominal of which 29(50%) suffered postoperative hypoxemia, 47 patients underwent for lower abdominal operations of which 16 (34.04%) became hypoxemic and only 8 (17.78%) patients out of 45 peripheral opearions.

We observed that the incidence of hypoxemia was more in upper and lower abdominal operations as compared to operations on other sites (peripheral) and it was statistically significant ($P<0.05$) by applying chi-square test.

Table 2: Showing induction characteristics and incidence of hypoxemia

Induction Agent	Number of Patients	Incidence of hypoxemia	
		Number of Patients	(%) Percentage
Thiopentone Sodium	127	43	33.86
Ketomine hydrochloride	23	10	43.48
Total	150	53	-

Table 3: Showing early postoperative arterial oxygen saturation characteristics

Timings (Minutes)	Number of patients who suffered hypoxemia (SaO_2 = 92-84%)
01-15	53
16-30	03
31-45	Nil
46-40	Nil

Table 4: Showing Correlation in between incidence of Hypoxemia and site of operations

Groups	Type of operations	Number of patients	Incidence of Hypoxemia	
			Number of Patients	(%) Percentage
I	Upper abdominal	58	29	50.00
II	Lower abdominal	47	16	34.04
III	Peripheral	45	08	17.78
Total		150	53	-

Table 5: Showing the degree of hypoxemia

Hypoxemia	Incidence of hypoxemia	
	Number of Patients	Percentage (%)
Mild (SaO_2 = 92-88%)	37	24.67
Moderate (SaO_2 = 88-84%)	16	10.67
Severe (SaO_2 < 84%)	Nil	Nil

Table 5 shows that, out of 150 patients hypoxemia found in 53 patients out of in which mild hypoxemia was in 37 patients and moderate hypoxemia in 16 patients and not a single patient became severaly hypoxemic as we have not allowed arterial oxygen saturation to go below 84% (by giving 100% supplemental oxygen through face mask 4 lit/min)

Table 6 Shows that, in the present study out of 150 patients the incidence of hypoxemia was found in 53 patients upto 15 min. after endotracheal extubation and only 3 patients became once again hypoxemic in next 15 min. (second episode in between 16-30 min) and not a single patient was found hypoxemic in between 30-60 min. We observed that the incidence of hypoxemia was more in the early postoperative study.

Table 6: Showing Correlation in between number of patients and onset of hypoxemia

Time (Min)	Incidence of Hypoxemia	
	Number of Patients	Percentage (%)
01-15	53	35.33
16-30	03	2.00
31-45	00	00
46-60	00	00

Table 7: Showing Correlation in between early postoperative hypoxemia and the anaesthetic agent

Anaesthetic Agent	Number of Patients	Incidence of hypoxemia	
		Number of Patients	Percentage (%)
Without halothane	110	29	26.36
With halothane	40	24	60.00
Total	150	53	-

Table 8: Showing incidence of hypoxemia in relation to duration of anaesthesia

Duration of Anesthesia (Min.)	Number of Patients	Incidence of hypoxemia	
		Number of Patients	Percentage (%)
00-60	46	11	23.91
61-120	80	31	38.75
121-180	21	09	42.85
181-240	03	02	66.67
Total	150	53	-

Table 7 shows that, we had given 0.5-1% halothane intermittently for maintenance of anaesthesia in 40 patients during operations. The incidence of postoperative hypoxemia was observed in 24 (60%) patients. The incidence of postoperative hypoxemia was in 29 (26.36%) patients out of 110 patients in which halothane was not given, concluding that incidence of hypoxemia is significantly more in halothane group.

Table 7 Shows that, maximum operative procedures took 61-120 min. and there were only 3 operative procedures who required 181-240 min. We concluded that incidence of hypoxemia is directly proportional to duration of anaesthesia.

Discussion

The postoperative hypoxemia occurs commonly and is well documented even after minor surgical procedures under general anaesthesia. The postoperative period is generally accepted to extend over several days can therefore be considered under two phases early following termination of anaesthesia until few hours in recovery and late upto about one week after surgery. Many factors contribute to postoperative hypoxemia but this study was mainly aimed to have or to establish full utility of pulse oximeter by monitoring arterial oxygen saturation perioperatively in 150 patients randomly choosen of ASA - grade - I and grade - II only, inturn to get the incidence and severity of hypoxemia as well as to know the role of oxygen therapy in prevention and correction of hypoxemia.

Postoperative hypoxemia was defined in our study as a state of reduced oxygenation, when the arterial oxygen saturation was at or below 92%. Delivery of oxygen to the tissues depends on the amount of oxygen in arterial blood and cardiac output. Tissue oxygenation depends on three major variable factors, haemoglobin concentration, arterial oxygen saturation and cardiac output. Arterial oxygen saturation determined by the oxygen tension, the level of alveolar ventilation and the distribution of ventilation and perfusion in the lungs.

Hypoxemia in the postoperative period, especially the early phase, can be difficult to diagnose or assess clinically. Cyanosis is hard to detect and impossible to quantify. Tachycardia is not a reliable or specific indicator of hypoxia. The rate and depth of breathing are also not totally helpful. Measurement of arterial oxygenation can reliably detect and assess any hypoxic state. Continuous monitoring of arterial oxygen saturation by a pulse oximeter is particularly useful and practical, especially in the early postoperatie period. Arterial blood gas (ABG) estimations are also useful and may be more appropriate in the late phase. An ABG should always be undertaken when the clinical picture and pulse oximeter indicates significant and persistent hypoxemia. The use of pulse oximeter has been extensively reviewed elsewhere.

The traditional view on this subject was summarized by Craig who considered postoperative hypoxemia to occur in two phases: an early phase which may be due to anaesthetic drugs and techniques and a late phase due to administration of opioids or other drugs as well as onset of various changes in the lungs. He suggested that low arterial oxygen tension is found immediately after anaesthesia and may last for one to two hours. This early hypoxemia may be related to the anaesthetic itself, as it is an extension of the gas exchange that occurs during anaesthesia. Diffusion hypoxia during nitrous oxide elimination is an important contributory factor.

There are so many factors for producing early postoperative hypoxemia like inadequate replacement of blood loss, myocardial depression, wound pain, supine position, restrictive bandages, impaired gas exchange due to decreased functional residual capacity (FRC); V/Q mismatch, diffusion hypoxia and residual muscle paralysis.

In our study we observed that the incidence of postoperative hypoxemia was more in extremes of age and more pronounced in paediatric patients which is very well supported by Etsuro K. Motoyama and Christophe H. Glazener in 1986 concluding that in children 21% of oxygen (i. e. room air) is a potentially hypoxic mixture during the early postoperative period and also indicates that the oxygen mask should be used routinely in the early postoperative period, at least until a child is awake enough to reject it; when the need for supplemental oxygen diminish.

We observed that incidence of hypoxemia was more in patients induced with i.v. ketamine hydrochloride in comparision with Thiopentone sodium because of its action like increase in salivation may be the cause for impaired gas exchange.

The arterial oxygen saturation at "0" hours (immediately after extubation) was between 98-100% in all 150 patients. Out of 150 patients 53 patients shown mild to moderate hypoxemia that to in very early postoperative period [i.e. between 1-15 min.] our results support the view of so many authors of the past by Jakob. Trier Modler, Minna Waittrup in 1990, B. H. Meikle John, G. Smith in 1987, IL Tyler Boonrk Tantisira, peter in the year 1985, Bideshwar K. Kataria, Harnik in the year 1986.

But only three patients had second episode of hypoxemia during 16-30 min. of early postoperative period who respond reinstitution of oxygen therapy at the rate of 4 lit/min for 15 min. more, concluding that oxygen therapy is to be given routinely to each and every patient who received general anaesthesia, out of 150 patients 58 patients underwent upper abdominal surgery, 47 lower abdominal surgery and 55 peripheral or non cavity surgery in which 26, 16 and 8 patients suffered from mild to moderate hypoxemia respectively. Concluding that hypoxemia incidence was highest in upper abdominal and lowest in peripheral operations which is very well supported by B. B. Drummond and D. J. Wrigh in the year 1977, B. H. Meikljohn; G. Smith in the year 1987 and Julie Knudsen in 1970; stating that incidence and severity of postoperative hypoxemia is more with abdominal operations in early as well as late phases. M. K. Reeder; Goldmen in the year 1992; stating that hypoxemia that too severe degree is common after upper abdominal surgery and may be continued into late postoperative period and also has shown that oxygen supplementation was almost 100% effective in keeping oxygen saturation more than 90% during the early postoperative period.

Findings in our study conclude that routinely each and every patient irrespective of age, sex, type of operation, duration of operation and type of anaesthetic agent recovering from general anaesthesia should receive minimum of 4 lit. of oxygen at least in early postoperative period of one hour which is very well supported by J. Rosenberg; bedersen and Kehlete in the year 1992 who stated that oxygen therapy is of major importance for a patient care in both the early as well as postoperative period.

M. P. Reeder, Goldmen 1992, concluded that oxygen supplementation should be considered beyond the usual clinical utility of one to two hours. Julie Stank in 1996 stating that supplemental oxygen in maintaining adequate oxygen saturation is more important during first 31 to 60 min. whereas Robert J; Shirley in 1994 with our findings and concludes stating that in their study supplemental oxygen was unnecessary in 63% of patients whereas R. Scott Marray; Danial B. in the year 1988 concluded that hypoxemia was neither predictable nor clinically apparent and recommended that; unless arterial oxygenation is monitored, ambulatory patient should routinely receive supplement oxygen during recovery from general anaesthesia. Also very well supported by Ramesh Petel; Janet Norden in the year 1988 concluded that infants and children are at a greater risk of developing postoperative hypoxemia and recommended infants and children recovering from general anaethesia should receive supplementary oxygen.

The present study also concluded that incidence of hypoxemia is significantly more in the patients

recovering from general anaesthesia of halothane, which is supported by J. F. Nunn in 1964, stating that arterial oxygen tension and arterial oxygen saturation during anaesthesia are always lower than conscious subject a comparable minute volume.

In the present study even though it was apparent that incidence of hypoxemia found is directly proportional to duration of anaethesia but no study categorically said that duration is a main factor for producing more frequent cause of postoperative hypoxemia but whatever has been observed in our study might be interplay of so many other factors like site of operation, age of the patient and halothane anaesthesia. It must be stressed that although cyanosis may be present in severe hypoxia its absence does not guarantee normal or even near normal levels of oxygenation.

Summary and Conclusion

Postoperative hypoxemia not only occurs commonly it is also very difficult to assess clinically. Hypoxemia in the early postoperative phase is the result of residual anaesthesia, analgesia and their effects. Continuous monitoring of arterial oxygen saturation is an mandatory one by simple non-invasive pulse. Oximeter even in remote area. Not only it helps to detect hypoxemia but also helps in preventing the patient to enter into severe hypoxemia and its consequence by altering at earlist as due to its continuos monitoring and display. To sum up as we do not know which patient will become hypoxic, the level at which hypoxemia damages that brain or other tissue but one thing is sure that postoperative hypoxemia is more common than assumed, requires routine use of supplemental oxygen combine with routine clinical surveillance is not sufficient to prevent severity of hypoxemia and its consequences. So routine use of simple compact, portable device (pulse oximetery) is to be made mandatory.

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