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A CLINICAL STUDY OF SIGNIFICANCE OF DIFFUSION HYPOXIA AFTER GENERAL ANAESTHESIA WITH NITROUS OXIDE AND OXYGEN

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ABSTRACT

Introduction: At the end of general anaesthesia rapid excretion of nitrous oxide into the alveoli dilutes the oxygen concentration in the alveoli . Breathing that hypoxic mixture will result in diffusion hypoxia. Administration of 100% oxygen after extubation will reverse this hypoxia . We carried out a clinical study to test the above hypothesis

Material and Methods : We have taken 44 young healthy patients and allocated them into two groups . Group 1 patients were given 100% oxygen for 5 minutes. Patients in group ll were allowed to breathe room air after extubation . Oxygen saturation of all patients was assessed with pulse oximetry every minute for fifteen minutes after extubation. Pulse rate and respiratory rate was also observed at the same intervals.

Results : We observed that in patients who breathed room air after extubation , there was a 2 to 3% fall of oxygen saturation compared with pre op saturation . None of the patients had clinically significant hypoxia .There was no fall of oxygen saturation in patients who were given 100% oxygen. When the fall of oxygen saturation in patients who breathed room air was compared with the oxygen saturation of patients who received 100% oxygen the fall of saturation was statistically significant . None of the patients had alterations in pulse or respiratory rate in relation to oxygen saturation.

Conclusions : We conclude that clinically significant hypoxia does not occur in young healthy patients after nitrous oxide anaesthesia. Five minute oxygen administration after extubation may not be required in young healthy patients

KEYWORDS : Nitrous oxide, Diffusion Hypoxia, 100% oxygen.

INTRODUCTION

Nitrous oxide is a useful gas in anaesthesia. It provides good analgesia ,is a carrier gas for anaesthetic gasses , and potentiates effect of other inhalational agents . Hence consumption of inhalational agents will be reduced thereby causing economy of inhalational agents. Nevertheless nitrous oxide has some disadvantages . It causes nausea vomiting , rises intracranial pressure, causes distension of intestinal loops , and most importantly it is supposed to produce diffusion hypoxia . Despite of these side effects nitrous oxide is most widely used gas in anaesthesia . The last side effect mentioned above is the point of discussion of our study. It is said that after general anaesthesia with nitrous oxide, patient breathing room air may develop diffusion hypoxia . This has been described by B.R . Fink l in 1995 . He postulated that after general anaesthesia excess nitrous oxide which is 30 times more soluble than nitrogen, diffuses into the alveoli and dilutes the oxygen concentration in the alveolus . When the patient breathing room air breathes this hypoxic mixture, develops diffusion hypoxia . This is also known as Fink effect ,and to counter this it is advised that 100% oxygen be given for five minutes after extubation . Fink reported a 8% fall in oxygen saturation in gynaecologic patients given general anaesthesia with thiopentone and nitrous oxide, oxygen mixture . Fink termed it as diffusion anoxia. But in 1961 this was opposed by Rackow et al 2, who reported only 2% fall in oxygen saturation and emphasized more on disparity of solubility of nitrous oxide and nitrogen and dilutional hypocapnoea and consequent hypoventilation contributed to diffusion hypoxia . They felt that fall in oxygen saturation is too insignificant to call it diffusion anoxia and they recommended that the term diffusion hypoxia be used in place of diffusion anoxia. Fanning and Colgan 3 experimented on dogs and then on human patients and reported a similar significant fall in oxygen saturation after general anaesthesia involving nitrous oxide. Fanning and colgan administered 75% nitrous oxide and 25% oxygen with thiopentone . It is opined by some that the fall in oxygen saturation in the above studies may be because of respiratory depression caused by thiopentone . Many other clinical studies have questioned the clinical significance of this diffusion hypoxia .In 1969 Frumin and Eldiest4 gave nitrous oxide and oxygen anesthesia in 18 surgical patients and mentioned in their study that fall in oxygen saturation that occurs during the phenomenon of diffusion hypoxia is not significant clinically. They opined that if there is no respiratory depression and airway is patent, diffusion hypoxia is not clinically significant . However the results of the study of Frumin were questioned by Thornbein5 Other studies also mentioned that diffusion hypoxia was clinically insignificant when ventilation was adequate and airway was patent. In many of the earlier studies arterial oxygen tensions were measured . Recent studies assessed the oxygen saturation by pulse oximetry. We also used pulse oximetry to assess oxygen saturation . Many studies were done to assess significance of diffusion hypoxia in patients undergoing dental procedures under nitrous oxide sedation . They all concluded that significant hypoxia does not occur after nitrous oxide sedation. To assess the significance of diffusion hypoxia we studied two groups of patients . In one group we allowed the patient to breathe room air immediately after extubation and the other group of patients were given 100% oxygen immediately after extubation in patients undergoing general anaesthesia with nitrous oxide , oxygen and halothane. We assessed the oxygen saturation utilising a pulse oximeter.

MATERIALS AND METHODS

After obtaining approval from our local ethics committee of our hospital , we allocated 44 patients randomly into two groups by flip of a coin ,into goup l and group ll . Patients in group l were given 100% oxygen after extubation and group ll patients were allowed to breathe room air after extubation. All patients were in the age group from 10 to 40 years , healthy and were ASA grade l or grade ll. All underwent short general surgery and ENT procedures of less than one hour . Patients less than 10 years of age or more than 50 years were excluded from the study. Patients of more than ASA grade ll and patients with cardiac or respiratory disease were excluded . A routine preanaesthetic check up was done and nil by mouth advice for six hours was given. After securing intravenous access , all patients were given glycopyrolate 5

micrograms per Kg body weight and ondansetron 80 micrograms per Kg body weight intravenously. Inj fentanyl one microgram per Kg body weight was given IV . All patients were preoxygenated with 100% oxygen for three minutes . Patients were induced with thiopentone 5 mg per Kg body weight and were given vecuronium 80 micrograms per Kg body weight . They were ventilated by mask with oxygen , nitrous oxide and sevoflurane for 3 to 4 minutes and were intubated with appropriate sized endotracheal tube. They were connected to Boyle's machine via Bain's circuit. Patients were ventilated with a mixture of 33% oxygen, 66% nitrous oxide and 0.5% sevoflurane . All patients were monitored by pulse oximetry and non-invasive blood pressure monitor. Clinical monitoring was also done by observing pulse ,respiration and colour of the skin. After the end of the surgical procedure ,once the patient regained respiratory efforts nitrous oxide and sevoflurane were cut off and ventilation with oxygen was continued . The duration of oxygen administration was noted down. Almost in all cases total duration of oxygen administration after cessation of nitrous oxide was about 5 minutes. At the appropriate time estuation was carried out. Oxygen saturation at the time of extubation was noted down. Patients in group l were given 100% oxygen for 5 minutes and patients in group all were allowed to breathe room air . All the patients were monitored for 15 minutes in operation theatre. Oxygen saturation was measured by pulse oximetry every minute for 15 minutes. Oxygen administration was carried out in any patient who had a saturation less than 90%. Pulse rate and respiratory rate were also measured every minute for 15 minutes. In patients of both groups any incident of significant hypoxia was noted down. After 15 minutes monitoring, patients were shifted to post-operative ward.

RESULTS

There was no difference of demographic data in each group . The duration of surgical procedure in all patients was about same in both groups, about 30 to 45 minutes .Hence total period of nitrous oxide administration in all the patients in both groups is the same . There was no difference in the preop oxygen saturation and oxygen saturation at the time of extubation in all patients in both groups .That is it was the same in both groups. In group l patients oxygen saturation remained same like that of at extubation for 15 minutes after extubation with minor fluctuation . On the other hand oxygen saturation in group ll patients there was fall in oxygen saturation of about 2 to 3 % , three to five minutes after extubation . After 5 minutes the oxygen saturation in group l and ll was about the same with little difference. But still this was statistically significant (P< 0.05)The fall in oxygen saturation immediately after extubation was brief and transient for few seconds and occurred for 3 to 5 minutes after extubation .. This fall in oxygen saturation was statistically significant when compared with the values of oxygen saturation in group l patients . Interestingly difference in oxygen saturation in both groups is statistically significant throughout the 15 minutes of monitoring. But the fall in oxygen saturation in group ll patients did not cause clinically significant hypoxia .All patients tolerated this fall in oxygen saturation without any clinical effects. All the patients had tachycardia immediately after extubation , but the heart rate settled down during the next 15 minutes. Understandably initial tachycardia was extubation response which settled down after few minutes. There was no significant fluctuation of heart rate in relation to the fall in oxygen saturation . There was no tachypnoea in any patient. None of the patients had any side effects.

A blinded Statistical analysis was done by 3rd party statistician using epi info version 6.04. Fall in oxygen saturation in group ll at 3,4and 5 minutes was statistically highly significant(P<o.o5). Also from 6th minute to 14th minute it is still significant (P<0.05). But clinically significant hypoxia did not occur at any time.

DISCUSSION

At the end of general anaesthesia with nitrous oxide, Diffusion hypoxia occurs because of flooding of the alveoli with nitrous oxide because of excessive solubility of nitrous oxide over nitrogen. This results in dilution of oxygen in the breathing mixture in the alveolus. When patient is allowed to breathe room air he will be breathing this hypoxic mixture and hence develops hypoxia. Other causes are dilution of carbon dioxide resulting in hypocapnoea and respiratory depression . Residual anaesthetic effects may also cause ventilator depression and hypoxia . Respiratory obstruction due to any cause can also cause oxygen desaturation .This phenomenon of diffusion hypoxia was first described by B. Raymond Fink in 1955. B.R. Fink reported an average of 6 to 8 % fall in oxygen saturation in gynaecological patients given general anaesthesia with thiopentone and nitrous oxide and oxygen mixture . Fanning and Cogan had the same results when animals and later human patients were anaesthetised with thiopentone and nitrous oxide and oxygen mixture(75%+25%). They reported diffusion hypoxia of same levels as reported by Fink in their patients . Hornbein et al at the same time opined that diffusion plays a part in fall in saturation but if respirations are adequate and there is no respiratory obstruction diffusion hypoxia is of little clinical significance . Many other studies questioned the significance of diffusion hypoxia. Many opined that the desaturation that occurred during so called diffusion hypoxia is clinically not significant . In 1969 Frumin and Edelist studied 18 patients undergoing different surgical procedures under general anaesthesia ,they concluded that nitrous oxide diffusion caused clinically insignificant hypoxia in healthy patients who were allowed to breathe room air after general anaesthesia with nitrous oxide. Stubbing and Sweeney6 questioned whether diffusion hypoxia really exists. J.B.Brodsky et al7 studied 60 healthy surgical patients and observed around 4% fall in oxygen saturation after cessation of nitrous oxide and oxygen. They observed clinically significant hypoxia in 3 patients ,but attributed this to respiratory obstruction. They opined that diffusion hypoxia is of little clinical significance in many patients. Many studies were done on diffusion hypoxia in dental patients who were given nitrous oxide sedation . Tamara dunn-Russel et al 8 studied 24 children who were given nitrous oxide sedation and assessed oxygen saturation after cessation of nitrous oxide sedation . They found that there was statistically significant drop of 1 % in oxygen saturation but it was not clinically significant. Fred C Quanstron et al 9 studied 100 dental patients who were given nitrous oxide sedation for oxygen desaturation after cessation of nitrous oxide . They found a mean 2% fall in oxygen saturation which was not clinically significant . None of their patients had oxygen saturation below 92%. Arthur H Jesse et al 10 evaluated effect of breathing room air instead of 100% oxygen in healthy human volunteers following nitrous oxide sedation . They did not observe significant hypoxia in their study. In none of these volunteers oxygen saturation was below 95% after cessation of nitrous oxide sedation . Milles M, Kohne G et al 11 and Takarada et al 12 concluded the same The results of our study correlate with the above studies .It is said that maximum nitrous oxide excretion occurs 3 to 5 minutes after cessation of nitrous oxide8 . That is why diffusion hypoxia is most likely to occur during the same period. Accordingly 5 minute monitoring for diffusion hypoxia would suffice. But we monitored for 15 minutes and understandably observed fall in oxygen saturation only during first five minutes. We recorded an average 3% fall of oxygen saturation in patients breathing room air . This was statistically significant when compared with those who were given 100% oxygen , but clinically significant hypoxia was not observed except in one case where 89% oxygen saturation for a transient period was observed .This could be because of transient respiratory obstruction .However , Clinically hypoxia is classified as mild (85 to 90%) moderate (81 to 85%) and Severe (<81%)13. Accordingly saturation of 89% for a brief period is not significant. From our study we feel that diffusion hypoxia is clinically insignificant and of little consequence to most of patients. And administering 100% oxygen to healthy patients after extubation may not be recommended .This will save some 30 litres of oxygen (6 litres/minute x 5) and also decrease theatre occupation time 10 . This may facilitate increase in number of cases done in the theatre.

CONCLUSIONS

We conclude that diffusion hypoxia is minimal and inconsequential in healthy patients after nitrous oxide oxygen ,halothane anaesthesia with thiopentone and fentanyl . . These patients can easily tolerate this transient fall In oxygen saturation without any clinical effects . Oxygen administration after extubation may not be recommended . Patient may simply be monitored in the post operative ward for desaturation and for oxygenation if required.

Table 1: Comparison of SpO2 among the two groups at various intervals of extubation

Time (minutes)	Group I	Group II	T value	P value	Interpretation
1	98.83+1.23	98.87+1.19	0.1047	0.9170	Not significant
2	98.75+1.64	97.91+1.41	1.9027	0.0634	Not significant
3	98.70+1.36	96.58+2.35	3.8251	0.0004	significant
4	98.62+1.46	96.91+2.30	3.0751	0.0035	significant
5	98.66+1.37	96.41+2.66	3.6840	0.0006	significant
6	98.66+1.12	97.16+2.89	2.3709	0.0220	Significant
7	98.41+1.52	97.20+2.48	2.0379	0.0473	Significant
8	98.62+1.31	97.08+2.44	2.7242	0.0091	Significant
9	98.66+1.04	97.25+2.28	2.7564	0.0083	significant
10	98.79+1.14	97.45+2.36	2.5047	0.0159	Significant
11	98.87+1.19	97.25+2.40	2.9626	0.0048	significant
12	99.08+1.17	97.29+2.19	3.5318	0.0010	significant
13	98.95+1.19	97.5+1.81	3.2793	0.0020	significant
14	98.91+1.17	97.79+1.67	2.6909	0.0099	significant
15	98.79+1.93	97.91+1.41	1.8037	0.0778	Not significant

Table 1 shows comparison of SpO_2 among the two groups at various intervals of extubation. The mean SpO_2 was significantly higher in Group I as compared to Group II patients from third minute of extubation to the 14th minute of extubation.

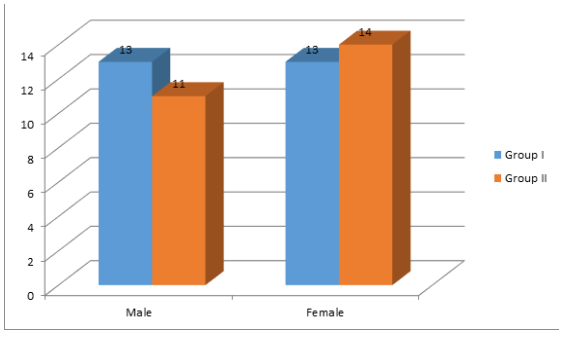

Figure 1: Sex wise distribution of study subjects in two groups

Fig 1 shows sex wise distribution of study subjects in two groups. both the groups were found to be similar in sex distribution. Both the groups had males and females number which was not significantly different.

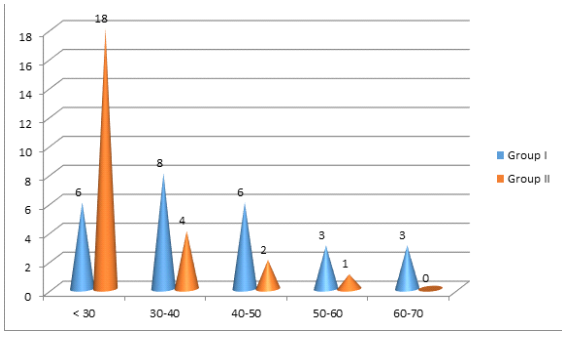

Figure 2: Total Age wise distribution of study subjects in two groups

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