IS SEDATION WITHOUT DESATURATION POSSIBLE?

D H B Tay, S Omar, T C Low

ABSTRACT

The correlation between the depth of sedation and the degree of oxygen desaturation was studied in 52 patients presenting for surgery under regional anaesthesia. After establishing successful regional anaesthesia, patients were sedated with incremental doses of intravenous midazolam. The various levels of sedation were scored from 0 to IV. Continuous pulse oximetry allowed for correlation of sedation and saturation. Each patient's presedation oxygen saturation served as the control value. This was compared with the saturation at sedation levels II, III and IV and was found to be significantly higher ($p<0.001$). There was also a significant drop in saturation as patients progressed from sedation level II to III ($p<0.02$) and from level III to IV ($p<0.001$). The incidence of saturation falling to less than 90% was 4.35%, 14.71% and 40% at sedation scores of II, III and IV respectively.

Keywords : Sedation, desaturation, midazolam, pulse oximetry, regional anaesthesia

SINGAPORE MED J 1991; Vol 32: 329-331

INTRODUCTION

Sedation is often employed as an adjunct to regional anaesthesia or to alleviate anxiety during unpleasant procedures. Recent reports have emphasized that the use of sedation is not without risks. There is however little in the literature correlating the depth of sedation and the degree of oxygen desaturation. Consequently there is little data as to the depth of sedation which may be considered "safe". With this in mind, this study sought to establish the relationship between the depth of sedation and the degree of desaturation and therefore to establish if a "safe level of sedation" existed.

METHOD

Institutional approval and informed consent from patients were obtained. Only ASA I and II patients were included in this study. After an intravenous line was inserted, regional anaesthesia was administered and tested. Patients were then brought into the operating room and continuous electrocardiograph monitoring, non-invasive blood pressure monitoring and pulse oximetry were commenced.

Oxygen saturation was monitored with the Ohmeda-3700 or the Nellcor N-200. Finger probes were used exclusively and were placed on the left index finger. When surgery involved the left arm, the right index finger was used. Saturation from the pulse oximeters were considered indicative of oxygen saturation only when the digital display was constant and accompanied by satisfactory perfusion signals as indicated by the plethysmographic waveform or the light emitting diode indicators. When these conditions were not met, the values were ignored (The use of different pulse oximeters was not a confounding factor as each patient served as his own control, and it was the trend value ie the intra-subject variability that was being studied). The lowest saturation for each sedation level was noted. Pre-medication and medical conditions that were likely to contribute to hypoxemia were also noted.

The patients were then allocated scores according to the depth of sedation as follows:

0 Awake and anxious.
I Awake but calm.
II Eyes closed. Responds to name.
III Asleep but awakes when tapped lightly.
IV Requires vigorous stimulation to be awakened.

The plan was to sedate each patient with small doses of intravenous midazolam at a rate which would allow the depth of sedation to be correlated to the saturation readings on the pulse oximeters. Patients therefore served as their own controls as they progressed from one sedation score to the next.

Pre-sedation baseline readings were first recorded. Sedation was then commenced using initial doses of midazolam ranging from 1 to 3 mg depending on the size of the patient, whether premedication had been administered and the initial anxiety state of the patient. This was usually followed by repeated boluses of 1 mg until the patient scored III on our sedation scale. We did not deliberately sedate any patient to a score of IV.

All patients were breathing room air at the onset. When pulse oximeter readings fell below 90% for a minute, patients were aroused and instructed to breathe deeply. If there was no improvement, or if the saturation fell to below 90% for a second time, oxygen supplement was administered.

RESULTS

Demographic data on the patients studied are presented in Table I. It must be emphasized that patients were predominantly young males. Extrapolation to other population groups may not be valid.

Table I
Demographic data

Age	(yrs)	Mean SD	= =	32.29 14.13
Sex	(nos)	Males Females	= =	43 9
Weight	(kg)	Mean SD	= =	63.50 14.79
Height	(cm)	Mean SD	= =	168.37 24.62
ASA	(nos)	I II	= =	46 6
Total	(nos)			52

Department of Anaesthesia
Alexandra Hospital
Alexandra Road
Singapore 0511

D H B Tay, MBBS, M Med(Anaes)
Senior Registrar

S Omar, MBBS
Medical Officer

T C Low, MBBS, M Med(Anaes)
Consultant and Head

Correspondence to : Dr D H B Tay
Department of Anaesthesia
Kandang Kerbau Hospital
Hampshire Road
Singapore 0821

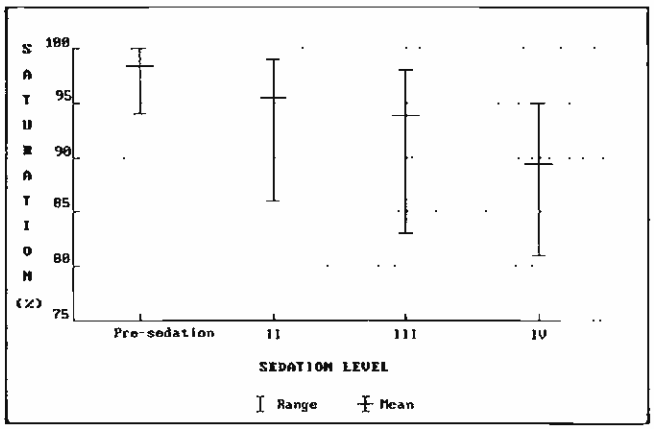
Table II enumerates the anaesthetic technique employed. Twenty patients presented for general surgery and 32 for orthopaedic surgery.

Table II
Anaesthetic technique employed

Anaesthesia	No.
Brachial block	5
Spinal anaesthesia	32
Caudal anaesthesia	11
Epidural	2
Nerve blocks	2

Fig 1 shows the relationship between sedation and saturation. The saturation at each sedation score was compared with the pre-sedation saturation level when patients scored 0 or I on the sedation scale.

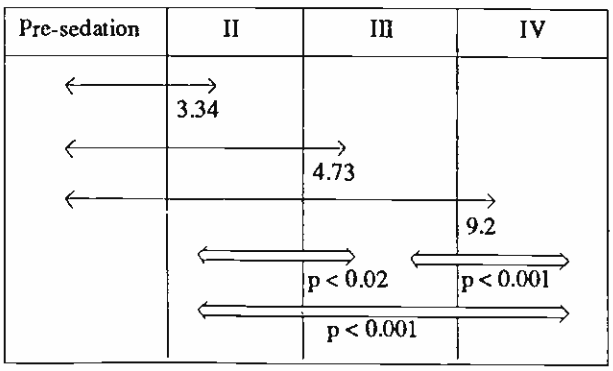
Fig 1 – Sedation and oxygen saturation



P < 0.001 for saturation values at sedation levels II, III and IV as compared to the pre-sedation level.

Not only was there a significant difference in oxygen saturation relative to pre-sedation levels but also relative to each level of sedation. Table III demonstrates the correlation between progressive sedation and desaturation. To correct for differences in the initial pre-sedation saturation levels, differences between the pre-sedation saturation and the lowest saturation at each sedation score were used in preference to the mean values.

Table III
Degree of desaturation as sedation increases



⟷ difference between pre-sedation saturation and saturation at each sedation level.

⟺ probability of the above difference (ANOVA).

Table IV shows the incidence of significant hypoxemia (saturation less than 90%), to increase as the depth of sedation increases. The number attaining each sedation level is also shown. Only 46 patients attained a sedation score of II. As sedation had to be administered slowly to allow for observation, some patients were unable to attain higher scores as surgery was completed before they could be further sedated. Sedation was discontinued in two patients who remained awake after 15 mg of midazolam was administered. Even fewer patients attained a sedation score of III (34 patients).

Table IV
Significant hypoxia and sedation

Sedation Level	Saturation < 90%	Contributing Factors
Pre-sedation	Nil	Nil
II	2/46 (4.4%)	1 near total spinal
III	5/34 (14.7%)	Nil
IV	2/5 (40.0%)	Nil

Five patients progressed to a score of IV. This was unintentional and indicated that the maximum effect had not been reached when the next increment was administered. (Increasing the dosing interval would have resulted in even fewer patients attaining scores of II and III).

Two of the 46 patients with a sedation score of II desaturated to levels below 90%. In one of them, an inadvertent near total spinal which led to hypotension and a sensory level of T1, contributed to the hypoxemia.

Table V shows the distribution of patients by the type of premedication administered. The number of patients in each group were too small to draw any conclusions about the contribution of premedication to clinically significant hypoxemia.

Table V
Premedication and clinically significant desaturation

	Nil	Midazolam (oral)	Diazepam	Pethidine
Total	20	2	25	5
Sat. < 90%	0	0	4	1

Of the 52 patients, 3 required oxygen supplementation. Two of the 3 patients (including the patient with a near total spinal) required oxygen supplementation at a sedation score of II onwards.

DISCUSSION

Is sedation without desaturation possible?

The price of sedation is desaturation. From this study it is clear that progressive depth of sedation is accompanied by progressive desaturation. These findings are consistent with that of other workers. Smith and Crul abandoned a clinical study after investigating only 10 patients[1]. This was because 3 out of 4 subjects were unable to maintain a saturation level above 90% on room air. Their patients were all arousable. Manara, Smith and Nixon studied patients under a combination of sedation and spinal anaesthesia[2]. They concluded that desaturation was significant enough to make routine oxygen supplementation advisable.

When is desaturation of clinical significance?

Not all statistical declines in oxygen saturation have clinical significance, as fluctuations in oxygen saturation may be expected during the course of the day, especially during sleep. However "normal" values from "sleep studies" are not available for comparison with sedated patients. Lack of consensus complicates the picture further. Taylor and Whitwam recommended that the alarm on pulse oximeters be set to 94%[3]. This was based on the shape of the oxygen-dissociation curve.

Smith and co-workers recommended that the lower limit of 90% would be more appropriate after a study on 150 healthy ambulant volunteers[4]. This was the value that we adopted to signify clinically significant hypoxemia. Adopting "normal" values from studies done on ambulant subjects however confounds interpretation.

Anaesthetic literature is replete with examples of such confusion. Vegfors and co-workers recommended that oxygen be administered based on their study on post-operative laparoscopy patients[5]. This resulted from the use of 94% as the criterion for hypoxemia. If 90% was used as the criterion, none of their patients would have been considered hypoxaemic. Murray, Raemer and Morris using 92% as their criterion for diagnosing hypoxemia, found 7% of their healthy, female patients presenting for ambulatory surgical procedures to require supplemental oxygen[6].

Why does desaturation occur?

There are several possible mechanisms that could lead to oxygen desaturation following benzodiazepine administration. A lowering of the oxygen saturation may be expected during sleep. In addition there is a possible effect of benzodiazepines on the respiratory centre at higher dosages. The patient's ability to maintain an unobstructed airway is also compromised. A minor contribution to the desaturation may be attributed to decreased respiratory muscle efficiency. It is interesting to note that in this study, arousal from sleep was sufficient to correct desaturation. Flumazenil was not available during the study and some patients required repeated arousal and oxygen supplementation. In contrast, Tolksdorf and co-workers found that arousal with flumazenil was not adequate to prevent clinically significant hypoxemia[7]. However, their patients had received 0.3 to 0.6 mg of fentanyl in addition to midazolam and it is not surprising that the opiate induced respiratory depression was not reversed.

How can sedation be administered safely?

For ethical reasons, there are no studies which correlate saturation and injurious effects. Until norms from "sleep studies" are available there is unlikely to be a consensus on what is clinically significant hypoxemia.

There is however uniformity of opinion with regards to how sedation can be administered safely. Despite different standards being adopted, the common conclusion is that routine oxygen supplementation is necessary[3-6] where oxygen saturation is not monitored.

In many countries, pulse oximetry is fast becoming a requirement in minimum mandatory monitoring standards. We support the view that pulse oximetry should be an integral part of mandatory monitoring for regional anaesthesia[8-10]. Only 3 out of 52 patients required oxygen supplementation in this study. It would therefore be more cost effective to routinely monitor oxygen saturation and selectively administer oxygen.

Is there a safe level of sedation?

In this study, when patients could be aroused when they heard their names called, the incidence of hypoxemia (saturation <90%) was 4.4%. When light tapping was required to arouse the patient, the incidence had increased to 14.7% and when vigorous stimulation was required, 40% were hypoxaemic. Sedation must therefore be deliberate and controlled. Small boluses of a drug titrated to patient response is preferable to sedation administered on the basis of body weight. Patients who need to be "soundly asleep" during the surgical procedure are poor candidates for regional anaesthesia.

The use of pulse oximetry together with routine oxygen administration should be encouraged when sedation is employed in the absence of an anaesthetist.

In summary, this study shows that the degree of desaturation parallels the depth of sedation. Unfortunately even at light levels of sedation, the degree of desaturation was at times significant (using 90% as the criterion). We therefore advocate the use of pulse oximetry in every sedated patient and oxygen therapy as appropriate to the circumstances.

ACKNOWLEDGEMENTS

We would like to acknowledge the assistance of Cybron Singapore in making available the Nellcor N-200 for this study. Dr KS Chia, Senior Lecturer in the Department of Community, Occupational and Family Medicine, National University of Singapore, provided his statistical wisdom and expertise on the computer.

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