OXYGEN DESATURATION FOLLOWING SEDATION FOR REGIONAL ANALGESIA

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Many patients, especially those in certain "high risk" groups, undergo operative procedures under regional analgesia in the belief that this approach is safer than general anaesthesia; this is particularly the case for patients with coexistent lung disease. During the procedure, sedation is often provided with an i.v. agent, for example midazolam, diazepam or propofol. It seems (from personal observation) that administration of oxygen to the patient in this situation is not routine in current European practice. There is only equivocal guidance on this topic from reference works on local anaesthesia [1,2] and most do not mention the use of oxygen, except to treat the complications of regional anaesthesia [3-5].

We decided to study the effects of sedation with midazolam on oxygen saturation ($S_aO_2$) as measured by pulse oximetry in patients undergoing surgery under regional conduction block.

PATIENTS AND METHODS

A study was planned of 100 patients aged 20-85 yr and in ASA categories I-III. Patients were randomly allocated to one of two groups according to the last digit of the hospital number: the first group (odd digit) received oxygen 2 litre min$^{-1}$ via a 12-French gauge nasal catheter (without foam nostril plug) during surgery, the second group (even digit) received no additional oxygen unless $S_aO_2$ decreased to less than 90%. All patients underwent surgery under regional conduction block in combination with sedation using increments of midazolam 1.0-2.5 mg i.v. as required.

SUMMARY

Moderate to severe arterial oxygen desaturation ($S_aO_2$, 75-88%) occurred in three of four patients who were given sedation during regional conduction block without the administration of oxygen. No desaturation occurred in six patients given oxygen 2 litre min$^{-1}$ via a nasal catheter during similar procedures. It is concluded that patients undergoing regional conduction block with concurrent sedation should receive additional oxygen routinely unless a pulse oximeter is available for monitoring.

The level of sedation was controlled such that the patient was drowsy but aroused easily and capable of communication. Upper airway patency was maintained at all times. $S_aO_2$ was monitored continuously during the procedure using a prototype Datex Satlite pulse oximeter. The standard adult finger probe on a randomly selected finger was applied according to the manufacturer's instructions. Monitoring was commenced after the patient had been positioned for induction of the block, and continued until the end of the surgical procedure.

Unfortunately, we felt compelled to abandon this study after only 10 patients had been investigated, as a result of profound reductions in $S_aO_2$ which occurred in some patients breathing only air. In all patients given supplementary oxygen, $S_aO_2$ remained greater than 90% throughout the procedure. A brief résumé follows of the three patients in whom desaturation occurred.

Patient 1

A 63-yr-old man, ASA category I, underwent elective total hip replacement. Clinical history and examination, laboratory investigations, chest x-ray and ECG were normal. Arterial pressure
Induction Time (h)

**FIG. 1.** Course of anaesthesia in patient 1. Incremental doses of midazolam are shown by arrows. The horizontal bars indicate administration of oxygen 2 litre min$^{-1}$; at point A the patient removed the oxygen catheter.

was 140/90 mm Hg. The patient refused pre-medication. Extradural anaesthesia was administered via the L3–4 space using 2.0% mepivacaine 20 ml with adrenaline 1:200000, producing a bilateral sensory loss to T6. No extension of the block occurred during surgery. Sedation was provided with an initial dose of midazolam 2.5 mg i.v., followed by increments of 1 mg i.v. as required (fig. 1—arrows). After the first dose the patient was sleepy but rousable; $S_{a_o}$ decreased to less than 90% and oxygen 2 litre min$^{-1}$ was administered via a nasal catheter. $S_{a_o}$ improved to an acceptable value. However, following each subsequent dose of midazolam a reduction in $S_{a_o}$ occurred, although not to values below 90%. At point A in figure 1, the patient removed the oxygen catheter, whereupon $S_{a_o}$ decreased precipitously. The oxygen catheter was replaced in his nose, resulting in an immediate improvement in $S_{a_o}$.

**Patient 2**

A 70-yr-old man, ASA category I, underwent transurethral prostatectomy. Arterial pressure was 140/85 mm Hg. Laboratory analyses, ECG and chest x-ray were within normal limits. Premedication with temazepam 20 mg by mouth was given 2 h before surgery; the patient arrived in the anaesthetic room awake and co-operative. Analgesia was achieved with hyperbaric 0.5% bupivacaine 20 mg injected intrathecally at L4–5 with the patient sitting. A bilateral sensory block to L1 was produced which remained stable during the operation. Sedation was provided with an initial dose of midazolam 2.5 mg i.v., followed by increments of 1 mg i.v. An initial decrease in systolic arterial pressure to less than 100 mm Hg accompanied induction of the block, despite a preload with Hartmann’s solution 500 ml (fig. 2). Superimposed upon this, the first dose of midazolam (arrows, figure 2) was associated with a reduction in $S_{a_o}$ to less than 90%. The patient refused additional oxygen, but both arterial pressure and $S_{a_o}$ increased to acceptable values after the administration of ephedrine 10 mg i.v. All subsequent doses of midazolam, with the exception of the third, also were associated with transient reductions in $S_{a_o}$ to less than 90%. However, only the second and third doses of midazolam were associated with further reductions in systolic arterial pressure, although on these occasions not to less than 100 mm Hg.

**Patient 3**

A 59-yr-old man, ASA category I, underwent urethroplasty. Laboratory analyses, ECG and chest x-ray were within normal limits. Premedication consisting of diazepam 10 mg and
droperidol 2.5 mg was given by mouth 2 h before the procedure; he arrived in the anaesthetic room awake and co-operative. Analgesia was produced with hyperbaric bupivacaine 20 mg injected intrathecally at the L2–3 space with the patient sitting. A bilateral sensory block to T10 was produced which remained at this level throughout the procedure. Sedation was provided with an initial dose of midazolam 2.5 mg i.v., followed by increments of 1 mg i.v. as required. Figure 3 details the course of this anaesthesia, showing reductions in $S_{aO_2}$ in response to administration of midazolam, not associated with changes in arterial pressure or heart rate.
DISCUSSION

These patients illustrate a potential danger during regional conduction blockade combined with sedation if additional oxygen is not administered to patients during surgery.

There are three factors which may interact to cause the observed reduction in $\text{Sa}_0_2$. First, compensatory reflex vasoconstriction in areas not subject to sympathetic blockade may result in a reduction in $\text{Sa}_0_2$, the degree depending upon the performance of the individual pulse oximeter in the face of reduced perfusion at the measuring site. The Satlite produces a pulse waveform derived from the light absorption curve, but no variation in the amplitude of the waveform was seen during the cases reported. Compensatory reflex vasoconstriction should persist for the duration of the block, while the phenomena reported here were transient.

A second possible factor is hypoventilation resulting from sedation. Although we did not assess formally the degree of ventilation, all patients were easily rousable and had a patent upper airway and an adequate rate of ventilation. However, hypoventilation would explain the transient reductions in $\text{Sa}_0_2$ occurring in the absence of cardiovascular changes, seen in all three patients reported here.

Third, ventilation-perfusion mismatch may occur as a result of a combination of relative hypovolaemia, depression of cardiac output and reduction in FRC consequent upon the supine position. This would result in a reduction in $\text{Sa}_0_2$, and may explain the initial decreases in both $\text{Sa}_0_2$ and arterial pressure in patient 3. This may be the same phenomenon of intrapulmonary shunting described during general anaesthesia [6, 7].

The question as to the value of $\text{Sa}_0_2$ at which intervention becomes necessary is unanswered [8–10], and has been complicated by the recent demonstration that the pulse oximeter reading is not dependent solely upon pulsation within the arteriolar side of the circulation [11]. Further investigation, combined with adequate statistical analysis [12] is required before conclusions can be drawn on accurate measurements of $\text{Sa}_0_2$ based upon data obtained from a pulse oximeter. Following a study of 150 "normal" individuals (Authors’ unpublished observations), we regard a lower limit for a pulse oximeter reading of 90% as being practicable in most circumstances.

We conclude that major regional conduction blockade, combined with sedation, should not be used without the administration of additional oxygen unless a pulse oximeter is available.

ACKNOWLEDGEMENTS

The authors wish to thank Datex Instrumentation Corporation for the loan of the Satlite oximeter.

REFERENCES