Patients undergoing surgery with regional anaesthesia are frequently sedated, as many prefer to have no recall of their stay in the operating theatre. Midazolam is used commonly for this purpose and may be preferable to diazepam, as it is associated with a greater degree of amnesia [1]. The administration of additional oxygen to these patients is practised by most anaesthetists, but reference works on local anaesthesia make only cursory mention of the use of oxygen. A reduction in arterial oxygen saturation has been reported in three patients undergoing surgical procedures with regional anaesthesia and sedated with midazolam [2]. The aim of this study was to investigate the effects of the combination of spinal anaesthesia and sedation with midazolam on oxygen saturation.

**METHODS AND RESULTS**

Thirty patients undergoing transurethral resection of the prostate (TURP) gave informed consent to participate in the study, which was approved by the Hospital Ethics Committee. FVC and FEV₁ were measured in all patients, using a Vitalograph. No premedication was prescribed. On the patient’s arrival in the anaesthetic room, baseline heart rate, arterial pressure (AP) and arterial oxygen saturation (Saₐₒ) in the ear lobe [3] were recorded using a continuous ECG display, an automatic arterial pressure recorder (Dinamap) and a pulse oximeter (Ohmeda Biox 3700), respectively. The rate of ventilation was counted over a 1-min period. Patients were given Hartmann’s solution 500 ml i.v. and spinal anaesthesia was induced with 0.5% bupivacaine 3 ml in 8% glucose injected over 30 s at the L2–3 or L3–4 space with patients in the lateral position. The upper level of the block was assessed by loss of sensation to pinprick and the patient placed in the lithotomy position. The measurements were then repeated.

Patients were allocated randomly to one of two groups: group A received oxygen 4 litre min⁻¹ from a Hudson mask; group B breathed room air. Incremental 1-mg i.v. doses of midazolam were administered at 3-min intervals until Verrill’s sign [4] was achieved. Further increments were given as required during the procedure to maintain this level of sedation. A clear, unobstructed airway was maintained at all times. Heart rate, ventilatory rate, AP and Saₐₒ were recorded at 3-min intervals from the institution of the intrathecal block until the end of the procedure. Saₐₒ was recorded only when the plethysmographic waveform on the oximeter was normal. Supplementary oxygen was administered to patients in group B if Saₐₒ was
sustained between 85 and 89% ("hypoxaemia") for 5 min or decreased to less than 85% ("severe hypoxaemia") at any time.

Student's unpaired t test was used to compare the demographic data and the $S_aO_2$ values.

The mean (SD) values of the following variables did not differ significantly between groups A and B (respectively): age 77.8 (7.9) v. 74.1 (9.4) yr; weight 73 (13.2) v. 70.3 (8.9) kg; height 173 (6.5) v. 170.9 (7.6) cm; spirometry (FEV$_1$) 1.85 (0.92) v. 1.99 (0.43) litre, total dose of midazolam 3.93 (1.2) v. 4 (1.8) mg; weight of prostatic tissue resected 24.7 (31.8) v. 29.4 (29.1) g. The median height of anaesthesia to pinprick was T8 (range T6–T11) in both groups.

The baseline rate of ventilation was similar in both groups (14.9 (2.9) v. 13.1 (1.7) b.p.m.), but increased significantly ($P < 0.01$) after the administration of midazolam to a maximum of 21.2 (4.1) and 22.7 (5.1) b.p.m. in groups A and B, respectively. Baseline $S_aO_2$ was comparable in both groups (94.2 (3.3) v. 94.8 (1.7)%) and was not reduced significantly following assumption of the lithotomy position. $S_aO_2$ in patients in group A was significantly higher than that in group B throughout the procedure ($P < 0.001$) following the administration of midazolam (fig. 1). The reduction in $S_aO_2$ from baseline in group B was significant also at each time of measurement ($P < 0.005$ at 3, 27 and 30 min; $P < 0.001$ at all other times).

Hypoxaemia developed in four patients, and severe hypoxaemia in two patients in group B. The reduction in $S_aO_2$ was not associated with hypotension, airway obstruction or apnoea, but was associated closely with the administration of midazolam and the achievement of the desired level of sedation. The administration of oxygen corrected the hypoxaemia in all patients. One patient with hypoxaemia developed a junctional rhythm and broadened QRS complexes. Sinus rhythm was restored with the administration of oxygen.

**COMMENT**

A significant reduction in $S_aO_2$ may occur when spinal anaesthesia is combined with sedation induced by midazolam. Similar desaturation has been reported during upper gastrointestinal endoscopy when midazolam was used for sedation, and was prevented by the routine administration of oxygen [5]. Although moderate transient reductions in $S_aO_2$ may be considered acceptable in young, healthy patients, they should be avoided in the elderly patient with ischaemic heart disease. This was illustrated by the association of arrhythmia with hypoxaemia in one of the patients withdrawn from group B.

No constitutional factor was significantly different in those patients developing hypoxaemia, but the number of patients studied was small. Midazolam is known to cause central respiratory depression resulting in a reduction in total lung capacity, maximal breathing capacity and tidal volume, whilst the rate of ventilation is increased; significant reductions in $S_aO_2$ have followed the administration of midazolam 0.2 mg kg$^{-1}$ to young healthy volunteers [6]. The elderly patients in this study received smaller doses but demonstrated similar findings, possibly because of the combination of a degree of deafferentation and sedation. The physiological effects of age on pulmonary function place elderly patients at greater risk of developing hypoxaemia. Increased closing volume encroaches on tidal ventilation, resulting in increased intrapulmonary shunting.
and an increased alveolar to arterial difference in oxygen tension. The assumption of the lithotomy position, with its attendant reduction in functional residual capacity, further aggravates ventilatory function.

We conclude that spinal anaesthesia combined with sedation should not be used without the administration of additional oxygen, unless a pulse oximeter is available to monitor $\text{Sa}_O_2$.

REFERENCES


