Haemodynamic stability with midazolam-sufentanil analgesia in cardiac surgical patients

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Since the administration of both diazepam and midazolam are claimed to cause adverse haemodynamic effects following fentanyl or sufentanil intravenous injection, we evaluated the effectiveness and safety of the reverse sequence, (midazolam-sufentanil) on haemodynamic variables, adequacy of analgesia, amnesia and recovery in 15 adult patients undergoing coronary artery surgery (with a mean $\pm$ SEM ejection fraction of 0.41 ± 0.03). After routine premedication, midazolam 0.14 ± 0.01 mg·kg⁻¹ IV was given over one min followed 5 min later by sufentanil in incremental IV doses of $1.5 \mu\text{g}\cdot\text{kg}^{-1}$ to a total pre-intubation dose of $4.0\text{--}5.0 \mu\text{g}\cdot\text{kg}^{-1}$ injected in 10 min. One minute after the initial dose of sufentanil, pancuronium $0.1 \text{ mg}\cdot\text{kg}^{-1}$ IV was given in 30 seconds. The incremental doses of sufentanil were based on a greater than 15 per cent increase in rate-pressure product. The mean dose of sufentanil before cardiopulmonary bypass was $9.6 \pm 2.1 \mu\text{g}\cdot\text{kg}^{-1}$ and $13.9 \pm 1.3 \mu\text{g}\cdot\text{kg}^{-1}$ for the entire procedure. A significant decrease in systolic and diastolic blood pressures occurred after midazolam administration which was sustained until sternotomy. A significant reduction in systemic vascular resistance occurred following midazolam. Sufentanil reduced the left ventricular stroke-work index. Tracheal intubation, skin incision and sternotomy elicited no adverse haemodynamic responses. Adequate analgesia, complete amnesia and early recovery of wakefulness were observed.

Key words

HYPNOTICS, BENZODIAZEPINES: midazolam; ANALGESICS, NARCOTIC: sufentanil; SURGERY: cardiac; BLOOD PRESSURE: drug effects.

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Diazepam has been used as a safe sleep-inducing agent in patients undergoing cardiac catheterization and cardioversion,^{1,2} but thrombophlebitis, its inadequate hypnotic potency and its long half-life were drawbacks not shared by its water soluble congener, midazolam.³ Studies by Fragen *et al.*⁴ in cardiac patients showed haemodynamic stability after IV midazolam. On the contrary, both diazepam and midazolam have, when combined with morphine⁵ or fentanyl,⁶⁻⁸ been claimed to cause adverse haemodynamic changes in patients undergoing cardiac surgery. Since no reports are available on similar adverse interaction of midazolam with sufentanil, the haemodynamic effects of a midazolam-sufentanil induction sequence in cardiac patients were evaluated.

Methods

Fifteen patients scheduled to undergo elective aorto-coronary artery bypass graft (CABG) surgery were studied. Patients were excluded who had a history of drug habituation or addiction, psychiatric treatment, or allergic reactions to narcotic-analgesic drugs. Patients who were undergoing emergency surgery or who were minors or prisoners, were also excluded. The patients were entered into the study at random. Written informed consent, as required by the Institutional Research Review Board, was obtained prior to the study. Fourteen patients were male and one female. Mean $\pm$ SEM age was 62.5 ± 2.1 yr (range 51 to 80), mean weight was 75.4 ± 2.8 kg, and mean height was 172 ± 1.4 cm, with a mean BSA of 1.91 ± 0.03 m². Patients were maintained on β -adrenergic blockers or calcium channel blockers until the time of anaesthetic induction. The morning digoxin dose was withheld. The mean ejection-fraction was 0.41 ± 0.03 (0.31–0.57).

The night prior to surgery, the patients received a benzodiazepine hypnotic, usually triazolam 0.25 mg PO for sleep. For preoperative preparations each patient received diazepam $0.15 \text{ mg}\cdot\text{kg}^{-1}$ PO and morphine sulfate $0.15 \text{ mg}\cdot\text{kg}^{-1}$ IM 60 minutes prior to induction. In addition, six patients also received $0.2\text{--}0.4$ mg scopolamine IM solely by chance, since one of the investigators routinely uses it in the preoperative medication.

Monitoring

In the preoperative preparation area, an intravenous line was established. An arterial cannula was inserted into a radial artery under local anaesthesia. A balloon-tipped, Swan-Ganz pulmonary artery catheter was placed into the pulmonary artery through an introducer previously inserted into the right internal jugular vein. ECG lead II and V₅ and non-invasive blood pressure (Dinamap) monitoring were continued in the preoperative preparation area until the patients were transferred to the operating room. In the OR, direct arterial systemic pressure, pulmonary artery pressure and ECG were recorded on a multichannel monitor linked to a computer terminal. Oxygen saturation was continuously measured by an oximeter to maintain > 95 per cent saturation. Exhaled CO₂ level was displayed and continually recorded by a capnograph in order to assure that a CO₂ partial pressure of 35–45 mmHg was maintained. Tidal volume and ventilatory rate were recorded by a respirometer. Blood gases were determined at each blood sampling and if needed. Cardiac output was determined by thermodilution with a cardiac output computer in triplicate.

Haemodynamic data were determined and calculated at the following times before and throughout anaesthesia and surgery.

- 1 Pre-induction: before IV midazolam administration;
- 2 Post-midazolam but pre-sufentanil: before sufentanil IV or 5 min after the start of midazolam IV injection;
- 3 Post-sufentanil but pre-intubation: 5 min after sufentanil and pancuronium IV injection;
- 4 Post-intubation: 5 min after intubation but before surgical incision;
- 5 Pre-sternotomy: 10 min after surgical incision;
- 6 Post-sternotomy: 10 min after sternotomy;
- 7 Pre-bypass: immediately before the start of cardiopulmonary bypass;
- 8 Post-bypass: after the termination of cardiopulmonary bypass;
- 9 At the end of surgical procedure.

Haemodynamic parameters evaluated were:

- Heart rate (HR)
- Systolic systemic blood pressure: (BP_s) mmHg
- Diastolic systemic blood pressure: (BP_d) mmHg
- Pulmonary artery systolic blood pressure: (PAP_s) mmHg
- Pulmonary artery diastolic blood pressure: (PAP_d) mmHg
- Pulmonary artery wedge pressure: (PAP_w) mmHg
- Central venous pressure (CVP): cmH₂O
- Cardiac output (CO): L·min⁻¹

The following haemodynamic data were calculated:

- Cardiac index (CI): L·min⁻¹·m⁻²
- Stroke volume (SV): ml

- Stroke index (SI): ml·m⁻²
- Left ventricular stroke work index (LVSWI): g·m·m⁻²
- Right ventricular stroke work index (RVSWI): g·m·m⁻²
- Systemic vascular resistance (SVR): dynes·sec·cm⁻⁵
- Pulmonary vascular resistance (PVR): dynes·sec·cm⁻⁵
- Rate pressure product (RPP): HR × BP_s

Anaesthetic management

Midazolam in a mean ± SEM dose of 0.14 ± 0.01 mg·kg⁻¹ was given IV over a one-minute interval to the patients spontaneously breathing 100 per cent O₂. Five minutes after the end of midazolam injection, incremental doses of 1.5 µg·kg⁻¹ sufentanil were given. The total of sufentanil dose was 4.0–5.0 µg·kg⁻¹ up to the time of intubation. One minute after the initial dose of sufentanil, pancuronium 0.1 mg·kg⁻¹ IV was given. After the development of complete muscle relaxation, tracheal intubation was carried out without spraying the vocal cords with a local anaesthetic. After confirming the correct position of the endotracheal tube, the administration of 100 per cent oxygen or oxygen in air was continued at an FiO₂ to maintain >95 oxygen saturation. None of the patients received N₂O. Additional incremental doses of 0.6 µg·kg⁻¹ of sufentanil were given based on a greater than 15 per cent increase in RPP. Additional doses of midazolam were injected when eye-signs and clinical evidence of sympathetic activity showed light planes of anaesthesia. Intermittent doses of pancuronium were given throughout anaesthesia when required based on the response of the thumb to ulnar nerve stimulation. No patient received an IV infusion of vasopressors and/or vasodilator drug, β-blockers or Ca-channel blockers intravenously throughout the study until cardiopulmonary bypass.

During the postoperative period, the following were recorded: times to recovery of spontaneous respiration, to wakefulness and to the first analgesic administration, adequacy of intraoperative amnesia, and postoperative analgesia. On postoperative interviews, the patients were asked to answer yes or no to the following questions: Were you pleased with anaesthesia? Did you feel any pain or discomfort during anaesthesia and surgery? Can you recall any events or conversations during surgery? Would you object to receiving this anaesthetic technique again?

Data analysis

The haemodynamic parameters were evaluated for the change from the baseline at each sampling time during the procedure, using the ANOVA-program. Statistical significance was defined at $p \leq 0.05$. All values are expressed as mean ± SEM.

TABLE Haemodynamic data

Parameters	Before induction	After midazolam	After SF	After intubation	Before sternotomy	After sternotomy
BP _s mmHg	145.1 ±7.8	124.3* ±5.8	121.1* ±4.8	125.6* ±3.9	126.2* ±4.0	127.7* ±3.6
BP _d mmHg	66.8 ±4.4	57.7* ±3.3	56.0* ±3.1	60.6 ±2.6	61.3 ±2.5	63.2 ±2.2
HR beats·min ⁻¹	69.2 ±3.9	70.9 ±2.7	69.9 ±3.2	67.6 ±2.9	67.9 ±2.8	69.9 ±3.2
PAP _s mmHg	29.6 ±3.0	27.3 ±1.7	26.3 ±1.2	25.5 ±1.1	25.6 ±1.0	25.6 ±1.3
PAP _d mmHg	13.8 ±1.5	12.8 ±1.1	12.5 ±0.8	12.9 ±0.7	13.5 ±0.8	13.9 ±0.6
CO L·min ⁻¹	4.9 ±0.3	5.0 ±0.2	4.7 ±0.3	4.6 ±0.2	4.6 ±0.2	4.5 ±0.2
CI L·min ⁻¹ ·m ⁻²	2.6 ±0.2	2.7 ±0.1	2.4 ±0.2	2.4 ±0.1	2.4 ±0.1	2.4 ±0.1
SV ml	72.4 ±4.4	72.7 ±3.5	71.3 ±3.2	67.7 ±4.3	68.9 ±3.0	66.7 ±4.0
SI ml·m ⁻²	38.3 ±2.6	38.1 ±2.3	36.0 ±2.6	36.5 ±1.9	36.4 ±1.8	35.3 ±2.6
SVR dyne·sec·cm ⁻⁵	1453.5 ±126.1	1164.7* ±75.2	1262.1 ±129.5	1303.0 ±53.2	1318.7 ±55.6	1361.6 ±72.7
PVR dyne·sec·cm ⁻⁵	168.3 ±39.8	121.5 ±16.7	138.0 ±20.5	126.5 ±15.1	133.2 ±17.6	129.0 ±14.1
LVS WI g·m ⁻²	43.6 ±3.9	36.3 ±3.1	33.0* ±2.6	36.1 ±2.5	36.3 ±2.4	35.9 ±3.0
RVS WI g·m ⁻²	9.8 ±1.3	9.2 ±0.9	8.2 ±0.6	8.3 ±0.4	8.5 ±0.4	8.5 ±0.6
RPP mmHg × beats·min ⁻¹	10070.8 ±871.8	8834.2 ±561.4	8425.8 ±445.4	8517.6 ±483.1	8600.5 ±490.8	8925.4 ±487.1

*p < 0.05. For abbreviations, see Methods.

Results

Intravenous injection of midazolam *per se* caused significant reductions in BP_s, BP_d and SVR, but no concomitant changes in other directly measured and calculated haemodynamic variables (Table and Figures 1–4).

Injection of incremental doses of sufentanil alone totaling a mean dose of $4.5 \pm 1.2 \mu\text{g}\cdot\text{kg}^{-1}$ for induction and $9.6 \pm 2.1 \mu\text{g}\cdot\text{kg}^{-1}$ before cardiopulmonary bypass caused a decrease in LVS WI after induction but before intubation (Table) but no other significant changes in haemodynamic variables.

The only change in haemodynamic variables caused by intubation was a moderate non-significant increase in BP_s. Nor were HR, SVR, PVR, BP, CO, CI, SV, SI significantly altered by intubation (Figures 1–4).

Following sternotomy, no statistically significant

changes in haemodynamic variables were observed as compared to pre-sternotomy or to the pre-induction values.

Throughout anaesthesia and surgery, no other changes in haemodynamic variables were observed until completion of study before the start of cardiopulmonary bypass (Table). The total dose of sufentanil was $13.9 \pm 1.3 \mu\text{g}\cdot\text{kg}^{-1}$ for the entire procedure. No ECG changes representing ischaemia occurred in any of the patients. No myocardial infarct or pulmonary oedema was observed during the study period before cardiopulmonary-bypass.

Recovery from anaesthesia was uneventful. The mean duration of anaesthesia was 300.0 ± 14.3 min. The mean time interval from the end of anaesthesia to recovery of wakefulness was 150.0 ± 35.4 min. The mean time interval from end of anaesthesia to spontaneous ventila-

FIGURE 1 Systolic and diastolic systemic blood pressures, heart rate, and pulmonary arterial pressures, during midazolam-sufentanil analgesia (mean $\pm$ SEM) (* $p < 0.05$; ** $p < 0.01$).

tion was 966.0 ± 46.3 min. The mean time interval from the end of anaesthesia to the first required analgesic dose was 120.0 ± 30.0 min. On postoperative interview, all patients were pleased with their anaesthesia. No patient had recall of pain or any events or conversations during anaesthesia and surgery (100 per cent amnesia) and none objected to receiving this anaesthetic technique again.

Discussion

This is the first report on the successful use of midazolam-sufentanil for both induction and maintenance of amnesia-anaesthesia in patients with coronary artery disease. Our assumption that midazolam and sufentanil induction sequence causes no adverse but rather beneficial haemodynamic changes in patients scheduled to undergo cardiac surgery is substantiated by our results. The reduction in BP_s (Figure 1) and RPP (Table) after midazolam paired with the reduction of SVR (Figure 3) was likely to result in reduction of the oxygen demand of the heart muscles.

Indeed, the reduced LVSWI (Figure 4) is further proof for this contention, although actual measurements on myocardial oxygen uptake were not carried out.

The dose of midazolam used in these cardiac patients was moderate, a sleep-inducing dose of 0.14 ± 0.01 mg·kg⁻¹. Slow careful titration of the minimum effective dose might have contributed to the haemodynamic stability reported. In contrast to the findings of several investigators,^{7,9,10} we observed no sudden decrease in BP or a compensatory tachycardia, which are reflections of the autonomic circulatory response to a sudden decrease in SVR. Some investigators blamed splanchnic pooling of blood as a cause of hypotension, especially in water depleted hypovolaemic cardiac patients.⁸ Recently this assumption received further proof in experiments in dogs.¹¹ Our results confirm the findings of several investigators^{4,12-14} who observed no significant decreases in systemic blood pressures following midazolam in cardiac patients, having received no fentanyl or fentanyl derivatives prior to midazolam.

FIGURE 2 Cardiac output and index, stroke volume and stroke volume index, during midazolam-sufentanil analgesia (mean $\pm$ SEM) (* $p < 0.05$).

The adverse haemodynamic effects of midazolam following the use of high doses of fentanyl anaesthesia for CABG operation by Heikkilä *et al.*⁸ can be explained by their induction sequence. First, they administered 15 $\mu\text{g}\cdot\text{kg}^{-1}$ of pancuronium for prevention of fentanyl-induced truncl rigidity. Then the IV infusion of 75 $\mu\text{g}\cdot\text{kg}^{-1}$ of fentanyl was completed in five minutes. This was followed by 0.15 $\text{mg}\cdot\text{kg}^{-1}$ midazolam, the drug studied. Evidently, midazolam was injected at a point when fentanyl still had been exerting its haemodynamic effects. Consequently, the experimental set-up did not allow the evaluation of the effect of midazolam alone on haemodynamics as even Heikkilä *et al.*⁸ admitted. Therefore, no conclusion as to the haemodynamic stability following IV midazolam in cardiac patients can be drawn from their studies. Moreover, the lack of reduction of CI or SI found (Figure 2) and also reported by Heikkilä *et al.*⁸ is further proof of the lack of myocardial depressant effect of midazolam even after high doses of fentanyl.

The doses of sufentanil used in our study could be considered as low or moderate doses for both induction and maintenance of analgesia for cardiac surgery. The

administration of sufentanil alone following midazolam injection was not associated with any significant haemodynamic changes except for a decrease in LVSWI (Figures 1–4). The slow incremental dosing might have contributed to the favourable results, although the contribution of midazolam to the haemodynamic stability cannot be underemphasized. The stabilizing effect of benzodiazepines on the limbic autonomic centres, thereby reduction of narcotic requirements in neuroleptanaesthesia, is well recognized.¹⁵

Stressful events such as tracheal intubation, skin incision and sternotomy caused no adverse haemodynamic changes in our patients (Figures 1–4). RPP (Table), LVSWI, RVSWI (Figure 4) and acid-base balance remained unchanged following tracheal intubation, skin incision and sternotomy (Figures 1–2). These events may elicit hypertension, tachycardia and increased catecholamine levels that in cardiac patients, in turn, may precipitate myocardial infarction and fatal cardiac arrhythmias. Although in our cardiac patients there was a blockade of autonomic responses to stressors, the safety of our technique ought to be confirmed in a larger series of

FIGURE 3 Systemic and pulmonary vascular resistance, during midazolam-sufentanil analgesia (mean $\pm$ SEM) (* $p < 0.05$).

patients induced and maintained with midazolam and sufentanil.

The safety of a midazolam-sufentanil anaesthetic induction sequence for induction followed by maintenance of analgesia with sufentanil- O_2 is also confirmed by the lack of adverse occurrences intraoperatively in these patients, such as hypo- or hypertension, brady- or tachycardia, arrhythmias, ST-segment changes, pulmonary oedema, and myocardial infarction. Therefore, no vasoactive drugs, inotropic agents or β -blockers were needed throughout the course of anaesthesia from induction to cardiopulmonary bypass except for the routine nitroglycerin IV infusion. No recall of pain or any unpleasant experiences were reported in our patients on postoperative interviews. Postoperative analgesia lasting for two hours was another major advantage of our technique. Postoperative return of wakefulness was prompt. The return of spontaneous ventilation was prolonged. This should be taken into consideration in the selection of this anaesthetic technique in patients who are not to be kept on the ventilator for several hours postoperatively, as is routinely done with cardiac patients.

In conclusion, midazolam-sufentanil-pancuronium- O_2 induction/intubation sequence caused no adverse haemodynamic changes in cardiac patients in the doses and sequence utilized in our study. Tracheal intubation, skin incision and sternotomy, the most stressful events, elicited no adverse haemodynamic effects. Adequate pain relief, lack of recall of pain or other intraoperative events, the patient's satisfaction and good operating conditions were provided by this anaesthetic technique.

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FIGURE 4 Left and right ventricular stroke work indexes, during midazolam-sufentanil analgesia (mean $\pm$ SEM) (**p < 0.01).

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Résumé

On a évalué les effets et la sécurité de la séquence midazolam-sufentanil sur les variables hémodynamiques, le degré d'analgésie, l'amnésie, et le réveil chez quinze patients adultes subissant une chirurgie coronarienne. Après une prémédication de routine le midazolam $0.14 \pm 0.01 \text{ mg} \cdot \text{kg}^{-1}$ par voie intraveineuse a été administré en une minute suivi cinq minutes plus tard par le sufentanil à des doses de $1.5 \mu\text{g} \cdot \text{kg}^{-1}$ pour une dose totale pré-innubation de $4.0\text{--}5.0 \mu\text{g} \cdot \text{kg}^{-1}$ injectée en dix minutes. Une minute après la dose initiale de sufentanil, du pancuronium $0.1 \text{ mg} \cdot \text{kg}^{-1}$ a été administré en 30 secondes. Les doses additionnelles du sufentanil étaient basées sur une augmentation supérieure à 15 pour cent dans le produit pression-fréquence. La dose moyenne de sufentanil avant la CEC était de $9.6 \pm 2.1 \mu\text{g} \cdot \text{kg}^{-1}$ et $13.9 \pm 1.3 \mu\text{g} \cdot \text{kg}^{-1}$ pour la totalité de la procédure. Une diminution significative des pressions systoliques et diastoliques est survenue après administration de midazolam et ceci fut soutenu jusqu'à la sternotomie. Une diminution significative de la résistance vasculaire systémique est survenue après midazolam. Le sufentanil a diminué le travail d'éjection indexé du ventricule gauche. L'intubation trachéale, l'incision cutanée et la sternotomie n'ont pas amené de réaction hémodynamique. Une analgésie adéquate, une amnésie complète et un réveil précoce furent observés.