# Hypotensive actions of diltiazem and nitroprusside compared during fentanyl anaesthesia for total hip arthroplasty

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*The potential for inducing hypotension during fentanyl anaesthesia by administering either diltiazem (n = 7) or sodium nitroprusside (n = 7) was investigated during total hip arthroplasty. Haemodynamic variables were obtained in the lateral position before, during and after administration of the hypotensive agent. Diltiazem 0.15 mg·kg$^{-1}$ given as an IV bolus followed by a 12.5 ± 3 µg·kg$^{-1}$·min$^{-1}$ continuous infusion decreased mean arterial pressure (MAP) from 77 ± 11 mmHg to 63 ± 16 mmHg (p < 0.05) while other haemodynamic parameters showed only minor and insignificant changes. Hypotension continued for at least 30 min after the cessation of diltiazem. With sodium nitroprusside MAP decreased immediately from 81 ± 11 mmHg to 59 ± 9 mmHg (p < 0.01) and rapidly returned to its control value after cessation of the infusion. CI and Qs/Qt rose significantly (p < 0.05) while the systemic vascular resistance index (SVRI) (p < 0.01) and pulmonary vascular resistance index (PVRI) (p < 0.05) fell significantly. The haemo-*

*dynamic profile was significantly different between hypotensive agents for MAP (p < 0.02), heart rate (HR) (p < 0.01), SVRI (p < 0.05), and PVRI (p < 0.05). HR was lower with diltiazem than with nitroprusside. A bradycardia less than 50 beats/min was observed in five patients in the diltiazem group. MAP, SVRI and PVRI were lower with nitroprusside than with diltiazem. Diltiazem can induce and maintain moderate hypotension without tachycardia and decreased cardiac output in humans during fentanyl anaesthesia but the modulation of the level of arterial pressure and the depression of atrioventricular conduction are unpredictable.*

## Key words

BLOOD PRESSURE: drug effects, calcium channel blockers, diltiazem, sodium nitroprusside; ANAESTHETIC TECHNIQUES: hypotension, induced, nitroprusside, diltiazem.

The anticipated benefits of moderate controlled hypotension during total hip arthroplasties no longer have to be demonstrated.[1,2] Sodium nitroprusside (SNP) is the reference pharmacological agent,[3] but its side effects of potential toxicity,[4] reflex activation of the sympathetic system,[5] and hypertensive rebound on withdrawal[6] encourage research on other controlled hypotension protocols. The literature provides little information about the use of calcium channel blockers to produce hypotension[7] despite their well known vasodilator effects.[8] Cottrel *et al.* investigated the effect of nifedipine as an IV bolus on systemic haemodynamics and intracranial pressure in the cat.[9] Oates compared the hypotensive actions of SNP and verapamil infusions in the cat.[10] Zimpfer *et al.* evaluated the haemodynamic effects of an IV bolus injection of verapamil during neuroleptanaesthesia in man in order to reduce impedance to ventricular ejection during the surgical procedure.[11] The potential for inducing hypotension by administering

diltiazem intravenously was investigated in the dog under either halothane anaesthesia,[12] or fentanyl anaesthesia.[13,14] Diltiazem has not been tested in patients,[15] since it has been feared that, like verapamil, it might lower arterial pressure by the preponderant lowering of cardiac output. Nifedipine acts more by the lowering of systemic vascular resistance.[16]

This investigation was designed to compare diltiazem with nitroprusside in producing controlled hypotension during total hip arthroplasty and to determine the haemodynamic profiles produced.

## Methods

Fourteen patients (ten women and four men), with a mean age of 65 years (range: 48 to 77), had total hip arthroplasties (prosthesis Charnley-Kerboull type), under general anaesthesia.

After premedication (15 mg diazepam po), the patients were taken to the pre-anaesthesia room. Heart rate (HR) was monitored by a CM5 lead. An 18 Gauge Teflon catheter was placed in a radial artery to measure systolic (SAP) and diastolic (DAP) arterial pressures. A 7 F thermodilution catheter was placed via the right jugular vein to measure right atrial pressure (RAP), systolic (SPAP) and diastolic (DPAP) pulmonary arterial pressures, pulmonary capillary wedge pressure (PCWP) and cardiac output (CO) (iced injectate in triplicate). Heart rate and pressure were simultaneously recorded on a multi-channel recorder (Mingograf 803 Siemens Elema). Standard formulae were used to calculate mean arterial pressure: $MAP = (SAP - DAP)/3 + DAP$, cardiac index: $CI = CO/BSA$, stroke index: $SI = CI/HR$, systemic vascular resistance index: $SVRI = (MAP - RAP)/CI \times 80$, and pulmonary vascular resistance index $PVRI = (MPAP - PCWP)/CI \times 80$.

$PO_2$, $PCO_2$ (ABL 30 acid-base analyzer radiometer), $SO_2$ and haemoglobin concentration ($OSM_2$ hemoximeter radiometer) were measured in arterial blood and in mixed venous blood. The arterial ($CaO_2$), mixed venous ($C\bar{v}O_2$) and capillary ($CcO_2$) contents were calculated using the usual formulae: oxygen content $= (SO_2 \times Hb \times 1.34) + PO_2 \times 0.003$, assuming $PcO_2 = PAO_2$ and $ScO_2 = 100$ per cent. The intrapulmonary shunt was calculated according to the formula: $Qs/Qt = CcO_2 - CaO_2/CcO_2 - C\bar{v}O_2$.

Patients were anaesthetized with thiopentone

(5 mg·kg⁻¹) and fentanyl (5 μg·kg⁻¹), intubated after pancuronium injection (0.1 mg·kg⁻¹) and mechanically ventilated. Anaesthesia was maintained with oxygen in nitrous oxide ($FiO_2$: 0.5). The initial adjustments were adapted to maintain a $PaCO_2$ between 4.5 and 5.2 kPa (35–40 mmHg), with a respiratory frequency of 16 per minute and an I/E ratio = 1/2. Reinjections of fentanyl and pancuronium were systematic, while atropine was not given (0.5 mg IV) unless the HR was less than 50 per minute.

## Protocol

An initial measurement of arterial pressure was obtained before anaesthesia in order to define the desired level of hypotension. The aim was to obtain moderate hypotension, equal to 50 per cent of the MAP of the patient when awake, without going below 55 mmHg. Patients were randomly divided into two groups: sodium nitroprusside (n = 7) and diltiazem (n = 7). The hypotensive agent was perfused using a teflon antecubital catheter. The infusion rate of SNP (concentration 200 mg·L⁻¹) was regulated from 1 to 6 μg·kg⁻¹·min⁻¹ to achieve the desired hypotension. After the standardized injection of 0.15 mg·kg⁻¹ of diltiazem during one

TABLE I   Basic patient data (mean values ± SD)

| | Patient groups | |
|---|---|---|
| | Diltiazem (n = 7) | Nitroprusside (n = 7) |
| Age (years) | 65.4 ± 9.1 | 65.8 ± 8.4 |
| Weight (kg) | 61.7 ± 12.1 | 62.9 ± 13.8 |
| Sex | 5F; 2M | 5F; 2M |
| Duration of operation (min) | 110 ± 17 | 95 ± 18 |
| $PaCO_2$ (kPa) | 4.8 ± 0.4 | 4.8 ± 0.4 |
| pH | 7.44 ± 0.04 | 7.43 ± 0.04 |
| Fentanyl dose (μg·kg⁻¹) | | |
| at incision | 10.4 ± 2.4 | 10.5 ± 2.3 |
| before first cementing | 17.9 ± 3.2 | 17.3 ± 3.2 |
| Volume expansion (ml) | | |
| fresh frozen plasma | 1500 ± 500 | 1650 ± 900 |
| packed red cells | 1100 ± 600 | 800 ± 600 |
| Postoperative hematocrit | 36.4 ± 1.7 | 36.1 ± 1.9 |

Non-paired *t* test: NS.
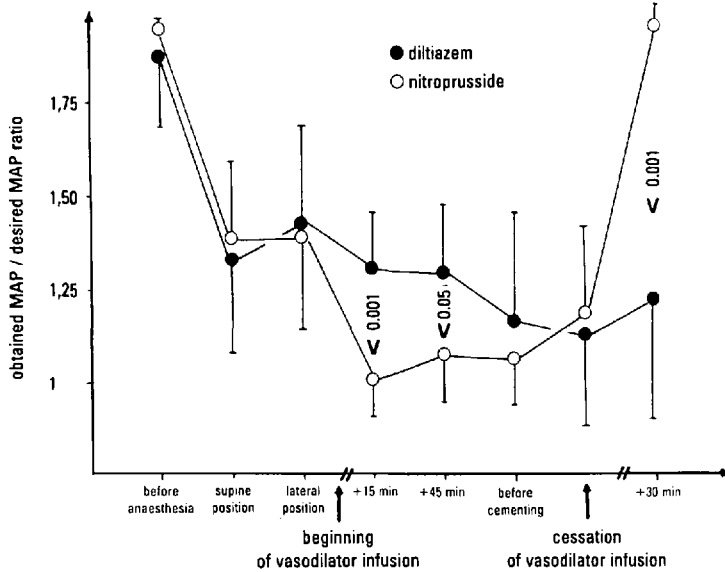
FIGURE   Evolution of the ratio MAP obtained/MAP desired throughout the study. The ratio was
one when the goal (a 50 per cent decrease in MAP) was obtained. As the desired MAP was 50 per
cent of the MAP of the patient when awake, the ratio was two before and after hypotension.

minute, the further infusion (rate about 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$) was then regulated according to the pressure result obtained. The overall haemodynamic data and the ratio MAP obtained/MAP desired were recorded once the patient was asleep, in the supine position and then in the lateral position, prior to the administration of the hypotensive agent, during its administration (15th and 45th minutes of hypotension, before the first cementing and cessation of the product), and 30 minutes after stopping its administration. Fresh frozen plasma was perfused into a peripheral vein in order to maintain PCWP in the range 8–12 mmHg; red blood cells were begun when blood loss was greater than 300 ml. The intrapulmonary shunt was calculated in the lateral position prior to the administration of the hypotensive agent, 15 minutes after the beginning of its administration, and then before the first cementing.

The haemodynamic data are expressed as means ± SD. Statistical analysis was done using ANOVA in order to test the haemodynamic profile within each group; when the variance was significant, a paired $t$ test was used to assess the significance of the changes over time. The analysis of variance then permitted the difference between the two hypotension methods to be determined; when the variance was significant, an unpaired $t$ test was used to localise the difference. Finally, the variance analysis permitted an investigation of interference. Significance was assumed at p values less than 0.05.

## Results

Some general information is summarized in Table I. There were no significant differences between the two groups of patients.

The course of the MAP during the investigation is given in Table II. The desired lowering of pressure was identical for both groups ($-46.1 \pm 6.4$ per cent in the diltiazem group and $-48 \pm 1.5$ per cent in the nitroprusside group). The figure shows the course of the ratio MAP obtained/MAP desired. A decrease in this ratio was produced by the anaesthetic itself and the changes of body position. With nitroprusside, the desired level of hypotension was reached immediately and was maintained until the first cementing. With diltiazem, the objective was

TABLE II   Course of mean arterial pressure (mean ± SD)

| Time | Diltiazem (n = 7) | Nitro-prusside (n = 7) |
|---|---|---|
| Supine position when awake | 106 ± 13 | 114 ± 16 |
| Desired hypotension | 56 ± 2 | 56 ± 7 |
| Supine position when anaesthetized | 71 ± 17 | 84 ± 25 |
| Lateral position when anaesthetized | 77 ± 17 | 81 ± 11 |
| Vasodilator + 15 min | 73 ± 10 | 59 ± 9** |
| Vasodilator + 45 min | 72 ± 12 | 63 ± 9* |
| Vasodilator prior to first cement | 66 ± 17 | 62 ± 9† |
| Before cessation of vasodilator | 63 ± 16* | 70 ± 17 |
| Cessation of vasodilator + 30 min | 68 ± 16 | 114 ± 12** |
| F | 5.44 | 17.86 |
| p | 0.001 | 0.001 |

Significant differences within groups: paired *t* test; *vs* lateral position when anaesthetized, *p < 0.05; †p < 0.02; **p < 0.01. Significant differences between groups: non-paired *t* test; vasodilator + 15 min: p < 0.05; cessation of vasodilator + 30 min: p < 0.02.

reached only for the latest points (prior to cementing and before cessation of the infusion), after having doubled, even tripled, the infusion flow (overall dose: $1.125 \pm 0.250 \; mg \cdot kg^{-1}$; mean dosage: $12.5 \pm 3 \; \mu g \cdot kg^{-1} \cdot min^{-1}$). Furthermore, the diltiazem infusion had to be stopped on three occasions due to excessive hypotension. Blood pressure rose immediately after the cessation of nitroprusside, while hypotension continued for at least 30 min after the cessation of diltiazem (cessation + 60 minutes: MAP = 76 ± 16 mmHg).

The haemodynamic course and the intrapulmonary shunt values during the diltiazem infusion are given in Table III, and the corresponding results for the nitroprusside group are given in Table IV. Although none of the parameters studied changed significantly in the diltiazem group, bradycardias of less than 50 beats/min required atropine injections in five of the seven patients; a junctional bradycardia persisted in two of these five patients and diltiazem was then stopped. With sodium nitroprusside, CI and Qs/Qt rose significantly (p < 0.05),

while the SVRI (p < 0.01) and PVRI (p < 0.05) fell significantly.

The time course of changes was significantly different between the hypotensive agents for MAP (p < 0.02), HR (p < 0.01), SVRI (p < 0.05), and PVRI (p < 0.05). MAP was significantly lower with nitroprusside than with diltiazem at 15 minutes (p < 0.05) and significantly higher with nitroprusside than with diltiazem 30 min after cessation of the vasodilator (p < 0.02) (Table II). HR was significantly lower with diltiazem than with nitroprusside at 45 minutes (p < 0.05), and before the first cementing (p < 0.02). The SVRI and PVRI were significantly lower with nitroprusside than with diltiazem at 45 minutes (p < 0.05) and before cementing (p < 0.05).

A significant interaction existed between the use of nitroprusside or diltiazem and the overall course of MAP (p < 0.001) and SVRI (p < 0.05).

## Discussion

In terms of handling or efficacy diltiazem is not comparable to sodium nitroprusside as a controlled hypotension agent for use in anaesthesia. The choice of the initial dosage (IV bolus of 0.15 $mg \cdot kg^{-1}$ followed by a continuous infusion of 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$) which was as a result of the work of Valette *et al*.[17] led to a weak and inconsistent drop in blood pressure. The corresponding plasma levels reported by Valette *et al*.[17] ($145 \; ng \cdot ml^{-1}$ after 20 minutes, and $321 \; ng \cdot ml^{-1}$ after 45 minutes) are nonetheless within the range of therapeutic efficacy.[18-20] The effect of under-dosage seems to have been avoided since the dosage was doubled, even tripled, during the procedure. Significant hypotension was obtained only after the 60th minute, but there were considerable individual variations. In some cases hypotension did not occur while in others hypotension was too severe. The hypotensive effect was accentuated as time passed, contrary to the study of Pierrot *et al*. (bolus IV of 0.15 $mg \cdot kg^{-1}$ followed by a continuous infusion of 1 $\mu g \cdot kg^{-1} \cdot min^{-1}$) in which the effect decreased with time.[21] Finally, the hypotensive effect was prolonged for at least 60 minutes after cessation of the infusion, in agreement with Bourassa *et al*.[22] While the persistence of this hypotension before closing of superficial layers may be considered to be harmful, the postoperative course was comparable in both groups.

TABLE III   Haemodynamic course during diltiazem infusion (mean values ± SD)

| | Baseline (lateral position) | Diltiazem infusion | | | | ANOVA p |
| | | + 15 min | + 45 min | Before cementing | Before cessation of diltiazem | |
|---|---|---|---|---|---|---|
| HR (beat/min) | 62.6 ± 9 | 65.1 ± 7.8 | 63.1 ± 7.7 | 61.7 ± 7.6 | 62 ± 7.8 | NS |
| RAP (mmHg) | 8 ± 6.6 | 9.1 ± 5 | 7 ± 5.3 | 8.3 ± 6.3 | 8.4 ± 5.6 | NS |
| MPAP (mmHg) | 18 ± 4.4 | 17.9 ± 4.6 | 16.9 ± 4.1 | 17.3 ± 6.1 | 18.1 ± 4.6 | NS |
| PCWP (mmHg) | 10.9 ± 3 | 10.7 ± 4.6 | 10 ± 5.1 | 10.4 ± 6.1 | 10.3 ± 5.3 | NS |
| CI ($L \cdot min^{-1} \cdot m^{-2}$) | 2.56 ± 0.36 | 2.91 ± 0.44 | 2.75 ± 0.48 | 2.69 ± 0.61 | 2.79 ± 0.73 | NS |
| SI ($ml \cdot beat^{-1} \cdot m^{-2}$) | 41.1 ± 5.4 | 44.6 ± 8.1 | 43.1 ± 4.4 | 42.9 ± 11.2 | 44.4 ± 13.1 | NS |
| SVRI ($dyne \cdot sec \cdot cm^{-5} \cdot m^{-2}$) | 2234 ± 498 | 1810 ± 330 | 1969 ± 582 | 1800 ± 564 | 1608 ± 622 | NS |
| PVRI ($dyne \cdot sec \cdot cm^{-5} \cdot m^{-2}$) | 219 ± 98 | 200 ± 78 | 203 ± 62 | 223 ± 35 | 248 ± 61 | NS |
| Qs/Qt (%) | 16.9 ± 5.1 | 15.6 ± 5 | | 18.2 ± 8.4 | | NS |

TABLE IV   Haemodynamic course during nitroprusside infusion (mean values ± SD)

| | Baseline (lateral position) | Nitroprusside infusion | | | | ANOVA p |
| | | + 15 min | + 45 min | Before cementing | Before cessation of nitroprusside | |
|---|---|---|---|---|---|---|
| HR (beat/min) | 70.9 ± 8.4 | 73.3 ± 11.0 | 80.2 ± 15.4 | 82.1 ± 18.4 | 74.7 ± 15.2 | NS |
| RAP (mmHg) | 5.1 ± 2.7 | 5 ± 3.3 | 5.2 ± 3.7 | 4.1 ± 4.8 | 5.6 ± 4.8 | NS |
| MPAP (mmHg) | 16.6 ± 4.5 | 14.9 ± 3.6 | 16 ± 3.3 | 14.9 ± 3.8 | 19.7 ± 3.3 | NS |
| PCWP (mmHg) | 8.7 ± 3.2 | 8.1 ± 4.7 | 9.5 ± 3.4 | 8.2 ± 4.6 | 9.9 ± 5.3 | NS |
| CI ($L \cdot min^{-1} \cdot m^{-2}$) | 2.36 ± 0.59 | 3.13 ± 0.90* | 3.76 ± 1.46** | 3.34 ± 1.21* | 2.92 ± 0.79* | <0.05 |
| SI ($ml \cdot beat^{-1} \cdot m^{-2}$) | 32.9 ± 6.4 | 42.2 ± 9.5 | 46.3 ± 13.8 | 36.9 ± 20.4 | 40.6 ± 13.3 | NS |
| SVRI ($dyne \cdot sec \cdot cm^{-5} \cdot m^{-2}$) | 2752 ± 512 | 1455 ± 371** | 1240 ± 355† | 1414 ± 444** | 1783 ± 230* | <0.01 |
| PVRI ($dyne \cdot sec \cdot cm^{-5} \cdot m^{-2}$) | 254 ± 77 | 181 ± 79 | 128 ± 41* | 165 ± 43 | 190 ± 82 | <0.05 |
| Qs/Qt (%) | 13.6 ± 3.4 | 21.4 ± 8.9 | | 21.9 ± 6.8* | | <0.05 |

Paired t test vs lateral position.   *p < 0.05.   †p < 0.02.   **p < 0.01.

Diltiazem seems to be poorly adapted to the technique of controlled hypotension, contrary to the statement of Griffin et al.[13] who studied dogs anaesthetized with fentanyl. The dose of diltiazem (30 to 90 $\mu g \cdot kg^{-1} \cdot min^{-1}$) necessary to obtain a significant drop in systemic vascular resistance (from 3249 ± 257 to 1138 ± 148 $dyne \cdot sec \cdot cm^{-5} \cdot m^{-2}$) were accompanied by electrophysiological modifications (five of six dogs developed first-degree AV block and four of these five dogs developed 2nd-degree AV block);[13] two dogs had episodes of junctional rhythm.[13] Furthermore negative chronotropic effects appear to be dose-related and HR decreased from 109 ± 6.1 to 90 ± 13 beats/min.[13] In our study smaller doses (12.5 ± 3 $\mu g \cdot kg^{-1} \cdot min^{-1}$) led also to bradycardia and junctional rhythm. However, cardiac index increased (from 2.40 ± 0.20 to 4.93 ± 0.89 $L \cdot min^{-1} \cdot m^{-2}$) due to the reduction in afterload, while the dp/dt was little modified.[13]

In our study smaller doses resulted in a smaller decrease in SVRI (−28 per cent *vs* −65 per cent in the study of Griffin *et al*.) and *per se* in no increase in CI. Hypotension provoked by diltiazem does not seem, therefore, to be linked to myocardial depression, contrary to what is observed in swine anaesthetized with halothane.[23] Kates *et al*.[23] demonstrated that blood pressure is reduced by decreasing cardiac output (−42 per cent) without affecting systemic vascular resistance. The hypotensive mechanism appears to be different with anaesthesia using fentanyl, since in the absence of modifications in the conditions of ventricular load, the stroke volume and cardiac output are not modified, despite a known-negative inotropic action.[24-26]

In conclusion, arterial pressure is difficult to control with diltiazem and the added depression of AV conduction make its use in controlled hypotension during anaesthesia with fentanyl undesirable. On the other hand, diltiazem provides no harmful effects on cardiac output and produces excellent haemodynamic stability during the recovery period.

## Acknowledgements

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Résumé

*Le diltiazem a été testé comparativement au nitroprussiate de sodium pour provoquer une hypotension contrôlée au cours des arthroplasties de hanche (n = 14). Les données hémodynamiques ont été recueillies sous anesthésie générale (thiopental – fentanyl – pancuronium) en décubitus latéral avant, pendant et après administration de l'agent hypotenseur (choix randomisé). Le diltiazem (bolus IV de 0.15 mg·kg$^{-1}$ suivi d'une perfusion continue de 12.5 ± 3 µg·kg$^{-1}$·min$^{-1}$) a entraîné une baisse de pression artérielle moyenne (PAM) de 77 ± 11 mmHg à 63 ± 16 mmHg (p < 0.05) sans modifications significatives des autres données hémodynamiques. L'hypotension s'est maintenue au moins 30 min après arrêt du diltiazem. Le nitroprussiate de sodium a entraîné une baisse immédiate de PAM de 81 ± 11 mmHg à 59 ± 9 mmHg (p < 0.01); le retour de PAM à la valeur contrôle est obtenue rapidement après arrêt du nitroprussiate. L'index cardiaque et Qs/Qt se sont élevés significativement (p < 0.05) alors que l'index des résistances vasculaires systémiques (RVS) (p < 0.01) et l'index des résistances vasculaires pulmonaires (RVP) (p < 0.05) se sont abaissés significativement. Ces profils hémodynamiques ont été significativement différents pour la MAP (p < 0.02), la fréquence cardiaque (FC) (p < 0.01), les RVS (p < 0.05) et les RVP (p < 0.05). FC a été plus basse sous diltiazem que sous nitroprussitate; une bradycardie inférieure à 50/min a été observée chez cinq des sept patients. PAM, RVS et RVP ont été plus basses sous nitroprussiate que sous diltiazem. Le diltiazem est susceptible d'induire, de maintenir une hypotension sans tachycardie et sans diminution du débit cardiaque chez l'homme pendant une anesthésie au fentanyl, mais, la modulation du niveau d'hypotension et la dépression de la conduction auriculo-ventriculaires sont imprévisibles.*