Systemic haemodynamic and metabolic effects of deliberate hypotension with isoflurane anaesthesia or sodium nitroprusside during total hip arthroplasty

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Isoflurane (ISO) was examined as an alternative hypotensive agent to nitroprusside (SNP) in 16 patients (mean age: 60 years) anaesthetized for total hip arthroplasty. MAP was decreased to 50 per cent of the awake level by infusion of SNP in Group I (n = 8) and with ISO in Group II (n = 8). Fentanyl ($10\text{--}16\text{ }\mu\text{g}\cdot\text{kg}^{-1}$) was administered to both groups. Haemodynamic measurements were repeated in the lateral position before, during and after hypotension. Polygeline and fresh frozen plasma were infused throughout the study period in volumes sufficient to maintain pulmonary capillary wedge pressure in the 7–9 mmHg range. The MAP decrease was the same in both groups, as were perioperative blood replacement (mean 500 ml), and postoperative haematocrits. Total perioperative fluid replacement was higher ($p < 0.01$) in Group I (mean 2500 ml) than in Group II (mean 1300 ml). Venous tone was more affected by SNP than by ISO. ISO decreased the systemic vascular resistance index and oxygen consumption ($\dot{V}\text{O}_2$) without any change in CI or in $\dot{Q}\text{s}/\dot{Q}\text{t}$, in contrast to SNP which increased CI, $\dot{V}\text{O}_2$ and $\dot{Q}\text{s}/\dot{Q}\text{t}$.

Key words

ANAESTHETIC TECHNIQUES: induced hypotension, nitroprusside; ANAESTHETICS: VOLATILE, isoflurane; SURGERY: orthopaedics.

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During total hip arthroplasty, blood loss can be decreased by employing hypotension.^{1,2} The choice of the hypotensive agent remains controversial. Sodium nitroprusside (SNP) is the pharmacological agent of reference.³ Its side effects: (i) potential toxicity,⁴ (ii) rebound hypertension during withdrawal,⁵ (iii) reflex increase of the sympathetic⁶ and renin-angiotensin activity,⁷ and (iv) impairment of pulmonary gas exchange,⁸ encourage research on other controlled hypotension protocols.

Fluorinated volatile anaesthetic agents may provide a useful alternative.⁹ The mechanism underlying the decrease in arterial pressure is different with each agent: depression of myocardial contractility is important with halothane and enflurane. The decrease in systemic vascular resistance is important with isoflurane, moderate with enflurane and only minimal with halothane.¹⁰ Although depressed myocardial contractility has been shown with isoflurane *in vitro*,^{11–13} *in vivo* studies have not shown a decrease in cardiac output.^{14–17} While there are reports of the use of isoflurane-induced hypotension^{18–20} there are few clinical comparisons with SNP.²¹

The purpose of this study was to evaluate the haemodynamic and metabolic effects of isoflurane during hypotensive anaesthesia for total hip arthroplasty, compared with a technique using SNP.

Methods

The study was approved by the Committee on Ethics of our University Hospital. After giving their informed consent, 16 patients (ten women, six men) underwent total hip arthroplasty (Charnley Kerboul hip prosthesis) under general anaesthesia with similar surgical conditions. The medullary cavity was plugged by a plastic plug. A plastic catheter was used to vent blood, air and fat. The cement (orthopaedic bone cement powder type I CMW Labora-

tories) was forced into the medullary cavity of the femur using the pulp of a thumb as a piston, 3–4 min after adding the liquid to the powder. The prosthesis was pushed into the cement and forced down.

Mean age was 60 (range 41–77). Six patients had a history of moderate essential hypertension either treated (alpha-methyl dopa: five, thiazide diuretic: three) or untreated. Patients with ischaemic cerebral vascular disease, ischaemic myocardial disease or severe systemic hypertension were excluded from the study.

Heart rate was monitored by a V_5 ECG lead. An indwelling teflon cannula was inserted into a radial artery for systolic (SAP) and diastolic blood pressure (DAP) measurement. A 7-F triple lumen thermodilution catheter was inserted via the right internal jugular vein to measure the 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). HR and pressures were recorded simultaneously on a polygraph (Mingograf 803 Siemens Elema). PO_2 , PCO_2 , pH, (ABL 30 Acid-base analyzer, Radiometer), SO_2 and haemoglobin concentration (OSM2 Hemoximeter, Radiometer) were measured in arterial blood and in mixed venous blood. Derived values: mean arterial pressure: MAP, cardiac index: CI, stroke index: SI, systemic vascular resistance index: SVRI, pulmonary vascular resistance index: PVRI, oxygen content, oxygen consumption ($\dot{V}O_2$), and intra-pulmonary shunt ($\dot{Q}_s/\dot{Q}_t$) were computed according to standard formulae.²²

Protocol

The patients were randomly assigned to one of two groups.

GROUP I SNP-FENTANYL

After premedication (diazepam 15 mg PO) anaesthesia was induced with thiopentone ($5 \text{ mg} \cdot \text{kg}^{-1}$), fentanyl ($5 \mu\text{g} \cdot \text{kg}^{-1}$) and pancuronium ($0.1 \text{ mg} \cdot \text{kg}^{-1}$). After tracheal intubation, the patients were mechanically ventilated using a non-rebreathing circuit and IPPV. They received an O_2/N_2O mixture with FiO_2 of 0.5. End tidal CO_2 was monitored with a capnometer (Hewlett Packard 47210A) and initial settings were modified in order to maintain $PaCO_2$ between 4.2 and 5.2 kPa (32–40 mmHg), with a respiratory rate of 16/minute and an I/E ratio of 1/2. Supplementary doses of fentanyl and pancuronium were given during the operation. SNP was infused (concentration $200 \text{ mg} \cdot \text{L}^{-1}$) through a teflon antecubital cannula used only for this purpose. Infusion was started with the skin incision, regulated from $1\text{--}6 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ to

achieve the desired hypotension, and discontinued five minutes after placement of the second bone cement.

GROUP II ISOFLURANE-FENTANYL

The protocol was identical except that the SNP was replaced by isoflurane. Isoflurane four per cent (vaporizer setting, Isoflurane Vapor 19.3 Drager) was started before the skin incision, then the concentration was quickly decreased and modified in response to blood pressure. Isoflurane was discontinued five minutes after placement of the second bone cement. The isoflurane end-tidal concentration was monitored by a Normac Datex analyzer.

Arterial blood pressure was measured prior to general anaesthesia in order to determine the desired level of hypotension. The aim was to reach a moderate level of hypotension, equal to 50 per cent of the MAP of the awake patient, without going below 50 mmHg. Haemodynamic data and blood samples were collected during stable anaesthesia with the patients in the lateral position, before hypotension, during hypotension (15 min after the onset of hypotension, 5 min before the first cement, 5 min after the second cement), and after hypotension during skin closure (mean 40 min after insertion of the second cement). Fluid replacement was with colloids (polygeline) and fresh frozen plasma (FFP), in order to maintain PCWP between 7 and 9 mmHg. The goal was to maintain a constant ventricular preload in both groups to avoid deleterious consequences of a substantial degree of venodilation. Blood replacement (red blood cells) was done when blood loss exceeded 300 ml. Operative blood loss was assessed during the operation by weighing sponges, measuring suction drainage and estimating the amount of blood in the area of the wound, and after the operation by measuring blood collected from the wound drainage.

Results are expressed as mean $\pm$ SD. Intragroup comparisons over time were determined by analysis of variance for repeated measures, followed when indicated, by paired t-test with Bonferroni corrections. Intergroup comparisons were made by analysis of variance where the two factors were treatment (SNP-fentanyl or isoflurane-fentanyl) and time (before, during and after hypotension). When variance was significant a modified Student's t test was used to locate the difference. Significance was assumed when $p < 0.05$.

Results

Summary data for the two groups are presented in Table I. There were no significant haemodynamic differences between the groups prior to general anaesthesia. There were no complications during or after the study. MAP changes during the study are shown in Table II. In both

TABLE I Demographic information and haemodynamic data before anaesthesia (Mean $\pm$ SD)

	Nitroprusside-fentanyl (n = 8)	Isoflurane-fentanyl (n = 8)
Age (years)	62 (48–77)	57 (41–75)
Weight (kg)	63 (50–85)	60 (45–82)
Sex	5 F; 3 M	5 F; 3 M
Essential hypertension	3	3
HR (beats·min ⁻¹)	76 $\pm$ 9	77 $\pm$ 10
PCWP (mmHg)	5.9 $\pm$ 1.9	6.2 $\pm$ 2.4
CI (L·min ⁻¹ ·m ⁻²)	3.45 $\pm$ 0.62	3.39 $\pm$ 0.55
SI (ml·beat ⁻¹ ·m ⁻²)	45.4 $\pm$ 10.3	44.1 $\pm$ 11.2
SVRI (dyn·s·cm ⁻⁵ ·m ⁻²)	2500 $\pm$ 482	2452 $\pm$ 357
Hypotension duration (min)	90 $\pm$ 18	85 $\pm$ 25
Total fentanyl dose (μ g·kg ⁻¹)		
– at incision	10.3 $\pm$ 2.1	9.6 $\pm$ 1.8
– before 1st cement	17.1 $\pm$ 3.1	15.9 $\pm$ 4.1
Operative blood losses (ml)		
– during operation	330 $\pm$ 100	310 $\pm$ 100
– after operation	530 $\pm$ 300	510 $\pm$ 260
– total	860 $\pm$ 320	820 $\pm$ 280
Total fluid replacement (ml)		
– red blood cells	550 $\pm$ 400	500 $\pm$ 350
– fresh frozen plasma	700 $\pm$ 100	300 $\pm$ 100*
– polygeline	1250 $\pm$ 400	500 $\pm$ 100*

*p < 0.01 vs nitroprusside.

Abbreviations: HR, heart rate; PCWP, pulmonary capillary wedge pressure; CI, cardiac index; SI, stroke index; SVRI, systemic vascular resistance index.

groups the same decrease in MAP was achieved from induction of anaesthesia and positioning of the patients in the lateral position. There were no differences between the groups during hypotension. The decrease in MAP had a similar effect in both groups on the operative blood loss (Table I). The postoperative haematocrits were respectively 36.1 ± 1.9 and 36.6 ± 5.4 (NS). Fluid replacement (colloids, FFP and red blood cells), how-

TABLE II Mean arterial pressure (Mean $\pm$ SD)

	Nitroprusside-fentanyl (n = 8)	Isoflurane-fentanyl (n = 8)
Awake in the supine position	113 $\pm$ 15	109 $\pm$ 13
Anaesthetized in the lateral position	82 $\pm$ 11	89 $\pm$ 15
Hypotension + 15 min	58 $\pm$ 9†	62 $\pm$ 7†
Hypotension 5 min before 1st cement	63 $\pm$ 9*	53 $\pm$ 4‡
Hypotension 5 min after 2nd cement	72 $\pm$ 16	58 $\pm$ 9†
Skin closure	118 $\pm$ 13†	87 $\pm$ 11‡

Within group variations: vs anaesthetized in the lateral position: *p < 0.05; †p < 0.01; ‡p < 0.001.

Between group variations: §p < 0.01 vs nitroprusside.

FIGURE Arterio-venous O_2 difference ($CaO_2 - CvO_2$), oxygen consumption (VO_2), cardiac index (CI) and intrapulmonary shunt (Q_s/Q_t) in Group I (nitroprusside-fentanyl) and in Group II (isoflurane-fentanyl) in the lateral position before hypotension (C), and during hypotension 15 minutes after it was started (T1), 5 minutes before first cement (T2) and 5 minutes after the second cement (T3). Mean value $\pm$ SD; when compared with control values in the lateral position before hypotension (C): *p < 0.05; **p < 0.01.

ever, was significantly more (p < 0.01) in the SNP-fentanyl group (2500 ± 900 ml) as compared to the isoflurane-fentanyl group (1300 ± 500 ml). The isoflurane end-tidal maintenance concentration was 0.6 ± 0.2 vol% and the total amount of isoflurane needed was 0.37 ± 0.14 ml·kg⁻¹·h⁻¹. Blood pressure started to rise immediately after the SNP infusion was stopped and rebound hypertension occurred, as compared to values with the patient in the lateral position before hypotension (p < 0.01; Table I). Blood pressure started to rise 8 to 10 min after isoflurane was stopped, reaching its prehypotensive level without rebound.

Within group variations

The changes in haemodynamic and metabolic data during hypotension are shown in Table III and in the Figure. With SNP-fentanyl, SVRI (p < 0.01) and PaO_2 (p < 0.05) were significantly decreased, CI, Q_s/Q_t (p < 0.05), and VO_2 (p < 0.01) were significantly increased. With isoflurane SVRI (p < 0.01) and VO_2 (p < 0.05) were

TABLE III Data summary during sodium nitroprusside-fentanyl hypotension (Group I) and isoflurane-fentanyl hypotension (Group II) (Mean $\pm$ SD)

	Group	Lateral position	Hypotension + 15 min	Hypotension 5 min before 1st cement	Hypotension 5 min after 2nd cement	Significance
HR (beats $\cdot$ min $^{-1}$)	SNP	70 $\pm$ 9	73 $\pm$ 10	81 $\pm$ 18	75 $\pm$ 15	NS
	ISO	75 $\pm$ 12	71 $\pm$ 11	75 $\pm$ 13	68 $\pm$ 9	NS
RAP (mmHg)	SNP	4.9 $\pm$ 2.7	4.7 $\pm$ 3.1	4.0 $\pm$ 4.5	5.3 $\pm$ 4.7	NS
	ISO	3.5 $\pm$ 2.6	4.2 $\pm$ 3.6	3.7 $\pm$ 2.7	4.4 $\pm$ 2.3	NS
MPAP (mmHg)	SNP	16.3 $\pm$ 4.5	15.0 $\pm$ 3.5	14.7 $\pm$ 3.8	19.8 $\pm$ 3.1 $\ddagger$	NS
	ISO	14.1 $\pm$ 2.9	13.5 $\pm$ 2.6	13.2 $\pm$ 2.5	16.0 $\pm$ 2.7	NS
PCWP (mmHg)	SNP	8.5 $\pm$ 3.0	8.0 $\pm$ 4.6	8.1 $\pm$ 3.6	8.9 $\pm$ 4.3	NS
	ISO	7.4 $\pm$ 3.4	6.5 $\pm$ 4.2	7.6 $\pm$ 3.8	7.2 $\pm$ 2.7	NS
CI (L $\cdot$ min $^{-1}$ $\cdot$ m $^{-2}$)	SNP	2.40 $\pm$ 0.60	3.20 $\pm$ 0.88*	3.42 $\pm$ 1.18* $\ddagger$	3.13 $\pm$ 0.85 $\ddagger$	<0.05
	ISO	2.59 $\pm$ 0.49	2.48 $\pm$ 0.46	2.37 $\pm$ 0.76	2.14 $\pm$ 0.62	NS
SI (ml $\cdot$ beat $^{-1}$ $\cdot$ m $^{-2}$)	SNP	34 $\pm$ 7	44 $\pm$ 9	42 $\pm$ 18	42 $\pm$ 12 $\ddagger$	NS
	ISO	36 $\pm$ 10	36 $\pm$ 10	32 $\pm$ 10	31 $\pm$ 6	NS
SVRI (dyn $\cdot$ s $\cdot$ cm $^{-5}$ $\cdot$ m $^{-2}$)	SNP	2570 $\pm$ 535	1335 $\pm$ 380 $\ddagger$	1380 $\pm$ 465 $\ddagger$	1704 $\pm$ 250*	<0.01
	ISO	2740 $\pm$ 712	1911 $\pm$ 354*	1850 $\pm$ 505 $\ddagger$	2132 $\pm$ 633	<0.01
PVRI (dyn $\cdot$ s $\cdot$ cm $^{-5}$ $\cdot$ m $^{-2}$)	SNP	260 $\pm$ 80	175 $\pm$ 82	155 $\pm$ 53	278 $\pm$ 92	NS
	ISO	218 $\pm$ 61	213 $\pm$ 88	188 $\pm$ 32	325 $\pm$ 93 $\ddagger$	<0.01
pH	SNP	7.41 $\pm$ 0.03	7.42 $\pm$ 0.03	7.42 $\pm$ 0.03	7.41 $\pm$ 0.04	NS
	ISO	7.40 $\pm$ 0.04	7.41 $\pm$ 0.04	7.40 $\pm$ 0.03	7.39 $\pm$ 0.04	NS
PaCO ₂ (kPa)	SNP	5.0 $\pm$ 0.2	5.0 $\pm$ 0.3	5.1 $\pm$ 0.3	5.1 $\pm$ 0.3	NS
	ISO	5.0 $\pm$ 0.2	4.8 $\pm$ 0.3	4.9 $\pm$ 0.3	4.9 $\pm$ 0.3	NS
PaO ₂ (kPa)	SNP	24.5 $\pm$ 4.5	20.1 $\pm$ 4.5	17.9 $\pm$ 4.3* $\ddagger$	18.9 $\pm$ 4.8* $\ddagger$	<0.05
	ISO	23.9 $\pm$ 5.7	24.4 $\pm$ 6.4	25.9 $\pm$ 5.5	25.8 $\pm$ 5.4	NS

Within group variations: vs lateral position * p < 0.05; $\ddagger p$ < 0.01.

Between group variations: vs Group II $\ddagger p$ < 0.05.

Abbreviations: See Table I RAP, right atrial pressure; MPAP, mean pulmonary artery pressure; PVRI, pulmonary vascular resistance index.

significantly decreased without any change in CI, $\dot{Q}_s/\dot{Q}_t$, pH, PaCO₂ and PaO₂.

Between group comparisons

The two groups, which were haemodynamically identical before hypotension, behaved differently during hypotension: CI, $\dot{V}O_2$ and $\dot{Q}_s/\dot{Q}_t$ were smaller in the isoflurane-fentanyl group before the first cement (p < 0.05) and after the second cement (p < 0.05); SVRI was higher in the isoflurane-fentanyl group 15 minutes after the hypotension onset (p < 0.05) and before the first cement (p < 0.05); SI was less in the isoflurane-fentanyl group after the second cement (p < 0.05); PaO₂ was reduced in the SNP-fentanyl group before the first cement (p < 0.05) and after the second cement (p < 0.05).

Discussion

An attempt was made to control left ventricular filling pressures by infusing fluid to maintain PCWP. The haemodynamic response to the administration of SNP repre-

sents a combination of venous pooling and reduced arterial impedance.²³ In patients with a normal or nearly normal left ventricle the further decrease in PCWP during hypotension in the absence of fluid loading will have a more profound effect on cardiac output than the concomitant reduction of impedance leading to a fall in cardiac output. Furthermore, as suggested by Khambatta *et al.*,⁸ the increase in pulmonary deadspace may be minimized by the maintenance of filling pressures and cardiac output. In the absence of such a fluid replacement protocol and controlled PCWP, the observed changes in cardiovascular function may have been different so that the results of our experiment are applicable only to the exact experimental circumstance we employed.

Isoflurane allowed us to reach quickly the desired level of hypotension. A maintenance inspired concentration of 1 ± 0.2 per cent (according to vaporizer setting) was enough to maintain the desired hypotension level if fentanyl doses were important ($10 \mu\text{g} \cdot \text{kg}^{-1}$ on skin incision; $16 \mu\text{g} \cdot \text{kg}^{-1}$ on cement insertion). During painful times

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Résumé

L'isoflurane (ISO) a été confronté au nitroprussiate de sodium (NPS) comme agent d'hypotension contrôlée chez 16 patients (âge moyen 60 ans) endormis pour remplacement prothétique de hanche. La pression artérielle moyenne (PAM) a été réduite à 50 pour cent de sa valeur chez le sujet éveillé par perfusion de NPS dans le groupe I (n = 8) et par inhalation d'ISO dans le groupe II (n = 8). Tous les patients ont reçu du fentanyl ($10-16 \mu\text{g} \cdot \text{kg}^{-1}$). Les mesures hémodynamiques ont été réalisées en décubitus latéral avant, pendant et après hypotension. Une expansion volémique par gélatine et plasma frais congelé a été conduite pendant l'étude pour maintenir la pression capillaire pulmonaire entre 7 et 9 mmHg. La baisse de PAM a été la même dans les deux groupes de même que les volumes sanguins transfusés (500 ml en moyenne pour la période périopératoire) et les hématocrites postopératoires. L'expansion volémique périopératoire globale a été plus élevée ($p < 0.01$) dans le groupe I (2500 ml en moyenne) que dans le groupe II (1300 ml en moyenne). Le secteur capacitif a été plus touché par le NPS que par l'ISO. L'ISO a diminué les résistances vasculaires systémiques et la consommation d'oxygène ($\dot{V}\text{O}_2$) sans modification de l'index cardiaque (IC) et du shunt intrapulmonaire ($\dot{Q}_s/\dot{Q}_t$), contrairement au SNP qui a augmenté IC, $\dot{V}\text{O}_2$ et $\dot{Q}_s/\dot{Q}_t$.