Deliberate Hypotension Induced by Epidural Anesthesia

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Deliberate hypotension is thought to provide the reduction of intraoperative blood loss and transfusion-related morbidity[1]. Though several intravenous vasodilators are used to induce hypotension[2–4], few attempts have been made to evaluate the clinical efficacy and safety of deliberate hypotension with epidural anesthesia. Epidural anesthesia is a useful technique to lower blood pressure. In addition, it has been shown to maintain circulatory stability and exert favorable results on postoperative outcome[5]. The purpose of this study is to elucidate the effects and safety of deliberate hypotension induced by epidural anesthesia.

Methods

Twenty patients undergoing rotatory acetabular osteotomy were investigated after obtaining the informed consent. All patients were without cardiovascular disorder. No patient had hepatorenal dysfunction and coagulopathy in preoperative screening tests. The effects on blood loss, urine output, and postoperative hepatorenal function were evaluated in the following two types of mean arterial pressure (MAP) management; normotension of 80 ~ 100 mmHg (control group: n=10) and deliberate hypotension of 55 ~ 65 mmHg, with epidural anesthesia (hypotensive group: n=10). All cases were operated by the same orthopedic surgeon.

Medications and feeding were discontinued 16h before the study. Intramuscular premedication with hydroxyzine 1 $mg \cdot kg^{-1}$ or midazolam 0.1 $mg \cdot kg^{-1}$ and atropine 0.1 $mg \cdot kg^{-1}$ was given 1h before anesthesia. The patients were administered lactated Ringer's solution 500 ml before the induction of anesthesia. After the patients' arrival to the operating room, radial artery and superior vena cava were cannulated for the monitoring of arterial and central venous pressure. Epidural catheter was inserted in the lumbar epidural space (L2–L3) of hypotensive group. Anesthesia was induced with thiamylal 4 ~ 6 $mg \cdot kg^{-1}$. Tracheal intubation was facilitated by succinylcholine 1 $mg \cdot kg^{-1}$ or vecuronium 0.15 ~ 0.2 $mg \cdot kg^{-1}$. Anesthesia was maintained with oxygen 40%, nitrous oxide 60% and enflurane 1 ~ 2% in the control group, and oxygen 40%, nitrous oxide 60% and fentanyl

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$3 \sim 5$ $\mu g \cdot kg^{-1} \cdot h^{-1}$ in the hypotensive group. All patients were paralyzed with pancuronium $0.1 \sim 0.2$ $mg \cdot kg^{-1}$ or vecuronium $0.2 \sim 0.3$ $mg \cdot kg^{-1}$. Ventilation was controlled and adjusted to maintain arterial CO_2 tension between 30 and 40 mmHg. The hypotensive group was given 15 ml of 2% lidocaine into the epidural space as an initial dose. When MAP was more than 70 mmHg at 15 minutes after initial epidural injection, 5 ml of 2% lidocaine was administered as the additional dose. The hypotensive group was administered $5 \sim 8$ ml of 2% lidocaine epidurally every 45 to 60 minutes to maintain hypotension during the operation. The transducer was positioned at the level of spinous process of middle thoracic spine during the operation because the patients were in the lateral position. Central venous pressure was maintained more than 6 mmHg with continuous infusion of lactated Ringer's solution in both groups. Blood loss was replaced by 70% volume of transfusion following the establishment of acetabular osteotomy. Arterial sample was taken 90 minutes after the induction of hypotension for measurement of serum lidocaine concentration. Venous blood was sampled before and 10 days after operation to measure total protein, serum albumin, total bilirubin, GOT, GPT, Al-P, LDH, BUN, creatinine, Na, K and Cl.

Data are presented as mean ± SD. Possible differences between the obtained values were assessed with factorial ANOVA or χ^2 analysis when appropriate. The level of significance was set at $P < 0.05$.

Results

Two cases in the control group were given propranolol 0.1 mg or trinitroglycerin 6.8 mg to control MAP. In the hypotensive group, two of ten cases were given ephedrine 4 mg because patients' MAP decreased less than 50 mmHg following the initial epidural injection. These four cases were excluded from the following evaluations except for the plasma concentration of lidocaine.

There were no significant differences in patients' age (control group *vs.* hypotensive group: 31 ± 6 *vs.* 34 ± 8 yr.), height (153 ± 9 *vs.* 149 ± 8 cm), weight (48 ± 9 *vs.* 53 ± 7 kg), sex (1/7 *vs.* 0/8, male/female), the duration of operation (166 ± 32 *vs.* 153 ± 14 min.) and the duration of anesthesia (236 ± 28 *vs.* 236 ± 11 min.) between the groups. Deliberate hypotension less than 65 mmHg was induced within 30 min following the initial injection of lidocaine and was sustained for 148 ± 18 minutes. Arterial pressure of the hypotensive group was significantly lower than that of the control group for 90 minutes during the surgery. Heart rate of the hypotensive group significantly changed relative to the control group for 150 min during surgery (table 1). Lidocaine required for deliberate hypotension was 39.5 ± 6.2 ml as a total dose. Mean plasma concentration of lidocaine was 2.8 ± 1.4 $\mu g \cdot ml^{-1}$ ($0.9 \sim 5.7$ $\mu g \cdot ml^{-1}$).

Intraoperative blood loss of the hypotensive group was significantly less than that of control group (1018 ± 267 *vs.* 745 ± 199 ml). Intraoperative bleeding rate, which was calculated by the blood loss (ml) per operation time (min), was significantly less in the hypotensive group as well (6.2 ± 1.3 *vs.* 4.8 ± 1.1 $ml \cdot min^{-1}$). The volume of blood transfusion was greater in the control group. In contrast, the volume of infusion (2581 ± 767 *vs.* 2931 ± 408 $ml \cdot min^{-1}$) and urine output (299 ± 213 *vs.* 492 ± 270 $ml \cdot min^{-1}$) were greater in the hypotensive group. However, there was no statistical significance in the difference of these values. Postoperative hemoglobin of both groups was almost equivalent (9.9 ± 1.3 *vs.* 9.2 ± 1.3 $g \cdot dl^{-1}$).

Table 1. Changes in mean arterial pressure (MAP), heart rate (HR), and central venous pressure (CVP) during operation

	Post-intubation	Post-incision	30 min	60 min	90 min	120 min	Post-operation
MAP (mmHg)							
Normotensive (n=8)	90 ± 8	71 ± 7	75 ± 10	70 ± 9	72 ± 12	71 ± 10	78 ± 12
Hypotensive (n=8)	84 ± 9	61 ± 6*	59 ± 6*	57 ± 4*	57 ± 6*	62 ± 5	84 ± 10
HR (bpm)							
Normotensive	82 ± 14	91 ± 15	92 ± 17	95 ± 19	89 ± 18	88 ± 19	92 ± 20
Hypotensive	70 ± 8	61 ± 5*	60 ± 6*	59 ± 6*	59 ± 6*	60 ± 6*	85 ± 13
CVP (mmHg)							
Normotensive	13 ± 4	11 ± 3	7 ± 2	7 ± 3	6 ± 2	6 ± 2	8 ± 3
Hypotensive	12 ± 4	10 ± 2	8 ± 2	8 ± 3	7 ± 2	7 ± 2	10 ± 4

Values represent mean ± SD

*$P < 0.05$ compared with the value of control group

There were significant increases of postoperative GPT, Al-P and LDH (24.4 ± 9.7, 184.9 ± 84.9 and 333.1 ± 21.9 IU, respectively) in the control group compared with their preoperative values (9.0 ± 5.4, 119.1 ± 50.9 and 255.1 ± 21.9 IU, respectively). Postoperative Al-P of the hypotensive group (136.3 ± 44.1 IU) also revealed a significant increase compared with the preoperative value (89.2 ± 26.2 IU). In the hypotensive group, mean values of postoperative GPT (74.2 ± 146.1 IU) and LDH (551.8 ± 482.7 IU) were greater than normal ranges because one case had transient aggravation of GOT, GPT, Al-P and LDH (127, 423, 162 and 1524 IU, respectively) postoperatively. However, the increase of those enzymes had no significant difference compared with preoperative values. There was no significant difference of each value between the groups. The recovery from anesthesia was prompt in all patients. No patient revealed neurological deficit or significant ST change on electrocardiogram. All patients discharged without complications caused by anesthesia.

Discussion

The cardiovascular effects of epidural anesthesia are well defined and minimal in healthy volunteers[6]. However, the efficacy of deliberate hypotension with epidural anesthesia on blood loss during surgery has not been established. In general, stable hemodynamics by epidural anesthesia is likely to result in less blood loss during operation. However, previous reports have revealed controversial effects of epidural anesthesia on intraoperative blood loss during joint surgery[7–9]. Venous dilatation by sympathetic block is a possible explanation of increased bleeding during epidural anesthesia[9]. However, these reports described little about intraoperative arterial pressure. In our study, arterial pressure was controlled within the proposed range. Moreover, we restricted the operation and surgeons to exclude the influences of operation time or skillfulness on intraoperative blood loss. Our study suggests that lower intraoperative arterial pressure caused by epidural anesthesia is a possible reason for less bleeding.

The block of sympathetic outflow may impair autonomic responses against hypovolemia or hypotension and is likely to deteriorate hypotension[10,11]. In our study, two of 10 cases were administered ephedrine

4 mg because of marked hypotension with epidural anesthesia before the skin incision. However, no more doses of ephedrine and other inotropes were necessary to control MAP within the intended value. There was no episode of critical hypotension during the operation otherwise and MAP was stable and easily maintained. Recently, epidural anesthesia has been applied even for high-risk patients and has improved the overall outcome[5]. It is well known that epidural anesthesia suppresses endocrine responses during surgery compared with general anesthesia[12,13]. From this point of view, epidural anesthesia may be another useful method to achieve deliberate hypotension. However, the reduction of cardiac output by high levels of epidural anesthesia can attenuate organ perfusion[14]. In the present study, urine output of hypotensive group was tended to be greater than that of control group, whereas there was no significant difference of fluid infusion and blood transfusion between the groups. These results suggest that hypotensive epidural anesthesia produced no adverse effects on urine output. Postoperative laboratory tests revealed liver dysfunction in one case of hypotensive group. However, the liver function of this case returned to normal ranges within 1 month, and the mean values of laboratory tests revealed no significant changes. We could not prove the cause of liver dysfunction conclusively because several treatments such as antibiotics and blood transfusion may worsen liver function postoperatively. Thus, we conclude that the reduction of hepatorenal perfusion seems to be permissible under hypotension, MAP of 60 mmHg or less, with epidural anesthesia when patients are without cardiovascular complication. However, we should consider that the safety of hypotension induced by epidural anesthesia has not been established in elderly or complicated cases.

In summary, deliberate hypotension with epidural anesthesia significantly reduced intraoperative blood loss. MAP was easily controlled by epidural anesthesia. No serious side effects were observed in the hypotensive group during the study. Regarding the favorable outcome of epidural anesthesia, deliberate hypotension with epidural anesthesia may be a beneficial technique to reduce the risks of massive bleeding during operation.

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