J Anesth (2007) 21:317–319
DOI 10.1007/s00540-006-0525-3

Journal of
Anesthesia

Changes in percutaneous oxygen tension induced by spinal anesthesia

Tomoki Nishiyama

Department of Anesthesiology, The University of Tokyo, Faculty of Medicine, 7-3-1 Hongo, Bunkyo-ku, Tokyo 113-8655, Japan

Abstract
Purpose. To check the level of spinal anesthesia is sometimes difficult in patients with dementia. In spinal anesthesia, peripheral oxygen tension may increase in the anesthetized area because of vasodilatation due to sympathetic block. The purpose of this study was to determine whether changes in percutaneous oxygen tension ($tcPO_2$) were suitable for checking the level of spinal anesthesia.
Methods. Thirty patients, aged 40 to 70 years, scheduled for surgery of the lower extremities under spinal anesthesia, were enrolled. Spinal anesthesia was performed at L4-5 with hyperbaric 0.5% tetracaine 10 to 12 mg, administered with the patient in the lateral position; the patients were then immediately returned to the supine position. The anesthesia level was checked by cold test 10 min after the spinal anesthesia, and it was confirmed that the upper level was between T3 and T11. Then oxygen 6 l·min^{-1} was administered by mask. Six electrodes of a $tcPO_2$ monitor, (TCM 400) were attached before anesthesia, three electrodes at the right, center, and left side of the T3 level, and the other three at the right, center, and left at the T11 level. $TcPO_2$ was measured before and 10 min after spinal anesthesia, and 5 min after starting oxygen inhalation.
Results. $TcPO_2$ increased significantly after spinal anesthesia only at T11, and was increased by oxygen administration at both T3 and T11. The increase of $tcPO_2$ after oxygen administration was larger at T3 than T11, without any differences in absolute values.
Conclusion. Measurement of $TcPO_2$ might be useful as one of the objective methods to distinguish anesthetized and nonanesthetized areas in spinal anesthesia.

Key words Spinal anesthesia · Anesthesia level · Percutaneous oxygen tension · Cold test

Address correspondence to: T. Nishiyama, 3-2-6-603, Kawaguchi, Kawaguchi 332-0015, Japan
This study was performed at Ofuna Chuo Hospital.
Received: October 12, 2006 / Accepted: March 15, 2006

Introduction

The level of spinal anesthesia is usually checked with a pain test (pin-prick test) or a cold-sensation test (cold test). However, these subjective methods are not useful for patients with dementia. In such patients, objective methods to measure blood flow or skin temperature changes induced by sympathetic blockade, using pulse wave [1], laser Doppler flowmetry [2], thermography [2], or various thermometers [3,4] have been investigated. These methods were able to detect changes in parameters induced by spinal anesthesia, but there were no definite criteria to distinguish anesthetized and nonanesthetized areas. We hypothesized that vasodilatation by sympathetic block in the spinally anesthetized area might increase percutaneous oxygen tension ($tcPO_2$) and that this could be another objective method to differentiate anesthetized and nonanesthetized areas in spinal anesthesia. Therefore, in the present study, to confirm this hypothesis, changes in $tcPO_2$ in anesthetized and nonanesthetized areas were compared.

Patients, materials, and methods

After obtaining institutional approval and informed consent, 30 patients, aged 40 to 70 years, scheduled for orthopedic surgery of the lower extremities under spinal anesthesia were enrolled in this study. Those who were obese and those with diabetes mellitus and cardiovascular or respiratory disease were excluded.

Midazolam 2–4 mg was intramuscularly administered as a premedication 15 to 30 min before the patient entered the operating room. Six electrodes of a $tcPO_2$ monitor (TCM 400; Radiometer, Copenhagen, Denmark), heated at 44°C, were attached as follows: three electrodes were placed on the right, center, and left side of the upper chest (T3 level) where spinal anesthesia could not be effective, and the other three were

placed on the right, center, and left under the navel (T11 level), where anesthesia had to be effective. After epidural catheter insertion at L1-2 or L2-3, to be used for postoperative analgesia, spinal anesthesia was performed at L4-5 with hyperbaric 0.5% tetracaine 10 to 12 mg, with the patient in the lateral position. Then the patient was immediately returned to the supine position. Anesthesia level was checked by cold test 10 min after spinal anesthesia, and it was confirmed that the level was between T3 and T11. Then oxygen, at 6 l·min^{-1}, was administered by mask. $TcPO_2$ was measured before spinal anesthesia, 10 min after anesthesia (before oxygen administration), and 5 min after starting oxygen inhalation. After the surgery, the anesthesia level was checked again to confirm that the level was between T3 and T11. Blood pressure and heart rate were also monitored.

Data values are shown as means ± SD or with ranges. Data values in the nonanesthetized area (mean values for right, center, and left at T3) were compared with those in the anesthetized area (mean values for right, center, and left at T11) using two-way repeated measures analysis of variance, followed by the Student Newman-Keuls test as a post-hoc test. A P value less than 0.05 was considered statistically significant.

Results

Patients were aged 60 years (range, 40–69 years); 18 were male and 12, female and body weight was 59.3 kg (range, 42.5–70.6 kg). Open reduction of fracture was performed in 12 patients and total knee replacement was performed in 18 patients. Duration of surgery was 119 min (88–154 min). Anesthesia level showed no differences between the right side and left side in any patient, and was T6 in 12 patients, T7 in 9 patients, T8 in 5 patients, and T9 in 4 patients. Blood pressure decreased significantly 10 min after spinal anesthesia, but no treatment was necessary. Heart rate did not change significantly.

$TcPO_2$ increased significantly after spinal anesthesia only at T11, and was increased by oxygen administration at both T3 and T11 (Fig. 1). The increase of $tcPO_2$ after oxygen administration was larger at T3 than at T11, while the absolute $tcPO_2$ values were not different between T3 and T11. No patients showed a decrease in $tcPO_2$ at T3 after anesthesia and none showed large changes at T11.

Discussion

$TcPO_2$ was increased in the anesthetized area by spinal anesthesia. After oxygen administration, the increase in $tcPO_2$ was larger in the nonanesthetized area, although

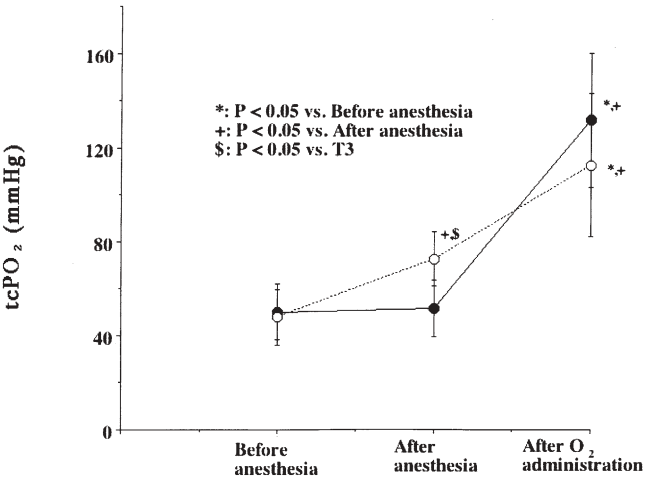

Fig. 1. Percutaneous oxygen tension ($tcPO_2$) in anesthetized (*open circles*; T11) and nonanesthetized (*closed circles*; T3) areas *Bars* indicate SD

the absolute values were not different between the anesthetized and nonanesthetized areas.

To judge the level of spinal anesthesia objectively, changes in blood flow or skin temperature have been used as an indicator of sympathetic block. During spinal anesthesia, a significant reduction in skin blood flow was obtained in the shoulder and chest, while an increase in skin blood flow was seen in the lower part of the body [5]. The most cephalad dermatome at which skin temperature elevation occurred showed the upper limit of diminished sympathetic activity in spinal anesthesia [6]. The sympathetic block might be far below the upper level of analgesia, and the duration of the sympathetic block was found to be far shorter than the duration of analgesia and motor block [5,7,8]. Preganglionic sympathetic B-fibers are more difficult to block by tetracaine or bupivacaine than A-fibers [9]. Therefore, checking the sympathetic block might be an alternative way to check the analgesia level, because at least where the sympathetic block is observed, there should be sensory block.

However, the contrary has been reported, in that, during spinal anesthesia, the level of sympathetic block lies cephalad to that of sensory block [6,10]. It has been shown that B-fibers are blocked at lower concentrations of local anesthetics than A-, and C-fibers; therefore, the level of sympathetic denervation during spinal anesthesia extends further cephalad than the level of analgesia or motor block [11]. Thus, it is still controversial whether sympathetic block or sensory block is greater in spinal anesthesia. The present study could not detect the precise upper level of sympathetic block by changes in $tcPO_2$ because of the limited number of electrodes. In our 12 patients with an anesthesia level of T6, the sympathetic block may have been as high as T3, and in the

4 patients with a level of T9, the sympathetic block may have been lower than T11, considered in the light of the above controversial reports [5–8,10,11]. One of the reasons that we could not obtain a clear line of $tcPO_2$ values to distinguish anesthetized and nonanesthetized areas, may have been the large SD. We could not find any relation between the anesthesia level and absolute values of $tcPO_2$. Therefore, it may be impossible to draw a line for the absolute value of $tcPO_2$ to judge the level of spinal anesthesia. According to the results of the present study, it seems that at least two measurements should be performed simultaneously to distinguish anesthetized and nonanesthetized areas; one electrode should be attached at the upper limit to be anesthetized and another electrode should be in an area fara way from the anesthetized area. Then changes in $tcPO_2$ at the two electrodes should be compared after spinal anesthesia, and, further, after O_2 administration.

Oxygen delivery, local metabolic conditions, and diffusion across the skin all determine $tcPO_2$ [12]. At the standardized electrode temperature of 44°C, there is important local vasodilatation [13]. Sympathetic tone is well known to influence $tcPO_2$ [14]. Increased sympathetic activity in surgery causes cutaneous vasoconstriction, thereby decreasing skin blood flow and $tcPO_2$. Sympathectomy induced by brachial plexus block increased $tcPO_2$. It was reported that, during hyperbaric oxygen exposure, $tcPO_2$ was substantially and significantly higher after sympathectomy compared to the control without sympathectomy [15]. However, that report is different from the present results, in which $tcPO_2$ increased significantly in the anesthetized area in room air, and the administration of oxygen induced a larger increase in $tcPO_2$ in the nonanesthetized area than in the anesthetized area. However, after oxygen administration, the absolute $tcPO_2$ value was not different between the anesthetized and the nonanesthetized areas. Brachial plexus block cannot always fully block sympathetic activity, while spinal block at a level higher than T9 could completely block lumbar sympathetic activity. Therefore, $tcPO_2$ increased significantly after spinal block, while it did not increase after brachial plexus block in room air. After oxygen administration, $tcPO_2$ should be higher in an area with sympathectomy than in an area without sympathectomy [15]. In the study by Thomas et al. [15], hyperbaric oxygen appeared to induce vasoconstriction, which was effectively inhibited by brachial plexus block, while without any block, vasoconstriction inhibited the increase in $tcPO_2$. In the anesthetized area in the present study, $tcPO_2$ was already increased by sympathectomy in room air, which resulted in a smaller further increase in $tcPO_2$ by oxygenation compared with that in the nonanesthetized area.

In conclusion, $tcPO_2$ might be useful as one of the objective methods to distinguish anesthetized and nonanesthetized areas in spinal anesthesia.

References

1. Meijer J, de Lange JJ, Ros HH (1988) Skin pulse wave monitoring during lumbar epidural and spinal anesthesia. Anesth Analg 67:356–359
2. Bengtsson M (1984) Changes in skin blood flow and temperature during spinal analgesia evaluated by laser Doppler flowmetry and infrared thermography. Acta Anaesthesiol Scand 28:625–630
3. Itoh Y, Yamakage M, Nakayama Y, Kanada Y, Chen X, Namiki A (2000) Estimation of the analgesic level of spinal anesthesia by measuring the skin temperature using an infrared thermometer (in Japanese with English abstract). Rinsho Masui Gakkaishi (Journal of the Japan Society of Clinical Anesthesia) 20:372–377
4. Satoh K, Ohe Y, Hayashida M, Fukuda T, Hasegawa R, Furuya Y (1993) Evaluation of skin temperature of patients under spinal anesthesia during cesarean section (in Japanese with English abstract). Masui (Jpn J Anesthesiol) 42:1148–1152
5. Bengtsson M, Nilsson GE, Lofstrom JB (1983) The effect of spinal analgesia on skin blood flow, evaluated by laser Doppler flowmetry. Acta Anaesthesiol Scand 27:206–210
6. Chamberlain DP, Chir B, Chamberlain BDL (1986) Changes in the skin temperature of the trunk and their relationship to sympathetic blockade during spinal anesthesia. Anesthesiology 65: 139–143
7. Bengtsson M, Lofstrom JB, Malmqvist LA (1985) Skin conductance responses during spinal analgesia. Acta Anaesthesiol Scand 29:67–71
8. Lundin S, Wallin BG, Elam M (1989) Intraneural recording of muscle sympathetic activity during epidural anesthesia in humans. Anesth Analg 69:788–793
9. Gissen AJ, Covino BG, Gregus J (1980) Differential sensitivities of mammalian nerve fibers to local anesthetic agents. Anesthesiology 53:467–474
10. Fink BR (1989) Mechanisms of differential axial blockade in epidural and subarachnoid anesthesia. Anesthesiology 70:851–858
11. Fink BR, Cairns AM (1983) Differential peripheral axon block with lidocaine: unit studies in the cervical vagus nerve. Anesthesiology 59:182–186
12. Byrne P, Provan JL, Ameli FM, Jones DP (1984) The use of transcutaneous oxygen tension measurements in the diagnosis of peripheral vascular insufficiency. Ann Surg 200:155–165
13. Huch R, Huch A, Lubbers HU (1973) Transcutaneous measurement of blood PO_2 ($tcPO_2$). Method and application in perinatal medicine. J Perinat Med 1:183–191
14. Rooke T, Hollier L, Osmundson P (1987) The influence of sympathetic nerves on transcutaneous oxygen tension in normal and ischemic lower extremities. Angiology 38:400–410
15. Thomas PS, Hakim TS, Trang LQ, Hosain SI, Camporesi EM (1999) The synergistic effect of sympathectomy and hyperbaric oxygen exposure on transcutaneous PO_2 in healthy volunteers. Anesth Analg 88:67–71