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Effects of spinal anesthesia on the peripheral and deep core temperature in elderly diabetic patients undergoing urological surgery

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Abstract
Purpose. The effects of spinal anesthesia on temperature homeostasis have been well studied, but whether body temperature during spinal anesthesia exhibits the same characteristic changes in patients with diabetes mellitus (DM) has not been clarified. The present study measured body temperatures at the forehead and at the lower limb using a monitor of deep body temperature and compared patients with DM ($n = 8$) and without DM ($n = 10$).
Methods. Subjects comprised 18 male patients (ASA physical status I or II) undergoing spinal anesthesia for urological surgery. Changes in deep body temperatures were measured using a Coretemp "deep-tissue" thermometer.
Results. Although the forehead temperature decreased slightly in both groups after spinal anesthesia, no significant differences were noted between groups. Conversely, although the foot temperature was elevated in both groups, temperature increases were smaller in DM patients (4.0° ± 0.3°C) than in controls (4.9° ± 0.6°C). Moreover, longer times were required to display increases of 1°C and 2°C for patients with DM (1°C: 19.1 ± 4.0 min; 2°C: 25.1 ± 4.2 min) compared with controls (1°C: 9.6 ± 1.3 min; 2°C: 13.1 ± 1.5 min).
Conclusion. These data suggest that body temperature changes in patients with DM during spinal anesthesia are different from those of control patients, probably due to disorders of the vascular response.

Key words Body temperature · Spinal anesthesia · Diabetes mellitus

Introduction

The effects of spinal anesthesia on temperature homeostasis have been extensively studied. Spinal anesthesia is known to significantly impair thermoregulation by inhibiting vasomotor and shivering responses and by redistributing heat from the core of the body to peripheral tissues [1–3].

Patients with long-term diabetes mellitus (DM) develop various complications of neuropathy, particularly disorders of microvascular function [4] and autonomic nervous activity [5]. Patients with DM may thus be expected to display different vascular responses and changes in body temperature of the lower limb during spinal anesthesia. The present study examined diabetic patients to determine if changes in body temperature might differ from those in nondiabetic patients during spinal anesthesia.

Frank et al. reported that one of the risk factors for hypothermia after spinal anesthesia, other than high spinal blockade level, is advanced age [6,7]. Commonly, patients who undergo urological surgery include a high proportion of elderly individuals. We thus selected those patients for this study and also investigated whether hypothermia was induced.

For these purposes, a CM-210 deep body temperature monitor (Terumo, Tokyo, Japan) was used to measure the body temperature at both the foot and the forehead, and results were compared between patients with and without DM.

Patients and methods

After receiving institutional approval, written informed consent was obtained from 18 male patients scheduled to undergo urological surgery. All patients were classified as ASA physical status I or II. The mean age was 67.6 ± 1.8 years (range, 51–80 years). Patients were assigned into two groups: a diabetes group ($n = 8$) and a control group ($n = 10$). The diabetes group comprised patients whose medical records showed a diagnosis of type 2 diabetes and who displayed hemoglobin $A1_C$

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levels greater than 7.0%. All patients in the diabetes group had been receiving medical treatments such as administration of insulin or hypoglycemic drugs or/and dietary therapy.

No premedication was administered before surgery. The ambient operating room temperature and humidity were maintained close to 23°C and 30%, respectively, during the study period. An intravenous catheter was inserted into the antecubital vein on the left arm and acetated Ringer's solution was infused. Those solutions were preheated to 37°C in a warmed cabinet and administered without in-line warming. Patients were covered with a sheet, but passive cooling was allowed. The body core temperature was measured using a CM-210 deep body temperature monitor (Terumo). The forehead temperature measured using this device correlates well with the deep body core, rectal, and pulmonary artery temperatures [8,9]. Probes were placed on the forehead and the sole of the left foot. Measured temperatures were recorded every 30 s and the time courses of increases in body temperature of the foot after spinal anesthesia were examined. For this purpose, ten temperature measurements were taken in the 5-min period before spinal anesthesia and were averaged to give the baseline temperature, then the time required for the temperature to increase by 1°C and 2°C compared to the baseline was measured as shown in Fig. 2 (upper panel). Before spinal anesthesia, a significantly lower body temperature due to vascular dysfunction was not observed in any patient. Spinal anesthesia was performed at the L3–L4 or L4–L5 interspace in the lateral position using a 23-gauge Quincke (Terumo) spinal needle to inject 12–12.5 mg of hyperbaric 0.5% bupivacaine. Patients were then immediately placed in a supine position. Body sensory block levels were evaluated 5 min after spinal block and after surgery by response to a cold sensation and the pin-prick test. Blood pressure, heart rate, and oxygen saturation were recorded at 5-min intervals during the study period. Hypotension (systolic blood pressure < 80 mmHg) and bradycardia (heart rate < 50 beats/min) were treated using 4 mg ephedrine and 0.5 mg atropine bolus doses, respectively, repeated as needed.

Data are expressed as mean ± SEM. Data were compared using an unpaired t test or the Mann-Whitney test, with values of $P < 0.05$ considered statistically significant.

Results

The demographic data of the patients are given in Table 1. No significant differences in patient size, age, operation time, dose of bupivacaine for spinal anesthesia, or sensory block level were noted between the diabetes and control groups.

Before spinal anesthesia, the foot temperature was lower than the forehead temperature in every patient. In all patients in the control group, the foot temperature dramatically increased after spinal anesthesia and became almost equal to the body temperature as measured at the forehead (Fig. 1A). In contrast, in the diabetes group, the foot temperature exhibited a slow onset of increase and a lower final temperature compared to the forehead temperature in many patients (Fig. 1B). Consequently, although no significant differences between groups in forehead or foot temperatures were identified before spinal anesthesia, the foot

Table 1. Demographic data from control patients and patients with diabetes undergoing urological surgery

Parameter	Control	Diabetes
Number of patients	10	8
Age (years)	66 ± 3	70 ± 2
Weight (kg)	66 ± 4	64 ± 4
Height (cm)	164 ± 2	167 ± 2
Operation time (min)	35 ± 5	45 ± 11
Dose of bupivacaine (mg)	12.1 ± 0.1	12.1 ± 0.1
Block height		
5 min	Th 10	Th 11
Final	Th 7	Th 7

Data are mean ± SEM

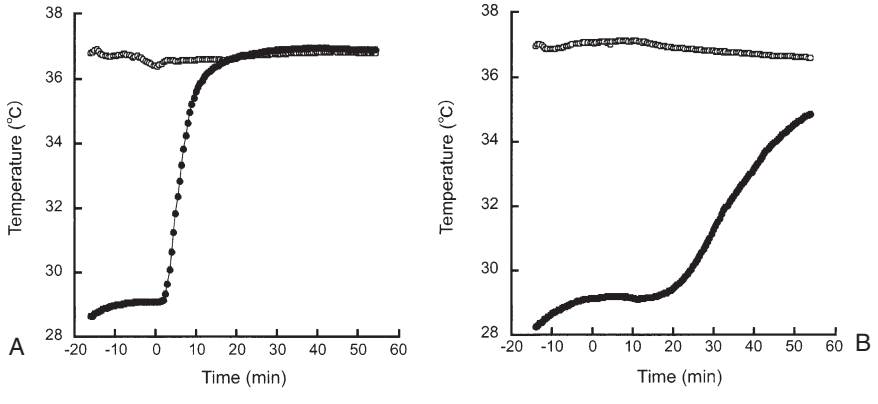

Fig. 1. Representative data of forehead and foot temperatures in a control patient (**A**) and a patient with diabetes (**B**). *Open circles* and *filled circles* indicate instantaneous temperatures of the forehead and foot, respectively. Induction of spinal anesthesia is identified as elapsed time zero

Table 2. Changes in forehead and foot temperatures (°C) on spinal anesthesia in control patients and patients with diabetes

Location	Control	Diabetes
Foot		
Before block	31.0 ± 0.4	30.5 ± 0.5
Final	35.8 ± 0.2	34.6 ± 0.5*
Difference	4.9 ± 0.6	4.0 ± 0.3
Forehead		
Before block	36.3 ± 0.2	36.1 ± 0.2
Final	36.1 ± 0.1	35.7 ± 0.2
Difference	–0.2 ± 0.1	–0.4 ± 0.1

Data are mean ± SEM
*$P < 0.05$ vs control group (unpaired t test)

temperature after spinal anesthesia was significantly lower in the diabetes group than in the control group ($P < 0.05$) (Table 2). Moreover, after comparing the time course of increases in foot temperature, we calculated the mean times for 1°C and 2°C increases in each group. Both mean times were longer in the diabetes group (1°C: 19.1 ± 4.0 min; 2°C: 25.1 ± 4.2 min) than in the control group (1°C: 9.6 ± 1.3 min; 2°C: 13.1 ± 1.5 min; $P < 0.05$ each) (Fig. 2).

Hypotension was observed in three patients in the diabetes group and bradycardia was also apparent in one patient in the diabetes group and two patients in the control group. In these cases, ephedrine and atropine were administrated, respectively. In just one patient in the control group, 15 mg of pentazocine was used for additional analgesia at almost the end of surgery. No obvious changes in body temperature were observed following the application of these drugs.

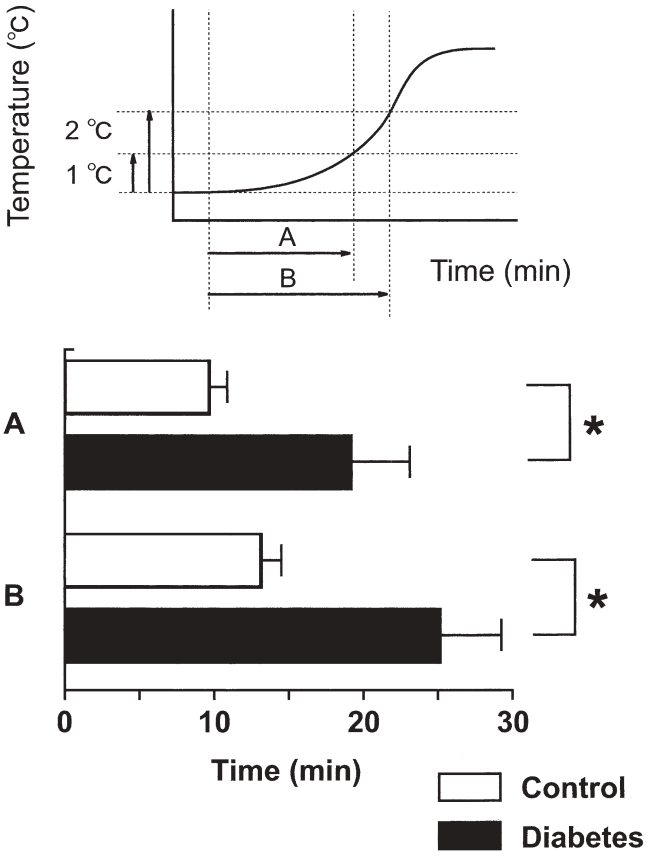

Fig. 2. Times required for the foot temperature to increase to 1°C and 2°C above the baseline after spinal anesthesia. The time required for the temperature to increase to 1°C and 2°C above the baseline was calculated as shown in *upper panel*. The results show the mean ± SEM. *$P < 0.05$, unpaired t test

Discussion

The principal finding in this study was that the onset of the increase in peripheral body temperature measured at the foot and the time courses of increases after spinal anesthesia were slower in patients with diabetes than in control patients.

The origin of such differences is obviously a key question. One possible mechanism may be deficient vascular responses in patients with DM during spinal anesthesia. Spinal anesthesia elicits a redistribution of central heat to the periphery, caused by vasodilation from sympathetic block [1–3]. In patients with diabetes, hyperglycemia leads to structural changes in endothelial cells and basement membrane thickening in the microvasculature. These changes thus reduce vasodilatory capacity and the ability of small vessels to autoregulate in the face of increased environmental demands under conditions such as infection or inflammation [4]. In the present study, no extremely low foot temperatures were observed before spinal anesthesia. Moreover, no patients exhibited diabetic foot ulcers. However, some patients in the diabetes group might have had some degree of subclinical angiopathy. Taken together, this seems to be the most likely cause of differences between patients with and without diabetes, with vascular disorder induced by hyperglycemia impairing the vasodilation of the lower limbs that should have been induced by spinal anesthesia.

Another possible mechanism is disorder of the autonomic nerves themselves in patients with diabetes. Orthostatic hypotension occurs in diabetes largely as a consequence of efferent sympathetic vasomotor denervation, causing reduced vasoconstriction of the splanchnic and other peripheral vascular beds [4,10]. Peripheral vascular beds of some patients in the diabetes group might thus have no capacity to dilate even after sympathetic block due to spinal anesthesia.

One of the risk factors for hypothermia after spinal anesthesia, other than high spinal blockade level, is advanced age [6,7]. Although the mean age of patients

in this study was relatively high (67.6 years), severe hypothermia (<35°C) was not observed in any patient during surgery. This may have resulted from the low average spinal blockade level in this study, as indicated by Frank et al. in previous reports [6,7].

Several studies in which ephedrine [11] and atropine [12] were systemically administered have displayed alterations to temperature homeostasis by cutaneous vasodilation. However, patients treated using these drugs in the present study exhibited no obvious changes in body temperature after administration, possibly due to the relatively low doses of the drugs used.

A possible limitation of the current study is that the relationship between the clinical stage of diabetes and the effects of spinal anesthesia on body temperature in patients with diabetes remains unclear. Further studies are needed to clarify this issue.

In summary, we showed that the increase in temperature of the lower limb after spinal anesthesia in patients with diabetes mellitus exhibited a significantly slower onset and time course that those seen in normal patients, probably due to disorders of the vascular response. Although severe hypothermia was not observed in any patients in this study, careful monitoring of body temperature should be performed in patients with diabetes mellitus during both spinal and general anesthesia.

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