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Limb tourniquet causes thermal perturbations under various types of anesthesia: a report of seven cases

TAKASHI AKATA, TOMOO KANNA, KAORU IZUMI, TARO NAGATA, and SHOSUKE TAKAHASHI

Department of Anesthesiology and Critical Care Medicine, Faculty of Medicine, Kyushu University, 3-1-1 Maidashi, Higashi-ku, Fukuoka 812-82, Japan

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Introduction

Pneumatic tourniquets are frequently used during limb operations to decrease blood loss and to provide a bloodless surgical field. Deflation of the limb tourniquet is known to cause a number of adverse systemic responses such as systemic hypotension, tachycardia, systemic acidosis with increases in arterial CO_2 pressure ($Paco_2$) and lactate, and decreases in arterial oxygen pressure (Pao_2) [1–4]. Although Bloch et al. previously demonstrated progressive increases in central temperature following tourniquet application in pediatric patients under general anesthesia [5], little information is available regarding changes in body temperature following tourniquet release. In this report of seven patients who underwent limb surgery under various types of anesthesia, we demonstrate gradual increases in both central and peripheral temperatures during tourniquet application, and progressive decreases in both central and peripheral temperatures following tourniquet release.

Case reports

We report one pediatric and six adult patients undergoing orthopedic surgery on either lower or upper extremities under either regional or general anesthesia. The patients' clinical profiles, preoperative diagnoses, proposed operations, and chosen anesthesia are summarized in Tables 1 and 2. Mepivacaine (1% or 2%) was exclusively used for epidural anesthesia, and the sensory blockade was maintained by additional epidural administration of mepivacaine (2%, 5–7 ml) every 45–50 min. In patient 3, unilateral spinal anesthesia using 0.3% dibucaine (1.5 ml) was additionally given to supplement the epidural anesthesia. In patients undergoing regional anesthesia, diazepam and/or pentazocine were intravenously given for sedation when necessary. All patients had unilateral limb tourniquets applied to facilitate the surgery. The tourniquets were serially applied in patients 3 and 5 (Table 2). The tourniquet pressures used for lower and upper limb surgery were 350–450 mmHg and 250 mmHg, respectively.

For intraoperative anesthetic management of all patients presented in this report, we followed the "Guideline for Intraoperative Monitoring for Anesthetic Safety" advocated by the Japan Society of Anesthesiology (April 21, 1993). In addition to heart rate (HR), blood pressure (BP), and arterial saturation of hemoglobin with oxygen measured by a pulse oximeter (Spo_2), both rectal and fingertip skin-surface temperatures were monitored in all patients. In patients undergoing general anesthesia, inspiratory and expiratory CO_2 concentrations were also monitored utilizing infrared capnography. In patients undergoing lower limb surgery, in addition to BP measured with the standard cuff method, tonometric BP was continuously monitored. The ambient temperature was thermostatically controlled at ≈25°C in our operating rooms. Body temperatures, HR, tonometric BP, end-tidal CO_2, and Spo_2 were recorded every 1 min by a computerized patient monitoring system, "System Q," developed by our institute. The monitoring sites of skin-surface temperature in each patient are summarized in Table 2. In patient 6, fingertip skin-surface temperature was monitored at the untourniqueted upper extremity where both motor and sensory blockades attributable to neck epidural anesthesia were observed.

Address correspondence to: T. Akata
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Table 1. Patients, profiles, preoperative diagnoses, and proposed operations

Case no.	Age (y.o.)	Sex (F/M)	Height (cm)	Weight (kg)	ASA class	Preoperative diagnosis	Proposed operation
1	47	F	151	51	I	rt. patella fracture	ORIF and arthroscopy
2	41	F	152	41.5	I	p/o rt. patella fracture	Removal of nails and arthroscopy
3	19	M	128	30	III[a]	rt. femoral bone fracture	ORIF
4	62	M	151	72	II[b]	rt. knee osteoarthritis	HTO and arthroscopy
5	51	F	152	48	II[c]	rt. wrist RA	Synovectomy and arthroplasty
6	21	M	172	74	I	lt. amputated thumb	Replantation
7	6	M	116	18	II[d]	rt. Brodie's abscess	Curettage

rt., right; lt., left; p/o, postoperative; RA, rheumatoid arthritis; ORIF, open reduction and internal fixation; HTO, high tibial osteotomy; ASA class, physical status classification of American Society of Anesthesiologists.
[a] Chronic renal failure with anemia, hypothyroidism with dwarfism, hypoparathyroidism with rickets.
[b] Hypertension.
[c] RA.
[d] Febrile convulsion.

Table 2. Anesthesia, tourniquet, and temperature measurement

Case no.	Anesthesia				Tourniquet		FSST monitoring
	Premedication	Induction	Maintenance	Blockade[a]	Sites	Time (min)	
1	Hydroxydine p. Atropine s.	—	Epi (L3/4)	T6	L	98	U
2	Nitrazepam Atropine s.	GOS slow	GO-Epi (L2/3)	T7	L	73	U
3	Hydroxydine p. Atropine s.	—	Epi + Sp (L3/4)	T4	L	88, 83[b]	U
4	Hydroxydine p. Atropine s.	Thiamylal Vecuronium	GOI	—	L	104	U
5	Nitrazepam Atropine s.	Thiamylal Vecuronium	GOS	—	U	87, 74[b]	L
6	Nil	—	Epi (C7/T1)	C5	U	28	U[c]
7	Nitrazepam Triclofos Na	Midazolam GOS slow Vecuronium	GOS	—	L	101	U

FSST, fingertip skin-surface temperature; Epi, epidural; Sp, spinal; G, nitrous oxide; O, oxygen; I, isoflurane; S, sevoflurane; L, lower limb; U, upper limb.
[a] The upper dermatomal level of sensory blockade.
[b] Serial application of tourniquets.
[c] Untourniqueted epidurally anesthetized upper limb.

Except for patient 3, who had chronic renal failure (CRF), ≈10–20 ml·kg^{-1} of balanced salt solution (unwarmed) was intravenously infused for the first 1 h (before tourniquet application). The infusion rate was then, reduced to ≈4–5 ml·kg^{-1}·h^{-1}. In patient 3, who had CRF and hypoalbuminemia, ≈400 ml of plasma protein fraction (PPF) and 150 ml of 50% saline solution were intravenously infused for the first 2 h after the patient entered the OR (before tourniquet application), and the PPF was subsequently infused at a rate of ≈3 ml·kg^{-1}·min^{-1}. Fingertip skin-surface temperature was monitored at the limb where the IV line was not placed except in patient 6.

As shown in the Fig. 1 and Table 3, increases in both rectal and skin-surface temperatures were observed during tourniquet application except in patient 6. In contrast, both rectal and skin-surface temperatures progressively decreased following tourniquet release. In patient 3, severe shivering was observed a few minutes after tourniquet release following rapid decreases in body temperature after deflation of the second tourniquet.

Table 3. Summary of changes in body temperature during application of and following deflation of limb tourniquet

Case no.		On tourniquet application (a)	On tourniquet release (b)	Δ Increases (b–a)	After tourniquet release			
					5 min	10 min	15 min	Δ max. decreases
1	Rectal	37.6	38.5	0.9	38.4	38.3	38.2	0.4
	Skin	30.0	37.7	7.7	36.2	35.0	33.7	4.3
2	Rectal	36.2	37.0	0.8	36.8	36.8	36.7	0.3
	Skin	35.7	36.4	0.7	35.4	35.6	35.6	1.0
3	Rectal	37.3	37.6	0.3	37.5	37.5	37.4	0.2
	Skin	35.0	36.6	1.6	35.6	34.7	34.2	2.8
	Rectal[a]	37.4	37.5	0.1	37.4	37.4	37.0	0.5
	Skin[a]	33.8	36.2	2.4	33.8	33.7	34.1	2.6
4	Rectal	36.4	36.5	0.1	36.5	36.4	36.4	0.1
	Skin	35.7	36.3	0.6	35.7	35.7	35.6	0.7
5	Rectal	36.5	36.8	0.3	36.7	36.6	36.6	0.2
	Skin	35.9	36.4	0.5	36.0	36.0	36.2	0.4
	Rectal[a]	36.6	36.8	0.2	36.7	36.6	36.5	0.1[b]
	Skin[a]	36.0	36.4	0.4	36.1	36.0	35.6	0.4[b]
6	Rectal	37.4	37.4	0	37.4	37.3	37.4	0.1
	Skin	35.4	35.0	−0.4	34.8	34.7	34.7	0.3
7	Rectal	36.3	37.8	1.5	37.8	37.6	37.6	0.2
	Skin	36.3	37.4	1.1	37.1	36.5	36.5	0.9

[a] Changes in body temperature during application of and after release of the second tourniquet.
[b] Maximal decreases in body temperature before discontinuation of general anesthetics.

Discussion

Pneumatic limb tourniquets appear to cause thermal perturbations under various types of anesthesia, i.e., inflation and deflation of the tourniquet appear to cause progressive increases and decreases in body temperature, respectively. While the changes in central/rectal and peripheral/skin-surface temperature occurred in the same direction in both situations, the changes in skin-surface temperature were much more distinct and larger than those in rectal temperature in both situations. We might have failed to detect these relatively modest thermal perturbations caused by tourniquet application or release without intensive monitoring of fingertip skin-surface temperature which is standard practice during anesthesia at our institute.

The gradual increases in rectal temperature observed during tourniquet application in our patients may be consistent with previous studies demonstrating progressive increases in central temperature during application of limb tourniquets in pediatric patients undergoing general anesthesia [5]. As Bloch et al. previously speculated, tourniquet-induced central hyperthermia might result from decreased effective heat loss through the distal skin and from the constraint of metabolic heat to the central thermal compartment [5]. In addition, tourniquet application after exsanguination with an Esmarch bandage would decrease the area for distribution of heat contained in blood, causing increases in heat distributed in the proximal area. Regional anesthesia is believed to interfere only modestly, but not strongly, with central control of thermoregulation by blocking thermal inputs and efferent responses, while general anesthesia has been proposed to increase the thresholds for active vasodilation by only ≈1°C [6–8]. Therefore, such possible alterations in kinetics or distribution of body heat following tourniquet application probably would lead to increases in mean body temperature, stimulating the thermoregulatory center to cause peripheral vasodilation to effectively dissipate heat even under general anesthesia.

The progressive decreases in both rectal and skin-surface temperature were observed following tourniquet release. The decreases in rectal temperature were relatively small (≈0.1–0.4°C), while the decreases in skin-surface temperature were much more distinct and larger (up to ≈4°C) as compared to those in rectal temperature. However, the skin-surface temperatures, which were rather high immediately before tourniquet release (i.e., ≈35.0–37.5°C under either anesthesia), finally reached to steady-state values of ≈33.5–34.5°C and ≈35.5–36.5°C under regional and general anesthesia, respectively, ≈10–20 min after tourniquet release. Both of these values appear to be normal values in both situations [7,9]. Therefore, we speculate that vasodilation had occurred during tourniquet application as a thermoregulatory response as discussed above, and that the decreased vascular tone at the time of tourniquet release might return to normal following tourniquet release as a result of discontinuation of thermoregulatory vasodilation. The decreases in the skin-surface temperature were larger in patients undergoing leg surgery

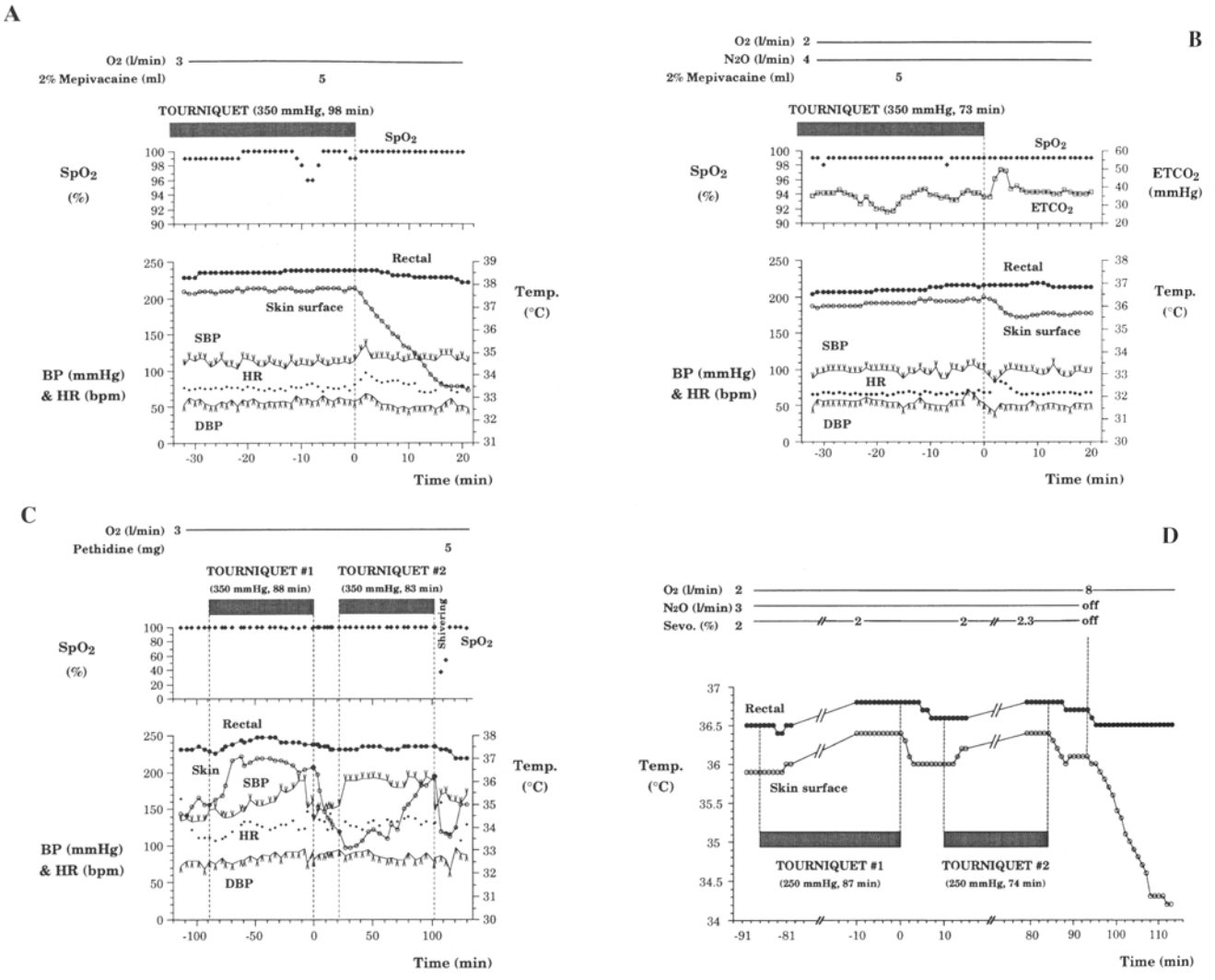

Fig. 1. Changes in both rectal (*closed circles*) and fingertip skin-surface (*open circles*) temperatures associated with the use of limb tourniquets in patients 1 (**A**), 2 (**B**), 3 (**C**), and 5 (**D**). **A** and **B** focus on the changes in body temperature following tourniquet release, while **C** and **D** demonstrate the effects of serial application of unilateral tourniquets on body temperature. Progressive increases and decreases in both rectal and fingertip skin-surface temperatures were observed following tourniquet application and release, respectively. Changes in skin-surface temperature were much larger compared to those in rectal temperature in both situations. Note the smaller decreases in skin-surface temperature following tourniquet release in the patient 2 (**B**) who underwent lower limb surgery under general anesthesia as compared to those in patient 1 undergoing comparable lover limb surgery under regional anesthesia. The changes in body temperature observed during application and following release of the first tourniquet were reproduced associated with the use of the second tourniquet in patients 3 (**C**) and 5 (**D**). Note emergence of severe shivering following the rapid decrease in skin-surface temperature after release of the second tourniquet in patient 3 (**C**). Note also further decreases in body temperature after discontinuation of inhalational anesthetics in patient 5 (**D**). Respiratory and hemodynamic data were omitted from **D** for clarity. *Spo_2*, arterial saturation of hemoglobin with oxygen measured by pulse oximetry; *BP*, blood pressure; *SBP*, systolic BP; *DBP*, diastolic BP; *HR*, heart rate; *Temp.*, body temperature; *Sevo.*, sevoflurane

under regional anesthesia (patients 1 and 3) as compared to patients undergoing comparable leg surgery under general anesthesia (patients 2 and 4) (Fig. 1, Table 3). Regional anesthesia has been reported to widen the interthreshold range only modestly to ≈0.6°C [8]; the interthreshold range was formed by temperatures that fall between the highest cold response and the lowest warm defense, and thus, in this thermoregulatory "null zone," neither cold nor warm responses occur [8]. On the other hand, general anesthesia is believed to widen the interthreshold range markedly by up to ≈4°C, shifting the thresholds for vasoconstriction down to ≈34.5°C (mean body temperature) [6,7]. Therefore, thermoregulatory vasoconstriction might possibly occur in our patients under regional anesthesia, leading to larger decreases in skin-surface temperature. In support

of this, shivering, a type of thermoregulatory cold responses, did appear following the progressive decrease in skin-surface temperature in patient 3 undergoing regional anesthesia (Fig. 1C). In addition, sympathetic stimulation in response to systemic hypotension or the efflux of ischemic products or hypothermic venous blood from the tourniqueted (ischemic/hypothermic) area into the systemic circulation at the time of tourniquet deflation might also possibly contribute to peripheral vasoconstriction. Ischemia, including tourniquet ischemia, has been suggested to stimulate production of endogenous vasoconstricting substances such as thromboxane A_2 or endothelin [10–16].

Skin-surface temperature did not increase, but rather decreased, during tourniquet application in patient 6, in whom skin-surface temperature was monitored at the upper extremity where both sensory and motor nerves were almost completely blocked in the presence of neck epidural anesthesia (Table 3). If the increases in skin-surface temperature observed in other patients had resulted from thermoregulatory vasodilation as discussed above, the blockade of efferent thermoregulatory responses caused by the epidural anesthesia might have prevented vasodilation, and infusion of unwarmed solutions from the same extremity might have resulted in the slight decreases in the skin-surface temperature. The observed small decreases in the skin-surface temperature following tourniquet release might suggest involvement of some mechanisms in the peripheral vasoconstriction other than activation of the sympathetic nervous system. However, since the tourniqueted area was small and the tourniquet time was relatively short in this patient (no. 6), the changes in body temperature are rather difficult to interpret; the tourniqueted area and time were too small and short, respectively, to cause distinct thermal perturbations in this patient. To clarify the underlying mechanisms behind the tourniquet-induced thermal perturbations, further investigations would be necessary, e.g., systematic comparison of the tourniquet-induced changes in temperature in patients receiving regional anesthesia with those in patients under general anesthesia (i.e., in the absence of thermoregulation), or of the changes in skin-surface temperature at epidurally anesthetized limbs with those at unanesthetized limbs.

Although intraoperative thermal perturbations have been suggested to cause a significant alteration in cardiovascular homeostasis and to increase cardiovascular mobility [6,17], the observed tourniquet-induced thermal perturbations were relatively modest and their clinical importance does not appear to have been immense. Indeed, no severe deteriorations in the cardiovascular system related to the thermal perturbations caused by tourniquet application or release were observed in our patients (Fig. 1A – C). Nevertheless, we did observe severe shivering with a concomitant decrease in skin-surface temperature following tourniquet release in patient 3, which hastened us to pharmacological intervention. We would suggest that careful monitoring of changes in body temperature during surgery requiring limb tourniquet would be useful to minimize the possible adverse effects of tourniquet-induced thermal perturbations in conjunction with other known adverse systemic responses caused by tourniquet application or release, particularly in patients at higher risk of cardiovascular morbidity or patients who are susceptible to thermal perturbations such as pediatric or aged patients.

In summary, we have demonstrated tourniquet-induced thermal perturbations in seven patients who underwent orthopedic surgery on either the lower or upper extremities with the aid of unilateral limb tourniquets under various types of anesthesia. Tourniquet application and release appear to cause progressive increases and decreases in body temperature, respectively. The precise mechanisms and clinical importance of those relatively modest thermal perturbations are currently unknown, and need to be further investigated.

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