vironment, peripheral vasodilatation, infusion of cold fluids, and anesthetic-induced inhibition of thermoregulation are the main causes of hypothermia during surgical procedures. In the past, surgical patients were commonly considered to be poikilothermic.[5] However, many studies have demonstrated that thermoregulation responses do exist if anesthetized patients become hypothermic.[6,19,20] The major effective thermoregulatory responses of the anesthetized and paralyzed hypothermic patients are nonshivering thermogenesis (increased metabolic heat production)[21] and cutaneous vasoconstriction (decreased environmental heat loss).[22] Only the factor of thermoregulatory vasoconstriction was evaluated in our study, because nonshivering thermogenesis is not considered to be important in adult humans and is difficult to evaluate.[6,21]

In this study, we demonstrated that without active warming, patients under midazolam-fentanyl-nitrous oxide anesthesia became hypothermic, and the thermoregulatory vasoconstriction threshold occurred around 35.2 °C. Once vasoconstriction occurred, the hypothermic patients reached a relatively stable thermal steady state which prevented deeper hypothermia from occurring (Fig. 1). Free flap surgery is time-consuming, superficial surgery with minimal blood loss, and takes several hours, which allows thermoregulatory vasoconstriction to occur. During midazolam-fentanyl-nitrous oxide anesthesia, the thermoregulatory vasoconstriction threshold (35.2 ± 0.7 °C) developed more rapidly and was higher than those reported with other intravenous anesthetics such as propofol-nitrous anesthesia[13] and fentanyl-nitrous anesthesia[11] (Table 2).

Hypothermic patients did not become hypertensive at the time of vasoconstriction. Thermoregulatory vasoconstriction is not a response of the entire vascular system.[6,23] Anatomic thermoregulatory arteriovenous shunts are located primarily in the fingers and toes; therefore, blood flow to distal extremities is affected more by thermoregulation than is flow to central skin.[18] So the skin-temperature gradient becomes significantly different between the fingers and forearm during thermoregulatory vasoconstriction. Fingertip temperature can range from near ambient (vasoconstricted) to near core temperature (vasodilated) because of the presence of thermoregulatory arteriovenous shunts and the wide range over which fingertip blood flow varies. Forearm skin does not possess arteriovenous shunts, therefore its temperature is less affected by the thermoregulatory status.[18]

Table 2. Historical Comparison of the Onset of Thermoregulatory Vasoconstriction and Core Temperature of the Thermoregulatory Threshold

	Thermoregulatory vasoconstriction (min)	Thermoregulatory threshold (°C)
Midazolam-N_2O ($n = 9$)	84 ± 42	35.2 ± 0.7 °C
Fentanyl-N_2O ($n = 6$)[11]	167 ± 33	34.2 ± 0.5 °C
Propofol-N_2O ($n = 9$)[13]	208 ± 43	33.6 ± 0.7 °C

Values are expressed as the mean ± SD.

In both groups, core temperatures decreased during the first 60 min of anesthesia (Figs. 1, 2). This initial decrease in core temperature after induction of general anesthesia probably resulted from central redistribution of cool peripheral blood.[24] It is also interesting that the skin-temperature gradient of nearly all of the patients widened in the initial 60 min of anesthesia (Fig. 4). This means that the skin temperature of the fingertip was warmer than that of the forearm during the phase of central redistribution. This could have been due to the fully dilated thermoregulatory arteriovenous shunts around the distal fingertips because of the dramatic change in the threshold of thermoregulatory vasoconstriction from the unanesthetized state (36.7 ± 0.3 °C)[25] to the anesthetized state.

When thermoregulatory vasoconstriction is triggered, the skin-temperature gradients usually steadily increase. However, some temporary reversals of the established skin-temperature gradient were noted in some patients in our study (Fig. 4), and this phenomenon also has been reported by others.[6] Temporary reversals may result from an increasing anesthetic dose, which presumably lowers the threshold. Other conceivable factors are temperature changes in other parts of the body which are not being recorded (e.g., those inside the surgical incision) that increase total thermoregulatory input to a level above that which would trigger vasoconstriction.[11] This observation provides further support for the use of skin temperature gradient measurements being a reliable index of vasoconstriction.

In conclusion, active thermoregulation occurred during midazolam-fentanyl-nitrous oxide anesthesia, which seemed to prevent a time-dependent continuous decline in body temperature and the development of deep hypother-