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Mechanism of Action of Nifedipine on Intraoperative Hypothermia

To the Editor:—Anesthesia increases cutaneous heat loss and decreases metabolic heat production. The core temperature especially decreases during the 1st hour of anesthesia. This decrease is explained by internal body heat redistribution and not only negative heat balance. Thus, Just *et al.* have shown that preinduction skin-surface warming reduces core-to-peripheral tissue temperature gradient and so reduces the body heat redistribution and the core temperature decrease after induction.[1] Peripheral heat storage capacity is also an important factor to be considered.[2] Thus, preinduction skin-surface warming reduces core-to-peripheral tissue temperature gradient but also increases peripheral heat storage capacity. Vassilieff *et al.* study the effect of nifedipine on body heat redistribution during the 1st hour of anesthesia.[3] A previous study had shown that patients who took nifedipine a few days before surgery exhibited a smaller decrease of core temperature after induction.[4] However, no explanation could be provided, because of the absence of mean skin temperature monitoring. The study by Vassilieff *et al.* confirms these results.[3] The authors advocate a decrease in core-to-peripheral temperature gradient. Nifedipine first increases cutaneous heat loss but leads to internal heat redistribution and promotes a new thermal equilibrium. The data of Vassilieff *et al.* surprisingly do not show a significant difference between initial mean skin temperature and initial core temperature between patients taking nifedipine and control subjects. An alternative mechanism to the intraoperative preservation of core temperature may be a change in the volume of the peripheral compartment induced by vasodilatation, which then would increase the heat storage capacity without significant mean cutaneous temperature variation. Should this assumption be verified, the primary mechanism would be an increase of peripheral heat storage instead of a change in temperature gradient.

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In Reply:—Guillaume *et al.* are mistaken when they state that induction of general anesthesia increases cutaneous heat loss: the increase is trivial.[1] General anesthesia does decrease metabolic heat production,[2] but the decrease is only about 20%, which is insufficient to explain the typical rapid onset of core hypothermia after anesthetic induction. It is for this reason that we proposed core-to-peripheral redistribution of body heat as a primary force reducing core temperature in the immediate postinduction period.[1,3] Consistent with the importance of redistribution, we have shown that peripheral tissue warming before induction of general[4] and epidural[5] anesthesia minimizes core hypothermia.

Pre-induction cutaneous warming is assumed to reduce redistribution hypothermia by increasing peripheral tissue heat content. However, in contrast with the statement of Guillaume *et al.*, peripheral tissue temperature was not measured in our prewarming studies[4,5] or that of Just *et al.*[6] However, we have recently measured arm and leg tissue temperature during prewarming; from these values we were able to estimate that heat content increases 77 ± 18 kcal in just 30 min and 133 ± 30 kcal in 1 h (table 1, unpublished data). These values are consistent with established rates of cutaneous heat transfer during forced-air warming[7,8] and the heat capacity of the peripheral thermal compartment (about 150 kcal).[9] The large in-

Table 1. Increase in Arm and Leg Heat Content during Prewarming

(h)	Δ Content (kcal)	Δ Content (kcal/kg)	Fraction of Maximum (%)
0.5	77 ± 18	0.9 ± 0.1	44 ± 6
1.0	133 ± 30	1.6 ± 0.2	77 ± 8
2.0	176 ± 48	2.0 ± 0.3	100

Five volunteers were warmed for 2 h using a Bair Hugger forced-air heater (Augustine Medical, Eden Prarie, MN) set on "high." Total arm and leg (peripheral) tissue heat content increased dramatically with forced-air warming. Most warming occurred within 1 h.

crease in arm and leg heat content explains how prewarming alone can maintain intraoperative normothermia for many hours.[6]

The specific heat of humans is essentially constant.[10,11] A nifedipine-induced increase in peripheral thermal compartment heat content, therefore, must be associated with increased peripheral tissue temperature and a reduced core-to-peripheral tissue temperature gradient. Therefore, we were surprised by the suggestion of Guillaume *et al.* that nifedipine increases peripheral heat storage *instead* of changing the core-to-peripheral tissue temperature gradient. Their error appears to be in assuming that mean skin temperature reliably indicates peripheral tissue temperature. Skin temperature is a complex function of core and ambient temperature, adjacent tissue temperature, and thermoregulatory status. That skin temperatures were similar in untreated patients and those given nifedipine indicates simply that skin temperature generally does not adequately quantify peripheral tissue temperature.

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Electroencephalogram Suppression during Anesthesia

To the Editor:—In a recent editorial, Drummond[1] stated that, with a concentration of isoflurane sufficient *to render the electroencephalogram (EEG) isoelectric,* it is reasonable to anticipate that the rate of cerebral oxygen use is strongly reduced. From the following, discussion it is obvious that he means *EEG suppression,* not *isoelectric EEG.*

Isoelectric EEG refers to a situation in which the brain, or at least the cerebral cortex, does not produce electrical activity, *i.e.,* electrocerebral inactivity or electrocerebral silence.[2]

Suppression is not synonymous with isoelectricity. During isoflurane-induced suppression, low-amplitude EEG activity is well described in the literature. Zaret has reported α coma pattern during suppression.[3] Spindles measuring 13 Hz are seen on the vertex during propofol-induced suppression.[4] Focal or generalized epileptic discharges sometimes are seen during barbiturate anesthesia for the treatment of status epilepticus. In the higher frequencies, the N_{20} wave of somatosensory evoked potentials to median nerve stimulation, for instance, can be recorded even during EEG suppression. Hence,