POSTER DISCUSSION III

A1003

TITLE: CONTINUOUS MEASUREMENT OF PUPILLARY AREA FLUCTUATIONS AS A MARKER OF THE AUTONOMIC NERVOUS SYSTEM FUNCTION

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Pupillary area (PA) is known to fluctuate at a frequency of about 1 Hz. This fluctuation reflects an equilibrium between sympathetic vs. vagal phasic activities. Thus, analysis of PA variations can be used as a marker for estimating central autonomic outflows from the brain.

We newly developed a light weight portable video camera sensitive to infrared light with a miniature charge-coupled device for pupillometry. With informed consent and institutional approval, we analyzed PA fluctuations by this method to assess the autonomic nervous system (ANS) function in anesthetized patients. PA was calculated by binary differentiation of 60,000 pixels at a rate of 60 Hz. ECG, plethysmogram and pneumogram were also simultaneously recorded in digital data recorder.

In the awake state, PA fluctuations mainly coincide with each cardiac beat with an increase during late systole and superimposed by respiratory change. This synchrony was absent in anesthetized patients. 10 to 15 min following 2% enflurane/nitrous oxide anesthesia, the trace of PA became smoothed out. Respiratory fluctuation was partially reserved 10 min later, but totally absent 15 min later from the beginning of anesthesia (Fig 1). After 10 mcg/kg fentanyl administration, complete disappearance of contours in the PA tracing was observed (Fig 2). This is characteristics of the effects of opiates on PA variations. PA changes fluctuated on skin incision after enflurane anesthesia. In contrast, skin incision had no effect on PA following fentanyl anesthesia (Fig 3).

Analysis of PA fluctuations is a time-tested method to evaluate ANS function. Continuous monitoring of PA fluctuations can give us an information concerning the dynamic nature of the pupillary response to anesthetics and noxious stimulation related to surgery.

Fig. 1

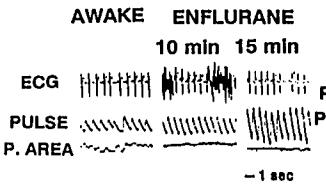


Fig. 2

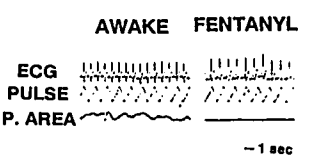
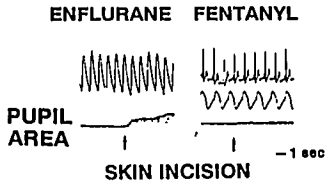


Fig. 3



A1004

TITLE: PRE-INDUCTION SKIN-SURFACE WARMING PREVENTS REDISTRIBUTION HYPOTHERMIA

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Hypothermia often develops rapidly following induction of general anesthesia. Our previous work suggests that its primary etiology is redistribution of heat from central to peripheral tissues.[1] We therefore tested the hypothesis that internal redistribution of heat and hypothermia can be reduced by skin-surface warming *before* induction of general anesthesia.

Following IRB approval, 10 volunteers participated in one of two studies of thermoregulation during isoflurane anesthesia.[1,2] In one group of five, (control) no external warming methods were used before induction or during the first 30 min of anesthesia. In the second, five volunteers were given skin-surface warming using a forced-air warmer for 45 min before induction. Warming was discontinued after induction. All volunteers were uncovered after induction of anesthesia. Tympanic membrane temperature and heat loss (thermal flux transducers, ten area-weighted sites) were measured.

Heights, weights, ages, ambient temperatures and administered fluid volumes were similar in both groups. Cutaneous heat loss was negative during pre-warming (indicating heat storage). At induction, tympanic membrane temperatures were 36.8±0.2 and 37.2±0.3 in the control and pre-warmed groups respectively. Despite greater heat loss in the pre-warmed volunteers after induction of anesthesia, central temperature remained greater than in the control group. After 30 min, the decrease in tympanic membrane temperature was ≈0.8°C less in the pre-warmed group. We conclude hypothermia following induction of general anesthesia can be reduced by skin-surface warming *before* induction.

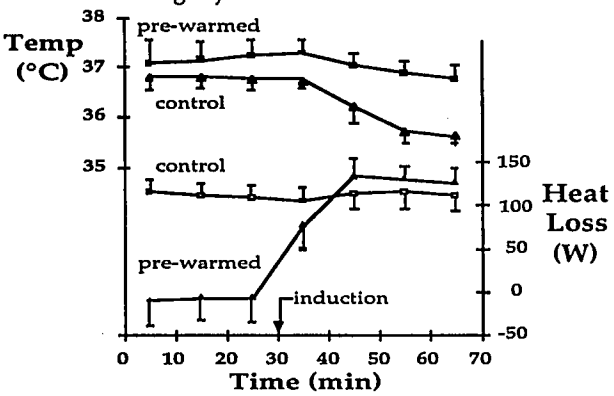

Figure. Pre-warming decreased initial hypothermia following induction of anesthesia, despite slightly greater heat loss. Volunteers were uncovered in a cool room except during pre-warming.

References:
1. Sessler DI, *et al*, Anesthesiology 74:226-232, 1991.
2. Hynson JM, *et al*, Anesth Analg 72:S119, 1991.

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