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TITLE: HEAT STORAGE CAPACITY OF THE PERIPHERAL THERMAL COMPARTMENT.

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Central temperature can decrease without any change in mean body temperature when heat distribution within the body is alterred. Such redistribution of heat leads to hypothermia following induction of general or epidural anesthesia.[1,2] Similarly, central temperature may decrease during surface warming for the treatment of accidental or perioperative hypothermia. The extent of afterdrop depends on the temperature and thermal capacity of the peripheral thermal compartment. To estimate this thermal storage capacity we measured cutaneous thermal flux and metabolic heat production during skin-surface warming of anesthetized, hypothermic volunteers.

Following IRB approval, hypothermia was induced in five volunteers during isoflurane anesthesia. Each was actively cooled using circulating water blankets set at 5°C. Blankets were placed under the back and over the anterior surface. Volunteers were cooled to a central temperature of 34.0±0.6 (SD)°C (below the threshold for thermoregulatory vasoconstriction during isoflurane anesthesia[3]). Volunteers were then rewarmed using two forced-air warmers on high (43°C) covering the anterior surface and a full-length circulating water blanket under the back set at 40°C. Heat balance was evaluated from the start of rewarming, through the afterdrop and until central temperature recovered to what it was at the start of rewarming. Heat gain across the skin was measured with thermal flux transducers (10 area-weighted sites). Oxygen consumption was measured and converted to heat production using a caloric value for oxygen of 4.18 kcal/l. Heat storage was calculated by integrating the sum of cutaneous heat gain and heat production over time.

Volunteers vasoconstricted at 35.2 ± 0.8°C. Despite positive heat gain during rewarming, tympanic membrane temperature decreased an additional 0.6±0.1°C before increasing. Total heat storage during rewarming was 144±60 kcal. Correlation coefficients with BSA, height and weight were: 0.91, 0.73 and 0.85 respectively.

Since central temperature was the same at the start and end of rewarming, the measured heat storage reflects the large thermal capacity of the peripheral compartment. This amount of heat (144 kcal) is approximately equivalent to 2-3 h of basal metabolic heat production and would represent a change in *mean* body temperature of ≈2.5°C for an average 70 kg human.

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TITLE: THE EFFECT OF METHYLPARABEN AND PROPYLPARABEN, TWO COMMONLY USED DRUG PRESERVATIVES, ON HUMAN CEREBRAL BLOOD FLOW

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Succinylcholine has been shown to increase intracranial pressure in both animals and humans. Although a number of mechanisms have been postulated, a recent in vitro study suggested that rather than succinylcholine, the preservatives methyl-paraben(MP) and propylparaben(PP) which are used in multidose vials are the vasodilators[1]. We explored this hypothesis in humans by the measurement of cerebral blood flow(CBF) before and after the intravenous(IV) administration of MP and PP.

Methods: After IRB approval, written informed consent was obtained from sixteen healthy volunteers. In the first 8 voluteers, CBF was measured by inhaled ^{133}Xe and a 16 lead EEG continuously recorded. In the second group, CBF was measured by transcranial Doppler. In each subject, after a baseline measurement, MP 9mg and PP 1mg (the amount present in 100 mg of succinylcholine) was given IV and CBF immediately measured.

Results: There were no significant changes in ^{133}Xe CBF (CBF averaged over 15 minutes or initial slope index) or in the transcranial Doppler with the intravenous injection of the paraben preservatives. There was also no evidence of EEG stimulation or other EEG changes.

Discussion: Because ^{133}Xe CBF takes time to perform, we were concerned that it may miss transient changes which have been reported in some studies of succinylcholine. However even using transcranial Doppler which provides beat to beat measurements, we were unable to detect any changes. Succinylcholine is believed to stimulate muscle spindles and to result in secondary EEG stimulation which increases CBF and ICP[2]. We detected no EEG changes suggestive of activation in these awake subjects. Our subjects had a normal cerebral vasculature and different results could be obtained in patients with an abnormal vasculature. We conclude that it is unlikely that the increases in CBF and ICP reported are due to the preservatives methylparaben and propylparaben.

References:

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