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Causes of Induced Hyperthermia

To the Editor: — In his article, Dr. Fraser has reported an effective, if inadvertent, method of inducing hyperthermia.[1] The temperature increases, however, can probably be more correctly attributed to the prevention of evaporative heat loss than to a decrease in convection or radiation. Presently at our institution we are conducting a phase 1 trial of whole-body hyperthermia to 41.8 C as an adjuvant mode of therapy in the treatment of metastatic cancer.[2] The technique employed depends primarily on the confinement of endogenous metabolic heat by restricting evaporative heat loss.

In our protocol patients wear a disposable, nonpermeable paper "jump suit" over cotton pajamas. The suit, which is made of material similar to the implicated surgical drapes, prevents evaporative heat loss by confining water vapor. The suit is then surrounded by several heated-water perfusion blankets that both further insulate and provide additional exogenous heat in the initial phase to increase the rate of heating. With this combination vapor barrier–insulation system, core temperature can be increased 3 degrees C/hour. The system is so effective that when the patient's temperature is 41.8 C, the temperature of the water perfusing the blankets often needs to be several degrees cooler to prevent "thermal runaway."[3] Were an anesthetized child, dressed in pajamas and also possibly placed on a conventional heated-water mattress in the operating room, to be then enveloped by a nonpermeable drape, hyperthermia might well ensue.

Under conditions of increased endogenous heat production (malignant hyperthermia or pyrexic infections), oxygen consumption must show correspondingly large increases. Our recent work, however, has shown that in anesthetized adult subjects, where core temperatures are increased by retarding heat dissipation, oxygen consumption does not increase dramatically. When pulmonary arterial blood tem-

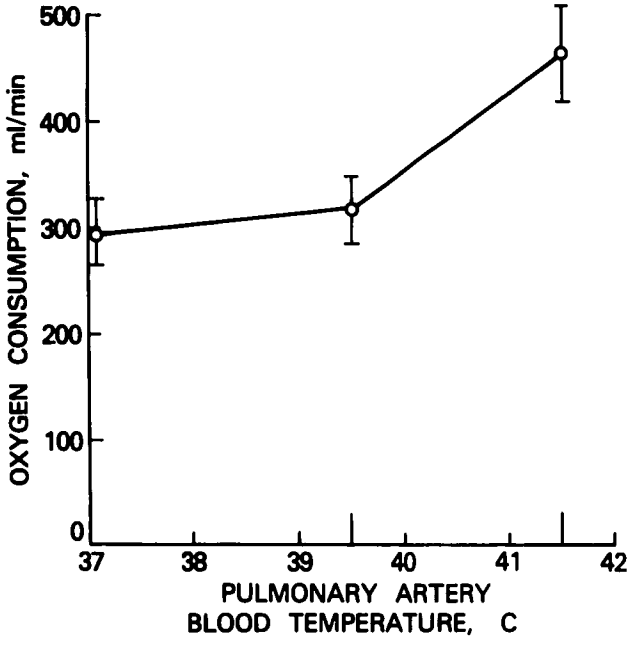

FIG. 1. Oxygen consumption during thiopental–fentanyl anesthesia with whole-body hyperthermia.

TABLE 1. Comparison of Cooling Rates (Maximum Rates Recorded by Esophageal Thermistors)*

Degrees C/Hour	
Ice Packing	Forced Air
7.3	8.5
5.5	10.0
8.0	7.4
8.2	6.3
6.7	9.5
7.1 ± 0.5 (SEM)	8.3 ± 0.7

* Results were compared by Student's t test for paired data. Significance was defined as $P < 0.05$.

peratures were increased from 37 to 41.5 C in ten patients during thiopental–fentanyl anesthesia, oxygen consumption increased to only 50 per cent above normothermic control values (fig. 1).

In the study reported by Fraser, the patients were cooled by increasing evaporative heat loss (removing the drapes and alcohol sponging) and by applying ice packs. We have compared these two methods of cooling at the conclusion of whole-body hyperthermia treatments. On five separate occasions, a 71-kg male patient was cooled from 41.8 to 37 C by surrounding the anterior trunk and extremities with ice. On five subsequent occasions, the same patient was cooled by directing a forced-air stream over the exposed body surfaces. Room temperature was maintained between 24 and 27 C and relative humidity between 30 and 40 per cent. There was no significant difference in the rates of cooling by the two methods (table 1).

Packing with ice offered no advantage over increasing evaporative cooling, and was a cumbersome technique. Evaporative cooling is rapid because of the high latent heat of vaporization for water (540 cal/g). It should be noted that alcohol sponging is not necessary and may, in fact, be hazardous.[4]

With modern air-conditioning, and to-and fro circuits no longer in use, intraoperative hyperthermia is an uncommon event. The report by Fraser illustrates the need to consider all possible etiologies before implementing a specific therapeutic regimen that may not be without its own risks. Complex problems often require only a simple solution.

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Averaging Values for Gastric *p*H Incorrect

To the Editor:—Please permit me to point out a frequently made, but nevertheless serious, error in the calculation of data from measurements of acidity of gastric contents using *p*H. Stoelting has reported a mean and standard error of *p*H in tables 3 and 4, and the editors permitted this computation.[1] *p*H is defined as the negative logarithm of the hydrogen ion concentration. A quick review of algebra tells us that in a logarithm, the number before the decimal is the *characteristic* (tells us where to put the decimal in the real number) and the number behind the decimal is the *mantissa* (gives us the identity of the real number from a table). Logarithms cannot be averaged until they have been converted back to real numbers. The same mistake, of course, has been made with blood data, where the *characteristic* is usually 7 and the magnitude of error is no more than a factor of 10. By contrast, gastric *p*H values range from 1 to 9 and errors in the magnitude of one million are possible.

In spite of a disagreement with the author on his mathematics, I concur with his conclusions, but would enjoy seeing his results analyzed appropriately.

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