- taneous lymph node syndrome. Am J Cardiol 47:323-330, 1981
- Kitamura S, Kawashima Y, Kawachi K, et al: Severe mitral regurgitation due to coronary arteritis of mucocutaneous lymph node syndrome. J Thorac Cardiovasc Surg 80:629-636, 1980
- Hondo S, Matsumato H, Mizoguchi Y, Hamasaki Y, Sunadawa H: Aortic regurgitation following acute febrile mucocutaneous lymph node syndrome (MCLS) in an infant. Jpn Circ J 43:463– 468, 1979
- Kato H, Koiki S, Yamamoto M, Ito Y, Yano E: Coronary aneurysms in infants and young children with acute febrile mucocutaneous lymph node syndrome. J Pediatr 86:892–898, 1975
- Fukushige J, Nihill MR, McNamara DG: Spectrum of cardiovascular lesions in mucocutaneous lymph node syndrome: Analysis of eight cases. Am J Cardiol 45:98-107, 1980
- Kato H, Koike S, Tanaka C, et al: Coronary heart disease in children with Kawasaki disease. Jpn Circ J 43:469-475, 1979
- Kegel SM, Dorsey TJ, Rowen M, Taylor WF: Cardiac death in mucocutaneous lymph node syndrome. Am J Cardiol 40:282– 286, 1977

- Kato H, Ichinose E, Yoshioka F, et al: Fate of coronary aneurysms in Kawasaki disease: Serial coronary angiography and longterm follow-up study. Am J Cardiol 49:1758–1766, 1982
- Kato H, Koike S, Yokoyama T: Kawasaki disease: Effect of treatment on coronary artery involvement. Pediatrics 63:175–179, 1979
- Konishi Y, Tatsuta N, Miki S, et al: Simultaneous surgical treatment of tetrology of Fallot and coronary artery aneurysm due to mucocutaneous lymph node syndrome in a 4-year-old child. Jpn Circ J 43:749–756, 1979
- Sandiford FM, Vargo TA, Shih JY, Pelargonia S, McNamara DG: Successful triple coronary artery bypass in a child with multiple coronary aneurysms due to Kawasaki's disease. J Thorac Cardiovasc Surg 79:283–287, 1980
- Wada J, Endo M, Takao A, Kawamura T: Mucocutaneous lymph node syndrome. Chest 77:443–446, 1980
- Suma K, Takeuchi Y, Shiroma K, et al: Early and late postoperative studies in coronary arterial lesions resulting from Kawasaki's disease in children. J Thorac Cardiovasc Surg 84:224– 229, 1982
- Mercer S, Carpenter B: Surgical complications of Kawasaki disease. J Pediatr Surg 16:444–448, 1981

Anesthesiology 58:271-275, 1983

Deep Body Thermometry during General Anesthesia

STANLEY MURAVCHICK, M.D., PH.D.*

Conventional techniques for measuring central or "core" patient temperatures during general anesthesia require passage of thermistor or thermocouple probes through body orifices into the nasopharynx, esophagus, rectum, or auditory canal. These techniques can cause tissue trauma and hemorrhage and are subject to sampling error because of imprecise placement of the probe. In this study of adults undergoing elective surgery, we measured the *in vivo* equilibrium and response characteristics of a commercially available non-invasive deep body thermometry (DBT) system using a heated skin surface probe designed to provide atraumatic approximations of core body temperatures. We then compared those characteristics to those of a conventional, passive, wedged nasal thermistor device.

Received from the Department of Anesthesiology, University of Miami School of Medicine, and the Veterans Administration Medical Center, Miami, Florida. Presented in part at the Scientific Session of the annual meeting of the American Society of Anesthesiologists in New Orleans, Louisiana, October 21, 1981. Accepted for publication September 15, 1982.

Address reprint requests to Dr. Muravchick: Department of Anesthesia, University of Pennsylvania School of Medicine, Hospital of the University of Pennsylvania, 3400 Spruce Street, Philadelphia, Pennsylvania 19104.

Key words: Hypothermia. Surgery: cardiac. Measurement techniques: thermometry, deep body.

METHODS

The apparatus evaluated was the Thermo-Finer Coretemp® Model DC-1 DBT system† using a 40-g, 4.5cm diameter surface probe with a cast aluminum shell containing an electrical heating element and two thermistors separated by an insulator. The probe is connected by a flexible electrical cable to amplifier, comparator, and heater circuitry within an AC line-powered control box with digital display to the nearest 0.1° C. For each patient, one of two available probes was connected interchangeably to one of two control boxes used for this study. To obtain control values, one of four Yellow Springs Instruments® series 701 vinyl-covered thermistor probes was connected to the amplifier and readout circuits provided in a Tektronix® 414 electrocardiogram and pressure monitor with function controls set for digital display of temperature to the nearest 0.1° C. Prior to patient use, the Yellow Springs thermistor probes and the Coretemp probes were applied simultaneously to a temperature-controlled water bath; they were used for this study only if found to indicate the same temperature to within 0.1° C at 34.0° and 36.0° C. Both the DBT and Yellow Springs probe systems met

^{*} Associate Professor of Anesthesiology.

[†] Terumo America, Inc., 120 New England Avenue, Piscataway, New Jersey 08854.

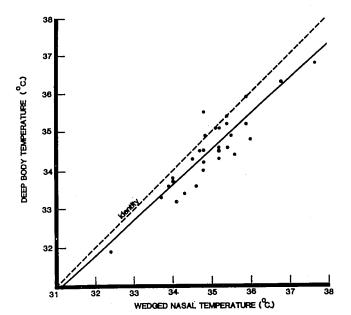


FIG. 1. Simultaneous wedged nasal and deep body temperatures at in vivo equilibrium, 31 pairs, r=0.91. Dashed line represents identity; solid line is best-fit curve by least-squares method.

the manufacturer's nominal response time specifications of 60 and 45 seconds, respectively, and both were checked for electrical safety and approved by a qualified bioengineering technician.

A total of 50 consenting patients, 25 to 74 years of age, were studied according to a protocol approved by the Human Studies Subcommittee of the Miami VA Medical Center. Data from 44 of these patients were used to determine equilibrium times and temperatures; the other six patients were studied only during the non-equilibrium conditions of hypothermic CPB. After induction of inhalational or intravenous general anes-

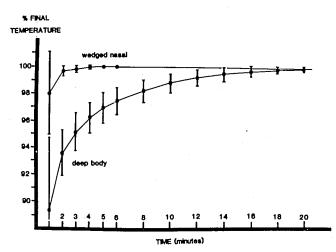


FIG. 2. Mean time required to reach equilibrium in vivo, wedged nasal thermistor and deep body thermometry systems. Each point represents the mean \pm SD of 31 measurements.

thesia and tracheal intubation, a Coretemp probe was secured to the midline forehead of the patient by means of a custom-designed Velcro® headstrap, and a thermistor probe advanced 8 to 12 cm into the nasopharynx until slight resistance was encountered, indicating wedged nasal (WN) position. Both probes were fixed in final position with tape as needed.

Ten of the patients studied underwent cardiac surgery with cardiopulmonary bypass (CPB) and moderate hypothermia (25° C–28° C), and 40 had a variety of orthopedic and general surgical procedures. All patients studied had both a Coretemp DBT and WN thermistor probe in place; the WN probe provided control values for all simultaneous DBT readings. Operating room air was circulated continuously with 16 changes per hour, and room temperature maintained within the range of 20.0° C to 24.0° C.

Time to equilibrium, t_{eq}, was defined as the interval required in minutes before a digital temperature display remained unchanged for four consecutive minutes. Temperature discrepancy, ΔT , was defined as the difference in simultaneously measured temperature, DBT-WN, in °C. The t_{eq} values for the DBT and WN probes were compared by t test, and the indicated equilibrium temperatures were compared statistically using the Wilcoxon Signed Rank Test for non-parametric paired samples¹ with P < 0.05 being the criterion of statistical significance for all comparisons. The relationship between equilibrium DBT and WN temperatures was established by calculating a correlation coefficient for regression analysis by the least-squares method. Demographic data and physical characteristics recorded, measured, or calculated for every patient included age, weight, height, body surface area, head circumference, and depth of thermistor probe insertion.

RESULTS

In the 44 patients studied to determine simultaneous in vivo equilibrium temperatures, the indicated values of the two systems were significantly different (P < 0.001), with a ΔT of $-0.5 \pm 0.1^{\circ}$ C, (mean \pm SE), the WN thermistor indicating a higher value than the DBT system. In 31 of these patients, indicated temperatures from both systems had been recorded at oneminute intervals beginning with probe placement, and the mean time required to achieve in vivo equilibrium, t_{eq} , for the DBT system was 16.6 ± 0.7 min, significantly longer (P < 0.001) than the t_{eq} of 3.2 \pm 0.2 min of the WN system. The simultaneous equilibrium values for the two systems showed non-normal distribution but were highly correlated (r = 0.91), ΔT increasing at higher temperatures (fig. 1). Mean times to achieve 99% of teq were approximately 10.5 and 1.5 min, respectively, for the DBT and the WN thermometry systems (fig. 2). Longest observed individual $t_{\rm eq}$ for the DBT system was 28 min; for the WN system, it was 6 min.

There was no significant correlation between ΔT at equilibrium and patient weight or head circumference, and no correlation between the t_{eq} of either system and patient head circumference, body weight, or body surface area. There was no difference between ΔT for the 32 patients receiving inhalation anesthesia (enflurane or halothane) and the ΔT of 12 patients given a narcotic/muscle relaxant anesthetic. Average age of the 44 patients in whom equilibrium measurements were made was 52.8 ± 2.0 years (mean \pm SE); average weight was 74.6 ± 2.0 kg; average body surface 1.90 ± 0.03 m²; average head circumference 57.9 ± 0.4 cm; and average depth of insertion of WN probe 8.7 ± 0.4 cm.

Non-equilibrium conditions such as rapid cooling and warming using extracorporeal perfusion during CPB markedly increased ΔT . Six patients who had not been observed at one-minute intervals according to the equilibrium study protocol but who had had both temperature monitors in place for at least one hour were studied during rapid cooling and warming with CPB. In addition, two of the 44 patients in whom equilibrium temperatures had been measured also were observed during rapid cooling but not during rapid warming, and two other patients from the equilibrium study were observed during rapid warming but not during rapid cooling with CPB. These ten patients, therefore, provided a total of 16 observations; eight of simultaneous DBT and WN temperatures during rapid cooling (fig. 3), and eight observations during rapid warming (fig. 4). During rapid cooling with CPB, mean ΔT went from an initial value of -0.5 ± 0.2 °C (\pm SE) to a maximum value of $+2.0 \pm 0.8^{\circ}$ C within six minutes after the start of cooling. The discrepancy then fell progressively but did not return to its initial value until 14 to 20 minutes after the start of cooling. The largest individual ΔT observed during rapid cooling was +4.9° C, indicating that DBT readings lagged behind the changes indicated by the WN system.

Temperature discrepancy patterns during rapid warming on CPB were even more varied than those seen during cooling. The maximum ΔT observed during warming was $+5.3^{\circ}$ C, indicating DBT temperature was leading WN temperature, but in one patient the ΔT reached -3.8° C, with the DBT lagging behind WN (fig. 4). The discrepancies for the eight patients studied were not of similar sign or magnitude until more than 40 min after the initiation of warming. Mean age of the ten patients in whom the CPB observations were made was 56.2 ± 1.7 years and mean weight 80.1 ± 4.5 kg, not significantly different from the corresponding values for the patients observed in non-CPB equilibrium.

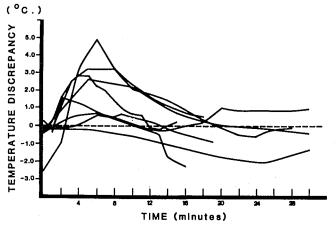


FIG. 3. Time courses of eight individual observations of temperature discrepancy (deep body-wedged nasal temperature, °C) during rapid cooling on cardiopulmonary bypass.

No electrical burns or thermal injuries were observed with the Coretemp DBT system, but stable positioning of the probe on the forehead proved to be difficult in two patients. The WN thermistor was discontinued in an anticoagulated patient with a persistent nosebleed and in another patient because of unexplainably erratic temperature indications.

DISCUSSION

Continuous monitoring of body temperature during general anesthesia is a widely accepted clinical practice which includes a variety of techniques. The large temperature gradients between superficial and deep body tissues during general anesthesia,² especially with hypothermic CPB,³ suggest that body temperature measurements are most reliable and useful when they reflect

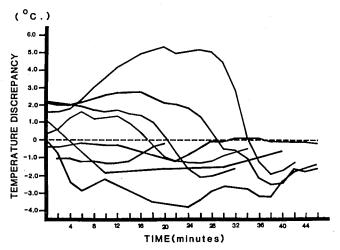


FIG. 4. Time courses of eight individual observations of temperature discrepancy (deep body-wedged nasal temperature, °C) during rapid warming on cardiopulmonary bypass.

the temperature of the central core of organs and tissues within the skull, chest, and abdomen.4 Tympanic membrane probes accurately track rapid changes in blood temperature but cannot be expected to reflect the temperature of the central organ core when blood temperature is abruptly manipulated using a heat exchanger during cooling or warming on CPB.5 Lower esophageal temperatures also are determined largely by heart and blood temperature and were not appropriate for control values in our study because application of ice and cold cardioplegia solutions to the heart was required during CPB. We chose instead to use a modification of the nasopharyngeal technique, the wedged nasal thermistor,6 as our control standard. It is a conventional technique which correlates well with brain temperature, is accessible during most surgical procedures, and is in anatomic proximity to the forehead placement site for the Coretemp DBT probe.⁷

Fox and Solman^{8,9} were the first to describe a technique for monitoring deep body temperature by means of a probe placed on the skin surface using a heated element to create a zone of zero heat flow under the probe. Subsequent modification of the original design and more rigorous mathematical analysis of the heat flow zones beneath the probe^{7,10} indicated that active heating of the aluminum cup which served as a housing for the probe would make the system independent of ambient temperature if the environment was colder than the patient. The equilibrium times of the DBT system reflect the time required to eliminate the initial temperature gradients between the skin surface and the deeper tissues of the area being monitored, as well as the caloric capacitance of the probe itself. In theory, DBT systems such as the Coretemp "exteriorize" the temperatures of core organs, simplifying probe placement and minimizing tissue trauma. Once thermal equilibrium has been achieved, intermittent heating triggered by a comparator circuit compensates for heat loss from the probe.

In vivo equilibrium times previously reported for the improved design represented by the Coretemp DBT were obtained from unanesthetized subjects or from animal models, and range from 30 min⁶ to 15 to 20 min. 7,10 Prior published values for equilibrium temperature discrepancy 7,11 vary from -0.1° to -0.4° C, depending upon the DBT probe design and the monitoring site used as a reference. In our study, human equilibrium deep body temperatures during general anesthesia were consistently lower than simultaneous wedged nasal temperatures, but while the average Δ T of -0.5° C was statistically significant, it was numerically small and not likely to be of clinical importance. In

agreement with earlier results from animal studies, 10 we found that ΔT increased at higher equilibrium temperatures. We found, however, that ΔT was independent of the type of anesthetic used, the physical mass of the patient, or measured head circumference. Others have suggested that increased thickness of subcutaneous tissues would alter the ability of the probe to establish and maintain zero heat flow. 10

The 20-min in vivo equilibrium or stabilization period suggested by the manufacturer of the Coretemp DBT system is a realistic recommendation, but this interval represents a much longer period than that required by the conventional wedged nasal thermistor system. Rapid and significant changes in temperature early in anesthesia, such as those associated with malignant hyperpyrexia, could be missed in a patient monitored only with a DBT system placed just after induction of anesthesia. We also found that even after initial equilibrium has been achieved, rapid alteration of body temperature such as cooling or warming during CPB can produce erratic and unpredictable relationships between the temperatures indicated by the DBT system and those of the conventional passive thermistor accepted as the standard for core temperature monitoring. Therefore, we conclude that DBT systems such as the Coretemp may be useful in monitoring slow, progressive changes in temperature such as may be seen in an intensive care environment, but the DBT technique is not clinically appropriate for monitoring the rapid changes in patient temperature that may occur during general anesthesia and cardiopulmonary bypass.

The author gratefully acknowledges the technical assistance of Robert Andritsch, B.S.

REFERENCES

- Colton T: Statistics in Medicine. Boston, Little, Brown and Co, 1974, pp 219-221
- 2. Smith NT: Subcutaneous, muscle, and body temperatures in anesthetized man. J Appl Physiol 17:306-310, 1962
- Muravchick S, Conrad DP, Vargas A: Peripheral temperature monitoring during cardiopulmonary bypass operation. Ann Thorac Surg 29:36-41, 1980
- Holdcroft A: Body Temperature Control in Anesthesia, Surgery, Intensive Care. London, Bailliére Tindall, 1980, pp 4–5
- Dickey WT, Ahlgren EW, Stephen CR: Body temperature monitoring via the tympanic membrane. Surgery 67:981-984, 1970
- Vale RJ: Monitoring of temperature during anesthesia. Int Anesthesiol Clin 19:61–83, 1981
- Togawa T, Nemoto T, Yamazaki T, Kobayashi T: A modified internal temperature measurement device. Med Biol Eng 14:361-364, 1976

- Fox RH, Solman AJ: A new technique for monitoring the deep body temperature in man from the intact skin surface. J Physiol (Lond) 212:8P-10P, 1971
- Fox RH, Solman AJ, Isaacs R, Fry AJ: A new method for monitoring deep body temperature from the skin surface. Clin Sci 44:81–86, 1973
- Kobayashi T, Nemoto T, Kamiya A, Togawa T: Improvement of deep body thermometers for man. Ann Biomed Eng 3:181– 188, 1975
- Singer B, Lipton B: Monitoring of core temperature through the skin: A comparison with esophageal and tympanic temperatures. Bull NY Acad Med 51:947-952, 1975

Anesthesiology 58:275-277, 1983

Postoperative Rigidity Following Fentanyl Anesthesia

CHARLES M. CHRISTIAN II, M.D., PH.D.,* JOHN L. WALLER, M.D.,† C. CRAIG MOLDENHAUER, M.D.,‡

Postoperative respiratory depression occurs after the intravenous injection of fentanyl. Becker $et~al.^1$ demonstrated the biphasic nature of this response; Adams and Pybus² cited three patients in whom postoperative respiratory depression followed an apparently normal recovery from fentanyl-nitrous-oxide anesthesia; McQuay $et~al.^3$ demonstrated a second peak of plasma fentanyl which occurred after surgery. Stoeckel $et~al.^4$ postulated an entero-systemic recirculation to explain these phenomena. Hall⁵ expressed concern about the impact of the recent trend to high-dose (50–150 μ g/kg) fentanyl anesthesia on the problem of a secondary or biphasic episode of respiratory depression.

We describe three patients who developed rigidity several hours after fentanyl administration which may be attributable to a secondary peak of fentanyl in the plasma.

REPORTS OF THREE PATIENTS

Patient 1. A 56-year-old, 78-kg man with coronary occlusive disease scheduled for coronary artery bypass grafting (CABG) was premedicated with 30 mg propanolol, po, 10 mg diazepam, po, and 15 mg morphine, im. Systemic and pulmonary arterial and peripheral venous lines were established, and electrocardiographic and electroencepha-

Key words: Analgesics: fentanyl. Anesthestics, intravenous: fentanyl. Complications: rigidity. Pharmacokinetics: distribution.

lographic monitoring were initiated prior to induction of anesthesia. Induction of anesthesia was accomplished with 35 μ g/kg fentanyl and 150 μ g/kg pancuronium; the patient breathed 100% oxygen through a semi-closed system. Truncal rigidity was not seen during induction of anesthesia and no hemodynamic response to endotracheal intubation occurred. Ventilation was controlled to maintain Pa_{CO2} between 33 and 37 mmHg.

Additional fentanyl (15 μ g/kg) failed to ablate hemodynamic responses to skin incision and a sodium nitroprusside infusion was begun. Additional doses of 10 μ g/kg fentanyl were given prior to sternotomy and prior to initiation of cardiopulmonary bypass. Neuromuscular blockade was maintained by incremental doses of pancuronium bromide titrated to produce the loss of the last twitch of a train-of-four. During the course of surgery, the patient received a total dose of fentanyl of 70 μ g/kg (5.46 mg) and 16 mg pancuronium, iv.

The anesthetic and surgical course was otherwise unremarkable. At the termination of the procedure, four hours after induction of anesthesia, neuromuscular train-of-four had returned to control value although no antagonist had been given. Ventilation was spontaneous. He was responsive to voice and would move all four extremities on command. His rectal temperature was 35.4° C.

The patient was taken to the cardiovascular acute care unit where ventilation was controlled with a volume ventilator which delivered an FIO2 of 0.6, a tidal volume of 12 ml/kg at an intermittant mandatory vantilatory rate of six breaths/min. Analysis of arterial blood gases revealed a $p\rm H_a$ of 7.47, a PaO2 of 113 mmHg, and a PaCO2 of 37 mmHg. The patient was placed under a radiant heater and sodium nitroprusside infusion was used to control hypertension.

Approximately 45 min later, five hours after the initial injection of fentanyl, the patient became obtunded and his extremities, chest, and abdominal musculature became rigid. His rectal temperature at this time was 36.7° C. Analysis of blood gases revealed a Pa_{CO2} of 65 mmHg, a *p*H_a of 7.23, and a Pa_{O2} of 98 mmHg. The peak inspiratory pressure (PIP) was 47 cmH₂O. His pupils were 2 mm and reactive to light. His cardiac index was 4.7 l·min⁻¹·m⁻², mean arterial pressure 111 mmHg, pulmonary artery occlusion pressure 17 mmHg, and central venous pressure 10 mmHg.

Within three minutes after 40 μ g naloxone, iv, the patient was responsive, moved all extremities upon command, and complained of mild incisional discomfort. Rigidity disappeared completely. After readjustment of the ventilator, analysis of blood gases revealed a Pa_{CO_2} of 42 mmHg, a pH_a of 7.42, and Pa_{O_2} of 123 mmHg. Cardiac index was 3.9 $1 \cdot min^{-1} \cdot m^{-2}$, mean arterial pressure 84 mmHg, pulmonary artery occlusion pressure 16 mmHg, and central venous pressure 11 mmHg.

^{*} Assistant Professor, Division of Cardiothoracic Anesthesia, Department of Anesthesiology, Duke University Medical Center.

[†] Associate Professor, Section of Anesthesiology, Cardiothoracic Division, Emory University Clinic.

[‡] Cardiac Fellow, Section of Anesthesiology, Cardiothoracic Division, Emory University Clinic.

Received from the Departments of Anesthesiology, the Duke University Medical Center, Durham, North Carolina, and the Emory University Clinic, Atlanta, Georgia. Accepted for publication September 16, 1982.

Address reprint requests to Dr. Christian: Department of Anesthesiology, Duke University Medical Center, Durham, North Carolina 97710