SKELETAL MUSCLE, ESOPHAGEAL AND RECTAL TEMPERATURES IN MAN DURING GENERAL ANESTHESIA AND OPERATION

HARRY WOLLMAN, M.D., AND THOMAS H. CANNARD, M.D.

Muscle temperature changes influence the action of muscle relaxants in both animals and man. A reduction in muscle temperature as small as two to three degrees centigrade has been shown to prolong the action and increase the intensity of blockade produced by succinylcholine and decamethonium in man. It has been suggested that some cases of prolonged apnea following the use of depolarizing neuromuscular blocking drugs may be related to lowered temperature of the muscles of respiration. This study was undertaken to ascertain the direction and extent of muscle and core temperature changes during general anesthesia and operation, in order to determine whether the temperature changes which occurred would be of the magnitude known to affect the action of muscle relaxants. No attempt was made to measure the intensity or duration of action of muscle relaxants in the patients studied.

Methods

Skeletal muscle, esophageal and rectal temperatures were recorded in 23 adult patients during intraperitoneal, renal, and intrathoracic operations. Temperatures were measured in the diaphragm, intercostal, rectus abdominis, deltoid, and gastrocnemius muscles. Measurements were made with a Yellow Springs Instrument Company telethermometer using no. 513 and 514 hypodermic probes and no. 401 internal esophageal-rectal probes. These were calibrated weekly against a standard laboratory mercury thermometer graduated in 0.1-degree centigrade divisions. Temperature measurements were accurate to 0.2 degrees centigrade.

The needle probes were placed as deeply as possible within the muscles, and either remained there during the entire procedure or were intermittently inserted into the same portion of the muscle. Intercostal muscle temperatures were obtained during abdominal operations and on the unopened side of the chest during thoracic operations by inserting the needle probes into the intercostal muscles percutaneously, parallel to the ribs, in the second to sixth interspace. On the open side of the thorax the probes were inserted by the surgeon into the intercostal muscle one to four interspaces from the incision. Diaphragmatic temperature probes were inserted by the surgeon intermittently.

Esophageal temperatures were measured 40 to 48 cm. from the nostril, and rectal temperatures 8 to 10 cm. from the anus. Rectal and esophageal probes remained in place during the entire surgical procedure.

Temperatures were recorded at intervals beginning in some instances just after the induction of anesthesia, and in other instances as soon as the chest or abdomen was opened. The last temperature measurements were made just before closure of the chest or abdomen, and where possible, at the end of the operation.

Room temperatures were obtained with the no. 401 probes placed near the operative field and under the 400 watt overhead light. Relative humidity was read with 1.5 per cent accuracy on a Serdex Model 201 hygrometer. The operating rooms were air-conditioned and the temperatures ranged from 19.7 to 25.9 C. Mean room temperature was 23.1 C. The range of relative humidities was from 17.5 to 54.0 per cent, and mean relative humidity was 40.6 per cent.

All patients had a normal oral temperature preoperatively. They were brought to the operating room and anesthetized clothed in a short hospital gown and short boots, and covered with a double thickness cotton sheet. A six-minute preparation of the skin with a hexachlorophene solution was carried out, and
patients were covered with 4 small towels, 1 drapes, and a laparotomy sheet.

Preanesthetic medication consisted of a belladonna drug and usually a barbiturate or narcotic given intramuscularly about one hour before the start of anesthesia. Anesthesia was administered by closed circle and semi-closed techniques. To-and-fro methods were not used. Induction was accomplished with thiopental or cyclopropane, and anesthesia was maintained with cyclopropane, ether, nitrous oxide, halothane, and muscle relaxants in various combinations. Endotracheal tubes were inserted in all patients, and respirations were assisted or controlled.

Eleven patients required blood transfusions, varying from 500 to 2,000 ml. One patient was given 1.5 mg. of phenylephrine intravenously during operation, but no other patients received pressor drugs. No patients exhibited clinical signs of hypovolemic shock, and all survived the immediate postoperative period. The average length of operation was 126 minutes for thoracic operations and 145 minutes for abdominal and renal procedures.

Results

Decreases in muscle, esophageal and rectal temperatures were observed during all operations, but were most marked during intrathoracic procedures. As temperature changes were similar in all types of abdominal and renal operations, these were combined and compared with the results obtained in thoracic operations. The results are presented in table 1. There were no marked differences in age, physical status or anesthetic management between the abdominal and thoracic groups, and the sex of the patients did not appear to influence the results. The observed reductions in temperature could not be related to trends in room temperature or humidity, nor could the difference between the abdominal and thoracic groups be related to room temperature and humidity.

Intercostal Muscle Temperature. A downward trend in temperature was observed during anesthesia and operation, most marked in thoracic procedures, particularly on the open side of the thorax, where the mean intercostal muscle temperature reached 32.0 C. at closure. Temperatures tended to stabilize after closure was begun. In abdominal operations, the intercostal temperatures obtained at closure were significantly lower than those during induction, (P < 0.05). In thoracic operations the temperature decrease between induction and closure was highly significant (P < 0.001) on both the open and unopened sides of the chest.

Diaphragm and Rectus Abdominis Temperatures. The diaphragm cooled most markedly during thoracic operations, reaching a mean temperature of 33.7 C. just before closure. Some cooling of the diaphragm had probably occurred before opening temperatures were measured. Despite this, during thoracic procedures the decline in diaphragmatic temperatures between opening and closure was statistically significant (P < 0.01). The small temperature decreases in diaphragm and rectus abdominis muscles during abdominal procedures were not statistically significant.

Esophageal and Rectal Temperatures. Esophageal and rectal temperatures declined for about the first 100 minutes of anesthesia. They then stabilized and in about half the cases began a slow rise. Minimal temperatures were reached in most cases about the time wound closure was begun. Esophageal and rectal temperatures both fell 1.2 degrees centigrade between induction and closure in thoracic operations. The decreases are highly significant (P < 0.001). The reductions in esophageal and rectal temperature between induction and closure in abdominal operations were smaller but statistically significant (P < 0.05).

Peripheral Muscle Temperatures. Deltoide and gastrocnemius temperatures decreased throughout anesthesia and operation. The deltoide temperature fell 2.3 degrees centigrade between induction and closure in thoracic cases (P for decrease < 0.01), and 1.5 degrees centigrade in abdominal cases (P for decrease < 0.05). Gastrocnemius temperatures were considerably lower than those of the deltoide at the start. Gastrocnemius temperature reductions during anesthesia and operation were smaller than deltoide and not statistically significant.

Unusual Cases. The temperature of cold banked blood during infusion, measured in the tubing 2.0 cm. from the intravenous needle, varied from 10.8 C. to room temperature. The
TABLE 1

TEMPERATURES IN DEGREES CENTIGRADE DURING GENERAL ANESTHESIA AND OPERATION

<table>
<thead>
<tr>
<th></th>
<th>Induction</th>
<th>Thoracic Operations</th>
<th>Abdominal Operations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.E.</td>
<td>No.</td>
</tr>
<tr>
<td>Intercoastal</td>
<td>36.4</td>
<td>.20</td>
<td>8†</td>
</tr>
<tr>
<td>Open side</td>
<td>34.9</td>
<td>.42</td>
<td>5</td>
</tr>
<tr>
<td>Unopened side</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abdominal operations</td>
<td>35.4</td>
<td>.34</td>
<td>9</td>
</tr>
<tr>
<td>Diaphragm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rectus abdominis</td>
<td>36.9</td>
<td>.13</td>
<td>7</td>
</tr>
<tr>
<td>Esophagus</td>
<td>37.4</td>
<td>.15</td>
<td>5</td>
</tr>
<tr>
<td>Rectum</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deltoid</td>
<td>36.3</td>
<td>.28</td>
<td>8</td>
</tr>
<tr>
<td>Gastrocnemius</td>
<td>35.1</td>
<td>.36</td>
<td>5</td>
</tr>
</tbody>
</table>

Induction temperatures—taken within first 10 minutes of anesthesia.
Opening temperatures—taken less than 10 minutes after peritoneum or pleura was opened.
Closing temperatures—taken less than 10 minutes before peritoneum or pleura was closed.
End temperatures—taken during skin closure.
* Upper abdominal cases only.
† Measured in patients who underwent abdominal operations, and on the unopened side in patients who underwent thoracic operations.
TEMPERATURES IN MAN DURING ANESTHESIA

blood became warmer during its passage through the tubing, and the lower temperatures were observed with more rapid flow rates. The infusion of relatively small amounts of cold blood rapidly (up to 400 ml in 10 minutes), or of larger amounts more slowly (up to 2,000 ml in two hours), resulted in no temperature decreases greater than those observed in other patients who were not transfused.

Unreplaced blood loss of 500 to 700 ml likewise produced no clear-cut temperature changes, so long as blood pressure was maintained at the patient's normal level. However, marked temperature decreases were observed in patients who became hypotensive or received massive transfusions of cold blood, and precipitous temperature declines have been measured in children. Several illustrative case histories follow:

**Case Reports** *

**Case 1.** A 65 year old hypertensive man who had had a recent myocardial infarction was brought to the operating room in borderline shock with a ruptured aneurysm of the abdominal aorta. His normal blood pressure was 200/110. During the seven-hour operative procedure 9,000 ml of cold blood were infused. Systolic blood pressure fell below 100 mm of mercury several times, and was once unobtainable for several minutes. During the last 2 hours of operation, esophageal temperature ranged from 33.2 to 33.5 C. Eight hours after the end of operation, rectal temperature had returned to 37.7 C.

**Case 2.** A 67 year old woman with a large hepatic cyst underwent left hepatic lobectomy. Brisk bleeding was encountered and 3,800 ml of cold blood and 200 ml of fresh frozen plasma were infused into left arm veins in a three-hour period. Lower esophageal temperature decreased to 33.7 C. Intercoastal muscle temperature was 32.6 C. Left deltoid temperature was 27.4 C and right deltoid was 32.9 C. Shivering did not occur during the four-hour operation, but was noted in the recovery room. Heat lamps were applied and rectal temperature returned to 37.3 C., 90 minutes after the end of operation.

**Case 3.** A 6 week old baby girl weighing 5 1/2 pounds had a ventriculoo-jugular shunt operation for communicating hydrocephalus. She was anesthetized with nitrous oxide and ether using a Y-tube and high flow rates. Fifteen minutes after anesthesia was started rectal temperature was 36.1 C. One hour later it had fallen to 33.3 C. Hot water bottles were applied during operation and in the recovery room, and rectal temperature was back to 36.1 C. in several hours. Shivering was not present until the patient had recovered from anesthesia.

**DISCUSSION**

Heat gain by anesthetized patients has been described by a number of workers. Certain conditions are recognized which favor heat gain. These include: an operating room temperature above 25 C., a high relative humidity in the operating room, use of uncooled to-and-fro canisters, multilayered surgical draping and severe dehydration of the patient. Less important sources of heat are the operating room lights and warm laparotomy pads.

The fact that heat loss may occur during operation has also been realized, for infants; and for all patients if the operating room is cool. Our data indicate that heat loss is perhaps more common than has been recognized, particularly in air-conditioned operating rooms. During operations other than those within the thorax the decreases in temperature which we found were not great, except under the special circumstances reported. During intrathoracic procedures, however, the degree of heat loss was greater. In these operations the mean temperature of the exposed intercostal muscles decreased to 32.0 C. and that of the diaphragm to 33.7 C.

Heat production may have decreased in our patients as the result of an over-all reduction of metabolism by general anesthesia, and the lessened heat production in curarized skeletal muscle. Heat loss can be considered in the standard fashion—the loss of metabolic heat due to heat production in the skin produced by general anesthesia. Radiation, the principal means of heat loss at comfortable room temperature, probably accounts for a considerable portion of the heat loss in the patients we studied.

Evaporative heat loss by sweating is small at comfortable room temperature. The depression of sweating caused by preanesthetic medication with belladonna drugs is probably of little significance in minimizing heat loss. Evaporative heat losses may occur during the six-minute preparation of the skin with cool liquids, and from exposed moist surfaces in the
wound. The greater heat loss during chest operations may be related in part to evaporation from the relatively large surface area of moist tissues exposed by the thoracic incision. Additional heat loss occurs when the inhaled gases are saturated with water vapor in the lungs. This loss accounts for about 1.9 Calories per hour in a closed circle system, and increases with the flow rate, reaching 9.8 Calories per hour in a nonrebreathing system. This loss may be compared to the basal heat production of about 70 Calories per hour in an average young male.

A rather small amount of heat is lost by conduction to the operating table mattress, to cold instruments in the wound, to the inhaled gases and to the room air. If the air is in motion, as it is in air-conditioned operating rooms, there is heat lost by convection. This process is retarded by the drapes, but can take place from the wound. The opened chest is an area where this phenomenon can occur. The muscles of respiration therefore are probably cooled by radiation, evaporation and convection during their exposure to room air for relatively long periods.

The infusion of a liter of intravenous fluid at room temperature would result in a heat loss of about 15 Calories. More marked heat losses can be related to the infusion of large volumes of cold blood.

One more factor which affects thermal equilibrium during operation is the depression of central thermoregulatory mechanisms, which is associated with general anesthesia. The poor development of these mechanisms in infants is also of clinical importance.

The contribution to total heat loss of many of the factors discussed above is relatively small, and it would be difficult to isolate the effect of each in a clinical study such as this. The important fact is that the total heat loss may be considerable under normal conditions and can be marked in special circumstances.

During intrathoracic operations the temperature of the muscles of respiration was found to reach levels which have been shown to prolong and intensify depolarization blockade, and antagonize d-tubocurarine blockade of the neuromuscular junction in man. Muscle temperature decreases, therefore, may provide an explanation for some cases of prolonged apnea following the use of succinylcholine or decamethonium. Rewarming would appear to be a rational therapeutic measure in the treatment of such apnea.

The muscle temperature changes which occurred normally during abdominal operations were not sufficient to affect neuromuscular blockade to any important degree. However, as shown in the case histories, in some situations temperatures can decrease markedly during operation, and the effects on the action of depolarizing muscle relaxants might be profound. We suggest that esophageal temperatures be monitored in those patients who are likely to incur large temperature decreases during operation. Although rectal temperature parallels heart temperature until 35.0 C. is reached, and if changes occur slowly, a lag occurs at lower temperatures and with a more rapid rate of fall. The best estimate of heart temperature appears to be that obtained in the lower esophagus.

Summary

Skeletal muscle, esophageal, and rectal temperatures were measured in 23 adult patients during general anesthesia and operation. Temperatures decreased in all cases, more markedly during intrathoracic operations.

During intrathoracic procedures the temperatures of the respiratory muscles reached levels at which the actions of succinylcholine and decamethonium should be prolonged and intensified. This temperature reduction in the muscles of respiration might cause prolonged apnea. Rewarming of patients is suggested as a reasonable therapeutic procedure if apnea persists under such circumstances.

During routine intra-abdominal procedures the decrease in muscle temperature was not great enough to affect the action of muscle relaxants significantly.

Case histories were presented illustrating the precipitous temperature decreases which can occur in certain circumstances. Monitoring of temperature is suggested in cases where extraordinary heat loss is likely.

This work was supported (in part) by a grant from the John A. Hartford Foundation, Inc., New York, and by grants from the National Heart Insti-
TEMPERATURES IN MAN DURING ANESTHESIA

REFERENCES


QUINIDINE Ventricular fibrillation in dogs with oesophageal temperatures reduced from 5-10 degrees C. by core cooling with a pump oxygenator and heat-exchanger system can be readily prevented by the intravenous infusion of quinidine. It appears that fibrillation during hypothermia is due to the uneven cooling and re-warming within the heart with areas of different metabolic and electrolytic environment. (Johnson, P., and others: Prevention of Ventricular Fibrillation During Profound Hypothermia by Quinidine, Ann. Surg. 151: 490 (April) 1960.)

DIGITALIS In normal subjects whose cardiac output was studied during walking exercise by means of earpiece oximetry, Lanatoside-C did not measurably alter the "normal" response of the cardiac output to exercise. (Goodyer, A. V. N., Chetrick, A., and Hucos, A.: Use of Ear Oximeter for Measurement of Cardiac Output during Walking Exercise in Human Subjects, Yale J. Biol. & Med. 32: 265 (Feb.) 1960.)

LEVARTERENOL Levarterenol-induced hypertension may slow, increase, or produce no change in the cardiac rate of persons with cardiovascular diseases. The degree of cardiac disability at least partly determines the type of response. (Soloff, L. A., Cortez, F., and Winters, W. L.: Importance of Myocardial Reserve in Determining Changes in Heart Rate During Lecarteronel Infusion, Circulation Res. 8: 377 (March) 1960.)

CORTISONE A death rate of 37 per cent resulted in guinea pigs under decompression to a simulated altitude of 25,000 feet for 6 hours. This mortality rate was increased to 50 per cent and to 75 per cent when morphine sulphate (2.5 mg.-5 mg./100 Gm. body weight, respectively) was injected intraperitoneally 10 minutes before the decompression was started. Prior treatment with cortisone for 3 days reduced mortality to 22 per cent and 29 per cent, respectively. Multiple injections with ACTH reduced the death rate to 19 per cent. (Sobel, H., and others: Effect of Cortisone on Survival of Morphine Treated Guinea Pigs under Decompression Hypoxia, Proc. Soc. Exp. Biol. & Med. 104: 31 (May) 1960.)