Undersea Biomedical Research, Vol. 11, No. 3, September 1984

Physiological effects of ventilation with liquid fluorocarbon at controlled temperatures

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Shaffer TH, Forman DL, Wolfson MR. Physiological effects of ventilation with liquid fluorocarbon at controlled temperatures. Undersea Biomed Res 1984; 11(3):287–298.—Body cooling, respiratory heat loss, and the physiological effects of liquid ventilation at various temperatures were studied in 10 adult cats with applications to the deep sea diver. The animals were stabilized on mechanical gas ventilation with 100% oxygen during a control period and then mechanically ventilated for 1 h with liquid fluorocarbon. Fluorocarbon (Rimar 101) temperatures of 10°C, 20°C, and 30°C were used to ventilate the animals while rectal and subcutaneous body temperatures were being measured. For the 3 temperature conditions, respective cooling rates of 9.0°C/h, 7.8°C/h, and 3.6°C/h, as well as respiratory heat losses of 65,637 $J \cdot kg^{-1} \cdot h^{-1}$, 33,488 $J \cdot kg^{-1} \cdot h^{-1}$, 18,036 $J \cdot kg^{-1} \cdot h^{-1}$ were observed while maintaining effective physiological gas exchange [mean Pa_{O_2} = 353 ± 28 (SEM) mmHg, mean Pa_{CO_2} = 30 ± 2 (SEM) mmHg]. Changes in cardiovascular variables were noted as mild (35°C–30°C) and moderate (30°C–25°C) levels of hypothermia were reached. Cardiac output, oxygen consumption, heart rate, and mean blood pressure were significantly correlated with rectal temperature. The data presented herein quantitate the effects of liquid ventilation on body cooling and respiratory heat loss. Furthermore, the physiological alterations associated with the observed hypothermic condition could severely limit the effectiveness of a human diver if not carefully controlled.

liquid ventilation
hypothermia
fluorocarbon
cooling rates

In 1958 Kylstra introduced the concept of liquid breathing in mammalian species (1). Since then he and other investigators have shown that liquid breathing could provide a possible alternative to air breathing in a man escaping and ascending to the surface from great depths (2, 3). Maintenance of thermal balance, however, is a major problem for the diver at depths exceeding 19.2 ATA (600 fsw) (4). In this environment heat lost via the respiratory tract outstrips that produced by metabolism, and hypothermia may result unless some method of heat replacement is provided (4).

Theoretically, ventilation with a cool liquid should produce more rapid body cooling and respiratory heat loss than a gas, because of the greater thermal conductivity of a liquid as compared to a gas. However, quantification of the degree and rate of hypothermia during liquid ventilation has not been experimentally established to date. Furthermore, there are few quan-

titative physiological data in the existing literature that differentiate the effects on the body of breathing cooled liquids from the effects of hypothermia induced by other methods. The present study investigates the effects of ventilation with fluorocarbon liquid on body cooling and respiratory heat loss while controlling the temperature of the inspired liquid. In addition, the cardiovascular responses to both hypothermia and liquid ventilation are characterized.

MATERIALS AND METHODS

Animal preparation

Anesthesia was induced intraperitoneally with pentobarbitol sodium (30 mg/kg) in 10 adult cats [mean wt = 2.4 ± 0.2 (SEM) kg]. Each animal was secured in the supine position, intubated (National Catheter Co., NY, 3.0 mm i.d.) and catheterized. A French No. 7 polyvinly catheter was inserted into the right external jugular vein and positioned in either the right ventricle or superior vena cava for blood sampling and measurement of central venous pressure (CVP). Catheter placement was confirmed by the pressure tracing. A French No. 5 catheter was inserted into the left carotid artery for mean arterial pressure (MAP) measurements. Finally, four subcutaneous needle electrodes were inserted for electrocardiographic (ECG) tracings and heart rate measurements.

Methods and experimental protocol

After instrumentation, the animal was paralyzed with either tubocurarine hydrochloride (2.0 mg/kg) or Anectine hydrochloride (Burroughs-Wellcome; 2.0 mg/kg) and mechanically ventilated with 100% oxygen with a Harvard small animal respirator (Harvard apparatus, South Natick, MA). During this period [65 ± 2 (SEM) min] we obtained control values for all variables. Following the control period, the animal was transferred to the liquid system and maintained for 1 h. Samples of both venous and arterial blood (1 ml) were analyzed at 10-to-20-min intervals during both control and experimental phases. A Radiometer (Copenhagen, Denmark) blood gas analyzer with membrane-covered O_2 and CO_2 electrodes, and a glass capillary electrode for pH, was used to measure Pa_{O_2}, Pa_{CO_2}, and pH. The electrodes were housed in constant-temperature bath cuvettes maintained at 37°C. All measurements were temperature corrected (5). Base-line blood gas and pH analyses during the control period ensured adequate gas exchange prior to liquid ventilation.

Oxygen consumption was measured using a closed loop system integrated into the liquid ventilator (6). Expired liquid flowed through a membrane oxygenator, allowing it to spread out and approach film flow. As carbon dioxide was stripped from the liquid, a countercurrent stream of 100% oxygen gas reoxygenated the fluorocarbon. The exhaust gas from the membrane oxygenator passed through a soda-lime chamber that removed carbon dioxide. The remaining gas solution, containing excess oxygen and vaporized fluorocarbon, flowed to the reservoir, thus completing the oxygenation loop.

As oxygen was being used by the animal, additional oxygen was supplied from a spirometer attached to the loop at a point downstream from the soda-lime chamber. A potentiometer attached to the spirometer transformed spirometer volume changes into electrical signals, which were recorded on a Varian (Varian Associates, Palo Alto, CA) model 9176 strip chart. Oxygen uptake (in ml/min) was calculated from the base-line change in the spirometer volume.

The spirometer-recorder system was also used for control measurements of oxygen consumption during gas breathing. This was achieved by connecting an auxiliary gas breathing circuit to the spirometer outlet tube (6). After obtaining oxygen consumption measurements, cardiac output was subsequently calculated using the Fick principle, i.e., oxygen consumption divided by the difference in oxygen contents of the blood samples withdrawn for blood gas analysis. Oxygen content was calculated from the following equation:

$$\text{oxygen content} = (Po_1 + sHbO_2) \times 100$$

where P = Pa_{O_2}, O_1 = 0.003 ml oxygen · $mmHg^{-1}$ · 100 ml^{-1} blood, s = % oxygen saturation, Hb = 16 g hemoglobin/100 ml blood, and O_2 = 1.34 ml oxygen · g^{-1} hemoglobin · 100 ml^{-1} blood.

Pressure measurements were transmitted to Statham P23 PB (MAP) and P23 DB (CVP) strain-gauge transducers (Statham Instruments, Oxnard, CA) and recorded on a Grass model 7 polygraph recorder incorporating model 7P1F low-level DC preamplifiers (Grass Instrument Co., Quincy, MA). Deflections in the ECG were also recorded on the Grass instrument using a model 7P10CD integrator/amplifier. Body temperature measurements were determined at 10-to-20-min intervals from a rectal thermistor (core), and a surface thermistor (skin). The latter was secured in a subcutaneous incision on the right thigh. Cooling rates were assessed from animals breathing 30°C fluorocarbon (Group I, n = 3), 20°C fluorocarbon (Group II, n = 4) and 10°C fluorocarbon (Group III, n = 3). Respiratory heat loss (ΔQ) was determined using the following equation: $\Delta Q = \rho mCp\Delta T$ where ρ = density of fluorocarbon (1.78 g/ml), m = mass of inspired tidal volume (ml), Cp = specific heat of fluorocarbon (1045.5 J · kg^{-1} · $°C^{-1}$), and ΔT = temperature difference between inspired and expired fluorocarbon. The latter was measured using two thermistors inserted into the inspiratory and expiratory liquid lines respectively. These thermistors were placed as close to the animal's endotracheal tube as possible to minimize error due to heat loss within the connecting lines of the liquid ventilation system.

Liquid ventilation procedure

Liquid ventilation with Rimar fluorocarbon (Rimar Chimica S.p.A., Vicenza, Italy) was instituted using a previously described but modified liquid-breathing system (6, 7). The modified system is shown in Fig. 1. Fluorocarbon temperature was adjusted appropriately by means of an apparatus for external water-bath circulation and heat exchange. To ensure skeletal muscle paralysis each animal was given a supplemental dose of tubocurarine or Anectine prior to liquid breathing. The endotracheal tube of the animal was then connected to the liquid system.

After an estimated functional residual capacity (FRC) volume of liquid was instilled into the animal's lungs, thoracic manipulations were performed to remove any oxygen that had become trapped during the transfer. This gas escaped into the system and was subsequently removed with a large syringe. Since the animal was previously ventilated with 100% oxygen, it was assumed that all gas remaining in the lungs was oxygen or carbon dioxide. Thus any residual gas could either be absorbed by the liquid or utilized by the animal, since little or no bubbling was observed through the clear polyvinyl tracheostomy tube. In addition, the animal rested on a strain gauge weight platform accurate to within 1 ml of fluorocarbon. This enabled us to monitor tidal volume (V_T) and FRC while the animal was breathing liquid. The liquid lung volumes were recorded on the Grass instrument using model 7P122B low-level DC amplifiers. Blood gases and pH were monitored throughout the experiment, and the ventilation scheme [i.e., V_T, FRC, and inspiratory-to-expiratory (I:E) frequency] was adjusted so as to maintain adequate tissue oxygenation and carbon dioxide elimination.

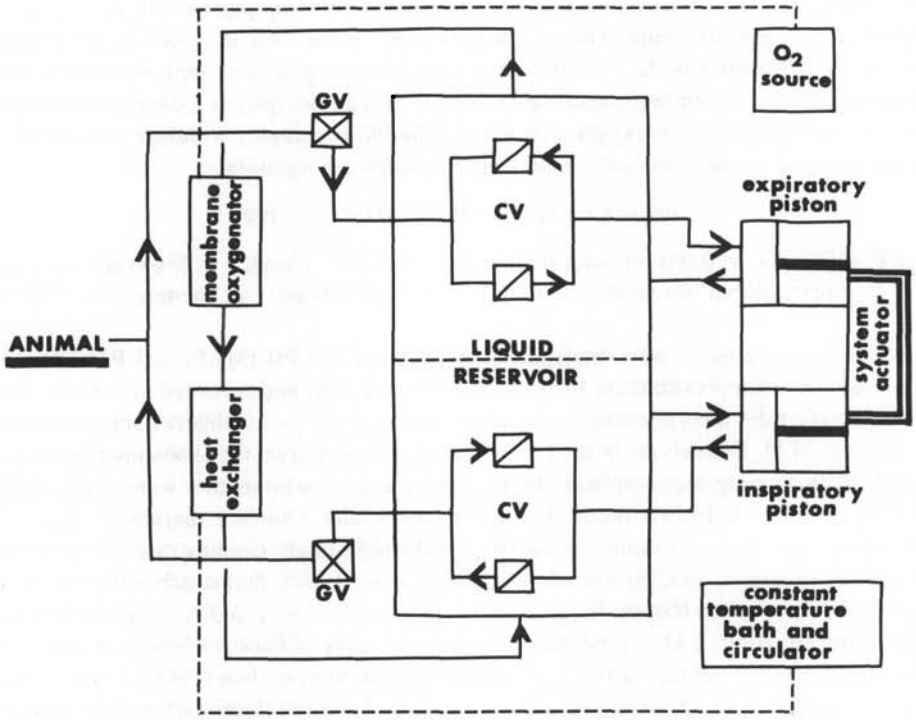

Fig. 1. Schematic drawing of modified liquid ventilation system.

Mean core and surface temperatures ± SEM were correlated with time for Groups I, II, and III. Linear regression analysis was performed on data from each group, and the cooling rates were calculated from the slope of each curve. Cardiovascular variables (i.e., heart rate, cardiac output, mean arterial pressure, and oxygen consumption) were each correlated with core temperature. The data presented for each variable reflects the combined measurements from all three groups of animals (n = 10). Linear regression provided the best-fitted curves for these data sets as well.

RESULTS

Measurements obtained during the gas ventilation control period indicate that the animals were stabilized with respect to temperature, gas exchange, and cardiovascular function. In Table 1, both heart rates [164.0 ± 4.0 (SEM) beats/min] and mean arterial pressures [MAP = 119.0 ± 0.7 (SEM) mmHg] were within normal ranges for an anesthetized cat. Furthermore, cardiac output [$\dot{Q}_T$ = 580.0 ± 62.0 (SEM) ml/min], and oxygen consumption [$\dot{V}_{O_2}$ = 34.2 ± 2.0 (SEM) ml/min] measurements were in keeping with previously reported values (6, 7). We noted a slight but stable depression in core temperature, 36.0 ± 0.2 (SEM)°C, which might be attributed to the effect of the induction anesthesia. Adequate physiological gas exchange was also maintained during the control period as indicated in Table 2. Hyperventilation with 100% oxygen eliminated nitrogen from the lungs and served to elevate arterial oxygen, [Pa_{O_2} = 353 ± 28 (SEM) mmHg] as well as to depress arterial carbon dioxide [Pa_{CO_2} = 30 ± 2 (SEM)

TABLE 1
GAS VENTILATION CONTROL DATA

Parameter	Unit	Mean Value ± SEM
Weight	kg	2.4 ± 0.2
Rectal temperature	°C	36.0 ± 0.2
Heart rate	beat/min	164.0 ± 4.0
Mean arterial pressure	mmHg	119.0 ± 0.7
Oxygen consumption	ml/min	34.2 ± 2.0
Cardiac output	ml/min	580.0 ± 62.0

All data are based on observations from 10 adult cats.

TABLE 2
BLOOD CHEMISTRY DATA DURING GAS AND LIQUID VENTILATION

Rectal Temperature, °C	Pa_{O_2}, mmHg	Pa_{O_2}, mmHg	pH
Gas ventilation			
36.0 ± 0.1	353 ± 28	30 ± 2	7.32 ± 0.02
Liquid ventilation			
34.0 ± 0.2	216 ± 23	34 ± 2	7.29 ± 0.02
31.3 ± 0.2	240 ± 22	32 ± 2	7.20 ± 0.02
29.0 ± 0.2	216 ± 38	32 ± 3	7.14 ± 0.03
27.2 ± 0.2	233 ± 44	27 ± 2	7.14 ± 0.04
23.6 ± 0.5	281 ± 49	28 ± 2	7.19 ± 0.03

All values based on observations from 10 adult cats and presented as means ± SEM.

mmHg]. The arterial pH during the control period [7.32 ± 0.02 (SEM)] remained within normal limits.

The temperature data obtained from Group I (30°C fluorocarbon), Group II (20°C fluorocarbon), and Group III (10°C fluorocarbon) are illustrated in Figs. 2 and 3 and linear regression coefficients are shown for each fitted line. The calculated cooling rates, measured during the 10-to-60-min interval of liquid ventilation, reflect the slope of each fitted curve shown. Core temperature for Group I decreased at a rate of 0.06°C/min ($R = 0.99$, $P < 0.01$) reaching mild hypothermia (35°C–30°C) within 20 min and settling at 32.6°C after 1 h of liquid ventilation. The core temperatures for Groups II and III, however, declined more rapidly at rates of 0.13°C/min ($R = 0.99$, $P < 0.01$) and 0.15°C/min ($R = 0.99$, $P < 0.01$) respectively. These animals reached the moderate hypothermic level (30°C–25°C) after about 40 min of liquid ventilation. In the next 20 min, core temperature continued to fall, reaching 26.5°C at the end of the experimental period. Surface cooling rates for Groups I, II, and III were 3.0°C/h ($R = 0.99$, $P < 0.01$), 4.2°C/h ($R = 0.98$, $P < 0.01$), and 5.4°C/h ($R = 0.99$, $P < 0.01$). Ambient temperature was maintained relatively constant at 22.1 ± 0.5 (SEM)°C.

Table 2 shows the calculated respiratory heat loss (ΔQ) for all groups. It is apparent by inspection that as the inspired liquid temperature was reduced, the amount of heat loss increased. Heat loss per breath during the first 20 min of liquid ventilation was 68.0 ± 13.5

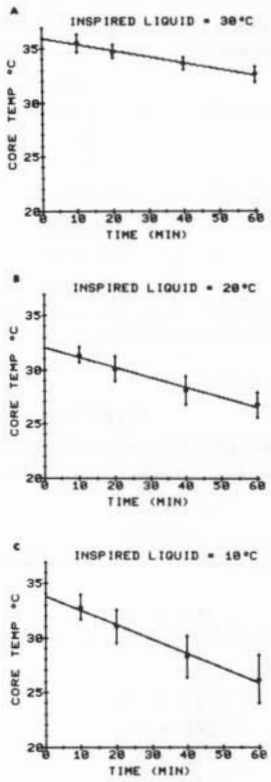

Fig. 2. Mean core temperature ± SEM as function of time for inspired liquid temperatures. *A*, 30°C; *B*, 20°C; *C*, 10°C. Line represents best fit using least squares method.

(SEM) joules per breath per kilogram ($J \cdot br^{-1} \cdot kg^{-1}$) for an inspired liquid temperature of 30°C, and inspired liquid temperatures of 20°C and 10°C resulted in respiratory heat losses of 122.6 ± 8.5 and 311.3 ± 42.6 (SEM) $J \cdot br^{-1} \cdot kg^{-1}$ respectively.

The cardiovascular changes in all 10 animals are illustrated in Fig. 4 as means, plus or minus standard error of the mean (SEM). Cardiac output measurements assessed during the liquid ventilation period are presented as a percentage of control values. With the induction of hypothermia, the cardiac output declined linearly as a function of temperature. Regression analysis indicated a fall of about 5% $\dot{Q}_T$/°C ($R = 0.87$, $P < 0.05$). The mild hypothermic state was characterized by a 40% decrease in cardiac output, and moderate hypothermia resulted in a 70% decrease.

Oxygen consumption ($R = 0.82$, $P < 0.001$) and heart rate ($R = 0.99$, $P < 0.001$) were also found to decrease linearly with temperature. Oxygen consumption decreased 36% and heart rate decreased 35% with mild hypothermia. During moderate hypothermia, oxygen consumption was characterized by a fall of 57%, and heart rate fell 59%. At temperatures below 30°C, ECG irregularities were observed and complete cardiac arrest occurred in three animals. In addition, mild hypothermia resulted in a 20% decline in mean arterial pressure, while the moderate hypothermic stage was characterized by a 40% decrease.

Adequate physiological gas exchange was also maintained during the liquid phase of the experiment as indicated by blood gas data in Table 2; $Pa_{O_2} = 281 - 216$ mmHg, $Pa_{CO_2} = 34 - 27$

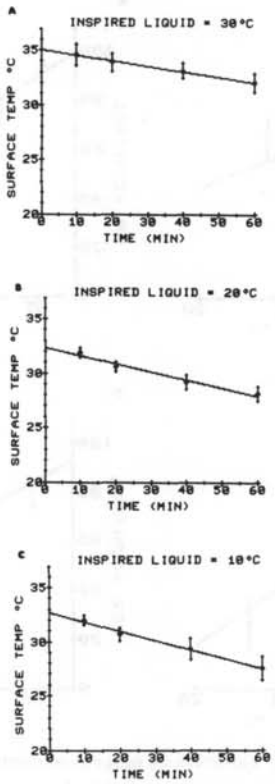

Fig. 3. Mean surface temperature ± SEM as a function of time for inspired liquid temperatures. *A*, 30°C; *B*, 20°C; *C*, 10°C. Line represents best fit using least squares method.

mmHg. During this period, however, the pH decreased, indicating the development of an expected metabolic acidosis.

DISCUSSION

One of the major problems facing the deep sea diver is maintenance of thermal balance. The diver must maintain his metabolic heat production while minimizing his heat loss to the environment, creating a steady-state equilibrium. Currently, heliox breathing is used to reduce the work of breathing at depth; however, the high thermal conductivity of helium (1.56 W · $cm^{-1} \cdot °C^{-1} \times 10^{-4}$) results in more rapid heat transfer than less-conductive gases (8). Hence, a steady-state equilibrium is more difficult to maintain. Rawlins and Tauber (4) have noted that a resting diver breathing heliox at 19.2 ATA (600 fsw) is in thermal equilibrium where both heat production and heat loss equal 125 W. At depths greater than 19.2 ATA, however, maintenance of thermal balance becomes increasingly difficult as more heat is lost via convection and conduction through the respiratory tract, resulting in a negative thermal balance (4, 9). Under these conditions hypothermia develops and the diver is faced with serious physiological alterations. As underwater research moves deeper into the sea the need has arisen for development of new concepts for maintenance of the human diver.

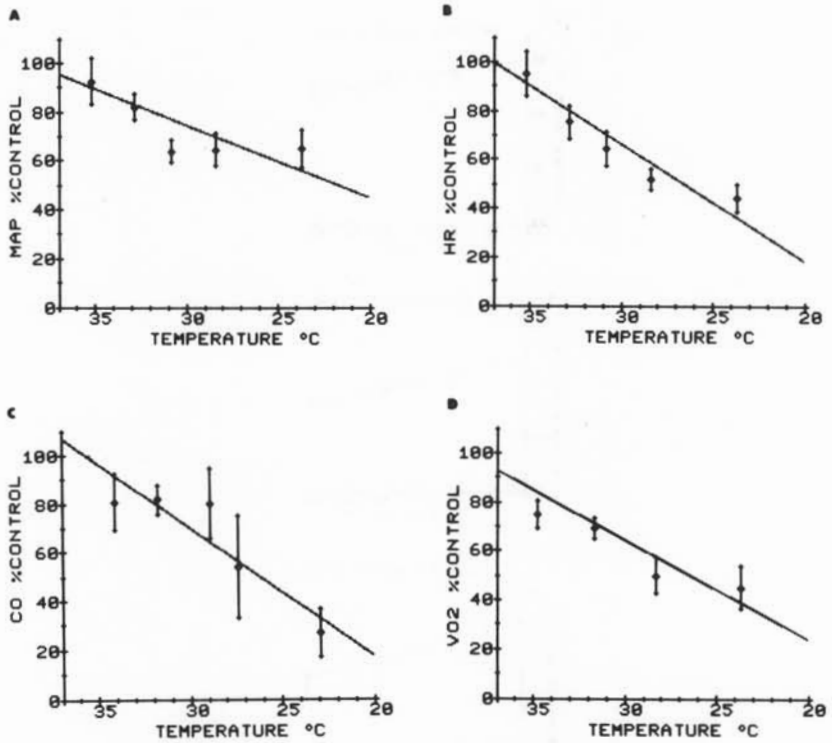

Fig. 4. Mean ± SEM cardiovascular variables as percent of control values compared to core temperature. Line represents best fit using least squares method. *A*, mean arterial pressure; *B*, heart rate; *C*, cardiac output; *D*, oxygen consumption.

Kylstra's studies with hyperbaric liquid breathing introduced the unique idea of using a liquid medium to ventilate divers at great depths (1, 2). He suggested that the use of liquid ventilation in this environment may help alleviate the problem of decompression after surfacing (3). In support of this hypothesis, Gollan and Clark (10) have shown that mice can survive rapid decompression after breathing liquid fluorocarbon. In addition, Lynch et al. (11) reported similar findings describing a reduction in the incidence of decompression sickness in hamsters who were decompressed from 7 ATA at a rate of 1.8 bar/min (60 ft/min) after breathing fluorocarbon liquid.

If liquid ventilation is used at great depths, however, the temperature of the breathing medium is also an important consideration, since convective and conductive heat loss through the respiratory tract may be exaggerated because of the differences in thermal properties between a liquid and a gas. This study quantitates the degree of respiratory heat loss and decrease in body temperature that would be expected while breathing liquids at various temperatures. In addition, the cardiovascular alterations resulting from the combination of liquid ventilation and hypothermia are discussed.

As noted in Table 3, respiratory heat loss increased with decreasing inspired liquid temperature. Furthermore, the amount of total heat lost increased almost fourfold with only a twofold drop in inspired liquid temperature. In examination of the respiratory heat loss for all three groups it is apparent that the amount of heat loss decreases with time as the temperature gradient (expiratory minus inspiratory temperature) decreases. Moskowitz and Greiner (12), in their study with lung lavage at 25°C, produced a respiratory heat loss of 118 $J \cdot br^{-1} \cdot kg^{-1}$

and 2,700 J · h^{-1} · kg^{-1}. In the present study, the first 10 min of liquid ventilation with 20°C fluorocarbon resulted in a respiratory heat loss of 122 J · br^{-1} · kg^{-1} and 33,488 J · h^{-1} · kg^{-1}. Although the heat loss per breath was comparable in both studies, the increased heat loss per hour may be explained by the fact that our average breathing frequency was 303 br/h, whereas in the unilateral lung lavage study the ventilation frequency was only 23 br/h.

After 10 min of liquid ventilation, the mean core temperature of Group I fell to about 35°C and Groups II and III dropped to nearly 33°C. Previous studies have indicated that amnesia may occur with the onset of mild hypothermia in humans (13). Therefore, if a diver were breathing liquid fluorocarbon cooled to a temperature of 10°C by the seawater (9), his body temperature would reach a critical level in less than 1 h, thus compromising his mental acuity.

Nervous system changes during hypothermia may be further complicated by changes in the cardiovascular system. It has been well documented that cardiac irregularities begin to develop with the induction of moderate hypothermia in humans (14). The slowed pacemaker firing and conduction defects resulting from cold exposure produce slowed heart rates and an abnormal ECG tracing (15). Our data support this finding, as indicated by the linear decline in heart rate during the experimental period and the increasingly irregular ECG tracings noted with decreasing core temperature. Since no significant alterations in heart rate or ECG have been documented during normothermic liquid ventilation (6, 7) the changes in these parameters may be attributed to the hypothermic condition.

During hypothermic gas breathing, most of the heat loss occurs via the conducting airways such that a fairly constant temperature is maintained within the alveolar space. During liquid breathing, however, the convective heat transfer in the large airways may not be sufficient to warm the ventilating medium before it reaches the smaller airways; thus, significant temperature gradients may develop at the interface of liquid and blood in the terminal alveoli. Since the pulmonary circulation as well as the bronchial circulation will be in close contact with cold

TABLE 3
CALCULATED RESPIRATORY HEAT LOSS

Time, min	Ti, °C	Te, °C	V_T, ml	*f*, breath/min	ΔQ, J·$breath^{-1}$·kg^{-1}	Total ΔQ, J·kg^{-1}
Group I						
0–20	31.6 ± 0.4	34.2 ± 0.4	40 ± 2.7	5.4 ± 0.2	68.0 ± 13.5	7344
21–40	31.4 ± 0.4	33.4 ± 0.5	43 ± 3.6	5.4 ± 0.2	59.0 ± 12.0	6056
41–60	31.4 ± 0.4	33.0 ± 0.5	44 ± 3.8	5.4 ± 0.2	45.2 ± 9.3	4636
Accumulated ΔQ						18,036
Group II						
0–20	21.0 ± 0.3	25.1 ± 0.7	30 ± 1.8	5.3 ± 0.4	122.6 ± 8.5	12,996
21–40	20.8 ± 0.2	24.8 ± 0.5	35 ± 2.7	5.3 ± 0.4	109.8 ± 10.1	11,057
41–60	20.0 ± 0.2	23.4 ± 0.5	36 ± 2.2	5.3 ± 0.4	93.7 ± 10.9	9,435
Accumulated ΔQ						33,488
Group III						
0–20	12.1 ± 0.5	24.6 ± 1.2	32 ± 2.9	4.5 ± 0.3	311.3 ± 42.6	28,017
21–40	11.0 ± 0.5	20.5 ± 0.2	30 ± 6.8	4.5 ± 0.3	256.2 ± 73.3	21,905
41–60	10.7 ± 0.7	18.0 ± 0.4	30 ± 6.8	4.5 ± 0.3	183.8 ± 48.0	15,715
Accumulated ΔQ						65,637

All data are presented as mean values ± SEM. Ti, of inspired temperature liquid. Te, temperature of expired liquid. V_T, tidal volume. *f*, Breathing frequency. ΔQ = respiratory heat loss.

fluorocarbon, a potential mechanism exists for cooling of the entire cardiac output (16). In fact the present study as well as heat transfer studies with unilateral lung lavage (12) have shown that fluorocarbon cooled to 20°C to 25°C gains approximately 0.12°C $\cdot\cdot br^{-1} \cdot kg^{-1}$ and 0.11°C $\cdot\cdot br^{-1} \cdot kg^{-1}$ respectively during the first 20 min of liquid ventilation.

Currently, the methods used clinically to induce hypothermia require cooling of the shell (17–19), the inhalate (20, 21), or the dialysis medium (22, 23) to very low temperatures (0°C), which produce uneven temperature distribution with a tendency to create large temperature gradients within the body (18, 19). The cooling rates for noninvasive induction techniques, calculated from reported data, range from 12.2°C/h (19) to 7.3°C/h (21), whereas invasive techniques using cardiopulmonary bypass may produce cooling rates up to 30°C/h (24). Although many of these studies did not control for anesthetic cooling, the raw data show relatively stable temperatures during the anesthetic control period. Therefore we have used these data to compare our cooling rates. In the present study inspired liquid temperatures of 10°C, 20°C, and 30°C produced comparable cooling rates of 7.8°C/h, 5.7°C/h, and 3.4°C/h respectively. Thus much smaller temperature gradients at the liquid/blood interface were able to produce cooling rates similar to those used clinically to induce hypothermia. Furthermore, because fluorocarbon liquid has a heat transfer capacity 20 times that of air at sea level, these small temperature gradients at the liquid/blood interface can result in significant heat loss (9).

Previous reports have shown that mean arterial pressure and cardiac output fall as the moderate hypothermic level is reached; however, peripheral compensation seems to be intact until the body temperature reaches about 25°C (25, 26). In fact, total peripheral resistance has been shown to increase as much as sixfold during deep hypothermia (25). In studies using normothermic liquid ventilation, however, mean arterial pressure was reported constant in spite of a 40% decrease in cardiac output, indicating that the liquid ventilation procedure itself does not significantly alter the peripheral regulatory mechanisms (6, 7). The data presented in this report showed a progressive decline in mean arterial pressure and cardiac output from the onset of hypothermia. Therefore it appears that the combination of hypothermia and liquid ventilation results in a decline in cardiovascular function that cannot be maintained by normal regulatory mechanisms.

Under the right conditions, ventilation with liquid fluorocarbon may decrease the incidence of decompression sickness (11). Moreover, because liquid breathing produces minimal changes in lung structure (27, 28) and function (29–31), it may provide a feasible alternative to the air-breathing man at great dephths. However, a diver may develop a hypothermic condition very quickly unless the temperature of the liquid is carefully controlled. A diver's performance and decision-making capabilities are critical at great depths and are dependent on maintenance of normal body temperature.

The authors are indebted to Dr. Peter Lynch for his helpful criticism. The authors are also grateful to the Mercantile Development Inc., for supplying the fluorocarbon liquid (Rimar 101) for this study. This work was supported in part by Public Health Service Grant HL-22843 and NHLI Grant HL-07414(04) and presented in part at American Physiological Society Meeting, San Diego, CA October 1982.—*Manuscript received for publication April 1983; revision received August 1983.*

Shaffer TH, Forman DL, Wolfson MR. Effets physiologiques de la ventilation avec le fluorocarbone liquide à des températures contrôlées. Undersea Biomed Res 1984; 11(3):287–298.—Le refroidissement du corps, la perte de chaleur par les voies respiratoires et les effets physiologiques de la ventilation liquidienne ont été étudiés à diverses températures chez 10 chats adultes en vue d'applications au plongeur en eau profonde. Les animaux furent d'abord stabilisés sous respiration gazeuse artificielle avec de l'oxygène à 100% pendant une période de contrôle, puis ventilés mécaniquement durant 1 h avec du fluorocarbone liquide. Le fluorocarbone (Rimar 101) fut utilisé à des températures de 10°C, 20°C, et 30°C pour ventiler les animaux, et les températures rectale et

souscutanée étaient mesurées en même temps. Pour les trois conditions de température. des taux respectifs de refroidissement de 9.0° C/h, 7.8° C/h, et 3.6° C/h, ainsi que des pertes de chaleur par voies respiratoires de 65,637 $J \cdot kg^{-1} \cdot h^{-1}$, 33,488 $J \cdot kg^{-1} \cdot h^{-1}$, et 18,036 $J \cdot kg^{-1} \cdot h^{-1}$ furent observés tout en maintenant un échange gazeux physiologique efficace [moyenne de la Pa_{O_2} = 353 ± 28 mmHg (ET), moyenne de la Pa_{CO_2} = 30 ± 2 mmHg (ET)]. Les changements dans les variables cardiovasculaires furent notés avec l'atteinte de niveaux d'hypothermie léger (35°C–30°C) et modéré (30°C–25°C). Le débit cardiaque, la consommation d'oxygène, la fréquence cardiaque et la pression artérielle moyenne étaient significativement reliés avec la température rectale. Les résultats présentés ici quantifient les effets de la ventilation liquidienne sur le refroidissement du corps et la perte de chaleur par les voies respiratoires. De plus, les modifications physiologiques associées avec la condition hypothermique observée pourrait sévèrement limiter l'efficacité du plongeur humain, si elle n'est pas contrôlée soigneusement.

ventilation liquidienne
hypothermie
fluorocarbone
taux de refroidissement

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