Effect of peripheral temperature on the formation of venous gas bubbles

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Mekjavić IB, Kakitsuba N. Effect of peripheral temperature on the formation of venous gas bubbles. Undersea Biomed Res 1989; 16(5):391–401.—Temperature of the tissue affects the many components involved in the formation of tissue gas phase formation: diffusion, perfusion, and inert gas solubility. Since the effects of perfusion and inert gas solubility may be counteracting in terms of enhancing growth of gas bubbles, the optimal thermal status of divers throughout a dive remains unresolved. To elucidate the role of peripheral body temperature on gas phase formation, four subjects were exposed to a 10° and 40°C environment for 3 h on two separate occasions, after a no-stop decompression from a 12-h dive to 9.14 m (30 fsw) on air. The 3-hour exposures to either a cold or warm air environment resulted in a significant difference in mean skin temperature ($P \leq 0.01$) with no alteration in rectal temperature. Total peripheral resistance during the 10°C exposure was 13.8 ± 1.9 mmHg $\cdot$ liter^{-1} $\cdot$ min^{-1} and significantly higher than that observed during the 40°C exposure (10.4 ± 3.5 mmHg $\cdot\cdot$liter^{-1} $\cdot\cdot$min^{-1}). Gas bubbles in the venous return were monitored with a Doppler ultrasonic transducer placed in the precordial region, both at rest and after a deep knee bend. Venous bubbles were only detected in 1 subject following the warm air exposure, whereas 3 of the 4 subjects developed Doppler-detectable bubbles during the cold air exposure. Although both the cold and warm air exposures (3 h postdecompression) were uneventful, a hot shower taken by the subjects on completion of the cold air exposure (6 h postdecompression) precipitated mild type I symptoms of decompression sickness. These symptoms were not present after a hot shower following the warm air exposure. The present results indicate that despite the assumed greater inert gas solubility of tissues expected during cold air exposure, the decrease in the perfusion may have played a more significant role in the observed levels of detectable venous gas bubbles. Development of type I symptoms following a 12-h saturation, a 3-h cold exposure, and a subsequent hot shower suggests that a rapid rise in peripheral temperature may cause a significant rise in tissue gas tension. This increase in tension does not seem to be sufficiently reduced by increased perfusion to the tissues to prevent bubble formation.

decompression sickness
inert gas solubility
cold exposure
hot exposure

The primary impetus in the development of decompression tables is to minimize decompression sickness, which presumably is a result of tissue gas phase formation. Recent table development has relied on Doppler-detected venous gas bubbles as indices of risk, but the underlying rationale for many such tables relies on optimizing

the partial pressure gradient of the inert gas between the inspired gas mixture and the tissue compartments (1). A wide range of physiologic and environmental factors have also been implicated in the intravascular inert gas phase formation during and after decompression (2). Thus several studies have emphasized that the rate of inert gas uptake and elimination is limited by perfusion (3, 4) and affected by hydrostatic pressure (5), body position (6), body temperature (6–9), and peripheral temperature (10, 11). With such increasing evidence of the significance of thermal status and exercise (12–15) in the instigation of decompression sickness, activities during and after a dive should be carefully monitored.

Recently, Dunford and Hayward (16) attempted to elucidate the role of divers' thermal status on the detection of venous gas bubbles at the precordial site following a 38-min dive to 78 fsw in open water. Based on a significant increase in the Spencer Doppler Bubble Grade observed after a warm dive (divers exercised on an underwater ergometer wearing an insulated dry suit) compared to a cold dive (exercise at depth while wearing a thin wetsuit), it was suggested that the uptake of inert gas was hindered due to peripheral vasoconstriction in the latter condition. The complexity of the interaction of temperature-induced changes in physical and physiologic mechanisms involved with inert gas uptake and elimination become apparent with the recent observations of Lin et al. (11), who investigated the effect of ambient temperature on bubble threshold in rats and observed depressed thresholds at temperatures both lower (15°C) and higher (35°C) than thermoneutral (24°C); however, since the chamber temperature was maintained at either 15°, 24°, or 35°C both during the compression and decompression phase of the dives, a significant difference may have existed in the amount of inert gas dissolved in the tissues for identical saturation depths.

The present study demonstrates the effect of peripheral temperature only on detected venous gas bubbles as determined with bubble scores, while maintaining a stable and normothermic core temperature. In addition, an attempt was made to eliminate the intersubject variability in the rate of inert gas uptake, and thus different levels of tissue saturation at onset of decompression, by detecting venous bubble levels following a no-stop decompression from a shallow saturation dive. During the saturation period, chamber temperature was maintained at identical levels for all dives to further eliminate any differences in the level of tissue saturation due to temperature.

MATERIALS AND METHODS

All dives were conducted in the hyperbaric facility (Perry Oceanographics Inc., FL) at the School of Kinesiology, Simon Fraser University, and were approved by the University Ethics Review Committee.

Four healthy, male volunteers participated in 2 simulated saturation dives to 9.14 m (30 fsw) on air. The subjects' anthropometric measurements are presented in Table 1. Subjects commenced the dives at 2100 h. They remained in the chamber for 12 h and were decompressed to the ambient pressure without decompression stops at 0900 h the following day. Although 12 h at a depth of 9.14 m (30 fsw) may not be adequate for complete saturation of all tissues, it was assumed that the majority of tissue compartments would be saturated. During the 12-h dive, ambient temperature in the chamber was maintained at 25°C. Upon surfacing, subjects were examined by the

TABLE 1
SUBJECTS' PHYSICAL MEASUREMENTS

Subject	Age, yr	Mass, kg	Height, cm	Skinfold Thickness, mm			
				Triceps	Biceps	Subscapula	Supraspinale
PS	24	95.9	189.8	10.7	4.3	16.1	5
NG	27	73.3	179.8	10.9	5.8	10.1	4.8
NK	33	70.2	174.3	7.8	4.5	11.6	11.2
IM	29	71.7	176.5	12.2	5.6	16.1	8.0

attending diving medical officer and transferred immediately to a temperature-controlled environmental chamber (Tenney Engineering Inc., NJ).

On one occasion the ambient temperature in the environmental chamber was maintained at 40°C, and during the second condition, which was separated from the first trial by an interval of 1 wk, ambient temperature was kept constant at 10°C. Subjects were requested to rest during the 3-h exposures in the environmental chamber, in the respective conditions. Divers' activity and diets predive, during, and postdive were identical in both trials, thus accounting for any endogenous production of N_2 within the alimentary canal.

During the 3-h thermal exposure after direct ascent to the surface, evaluation of detectable venous gas bubble levels was conducted at 15-min intervals with a Doppler Bubble Detector (model 1032G, Institute of Applied Physiology and Medicine, Seattle, WA) at the precordial region (17). Ultrasonic monitoring was conducted during a 1-min rest period and for 30 sec following a deep knee bend. In the event that the ultrasonic transducer slipped during the deep knee bend maneuver, and the signal could not be obtained immediately, the measurement was repeated after a short rest period. Signals were recorded on cassette tapes and then graded according to the Kisman and Masurel table (18, 19). Tapes from the present series of experiments were evaluated independently by both our laboratory and the Diving Unit at the Defence and Civil Institute of Environmental Medicine, Canada.

Rectal temperature was measured with a YSI rectal thermistor probe (Yellow Springs Instruments, Yellow Springs, OH) inserted 15 cm. Unweighted mean skin temperature was determined from measurements made on the arm, chest, thigh, and calf, also with YSI thermistors.

Cardiac output (Q, liter · min^{-1}) was determined at hourly intervals with an impedance plethysmograph (Bomed, CA) based on the analysis proposed by Kubicek et al. (20). As the core temperature did not vary significantly throughout the 3-h exposure in either trials, nor between trials, the resistivity of the blood at 37°C was incorporated into the calculation of cardiac output (21).

Mean arterial blood pressure (MAP, mmHg) was determined using an automatic sphygmomanometer and adding one third of the pulse pressure (the algebraic difference between systolic and diastolic pressures) to the diastolic pressure. An indication of the total peripheral resistance was derived at each hour of exposure by dividing MAP by cardiac output (Q, liter · min^{-1}).

RESULTS

The thermal stress imposed on the subjects on both occasions, namely 10° and 40°C ambient temperature, did not affect the rectal temperature, as evident from Fig. 1. However, unweighted mean skin temperature was maintained at 27.3° ± 0.4° and 35.4° ± 0.5°C for the 10° and 40°C environmental exposures, respectively. The intensity and duration of the cold stress was not sufficient to induce noticeable shivering tremor in the subjects.

Table 2 indicates that the total peripheral resistance (TPR, mmHg · liter^{-1} · min^{-1}) was significantly ($P \leq 0.05$) lower in the 40°C environment in comparison with values observed during the exposure to 10°C, although there was no significant difference in cardiac output for the two experimental conditions.

Results of the ultrasonic monitoring in the precordial region are presented in Table 3. Bubbles were observed in only 1 subject, while the subjects were seated motionless, during the 3-h postdecompression thermal exposure. During the 40°C exposure, bubbles were noted only in subject IM after the knee bend maneuver, whereas high venous gas emboli (VGE) scores were noted in 3 of the 4 subjects after the knee bend maneuver during exposure to 10°C ambient air, with subject NG having the highest VGE grade (a score of 4 on the Kisman-Masurel Table). There appeared to be no difference in the onset time of bubble formation between trials for subject IM, who experienced venous gas bubbles in both conditions.

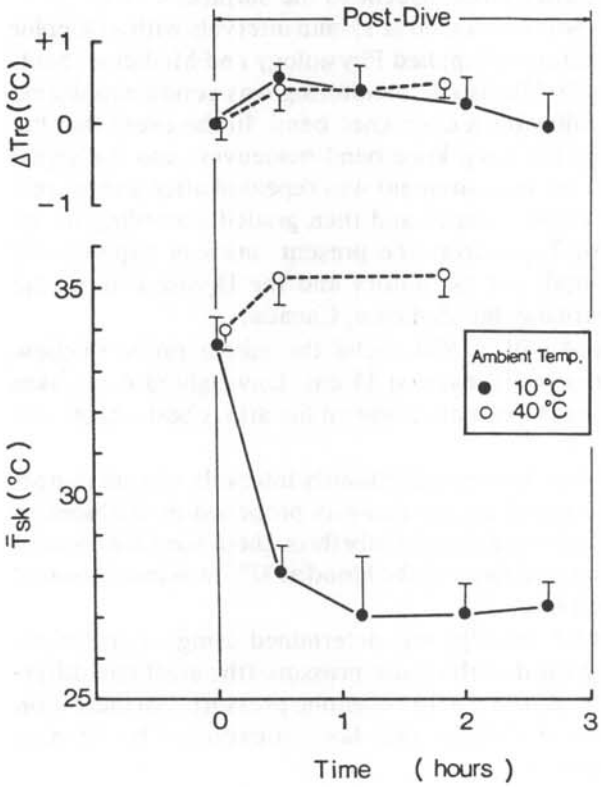

Fig. 1. Thermal status of divers following the no-stop decompression from a 12-h dive to 9.14 m (30 fsw) and during the subsequent 3-h exposure to either 10°C *(solid circles)* or 40°C *(open circles)* air. *Top graph* depicts the mean ($n = 4$) change in rectal temperature (ΔTre, °C) from the onset of the cold and warm exposures; *bottom graph* shows the level of the mean skin temperature ($\bar{T}$sk, °C). *Bars* denote the SD from the mean.

TABLE 2
CARDIOVASCULAR RESPONSES FOLLOWING 3-HR EXPOSURE TO COLD (10°C) AND WARM AIR (40°C)

	10°C	40°C
Blood pressure (mmHg):		
- systolic	132 ±10	120[a] ±14
- diastolic	87 ±8	78 ±8
- M.A.P.	102 ±7	92[a] ±10
Q (liter · min^{-1})	7.5 ±0.9	9.5 ±2.2
S.V. (ml)	124 ±36	103 ±25
H.R. (min^{-1})	63 ±15	92[a] ±9
T.P.R. (mmHg · liter^{-1} · min^{-1})	13.7 ±1.9	10.4[a] ±3.5

[a] Denotes differences between values observed during the 10° and 40°C air exposures, which were statistically significant at the 5% level.

No symptoms of decompression sickness (DCS) were apparent during the 3-h postdecompression period in the two conditions. However, 3 of the 4 subjects reported mild symptoms of DCS following a hot shower after the cold air exposure (Table 3). Symptoms were not observed after the hot air exposure. Pruritus and mild shoulder pain developed approximately 6 h following the no-stop decompression to surface, approximately 3 h after the cold exposure, and 0.5 to 1 h following the hot shower. The use of hot showers by the subjects following the experiments was not part of the protocol, thus temperature and duration of the showers was not monitored. Since no restrictions were placed on the use of the showers, subjects made use of the available showers on both trial days. Thus, the results of mild DCS following a hot shower after the cold exposure, though serendipitous, are nevertheless noteworthy. As soon as the subjects reported the above symptoms they were given pure oxygen (surface oxygen) and the appropriate personnel alerted to activate the decompression chamber for therapy. Because the symptoms subsided within a short while and were also mild, a decision was made not to commence decompression therapy but to retain the subjects under observation in the laboratory for an additional 6 h. When discharged from the laboratory, the subjects were given medical alert bracelets, indicating that they had been exposed to a hyperbaric environment, and instructed to contact the diving medical officer should the symptoms reappear.

Due to the high incidence of DCS symptoms observed, the present study was terminated and thus the results of only 4 subjects are available.

TABLE 3

ONSET OF DETECTABLE VENOUS GAS BUBBLES AND PEAK BUBBLE SCORES OBSERVED IN THE PRECORDIAL REGION AT REST AND AFTER A DEEP KNEE BEND, FOLLOWING TWO 12-H DIVES TO 9.14 METERS (30 FSW)[a]

Ambient Temperature, °C	Subject	Onset Time of Venous Gas Bubbles Postdecompression, min	Peak Bubble Score Observed in the Precordial Region Over 3-h Period[b]		DCS Symptom	
			At Rest	After Movement	Onset Time, h	Description
40	PS	–	0	0	–	–
	NG	–	0	0	–	–
	NK	–	0	0	–	–
	IM	–	0	3	–	–
10	PS	101	0	1	5–6	mild shoulder pain
	NG	105	0	4	4–5	pruritus
	NK	–	0	0	4–5	mild shoulder pain
	IM	94	1	3+	5–6	pruritus and mild shoulder pain

[a] Continuous detection of venous gas bubbles with Doppler ultrasound was conducted during the 3-h postdecompression period in an environmental chamber. Chamber air temperature was maintained at either 40° or 10°C. Measurements were obtained with the subjects seated and after a deep knee bend.

[b] Detectable venous gas bubbles were graded using the procedure recommended by Kisman et al. (18). The procedure has been outlined in detail by Eatock and Nishi (19).

DISCUSSION

During decompression, Hesser and Lanphier (as documented in ref 22) observed reductions in incidences of paresthesia, pruritus, and skin rash in areas of skin with added thermal protection, suggesting that the cause of the symptoms in the unprotected areas was local vasoconstriction resulting in decreased blood flow and nitrogen washout. In addition, skin mottling has also been attributed to local occlusion of blood supply by intravascularly generated bubbles (23). Thus, our present observations of higher peak bubble scores during the cold exposure would support the suggestion of Hesser and Lanphier (22) of perfusion limitation due to vasoconstriction. Discussion of the subsequent development of pruritus following active heating with a hot shower of peripherally cold tissues, however, becomes more complex, as the phenomenon may be due to a combination of local occlusion by a gas phase, as proposed by Hempelmann (23), and of a temperature-induced change in inert gas solubility of the tissues.

The two major effects, which have to be considered in any discussion of temperature-induced gas phase formation in divers, are tissue perfusion and inert gas solubility. The classical concept of N_2 solubility holds that the solubility of N_2 decreases with increasing temperature of water. Assuming that this relationship holds for biological tissue, then placing a subject in a hot environment will enhance peripheral tissue perfusion, while decreasing the solubility of the inert gas in the tissue. Similarly, decreasing the peripheral tissue temperature will increase the solubility of the inert gas in the tissue, but will decrease perfusion. Although there is ample evidence demonstrating a decrease in the solubility of N_2 with increasing temperature of water, the data available for biological tissue are not conclusive. Extensive reviews of the literature concerning the temperature dependence of N_2 solubility, conducted by Bartels (24) and Weathersby and Homer (25), suggest both an increase and a decrease for the N_2 solubility in oil, and suggest that N_2 solubility in blood is fairly temperature independent. In addition to the substantial variation that exists in the determination of N_2 solubility for a given solvent between different research groups (25), it has been emphasized by Lawrence et al. (26) that the dissolved gas distribution ratio in the fat of critical areas in the central nervous system may be markedly different from that in olive oil. In light of the above data, it becomes speculative to attempt any firm conclusions regarding the effect of peripheral tissue temperature on N_2 solubility in humans and how this may subsequently effect gas phase formation. As a result, the choice of optimal peripheral tissue temperature during and following a dive becomes complex.

Recently, Mack and Lin (9) demonstrated in rats that hypothermia reduces N_2 elimination and that the hyperthermia has no clear advantage over normothermia, the principal factor being cardiac output. Although they illustrate that the hypothermia-induced reduction in cardiac output is responsible for the reduced nitrogen elimination rate, they do not credit tissue temperature per se with any significant contribution. Tissue temperature was not monitored in the present study, but the significant difference in the level of unweighted average skin temperature during the exposure to 10° and 40°C air suggests a substantial variation in the temperature of the intermediate tissue layers, despite very similar levels of core temperature (Fig. 1). Thus, the effect of a change in tissue temperature cannot be excluded as a

contributory factor in the development of DCS symptoms observed as a result of the hot shower following the cold air exposure.

Once the human body is saturated at a given pressure, inert gas tension of various tissue compartments may be assumed to be identical and independent of the rate of inert gas uptake or body composition. In the present study, subjects were exposed to the same controlled temperature atmosphere during the 12-h exposure to 30 fsw in both conditions. For the purpose of discussion, assuming a hypothetical situation during which tissue perfusion is similar for both cold and hot air exposures, a decreased inert gas solubility with decreasing tissue temperature would result in a greater generation of intravascular bubbles during the cold air compared to the hot air exposure. However, since the cold exposure induces a significant decrease in tissue perfusion, the occurrence of venous gas bubbles would be greatly enhanced. Thus, the hot air exposure, with a greater rate of N_2 elimination as a result of vasodilatation and assuming an increased solubility of inert gas, would result in a decrease in the occurrence of venous gas bubbles. This argument would hold were it not for the observations of a high incidence of DCS symptoms following a transition from the cold environment to a hot shower. For the hypothesis of decreased inert gas solubility with decreasing temperature to be tenable, a rapid increase in tissue temperature should favor a reduction in the occurrence of venous gas bubbles as a result of increased perfusion and inert gas solubility. The observed symptoms of DCS, specifically pruritus and mild pain in the shoulder joint (type I), as a result of the transition from the cold air ambient to a hot shower implies the development of a condition conducive to gas phase formation. In the absence of temperature (skin and rectal) and Doppler ultrasound data during this phase of the experiments, it can only be speculated that since the perfusion would have increased dramatically during the hot shower and thus enhanced the N_2 elimination rate, the DCS symptoms are most probably related to a decrease in inert gas solubility. The transition from a hot air environment to a hot shower may not have effected any significant changes in tissue perfusion and temperature, in accordance with observations of no DCS symptoms after the hot shower.

Accepting the evidence derived from the transition from cold air to a hot shower as supportive of the concept of decreasing inert gas solubility with increasing tissue temperature, the observations of higher peak Doppler-detectable bubbles during the cold exposure compared to the hot air exposure suggests that perfusion, and not inert gas solubility, was the dominant factor in intravascular gas phase formation. More specifically, the significance of the effect of temperature-induced changes in inert gas solubility on gas phase formation was secondary to the significance of the effect of temperature-induced changes in tissue perfusion. Although the present results are supportive of the concept of decreasing inert gas solubility with increasing temperature, the possibility of either increased solubility or unchanged solubility cannot be disregarded because there remains inconclusive evidence regarding the effect of temperature on inert gas solubility.

The nature of the experimental protocol utilized in the present investigation does not permit any firm conclusions to be drawn with regard to the contribution of intravascular bubbles generated in core regions to the overall Doppler-detected venous gas bubbles. Their contribution to the observed differences between the cold and hot air exposures may become relevant during conditions that either alter the perfusion of core regions or that induce substantial changes in the core temperature. Since

splanchnic and renal blood flow have been observed to decrease during passive heating (27), gas phase formation as a result of reduced perfusion may have been more enhanced in core regions during hot air exposure. However, as no Doppler-detected bubbles were observed, the effect of different rates of core perfusion would seem to be negligible in the present analysis. Furthermore, since core temperature was similar and constant during both air trials, the contribution of temperature-induced venous gas bubbles from the core region would be similar. Assuming that the duration and temperature of the showers were similar on both occasions, similar elevations in rectal temperature may be expected, and consequently a similar level of core-generated intravascular bubbles. Unfortunately, in the absence of either temperature measurements (core and skin) or ultrasonic surveillance during the shower phase, these suggestions are speculative.

The lack of DCS symptoms despite high peak Doppler-detected bubble scores after a deep knee bend during the cold air exposure, supports the observations of Spencer (28) that DCS is not always associated with Doppler-detected bubbles. It is not surprising that DCS symptoms were absent following the hot air exposure, because the resting bubble scores were 0 (with the exception of subject IM). Nevertheless, the high peak bubble scores in the cold air exposure trial indicate that Doppler-detected bubbles could be useful for detecting conditions that may be precursive to DCS symptoms, especially when significant skin temperature changes are induced.

On the basis of the present results, it would appear that over-warming of divers, as suggested by Lambertsen (29), especially if applied rapidly to divers who are initially peripherally cold, may create a condition of decreasing inert gas solubility in tissue with increasing tissue temperature. Body temperature, as a result of its significant effect on perfusion and inert gas solubility, is an extremely important factor in gas phase formation following decompression. Since the interaction of the effects of temperature on tissue perfusion and inert gas solubility remains unresolved, in terms of bubble growth, predicting its overall effect on venous gas bubble development requires further knowledge of its effects on the individual components involved in bubble growth, as mentioned previously.

The present study confirms that although different steady state levels of shell temperature may not cause alarming elevations in bubble scores, a sudden rapid elevation in tissue temperature may precipitate a condition conducive to gas phase formation and may ultimately result in symptoms associated with DCS. The significance of these findings in nonsaturation-type dives requires further investigation.

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