During the IXth International Hyperbaric Congress held in Sydney in 1987, the Executive Committee held a meeting to which Leon Greenbaum, Executive Secretary of UHMS, was invited as observer. At that meeting, a suggestion was made that it may be feasible in the future for SPUMS and UHMS to hold a joint meeting at a mutually convenient location. Dr Greenbaum agreed that he would take the proposal back to his Executive. At no time in the discussion was any decision made on precise date or location.

Since that time neither the President nor Secretary of SPUMS has been informed that the UHMS Executive has discussed the subject nor come to a decision. In the interim advice has been received that such a combined meeting is not in the best interests of this Society. When a similar combined meeting was held between UHMS and the EUBS some years ago, the EUBS members found that their meeting and members were overwhelmed by the tidal wave of intense esoteric didactic American hyperbaric research which bore little relevance to practical diving and, especially, recreational air diving. To them, the meeting was a bitter disappointment.

In order to avoid this and perhaps to prevent any ill will, the SPUMS Executive has agreed to hold the 1989 Annual Scientific Meeting at a venue separate from the UHMS Meeting but timed such that members of both Societies can attend both meetings.

Since this decision was made, several letters have been received from Senior members of the UHMS Executive accusing SPUMS of renegeing on a definite agreement between the two Societies. I repeat that no such definite agreement was ever made or confirmed.

It is unlikely, in light of this misunderstanding that the UHMS Meeting will be held so close to Australian shores again for a long time, so I strongly urge all our members to extend their conference tour in 1989 to include both the SPUMS Meeting in Vanuatu and the UHMS in Honolulu. I am writing to Dr Greenbaum inviting his members to attend the SPUMS Meeting and contribute to its scientific program.

I conclude that this unfortunate occurrence has resulted from misinterpretation and perhaps a lack of communication but I hope that at some time in the future a joint, informative and convivial meeting can be arranged so that any differences can be resolved.

David Davies,
Secretary of SPUMS

DIVER HYPOTHERMIA
CURRENT STATUS AND FUTURE CONTROL

Richard F. Taylor and David W. Yesair

In the population at large, hypothermia is mainly limited to infants and the elderly. Hypothermia is also common, however, in specialized sectors of the population which are routinely or accidentally exposed to cold environments during the normal course of their activities. One such group includes sport, military and commercial divers. In fact hypothermia remains a major limitation to divers in cold waters such as the coastal areas of North America and Europe, in the North Sea and in deep waters (greater than 180 metres) throughout the world. Diver hypothermia may be caused from normal exposure during prolonged dives, or as the result of accident, such as the case of a diver in a lost bell.

The primary cause of diver hypothermia is the rapid loss of body (core) heat into the surrounding water, either by direct transfer through the diving suit or by loss of heat to the diving gas. Water conducts heat approximately 25 times more rapidly than does air. The body’s first reaction to being submerged in cold water is the vasoconstriction of the blood vessels in the skin which results in a net decrease in body heat lost to the water.

Heat loss can also be minimised by proper insulation, such as a diving suit or a layer of subcutaneous fat. In active situations, however, these latter measures do not maintain a balance between body heat produced and lost. Thus, the body produces more heat such as that produced by shivering. Increased heat production also results from increased activity, such as exercise or hard work.

Shivering and increased physical activity can, however, also lead to increased exposure of the body and even more heat loss. In addition, physical activity requires more oxygen, more rapid breathing and causes additional heat loss from the lungs to the diving gas. This is especially important at depths greater than 183 metres of sea water (MSW) since the denser gas can lead to appreciable heat loss.

For example, normal heat loss to air from the lungs is approximately 10% of the total body heat loss. Under hyperbaric heliox or trimex at 183 MSW or more, heat loss from the lungs to the breathing gas can approach the same order of heat loss from the body to the water. It is thus apparent that the human body, unaided, cannot offset hypothermia in cold waters for any appreciable time.

The onset of hypothermia in divers is dependent on a number of practical, environmental and physiological factors. In each case some, but not all, of these factors can be controlled. For example, factors such as the length of dive
and the type of diving suit used can be optimized for the conditions at hand; but water temperature and the composition of the diving gas required cannot be altered significantly. The use of helium, for example, has allowed divers to achieve depths far in excess of those achieved with air; but, in return, the high thermal conductivity of helium dramatically increases heat loss from the diver into the breathing gas as noted above.

Physiological factors affecting diver hypothermia are more complex and include the degree of cold adaptation of the diver, his work activity during the dive, his physical makeup (such as amount of body fat), his metabolic response to cold stress and any pre-dive use of drugs. Such factors are variable from diver to diver, and thus would appear poor control points for hypothermia.

In many cases, however, physiological control of hypothermia may be partially successful or provide a basis for the design of new approaches toward offsetting hypothermia. For example, cold adaptation can help against mild hypothermia and may involve actual changes in metabolism. This is illustrated by the Japanese and Korean Ama (diving women) who have a basal metabolic rate approximately 30% greater in winter than that of nondiving women living in the same climate and eating the same diet. Studies on divers under saturation conditions have shown that their metabolic rates also increase if the temperature of their environment falls below 30°C.

Changes in metabolism or in the rate of nutrient absorption and metabolism may also account for the well-documented losses of weight in saturation chamber dives. For example, in experimental dives such as Helgoland, Tekite I and II, Sea Lab II, and Hana Kai II, the divers ate the equivalent of from 2500 to 6000 Kcal/day but still recorded total dive weight losses of from 0.6 to 4 kg, depending on the duration of the dive and depth. * The apparent correlation of metabolic change with depth, and time at depth, becomes very intriguing when viewed as a detriment toward diver resistance to hypothermia. For example, it has been shown that under saturation conditions, essential metabolic levels of thiamine (vitamin B) and thiamin-dependent enzymes decrease in proportion to time at depth. Since thiamine requirements may be directly proportional to energy expenditures, saturation divers may experience physiological stresses which decrease efficient nutrient utilization and, thus, become more susceptible to hypothermia.

It also has been suggested that divers may respond to the hyperbaric environment by gradually losing small amounts of heat (approximately 0.5 to 1.0 Kcal/min) to result in a long term body heat loss without apparent, gross change in metabolism or core temperature. This would then explain weight loss in saturation environments. This could also support the physiological stress theory advanced above and certainly decrease the diver’s resistance to hypothermia should his temperature or his environment change rapidly.

There is reason to believe that the hyperbaric environment may act directly on those physiological processes controlling thermoregulation and thus potentiate diver hypothermia. For example, changes in thyroid and adrenal cortex hormone levels in the blood occur at depth and may indicate direct effects of pressure on central nervous system (CNS) control of body temperature. Such pressure effects on neurological processes are not unknown and are best illustrated by the pressure-induced high pressure nervous syndrome (HPNS). Studies conducted at Arthur D. Little, Inc. have shown that pressure can directly affect the molecular processes involved in neural transmission and, thus, thermoregulation.

Other drugs such as morphine, the tranquilizers serpine and chlorpromazine and the stimulatory amphetamines are all known to cause hypothermia. Their action sites appear to be in the CNS, suggesting that future studies using such drugs as models could design new therapies to control hypothermia at a cellular or molecular level.

In the meantime, divers are to be warned of the high risk in using such drugs prior to diving. Divers should also minimize their use of alcohol prior to diving since alcohol will induce vasodilation and resulting heat loss.

**Treatment of Diver Hypothermia**

The classical and still practical method for the treatment of diver hypothermia relies on restoring the victim’s temperature back to normal as quickly as possible. In the field, this therapy involves placing the diver in a warm bath with elevation of the head and limbs out of the water. For milder cases of hypothermia, hot showers may suffice. Warm drinks and the administration of (warmed) intravenous fluids can also be used, especially if fluid and electrolyte imbalances exist in the victim.
In some cases more drastic measures may be required if hospital facilities are immediately available such as inhalation of warm oxygen and the circulation of warm fluids through the body (haemodialysis, peritoneal lavage, intrathoracic irrigation). To date, no proven therapy exists for the treatment of hypothermia with drugs.

**Thermoregulation Controls**

Currently, there is no standard procedure to prevent and/or treat diver hypothermia by controlling the diver’s physiology with one possible exception; the use of steroids or antihistamines to treat cold urticaria. Our interests are directed at such physiological prevention and control of hypothermia using nutritional and pharmacological approaches. This is exemplified in a new nutritional product now being developed based on studies utilizing this product as a drug delivery system. For example, it is known that the efficacy of certain drugs, such as griseofulvin and several steroids, are improved when given orally in fats rather than per se.

Based on this knowledge, Arthur D. Little, Inc. has developed a hydrolyzed lipid formulation, termed “lymphasomes” (patent pending) which dramatically improves the absorption and efficacy of a number of drugs, including the contraceptive, oestradiol and fat soluble vitamins such as the retinoids.

In the course of these studies, the ability of the lymphasomes to act as a rapidly absorbed and utilized energy source in the body tissues was investigated. For example, the metabolic rate of the lymphasomes in rats was followed using lymphasomes labelled with radioactive fatty acid. It was shown that within six hours, approximately 45 per cent of the lymphasome lipid had been metabolized to expired carbon dioxide. In actual fact, measurement of radioactive carbon dioxide only represents a minimum estimation of lipid utilization and does not account for use of the lipid metabolic products for other purposes which would not immediately release the tracer.

Thus these studies more realistically indicate that the lymphasome lipids are probably completely absorbed and metabolized within 12 to 24 hours, with the majority of utilization occurring 6 hours after administration. Parallel experiments showed that the delivery of lymphasome lipids into blood was a sustained process resulting in constant lipid blood levels starting at approximately 1 hour and lasting for at least 8 hours. Insignificant amounts of the lymphasome lipids were found in faeces, again indicating nearly complete absorption. These data supported earlier conclusions that the lymphasome high fat product is readily absorbed from the intestine into the lymphatic system and is then delivered by the blood to the tissues for immediate and sustained heat and energy production.

How can this lymphasome concept be applied to the problems associated with diver hypothermia? In order to answer this question, a discussion of some basic principles of fat absorption and utilization is needed and follow.

It is well documented that physically active, trained individuals (such as divers) oxidize more fat and less carbohydrate than any untrained individuals when carrying out comparable, submaximal work. In fact, regularly performed endurance exercise can increase the capacity for aerobic metabolism based on increased fat utilization with decreased carbohydrate consumption.

Questions have been raised, however, concerning the proper diet for supporting a rigorous work or activity schedule. In general, it is agreed that high protein, high fat diets should be avoided. Thus diets intended for use in high stress situations have tended to emphasize a high carbohydrate content such as, for example, a diet receiving 15 per cent of its calories from protein, 33 per cent from fat and 52 per cent from carbohydrate. However, such diets are based on the assumption that all the carbohydrate will be fully utilized to carbon dioxide and water.

**TABLE 1**

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Metabolic Products</th>
<th>Heat Generated (Kcal/g)</th>
<th>Work Potential* (Kcal/g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrate</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(glucose)</td>
<td>Anaerobic Lactic</td>
<td>Acid</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Metabolism</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Aerobic Metabolism</td>
<td>CO₂/H₂O</td>
<td>1.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Total</td>
<td>1.4</td>
</tr>
<tr>
<td>Fat</td>
<td>(Palmitic Acid)CO₂/H₂O</td>
<td>5.1</td>
<td>4.1</td>
</tr>
</tbody>
</table>

*Work potential represents production of high energy yielding adenosine triphosphate (ATP), which contains 11 Kcal of potential work per molecule.

As shown in Table 1, the metabolism of carbohydrate can be both anaerobic and aerobic. In stressful situations such as sustained hard work, anaerobic carbohydrate metabolism predominates. While such metabolism can provide the initial burst of energy for muscle during physical activ-
ity, it is insufficient to maintain energy requirements for sustained physical activity in a stressful situation such as that of a hard working diver. In contrast, aerobic fat metabolism does provide the sustained energy required under stressful conditions. In fact, mobilization of fat in the body has been shown to increase endurance by 50 per cent and to decrease the rate of glycogen depletion under stress conditions. On the other hand, prevention of hypoglycemia in humans does not delay exhaustion from sustained physical activity.

We thus propose that in high activity, stressful situations, a diet receiving 13 per cent of its calories from protein, 33 per cent from carbohydrate and 54 per cent from fat would be more effective. This diet is based on physiological estimates that for normal work output, a total caloric intake of 4368 Kcal per day is required: 1968 Kcal for basal metabolism and 2400 Kcal for work. Such work could be equivalent to, for example, six hours for a diver working at depth.

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>% Kcal</th>
<th>Kcal</th>
<th>Weight g</th>
<th>%</th>
<th>% Kcal</th>
<th>Kcal</th>
<th>Weight g</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrate (glucose)</td>
<td>52</td>
<td>2272</td>
<td>608</td>
<td>66</td>
<td>33</td>
<td>1441</td>
<td>385</td>
<td>49</td>
</tr>
<tr>
<td>Fat (palmitic acid)</td>
<td>33</td>
<td>1441</td>
<td>158</td>
<td>17</td>
<td>54</td>
<td>2358</td>
<td>258</td>
<td>33</td>
</tr>
<tr>
<td>Protein</td>
<td>15</td>
<td>655</td>
<td>160</td>
<td>17</td>
<td>13</td>
<td>569</td>
<td>139</td>
<td>18</td>
</tr>
<tr>
<td>Totals</td>
<td>100</td>
<td>4368</td>
<td>926</td>
<td>100</td>
<td>100</td>
<td>4368</td>
<td>782</td>
<td>100</td>
</tr>
</tbody>
</table>

One problem concerning a high fat, high energy diet needs to be surmounted before it can be used in a cold-stress situation. Simply stated, fats are the slowest and most difficult of all nutrients to be absorbed in the intestinal tract. This is due to the requirement that fat must be first hydrolyzed before it is absorbed into the intestinal lymph and then into the blood. Such hydrolysis is dependent on two components which are normally secreted into the intestinal tract: pancreatic lipase and bile salts. Of these two, bile salts appear directly affected by stressful conditions including cold stress. Thus, under hypothermic conditions, bile salts secretion decreases significantly (approximately 5 to 7% per °C) and this decrease results in decreased fat hydrolysis and hence decreased fat absorption and utilization. In addition, the dehydration and accelerated loss of body fluids which occurs in stressful (including hyperbaric) situations also decreases fat hydrolysis and absorption.

Our proposed high fat diet is compared to the recommended high carbohydrate diet for stressful condition in Table 2. The primary difference between the diets is their yield of heat: the high fat diet yields approximately 500 Kcal more heat even though its total food weight is 15 per cent less than the high carbohydrate diet. The difference could be especially important to the working diver exposed to a cold environment.

For example, the initial stages of hypothermia include shivering, which can increase the requirement for body heat production as much as 500 Kcal/hour. This requirement or others for body heat can in turn lead to a rapid imbalance between body heat produced and lost, and potentiate the onset of more serious hypothermia. Thus, a high fat diet could significantly aid in offsetting this condition.

A paradoxical situation thus exists concerning proper utilization of nutrients under stressful conditions. While fats are a superior source of rapid heat and energy, the very condition itself decreases fat utilization. The lymphasome concept for fat absorption specifically addresses this impasse. The lymphasomes do not require hydrolysis in the intestinal tract since that process has already been carried out prior to ingestion. In the intestinal tract the lymphasomes are rapidly absorbed and utilized by muscle for heat and energy production. In addition, the product is stable to storage and could be provided in a food bar form.

The lymphasomes (high fat) diet would be utilized by sport, commercial and military divers during routine dives in cold waters. It would provide not only additional work potential for the diver at depth, but would also provide heat
for both the diver’s comfort and to aid in offsetting hypothermia. The lymphasomes could also be used as an emergency ration, e.g. in lost bell situations and for the treatment of hypothermia.

Future exploitation of the lymphasomes might include the use of their drug delivery properties to administer specific metabolic stimulants to fat metabolism, or drugs which can control thermoregulatory responses. The lymphasome concept thus represents control of hypothermia by stimulating normal physiological functions. Such an approach is particularly relevant to the problems associated with diver hypothermia.

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HEAT LOSS AND DIET

In a study performed by the medical faculty at the Institute of Physiology in Buenos Aires, Argentine, the reaction to immersion in 72°F water was studied in ten healthy male volunteers who ingested a balanced diet for three weeks (then were tested), followed by successive testing after three week diets of high carbohydrate, high protein, and high fat. Caloric intake was maintained from diet to diet.

The researchers concluded that the “reaction to cold water immersion was demonstrable for all groups, but more efficient in subjects who had received either balanced or high carbohydrate diets”, suggesting that high protein or high fat diets reduce cold adaptation. But one has to wonder. Would an Eskimo agree?

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LESSENING THE RISK OF DECOMPRESSION SICKNESS

John Lippmann

ADDING SAFETY TO THE U.S. NAVY TABLES

Many divers have devised methods to add some degree of extra safety to their U.S. Navy (USN) Table calculations. Some methods are obviously better than others. I have chosen some methods that I know of to present in the following section. These methods should provide quite a lot of extra safety, but still cannot be guaranteed to prevent decompression sickness.

I believe that sport divers are better off using a more appropriate set of tables. At present I know of two tables, which are based on the U.S. Navy system, which I think should be used instead of the USN tables for sport diving purposes. They are the “Huggins Tables” and the “Bassett Tables”. There are also tables, based on other systems, that may be more suitable than the U.S. Navy Tables. These are the DCIEM and Buehlmann Tables.

Methods for making no-decompression dives safer

A. Choose an initial no decompression limit (NDL) by choosing the NDL for the next greater table depth. For