HEAT STRESS AND FLUID REPLACEMENT IN GOLFERS

YOSHIO KOBAYASHI*, TERUO HOSOI*, TOSHIKO TAKEUCHI*, HIDEKIYO YOSHIZAKI** and MASAHIKO SHIMIZU**

Abstract

The purpose of this study was to determine the effect of water intake on thermoregulatory response during a round of golf (18 holes) in the heat (30–33°C, 55–70% RH). Ten middle-aged male subjects participated in two separate golf rounds. During the first round the subjects played without fluid replacement (D), while in the second they received water replacement equal to weight loss in D (R). With D, body weight loss of 3.8% was accompanied by a 13.3% reduction in calculated plasma volume (PV), while a small loss of body weight (0.5%) with an increase in PV of 4.6% was observed in R. A third round of golf by 5 of the subjects in cooler weather (20.4°C) without fluid replacement two months later resulted in heart rate and rectal temperature changes only slightly lower than during R. Golfing with D significantly elevated rectal temperatures and heart rates as compared to R. The rectal temperatures and heart rates at end of the 4.5-hr rounds were 39.4°C and 145 bpm, and 38.6°C and 116 bpm, in D and R, respectively. The serum enzymes, aspartate aminotransferase, alanine aminotransferase, lactate dehydrogenase, and creatine kinase were measured at rest and after golfing. All enzyme levels increased significantly after golfing in D and to a lesser extent in R. The advantages of frequent fluid replacement during golf in high environmental temperatures was clearly demonstrated.

Key words : Golfers, Heat stress, Fluid replacement

Golf is enjoyed by individuals of various ages and physical conditions because it is not a strenuous activity. It is estimated that more than 12 million Japanese play golf for recreation or health and fitness. Unlike in America or Europe, amateur golf in Japan is played throughout the year because of its popularity and limited facilities, even in high temperatures and high humidity. Because of its relatively low work intensity, golf does not produce the number of heat injuries that are common in more vigorous sporting activities such as distance running, soccer or cycling. However, golf injuries in a hot environment do warrant attention because many people participate. The long duration of a game and the relatively low levels of physical fitness of some players can cause heat injuries when the game is played in hot weather.

Previous studies on golfers have emphasized muscular injuries, accidents and training for injury prevention14,20). Heat injuries in golf have received little attention in the literature. However, it is not uncommon for heat accidents to occur at golf during hot and humid summers. Sakamoto23) estimated that the core temperature of golfers in hot, humid weather could rise to 39°C and he emphasized the possibility of heat stroke. There is a need for more information concerning the thermoregulatory stress of golfers, not only to improve performance, but to insure safety in hot weather.

The purposes of this study were to determine the extent of thermoregulatory and serum enzymes responses during a round of golf in hot weather.

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and to determine the impact of fluid replacement on these responses.

**METHODS**

Ten healthy male subjects took part in the study. Mean values (SD) for age, height and weight were 46 (12) yr, 168 (4) cm, and 64 (11) kg, respectively. A round of golf (18 holes) was played by each subject twice during the last week of July and the first week of August (a week apart). All subjects walked with caddies during the round of golf. The subjects were allowed to sit on a bench while waiting for the next tee shot. Whenever they urinated, the volume of urine was measured and added to the post body weight. The dehydration experiment (D), without fluid replacement, was conducted first and the rehydration experiment (R), with fluid replacement, was conducted second. In the latter experiment, each subject played while drinking cold water at regular intervals to equal the total volume of water lost in D as estimated from the weight loss.

In addition to D and R, five of the subjects participated in a third experiment without fluid replacement in late October in a relatively cool temperature (D-C). The round of golf for three experiments took from 4.3 to 4.7 hrs to complete.

Venous blood samples were withdrawn from the antecubital vein in a sitting position one hour before and immediately after playing. The blood samples were drawn in vacuum tubes, remaining at ambient temperature for 30 min, and then were centrifuged at 3,000 rpm for 10 min in the laboratory. Hematocrit (Hct) was measured in triplicate with a microhematocrit centrifuge. Hemoglobin (Hb) was measured with a hematological cell counter. Because the levels of particular serum enzymes have been shown to be elevated in heat stroke patients, these were also measured with an autoanalyzer (Hitachi 736). These enzymes were: aspartate aminotransferase (AST), alanine aminotransferase (ALT), lactate dehydrogenase (LDH) and creatine kinase (CK).

Plasma volume (PV) changes were calculated according to Greenleaf et al. Percent PV change (%ΔPV) was calculated according to Eq. 1.

\[ %\Delta PV = 100 \left( \frac{H_{b_{pre}}}{H_{b_{post}}} \times \frac{100 - H_{c_{pre}}}{100 - H_{c_{post}}} \right) - 1 \]  

Electrodes for ECG were placed in the CM5 position and a copper-constantan thermocouple was inserted to a depth of 10 cm from the external temperatures (Tre) were continuously measured using a portable 2-channel heart rate and temperature memory (Duo models VM2-001, Vine Ltd, Tokyo) and later the recording values were analyzed by computer to obtain mean minute-by-minute HR and Tre values.

A paired-t test was used in order to determine the significance of difference between values at the P<0.05 level (two-tailed test).

**RESULTS**

The mean ambient temperature and relative humidity reading during the three experiments are presented in Table 1. Wind velocity was also measured at three-hole intervals and ranged from 0.8 m/sec to 1.5 m/sec during D and from 1.0 m/sec to 1.8 m/sec during R experiments.

The mean values for body weight (BW), Hb, Hct, Hb/Hct ratio and %ΔPV in D and R are shown in Table 2. In D the mean weight loss of 2.4 kg (3.8%) and the 13.3% reduction in PV after

<table>
<thead>
<tr>
<th>Time</th>
<th>Dehydration - Hot</th>
<th>Fluid Replacement - Hot</th>
<th>Dehydration - Cool</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Temperature (°C)</td>
<td>RH (%)</td>
<td>Temperature (°C)</td>
</tr>
<tr>
<td>1000</td>
<td>31.2</td>
<td>67</td>
<td>32.0</td>
</tr>
<tr>
<td>1500</td>
<td>30.5</td>
<td>57</td>
<td>30.7</td>
</tr>
<tr>
<td></td>
<td>(Maximum)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 2. Mean values (SD) and ranges of body weight (BW), hemoglobin (Hb), hematocrit (Hct), Hb/Hct ratio, and calculated plasma volume change (%ΔPV), before and after 4.5 hrs of golfing.

<table>
<thead>
<tr>
<th></th>
<th>Dehydration Before</th>
<th>Dehydration After</th>
<th>Δ</th>
<th>Fluid replacement Before</th>
<th>Fluid replacement After</th>
<th>Δ</th>
<th>D vs R</th>
</tr>
</thead>
<tbody>
<tr>
<td>BW (kg)</td>
<td>63.9 (10.9)</td>
<td>61.5 (10.5)</td>
<td>-2.4 (0.6)</td>
<td>63.9 (10.9)</td>
<td>63.4 (10.9)</td>
<td>-0.5 (0.2)</td>
<td>***</td>
</tr>
<tr>
<td>Hb (g/100ml)</td>
<td>14.3 (0.8)</td>
<td>15.5 (0.6)</td>
<td>1.2 (0.6)</td>
<td>14.9 (0.5)</td>
<td>15.5 (0.5)</td>
<td>0.6 (0.2)</td>
<td>***</td>
</tr>
<tr>
<td>Hct (%)</td>
<td>46.5 (4.4)</td>
<td>49.8 (4.2)</td>
<td>3.3 (1.4)</td>
<td>47.8 (2.7)</td>
<td>46.5 (2.0)</td>
<td>-1.3 (0.9)</td>
<td>***</td>
</tr>
<tr>
<td>Hb/Hct ratio</td>
<td>0.31 (0.02)</td>
<td>0.31 (0.01)</td>
<td>0.00 (0.01)</td>
<td>0.31 (0.01)</td>
<td>0.30 (0.01)</td>
<td>0.01 (0.01)</td>
<td>0.00 (0.02)</td>
</tr>
<tr>
<td>%ΔPV (%)</td>
<td>-13.3 (5.4)</td>
<td>(6.6 - 24.3)</td>
<td>4.6 (4.6)</td>
<td>(3.9 - 11.3)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Significance of differences between before and after or change between ΔD and ΔR is denoted by *: P<0.05, **: P<0.01, ***: P<0.001.

Table 3. Mean values (+1.0 SD) and ranges of serum enzymes before and after 4.5 hrs of golfing.

<table>
<thead>
<tr>
<th>Enzyme</th>
<th>Dehydration Before</th>
<th>Dehydration After</th>
<th>Δ</th>
<th>Fluid replacement Before</th>
<th>Fluid replacement After</th>
<th>Δ</th>
<th>D vs R</th>
</tr>
</thead>
<tbody>
<tr>
<td>CK</td>
<td>102.2 (43.8)</td>
<td>137.4 (49.2)</td>
<td>35.2 (19.1)</td>
<td>101.0 (28.3)</td>
<td>121.0 (35.6)</td>
<td>20.0 (18.3)</td>
<td>*</td>
</tr>
<tr>
<td>LDH</td>
<td>301.9 (58.4)</td>
<td>356.9 (63.5)</td>
<td>55.0 (29.4)</td>
<td>326.2 (70.1)</td>
<td>369.6 (75.9)</td>
<td>43.4 (27.8)</td>
<td>10.96</td>
</tr>
<tr>
<td></td>
<td>(198 - 344)</td>
<td>(233 - 446)</td>
<td>(12 - 121)</td>
<td>(209 - 409)</td>
<td>(251 - 505)</td>
<td>(10 - 96)</td>
<td></td>
</tr>
<tr>
<td>AST</td>
<td>24.5 (7.8)</td>
<td>28.1 (10.1)</td>
<td>3.6 (1.9)</td>
<td>24.7 (7.6)</td>
<td>25.9 (9.6)</td>
<td>1.2 (1.7)</td>
<td>1.7</td>
</tr>
<tr>
<td></td>
<td>(17 - 35)</td>
<td>(17 - 51)</td>
<td>(0 - 4)</td>
<td>(14 - 40)</td>
<td>(16 - 47)</td>
<td>(2 - 5)</td>
<td></td>
</tr>
<tr>
<td>ALT</td>
<td>19.2 (10.6)</td>
<td>21.2 (12.2)</td>
<td>2.0 (2.4)</td>
<td>20.1 (8.8)</td>
<td>20.7 (10.1)</td>
<td>0.6 (3.6)</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>(6 - 41)</td>
<td>(7 - 50)</td>
<td>(0 - 21.9)</td>
<td>(4 - 37)</td>
<td>(6 - 42)</td>
<td>(-20 - 13.5)</td>
<td></td>
</tr>
</tbody>
</table>

Significance of differences between before and after or change between ΔD and ΔR is denoted by *: P<0.05, **: P<0.01, ***: P<0.001.

5 hr were significant. The average BW during R declined 0.5 kg (0.7%) and the PV increased 4.6%, neither change being significant.

The Hb/Hct ratio was not significantly altered during D or R and difference between the two conditions was negligible.

The mean values and ranges of CK, LDH, AST and ALT for D and R are given in Table 3. It is clear from the table that the values of all enzymes were significantly increased by a greater amount during dehydration than with rehydration in a hot environment. However, the difference in these
changes between D and R were only significant for CK and ALT.

The time course of Tre and HR during D and R are plotted in Figs. 1 and 2, and compared with corresponding values at cool temperature with dehydration (D-C) in 5 subjects. Except during the first hour, Tre during D was significantly higher than Tre during R (Fig. 1). There were no significant differences between R and D-C. Mean values of Tre during the last 60 min for D, R and D-C were 39.4 (n = 10), 38.6 (n = 10) and 38.3°C (n = 5), respectively.

The mean HR during D was also higher than the mean HR during R, except during the first 60 min (Fig. 2). The values during the last 60 min ranged from 139 bpm to 145 bpm during D and from 115 to 116 during R. The corresponding values during D-C were between 99 to 107. Differences in HR between R and C-D (n = 5) were least apparent during the first 60 min.

The mean score of the 10 golfers during D was 107. During R, this improved to 103 (p < 0.002), as

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**Fig. 1.** Means of rectal temperature during for three different conditions. ** and *** denote significant differences between D and R at levels of P < 0.01 and P < 0.001, respectively.

**Fig. 2.** Means of heart rate during golfing for three different conditions. *, ** and *** denote significant differences between D and R at levels of P < 0.05, P < 0.01 and P < 0.001, respectively.
all but one subject improved their score. The five subjects who took part in C-D improved by 3 strokes over their performance during R (P < 0.10). However, some of these improvements in both situations may be attributed to practice and not solely to environmental conditions.

**DISCUSSION**

Thermal stress was clearly demonstrated in these men while playing golf without fluid replacement, as indicated by Tre, HR and plasma loss. After 4.5 hr, the Tre was elevated by 1.5°C in D (P < 0.05), but only 0.8°C in R and Tre rose continuously in D after the first hour, compared to a plateau in R. In D-C, the Tre increased only the first hour (Fig. 1). Similar results were seen for HR (Fig. 2). Both HR and Tre were quite similar during R and D-C. Hemoconcentration was clearly evident in D, compared to R, because of the great loss in PV. These findings indicated that playing golf in hot weather results in thermoregulatory stress if fluid is not replaced.

Plasma volume changes based on measurements of venous Hb and Hct changes are well-established\(^{13,29}\). The calculated percent change in plasma volume was 3.2 times greater than the changes in BW in D. The ratio is supported by data from earlier studies by Senay and Christensen\(^{27}\) and van Beaumont et al.\(^{30}\). These studies found that during 8.5 and 3 hrs of heat exposure (45°C, 35% RH) in resting men, the average percentage decrease in plasma volume was 3.5 and 2.9 times larger, respectively, than the percentage of total weight loss. During prolonged exercise water is lost from the body by thermoregulatory sweating, and the water content of the exercising muscle is thought to increase at the expense of a reduction in plasma volume\(^{17}\). Calculations from our measurements of Hb and Hct in D demonstrated a loss of PV of 13.3%. Assuming 4500 ml blood volume before (70 ml/kg) and a PV of 2408 ml [Blood volume × (1−Hct)], the PV was reduced about 320 ml. This represents a shift of about 300 ml of water from the vascular space to the tissues, when considering the relative density of plasma. The weight loss demonstrated a total loss of 2,400 ml of water from the body. This means that the net interstitial water loss was at least 2.0 L (2.4–0.3). It is difficult to see how exercising muscle can increase in water content under these conditions. The restoration of plasma volume is probably the main factor involved in the reduction of heat stress during exercise by rehydration.

Thermal and circulatory responses associated with dehydration have been studied at various levels of water deprivation, ranging from 1% of body mass\(^{5,13}\), 4%\(^{3,15}\) to 5 and 8%\(^{9,11,26}\). It has been well documented that performance is diminished when body weight losses exceed 5%\(^{1}\). In the present study the subjects showed an average loss of 3.8% in D with one round of golf (18 holes) without fluid replacement. Comparing this values with those mentioned above, it is clear that golfing without fluid intake in hot weather can be stressful and potentially dangerous to golfers of marginal fitness. This suggests that golf club should establish more water supply stations (at least every 2 holes). At the same time golfers should be advised to drink more frequently than they are accustomed.

The importance of fluid replacement during prolonged exercise, regardless of environmental temperatures, has been recognized widely in the field of sports and fitness. The optimal drink should be one that: (1) is palatable, (2) is absorbed quickly and (3) has no adverse effects on cardiovascular and thermoregulatory function\(^{19}\). The rate at which a fluid replacement beverage is absorbed into the body fluid is particularly important during prolonged exercise in a warm environment.
Our results indicate that temperature regulation and performance during golfing in a hot environment are well maintained by consuming water only. Since the intensity of work in golfing is moderate, our study demonstrates that water replacement is adequate to attenuate the rise in Tre, HR and maintain plasma volume. In the present study, an amount of water equal to that lost without fluid replacement was given to the subjects. However, since previous research has shown that when fluid intake is voluntary individuals ingest only two-thirds or less of the fluid lost, in general it may be beneficial to prepare more palatable beverages for fluid intake and encourage golfers to drink more. In a study by Candas et al., final rehydration only prevented 80% of the total body weight loss, yet they found that plasma volume was rapidly restored and expanded in exercising men in the heat. We replaced the body weight lost in D with an equal rehydration volume during R and noted a small increase in RV in R. Therefore, plasma volume expansion seems possible without excessive fluid ingestion.

Heart rates were higher in D and this is in agreement with previous studies. Because of plasma volume reduction during dehydration, an increased HR in exercising subjects is required to maintain cardiac output. Therefore, circulating blood volume restoration provides a clear advantage during exercise. Saltin concluded that heart rate elevation was primarily the result of the decrease in circulating blood volume and the results reported by Harrison et al. and Gaebelin and Senay support this conclusion. It is interesting to note that the thermoregulatory stress during D, as indicated by HR, was negligible in one subject (55 years) who has regularly jogged for years. On the other hand, the stress was more notable in another subject who was older (65 years) and not previously aerobically trained. The Tre and HR at the end of the round during D had increased by 1.0°C and 47 bpm, respectively, for the former subject and 1.6°C and 74 bpm, respectively, for the latter subject.

The levels of serum enzymes, including LDH, CK, AST and ALT, are reported to be moderately raised after strenuous exercise in cool air temperatures when body temperature is only slightly elevated. These enzymes have also been routinely monitored during recovery from acute heat illness. In men and animals, some reports have described the effects of high environmental temperatures on plasma enzyme concentrations in men and animals. In those studies hyperthermia was associated with significantly increased levels of LDH, CK, AST and ALT. Francesconi et al. found no significant increase in levels of LDH, AST and ALT after 90-min walking (5.6 km/h) in temperatures of 49°C/27°C dry/wet bulb. They attributed the absence of enzymatic responses to the combination of moderate exercise and small increments in Tre. In resting dogs, Spurr reported that the artificial heating to rectal temperatures of 41.5°C on 4 consecutive days was associated with markedly increased levels of both AST (23.5%) and ALT (27.7%). Wyndam et al. also noted that exercise in heat which raised body temperatures above 39°C resulted in a significant increase (9.1 to 14.9%) in AST, CK and LDH. However, they failed to find such an increment after the identical exercise (less than 50% of VO2max) when the exercise was conducted at room temperature.

In the present study there was no significant increase in the levels of the serum enzymes except for LDH in the subjects after golfing with fluid replacement. A likely explanation for the failure of the enzyme values to rise after golfing in the heat during R is that the exercise intensity of the sport was limited. However, golfing without fluid replacement in summer might be considered to be stressful, from our significant increase in the
levels of the serum enzymes (Table 3).

Serum enzyme levels are also affected by changes in plasma volume. If we "correct" our percentage increases in enzymes in D (Table 3) for hemoconcentration as based on $\%\Delta PV$, the increase in CK is only 16% higher than the corrected value and the other three are within 4% of the corrected values. During R the differences shown in Table 3 are increased because there was a small net increase in $\%\Delta PV$ of 4.6%. In this case the measured changes in CK and LDH concentrations increased more than shown in Table 3. This suggests that serum enzyme concentrations were not elevated more during D than R when corrected for hemoconcentration. Previous studies have typically not taken $\%\Delta PV$ changes into account when reporting enzyme concentrations. It appears that the enzymes measured did not increase as much as would be expected for intense and/or prolonged exercise of moderate intensity and since the changes in D were not of a magnitude consistent with muscle loss or lactate accumulation, they are probably not clear indicators of heat stress under these conditions.

In summary, our results indicate that frequent intake of cold water during golfing in summer is effective in reducing thermal stress. The beneficial changes in rectal temperature, heart rate and plasma volume were clearly associated with rehydration. Therefore, we can conclude that golfers should drink frequently when playing in summer or high temperature, especially those whose physical fitness is low.

ACKNOWLEDGEMENT

This study was supported by a grant for Faculty Research from Chukyo University. Valuable assistance was provided by Mr. H. Anabuki.

(Received, July 18, 1995)

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