Eur J Appl Physiol (1999) 80:353–359

ORIGINAL ARTICLE

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Intracellular hyperhydration induced by a 7-day endurance race

Accepted: 12 April 1999

Abstract To test the hypothesis that a chronic expansion of extracellular water (ECW), usually observed during prolonged endurance exercise, is associated with an increase in intracellular water space (ICW), total body water (TBW) and ECW were estimated before (within a week, day C-7) and after (on the 1st day of recovery, R + 1) a competition lasting 7 consecutive days in nine healthy sportsmen. The competition involved running, cycling and cross-country skiing over 620 km. Between days C-7 and R + 1, the following increases occurred – mean TBW by 4.2 (SEM 1.1) *l* (i.e. +10%, $P = 0.01$, bioelectrical impedance analysis, BIA, at 100 kHz) and by 4.1 (SEM 0.7) *l* ($P = 0.01$, dilution of ^{18}O); mean ECW by 2.2 (SEM 0.5) *l* (i.e. +14%, $P = 0.01$, BIA at 5kHz), and mean plasma volume (PV) by 0.7 (SEM 0.1) *l* (i.e. +22%, Evans blue dye dilution, $P = 0.008$). Consequently, mean ICW had been expanded by 2.1 (SEM 0.6) *l* (i.e. +8%, $P = 0.01$). The intensity of daily exercise evaluated from recordings of heart rate varied between 49.0% to 57.8% of maximal oxygen consumption $\dot{V}O_{2max}$. Water retention was highly correlated with relative exercise intensity $\dot{V}O_{2max}$ (ICW, $r = 0.86$; ECW, $r = 0.93$; TBW, $r = 0.94$). Total mean plasma content of sodium increased by 104 (SEM 17) mmol ($P = 0.008$) while albumin and total protein contents were unchanged. We concluded that prolonged and repeated exercise induced a chronic hyperhydration at both extracellular and intracellular levels, which was related to exercise intensity. Sodium retention was the major factor in the increase of PV.

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Key words Plasma volume · Bioelectrical impedance · ^{18}O water · Fluid compartments

Introduction

It has been well documented that prolonged strenuous exercise over several consecutive days induces a progressive increase in extracellular water space (ECW), in particular in plasma volume (PV; Williams et al. 1979; Milledge et al. 1982; Leiper et al. 1988; Fellmann et al. 1992) and total body water (TBW; Williams et al. 1979; Milledge et al. 1982; Convertino 1991). In contrast, alterations in the intracellular water compartment (ICW) have not been extensively studied. Only two studies have found that ICW was decreased by 4% (Milledge et al. 1982) and 8% (Williams et al. 1979) during 5 and 7 days of hill-walking, respectively. These results have been obtained from calculations based on cumulative water and sodium balances. However, such a chronic dehydration of the intracellular compartment is difficult to believe since interstitial water space (ITW) was shown to have expanded at the same time by 17% (Williams et al. 1979) and 9% (Milledge et al. 1982). No satisfactory explanation has been offered for the decrease in ICW. In contrast, it seems more reasonable to suggest that the osmotic pressure gradient existing between interstitial and cellular compartments could not be maintained over several days and must have favoured water movement into the cells.

The aim of this work was, therefore, to examine the changes in ICW after an endurance race lasting 7 consecutive days. We hypothesized that chronic expansions of ITW and PV would be associated with an increase in ICW. Multiple frequency bioelectrical impedance analysis (BIA) was used in this study for estimating TBW and ECW. The ICW was calculated by subtracting ECW from TBW. It has been shown that this method is non invasive, rapid and accurate for estimating water distribution (Segal et al. 1991; Baumgartner 1996). To avoid the post-exercise conditions that have been shown

to affect measured body resistance (Deurenberg et al. 1988; Kahled et al. 1988), TBW and ECW were measured only in basal and resting conditions. In addition, TBW estimations were also made using the dilution and principle with ^{18}O labelled water, a method which has been found to be the best probe for TBW measurement (Schoeller et al. 1980; Schoeller 1996).

Methods

Subjects

Nine men who had participated in a 7-day national French endurance race, the Trans-Massif Central, volunteered for the study and gave informed written consent. The experiment protocol was approved by the Ethics Committee on Human Research for Medical Sciences. The subjects [mean age 42.1 (SEM 2.5) years, range 30–35 years] were endurance-trained sportsmen. Their level of physical fitness was evaluated by maximal oxygen consumption mean [$\dot{V}O_{2max}$ 52.4 (SEM 7.2) ml · min^{-1} · kg^{-1}, range 42.1–68.0 ml · min^{-1} · kg^{-1}].

The competition

The total distance was 620 km and the distance covered each day varied from 66 to 120 km at altitudes up to 1,500 m. Overall the competitors ran 133 km, cycled 453 km using a mountain bicycle and cross-country skied 34 km (Table 1). Air temperature varied from 0° to 20°C. Each day the races started between 7 a.m. and 8 a.m. and finished between 4 p.m. and 6 p.m. depending on the distance covered. Durations of the races varied daily from 366 to 614 min. The subjects received water and food ad libitum with no possible control.

Protocol

Measurements were performed on two occasions:

1. Before the race (control period, C-7) within the week prior to the start of the competition
2. The morning following the end of the race (recovery period, R + 1).

All measurements of body mass and body water spaces were performed in the post-absorptive state, after an overnight fast of at least 12 h. The subjects were weighed to ±0.2 kg on a SECA 709 scale (Seca, Les Mureaux, France) at 8 a.m. on C-7 and R + 1.

Bioenergetic measurements

Maximal exercise On C-7, maximal oxygen uptake ($\dot{V}O_{2max}$) was measured in the laboratory using a direct method on a cycle ergometer. Heart rate (HR) was obtained from continuous electrocardiogram recording. After a 10-min warm-up yielding an exercise HR of 120–130 beats · min^{-1}, the subjects progressively increased their exercise intensity by four or five successive 4-min steps until they felt exhausted. Oxygen consumption ($\dot{V}O_2$) was measured continuously by open-circuit spirometry and averaged every 30 s using an automated on-line system (Med Graphics CPX I D). The $\dot{V}O_{2max}$ was considered as having been reached when exhaustion, a respiratory gas exchange ratio above unity and HR close to the theoritical maximal value were achieved.

Relative exercise intensity during the race Each day of the race, during each exercise session, HR was recorded every minute with a commercially available device (Sport Tester PE4000 Polar Electro, Kempele, Finland). The average daily exercise intensity (% $\dot{V}O_{2max}$) was assessed from the mean daily race HR and the relationship between HR and $\dot{V}O_{2max}$ in each individual established at the laboratory on C-7 during the maximal exercise test.

Body water compartments

Bioelectrical impedance analysis Body resistances were measured on days C-7 and R + 1 after a 12-h overnight fast, with an Analycor 3 impedancemeter with four electrodes at two frequencies (Eugédia, France). On R + 1, the last exercise period had occurred at least 14 h before the measurement. The subjects had been in a relaxed supine position for at least 30 min in a temperature controlled room (20–22°C) when the measurements were made and the limbs were abducted from the body during the measurements. The injector electrodes were placed just below the phalangeal-metacarpal joint in the middle of the dorsal side of the right hand and just below the transverse (metatarsal) arch on the superior side of the right foot. Detector electrodes were placed on the posterior side of the right wrist, midline with the prominent pisiform bone on the medial (fifth phalangeal) side and ventrally across the medial ankle bone of the right ankle. The precision of the measurements was assessed on 50 healthy volunteers on whom the resistance was measured 8 h apart: the coefficient of variation was shown to be 0.6% (Vaché et al. 1998). Resistance and reactance were measured at 5 and 100 kHz. The TBW and ECW were calculated from resistance measured at 100 kHz (R100) and 5 kHz (R5), respectively, using the equations developed and cross-validated by Segal et al. 1991:
TBW (l) = 0.454796 height^2 (cm^2)/R100 + 0.139523 mass (kg) + 3.432026. The residual SEE of this equation was 2.64 l.

Table 1 Mean daily distances and competition times in the Trans-Massif Central Race, together with mean daily race heart rate and exercise intensity in the nine subjects during the race. % $\dot{V}O_{2max}$ Exercise intensity related to maximal oxygen consumption

Days	1	2	3	4	5	6	7	ANOVA P
Distance (km)	103	70	66	102	74	85	120	
Time (min)								
Mean	614	366	512	552	326	498	521	
SEM	14	6	10	15	5	10	6	< 0.0001
Mean daily heart rate (beats · min^{-1})								
Mean	126	123	122	115	119	117	114	
SEM	4	4	3	3	4	3	3	< 0.0001
% $\dot{V}O_{2max}$								
Mean	57.4	54.6	53.9	51.0	51.4	51.6	48.0	
SEM	2.1	2.2	2.1	1.8	2.1	3.0	2.4	< 0.0001

ANOVA, *P* analysis of variance for repeated measures

ECW (l) = 0.284021 height^2 (cm^2)/R5 + 0.111926 mass (kg) − 6.115278. The residual SEE of this equation was 1.94 l.

The ICW was calculated as the difference between TBW and ECW (calculated from BIA measurements).

$H_2^{18}O$ dilution The TBW was calculated from ^{18}O dilution space as described by Coward (1990), with a 1% correction for exchanges with non-aqueous compounds (Schoeller et al. 1980). After collection of a baseline urine sample, accurately weighed amounts of 2% ^{18}O enriched water (1 g · kg^{-1} body mass, Enritech, Rehovot, Israel) were taken orally by the volunteers. Urine samples were collected again 4, 5, and 6 h post-dose. Volunteers remained fasted (water and food) during these 6 h, but were permitted light activity within the laboratory. Urine samples were kept at −20°C until analysis.

The ^{18}O enrichments were measured using the CO_2-H_2O equilibration technique according to Ritz et al. (1994) adapted for use with Vacutainers on a continuous flow gas chromatography-isotope ratio mass spectrometer (μgas, VG Isotech, UK). The mean enrichment (4–6 h post dose) net of predose was used to calculate ^{18}O dilution spaces. In our laboratory, the precision of repeated TBW measurements has been shown to be 0.7% (Vaché et al. 1998).

Plasma volume

The PV was measured using the Evans blue dye dilution technique (Foldager and Blomqvist 1991). A catheter was introduced into a cubital vein of each arm (one for the injection of the dye and the other for collection of blood samples) of the volunteers who had been in a supine position for at least 30 min, this being the equilibration period (Diaz et al. 1979). Baseline samples were collected 15, 10 and 5 min before an accurately weighed amount of Evans blue solution (2.5 ml at 5 mg · ml^{-1} concentration) was injected. To rinse the catheter it was flushed with 10 ml of saline. Blood samples were drawn 5, 7, 10, 15 and 20 min after injection. Evans blue concentrations were calculated from optical densities measured at 620 nm (and corrected for optical density at 740 nm; Foldager and Blomqvist 1991) of plasma and standard solutions on a Unicam 8625 UV/VIS spectrophotometer (Unicam Ltd, Cambridge, UK). Time 0 concentration was calculated by back extrapolation of the dilution curve with time and used to calculate PV.

The PV changes were also calculated from changes in haematocrit (Hct) and haemoglobin concentration (Hb) between C-7 and R + 1. The relative changes in PV (Δ PV%) were calculated according to the equation (Strauss et al. 1951):

$$\Delta PV\% = 100\left[\frac{Hb_{c-7}}{Hb_{R+1}} \times \frac{1 - Hct_{R+1} \cdot 10^{-2}}{1 - Hct_{C-7} \cdot 10^{-2}}\right] - 100$$

The ITW was calculated as ECW-PV.

Biochemical parameters

Blood was drawn from an antecubital vein at 8 a.m. before injecting Evans blue dye. The Hct and Hb concentration were determined by the Cobas Minos STE (ABX). Plasma concentrations of Na^+, urea, total proteins were measured on a Hitachi 911 autoanalyser. Albumin concentrations were measured with an immunological technique. Plasma contents of proteins, albumin and Na^+ were calculated as the product of PV and plasma concentration.

Statistical analyses

All data analyses were performed with the Statview statistical package (Abacus concept). ANOVA was used for the day-to-day comparison of the characteristics of the race (Table 1). The non-parametric paired Wilcoxon test was used to assess the significance between control and recovery values. The relationships between variables were analysed by correlation analyses. Statistical significance was accepted at $P < 0.05$.

Results

Performance – daily mean HR and % $\dot{V}O_{2max}$ varied during the competition (Table 1). The highest mean values were observed on the first day of the race [HR = 126 (SEM 4) beats · min^{-1} corresponding to 57.4 (SEM 2.1)% $\dot{V}O_{2max}$]. The lowest mean values were observed on the last day [HR = 114 (SEM 3) beats · min^{-1} corresponding to 48.0 (SEM 2.4)% $\dot{V}O_{2max}$]. The mean exercise intensity over the 7 days was 52.5 (SEM 1.8)% $\dot{V}O_{2max}$.

Mean body mass increased from 68.1 (SEM 2.3) kg (on day C-7) to 68.4 (SEM 2.2) kg (on day R + 1) without reaching statistical significance.

Body water compartments – mean TBW measured with ^{18}O labelled water increased by 4.1 (SEM 0.7) l [i.e. +9.8 (SEM 1.5)%, $P = 0.01$]. Similarly, mean TBW measured from BIA measurements increased by 4.2 + 1.1 l [i.e. +10.3 (SEM 2.6)%, $P = 0.01$]. The mean TBW calculated using the two techniques did not differ significantly and the individual changes obtained using the BIA and ^{18}O dilution techniques were highly correlated: $r = 0.87$, $P = 0.005$ (Fig. 1). The mean ECW increased by 2.2 (SEM 0.5) l [i.e. +13.8 (SEM 2.9)%, $P = 0.01$]. Mean calculated ICW was expanded by 2.1 (SEM 0.6) l [i.e. +8.2 (SEM 2.5)%, $P = 0.01$, Table 2].

The mean PV increased by 0.7 (SEM 0.1) l (Evans blue dye dilution, $P = 0.008$, Table 2). The magnitude of the changes in PV was similar to that calculated from the changes in Hct and Hb [+22.4 (SEM 3.6)%]. The mean ITW was also increased by 1.5 (SEM 0.5) l [i.e. + 11.8 (SEM 4.0)%, $P = 0.04$].

Changes in plasma variables are displayed in Table 3. Mean plasma concentrations in albumin (and total proteins) decreased by 18.5 (SEM 1.3)% [and 17.9 (SEM 0.2)%] between days R + 1 and C-7. No such changes in mean plasma Na^+ concentrations were observed. Therefore, mean plasma Na^+ content increased by 104 (SEM 17) mmol [i.e. 23.6 (SEM 3.7)%] while mean total protein and albumin contents remained unchanged. Mean plasma urea concentrations increased significantly by 3.5 (SEM 0.9) mmol · l^{-1} [+63.4 (SEM 16.4)%].

Correlations between variables – there were significant correlations between the mean exercise intensity (calculated over the 7 race days) and the extent of the water volume expansions (R + 1 compared to C-7) for TBW ($r = 0.94$, $P = 0.0006$), ECW ($r = 0.93$, $P = 0.0009$; Fig. 2), ICW ($r = 0.86$, $P = 0.006$; Fig. 3), ITW ($r = 0.83$, $P = 0.01$) and PV ($r = 0.70$, $P = 0.05$). No significant correlations were found between individual changes of any water compartments and either individual exercise duration or subject initial fitness ($\dot{V}O_{2max}$ in millilitres per minute per kilogram).

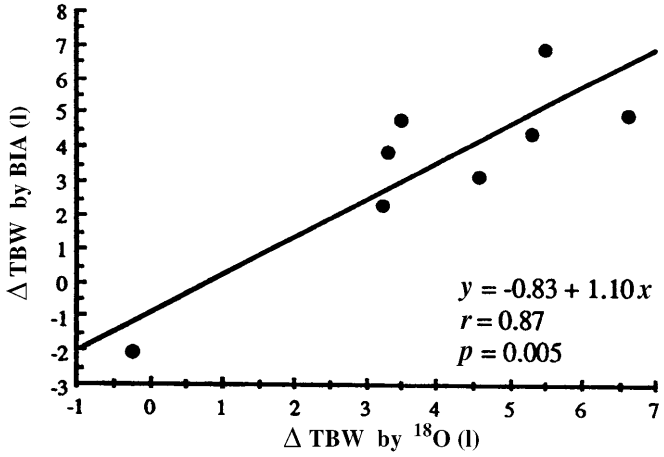

Fig. 1 Relationship between the individual changes in total body water (ΔTBW) measured by ^{18}O dilution and bioelectrical impedance analysis (BIA) methodology on the recovery day compared to control data (1 week before the competition)

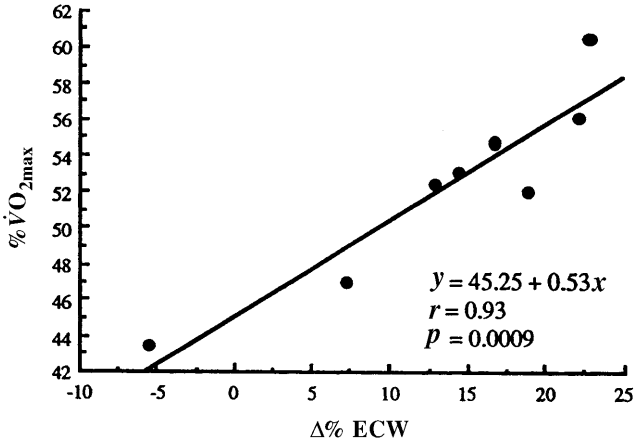

Fig. 2 Relationship between the changes in extracellular water compartment ($\Delta\%ECW$) on the recovery day compared to control data (1 week before the competition) and the mean exercise intensity of the 7-day competition related to maximal oxygen consumption ($\%\dot{V}O_{2max}$). One subject was omitted because of poor heart rate recording

Table 2 Body water compartments 1 week before the competition (*C-7*) and on the 1st day of recovery (*R+1*). *TBW*, *TBW* (^{18}O). Total body water space measured by bioelectrical impedance analysis and by dilution of ^{18}O labelled water, respectively, *ECW* extracellular water compartment measured by bioelectrical impedance analysis, *ICW* intracellular water space calculated from bioelectrical impedance analysis as *TBW-ECW*, *PV* plasma volume measured by Evans blue dye dilution, *ITW* interstitial water space calculated as *ECW-PV*

	TBW (l)	TBW (^{18}O) (l)	ECW (l)	ICW (l)	PV (l)	ITW (l)
C-7						
Mean	41.6	41.3	15.8	25.8	3.15	12.6
SEM	1.5	1.2	0.7	0.8	0.14	0.7
P	0.01	0.01	0.01	0.01	0.008	0.04
R+1						
Mean	45.8	45.4	18.0	27.9	3.86	14.1
SEM	1.9	1.5	1.0	0.9	0.2	0.9

The C-7 and R+1 values were compared by a non-parametric paired Wilcoxon test and the resulting *P* values are shown

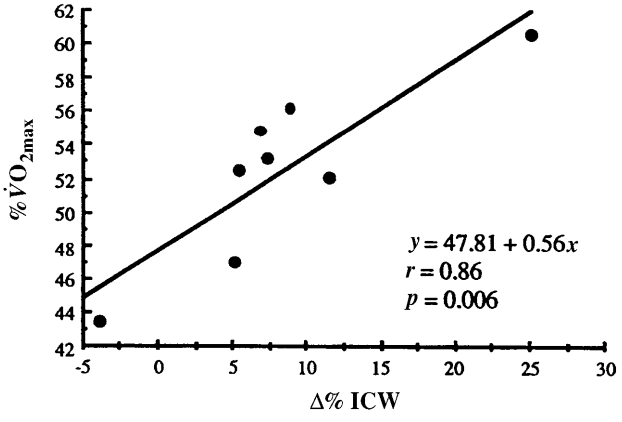

Fig. 3 Relationship between the changes in intracellular water compartment ($\Delta\%ICW$) on the recovery day compared to control data (1 week before the competition) and the mean exercise intensity of the 7-day competition related to maximal oxygen consumption ($\%\dot{V}O_{2max}$). One subject was omitted because of poor heart rate recording

Table 3 Plasma variables obtained 1 week before the competition (*C-7*) and on the 1st day of recovery (*R+1*)

	Na^+ concentration ($mmol \cdot l^{-1}$)	Na^+ content (mmol)	Total proteins concentration ($g \cdot l^{-1}$)	Total protein content (g)	Albumin concentration ($g \cdot l^{-1}$)	Albumin content (g)	Urea concentration ($mmol \cdot l^{-1}$)
C-7							
Mean	139.8	440.5	73.6	232.6	42.3	133.0	5.8
SEM	0.3	20.3	1.4	13.8	0.7	5.8	0.4
P	NS	0.008	0.008	NS	0.008	NS	0.008
R+1							
Mean	141.1	544.5	60.4	233.9	34.4	132.3	9.3
SEM	0.9	29.7	1.9	15.6	0.6	6.3	0.9

The C-7 and R + 1 values were compared by a non-parametric paired Wilcoxon test and resulting *P* values are shown. *NS* non significant

Discussion

The present study was intended to measure changes in body water compartments after an endurance race lasting 7 days. It was the first study to show an expansion of ICW (+8%) simultaneously with increases in ITW and PV as a consequence of a long-lasting endurance race.

The expansion of ECW induced by endurance exercise has been well documented (Williams et al. 1979; Milledge et al. 1982; Leiper et al. 1988; Fellmann 1992). Our results clearly indicated the major contribution of Na^+ to the expansion of PV since neither plasma albumin nor protein contents had changed by the end of the race (Table 3). A different mechanism has been shown to prevail after intermittent supra-maximal exercise (Gillen et al. 1991) and during training induced hypervolaemia (Convertino et al. 1980) where increase in plasma albumin content has been found to be a driving force for PV expansion (Haskell et al. 1997). It has been suggested that ECW expansion could be a direct consequence of plasma aldosterone activation by renin-angiotensin during successive days of exercise (Williams et al. 1979; Milledge et al. 1982; Wade et al. 1985). It is of interest that it has been found that catecholamine secretion is the most important regulator for renin secretion (Kotchen et al. 1971). Galbo et al. (1975) have reported that the magnitude of catecholamine secretion is related to exercise intensity. Therefore, this may explain the correlation between ECW (Fig. 2) or PV, and exercise intensity observed in the present study. Within the extracellular space we found a discrepancy between the increase in PV (+22%) and the increase in ITW (+12%). However, a similar discrepancy has already been described by Williams et al. (1979; +22% vs +17%), and by Milledge et al. (1982; +25% vs +9%). Since plasma albumin content was not increased, a reduced hydrostatic pressure in the great veins or small change in capillary hydrostatic pressure may have shifted the net flux of water in favour of inward movement to the vessels as has been suggested by Williams et al. (1979).

The present finding of an increase in ICW is not in agreement with the only two other studies that have tackled this issue. In these studies (Williams et al. 1979; Milledge et al. 1982), an increase in TBW and ECW but a decrease in ICW were calculated as a consequence of successive days of exercise. Furthermore, part of the ECW expansion was suggested to be the consequence of Na^+ retention, the remainder being caused by a shift of water from the intracellular to the extracellular space. Although ICW is calculated as the difference between TBW and ECW, the methodological approaches have differed. Williams et al. (1979) and Milledge et al. (1982) based their calculations on cumulative water balances (for TBW) and Na^+ balance (for ECW). These calculations relied on a certain number of assumptions that might not be tenable during exercise: metabolic water production and water excreted from the lungs and evaporated from the skin were not measured but calculated. The Na^+ loss in sweat was not taken into account and fat mass was assumed to be constant, which is unlikely since it would lead to erroneously low water balances. Indeed, in the study of Milledge et al. (1982) the calculated water balance (+920 ml) was almost twice as small as the actual changes in TBW measured using ^3H dilution (+1.92 *l*). These assumptions make this chronic cellular dehydration questionable.

In the present study, ECW and TBW were estimated by BIA. It is now generally accepted that BIA provides relevant hydration information in athletes when test conditions are well controlled (Segal 1996). To avoid post-exercise conditions that have been shown to affect measured body resistance (increase in skin temperature, vascular perfusion, cutaneous blood flow, glycogen depletion; Deurenberg et al. 1988; Kahled et al. 1988), resistances were only measured at rest. Both on control and recovery days, resistance measurements were performed on overnight fasted volunteers, at least 14 h after the last exercise session. They lay supine on a bed for at least 30 min in a temperature controlled room (20–22°C). Therefore, we are convinced that optimal test conditions were strictly adhered to, and that our measurements were reliable. Furthermore, estimates of TBW obtained with the dilution of ^{18}O (which has been shown to be the best probe for measuring TBW; Schoeller et al. 1980) were very similar to those obtained by BIA and these values were well correlated (Fig. 1). Controls for hydration and diet (including Na^+ intake) were not feasible under field circumstances and would not have been accepted by the competitors. In any case, an accurate water balance would have been impossible since evaporative water losses could not be measured. Furthermore, water balance informs about TBW, and not about water distribution within the body.

There is an apparent paradox in the increase in TBW (+4.2 *l*) unmatched by changes in body mass. However, similar observations have been made by Milledge et al. (1982). After a shorter period of exercise (5 days) at a lower intensity (hill walking, compared to the present competition), an increase in 1.92 *l* TBW was noted while body mass did not change (72.5 vs 72.6 kg). If the TBW increase was matched by an equivalent fat loss, this would correspond to an unacceptable negative energy balance ($\sim$4,000 kcal $\cdot$ day^{-1}). However, it is possible that a loss of protein also occurred. A highly catabolic hormone environment has often been described during competition (Lemon and Proctor 1991). During a similar race (Fellmann et al. 1992), we have previously shown that plasma cortisol concentrations were continuously maintained at high levels. This favours a breakdown of protein. The significant increase in plasma urea concentration (+63% observed here) provides indirect evidence for proteolysis. Furthermore, a decrease in protein synthesis has also been described during endurance events (Dohm et al. 1987; Lemon and Proctor 1991). Therefore, protein loss, which represents less energy than fat (1 g = 4 kcal), and which costs energy (for

proteolysis) is likely to occur at the same time as fat loss. The actual energy balance would therefore be less negative than that calculated above.

What can be the driving force for ICW retention? The increase in cell osmotic pressure gradient could shift water into the cells. Accumulation of substrates produced during muscle contraction would increase muscle cell osmolality and drive water into cells. This has been shown in rats where running uphill has induced swelling of the muscle fibres (Peeze-Binkhorst et al. 1990). Similarly, during intense dynamic exercise in humans, muscle biopsies have been used to show an increase by 10% in ICW of active muscles while no change occurred in the inactive ones (Sjøgaard and Saltin 1982). The end products of proteolysis could be among the substrates accumulated in cells. Increased intracellular lactate concentration has also been reported to increase muscle osmolality. This is unlikely to be the explanation in the present study since exercise intensity was moderate (52.5% of $\dot{V}O_{2max}$ as a mean) and even if lactate was produced during the race, it would have already returned to its resting level when the measurements were made. Nevertheless, the strong correlations between ICW changes and mean relative exercise intensity (Fig. 3) would suggest that factors favouring ICW retention were dependent on exercise intensity.

Finally, water retention in muscle cells can arise from glycogen overcompensation. However, to match the increase in ICW observed here, an overcompensation by about 800-g glycogen would be required. This is impossible. Glycogen overcompensation only plays a small role, if any, in water retention. Conversely, glycogen consumed during the last exercise session should have been resynthesised almost completely during the night preceeding the R + 1 measurements.

In conclusion, the present study has demonstrated an increase in TBW, ECW and ICW as a consequence of a prolonged endurance race. These findings support the concept that increased interstitial water content may induce a subsequent redistribution of extracellular water to the cell compartment. The PV retention was related to Na^+ retention. Further studies are necessary to elucidate the mechanism underlying exercise-induced cellular water retention. Beneficial aspects of hyperhydration can be foreseen since studies on liver (Haüssinger et al. 1994) and muscle cells (Parry-Billings et al. 1991; Low et al. 1996) have given in vitro evidence that cell swelling acts as an anabolic signal whereas cell shrinkage leads to catabolism.

Acknowledgements This work was supported by the GREESE (Société des Eaux de Volvic), by the Direction Régionale Jeunesse et Sport Auvergne, and by the Région Auvergne. The authors gratefully acknowledge the participation of the subjects and their splendid cooperation, without which this study could not have been done. Special thanks are due to Dr C. Viladent from the ABX manufacturer for supplying the device for haematological measurements. They thank Dr C. Motta (Laboratoire de Biochimie, Hôtel Dieu, Prof. B. Dastugue) and Prof. C. Gentou (Laboratoire de Biochimie Médicale, Hôpital Gabriel Montpied) for biochemical measurements, Prof. A. Chamoux (Laboratoire de Médecine du Travail) for heart rate recording and Prof. C. Gharib (Laboratoire de Physiologie et de l'Environnement, Lyon) for providing the Evans blue dye. They also thank Miss Solari for her technical assistance and Mrs Cohendy for typing the manuscript.

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