Water and electrolyte exchanges of obese patients on a reducing regimen

By R. PASSMORE, J. A. STRONG AND FIONA J. RITCHIE
Departments of Physiology and Medicine, University of Edinburgh and the Western General Hospital

(*Received* 24 *March* 1958—*Revised* 13 *August* 1958)

Fat people who attempt to reduce their weight by dieting are often successful at first. After a short period of rapid weight loss, progress is usually less well maintained and may slow down or come to a complete stop before the desired new weight is reached. This frequently happens even when the patient has been carefully supervised and has been collaborating conscientiously. The obese normally carry a moiety of body water which they rapidly lose early in the course of treatment by strict dieting (initial dehydration), but thereafter they may increase their relative body water content (secondary water retention) (Strong, Passmore & Ritchie, 1958; Passmore, Strong & Ritchie, 1958). In these papers we gave quantitative assessments of the initial water losses and subsequent water retention by seven obese patients, each of whom was studied for 6 weeks on a strictly controlled and rigorous reducing regimen. In five of these patients, a complete water balance was derived for the whole period. It comprised the total fluid intake, the metabolic water, the urine, the faecal water and the evaporative water loss. In addition, the sodium and potassium contents of the diet and of the urine and faeces were measured. This paper gives the results of these studies.

METHODS

The clinical histories of the five patients (Mrs L., Miss McN., Miss B., Miss M. and Mr W.) and the details of their dietary regimens and physical activities are recorded in a previous paper (Strong *et al.* 1958). The changes in their body water (ΔH_2O) during the reducing regimen were calculated from the equation:

$$\Delta H_2O = \text{change of body-weight} - \text{tissue protein metabolized} - \text{tissue fat metabolized}.$$

The tissue protein metabolized was calculated from the nitrogen balance and the tissue fat metabolized from the energy balance (Passmore *et al.* 1958).

Here we present values for the full water balance calculated as follows:

$$\Delta H_2O = (\text{ingested water} + \text{metabolic water}) - (\text{urinary water} + \text{faecal water} + \text{evaporative water}).$$

Ingested water. All liquids consumed were measured. Portions of replicate homogenized diets were dried to constant weight on a water bath and then in a desiccator and the water in the food was determined by the difference in weight.

Metabolic water. It was determined from the total amounts of protein, fat and carbohydrate in the diets, together with the amounts of tissue protein and tissue fat metabolized, which had already been calculated (Passmore *et al.* 1958). The water of oxidation of 1 g protein, carbohydrate and fat was taken as 0·41, 0·60 and 1·07 g respectively.

Urinary water. See p. 19.

Faecal water. Portions of faeces were dried to constant weight.

Evaporative water. It's weight was derived from the invisible weight loss by adding the weight of oxygen consumed and subtracting the weight of carbon dioxide produced. These weights of oxygen and carbon dioxide were calculated from the metabolic mixture, determined by adding the losses of tissue protein and fat to the dietary protein, fat and carbohydrate. As the metabolic mixtures were composed predominantly of fat (the respiratory quotients were around 0·70 (see Table 3, Strong *et al.* 1958)), weights of oxygen approximated very closely to weights of carbon dioxide. This correction for the invisible weight loss was therefore small.

The invisible weight loss was determined from the equation:

Invisible weight loss = change of body-weight + weight of solids and liquids ingested − weight of urine and faeces.

These values are easily obtained by simple measurement.

The calculation of the full water balance might be thought to provide a check on the water balance determined previously from the changes in body composition. It is not so, as the same calculation of the metabolic mixture is common to both determinations. That the two methods should give identical results can be proved by the following calculations, where P = protein, F = fat, C = carbohydrate, S = salts, R = roughage, W = weight and, written as subscript, i = ingested, u = urinary, f = faecal, t = tissue, m = metabolic, e = evaporative.

Changes in body water are calculated from changes in body composition as follows:

$$\Delta H_2O = \Delta W_t - \Delta P_t - \Delta F_t - \Delta C_t - \Delta S_t \quad (1)$$

In practice ΔC_t and ΔS_t are assumed to be negligible in comparison with the other components.

Changes in total body water are calculated from the full water balance as follows:

$$\Delta H_2O = (H_2O)_i + (H_2O)_m - (H_2O)_u - (H_2O)_f - (H_2O)_e \quad (2)$$

but $(H_2O)_i$ is obtained from

$$(H_2O)_i = W_i - P_i - F_i - C_i - S_i - R_i \quad (3)$$

and $(H_2O)_m$ from the equation of the metabolic mixture

$$(1-a)(P_i - P_f - \Delta P_t) + (C_i - C_f - \Delta C_t) + (F_i - F_f - \Delta F_t) + (O_2)_m = (H_2O)_m + (CO_2)_m \quad (4)$$

where a is that portion of the protein molecule that is not oxidized.

Now $(H_2O)_u$ is given by

$$(H_2O)_u = W_u - a(P_i - P_f - \Delta P_t) - (S_i - \Delta S_t) \quad (5)$$

$(H_2O)_f$ by

$$(H_2O)_f = W_f - P_f - F_f - C_f - R_i \quad (6)$$

and $(H_2O)_e$ by

$$(H_2O)_e = IWL + (O_2)_m - (CO_2)_m \quad (7)$$

but IWL (invisible weight loss) is measured indirectly thus

$$IWL = W_i - \Delta W_t - W_u - W_f \quad (8)$$

Now by substituting in equation (2) from equations (3)–(8) we derive again equation (1). Thus the two equations are not independent. $\Delta(H_2O)_t$ calculated from values derived from equation (2) must agree with the value for $\Delta(H_2O)_t$ derived from equation (1). If the two values do not agree, then there is an error in collection of the basic values.

When the calculations were complete, we found that the value of ΔH_2O obtained from the full water balance was always about 20–30 g/day less than that calculated from the change in body-weight. After much thought and inquiry, we discovered that this discrepancy was due to equating volume of urine with weight of urinary water. Trapp (1850) pointed out that urinary solids cause expansion of urinary volume. For calculating the solids in 1 l. urine he set out the formula:

$$\text{Urinary solids (g/l.)} = (1000 \times \text{sp.gr.} - 1000) \times 2.$$

Our patients usually passed urine of high specific gravity and so the volume of urinary water was significantly below that of urine. A correction for this factor has been made in the results presented in Table 1. In this table, all the figures have been rounded off and the changes in body water are the same as previously recorded (Table 3, Passmore *et al.* 1958). Apart from the conversion of the volumes of urine into volumes of urinary water, this equating of the two sets of results did not involve changes larger than 15 g in any one balance, which is certainly within the limits of accuracy of the observations.

Sodium and potassium. They were determined with a flame photometer in food, faeces and urine. The food and faeces were dried at 100° for the determination of water content and subsequently ashed at 500°. The ash was extracted with 1 ml. conc. HCl and diluted to the appropriate volume.

RESULTS AND DISCUSSION

Water balance

Table 1 sets out the water balances for each of the five subjects. It will be seen that during the first experimental period each subject had a large negative balance, but in subsequent periods balances were less negative and were sometimes even positive. Of the five components that make up the water balance, two (the metabolic and faecal water) are relatively small and constant, as a brief inspection of Table 1 shows. The other three components (ingested, urinary and evaporative water) are larger and more variable and share responsibility for the regulation of body-water content. Their relation to changes in body water are shown in Fig. 1.

Urinary water. The view is commonly held by clinicians (Newburgh, 1950) that the rapid loss of weight which may occur from time to time in obese patients under treatment can be attributed to diuresis. Conversely, their failure to lose weight may be ascribed to water retention by the kidneys.

This conception of the causes of changes in weight would not apply to most of our patients. Fig. 1 shows the relation between changes of body-water content and the three principal routes of water exchange. In Mrs L., Miss McN. and Miss B. there was obviously little association between urinary water and the loss of body water. In Miss M., however, there was a positive correlation between these two quantities which approached significance ($r = 0{\cdot}65$; $0{\cdot}1 > P > 0{\cdot}05$); but for Mr W. the relation was not significant. These correlations were calculated for all the periods, including those when urea was given. It would seem therefore that, with the exception of Miss M., changes of body water in these patients were not associated with marked diuresis or antidiuresis.

Table 1. *Water exchanges (ml./day) of obese patients on a reducing regimen*

Subject	Days	Water balance	Ingested water	Metabolic water	Urinary water	Faecal water	Evaporative water
Mrs L.	1–7	−440	1770	390	550	60	1990
	8–14	−240	1530	390	630	70	1460
	15–21*	+130	1280	370	500	40	980
	22–28*	+70	1240	310	580	20	880
	29–35	+100	1240	360	450	20	1030
	36–42	−150	1300	310	700	10	1050
Miss McN.	1–7	−300	1670	380	700	90	1560
	8–14	−10	1830	380	680	30	1510
	15–21	+10	1800	380	540	30	1600
	22–28	−50	1550	370	480	30	1460
	29–35	−10	1530	340	440	30	1410
	36–42†	+50	1580	360	550	30	1310
Miss B.	1–7	−390	1370	250	630	50	1330
	8–14	+40	1420	330	430	40	1240
	15–21	+50	1200	340	370	20	1100
	22–28	+10	1210	340	590	10	940
	29–35	+110	1240	370	570	20	910
	35–42	−10	1200	340	700	30	820
Miss M.	1–5	−570	1550	400	570	30	1920
	6–10	−160	1350	380	450	40	1400
	11–15	+70	1600	390	380	20	1520
	16–20	+240	1940	380	410	60	1610
	21–25	−20	1630	390	540	70	1430
	26–30§	−140	1740	390	580	40	1650
	31–35	+150	1990	360	300	90	1810
	36–40‡	+100	1510	380	370	110	1310
	41–45	−340	1950	330	800	160	1660
Mr W.	1–5	−580	1840	430	1110	80	1660
	6–10	+200	1770	430	730	30	1240
	11–15	−90	1580	410	530	50	1500
	16–20‡	+180	1840	410	430	20	1620
	21–25	−210	1630	370	880	60	1270
	26–30§	−140	2720	380	1620	40	1580
	31–35§	+60	2160	360	900	50	1510
	36–40	0	1940	370	750	40	1520

* Diet supplied about: protein 50, carbohydrate 35, fat 15 g/day.
† Diet supplied about: protein 80, carbohydrate 50, fat 30 g/day.
‡ Diet supplied about: protein 25, carbohydrate 200, fat 10 g/day.
§ 15 g urea given daily.
In other periods, the diet supplied about: protein 25, carbohydrate 40, fat 15 g/day.

Evaporative water losses. Observations by Kekwick & Pawan (1956) suggested that water lost by evaporation might be an important factor in determining changes in the body water of obese patients. They showed that on diets providing 1000 Cal., of which 90% was derived from carbohydrate, invisible weight losses were much less than on isocaloric diets in which fat was substituted for carbohydrates. It is clear from Fig. 1 that in our patients evaporative water losses were very variable though only in Mrs L. was the correlation with the loss of body water close and statistically significant ($r = +0{\cdot}80$; $0{\cdot}02 > P > 0{\cdot}01$). This finding contrasts with Newburgh's (1950) that in healthy subjects and in diabetics losses of water by evaporation were remarkably constant and accounted for between 23 and 25% of the total calories dissipated. In our investigation calorie expenditure was relatively constant in each patient (see Table 3, Passmore *et al.* 1958) and it might therefore have been expected that evaporative water losses would have been similarly uniform.

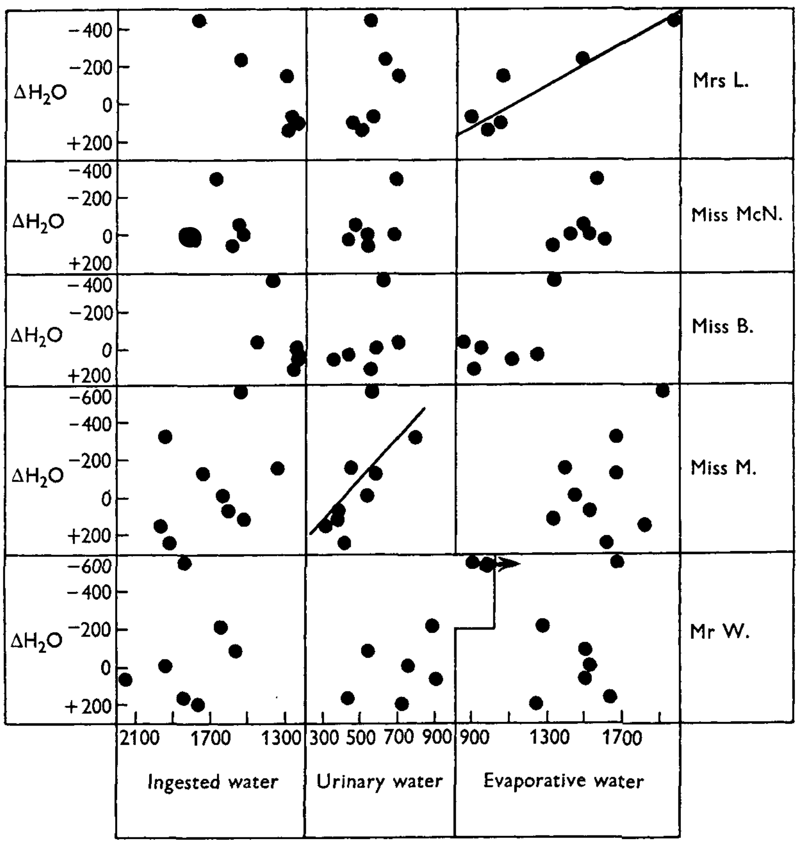

Fig. 1. Relation between the change in body water (ΔH_2O) and ingested water, urinary water and evaporative water. Each point represents a daily mean for observation periods lasting 7 days (Mrs L., Miss McN. and Miss B.) or 5 days (Miss M. and Mr W.). ●→, aberrant value.

The variations we have observed might be due to changes in environmental temperature or in visible sweat loss during walking exercises. We have no records of the temperature of the hospital ward throughout these observations, but we believe that there were no considerable changes during successive periods of observation in each patient; these were also limited to a relatively small part of the season of a year. The patients did not appear to sweat unduly. During the periods of exercise, evaporative water losses were low; they were less than would be predicted from Newburgh's (1950) observations on normal people—and much less for Mrs L. and Miss B. Furthermore, no association was found between evaporative water losses and calories expended during walking.

We consider, therefore, that irregularities in the evaporative water losses are real, and that in this respect obese patients differ markedly from Newburgh's subjects. We know of no accurate studies of temperature regulation in obese patients, and our results, together with those of Kekwick & Pawan (1956), suggest that such an investigation might be profitable.

During these studies, two patients were given high-carbohydrate diets similar to those prescribed by Kekwick & Pawan (1956). On this diet, Miss M. showed a fall in evaporative water loss and a positive water balance (Table 1). This result therefore agrees with the findings of Kekwick & Pawan. By contrast, Mr W. had a high rate of evaporative water loss on this diet though he also was in positive water balance.

Table 2. *Energy intake and invisible weight loss and heat exchanges of obese patients on a reducing regimen*

Day	Energy intake (Cal./day)	Weight (kg)	Metabolic rate (Cal./min)	Invisible weight loss (g/h)	Evaporative heat loss as percentage of total
		Miss M. (sitting)			
1	400	118·7	1·40	33	23
3	400	116·4	1·72	44	24
10	400	112·6	1·41	30	21
37	1000 90% carbohydrate	105·8	1·33	24	17
38	1000 90% carbohydrate	105·4	1·33	23	17
		Mr W. (sitting)			
0	(Home diet)	131·1	2·15	57	26
4	400	126·9	1·75	41	22
7	400	125·6	1·54	35	22
18	1000 90% carbohydrate	123·0	1·41	52	36
19	1000 90% carbohydrate	122·8	1·60	55	33

The values for evaporative water loss recorded in Table 1 are the means for 5-day periods, during which daily weight changes were recorded on a balance only sensitive to 30 g. A few values were obtained for both subjects when sitting quietly for 3 h, with a balance which could register changes in weight of as little as 2 g. The results are presented in Table 2 and confirm the changes shown in Table 1. Miss M. showed a fall and Mr W. an increase in invisible weight loss when on the high-carbohydrate diet.

Further studies on the evaporative water losses of obese patients under conditions where the environmental temperature is strictly controlled are obviously needed.

Ingested water. There were no significant correlations between the amount of water drunk and the calculated water balance. All the patients had free access to water and were instructed to drink as much as they liked. It is possible, however, that they may have been influenced by previous advice (commonly given to obese patients) to restrict their water intake. Certainly the intakes were low. Our findings suggest that restriction of fluid intake is unlikely to have any effect in increasing the rate of weight loss.

Electrolyte balance

Table 3 sets out the basic values, and Table 4 shows the estimated changes in total body values for water, N, K and Na for each subject during the period of initial dehydration and during the period of secondary water retention. The figures for N are calculated from the data in Table 1 of Passmore *et al.* (1958) which make no allowance for losses through the skin. For N and K these are not likely to be important but skin losses of Na may well have been appreciable.

Table 3. *Electrolyte exchanges (m-equiv./day) of obese patients on a reducing regimen*

		Sodium				Potassium		
Subject	Days	Diet	Table salt	Urine	Faeces	Diet	Urine	Faeces
Mrs L.	1–7	6	48	57	2	37	36	9
	8–14	8	24	57	1	44	47	11
	15–21	15	19	15	2	37	37	14
	22–28	15	24	37	1	42	36	3
	29–35	26	24	29	1	40	35	3
	36–42	19	26	54	0	30	24	3
Miss McN.	1–7	—	—	62	3	26	40	5
	8–14	26	68	73	1	29	31	3
	15–21	32	68	54	1	34	33	5
	22–28	28	68	78	1	35	36	6
	29–35	40	68	54	1	34	35	5
	36–42	74	39	109	1	36	29	4
Miss B.	1–7	40	24	66	1	34	43	7
	8–14	30	19	31	1	31	35	8
	15–21	29	18	25	0	26	33	3
	22–28	20	17	24	0	28	32	2
	29–35	29	22	23	0	30	35	5
	36–42	31	24	52	0	32	40	5
Miss M.	1–5	14	—	47	1	20	47	3
	6–10	15	—	6	1	20	35	3
	11–15	14	—	3	1	20	24	3
	16–20	21	17	4	1	48	29	8
	21–25	27	17	25	3	35	32	8
	26–30	24	17	44	1	42	42	6
	31–35	25	17	22	6	42	28	7
	36–40	58	17	57	10	45	29	8
	41–45	35	17	81	11	42	35	8
Mr W.	1–5	22	14	54	1	48	50	11
	6–10	32	41	51	1	40	44	6
	11–15	37	34	48	3	44	26	10
	16–20	59	45	57	1	56	34	1
	21–25	23	45	80	5	42	28	6
	26–30	37	34	113	2	44	38	2
	31–35	26	38	53	3	40	27	3
	36–40	37	41	52	1	44	24	3

Table 4 shows that during the period of initial dehydration there were negative balances of N, K and Na. Actual negative Na balances must have been larger than recorded owing to neglect of losses through the skin. These findings are consistent with the view that both intracellular and extracellular fluid was being lost during this period.

Table 4. *Body changes in water, nitrogen, potassium and sodium of obese patients on a reducing regimen*

Subject	Days	H_2O (l.)	N (g)	K (m-equiv.)	Na (m-equiv.)
		First period: dehydration			
Mrs L.	1–14	−4·8	−67	−154	−217
Miss McN.	1–7	−2·1	−56	−133	—
Miss B.	1–7	−2·7	−41	−112	−21
Miss M.	1–10	−3·7	−55	−240	−130
Mr W.	1–5	−2·9	−47	−65	−95
		Second period: water retention			
Mrs L.	15–42	+1·1	−34	−42	+203
Miss McN.	8–42	0	−66	−133	+966
Miss B.	8–42	+1·4	−107	−357	+581
Miss M.	11–45	0	−55	+35	+180
Mr W.	6–40	0	−112	+290	+295

During the period of secondary water retention, N balances were negative and Na balances positive in all subjects. Owing to skin losses, the Na retention would not be as large as recorded. However, Miss McN. and Miss B. both showed considerable losses of K and gains of Na during this period. It is reasonable to conclude that these changes represented gains of extracellular water and losses of intracellular water, each of the order of 1–3 l. for the whole period of 35 days. With Mrs L. and Miss M., the electrolyte balances were small (after allowance for unrecorded Na losses through the skin). Presumably both intracellular and extracellular water remained approximately constant despite losses in N and fat (for data see Table 1, Passmore *et al.* 1958). There is no obvious explanation for the positive potassium balance of Mr W.

CONCLUSION

We have studied the variations in body water of obese patients on a reducing regimen. Changes in water intake appear to be unrelated to rates of loss of body water, and fluctuation in urine output by most patients can only account for an inconstant proportion of the changes in body water. Evaporative water losses were very variable; in one patient they were more closely related to changes in total body water than were the changes in urine output. The changes in evaporative water losses might be associated with disturbances in temperature regulation, and it is clearly important to study these losses under conditions in which environmental temperature can be adequately controlled.

SUMMARY

1. The water and electrolyte exchanges were recorded in five patients on a reducing regimen lasting over 40 days.

2. Losses and gains of total body water were not associated with changes in fluid intake. They could only be partly explained by variations in the urinary output. In one

patient, variations in evaporative water losses appeared to be important, and the skin may have a role in determining the water balance.

3. The electrolyte exchanges indicated that during initial periods of dehydration there were losses of both intracellular and extracellular water. In subsequent periods, water retention was found to be associated with increases of extracellular water, despite probable continuing losses of intracellular water.

We are indebted to Miss N. Stokoe, B.Sc. and Dr Helen Stojicevic for assistance with the chemical analyses. We wish to thank the Medical Research Council for a personal grant to one of us (F.J.R.) and also for an expense grant.

REFERENCES

Kekwick, A. & Pawan, G. L. S. (1956). *Lancet*, **271**, 155.

Newburgh, L. H. (1950). In *Clinical Nutrition*, p. 689. [N. Jolliffe, F. F. Tisdall and P. R. Cannon, editors.] New York: Paul B. Hoeber Inc., Medical Book Department of Harper Brothers.

Passmore, R., Strong, J. A. & Ritchie, F. J. (1958). *Brit. J. Nutr.* **12**, 113.

Strong, J. A., Passmore, R. & Ritchie, F. J. (1958). *Brit. J. Nutr.* **12**, 105.

Trapp, H. (1850). Beiträge zur Kenntniss der Veränderungen welche der Urin in Krankheiten erleidet. Thesis, Giessen. Quoted by Vogel, J. (1854). *Arch. Ver. gem. Arb. wiss. Heilk.* **1**, 96.