with residual fragments. The delayed techniques of percutaneous drainage with subsequent stone removal will be valuable for patients with a blocked cystic duct, so completing the repertoire of the operator but begging the question whether the gall bladder itself requires removal to avoid subsequent stone formation.

Over the past 36 years 23 publications have reviewed the results of open surgical cholecystolithotomy carried out on 2053 patients without recourse to cholecystectomy. The overall recurrence rate has been 34.6% (J Dempsey, paper in preparation) and the range 0-83% over follow up periods of 1-19 years. Further, patients treated by endoscopic sphincterotomy for stones in the common bile duct have had only a 14% incidence of cholecystectomy in follow up periods extending to five years.^{9,10} Thus cholecystectomy is not a definite requirement for patients with stones, and with the use of non-invasive imaging such as ultrasound the early reformation of the gall stone can be detected and eliminated by extracorporeal shockwave lithotripsy.

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Reduced concentrations of potassium, magnesium, and sodium-potassium pumps in human skeletal muscle during treatment with diuretics

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Abstract

Animal studies have shown that potassium depletion induced by diuretics or potassium deficient fodder leads to a selective decrease in the concentrations of potassium and in the concentration of sodium-potassium pumps in skeletal muscle. In 25 patients who had received diuretics for 2-14 years the mean concentrations of potassium, magnesium, and sodium-potassium pumps were measured in skeletal muscle biopsy specimens and were significantly lower than in those from a group of age matched controls. The reductions in all three variables were significant in those patients receiving diuretics for arterial hypertension as well as in those being treated for congestive heart failure. In 14 patients the mean muscle potassium concentration was below the control range, but only one of those was hypokalaemic (3.4 mmol/l), and 13 were receiving potassium supplements. In 15 patients the mean muscle magnesium concentration was below normal, and the mean muscle potassium and magnesium concentrations showed a linear correlation. In 12 patients in whom the mean muscle potassium concentration was below 80 µmol/g wet weight there was a linear correlation between the cellular potassium:sodium ratio and the concentration of ³H-ouabain binding sites indicating that potassium deficiency also leads to a down regulation of sodium-potassium pumps in human skeletal muscle.

In spite of potassium supplements long term treatment with

diuretics may lead to potassium and magnesium deficiencies, which are not detectable using the standard methods of serum analysis. The changes in concentrations of electrolytes and sodium-potassium pumps associated with treatment with diuretics may impair muscle function and potassium homeostasis and interfere with the distribution of digitalis glycosides.

Introduction

Thiazides and loop diuretics facilitate the loss of potassium and magnesium through the kidneys leading to deficiencies that may require treatment with supplements.¹⁻⁵ These losses are often overlooked, however, because plasma concentrations may remain normal even when the total body pools of potassium and magnesium are appreciably reduced. Animal studies have shown that deficiencies in potassium and magnesium are associated with a selective reduction in the concentration of potassium and in the concentration of sodium-potassium pumps in skeletal muscle.⁶⁻⁹ The concentration of sodium-potassium pumps determines the maximum capacity for the uptake of potassium by cells¹⁰ and plays an important part in the distribution of acutely administered digitalis glycosides.¹¹ The skeletal muscles are of particular importance because they contain the largest single pool of sodium-potassium pumps in the body.¹²

The present study was undertaken to assess the prevalence of magnesium and potassium deficiency among patients receiving long term treatment with diuretics and potassium supplements that were regarded as adequate for maintaining normal potassium homeostasis.

Subjects and methods

The study, which was approved by the local ethical committee, was performed in 25 consecutive patients undergoing outpatient treatment of hypertension or congestive heart failure at a department of internal medicine. There were 10 men and 15 women aged 44-83 years (median 67)

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who had been receiving diuretics for 2-14 years. These patients were compared with 25 controls who were not taking any drugs and who had been admitted to hospital for minor surgery. The controls comprised 12 men and 13 women aged 42-86 years (median 64).

Eleven patients received 1-2 standard doses of thiazides (median 1.5) a day and 14 were treated with loop diuretics in 1-6 standard doses a day (median 2.5). A standard dose of a thiazide was defined as 2.5 mg bendroflumazide or 25 mg hydroflumethiazide, and a standard dose of a loop diuretic was defined as 40 mg frusemide or 1 mg bumetanide. In 20 patients the diuretic was combined with a supplement of potassium chloride (mean dose 31 mmol a day, range 8-90 mmol). No magnesium supplements were given. The indication for treatment with diuretics was hypertension in 15 patients and congestive heart failure in 10. Five of the patients with congestive heart failure and two of those with arterial hypertension also received digoxin 62.5 µg 2-4 times a day. None of the patients received potassium sparing diuretics, and patients who had other diseases or conditions that could have led to potassium or magnesium deficiency were not included. Patients with thyroid disorders were excluded because the concentration of sodium-potassium pumps in muscle increases appreciably in proportion to the free thyroxine index.¹³ No patients had signs of impaired renal function; serum creatinine concentrations were below 120 µmol/l in all cases.

Samples of serum were withdrawn for measuring potassium, sodium, and magnesium concentrations. After local anaesthesia of the skin, subcutaneous tissue, and fascia by 1% lignocaine with noradrenaline biopsy specimens of the vastus lateralis muscle were taken 15 cm above the knee joint using a Bergström needle. One biopsy yielded three or four specimens of about 20 mg wet weight; these were immediately weighed, frozen, and stored in sealed plastic tubes until the electrolyte and sodium-potassium pump measurements could be made. Magnesium, sodium, and potassium concentrations were measured using trichloroacetic acid extracts of the specimens and atomic absorption or flame photometry.¹⁴ The concentration of sodium-potassium pumps was calculated by measuring the ³H-ouabain binding capacity using established procedures.^{13,15}

The methodological variation in measuring the concentrations of magnesium and potassium was determined by comparing the results of the analyses of multiple specimens obtained from 12 of the control subjects. The total variation from the mean concentrations comprises the variation between subjects and the variation within each subject. If two specimens taken at the same biopsy were used the variation within one subject for magnesium was 13%, and the variation between subjects was 87% of the total variation.¹⁴ With three samples the variation within one subject was reduced to 9%. For potassium the variation within one subject for two and three samples was 27% and 20% respectively. With two samples for magnesium the variation within one subject was no different from that obtained by subdividing one large sample of vastus lateralis into specimens of the same size as the standard biopsy specimens. This indicated that the variation within one subject reflected differences in the composition of the samples of tissue rather than errors arising during sampling, weighing, and homogenisation. Measurements of ³H-ouabain binding sites in specimens obtained from the same subject showed an average coefficient of variation of 11%.¹⁵

The significance of differences was assessed by the two tailed *t* test for groups of non-paired observations and by one way analysis of variance after ascertaining homogeneity of variance. Linear correlation analysis of unweighted values was performed by the method of least squares. Figures are given as means (SE) unless otherwise specified.

Results

Figure 1 shows the concentrations of potassium and magnesium in each individual's biopsy specimens obtained from the 25 patients receiving diuretics and the 25 controls. For each group the mean value is given and for the controls the 95% confidence intervals are indicated (1.96 × SD above and below the mean). Among the patients muscle potassium concentrations were below the control range in 14 cases. Thirteen of these patients were given potassium supplements, and in only one case was the serum potassium concentration slightly lower (3.4 mmol/l) than the control range (3.5-5.0 mmol/l). In 15 of the patients muscle magnesium concentrations were below the control range, but in only three of these were the serum magnesium concentrations subnormal (0.36, 0.44, and 0.68 mmol/l, respectively). Among the patients with muscle potassium and magnesium concentrations below the control range there was a slight preponderance of patients with congestive heart failure. Among the control subjects as well as among the patients there were no significant differences between men and women with respect to the concentrations of potassium, magnesium, sodium, and ³H-ouabain binding sites in muscle.

Table I shows the mean (SE) concentrations in samples of serum and muscle obtained from the patients and the control subjects. Whereas the

TABLE I—Serum and muscle concentrations of potassium, magnesium, sodium, and ³H-ouabain binding sites in 25 patients receiving diuretics and 25 control subjects. Figures are expressed as means (SE)

Variable	Patients receiving diuretics	Controls	p Value
Serum magnesium (mmol/l)	0.85 (0.04)	0.8 (0.01)*	NS
Serum potassium (mmol/l)	4.1 (0.1)	4.3 (0.1)	<0.05
Serum sodium (mmol/l)	142.0 (0.4)	142.0 (0.4)	NS
Muscle magnesium (µmol/g wet weight)	7.6 (0.2)	9.5 (0.2)	<0.001
Muscle potassium (µmol/g wet weight)	74.4 (2.4)	91.7 (1.5)	<0.001
Muscle sodium (µmol/g wet weight)	40.9 (1.4)	33.0 (1.4)	<0.001
³ H-ouabain binding sites (pmol/g wet weight)	206.0 (9.8)	251.0 (9.6)†	<0.005

*n=11; †n=18.

FIG 1—Concentrations of potassium and magnesium in biopsies of vastus lateralis muscle from 25 patients receiving diuretics and 25 age matched control subjects. Mean value for each group is indicated by a horizontal bar, as are 95% confidence intervals for control subjects (●). ○=Patients with hypertension; ●=those with congestive heart failure.

mean serum potassium concentration in the patients was only a few per cent lower than that in the controls, the mean muscle potassium concentration showed a 19% reduction ($p<0.001$). There were no significant differences between serum magnesium concentrations, but in the patients the mean muscle magnesium concentration was reduced by 20% ($p<0.001$). Whereas there were no significant differences between serum sodium concentrations, the mean sodium concentration in the muscle biopsy specimens was 24% higher among the patients ($p<0.001$). Assuming values for extracellular (15.7%) and total (75.6%) water space in the normal human vastus lateralis muscle,¹⁶ we calculated that the mean intracellular sodium concentrations in the specimens from the control subjects and the patients receiving diuretics were 17.8 and 31.0 mmol/l, respectively.

Table II shows the mean muscle concentrations of potassium and magnesium for the patients with arterial hypertension and those with congestive heart failure. In both diagnostic groups the mean concentrations of potassium and magnesium were significantly lower than in the controls. In the group with congestive heart failure the concentrations were slightly lower than in those with hypertension, but this difference was not significant ($p>0.05$).

Figure 2 shows the relation between potassium and magnesium con-

centrations in the individual biopsy specimens obtained from the patients receiving diuretics. The two measurements showed a highly significant correlation ($r=0.88$, $p<0.001$). For each of the two groups of patients the correlation was also significant ($p<0.001$). Regression analysis showed that there was a negative correlation between the potassium and sodium content in the biopsy specimens from each patient ($r=0.71$, $p<0.001$).

Tables I and II show the mean concentration of ^3H -ouabain binding sites in the muscle biopsy specimens obtained from the patients receiving diuretics, which was significantly lower than in the control subjects ($p<0.005$). The patients with congestive heart failure and hypertension

Discussion

The first objective of our study was to assess the prevalence of magnesium and potassium deficiency in a typical group of patients receiving long term treatment with diuretics. The second aim was to find out whether these disorders were associated with a reduction in the concentration of sodium-potassium pumps, as had been predicted by experiments in animals.

In the control subjects the measurements of muscle magnesium,

TABLE II—Comparison of muscle concentrations of potassium, magnesium, and ^3H -ouabain binding sites between patients receiving diuretics for hypertension and congestive heart failure and control subjects. Figures are expressed as means (SE)

Variable	Patients receiving diuretics		
	For hypertension (n=15)	For congestive heart failure (n=10)	Controls (n=25)
Muscle potassium ($\mu\text{mol/g}$ wet weight)	77.7 (2.4)***	69.4 (4.4)***	91.7 (1.5)
Muscle magnesium ($\mu\text{mol/g}$ wet weight)	8.0 (0.3)***	7.0 (0.4)***	9.5 (0.2)
^3H -ouabain binding sites in muscle (pmol/g wet weight)	207 (14)*	205 (14)**	251 (9.6) (n=18)

Controls v subgroups * $p<0.02$; ** $p<0.01$; *** $p<0.001$. Differences in measurements between the subgroups receiving diuretics were not significant.

showed almost the same reduction (table II). To assess whether this reduction was related to potassium loss, the ratio between cellular potassium and sodium concentrations was compared with the concentration of ^3H -ouabain binding sites in those patients in whom potassium deficiency was obvious—that is, those with muscle potassium concentrations below $80 \mu\text{mol/g}$ wet weight ($n=17$). Linear regression analysis showed a significant correlation between the cellular potassium:sodium ratio and the concentration of ^3H -ouabain binding sites for this group ($r=0.72$, $p<0.02$).

FIG 2—Relation between magnesium and potassium concentrations in biopsies of vastus lateralis muscle from 25 patients receiving diuretics. Each point represents mean of measurements performed on at least two specimens obtained from each patient. ○=Patients with hypertension; ●=those with congestive heart failure.

Because treatment with digoxin might interfere with the measurements of ^3H -ouabain binding sites and produce changes in the concentrations of sodium, potassium, and ^3H -ouabain binding sites in muscle, another regression analysis was performed after excluding the patients who had received digoxin. This showed a somewhat closer correlation ($r=0.72$, $p<0.01$) (fig 3). In these 12 patients the concentrations of potassium and ^3H -ouabain binding sites in muscle also showed a significant correlation ($r=0.69$, $p<0.02$).

FIG 3—Relation between cellular potassium:sodium ratio and concentration of ^3H -ouabain binding sites in 12 patients in whom muscle potassium concentrations were below $80 \mu\text{mol/g}$ wet weight and who were not treated with digitalis glycosides. ○=Patients with hypertension; ●=those with congestive heart failure.

potassium, and sodium concentrations were in the same range as previously reported.¹⁶⁻²⁰ The mean concentration of ^3H -ouabain binding sites of $251 (10) \text{ pmol/g}$ wet weight was not significantly different from that obtained in our previous measurements on normal subjects ($278 (15) \text{ pmol/g}$ wet weight).¹⁵

In the entire group of patients receiving diuretics the mean muscle magnesium concentration was significantly reduced, and in 15 of the patients the values were below the range determined in an age matched control group. Only three of these patients, however, had serum magnesium concentrations below the normal range. In 14 of the patients muscle potassium concentrations were below the normal range. In only two of the 25 patients did the serum potassium concentrations show slight reductions. The reduction of about 20% in muscle potassium and magnesium concentrations could conceivably be the result of increased extracellular volume. To account for the observed changes, however, extracellular water space would have to have increased by 73%. Earlier studies have shown that after prolonged treatment with diuretics the extracellular volume of muscle does not change significantly.²¹

In both diagnostic subgroups of patients muscle magnesium and potassium concentrations were significantly reduced (table II), indicating that the electrolyte changes were caused by the diuretics rather than by the illness that required the treatment with diuretics. We confirm earlier observations¹⁸ that patients with congestive heart failure had slightly lower concentrations, which may be related to the secondary hyperaldosteronism that often occurs in such patients, but the differences were not significant.

These observations confirm previous studies that have shown that in spite of normal serum concentrations the magnesium and potassium concentrations of muscle may be reduced.^{22,23} The fact that magnesium and potassium deficiencies are often overlooked emphasises the need for data on the composition of skeletal muscle. The present study has shown that a recently developed simple needle biopsy procedure¹⁴ permits the detection of disorders of electrolytes during long term treatment with diuretics despite normal serum concentrations. The normality of serum values is possibly due to other regulatory factors controlling cellular and renal magnesium-potassium exchange.

Magnesium favours the restoration of muscle potassium in patients who have developed a deficiency due to treatment with diuretics.^{2,3,24} The close correlation between muscle potassium and magnesium concentrations in our patients indicates that their potassium deficiency was related to or caused by magnesium deficiency. In experimental studies we have shown that after giving animals food that was deficient in magnesium muscle potassium concentrations were reduced.⁹ This was accompanied by a reduction in the concentration of sodium-potassium pumps, which was closely correlated with the reduction in the potassium concentration. Similar correlations have been shown in rats, mice, and guinea pigs that had been depleted of potassium by feeding with potassium deficient fodder or diuretics.²⁵ Among the 12 patients in whom the muscle potassium concentrations were below 80 $\mu\text{mol/g}$ wet weight and who did not receive digoxin muscle potassium and ³H-ouabain binding site concentrations were significantly correlated. This indicates that the reduction in sodium-potassium pump activity was related to the loss of potassium.

The down regulation of sodium-potassium pumps that we observed would reduce the maximum capacity of the cell to perform active sodium-potassium transport¹⁰ and favour a further net loss of potassium from the muscles. From the present data we estimate that in the muscle biopsy specimens from the patients receiving diuretics the mean intracellular potassium concentration was reduced by 18.9% and the mean intracellular sodium concentration was increased by 74%. The reduced potassium concentration and sodium-potassium pump concentration in muscle tissue during treatment with diuretics may impair muscle function and may explain the muscle fatigue often associated with this treatment.

Potassium and magnesium deficiency are both associated with increased sensitivity to digitalis.²⁶ This may in part be explained by raised plasma concentrations due to the reduction in the skeletal muscle pool of digitalis receptors. In animal experiments we showed that after potassium depletion leading to a 63% reduction in the concentration of ³H-ouabain binding sites in skeletal muscle, the

injection of ³H-ouabain was followed by a 77% greater immediate increase in ³H activity in plasma than in normokalaemic controls.¹¹ In four of our patients the concentration of ³H-ouabain binding sites was reduced by about 40%. It is possible, therefore, that in these subjects a loading dose of digitalis would result in higher plasma concentrations and toxic effects.

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ONE HUNDRED YEARS AGO

Mr. George Smith, of Coalville, giving his experiences of a number of visits made to gipsy encampments in all parts of the country, says he found that the leading traits in the character of many of the gipsies were: living in dirt, heathenish ignorance, idleness, lying, dishonesty, immorality, fortune-telling and deception. With some notable exceptions, these people huddled together in tents, regardless of any sense of decency or morality. He calculated that there were at the present time about 30,000 children in this country living in vans and tents, not 5 per cent. of whom could read or write. Bad as was the condition of the people here, it was, he said, even worse in Scotland, and he intended to include them in a Bill he was promoting for the

poor travelling children's welfare. What he wanted, without interfering with liberty, was, in the first place, that vans and other abodes of the kind should be registered annually in a simple, easy, and inexpensive way, so as to give the sanitary officer power, instead of the policeman, to see to the health and well-being of those who live in them. The healthy appearance of many of our gipsy, van, and other travellers was vanishing, and disease was carried from village to village. He desired to give them all free education. The only means of improving the condition of these people was the sanitary officer's influence upon their homes, and the schoolmaster's influence upon the children. (British Medical Journal 1888;i:148)