Studies by Darrow and his coworkers (1, 2) have shown that, when renal adjustment is reached in the presence of a deficit of sodium, chloride, or potassium, a correlation exists between the electrolyte compositions of plasma and skeletal muscle. The present authors (3) have been interested in the relationship between the electrolyte compositions of plasma and muscle which obtains in the presence of a deficiency of potassium. Rats with a deficit of potassium exhibit an elevated plasma bicarbonate content and a lowered plasma chloride concentration. These latter plasma changes are typical for the potassium-deficient rat and seem to be closely related to a potassium loss and a sodium gain in skeletal muscle.

Conway and Hingerty (4) induced a low potassium and high sodium content in muscle by feeding a potassium-deficient diet to rats, and then observed the restoration following dietary supplementation with potassium. Upon administering a diet rich in potassium, the level of skeletal muscle potassium was found to be rapidly restored, while the elevated muscle sodium returned to normal only after several days. From this observation, the latter authors concluded that mammalian muscle has the power of slowly extruding sodium from within the muscle fibers and that the extrusion of the sodium is a relatively slow process when compared with the rate of potassium entrance.

The finding of Conway and Hingerty (4) that potassium supplementation in potassium-deficient rats results in a rapid gain of muscle potassium and a slow loss of sodium raises the question of the existence of a similar situation during potassium depletion; that is, a rapid loss of muscle potassium and a slow gain of sodium. Previous studies have primarily been concerned with the changes encountered in animals with a pronounced potassium deficiency. Few or no data are available showing the successive changes in the electrolyte and water content of plasma and muscle during the course of potassium depletion and during the course of potassium restoration. The present experiments were therefore carried out with the primary object of providing such data.
Methods

Young adult male rats of the Wistar strain, of a uniform age and weight, were placed on a control diet for a period of at least 7 days. With the exception of differences specified later, the control and experimental diets had the same composition as those employed in a previous study (3). In general, the procedure of varying the dietary supply of sodium, chloride, and potassium entailed altering the salt mixture (5) of the diet with respect to its content of these elements. Potassium deficiency was induced in one group of rats by feeding a low potassium, normal sodium and chloride diet and, in a second group, by the daily injection of 2 mg. per day per rat of desoxycorticosterone acetate (DCA).1 Potassium was administered to certain animals after they had been maintained on the potassium-deficient diet for 5 weeks. In most instances this was accomplished by resuming the control diet or by feeding a diet containing twice the potassium content of the control diet. A few of the potassium-deficient animals received an intraperitoneal injection of potassium chloride (1 m.eq. of K per 100 gm. of body weight) and were sacrificed 4 hours later.

At the completion of an experimental period, the rats were placed under sodium pentobarbital anesthesia and arterial blood was collected under oil by direct puncture of the left ventricle. The gastrocnemius muscles were then removed. In order to provide adequate samples for the several analyses, equal aliquots of plasma, separated under oil (heparin served as anticoagulant) from two rats of the same control or the same experimental group, were combined under oil. The gastrocnemius muscles from the two rats were also combined and sampled. In tabulating the data, therefore, the designation of the number of animals employed in a given experiment represents the number of “pairs” of animal.

By methods previously described (3), the following determinations were made on the plasma: water, sodium, potassium, chloride, and total CO2; on the muscle, water, chloride, sodium, potassium, and total fat. The plasma bicarbonate was calculated from the figure for plasma total CO2 by the Henderson-Hasselbalch equation, assuming a constant pH of 7.4. The data obtained on muscle are presented in terms of 100 gm. of fat-free solids. No correction was made for the chloride, which apparently does not react like extracellular chloride (6) in calculating the water and mineral partition in muscle. The volumes of the extracellular (F) and intracellular (C) phases of muscle were calculated in the manner outlined by Hastings and Eichelberger (7) and on the basis of 1000 gm. of fat-free tissue. In order to approximate the volume changes produced in the extracellular and intracellular phases of 1000 gm. of original muscle, the

1 We are indebted to the Ciba Pharmaceutical Products, Inc., for the generous supply of “percorten” which was employed in these experiments.
calculations were made relative to the control series, assuming a constant solid content of the intracellular phase.

Results

The results of the analyses of plasma and muscle of rats subjected to different periods of potassium depletion are summarized in Table I, while the results of similar analyses of samples obtained from rats with potassium deficiency followed by potassium administration are presented in Table II. In Table III is presented a summary of the calculated muscle phase values.

Plasma and Muscle Changes during Potassium Loss—From the data presented in Table I, it will be noted that, during the course of potassium depletion induced by dietary potassium lack or by DCA administration, there occurred a progressive decrease of the plasma chloride concentration and an essentially equivalent rise of the plasma bicarbonate content. The plasma potassium concentration promptly decreased to a relatively constant low level. Simultaneous with the plasma electrolyte changes, the muscle potassium fell and the muscle sodium increased. During the early period of potassium depletion, there was a precipitous fall of the muscle potassium when compared with the further decrease which occurred later.
Thus, during the first 2 week period of feeding a low potassium diet, there was an average loss in muscle potassium of 8.0 m.eq. per 100 gm. of fat-free solids when compared with the average control value. During the succeeding 3 week period there was an additional average loss of only 4.2 m.eq. of potassium. Similarly, after the first 7 days of DCA administration, the average change of muscle potassium from the control level amounted to 8.7 m.eq. per 100 gm. of fat-free solids, compared with the further average decrease of 1.5 m.eq. which occurred during the second 7 day period. The muscle sodium appeared to increase at a fairly constant rate in those rats maintained on a potassium-deficient diet. Consequently, the ratio of muscle potassium loss to sodium gain in these animals was higher in the initial than in the later periods of dietary potassium lack. Upon DCA administration, on the other hand, the increment of muscle sodium increase was found to be significantly greater for the first 7 days than for the second 7 day experimental period. Thus, no precise relationship was obtained between the deficit of muscle potassium and the increase of sodium during potassium loss. With a pronounced deficiency there is not a complete replacement of muscle potassium by sodium and in the present experiments the ratio of potassium loss to sodium gain tended to approach 1.5.

Recorded in Table I are the average data from the analyses of plasma and muscle of rats maintained on a low potassium diet for 5 weeks and then transferred to a low potassium, low sodium, high chloride diet for 1 to 12 days. After 1 day on the latter high chloride diet, the average plasma chloride concentration was found to be 99.3 m.eq. and the plasma bicarbonate content to be 36.0 m.eq. per 1000 gm. of water. When compared with the corresponding concentrations found in the animals given a low potassium diet for 35 days, these figures indicate a prompt and definite tendency toward normal plasma chloride and bicarbonate levels. However, the latter levels apparently represented a maximum response, or perhaps an equilibrium situation, for the specific dietary mineral supply, since no further change toward normal was found even in animals sacrificed after consuming the diet for 12 days. Within the group of animals there were no significant differences in the levels of muscle potassium and sodium. The average potassium content of muscle of 33.6 m.eq. per 100 gm. of fat-free solids is almost identical with, while the average muscle sodium of 15.8 m.eq. is 1.6 m.eq. lower than, the corresponding average values observed in the animals given the low potassium diet for 5 weeks.

Changes in Plasma and Muscle during Potassium Restoration—From the average data presented in Table II, it will be seen that the resumption of the control diet by the potassium-depleted rats was promptly followed by a trend toward a normal electrolyte composition of both the plasma and muscle. However, complete restoration was not evidenced in these ani-
mals even after they had consumed the diet for 3 days. The results of
the observations on these animals differ in several respects from those
reported by Conway and Hingerty (4). The latter authors found that,
when potassium-depleted rats were placed on a potassium-rich diet, the
muscle potassium was completely restored in 24 hours, while a significant
decrease in the muscle sodium was found to occur only after 3 days. In
addition, their animals exhibited an elevated serum potassium concen-
tration following the resumption of the potassium-rich diet. In the

### Table II

Average Electrolyte Content of Plasma and Skeletal Muscle of Rats with Potassium Loss Followed by Potassium Supplementation

<table>
<thead>
<tr>
<th>Supplementation</th>
<th>No. of rats</th>
<th>Diet</th>
<th>Plasma per kilo water</th>
<th>Muscle per 100 gm. fat-free solids</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Na</td>
<td>K</td>
</tr>
<tr>
<td>Control diet</td>
<td>2</td>
<td>1</td>
<td>157.4</td>
<td>2.9</td>
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<tr>
<td></td>
<td>2</td>
<td>2</td>
<td>151.2</td>
<td>3.4</td>
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<td></td>
<td>2</td>
<td>3</td>
<td>156.1</td>
<td>3.8</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>6–12</td>
<td>154.3</td>
<td>4.2</td>
</tr>
<tr>
<td>High K and normal Na and Cl diet</td>
<td>5</td>
<td>1</td>
<td>155.9</td>
<td>4.3</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>2</td>
<td>151.9</td>
<td>3.8</td>
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<td>3</td>
<td>152.2</td>
<td>4.2</td>
</tr>
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<td></td>
<td>3</td>
<td>6–12</td>
<td>150.5</td>
<td>4.4</td>
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<tr>
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<td>3</td>
<td>2</td>
<td>149.9</td>
<td>3.1</td>
</tr>
<tr>
<td>Intraperitoneal injection of KCl†</td>
<td>2</td>
<td></td>
<td>150.3</td>
<td>3.3</td>
</tr>
</tbody>
</table>

*Potassium administered after the animals were maintained on a low potassium diet for 35 days.
†Sacrificed 4 hours after an intraperitoneal injection of 1 m.eq. of K per 100 gm., body weight, employing a 200 m.eq. per liter solution of KCl.

In the present study an elevated plasma potassium concentration was not ob-
served in any of the animals. Further, it will be noted that the resump-
tion of the control diet for 1 day resulted in an increase of 5.3 m.eq. of
muscle potassium per 100 gm. of fat-free solids and a loss of 1.8 m.eq. of
sodium, when compared with the average values encountered in the ani-
mals fed a low potassium diet for 35 days (Table I). Thus, the muscle
potassium of these 35 day potassium-depleted rats observed 1 day after
resuming the control diet was only partially restored. At the same time,
there was evidence that the gain of potassium was accompanied by a loss
of muscle sodium.

The results of the latter experiment prompted additional studies on
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potassium-depleted rats given access to a diet containing potassium in twice the amount present in the control diet. From the average data presented in Table II it is seen that, in the group of rats which had consumed the high potassium, normal sodium and chloride diet for 1 day, the muscle potassium was 43.4 m.eq. and the muscle sodium was 13.5 m.eq. per 100 gm. of fat-free solids. Compared with the corresponding values found in the group of animals receiving the low potassium diet for 35 days, this represented a 9.5 m.eq. increase in potassium and a 3.9 m.eq. fall in sodium. With an extension of the dietary period there was a further small increase in muscle potassium, but the average level found in the controls was not reached even in those animals which had consumed the high potassium diet for 6 to 12 days. Meanwhile there was a progressive fall of the muscle sodium toward the control level. An evident explanation for the fact that the muscle potassium in these rats did not attain the level found in the control series is not at hand. It might be mentioned, however, that Miller and Darrow (8) noted that a high dietary potassium intake in rats results in a muscle potassium concentration which falls in a low normal range.

The changes toward normal plasma chloride and bicarbonate concentrations during the 1st day or two following potassium supplementation were more pronounced with the high potassium diet than with the control diet. The more pronounced change toward normal plasma chloride and bicarbonate concentrations paralleled the more rapid restoration of the muscle composition.

As might be anticipated, the sodium and chloride content of the potassium-containing diet had an effect on the restoration of the plasma composition. The amount of chloride in the diet also seemed to affect the restoration of the muscle electrolyte content. Thus, when potassium-depleted rats were given a diet high in potassium but low in both sodium and chloride for 2 days, the average changes toward normal plasma and muscle compositions were less pronounced than those observed in animals similarly treated with a high potassium, normal sodium and chloride diet.

The increase in muscle potassium following dietary potassium supplementation in potassium-depleted rats was in each experiment accompanied by a fall of muscle sodium. It is of interest to note that a similar response was encountered when the potassium was administered by intraperitoneal injection. Thus, in the two potassium-depleted animals sacrificed 4 hours after the injection of KCl, there was an average gain in muscle potassium of 4.0 m.eq. and an average loss in sodium of 1.9 m.eq. per 100 gm. of fat-free solids when compared with the corresponding average values found in the animals fed the low potassium diet for 35 days.

Changes of Muscle Phases—The foregoing data indicate that during
conditions of potassium loss and with potassium restoration the exchange of potassium between the extracellular and intracellular phases of muscle

**Table III**

*Average Phase Volume Data of Skeletal Muscle of Rats during Potassium Loss and of Rats with Potassium Loss Followed by Potassium Administration*

The values are expressed in terms of 1000 gm. of fat-free muscle.

<table>
<thead>
<tr>
<th>Diet</th>
<th>No. of rats</th>
<th>Diet</th>
<th>(Na)†</th>
<th>(K)†</th>
<th>(H₂O)‡</th>
<th>(P)</th>
<th>ΔM‡</th>
<th>ΔC</th>
<th>ΔF</th>
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</thead>
<tbody>
<tr>
<td>Control</td>
<td>9</td>
<td>7-35</td>
<td>10</td>
<td>163</td>
<td>739</td>
<td>109</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low K</td>
<td>2</td>
<td>7</td>
<td>8</td>
<td>161</td>
<td>727</td>
<td>149</td>
<td>0</td>
<td>-39</td>
<td>39</td>
</tr>
<tr>
<td>Low K, followed by</td>
<td>2</td>
<td>14</td>
<td>14</td>
<td>146</td>
<td>725</td>
<td>151</td>
<td>0</td>
<td>-13</td>
<td>42</td>
</tr>
<tr>
<td>Control + DCA</td>
<td>2</td>
<td>21</td>
<td>17</td>
<td>143</td>
<td>735</td>
<td>153</td>
<td>3</td>
<td>-41</td>
<td>44</td>
</tr>
<tr>
<td>Low K, followed by</td>
<td>2</td>
<td>28</td>
<td>32</td>
<td>136</td>
<td>735</td>
<td>153</td>
<td>3</td>
<td>-41</td>
<td>44</td>
</tr>
<tr>
<td>Control, followed by</td>
<td>2</td>
<td>35</td>
<td>39</td>
<td>124</td>
<td>733</td>
<td>110</td>
<td>0</td>
<td>-8</td>
<td>-2</td>
</tr>
<tr>
<td>high Cl diet</td>
<td>2</td>
<td>7</td>
<td>18</td>
<td>157</td>
<td>738</td>
<td>101</td>
<td>-10</td>
<td>-1</td>
<td>9</td>
</tr>
<tr>
<td>Low K, followed by</td>
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<td>11</td>
<td>32</td>
<td>135</td>
<td>736</td>
<td>102</td>
<td>-10</td>
<td>-7</td>
<td>-9</td>
</tr>
<tr>
<td>control diet</td>
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<td>14</td>
<td>36</td>
<td>127</td>
<td>739</td>
<td>100</td>
<td>-5</td>
<td>5</td>
<td>-10</td>
</tr>
<tr>
<td>Low K, followed by</td>
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<td>1-12</td>
<td>37</td>
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<td>729</td>
<td>101</td>
<td>-45</td>
<td>-32</td>
<td>-13</td>
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<tr>
<td>high K and normal Na and Cl</td>
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<td>2</td>
<td>30</td>
<td>140</td>
<td>737</td>
<td>114</td>
<td>0</td>
<td>-5</td>
<td>5</td>
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<tr>
<td>diet</td>
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<td>2</td>
<td>29</td>
<td>132</td>
<td>738</td>
<td>104</td>
<td>0</td>
<td>-6</td>
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<td>737</td>
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<td>103</td>
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<tr>
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<td>107</td>
<td>2</td>
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<td>high K and low Cl diet</td>
<td>2</td>
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<td>14</td>
<td>159</td>
<td>739</td>
<td>103</td>
<td>-7</td>
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<tr>
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<td>6-12</td>
<td>11</td>
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<td>-15</td>
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<td>-10</td>
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<tr>
<td>intraperitoneal injection of KCl</td>
<td>2</td>
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<td>141</td>
<td>741</td>
<td>97</td>
<td>-4</td>
<td>9</td>
<td>-13</td>
</tr>
</tbody>
</table>

* Also see the foot-notes at the bottom of Tables I and II.
† Per 1000 gm. of intracellular water.
‡ Per 1000 gm. of intracellular phase.
§ Calculations of the changes of the muscle phases were made relative to the control series, assuming a constant solid content of the intracellular phase.

is quantitatively greater than that of sodium. This situation may possibly result in an altered distribution of muscle water. In Table III are presented the calculated changes of the muscle phases together with the intra-
cellular sodium and potassium contents expressed per kilo of intracellular water. From a perusal of the data it will be noted that conspicuous changes of the muscle phases were encountered in only two groups of animals. The first group consisted of those animals examined during the early periods of potassium depletion induced by feeding a low potassium diet. Here the average values of the extracellular phase (F) were significantly greater than the control figure. However, the changes in the bulk of 1 kilo of muscle were not large and represented an approximate balance between an increase of the extracellular phase and a decrease of the intracellular phase. The latter changes were less pronounced after the longer periods of feeding the low potassium diet. Thus, after 5 weeks, the change in the bulk of 1 kilo of muscle amounted to an average decrease of 10 gm. per kilo of control muscle and consisted of average decreases of 8 and 2 gm., respectively, of the intracellular and the extracellular phases. Changes from the control of this magnitude are probably not significant.

The second group represented animals which had been given the low potassium diet for 5 weeks and then shifted to a low potassium, low sodium, high chloride diet for 1 to 12 days. In these animals the changes in the bulk of 1 kilo of muscle amounted to an average decrease of 45 gm. per kilo of control muscle, consisting of an average 32 gm. decrease of the intracellular phase and an average 13 gm. decrease of the extracellular phase.

It was not possible to relate closely the partition of muscle water with the intracellular distribution of sodium and potassium. With potassium loss there was a deficit of the sum of the intracellular concentrations of sodium plus potassium, while the sum tended to be increased when the potassium-containing diets were resumed. However, with potassium restoration the loss of intracellular sodium was of such a magnitude that the sum of the intracellular concentrations of these two cations was not greatly in excess of the average normal level. It is of interest to note that the relative concentrations of intracellular sodium and potassium underwent marked changes without serious alterations in the distribution of muscle water. For example, an average intracellular sodium concentration of 39 m.eq. and an average intracellular potassium concentration of 124 m.eq. per 1000 gm. of water were found in animals given the low potassium diet for 5 weeks. In similarly potassium-depleted rats given a high potassium diet for 1 day, the intracellular sodium and potassium concentrations were found to be 25 m.eq. and 153 m.eq. per 1000 gm. of water, respectively. With the average potassium increase of 29 m.eq. and the sodium loss of 14 m.eq., the intracellular water increased from 733 to 739 gm. per 100 gm. of intracellular phase. However, the calculation of the muscle phases relative to the control revealed no unusually large changes in volume.
DISCUSSION

Work from several laboratories (3–5, 8, 9) indicates that when rats are placed on a low potassium diet the increase in muscle sodium does not completely compensate the loss of potassium, even if the dietary period is extended to over 100 days (5). The results of the present study demonstrated that the ratio of muscle potassium loss to sodium gain is significantly greater during the earlier than during the later periods of potassium depletion induced by dietary means. It is of interest to point out that the data of Conway and Hingerty (4) obtained on rats fed a low potassium diet reveal similar changes in muscle composition. Thus, in their study the changes of the mean values from the control levels in 4 days represented a loss of 13.7 m.eq. of muscle potassium and a gain of 2.6 m.eq. of sodium per 1000 gm. of tissue. The further changes of the mean values for the dietary period from 4 days through 26 days amounted to a loss of 11.4 m.eq. of potassium and a gain of 11.1 m.eq. of sodium per 1000 gm. of tissue. It would therefore appear that the change in the muscle potassium content accompanying dietary potassium restriction in rats simulates a two-phase process. That is, there is an initial rapid loss of potassium which is quantitatively greater than the gain of sodium and this is followed by a slower loss of potassium which tends to be compensated by an equivalent gain of sodium.

Somewhat similar changes in muscle potassium and sodium, but in a reverse direction, were encountered when potassium-deficient rats were transferred from the low potassium to potassium-containing diet. The data reported here differ in two outstanding respects from those observed by Conway and Hingerty (4), for which there is no evident explanation. The latter authors found that the transfer from a low potassium to a high potassium diet led to a high plasma potassium. Further, it was found that, although the muscle potassium was restored in 24 hours, it was only after 3 days that a significant decrease in muscle sodium was observed. In contrast, no increased plasma potassium levels were observed in the present study and in each experiment there was a tendency for the muscle sodium to return toward normal as the muscle potassium became increased. Conway and Hingerty (4) base their explanation of the process of restoration of normal muscle composition on the relative rates of passage of potassium and sodium ions to and from the muscle fibers and on the influence of the plasma potassium concentration on the rate of sodium ion transfer. The present data indicate that a greater rate of potassium than of sodium transfer between the extracellular and intracellular compartments of rat muscle is more conspicuous during the initial than during the later periods of potassium deficiency and of potassium restoration. This seems to raise the question of the existence of a fraction of intra-
cellular potassium which has a greater freedom of exchange between the two compartments of muscle.

Correlation between the electrolyte compositions of plasma and skeletal muscle, as first noted by Darrow and his coworkers (1, 2), was in evidence in rats during the induction of potassium deficiency and during potassium restoration. It would appear that the increased plasma bicarbonate content and reduced plasma chloride concentration found in potassium-deficient rats cannot be restored to normal by increasing the chloride intake without also providing potassium. In a similar way, muscle potassium restoration is retarded during dietary potassium administration unless chloride is also provided. The changes of the plasma and muscle electrolyte compositions encountered in rats rendered potassium-deficient seem to depend upon an altered renal excretion. A study of the renal excretion of bicarbonate, chloride, sodium, and potassium in potassium-deficient animals is now in progress.

SUMMARY

A study was made of the successive changes in the electrolyte and water content of the plasma and skeletal muscle of rats undergoing potassium depletion and of rats previously rendered potassium-deficient by feeding a low potassium diet upon realimentation with potassium. The various findings may be briefly summarized as follows.

1. During the course of potassium deficiency there were a progressive decrease of the chloride concentration in plasma and an almost equivalent increase of the bicarbonate content in plasma. Simultaneously, there were a loss of muscle potassium and a gain of muscle sodium.

2. The above changes were reversed when the animals previously rendered potassium-deficient were placed on a diet containing potassium and normal amounts of sodium and chloride, the initial changes toward normal plasma and muscle electrolyte composition being more rapid with a high than with a normal potassium diet.

3. When potassium deficiency was induced by dietary means, there was found to be an initial rapid loss of muscle potassium which was quantitatively greater than the gain of sodium. This was followed by a slower loss of muscle potassium which tended to be balanced by an equivalent gain of sodium.

4. After allowing potassium-deficient rats to resume the control diet, there followed a rapid increase of muscle potassium which was quantitatively greater than the loss of sodium. By increasing the potassium content of the diet to twice that present in the control diet, the changes toward normal muscle potassium and sodium contents were more pronounced, but complete restoration of the muscle composition was not
found even after the animals had consumed the diet for 3 days. The differ-
ences between these and the other results of the present study and those
found by Conway and Hingerty (4) were pointed out.

5. The total sodium plus potassium content of the intracellular phase
of muscle was lowered in potassium-deficient animals and tended to be
elevated with potassium restoration. Relative to the control muscle, an
expansion of the extracellular phase (F) and an equivalent contraction
of the intracellular phase (C) of muscle occurred in the early periods of
dietary potassium lack. On the whole, the partition of muscle water was
not found to be seriously disturbed and could not be closely related to
the intracellular content of sodium and potassium.

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