The Relation of Cardiovascular Phenomena to Metabolic Changes in a Patient with Chronic Hypokalemia

By Edward J. Huth, M.D., and Russell D. Squires, M.D.

Cardiovascular phenomena were studied and correlated with metabolic data in an adult male with chronic hypokalemia. The appearance and disappearance of postural hypotension, the changes in heart rate, the S-T segment shifts, and the U-wave amplitudes correlated well with the changing serum potassium level during experimental acute potassium depletion. QRS and T-wave changes could not be correlated with metabolic data. Comparison with the cardiovascular manifestations of experimental potassium depletion in normal subjects suggested that this patient had a chronic potassium depletion of at least 300 mEq.

Clinical interest in the effects of hypokalemia on the circulatory system has been focused mainly on changes in electrocardiographic patterns and on relationships to digitalis effects. For the most part, too, the interest has been aroused by observations on patients having acute hypokalemia. The present paper is concerned with cardiovascular phenomena observed during more than 4 years in a patient with chronic hypokalemia. These phenomena are examined in relation to the metabolic disturbances.

The clinical problem presented by the patient was recurrent muscle weakness that for over a decade had occurred in episodes lasting from 3 to 4 days, at first monthly, later as often as weekly. The relation of the muscle weakness to hypokalemia was established during the patient’s first visit to this hospital, 10 years after the onset of his illness. During the next 4 years, the period covered by this study, the serum potassium concentration did not rise above 3.0 mEq./L., despite potassium chloride medication, though the latter freed the patient almost entirely from the attacks of paralysis.

Case History

H. B., a man, was first seen by the authors when he was 38 years old. He had suffered since the age of 28 from recurrent muscle paralysis in the arms and legs. Each episode was heralded by dryness of the throat, lasted 3 to 4 days, and spontaneously remitted. The frequency of the episodes was monthly at first and later weekly. At the age of 14 or 15 the patient had passed “bloody” urine once, but at that time had felt otherwise well.

At the initial physical examination the recumbent blood pressure was 130/80. The head was tilted back and to the left in the characteristic position of torticollis. The right eye showed a superior oblique muscle palsy. The heart rate was 80/min. and the rhythm was normal. The heart size was normal. A soft grade 1 systolic murmur was heard in the pulmonary area where the second sound was louder than in the aortic area. The slightly enlarged liver was not tender.

The blood showed slight hemoconcentration and the bone marrow was hypercellular. The specific gravity of the urine was 1.004, with an alkaline reaction, 0 to +1 albumin and a few white and red cells and finely granular and hyaline casts in the sediment. The urea clearance was 38 per cent of normal and the phenolsulfonphthalein excretion was 36 per cent in 2 hours. Intravenous urography showed enlargement of both renal shadows, poorly outlined calyceal systems, and delayed dye excretion.

Blood chemical studies showed a serum potassium of 2.8 mEq./L., sodium of 136 mEq./L., CO₂ content of 30-33.4 mM/L., chloride of 89 and 91 mEq./L., blood urea nitrogen of 17 — 26 mg./100 ml., and serum creatinine of 1.9 mg./100 ml.
latter hereafter being referred to as “acute depletion.”

**Orthodiography.** Frequent fluoroscopic examinations with orthodiography showed a frontal cardiac area 30 per cent below the predicted normal and a normal silhouette of the heart. During a metabolic balance study in April 1952, diaphragmatic elevation and weakness were detected by fluoroscopy after 3½ days of acute reduction of potassium intake (fig. 1). The serum potassium concentration was then between 1.4 and 1.2 mEq./L. Muscle weakness in the limbs had already been present for 1½ days. The diaphragmatic level at the end of quiet expiration was higher than normal, and the excursion of the diaphragm was reduced. The next day, the acute depletion period was ended by the intravenous and oral administration of potassium chloride. Three hours later the patient felt much better, muscle power improved in the limbs, and the diaphragm returned to its normal level.

**Electrokymography.** Electrokymographic tracings of the movement of the patient’s lower left ventricular border were made.* The typical tracing in figure 2 was recorded in the chronic

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* Courtesy of Dr. Peter Kuo, Robinette Foundation, Hospital of the University of Pennsylvania.
hypokalemic state under potassium chloride therapy when the serum potassium was slightly over 2.0 mEq./L. The movement of the left ventricular border was so small that much higher amplification was necessary than usual. The notable abnormality in form was the diastolic plateau. During a period of sharp reduction of potassium intake, a curve resembling a sine wave of very low amplitude was recorded just before the patient fainted.

Heart Sounds. The patient's heart sounds were usually soft, particularly the first. Occasionally, a grade 1 or 2 blowing systolic murmur was heard at the apex and along the medial portions of the fourth and fifth left interspaces. During acute depletion of potassium, the sounds became louder and the systolic murmur increased to grade 3.

Postural Hypotension. The patient's blood pressure varied little from 125/85 during the 4 years of study. During the potassium deprivation study of April 1952, the postural response of the blood pressure was examined. The blood pressures at 1-minute intervals after 5 to 10 minutes rest in recumbency were usually within 5 mm. Hg of 125/85. In the standing position, the systolic pressure fell markedly. The amount of fall increased as the potassium depletion increased; with resumption of usual potassium intake, the fall diminished. Persistent falls in the diastolic pressure also occurred in the acute depletion period. The patient felt lightheaded during the postural hypotension, but did not faint. The postural changes in pressure correlate well in time with changes in serum potassium concentration and muscle strength (fig. 3).

Ballistocardiography. Ballistocardiographic tracings were normal in form. The amplitude of the I-J complex has been about 40 per cent below average for a subject of his age and sex, but within the normal range.

Consecutive tracings during metabolic study were made in October 1952, when the usual potassium intake was withdrawn for 2 days. The tracings were made before lunch and after the subject had rested on the Starr ballistocardiographic table for at least 10 minutes. Amplitude was expressed in Starr's term of "cardiac force" with a slight change in the Starr formula. * The tracings showed no changes in form. The amplitude showed a marked increase in the recovery period following the acute depletion period of 2 days (fig. 4).

Other Studies. The venous pressure and circulation times (Decholin, arm to tongue) were normal in both the control and acute depletion periods.

Electrocardiography. Limb and CR leads of electrocardiograms were recorded in the supine position once or twice a day throughout the study of April 1952, in the control, acute depletion, and recovery periods.

Heart rates taken from the electrocardiograms were resting values in that the tracings were taken under basal conditions in bed, be-

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* In this modification, "cardiac force" = \sqrt{\frac{2(I+J)}{n}} where (I + J) is the sum of the I + J wave amplitude (mm.) in all complexes of one or more respiratory cycles and n is the number of complexes measured.
fore breakfast. The basal heart rates rose during acute depletion of potassium and fell to control levels during the recovery period (fig. 5).

The QRS complex was unchanged in the acute depletion period, but in the early part of the recovery period, a "left axis shift" developed and the S wave deepened in the medial chest leads (fig. 6). The sum of the major downward deflection of the QRS complex in leads I and III increased and regressed with the "left axis shift" (fig. 5).

The usually depressed S-T segments were depressed further during acute potassium depletion and returned toward the control level in the recovery period (fig. 5).

In the patient's usual state, with the serum potassium about 2.0 mEq./L., the T waves were of low amplitude and either positive or diphasic and there was often a small negative U wave in the precordial leads (fig. 6). Late during acute depletion, the T wave developed a double positive peak with a slight increase in amplitude and the U wave deepened and broadened, reaching beyond the succeeding P wave. In the early part of the recovery period,

![Graph showing changes in serum potassium level and electrocardiographic variables](image)

Fig. 5. Changes in serum potassium level ([K]s), resting heart rates, and in magnitude of various electrocardiographic variables (in mv.) during a metabolic study of acute potassium depletion.

QRS represents sum of major downward deflections in the QRS complexes of leads I and III. S-T, T', T", and U represent levels of S-T junction and the amplitudes of the 2 T-wave components and of the U wave, relative to the Q junction in CR4.

![Retraced electrocardiograms](image)

Fig. 6. Retraced electrocardiograms from the limb leads and CR4 showing characteristic patterns in the patient's usual chronic hypokalemic state (control), at the height of acute potassium depletion, early in recovery, and after a large oral dose of KCl. In the 3 lower tracings from CR4, the full amplitude of R is not included.
the T wave became taller, while the deep negative U wave began to regress; by the end of the recovery period, both waves reverted to their previous form (fig. 7).

When a large oral dose of potassium chloride was given to raise the serum potassium to a higher than usual level, a deep negative T wave developed (fig. 8).

**Discussion**

The cardiovascular abnormalities appear to be attributable solely to the potassium depletion. In analysis, 2 problems can be considered: the mode of production of these changes by the depletion and how well the cardiovascular abnormalities indicate the severity of the depletion. To consider the first
problem, the abnormalities can be taken up individually.

Heart Sounds. Systolic murmurs in association with hypokalemia have been described but the complexity of the cases has obscured any direct relationship. Frenkel reported a case with severe hypokalemia in which “high output failure” with a rapid circulation time was a possible explanation for a systolic murmur. Our subject, H. B., however, showed no signs of failure and his circulation time was normal. His systolic murmur and its increase in loudness during acute depletion remain unexplained.

The Electrokymogram. The notable abnormality in the electrokymogram was the diastolic plateau (fig. 4). This feature has been recorded previously from hearts with marked bradycardia (as in complete heart block) where left ventricular filling is completed relatively early in a long diastole and from hearts with constrictive pericarditis where an external restraint to left ventricular expansion exists.

No evidence of pericardial disease could be demonstrated in our patient and the tracing in figure 4 was recorded at a heart rate of 68/min. Considered speculatively, the limitation of the outward movement of the left ventricular wall early in diastole may arise from an abnormality in the elastic properties of the myocardium itself. The first possible abnormality is diffuse and microscopic scarring following focal necrosis, a known consequence of chronic potassium depletion. A second possible abnormality is a functional resistance of the myocardium to distention beyond a certain point. Pertinent in this regard are the decrease in diastolic size of the frog ventricular muscle as potassium and sodium are lost and the increased diastolic tone of frog heart in Ringer’s solution with a low concentration of potassium.

The Ballistocardiograph. The cause of the increased ballistocardiographic amplitude in the recovery period is unknown. Attempts to correlate ballistic amplitude with ionic and fluid shifts were fruitless. In the only previous report of increase in ballistic amplitude in association with major ionic and fluid shifts, Jones found maximal amplitude during diureses in cardiac patients, instead of after the major fluid and ionic shifts as found in this study.

Postural Hypotension. Postural hypotension was reported by Earle and co-workers in association with chronic hypokalemia. In our patient the blood pressure was usually normal. The postural hypotension developed and regressed with the fall and return of the serum potassium level and with the appearance and disappearance of muscle weakness in the limbs (fig. 3). Three mechanisms might account for the postural hypotension:

1. Reduced muscle tone. The muscle weakness in the legs might lower tissue pressure against the leg veins and lead to pooling of blood in the legs during standing.

2. Reduced peripheral vascular reactivity. Rats kept on a diet deficient in potassium develop hypotension in close relation to the serum potassium concentration. This effect may be related to a lessened response of peripheral vessels to pressor substances. In a patient with marked hypokalemia during recovery from diabetic acidosis “high-output failure” was ascribed to decreased peripheral resistance. The postural hypotension developed by our patient might be attributable in part to a failure of vasoconstriction in leg vessels in response to standing.

3. During the entire period of acute potassium depletion, the patient was in a state of mild hyperventilation, manifest by tachypnea and a rise in pH and fall in CO₂ content of the arterialized cutaneous blood. It is suggested that hyperventilation, arising from anxiety due to the increasing muscle weakness or stimulated by intracellular acidosis in the respiratory center, could contribute to the postural fall in pressure.

Neither the serum sodium level nor the calculated blood volume changes correlated satisfactorily with the postural blood pressure changes.

Electrocardiography. During the 4-year study of patient H. B., with serum potassium level between 1.8 and 2.8 mEq./L., the tracings always showed S-T segment depression and T-U wave alterations (fig. 6).

The moderate bradycardia of 50 to 55 beats/
min. during the control period of the April 1952 metabolic study (fig. 5) is consistent with the previous findings of bradycardia during chronic potassium depletion in young rats and chicks and in human beings, though it may simply be a normal rate for this patient. The more rapid rates in the acute depletion period (fig. 5) have a parallel in the rising rates in dogs during the development of acute total body potassium deficit (by extracorporeal dialysis), and of acute severe hypokalemia in human beings.

The changes in the QRS complex, the "left axis shift," were similar to those observed by Weller in dogs that underwent dialysis. The time sequence differed in that these changes in our patient came at the beginning of the recovery period when the serum potassium had begun to rise even though estimated intracellular repletion had not begun, whereas in the dogs of Weller the changes developed during the withdrawal of potassium from the intracellular compartment while the serum level was constant. The potassium concentration changes accompanying the QRS alteration in both the patient and the dogs would be expected to lead to a rise in the ratio of extracellular-intracellular myocardial concentrations of potassium; such a change in ratio may be a common denominator in both observations. It must be stated that minor changes in heart position as the tracings were taken from the patient in the supine position may have been responsible for the QRS changes. The time sequence of the T-wave changes closely paralleled that of the QRS changes (fig. 5).

The changes in the level of the S-T segment (or more precisely, the S junction) in CR₄ relative to the Q junction were found to correlate equally well with the serum potassium level and the cumulative potassium balance (table 1). From these correlations it is not possible to pick out a certain causal basis for the S-T segment shifts. In the narrow range of heart rates observed, 53–74, the S-T segment shifts cannot be attributed reasonably to rate changes. The findings of Weller in dogs that the S-T segment depressions, developing during depletion of potassium, persisted when the serum level of potassium was raised rapidly by infusion of potassium, suggested that the S-T shift might be closely related to a continuing myocardial depletion of intracellular potassium. A similar situation was present in patient H. B., in that the S-T segment level did not return completely to control levels during the recovery period (fig. 5). In this period the cumulative negative balance of intracellular potassium persisted (−300 to 400 mEq.) even though the serum potassium level returned to control levels (table 1).*

With more investigation of this problem, the

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* The failure of intracellular repletion of potassium in the recovery period when the serum potassium level returned to control level accounted for the poor correlation between the 2 variables. The partial return of the S-T segment toward control levels accounts for its satisfactory correlation with both metabolic variables.

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<table>
<thead>
<tr>
<th>TABLE 1.—Correlation of Electrocardiographic and Metabolic Data: Regression Equations, Correlation Coefficients (r), and Significance (p).</th>
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</thead>
<tbody>
<tr>
<td><strong>Resting heart rate:</strong></td>
</tr>
<tr>
<td>With serum potassium level:</td>
</tr>
<tr>
<td>Rate = 88 – 16 [K]ₙ</td>
</tr>
<tr>
<td>With arterial cutaneous blood CO₂:</td>
</tr>
<tr>
<td>Rate = 171 – 5.6 [CO₂]ₙABC</td>
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<tr>
<td><strong>S-T segment level (CR₄):</strong></td>
</tr>
<tr>
<td>With serum potassium level:</td>
</tr>
<tr>
<td>S-T segment level = 0.214 [K]ₙ – 0.80</td>
</tr>
<tr>
<td>With cumulative K balance:</td>
</tr>
<tr>
<td>With S-T segment level = 0.00042 (Cum. K Bal.) – 0.34</td>
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<tr>
<td><strong>U-Wave amplitude (CR₄):</strong></td>
</tr>
<tr>
<td>With serum potassium level:</td>
</tr>
<tr>
<td>U amplitude = 0.85 [K]ₙ – 1.96</td>
</tr>
<tr>
<td><strong>Cumulative potassium balance:</strong></td>
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<tr>
<td>With serum potassium level:</td>
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<table>
<thead>
<tr>
<th>r</th>
<th>p Value</th>
</tr>
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<tbody>
<tr>
<td>-0.83</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>-0.91</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>+0.76</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>+0.79</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>+0.83</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>+0.47</td>
<td>&lt;0.2</td>
</tr>
</tbody>
</table>

Note:
- [K]ₙ = serum potassium level in mEq./L.
- [CO₂]ₙABC = arterialized cutaneous blood CO₂ content in mM/L.
- S-T segment level = depression of S junction below Q junction in mv.
- Cumulative K balance is in total mEq. of external balance. U-wave amplitude is measured relative to the Q junction, in mv.

[Diagram or image of QRS complex and metabolic data correlation equations provided here]
S-T segment depression may be found to be a useful guide to the magnitude of cellular potassium depletion.

In the preceding discussion the repolarization waves were arbitrarily named T and U waves. Other possibilities in nomenclature for these waves were considered: 1. The first 2 waves forming a double positive peak as T waves, and the following negative wave as U wave. 2. The first positive peak as T, the second positive peak as U, and the negative wave as V wave. 3. The first positive peak as T wave of the atrium (with markedly prolonged P-Ta interval), the second positive peak as T wave of the ventricle, and the negative wave as U wave. Evidence for the last 2 possibilities was insufficient, so the first conventional nomenclature was used. On this basis, measurements of the interval from Q to either apex of T showed a prolongation as judged by the criteria of Surawicz and Lepeschkin. This prolongation occurred without the presence of hypocalcemia.

The T waves did not show the expected further lowering and inversion during the acute depletion study but became more positive early in the recovery period (fig. 7). This sequence is not so unusual if the deeply inverted T wave present at higher serum potassium levels (fig. 8), possibly reflecting myocardial tissue damage, represents the patient’s "normal" T wave. If so, the positive T waves after acute depletion may be simply following a general rule of change of polarity with acute potassium depletion. Additional evidence for this view was found in the tracings of a potassium-depleted patient studied by McNally. No explanation could be devised for the delay in the appearance of the major part of the T wave change until the early recovery period; it may be pertinent that Schwartz found that pattern changes in potassium depletion often appeared to lag behind changes in the total balance of potassium.

It is readily seen (fig. 5) that the T waves of this patient did not correlate with changes in the serum level of potassium, as the behavior of the T wave during the acute loading of potassium further emphasizes. Figure 8 shows that when the potassium serum level was 2.06 mEq./L. and rising, the pattern was identical to that seen when the serum level was 2.28 mEq./L. and falling. Therefore the T-wave pattern in this patient must be related to an aspect of electrolyte distribution in the heart not reflected by balance data or serum potassium level; nevertheless, attempts to provide evidence for this hypothesis by calculating estimated intracellular:extracellular ratios for potassium during the T-wave changes were fruitless.

The amplitude of the negative U waves (CR4) showed a good correlation with the serum potassium level (fig. 5, table 1). The change in amplitude of U waves paralleled the changes in the S-T segment level (CR4) and did not parallel the T-wave changes (fig. 5).

As has been mentioned, attempts to relate electrocardiographic variables to calculated total body intracellular concentrations of sodium and potassium and intracellular and extracellular concentration ratios have been unrewarding. In view of ignorance as to how closely total body external and internal balances of sodium and potassium can be relied on to reflect changes in the intracellular concentrations of these ions in the myocardium, this failure is not surprising.

The remaining problem is whether it is possible to estimate the magnitude of the chronic potassium depletion in this patient from the cardiovascular phenomena. The number of studies relating the development of cardiovascular disturbances to the degree of potassium depletion is small, if only those giving satisfactory quantitative information are considered.

Black and Milne depleted 2 normal subjects of potassium with a low potassium diet. In 1 subject with a total depletion of 280 mEq. K⁺ and a minimal serum potassium level of 3.2 mEq./L, no electrocardiographic changes were seen. A second subject developed a U wave in lead II with a potassium depletion of 270 mEq. K and a serum level of 3.0 mEq./L. The subject of Blahd and Bassett reached a potassium deficit of 270 mEq. and a minimal serum potassium level of 3.0 mEq./L in 55 days on a low potassium diet; he showed no changes in
Table 2.—Relation of Size of Potassium Depletion in Normal Experimental Subjects to Electrocardiographic Changes

<table>
<thead>
<tr>
<th>Size of K depletion</th>
<th>Type of electrocardiogram</th>
<th>Changes attributable to K depletion</th>
<th>No Changes attributable to K depletion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>&lt;100 mEq.</td>
<td></td>
<td>15</td>
<td>30</td>
</tr>
<tr>
<td>100-200 mEq.</td>
<td></td>
<td>20</td>
<td>30</td>
</tr>
<tr>
<td>200-300 mEq.</td>
<td></td>
<td>15</td>
<td>30</td>
</tr>
<tr>
<td>&gt;300 mEq.</td>
<td></td>
<td>50</td>
<td>10</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

Size of K depletion determined by balance studies. Changes attributable to potassium depletion include characteristic changes in S-T segment level, and in T and U-wave contour and amplitude. Data from 10 to 30 subjects have been used for size of samples and percentages rounded to nearest 5 per cent.

blood pressure or in electrocardiographic tracings. Three of 4 normal medical students on low potassium diets developed total potassium depletions of 116 to 195 mEq. over 7 to 8 days, with minimal serum potassium levels of 3.5 mEq./L. None of these 3 showed changes in blood pressure or in electrocardiographic tracings. The fourth developed small increases in the amplitude of a normal positive U wave during a depletion of 300-400 mEq. with a serum level of 3.1-2.8 mEq./L. Schwartz and associates studied the relation between electrocardiographic changes and the magnitude of potassium depletion in subjects in whom potassium loss was induced by adrenocortical hormones, acidifying salts, and excessive use of laxatives. They could not find a distinct correlation between the negative potassium balance and the changes in the electrocardiograms that were characteristic of potassium depletion. Fourman induced potassium depletion in 2 normal subjects by feeding an ion-exchange resin. Despite the production of extracellular acidosis by the resin and the tendency of metabolic acidosis to mask the electrocardiographic patterns of potassium depletion, electrocardiographic tracings with characteristics attributable to potassium depletion were found in 2 experiments on 1 subject with depletions of 338 and 839 mEq. In studies by Womersley and Darragh potassium depletions of 330, 266, and 162 mEq. were reached by normal subjects with low potassium diets without the development of electrocardiographic changes.

The data from these studies have been arranged in Table 2 to show the relationships between electrocardiographic tracings of the hypokalemic type and the magnitude of the potassium depletion. They suggest that patient, H. B., showing chronically a hypokalemic-type tracing, has a 65 per cent chance of having a chronic potassium depletion of at least 200 mEq., and a 50 per cent chance of a depletion greater than 300 mEq. When the additional depletion of 374 mEq. attained acutely in the April 1952 study is added to the estimate, the depletion accompanying the appearance of the postural hypotension may have reached a minimum of 600 to 700 mEq.

Summary

A male adult with intermittent muscular paralysis was found to have a chronic hypokalemia due to failure of renal conservation of potassium.

Diaphragmatic elevation and diminished excursion were observed fluoroscopically during an acute potassium depletion study.

The motion of the left ventricular border as recorded in the electrokymograph showed low amplitude and a diastolic plateau, possibly due to patchy fibrosis in the myocardium as a consequence of chronic potassium depletion, or a functional change in the elastic properties of the myocardium.

A mild postural hypotension developed during acute reduction of the potassium intake, possibly caused by reduction in leg muscle tone, reduced peripheral vascular resistance, and a background of hyperventilation.

Increases in ballistocardiographic wave amplitude were found during recovery from an acute potassium depletion study.

The changes in resting heart rate, S-T segment level, and U wave amplitude correlated closely with changes in the serum potassium level. The S-T segment level also correlated
well with the cumulative potassium balance, even though the correlation between the cumulative potassium balance and the serum potassium level was not good.

The time sequence of development and regression of a “left axis shift” in the QRS complex paralleled changes in amplitude of the T wave. These changes could not be correlated with metabolic variables.

Comparison of the cardiovascular findings in this patient with those in normal subjects depleted of potassium in several ways suggests that the patient had a minimal chronic potassium depletion of 300 mEq. and reached a depletion of at least 700 mEq. during a marked reduction in his potassium intake.

ACKNOWLEDGMENT

We wish to thank Dr. J. Russell Elkinton for his generous support during this study. He, Drs. A. G. Hills, C. F. Kay, I. Starr, C. C. Wolfert, F. C. Wood, and Miss Elizabeth Trotter kindly gave us thorough and useful criticism of the manuscript.

SUMMARIO IN INTERLINGUA

Esseva constatate, in un macho adulte con intermittente paralyse muscular, le presentia de chronic hypokalemia debite a non-conservation renal de kalium.

Elevation diaphragmatic e excursion diminuita esseva observate fluoroscopicalemente durante un studio de acute depletion de kalium.

Le motion del margine sinistro-ventricular, registrate in le electrokymographo, monstrava un basse amplitude e un plateau diastolic, possibilmente debite a placas de fibrosis in le myocardio (disveloppamento in consequentia de chronic depletion de kalium) o a un alteration functional del qualitates elastic del myocardio.

Se disveloppava un leve hypotension postural durante acute reduction del ingestion de kalium, possibilmente causate per reduction del tono muscular del gambas, reduceite resistentia periphero-vascular, e un fundo de hyperventilation.

Augmentos del amplitude in le undas ballistocardiographic esseva constatate durante le recovramento ab un studio de acute depletion de kalium.

Le alterationes del frequentia cardiac in stato de reposo, del nivello del segmento S-T, e del amplitude del unda U esseva nettemente correlationate con alterationes in le nivello del kalium seral. Le nivello del segmento S-T monstrevat etiam un bon correlation con le balancia de kalium cumulative, ben que le correlation del balancia de kalium cumulative con le nivello de kalium seral non esseva bon.

Le sequentia temporal del disveloppamento e del regression de un “displaciamiento sinistro-axial” in le complexo QRS esseva parallel a alterationes de amplitude in le unda T. Iste alterationes non esseva correlationabile con variabiles metabolic.

Le comparation del constatationes cardiovascular in iste paciente con correspondentie constatationes in subjectos normal con depletion de kalium effectuate in varie manieras pare indicar que le paciente habeva un minimal depletion chronic de kalium de 300 mEq e que ille attingeva un depletion de 700 mEq o plus durante un marcate reduction del ingestion de kalium.

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40 Schwartz, W. B., Levine, H. D., and Relman,


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Medical Eponyms

By Robert W. Buck, M.D.

Adams-Stokes Disease. Robert Adams (1791–1875), one of the surgeons to Jervis Street Infirmary, Dublin, described “Cases of Diseases of the Heart, Accompanied with Pathological Observations” in the Dublin Hospital Reports 4: 353–453, Dublin, 1827. On page 391 appears the following:

“February 20, 1822, I was called to visit a gentleman in my neighborhood, aged fifty years, who had suddenly fallen down, as reported to me, in an apoplectic fit. I found him in a state of complete insensibility; his face (naturally pale and sickly) was now red and bloatted; his breathing stertorous, with a slow pulse.... In the last year he had two apoplectic attacks, exactly resembling that which I had just witnessed: from these he recovered without any paralysis of the muscles....

“An officer in the revenue, aged sixty-eight years, of a full habit of body, had for a long time been incapable of any exertion, as he was subject to oppression of his breathing and continued cough. In May 1819.... I saw this gentleman: he was just then recovering from the effects of an apoplectic attack, which had suddenly seized him three days before.... What most attracted my attention was, the irregularity of his breathing, and remarkable slowness of the pulse, which generally ranged at the rate of 30 in a minute.... [During seven years he had been seen] in not less than twenty apoplectic attacks.... He would then fall down in a state of complete insensibility.... His pulse would become even slower than usual.... He recovered from these attacks without any paralysis.... In both these cases... apoplexy must be considered less a disease in itself than symptomatic of one, the organic seat of which was in the heart.”

William Stokes (1804–1878) in his article “Observations on Some Cases of Permanently Slow Pulse” which appeared in the Dublin Quarterly Journal of Medical Science 2: 73–85 (August 1), 1846, refers to these case histories as follows:

“In the fourth volume of the Dublin Hospital Reports, Mr. Adams has recorded a case of permanently slow pulse, in which the patient suffered from repeated cerebral attacks of an apoplectic nature, though not followed by paralysis. The attention of subsequent writers on diseases of the heart, has not been sufficiently directed to this case, which is an example of a very curious and, as there is reason to believe, special combination of symptoms.”
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