A Clinical Correlative Study of the Electrocardiogram in Electrolyte Imbalance

By L. S. Dreifus, M.D. and A. Pick, M.D.

There is no general agreement concerning the action of specific electrolyte disorders and acid-base balance upon the electrocardiogram nor concerning their interrelationships in this respect. In order to test the validity and limitations of the electrocardiographic diagnosis of such disorders in clinical practice, 133 records on 48 patients with suspected or established electrolyte imbalance were compared with chemical determinations, in part serial. Some difficulties encountered in the electrocardiographic diagnosis of mixed electrolyte disorders, and in the distinction of electrolyte from drug effects, are illustrated.

In electrolyte imbalance the failure of the electrocardiogram to correlate with clinical and laboratory findings may result in both diagnostic and therapeutic dilemmas. Several authors1-4 in particular have stressed the lack of conformity of electrocardiographic manifestations with the serum levels of potassium. This disparity has been attributed to the presence of electrocardiographic alterations from other causes or to concomitant abnormalities of other electrolytes. For example, Roberts and Magida5,6 studied the influence of the acid-base balance experimentally and concluded that electrocardiographic changes suggesting hyperkalemia and hypokalemia could be induced respectively by acidosis and alkalosis, and that such alterations were dependent upon the pH rather than the actual serum potassium concentration. Tarail,7 on the other hand, was unable to establish any correlation between serum pH, sodium, or calcium with the intensity of potassium effects reflected in clinical electrocardiograms.

Attempts have also been made to clarify the apparent lack of correlation between the electrocardiogram and the actual serum electrolyte concentrations by determining their intracellular/extracellular relationships. Crismon and associates8 could not relate the intracellular potassium concentration to typical electrocardiographic events in hyperkalemia. Bellet, too, could not establish a definite relationship of the electrocardiographic changes to skeletal muscle potassium content.9 He considered, among other possibilities, that differences in the metabolic activity of heart and skeletal muscle were the cause of this lack of correlation. His comparison of intracellular to extracellular potassium concentrations with the electrocardiogram gave no better results than serum potassium concentration alone. Kühns10 on the other hand, noted such a relationship in animal experiments. In relating intracellular/extracellular potassium concentrations he used a value termed “cardiac potassium quotient” with a normal range of 25 to 32. Reduction of this quotient was associated with typical hyperkalemic changes, and elevation with typical hypokalemic changes in the electrocardiogram. There was, however, no consistency between the electrocardiographic alterations and absolute potassium or sodium concentrations, intracellular or extracellular.

Changes in the autonomic nerve tone and pharmacologic actions of epinephrine, insulin, veratrine, and in particular quinidine, have been noted to simulate electrocardiographic patterns of hypokalemia.9,11-13 It is also well known that pre-existent or concomitant alterations of the ST-T segment caused by heart
strain, intraventricular block, digitalis medication, coronary disease, hypotension, and hypoxia may prevent, modify, or simulate changes induced by electrolyte imbalance.9, 14-16

In view of this multiplicity of factors acting upon the electrocardiogram in conjunction with, or in addition to, the effects of specific electrolyte derangements, an objective study in a diversified hospital population appeared worthwhile in order to gain some idea of the practical value and the limitations of electrocardiographic diagnosis of electrolyte imbalance. In this study special attention was directed to cases in which a correlation could not be made and to an analysis of the factors that conceivably may have contributed to the discrepancy between the chemical and electrocardiographic findings. Serial electrocardiograms were compared with serial electrolyte determinations in order to gain information concerning the relative diagnostic value of the 2 methods in clinical practice.

Material and Methods

One hundred and thirty-three electrocardiograms and corresponding electrolyte determinations obtained in 46 patients were analyzed. The criteria for inclusion in the study were one or more of the following: (1) The electrocardiogram suggested a disturbance in potassium or calcium metabolism, (2) the laboratory data revealed abnormal potassium or calcium blood levels, and (3) electrolyte imbalance was suspected on clinical grounds. The variety of clinical states included is exemplified in the data presented in the legends to figures 1 to 4.

The electrocardiograms were read by one of us without knowledge of the chemical or clinical findings and tabulated according to the suspected electrolyte derangement and the degree of alterations. Afterwards, the serum potassium, sodium, calcium, sodium/potassium ratio and, when possible,

Fig. 1. Examples of gradations of electrocardiographic alterations attributed to potassium depletion A and potassium intoxication B. Segment B2 is lead I, all others are lead II. Segments A1 and B1 are from infants. A1. Abdominal carcinoma with massive intestinal hemorrhage. On potassium therapy, K = 4.2 mEq./L., grade +. A2. Anorexia nervosa, K = 2.7 mEq./L., grade ++. A3. Severe intractable diarrhea and diabetes insipidus due to carcinoma of the pancreas with metastasis to the hypophysis (demonstrated at autopsy), K = 1.7 mEq./L., grade ++++. A4. Dehydration on potassium therapy, K = 4.0 mEq./L., grade +. B1. Chronic glomerulonephritis, K (several days before this tracing) = 9.0 mEq./L., grade +. B2. Polycystic kidneys, uremia, K undetermined, grade ++. B3. Malignant hypertension, K = 7.38 mEq./L., grade +++. B4. Dehydration, K = 6.72 mEq./L., grade +.

Fig. 2. The sequence of electrocardiographic events (lead II) during an attack of familial periodic paralysis. No potassium determinations were made at this time, but the serum concentration fell to 1.8 mEq./L during another attack. A. 7:30 p.m., control; B. 2:30 a.m., 2 hours after high carbohydrate meal, paralysis almost complete, only slight movement of right forearm; C. 3:15 a.m., complete paralysis of extremities; D. 4:35 a.m., 8 minutes after a teaspoon of KCl orally, neck muscles involved, difficulties in coughing; E. 4:45 a.m., slight movements of head; F. 5:10 a.m., 10 minutes after another teaspoon of KCl, slight movements of fingers and toes; G. 5:25 a.m., movements of all extremities; H. 9:30 a.m., complete recovery.
pH were compared with the electrocardiographic findings. This correlation was also made with respect to changes developing subsequently in serial electrocardiograms and laboratory determinations.

Criteria for the electrocardiographic diagnosis of specific electrolyte disorders were based on numerous exhaustive descriptions in the literature. For the determination of the Q-T interval relative to the cycle length, the table of Heggin and Holzmuller was used, and accordingly deviations of more than ±0.04 sec. from the predicted values were considered abnormal. A full account is not given of the varieties of patterns encountered, their sequential development, and preferential manifestation in specific leads. Representative examples of hypokalemic and hyperkalemic alterations, and their gradation as used in this study are illustrated in figure 1, segments 1 to 3. Figure 2 demonstrates acute development of less common features of potassium depletion and their rapid restitution in consecutive records. Diagnostic problems were encountered particularly in infant electrocardiograms, in some cases with pre-existing abnormalities, in simultaneous disorders of the potassium and calcium balance, and in the distinction of hypokalemic alterations from those caused by quinidine medication. Examples of cases in which the electrocardiographic diagnosis was verified, or, on the other hand, proved to be partially or completely incorrect, are shown in figure 1 (segment 4), and figures 3 and 4.

The serum sodium, potassium, and calcium were determined by flame photometry. The serum pH was measured with a Beckman pH meter and microelectrodes and corrected to 38 C. The normal ranges in this study were considered to be: sodium, 137-145 mEq./L; potassium, 3.8-5.5 mEq./L; calcium, 9-11 mg. per cent (4.5-5.5 mEq./L); pH, 7.38-7.47; Na/K ratio, 24-38. Where possible, the serum calcium value was corrected for the serum protein concentration, with a factor of 0.87 for each gram of protein above or below 7.0 Gm. Both corrected and uncorrected calcium determinations were applied in correlations with the alterations of the Q-T duration.

The distribution of the electrocardiographic correlation within the range of serum potassium concentrations encountered in our material and the
**Results**

**Hypokalemia.** There were 35 electrocardiograms (table 1) that suggested hypokalemia, and in 22 of them the serum potassium was actually depressed. In 19 of the 35 instances the Na/K ratio was greater than 38, indicating relative hypokalemia. Thus, the Na/K ratio showed no better correlative value than the actual potassium concentration. The serum pH was in the alkalotic range in 4 of the 13 instances with a normal potassium concentration, but it was within the normal range in an equal number with hypokalemia. Hence, alkalosis per se could not be considered responsible for the electrocardiographic alterations.

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<tr>
<th>Table 1.—Serum Electrolyte Values in Electrocardiograms Suggesting Hypokalemia</th>
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<tbody>
<tr>
<td>No. of Ecgs</td>
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<tr>
<td>-------------</td>
</tr>
<tr>
<td>35</td>
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<table>
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<th>Table 2.—Electrocardiographic Manifestation of Established Hypokalemia</th>
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<tr>
<td>No. of instances with K &lt; 3.8 mEq./L.</td>
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<td>---------------------------------------</td>
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<tr>
<td>35</td>
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On the other hand, in 35 instances in which the serum potassium was lowered, examination of the electrocardiogram (table 2) showed agreement in 26. Here again, the serum Na/K ratio and pH value gave no better correlation than the actual serum potassium concentration.

Follow-up studies were possible in 20 cases with electrocardiographic signs of hypokalemia (table 5), of which 16 (80 per cent) showed a correlation of the electrocardiogram with the serum potassium concentration at one time or another. In 2 instances the electrocardiographic abnormality preceded the fall of serum potassium and in 1 case typical hypokalemic changes persisted after the serum potassium had returned to the normal range. One of the 4 patients, in whom the electrocardiographic diagnosis could not be confirmed, was in congestive heart failure due to rheumatic heart disease with atrial fibrillation and was on digitalis and quinidine medication. In 2 of the other cases a severe disorder of the electrolyte pattern was found, though not of the hypokalemic type. (One of them, with malignant hypertension, had a severe depression of serum sodium to 102 mEq./L.; the other, a diabetic with dehydration, hyperglycemia, and hypernatremia of 165 mEq./L., presented on the whole a hyperosmolarity syndrome.) The fourth patient was hospitalized because of neurasthenia and muscle weakness, and his electrocardiogram became normal after potassium administration; unfortunately, however, electrolyte determinations were not made at this time.

The diagrams in figures 5 and 6 reveal complete agreement between the electrocardiogram...
and the serum potassium when the concentration of the latter was less than 2.3 mEq./L. Furthermore, in this low range, the severity of electrocardiographic changes shifted pari passu with the actual serum potassium concentrations. This was not so true in the group with moderately low serum potassium levels (2.3 to 3.0 mEq./L.) and in the borderline group (3.1 to 3.7 mEq./L.), both as regards to the frequency of noncorrelation and the magnitude of electrocardiographic alterations.

**Hyperkalemia.** There were 27 instances (table 3) in which the electrocardiogram suggested the presence of hyperkalemia, and the serum potassium was actually elevated in 22 of them. In 21 of these 27 instances the serum Na/K ratio was less than 24, indicating a relative hyperkalemia. Thus, the Na/K ratio was of no greater value in the correlation than the actual serum potassium values. The serum pH was in the acidotic range in 3 of the 5 instances in which the electrocardiogram did not agree with the serum potassium. Since, however, there were other instances with pH in the normal or even alkalotic range in which the potassium determinations were in keeping with the electrocardiographic alterations, the latter could not be ascribed to the acidosis per se.

On the other hand, in 39 instances with elevation of the serum potassium, examination of the electrocardiogram (table 4) showed agreement in 21. Again, the Na/K ratio and the serum pH gave no better correlation than the actual serum potassium concentration.

Follow-up studies were possible in 18 patients with electrocardiographic evidence of hyperkalemia (table 5), and agreement with the serum potassium could be established in 15 (83 per cent). In 2 of these patients the electrocardiogram had anticipated the potassium elevation, while in 2 others the electrocardiographic alterations persisted after the serum potassium concentration had returned to the normal range. Among the 3 patients in whom no such agreement could be established, 2 had chronic glomerulonephritis and were dying in uremia. The third had been receiving potassium medication at the time the electrocardiogram suggested hyperkalemia.

The diagrams in figures 5 and 6 reveal complete agreement between the electrocardiogram and the serum potassium concentration in the highest range group (serum potassium above 6.7 mEq./L.) with the severest electrocardiographic changes mostly occurring in this range.

**Hypocalcemia (and Hypercalcemia).** There were 16 instances in which the electrocardiogram suggested the presence of hypocalcemia (table 6) and in 15 (94 per cent) the serum calcium was actually less than 9 mg. per cent. In 8 of these 16 cases it was possible to correct the calcium values with respect to associated abnormalities of the protein concentration, and in 5, after this refinement, the corrected serum calcium concentration was still less than 9 mg. per cent.

On the other hand, there were 59 instances in which the serum calcium concentration was less than 9 mg. per cent (13 instances in the serum protein-corrected group) (table 7). The electrocardiogram suggested the presence of hypocalcemia in only 15 of these cases (26 per
Table 6.—Laboratory Data in Electrocardiograms Suggesting Hypocalemia

<table>
<thead>
<tr>
<th>No. of electrocardiograms</th>
<th>Ca &lt; 9.0 mg. %</th>
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<tbody>
<tr>
<td>Total.....................</td>
<td>16</td>
</tr>
<tr>
<td>Corrected for serum protein concentration</td>
<td>8</td>
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</table>

<table>
<thead>
<tr>
<th>Ca &lt; 9.0 mg. %</th>
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<tbody>
<tr>
<td>15 (94%)</td>
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<td>5 (63%)</td>
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Table 7.—Electrocardiographic Manifestation of Established Hypocalemia

<table>
<thead>
<tr>
<th>No. of instances with Ca &lt; 9.0 mg. %</th>
<th>Positive electrocardiograms</th>
</tr>
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<tbody>
<tr>
<td>Total.....................</td>
<td>59</td>
</tr>
<tr>
<td>Corrected for serum protein concentration</td>
<td>13</td>
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</tbody>
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<table>
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<tr>
<th>Positive electrocardiograms</th>
<th>15 (26%)</th>
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In 7 instances hyperkalemia alone was considered in the electrocardiographic interpretations, and in 2 other instances hypocalemia alone.

Discussion

On the basis of the present study it would appear that it is easier to diagnose an isolated disorder of the potassium or calcium metabolism in the electrocardiogram than their combination. Figures 5 and 6 reveal, within the ranges of blood potassium levels represented in this series, an excellent correlation in both the lowest and highest ranges (below 2.3 and above 6.7 mEq./L, respectively). Furthermore, the characteristic electrocardiographic alterations were most pronounced in these range groups, which is in agreement with the observations of others. Instances in which such a correlation could not be established were distributed equally within the ranges of 5.6–6.7 and 2.3–3.7 mEq./L. In the normal range (3.8–5.5 mEq./L), suggestive electrocardiographic alterations were found only in a small percentage. The degree of electrocardiographic abnormalities found in the borderline ranges did not necessarily reflect the extent of the electrolyte disorder as determined in the laboratory. Thus, in the quantitation of the electrolyte derangement, the electrocardiogram cannot be considered a reliable guide, an experience we share with other workers in the field.

Whereas the association of hypocalemia with hypokalemia proved difficult to recognize, its combination with hyperkalemia was frequently noted in the electrocardiogram. However, even when the diagnosis of the latter combination of electrolyte derangement was missed, at least one or the other of the electrolyte shifts was usually suspected (fig. 4). Surawicz and Lepeschkin and Sjöstrand have dealt with the problem of detecting hypocalemia in the presence of hypokalemia and vice versa. An excellent correlation is generally assumed between the electrocardiogram and serum calcium concentrations below 8 mg. per cent. The effects of potassium and calcium are independent of each other and both ions must be administered before the electrocardiogram reverts to normal. In 94 per cent of our cases, in which the electrocardiogram suggested hypocalemia, the serum calcium concentration was less than 9 mg. per cent. But when all instances
with calcium below this range were matched with their electrocardiographic manifestation, a prolonged Q-T interval was found in only 26 per cent. This lack of correlation persisted even in the presence of serum calcium concentrations of 5 mg. per cent or less, and even after correction was allowed for the associated serum protein content. A similar discrepancy between electrocardiographic contour and serum calcium concentrations can also be found in the data presented by others.21, 22 Determinations of the ionized calcium fraction, which may provide a clue to this problem, were not possible in the present study.

From our data it became clearly evident that, in a correlation of serial electrocardiograms with the broad electrolyte spectrum (including pH and the Na/K ratio), the actual serum potassium concentration correlated best with the electrocardiographic manifestations of electrolyte derangement. Mechanisms by which the electrocardiogram may change under such circumstances have been brought closer to solution by recent studies on cell membrane potentials under the influence of varying electrolyte concentrations of the extracellular perfusion fluid.26 The mechanisms of similar electrocardiographic alterations induced by epinephrine, veratrine, and quinidine are not well understood. Judging from the electrocardiographic pattern per se, one might suspect that these agents affect the potassium gradient at the cell surface, without any reflection in the serum potassium concentration. In our experience, only the clinical history proved helpful in distinguishing such drug-induced electrocardiographic manifestations from those caused by electrolyte derangement.

Although we tried to correlate serum electrolytes with the electrocardiogram, we are fully aware that the 2 do not measure the same parameters of ionic balance. A determination of merely the serum electrolyte concentrations cannot represent more than a part in the complex system of intracellular-extracellular relationships. Moreover, Joseph, Engle, and Catchpole27, 28 have recently presented evidence that the ground substance may function as a cation-exchange resin with respect to calcium, sodium, and potassium. Thus, another compartment between intracellular and ordinary extracellular spaces may be operative in the ionic exchange system to induce electrocardiographic alterations in the absence of measurable serum electrolyte changes.

The experimental work of several groups of investigators6, 6, 29, 30 implies that changes in the serum pH may produce electrocardiographic alterations suggesting potassium derangement in the face of normal serum potassium concentrations. Our data did not permit definite conclusions in this regard. Although it was true that alkalosis was usually associated with hypokalemia, and acidosis with hyperkalemia, no consistency was found in the type and the severity of the electrocardiographic changes with regard to abnormal pH values. At best the latter approximated the correlation accuracy of the serum potassium concentrations with the electrocardiogram.

Some controversy in the literature in relating pH alterations to the intracellular/extracellular potassium (Kᵢ/Kₑ) ratios seems to have resulted from the fact that acidosis and alkalosis have been produced experimentally under various and not always comparable conditions.31 In our opinion, changes in the ionic equilibrium resulting from respiratory acidosis or alkalosis may have consequences distinct from those engendered by the production of a metabolic acidosis or alkalosis. Also, insufficient attention has been paid to the differentiation of electrolyte and acid-base disturbances induced in intact and depleted electrolyte states, and in particular in nephrectomized and non-nephrectomized preparations.

From the information at hand it appears that the production of alkalosis causes a shift of the potassium into the cell and a decrease in the Kₑ/Kᵢ ratio while acidosis acts in the opposite direction, and that these alterations will persist as long as the pH remains abnormal. In nephrectomized animals or in the presence of renal disease with acidosis, small increases in the serum potassium concentration may quickly alter the Kₑ/Kᵢ ratio and thus produce early and typical changes of hyperkalemia.
Contrariwise, in the presence of alkalosis with intact renal regulatory mechanisms a disturbance of the $K_a/K_i$ ratio may not occur until there is a marked depletion of body potassium, and hence the electrocardiographic alterations can be expected to be delayed. Thus, it is conceivable that the effects of the electrolyte derangement on the electrocardiogram may depend to a significant degree on the presence or absence of renal regulation of the serum electrolytes.

In our material the electrocardiogram anticipated chemical electrolyte disorders in a number of cases, suggesting that the electrocardiogram may reflect changes in the $K_a/K_i$ gradient before actual derangements of the electrolyte balance become evident in their absolute serum concentrations. Our data, in conformity with those of Schwartz and associates do not permit definite conclusion whether an abnormal serum potassium value without concomitant electrocardiographic changes is of greater clinical importance than electrocardiographic findings suggesting an abnormal potassium metabolism in the absence of demonstrable chemical changes. We can only state that once the electrocardiogram per se suggested hyperkalemia or hypokalemia, the latter became evident in the majority of cases in subsequent serial determinations. When serum potassium and the electrocardiogram were in accord, there was of course no doubt about the electrolyte imbalance.

We had relatively little difficulty in diagnosing hypokalemia and hyperkalemia in the face of pre-existing or concomitant electrocardiographic alterations although the typical contour changes were modified to some extent (fig. 3). Sharpey-Schafer showed that T inversion due to heart strain, in contrast to that caused by myocardial infarction, can transiently be abolished by oral administration of potassium chloride, and Langendorf and Pirani were the first to point out that the T-wave configuration in spontaneous hyperkalemia and uremia is the result of a composite effect of several factors acting in opposite direction upon its amplitude and configuration. The combination of S-T elevation caused by uremic pericarditis with hyperkalemic T-wave contour, as illustrated in figure 3, was recognized previously and its reversibility by dialysis demonstrated recently. Whereas the modification of typical strain patterns by hyperkalemia are well known, no definite criteria have been established for the electrocardiographic diagnosis of the not infrequent occurrence of hypokalemic changes in the presence of left heart strain (fig. 3).

It has been shown experimentally that histologic changes consisting in necrosis, cellular infiltration, and fibrosis can be produced in both skeletal and heart muscle by feeding rats potassium-deficient diets. In the heart these lesions are initially localized in the subendocardial layers but may later become diffuse; they may be aggravated by sodium feeding. Necrosis and loss of myofibrils have been attributed to a coagulative action of increased intracellular sodium in the presence of a low potassium or to an intracellular ionic imbalance causing clumping of contractile material and destruction of the adenosine triphosphate-phosphopyruvate system. has correlated histologic, laboratory, and electrocardiographic alterations produced by hypokalemia in man, while Levine and associates have recently demonstrated that injury currents produced by hyperkalemia in clinical electrocardiograms may result in patterns indistinguishable from those caused by recent myocardial infarction. From all this it would appear that ST-T changes occurring in the presence of hypokalemia and hyperkalemia may have an organic counterpart. Hence, electrolyte imbalance requires immediate correction in order to prevent extensive and perhaps permanent myocardial lesions.

Any study dealing with comparison of electrocardiographic and chemical alterations in the presence of electrolyte imbalance should take into account the known effects of advanced changes of ionic concentrations on cardiac excitability and conductivity. In our material, and within the stated ranges of abnormal serum concentrations of potassium and calcium, disturbances of the cardiac rhythm attributable to the electrolyte im-
balance did not play a significant role and hence their consideration was omitted in the present report.

**Summary and Conclusions**

An objective comparison in a diversified hospital population with suspected or proved electrolyte imbalance of serum potassium, calcium pH, and sodium/potassium ratios with electrocardiographic findings revealed that, in the presence of abnormal potassium balance, the electrocardiogram correlated best with the actual serum potassium concentrations. Hypocalcemia was almost invariably present when suggested by the electrocardiogram, but in only 24 per cent of all cases with depressed serum calcium was this reflected in the electrocardiogram. Correction for concomitant abnormalities of the serum protein concentration did not lead to a better correlation of the electrocardiogram with the serum calcium concentration.

Full agreement of electrocardiographic and laboratory findings was noted in both the highest (>6.7 mEq./L.) and lowest (<2.3 mEq./L.) serum potassium ranges observed in this study. Failures of the electrocardiogram to reflect abnormal serum potassium levels were equally distributed in the borderline and in the moderately severe range groups. No absolute range could be established for calcium concentrations at which the electrocardiogram can be expected to be diagnostic.

In 15 of 18 cases of hyperkalemia and in 16 of 20 cases of hypokalemia, agreement of electrocardiographic with the chemical laboratory findings could be established after serial studies. In a number of cases the electrocardiogram anticipated the electrolyte derangement. However, no conclusion could be reached as to the relative diagnostic importance of the electrocardiogram and the chemical determinations in a given case.

Possibilities and difficulties in using the electrocardiogram to diagnose specific electrolyte derangements in their most common combinations, or in the presence of pre-existent or concomitant electrocardiographic abnormalities of other etiology, are illustrated and discussed.

Possible physiologic bases for the lack of conformity of the electrocardiogram with abnormal serum electrolyte concentrations are considered.

**Acknowledgment**

We are indebted to Dr. C. Cohn, Director of the Department of Biochemistry, for his aid and his permission to use his data in this study, and to Drs. L. N. Katz and J. Stamler for their critical remarks.

**Summario in Interlingua**

In un diversificate population hospitalari con suspicite o demonstrate imbalancia electrolytico, un comparation objective del kalium, del pH de calcium, e del proportion de natrium e kalium in le sero con datos electrocardiographic revelava que in le presentia de un anormal balancia de kalium le electrocardiogramma eseva correlationate le melio con le concentrationes de kalium seral. Hypokalemia eseva trovate de facto in quasi omne casos in illo eseva sugerite per le electrocardiogramma, sed inter le casos con depression del calcium seral, solmente 24 pro cento reflecteva iste facto in le electrocardiogramma. Correctiones pro simultane anormalitates del concentration de proteina seral non resultava in un meliorate correlation del electrocardiogramma con le concentration seral de calcium.

Accordos complete del constatationes electrocardiographic e laboratorial eseva notate in le areas maximal e minimal del concentrationes seral de kalium observate in iste studio (i.e. in le areas del valores de plus que 6,7 mEq/L e de minus que 2,3 mEq/L). Le constatation que le electrocardiogramma non reflecteva anormal nivellos seral de kalium eseva equalmente frequente in le areas marginal e in le areas del valores de severitate moderate. Il non eseva possibile establir un area absolute de valores del concentration de calcium ubi on pote expectar que le electrocardiogramma se monstra diagnostic.

In 15 ex 18 casos de hyperkalemia e in 16 ex 20 casos de hypokalemia, accordo del constata-
tiones electrocardiographic con le constata-
tiones chimic de laboratorio esseva establibile
post studios serial. In un numero de cases le
electrocardiogramma reflecteva le disturba-
tion electrolytic ante illo esseva chimicamente
manifeste. Tamen, nulle conclusion poteva
esser derive in re le importantia diagnostic
of the electrocardiogramma e del determinaciones
chimic in le cases individual.

Es illustrate e discutite le possibilitates e le
difficultates de usar le electrocardiogramma in
le diagnose del disturbiones electrolytic in
lor plus commun combinationes o in le presentia
de pre-existente o simultanea anormalitates
electrocardiographic de altere etiologia.

Es considerate possibile bases physiologic
pro le non-conformitate del electrocardiogramma
con anormal concentrationes seral del
electrolytos.

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Medical Eponyms

By Robert W. Buck, M.D.


“A polyglandular syndrome hitherto supposed to be of corticoadrenal origin characterized in its full-blown state by acute peltic adiposity, by genital dystrophy, by osteoporosis, by vascular hypertension, and so on, has been found at autopsy in six out of eight instances to be associated with a pituitary adenoma which in the three most carefully studied cases has been definitely shown to be composed of basophilic elements, the lesion in one instance having been clinically predicted before its postmortem verification. . . .

"While there is every reason to concede . . . that a disorder of somewhat similar aspect may occur in association with pineal, with gonadal, or with adrenal tumors, the fact, that the peculiar polyglandular syndrome, which pains have been taken herein conservatively to describe, may accompany a basophil adenoma in the absence of any apparent alteration in the adrenal cortex other than a possible secondary hyperplasia, will give pathologists reason in the future more carefully to scrutinize the anterior-pituitary for lesions of similar composition."

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