Dialyzable Currents of Injury in Potassium Intoxication Resembling Acute Myocardial Infarction or Pericarditis

By Harold D. Levine, M.D., Sidney H. Wanzer, M.D. and John P. Merrill, M.D.

A byproduct of experience with the artificial kidney was the detection of reversible electrocardiographic "currents of injury" in patients with electrolyte imbalance. These changes occurred irrespective of anatomic alterations and resembled the changes of acute pericarditis or myocardial infarction. The electrolyte basis for the "current of injury" was established by the prompt abolition of the RS-T segment elevations, when the electrolyte imbalance was corrected by means of artificial hemodialysis.

A REGULAR electrocardiographic feature of moderately advanced potassium intoxication is depression of the RS-T segments. The erroneous inference of myocardial infarction is generally avoided because of the direction of the RS-T shifts, the clinical setting in which this phenomenon is recorded and its association with other electrocardiographic abnormalities distinctive of potassium intoxication. In a few exceptional instances of potassium intoxication, however, elevation rather than depression of the RS-T segments may be produced. The resultant "currents of injury" may seem much more suggestive of acute myocardial infarction or pericarditis than potassium intoxication. In four such cases recently observed at the Peter Bent Brigham Hospital these changes could be wholly or partially eliminated by dialysis with the artificial kidney. Because of the practical and theoretical implications of this phenomenon these experiences are presented in some detail.

Case 1. J. C., a 40 year old electrician with acute renal failure due to carbon tetrachloride poisoning, was transferred to the Peter Bent Brigham Hospital after his electrocardiograms (fig. 1 A) had shown tall, peaked T waves characteristic of potassium intoxication. He gave no history of chest pain or compression. On examination the heart rate was 150, the cervical veins were distended, coarse bubbling rales were heard throughout the lung fields, a gallop rhythm was heard at the cardiac apex and the liver edge was felt two fingerbreadths below the right costal margin. Tracings recorded on the afternoon of admission showed sinus tachycardia (fig. 1 B), with left bundle branch block. The RS-T segments in leads V₁, V₅ and lead aVR showed rather pronounced elevation, probably more than could be accounted for purely on the basis of left bundle branch block. Later in the afternoon the heart rate was 108 and the cardiac rhythm irregular and disturbed by runs of probable paroxysmal ventricular tachycardia. Over the right precordium the RS-T segment elevation was now more pronounced and associated with deep broad Q waves (fig. 1 C). The persistence of these changes was regarded as strong evidence for a "current of injury" and, it was believed, could not be attributed to left bundle branch block. At 6:40 p.m. hemodialysis was begun. At this time the serum potassium level was 7.8 mEq. per liter, sodium 127 mEq. per liter and the carbon dioxide content 9 mM per liter. Because the patient had marked pulmonary edema, the concentration of sodium in the bath fluid of the artificial kidney was maintained at 127 in order to avoid sodium loading in the presence of heart failure. The bath potassium concentration was initially set at 2 mEq. per liter but following the development of arrhythmia, was raised to 5. Two hours after the beginning of dialysis when the serum potassium level had fallen to 2.6 mEq. per liter and the sodium level had remained at 128 mEq. per liter, the electrocardiogram showed striking improvement. A supraventricular tachycardia (ventricular rate 210) had developed, the QRS complex had sharpened and shortened and the RS-T segment shifts over the right ventricle had almost disappeared (fig. 1 D). Though the patient improved temporarily after the dialysis...
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Fig. 1. Potassium intoxication simulating acute myocardial infarction; partial elimination of electrocardiographic changes by artificial dialysis. J. C. (case 1), a 40 year old man with acute renal shutdown. (A). Initial tracings characteristic of early potassium intoxication. (B). Tracings recorded on day of admission showing supraventricular tachycardia and left bundle branch block. (C). Tracings before dialysis show changes very suggestive of acute antero-septal myocardial infarct blended with those of moderately advanced potassium intoxication. (D). Almost complete recession of these changes after dialysis. (E). Partial return of changes on following day. Autopsy showed massive pulmonary embolism, moderate coronary sclerosis, thrombophlebitis and hypoxic nephrosis.

and even exhibited a moderate diuresis, on the following day he showed electrocardiographic (fig. 1 E), and on the succeeding day chemical (serum potassium 7.3 mEq. per liter) evidence of redeveloping potassium intoxication. On the afternoon of the third hospital day he suddenly sat up, complained of pain in the front of the chest, fell back gasping for air and died shortly thereafter. Postmortem examination showed massive pulmonary embolism, left femoral thrombophlebitis, marked renal tubular necrosis and moderate sclerosis of the coronary arteries. Meticulous examination showed no evidence of coronary thrombosis or myocardial infarction.

The rapid reversibility of the RS-T segment shifts here make it extremely unlikely that they were due to profound anatomic changes. It seems much more probable that these shifts resulted from chemical alterations. The acute pulmonary embolism was interpreted as a few days old and probably present when the electrocardiogram showed the unique features described above; and, hence could have accounted in part for the electrocardiographic changes described at that time.

Case 2. J. M., a 21 year old man developed acute renal failure following severe vertebral and abdominal injuries incurred in an automobile accident, associated with massive intraperitoneal hemorrhage and complicated by transfusion with incompatible blood. On admission his serum potassium level was 8.1 mEq. per liter, sodium 133 mEq. per liter, calcium 3.7 mEq. per liter and carbon dioxide 17.7 mM per liter. Electrocardiograms (fig. 2) were characteristic of acute potassium intoxication, showing atrioventricular and intraventricular block, peaked T waves and striking elevation of the RS-T segments in leads V1 to V3. At times there developed electrical alternans with alternation in the degree of elevation of the RS-T segments and in the direction of the T waves. The lowest strip in this figure shows a prolonged recording of this phenomenon. It is noteworthy that the T waves were upright when the RS-T "take-off" was higher and inverted when the RS-T "take-off" was lower. There was some variation in the amplitude of the QRS complexes but this was irregular and nonalternating. The respiratory rate was 24 and thus not an integral fraction of the heart rate (85). It seems reasonable, therefore, that this is a true example of electric alternans involving the T wave segment only, unrelated to cardiorespiratory synchronization. It was believed that though these tracings were com-
patible with right bundle branch block, the RS-T segment elevation could not be explained on that basis. These changes receded after dialysis with the artificial kidney, but returned on the following day in more severe form.

Three days later his condition deteriorated and he was again dialyzed. Preceding dialysis, the electrocardiogram was again characteristic of potassium intoxication (fig. 3 A). The T waves were tall and peaked, the QRS duration was 0.11 second with incomplete right bundle branch block, and the RS-T segments were depressed in leads I, II and III and V₁ to V₆, elevated in leads V₁ and V₂. These changes resembled those of acute anteroseptal myocardial ischemia in the presence of right bundle branch block or possibly early infarction in the same area. Within 30 minutes after the start of the dialysis, the tracings showed improvement and at the end of the six hour run the electrocardiogram had returned to within normal limits (fig. 3, B), but the RS-T segments were still slightly elevated. There was corresponding clinical improvement.

Five days later, on July 2, 1954, a third dialysis was carried out because of the redevelopment of potassium intoxication with disorientation, tinnitus and blurring of vision. The electrocardiograms again showed peaked T waves, intraventricular block and pronounced elevation of the RS-T segments over the right precordium (fig. 4, A). As before, the changes reverted toward normal (fig. 4, B) as dialysis proceeded and the serum potassium value fell from 8.4 to 4.2 mEq. per liter. Because of continuing oliguria, dialysis was again necessary on July 10, 1954. Similar but less striking electrocardiographic changes were recorded before dialysis and disappeared thereafter. On the twenty-fourth day after his injury the patient entered the diuretic phase of his illness. His strength and sense of well-being gradually returned over a period of several weeks. Electrocardiographic tracings gradually became completely normal.

Case 3. J. W. G., a 50 year old contractor, was admitted on Oct. 31, 1950, with severe glomerulonephritis and uremia. A pericardial friction rub was
Fig. 4. Duplication of same phenomenon in same patient; similar recession of RS-T segment shifts with dialysis. Five days later. (A). Tracing on July 2, 1954, when serum potassium level was 8.4 mEq. per liter. At this time RS-T segment elevation over right precordium was not clearly a change reciprocal to RS-T segment depressions over the left precordium. (B). Tracings after dialysis show, coincident with fall of serum potassium to 4.2 mEq. per liter, almost complete disappearance of RS-T segment elevations. Patient recovered.

Fig. 5. Dialysis eliminating changes of pericarditis and unmasking digitalis effect. J. G., a 50 year old man with chronic nephritis and uremia. (A). Initial tracings showing changes compatible with early acute pericarditis (RS-T segment elevation in leads II, III, aVf and V1 through V6). Tall peaked T waves suggest potassium intoxication. Serum potassium 5.2 mEq. per liter. (B). Two days later tracings show atrial flutter. Tracings on succeeding day, following digitalis therapy and before dialysis, show disappearance of flutter, no change in ventricular complexes. (C). Tracings following one hour of dialysis show decrease in T waves to normal amplitude, persistent but less pronounced RS-T shifts. (D). Tracings at end of dialysis show replacement of RS-T segment elevations by RS-T segment depressions suggesting digitalis effect. Autopsy showed fibrinous pericarditis, coronary sclerosis and myocardial fibrosis.
not heard. Electrocardiograms showed elevated RS-T segments in the left precordial leads characteristic of pericarditis (fig. 5, A). At the same time the T waves over the right precordium were tall and peaked, suggesting potassium intoxication. During this hospitalization the serum potassium level ranged from 3.8 to 5.2 mEq. per liter, the serum sodium ranged between 124 and 142 mEq. per liter but was usually low. Carbon dioxide varied from 10.3 to 21.1 mM per liter and was generally below normal. Two days after admission the tracings showed the development of auricular flutter (fig. 5, B); this arrhythmia disappeared when the patient was digitalized. On the following day the patient was treated with the artificial kidney. At the end of the first hour the peaked T waves had disappeared (fig. 5, C) but slight RS-T segment elevation persisted. However, five hours later, at the end of the dialysis, the RS-T segment elevation was replaced by RS-T segment depression (fig. 5, D), with a contour suggesting the effect of digitalis. The following blood chemistry values were determined immediately before and after the dialysis. Since the concentration of potassium in the bath fluid was 4 mEq. per liter there was relatively little change in the serum potassium as a result of the procedure.

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<tr>
<td>Nonprotein nitrogen mg. %</td>
<td>256</td>
<td>152</td>
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<tr>
<td>Carbon dioxide mM/L</td>
<td>8.1</td>
<td>13.2</td>
</tr>
<tr>
<td>Chloride mEq./L.</td>
<td>92</td>
<td>107</td>
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<tr>
<td>Sodium mEq./L.</td>
<td>133</td>
<td>142</td>
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<tr>
<td>Potassium mEq./L.</td>
<td>4.7</td>
<td>4.3</td>
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Following another dialysis the patient was discharged. He was hospitalized twice more because of further progression of his disease. He died on April 15, 1951. Postmortem examination showed chronic glomerulonephritis, fibrinous pericarditis, coronary arteriosclerosis and fibrosis of the myocardium especially involving the interventricular septum.

In this patient the electrocardiographic changes suggesting pericarditis were eliminated during the course of the dialysis. They were not seen again at any time in this patient. Fibrinous pericarditis was found at autopsy five and one half months after the illustrated sequence. In this case, acidosis rather than potassium intoxication may have been the

Fig. 6. RS-T shifts of pericarditis washed out by artificial kidney. L. G., a 19 year old soldier with acute renal failure and potassium intoxication (serum potassium 8.6 mEq. per liter). (A). Tracings (leads V₂ through V₅ only) show tall T waves, intraventricular block (probably right bundle branch block) and elevated RS-T segments in these leads. (B) Tracings after dialysis showing disappearance of all these changes excepting tall T waves, emergence of left ventricular hypertrophy. (C). Subsequent curves show tall T waves, no other electrocardiographic evidence of potassium intoxication. Postmortem examination showed fibrinous pericarditis, pulmonary infarcts and staphylococcus pneumonia and sepsis.
important chemical determinant. Whether the RS-T segment shifts reflected the anatomic changes in the pericardium or myocardium or were manifestations of electrolyte imbalance, the remarkable fact was their disappearance during the dialysis.

Case 4. L. O., a 19 year old soldier, sustained multiple fractures and soft tissue injuries in an automobile accident, followed by the development of acute renal failure for which he was transferred from an army hospital to the Peter Bent Brigham Hospital on Oct. 20, 1954. On admission his electrocardiogram showed tall, peaked T waves characteristic of potassium intoxication. During the early course of his hospital stay he was twice dialyzed, each time with marked subjective improvement, the second dialysis followed by electrocardiographic improvement as well. On Oct. 24 his electrocardiogram showed RR' complexes in lead V₁ and sinus tachycardia (rate 110). On the following day tachypnea, tachycardia and a split second pulmonic sound were noted. These clinical and electrocardiographic findings justified the suspicion of acute pulmonary embolism. On October 27 he was again dialyzed; the T waves became lower and the RR' complex in lead V₁ replaced by RS complex. On October 30 the patient's condition and his electrocardiograms indicated advancing potassium intoxication. His serum potassium level rose to 8.6 mEq. per liter and the electrocardiograms showed broader QRS complexes and taller T waves. On the day before his fourth dialysis (fig. 6, A), there were RR' complexes and elevated RS-T segments in leads V₁ to V₄. This change persisted until the time of the dialysis. Tracings taken after the procedure (fig. 6, B) showed disappearance of these changes. They did not recur (fig. 6, C), although the peaked T waves continued. The dialysis resulted in the following chemical changes:

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<td>Blood urea nitrogen mg. %</td>
<td>290</td>
<td>165</td>
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<tr>
<td>Carbon dioxide mM/L</td>
<td>17.3</td>
<td>20.9</td>
</tr>
<tr>
<td>Chloride mEq./L</td>
<td>92.1</td>
<td>96.7</td>
</tr>
<tr>
<td>Sodium mEq./L</td>
<td>131</td>
<td>135</td>
</tr>
<tr>
<td>Potassium mEq./L</td>
<td>8.3</td>
<td>5.0</td>
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Three days later, amputation was performed at the junction of the upper third and lower two-thirds of the left leg. Within the next several days he had definitely entered into the diuretic phase of the kidney disease and he seemed to be recovering. But on November 10 he developed sepsis and pneumonia to which he succumbed within 24 hours. Postmortem examination showed staphylococcus septicemia and pneumonia, ischemic nephrosis, fibrinous pericardi-}

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tis and multiple recent hemorrhagic infarcts of the lungs.

**Discussion**

Previous studies have demonstrated that depression of RS-T segments in certain of the precordial and limb leads and, frequently, elevation of the RS-T segments in lead aV₄₀ are observed in moderately advanced potassium intoxication, and that these shifts may be eliminated by hemodialysis with the artificial kidney.¹ ² ³ Some of the patients described in these earlier reports showed, as well, elevation of the RS-T segments in lead V₁ (figs. 2, 3, 5 and 9 of the first report,¹ fig. 3 of the second²) or in lead V₂ (fig. 4 of the third paper³). These changes were generally associated with intraventricular block. They were never widespread or profound enough to have suggested myocardial infarction or pericarditis but in the last case listed the possibility was suggested that they might be due to acute cor pulmonale. In one of a series of studies dealing with conditions that may be mistaken for myocardial infarction, Myers⁴ presented a case of potassium intoxication which showed, in addition to the more usual manifestations, an apparent elevation of the RS-T junction and a cove-like RS-T segment and T wave in lead aV₄₀ resembling that seen in myocardial infarction. Comparison with other leads, however, showed that this shift was recorded within, and was thus part of, a prolonged QRS complex; this change was thus referable to the conduction defect of potassium intoxication and not a sign of infarction. Following this lead, all tracings in the present series were re-examined to determine whether the RS-T segment shift was an apparent rather than real one and produced in a slurred prolonged QRS complex; but this possibility could not be confirmed in any of the cases here presented.

In cases 1 and 4 the sequence was somewhat muddied by the development of acute cor pulmonale which may conceivably have contributed, in part, to the electrocardiographic picture. RS-T segment elevation in the right precordial leads may develop as a reciprocal change to RS-T segment depression over the left precordium. This may occur in left bundle
branch block and may account for some (a small part, it is believed) of the RS-T segment elevation in leads V₁ to V₄ in case 1. But these RS-T segment shifts cannot be explained by the development of incomplete right bundle branch block such as was recorded in cases 2 and 4. There is nothing about right bundle branch block as such, complete or incomplete, which would be expected to result in elevation of the RS-T segments over the right pre-
cordium.

In some cases the sole electrocardiographic evidence of acute cor pulmonale may consist
of inversion of the T waves over the right pre-
cordium, with, in rare instances, elevation of the RS-T segments in the same region. The possibility that the changes in cases 1, 2 and 4 are examples of this phenomenon seems extremely unlikely but cannot be denied categorically. But this would not explain the presence or distribution of RS-T segment shifts in case 3, which neither clinically nor pathologically presented evidence of acute cor pul-
monale. Nor would it explain the rapid reversibility of these changes on dialysis in all four

cases.

It has been demonstrated in the experimental animal that subendocardial injection of potas-
sium salts results in RS-T segment depression in leads related to the overlying epicardium, while the subepicardial injection of potassium salts or their application to the pericardial surface produces RS-T segment elevation in the same leads. There is no evidence that the blood perfusing the deeper layers of the ventricular wall has a different potassium content from that bathing the more superficial layers. From the fact that in moderately advanced potassium intoxication RS-T segment depression is generally recorded in the precordial leads and RS-T segment elevation in lead aV₅ (to which, in general, more of the endocardial potentials are projected) and from the fact that block in the subendocardially located bundle branches may be recorded occasionally during the development of potassium intoxication, it was suggested, by exclusion, that this phenomenon may be explained by the greater vulnerability of the subendocardial

laminae to the toxic effect of potassium. This has not been proved.

One of the major struts of the science of electrocardiography has been electrocardiographic-pathologic correlation. For years electrocardiographers have sought vindication in the anatomic verification of their deductions from the electrocardiogram. The RS-T segment shifts of acute myocardial infarction and acute pericarditis, for example, are assumed to result from anatomic changes in the myo-
cardium. At times electrocardiographic changes characteristic of either of these conditions are not associated with histologically demonstrated lesions. In explanation of this discrepancy, it has at times been averred that the lesion is still in its "biochemical phase"; the anatomic phase has not yet been attained. In the past such claims have been made rather loosely, on a priori grounds. But support for this attitude has recently been offered by the results of the new technics of histochemistry. The studies of succinic dehydrogenase activity by Wach-
stein and Meisel and those of Yokoyamo and co-workers, using a histochemical technic, demonstrated reduction in enzyme activity in the early hours after experimentally induced or spontaneous myocardial necrosis. This reduction in enzyme activity frequently extended to fibers which showed no significant changes with routine staining technics.

Another new technic is dialysis with the artificial kidney. This procedure was intro-
duced as a practical approach to the correction, for longer or shorter periods of time, of certain abnormalities in body chemistry. The present series of observations with this technic raises certain theoretic questions. It seems reasonable that a change which can be elimi-
nated by dialysis should be considered a chemical change. Would this same reasoning apply to a dialyzable "current of injury?" And should not these changes be considered chemical in origin, whether or not they are associated with anatomic changes? A demon-
stration that electrocardiographic changes of this type, occurring in proved myocardial infarction or pericarditis in the absence of electrolyte imbalance, can similarly be reversed
rapidly on chemical manipulation, would be of profound interest. We know of no observations on the effect of artificial hemodialysis upon these electrocardiographic phenomena.

Summary and Conclusions

Four patients with potassium intoxication associated with acute or chronic renal failure showed RS-T segment elevations over the right or left side of the precordium, resembling that occurring in acute myocardial infarction or pericarditis. One patient survived. The three fatal and autopsied cases (cases 1, 3 and 4) showed at postmortem examination pulmonary embolism (cases 1 and 4), fibrinous pericarditis (cases 3 and 4) or coronary artery disease with myocardial fibrosis (case 3) or without it (case 1); none showed fresh myocardial infarction. Regardless of this complicated anatomic substrate, it was possible in all four cases specifically to eliminate this “current of injury” entirely or, in large part, by artificial hemodialysis with the artificial kidney. This emphasizes the ultimate chemical origin of these changes, whether or not associated with anatomic changes.

In dealing with patients with potassium intoxication the possibility of confusion with acute myocardial infarction should be borne in mind.

Summary in Interlingua

Quatro patientes con intoxication a kalium associate con acute o chronic disfallimento renal monstrava supra le dextere o sinistre latere del precordio elevationes del segmento RS-T que eseva simile a illos occurrente in acute infarcimento myocardial o in pericarditis. Un del patientes superviveva. Le tres casos mortal (casos 1, 3, 4) eseva autopsiae e monstrava embolismo pulmonar (casos 1, 4), pericarditis fibrinose (casos 3, 4), o morbo del arteria coronari, con fibrosis myocardial (caso 3) o sin illo (caso 1). Nulle de iste casos monstrava recente infarcimento myocardial. In despecto de iste complexe substrato anatomic, il eseva possible in omne quatro casos eliminar iste “currente de lesion” completamente o extensemente per medio de hemodialyse artificial in le ren artificial. Iste facto sublinea le origine ultimemente chemic del alterationes sub discussion, si o non illos es associate con alterationes anatomic.

In casos de patientes qui mostra intoxication a kalium, on debe considerar le possibilitate de un confusion con acute infarcimento myocardial.

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