This 74-year-old white man had carcinoma of the bladder for four years. A few days prior to this electrocardiogram, he showed signs further substantiated by laboratory determinations consistent with urinary tract obstruction. On the day this electrocardiogram was taken, the following laboratory findings were reported: potassium 7.4 mEq/L.; calcium 12 mg. per cent; CO₂ 23 mEq/L.; albumin 2.2 gm. per cent; globulin 3.8 gm. per cent; creatinine 6.5 mg. per cent; phosphorus 7 mg. per cent; pH 7.46; magnesium 2.95 mg. per cent; (1.7 to 2.7 mg. per cent). The patient expired two days later. Necropsy revealed a normal heart.

This electrocardiogram demonstrates the classic changes of hyperpotassemia and hypercalcemia.

**THE QRS CHANGES**

The QRS is slightly broadened with the terminal vectors directed to the right, posterior, and superior; note that S waves are present in V₂, V₅ and aVF. As the serum potassium level increases, the initial QRS vector forces are also increased in duration and changed in direction, simulating at times myocardial infarction. Note in this case that the Q waves present in standard leads 2, 3 and aVF are approximately .02 to .03 seconds in duration. Since hyperpotassemia decreases the rate of ventricular depolarization, the prolonged QRS com-

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**FIGURE 1**

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plex would be expected to terminate rightward, posterior and superior. Interestingly enough, this is the location of the last portion of the heart to be depolarized normally — the crista supraventricularis and the basal portion of the heart.

The T-wave changes of hyperpotassemia, that is, the presence of tall peaked T-waves over the precordium, mimic the electrocardiographic changes of myocardial ischemia, sinus bradycardia, diastolic overload of the left ventricle, etc. However, hyperpotassemia has the following changes not usually present in the above: (1) narrow-based T-wave; (2) no change in the T-wave axis in the horizontal plane (note in this case the flattened T-wave in V₅R); (3) presence of an S-wave in V₅; (4) absence of U-waves. U-waves are rarely present in hyperpotassemia, but frequently are present in sinus bradycardia, myocardial ischemia and diastolic overload of left ventricle. It should be pointed out that potassium transport across the cell membrane is one of the major determinants for the inscription of the T-wave. Therefore, at times, the above differential diagnosis is difficult to make electrocardiographically.

**The ST Segment and QT Interval**

In this case, the corrected QT interval and ST segment are shortened. The specific shortening of the ST segment is classic and almost pathognomonic for hypercalcemia. However, digitalis also shortens the ST segment. Usually digitalis displays the classic sagging of the ST segment with an ST segment vector directed to the right. From basic experimental studies, hyperpotassemia also shortens the ST segment and QT interval. However, from a practical clinical standpoint, this slight shortening is not readily seen in a single electrocardiogram.

Little experimental work has been done with electrocardiogram changes in hypermagnesemia. However, data indicate that all electrocardiographic changes in hyperpotassemia qualitatively but to a lesser extent quantitatively can be produced by hypermagnesemia.

The interesting electrocardiographic clinical point in this case is that hyperpotassemia is usually associated with chronic renal disease which lowers the serum calcium and prolongs the ST segment. The presence of shortened QT interval and ST segment and the electrocardiographic changes of hyperpotassemia decrease the differential diagnostic possibilities.

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**MITRAL INSUFFICIENCY IN CHILDREN**

When the heart is involved as a result of rheumatic fever in children, mitral insufficiency is the most common lesion. Significant mitral stenosis is not common in this age group. Twenty-eight children with rheumatic mitral insufficiency were divided into three groups of severity according to symptoms and heart size. Cardiac volume index and cardiothoracic ratio were compared as indexes of heart size in these patients. The course and prognosis were studied in each group by using cardiac volume index, cardiothoracic ratio, electrocardiographic phonocardiographic, hemodynamic and clinical data as parameters of severity. In severe cases, the course is one of progressive deterioration with development of atrial fibrillation or left atrial and right ventricular hypertension, or both. This course was illustrated by eight fatal cases, death occurring between 11.5 and 15.5 years of age. In two patients, the mitral valve was replaced by a Starr-Edward valve. The patients are surviving and well, 12 and 17 months later.