Electrocardiographic Changes and Plasma Potassium Levels in Patients on Regular Haemodialysis

M. PAPADIMITRIOU,∗M.D. ; R. R. ROY,†M.B.,F.R.C.S.,F.R.C.S.Ed. ; M. VARKARAKIS,‡M.D.

Summary: Two out of four patients who had severe ischaemic E.C.G. changes when first accepted on to the dialysis programme showed much improvement after six months. From an analysis of 40 electrocardiograms (20 before and 20 after dialysis) of a further 17 patients it was found that the height of the T wave is a good index of the plasma potassium level. The tolerance of higher plasma potassium levels by these patients compared with patients with acute renal failure may be explained by the fact that the former do not have a hypercatabolic process and have a lesser degree of acidosis.

Introduction
Haemodialysis removes excess salt and water, urea, creatinine, urate phosphate, and other toxic substances accumulated in the plasma between dialyses. Hyperkalaemia is a great danger since it may cause sudden cardiac arrest in any oliguric patient with acute or chronic renal failure (Shaldon, 1966; Douglas and Kerr, 1968). During dialysis potassium loss is controlled by using a low concentration of potassium in the dialysate. Hypokalaemia with associated electrocardiographic (E.C.G.) changes has been noted during potassium-free dialysis (Klutsch, 1965).

Though there is no exact correlation between the serum potassium level and E.C.G. changes, the sequence of changes in the E.C.G. pattern is characteristic (Douglas and Kerr, 1968), particularly with a raised level of serum potassium (Black, 1968). We frequently noted that some of our patients had very high serum potassium levels before dialysis, without symptoms. We decided to investigate early E.C.G. changes in these patients, because it is known that the E.C.G. pattern is a more sensitive indicator of cardiotoxicity than the serum potassium level (Bellet, 1963).

Patients and Methods
Twenty-one patients on regular haemodialysis were studied; each patient had twice weekly 12-hour overnight haemodialysis by a modified Kilii two-layered parallel flow machine with a warm single pass automatic dialysate supply (Papadimitriou and Kulitlake, 1970). So far as possible all patients received a carefully controlled diet containing 0·7—0·9g. of protein per kg. body weight with intake of about 25 mEq of sodium, 70 mEq of potassium, and 300 mg. of calcium per day. No patients were on digitals.

Discussion

Though hyperkalaemia is undoubtedly the most important factor in producing E.C.G. changes such as taller and peaked T waves, smaller P wave, and widened QRS complex, probably other factors are also concerned, including acidosis, hypopatraemia, and hypocalcaemia (Douglas and Kerr, 1968). In patients on regular haemodialysis these three factors are kept mainly within normal limits, and cardiac arrhythmias rarely occur in such cases (Hampers and Schupak, 1967). The symptoms and signs of hyperkalaemia, such as general weakness, tingling and numbness of the extremities, and paralysis of the skeletal muscles, are neither early nor pathognomonic. We found a statistically significant negative correlation between plasma potassium and pulse rate (Fig. 1). Neither height of the P wave nor the length of the QRS complex are very useful indications of potassium effect because of the wide scatter of values (Figs. 2 and 3). The height of the T wave was found to be the most useful index (Fig. 4). T waves higher than 4 mm. in lead II should be considered as corresponding with a serum potassium above 6 mEq/l. The above findings agree with those of Black (1967), who suggested that and, in addition, hypocalcaemia and serum sodium disturbances, if present, contribute to the easy appearance of arrhythmias, which could be fatal. Conversely, patients on regular haemodialysis are not in a hypercatabolic state, their serum sodium and calcium are usually within normal limits, and the rise in serum potassium depends mainly on dietary indiscretion and mild acidosis developing during dialysis.

Hence higher serum potassium levels can be present in patients on regular haemodialysis without serious arrhythmias (Fig. 5). Between February 1968 and June 1969 (15 months) we used a concentration of 1.5 mEq of potassium per litre of dialysate fluid (Papadimitriou et al., 1968) and had no deaths from hyperkalaemia in 296 patient-months. Hypokalaemia has never been a serious problem, and this is in agreement with Seedat (1969). In a very few patients who present with low serum potassium levels we usually add potassium chloride to the dialysate.

A few patients undergoing regular haemodialysis have serum potassium levels consistently between 6 and 7 mEq/l. on attendance. Since our patients are dialysed overnight we usually estimate the pre-dialysis potassium levels the next day. Under these circumstances it is useful to monitor the E.C.G. before dialysis. A close approximation to the serum potassium level up to about 8 mEq/l. can be made by applying the algorithm \[ K^+ = \frac{3t^2}{2} \]
where \( K^+ \) is serum potassium in mEq/l. and the height of the T wave in lead II of the E.C.G. in millimetres.

We wish to thank Professor R. Shackman and Mr. G. D. Chisholm for permission to study patients under their care. Thanks are also due to the nursing staff of the artificial kidney unit and to Mrs. J. Vickers for her technical help.

Requests for reprints should be addressed to Mr. R. Roy.

References

Seedat, Y. K. (1966), British Medical Journal, 2, 344.