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Effect of Potassium on the Ventricular Deflections of the Electrocardiogram in Hypertensive Cardiovascular Disease.*

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Many reports have been published concerning the effects of potassium on the electrocardiogram in various clinical states[1-13] and in experimental animals.[14-17] These have been concerned chiefly with its effect upon the T-wave. Relatively little attention has been devoted to its effect upon the QRS complex except in those cases in which toxic doses were given. Inspection of the published records, however, shows that in many instances a reduction in the size of the QRS deflections followed the administration of this drug. We refer particularly to instances in which potassium was given to patients with hypertensive heart disease. In the articles referred to the QRS changes were not mentioned in the texts or legends. In fact in some of these it was reported that no change in the form of these deflections took place.[4,13] In a previously reported study[18] sodium restriction in hypertensive vascular disease was followed by retrogressive electrocardiographic changes unaccompanied by a significant change in blood pressure. This raised the question as to whether a relative increase in tissue potassium secondary to sodium restriction was responsible for these electrocardiographic phenomena. Consequently the effect of potassium alone upon the electrocardiogram in cases of hypertension was investigated. Potassium in the form of the chloride, dibasic phosphate, or citrate was administered, on 25 occasions, to patients with abnormal electrocardiogams characteristic of hypertensive vascular disease, and, on ten occasions, to normal subjects. Electrocardiograms were obtained immediately before and at times ranging from 15 minutes to 3 hours after the ingestion of 5 to 20 g of one of the potassium salts mentioned. In several instances 10 to 24 g of potassium salts daily had been prescribed previously and the patient had continued on this dosage up to the time of the test.

In every instance in which potassium was

* The observations upon which this paper is based were carried out with the aid of grants to Frank N. Wilson from the Horace H. Rackham School of Graduate Studies and the S. S. Kresge Foundation.

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given to a patient with hypertensive heart disease accompanied by the characteristic changes in the electrocardiogram there was a reduction in voltage of the QRS deflections which persisted for several hours. Frequently there was also a reduction in the length of the QRS interval. In the vast majority of cases the T-wave became less inverted or upright when it was originally inverted, or taller when it was originally upright. Serial electrocardiographic observations showed that following the administration of potassium the electrocardiogram passes in reverse order through the same series of configurations that it passes through in the course of the development of the changes characteristic of hypertensive heart disease. In a few instances the T-wave became more deeply inverted after potassium even though the voltage of the QRS deflections was reduced. In the majority of instances the mean axis of the QRS shifted to the right of its original position while that of the T-wave was shifted to the left. The magnitude of the ventricular gradient was usually increased and its direction was shifted slightly to the right.

In normal subjects the administration of potassium was followed by electrocardiographic changes similar in character but less pronounced than those seen in cases of hypertension. In one patient with left ventricular enlargement due to luetic aortic insufficiency, but with no definite increase in blood pressure, the administration of potassium was followed by pronounced toxic symptoms and transient left bundle branch block.

In hypertensive vascular disease sodium restriction alone is followed by electrocardiographic changes identical with those produced by the administration of potassium. No close correlation between the changes in blood pressure and the changes in the electrocardiogram is apparent. It should be pointed out that the electrocardiographic changes following sympathectomy in hypertensive patients are of the same type, and, that in this case also these changes are not always very closely related to the effects on the blood pressure.

In calling attention to the similarity between the changes in the electrocardiogram produced by the administration of potassium in cases of hypertensive heart disease, and the changes in the electrocardiogram which often follow sympathectomy or the restriction of sodium in cases of the same kind, we do not imply that the mechanism by which these electrocardiographic changes is brought about is necessarily the same in both instances. This is a question which can be decided only by future investigations.

The potassium dibasic phosphate used in this study was supplied through the generosity of Parke, Davis and Company.

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A Gaseous Nitrogen Elimination Test to Determine Pulmonary Efficiency.

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Nearly all physiologists interested in respiration have contributed to our knowledge of nitrogen elimination produced by decompression or by oxygen inhalation. As space prevents an adequate review of the literature, we can only refer the reader to typical references of studies by Haldane and Priestley,[1]

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