For comparison rectus femoris muscles of the same animals were excised in the same manner without previous stimulation and tested for lactates according to the same technique.

Results are as follows:

Stimulation period 30 seconds.

	Lactate in mgm.%		Lactate in mgm. %
Non-worked control	44	worked muscle	220
	56		165

Stimulation period 1 minute.

Non-worked control	61	worked muscle	246
	63		264

The increases in lactate resulting from these brief periods of activity are equal to or greater than the increases reported by us after 15 minutes of tetanization under similar conditions.[2]

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The Therapeutic Use of Potassium in Certain Cardiac Arrhythmias.*

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The investigation of the action of potassium salts on the heart dates from the time of Ringer,[1] whose classical experiments on the frog heart are well known. Irrespective of osmotic tension, the necessity of sodium, potassium and calcium ions in a balanced solution was proved. Hering[2] stopped paroxysmal ventricular tachycardia and ventricular fibrillation in the dog, and many investigators including Rothberger and Winterberg,[3] Anrep,[4] and Wiggers[5] have similarly obtained immediate cessation of both auricular and ventricular ectopic rhythms by intravenous or intracardiac injections of

[2] Martin, E. G., Field, J., II, and Hall, V. E., PROC. SOC. EXP. BIOL. AND MED., 1929, **26**, 292.

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[1] Ringer, S., *J. Physiol.*, 1882, **3**, 380; 1883, **4**, 429.

[2] Hering, H. E., *Zentralbl. d. Physiol.*, 1903, **17**, 1.

[3] Rothberger, C. J., and Winterberg, H., *Arch. f. d. ges. Physiol.*, 1911, **142**, 461.

[4] Anrep, A., personal communication.

[5] Wiggers, C. J., *Am. J. Physiol.*, 1930, **92**, 223.

solutions of potassium chloride. Wiggers[5] suggested the possibility of its therapeutic use in ventricular fibrillation of accidental electrocution. It thus seemed reasonable to attempt to disturb the calcium-potassium ratio in favor of potassium for certain other therapeutic reasons. Scherf[6] and others have demonstrated a reversal of the customary actions of potassium and calcium when used intravenously on ventricular ectopic rhythms caused by aconite. The calcium eliminated the aberrant beats, while the potassium precipitated showers or paroxysms of them. It is predicted that some cases may be encountered clinically in which the arrhythmias show such a paradoxical response to potassium.

Norn[7] showed an increment in blood potassium of 21% 40 minutes after the oral administration of 12 gm. of potassium chloride; and 61% increment in 2 hours. An appreciable increment was obtained in 30 minutes after 2 gm. of KCl in 2 of our cases.

Potassium salts were used clinically to check attacks of paroxysmal ectopic ventricular tachycardia, and to prevent the occurrence of auricular and ventricular ectopic beats. Four different soluble potassium salts were used: potassium chloride, potassium iodide, potassium citrate, and potassium acetate, all with apparently identical effect. It is presumed that the anion and acid-base influences are negligible. For later use, we have employed only the acetate, because it causes no gastric irritability when administered in raspberry syrup or similar menstruum.

It was found that potassium may be safely administered orally in doses of 1 to 5 gm. of certain soluble salts, to human cases of heart irregularities. In 12 cases of such oral administration in auricular or ventricular ectopic beats or tachycardias, definite control of the arrhythmias was obtained in 5 cases. In Case No. 1, of our series, two attacks of paroxysmal ventricular tachycardia, with duration of 8 hours and 4 days respectively, were checked within the period of expected absorption time for potassium. Of the remaining 7 cases, 4 were free of any other evidence of cardiac pathology; and of these 4, none showed even a suggestion of positive response to potassium. The failure to affect this group may be used to differentiate such patients from those with true myocardial damage. A low potassium content of myocardium is known to exist in cases of heart muscle failure, and may be the explanation for this difference in response. The influence of potassium on the arrhythmia occurred in 30 to 90 minutes after administration; and the

[6] Scherf, D., *Z. f. d. ges. Med.*, 1929, **65**, 255.

[7] Norn, M., *Skand. Arch. f. Physiol.*, 1929, **55**, 213.

necessary dose required varied in different individuals. The effect was maintained from 6 to 8 hours, and occasionally some effect was observed for the succeeding 24 to 48 hours. A definite effect on the electrocardiograms in certain cases after potassium administration was observed. Certain ill effects are occasionally noted, namely, gastric distress and diarrhea. The acetate and citrate caused less distress than the chloride or iodide. The paroxysmal nodal tachycardia observed in one case and the epistaxis and petechiae in another may have been due to potassium administration.

Conclusion. Potassium salt administration by mouth is effective in checking auricular and ventricular ectopic beats and tachycardia in the majority of cases of organic heart disease. The failure to affect arrhythmias in patients without other evidence of cardiac pathology may be used to differentiate this group from the former. Potassium administration does not prevent the occurrence of auricular fibrillation.

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The Relation Between Acidosis and Glucose Tolerance.

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In earlier work[1] it was shown that phlorhizin glycosuria the glucose tolerance was directly related to the nutritional condition of the animal. Low tolerance was noted in those animals which had been fasted and phlorhizinized for 5 to 6 days while the opposite was found in those dogs which had received large doses of carbohydrate 12 to 15 hours previous to the test, although these animals were still phlorhizinized. These variations were similar to those found in the same dogs in the non-phlorhizinized state after fasting or after feeding large amounts of carbohydrate before carrying out the glucose tolerance test.

In each case the increased tolerance is associated with an abolition or a considerable decrease in the ketosis. The present investigation was designed to determine whether the acidosis *per se* was the cause of the decreased ability to store carbohydrate. Since it is impossible to produce a ketosis and at the same time have a large carbohydrate storage, an acidosis was produced by the injection or

[1] Deuel, H. J., Jr., *J. Biol. Chem.*, 1930, in press.