brought into this state have, so far, not been observed to return to their previous condition. The rate of flow of the currents gradually slows down until the animal dies.

The protoplasm of an *Amœba* exists in a certain normal state of consistency from which it may deviate so as to solidify on the one hand or liquefy on the other. This normal state may be shifted not only by agitating the *Amœba* but also by injecting certain solutions. This I have been able to do with hydrochloric acid and with sodium hydrate.

A trace of acid throws the normal state to the more solid side, while the alkali throws it to the more liquid side. An acidified *Amœba* forms long slender pseudopodia because the peripheral back flow in the developing pseudopodium is quickly arrested by a setting of the protoplasm. The area of the base of the pseudopodium is, therefore, quickly limited and the extending pseudopodium conforms to this narrow base. In an alkalinized *Amœba*, on the other hand, the peripheral back flow of a developing pseudopodium tends to be arrested much more slowly. As a result of this the base of the pseudopodium spreads considerably before the protoplasm sets. The extending pseudopodium, having a larger base upon which to build, then becomes broadly lobate.

These observations harmonize with my experiments on injecting "acid" and "basic" organic dyes. The basic dyes, which contain a relatively strong acid radicle, jelly the protoplasm, whereas acid dyes, with a strong basic radicle, liquefy it.

It is interesting to note that these changes can be brought about in protoplasm while it is yet alive and that one can thereby change the character of the pseudopodia produced.

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Alterations in the cardiac mechanism after administration of quinidine to patients with auricular fibrillation.[1]

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Sufficient evidence is now at hand to indicate that in a certain number of patients suffering from fibrillation of the auricles

[1] This paper was presented at the One Hundred Thirteenth meeting of the Society for Experimental Biology and Medicine, October, 1921.

(about 50 per cent.), oral administration of quinidine sulphate serves to restore the normal rhythm. It is the purpose of this communication to record the mechanism of the heart's action which has been observed in the first eleven patients who have received quinidine in this hospital.

Five hundred and seven electrocardiograms taken on eleven patients have been measured and analyzed. After a preliminary dose of .2 to .4 gm. of quinidine to test for the presence of an idiosyncrasy to members of the cinchona group, .4 gm. of the drug has been given by mouth, in gelatin capsules, either three times daily or every two hours, until either the establishment of normal rhythm or the appearance of untoward symptoms indicated cessation of therapy. No more than 2.0 gm. of quinidine were administered in twenty-four hours, though treatment has been continued daily for as long as ten days.

Electrocardiograms were taken in some instances as often as every five minutes during the time when a change in rhythm was anticipated. Usually curves were made at two-hour intervals on the days on which the drug was given, and at least daily throughout the periods of observation.

Cases in which the Normal Mechanism was Restored.—Three patients received ten courses of quinidine. Restoration of the normal mechanism was accomplished nine times. The first effect noted was usually an acceleration of ventricular rate. This was followed at times by the appearance of premature beats, arising more commonly in the right, but occasionally in the left ventricle, and at times in both. If electrocardiograms were taken at sufficiently frequent intervals, the transitional mechanisms in the common order of their appearance were: coarse fibrillation, impure flutter, flutter, and normal rhythm. This sequence was not invariably demonstrated in all its phases, and it is possible that one or more of the intermediate mechanisms may be omitted. In one patient the transition from auricular flutter to the normal rhythm was photographed in the second lead. The change was rather abrupt, there being a period of altering auricular activity, slowing of ventricular rate for several beats, a relatively long period of a systole of both auricles and ventricles and then prompt resumption of the sinus rhythm. The P waves, denoting auricu-

lar activity, were well formed, even in the first normal beats, though showing some tendency to slight alterations both in form and in voltage during the early cycles of the restored sequential mechanism.

On the day on which normal rhythm was established the rate usually fell to 80 or 90, and on the following day to 60 or 70, and subsequently remained at or about this level. During the time the normal rhythm prevailed, it was common to see occasional auricular premature beats. These, together with ventricular premature contractions, when these were present, could be abolished by giving more of the drug.

P-R (conduction) time, after appearance of the sinus rhythm, was within normal limits in two patients, and was lengthened appreciably in the third. It is not possible to say whether the drug was responsible for delaying conduction of the impulse through the auriculo-ventricular bundle in this instance.

In one patient the QRS interval, which may be taken to indicate the time of the conduction of the impulse through the ventricles, was lengthened. In two patients, after establishment of normal rhythm, there was alteration in the form of the ventricular complex, exhibiting itself commonly as a change in the voltage and contour of the R and S waves.

The T wave often tended to reverse its direction and change its voltage after the normal rhythm was restored, returning to its original direction and form after reversion to the fibrillatory mechanism.

In one patient paroxysms of ectopic ventricular tachycardia preceded the onset of impure flutter, which, in turn, was followed by the normal rhythm. In this same patient sino-auricular block and paroxysms of auricular tachycardia were also observed when the sequential rhythm prevailed.

Digitalization prior to quinidinization was not an essential factor for success in therapy, for in the same individual the normal mechanism was restored on one occasion with ventricular rate of 180 and at another time, after the administration of digitalis, when the ventricular rate ranged from 90 to 100.

The duration of the normal rhythm after a single course of quinidine varied from a few hours to 23 days. In one patient it

has been possible, by means of intermittent quinidine therapy, properly spaced, to maintain the normal rhythm for over five months, with coincident marked clinical improvement.

Intravenous injection of atropine sulphate (1.0 to 1.5 mgm.) in these patients, at a time when fibrillation was present and again when the normal rhythm prevailed, resulted in the usual increase in ventricular rate, but in no significant alteration in the cardiac mechanism or in the form of the electrocardiogram.

Cases in which Restoration of the Normal Mechanism was Not Accomplished.—Eleven courses of quinidine were administered to 8 patients. As in the group just decsribed, tachycardia was commonly the first effect observed. Ventricular premature beats, at times in the form of coupled rhythm, were more commonly seen than in those patients in whom the sinus rhythm was eventually established. Occasionally the fibrillatory waves became coarser. In two patients auricular flutter followed administration of the drug, but a larger dosage was not followed by the normal rhythm. In one of these cases auricular flutter persisted for three days and was followed, after administration of digitalis, by reversion to the fibrillatory mechanism. Paroxysms of ectopic ventricular tachycardia occurred three times. Although of short duration, they served to indicate that quinidine as a therapeutic agent was not to be administered with impunity, for ventricular tachycardia occurring in dogs poisoned by digitalis or strophanthin is not infrequently the precursor of ventricular fibrillation.

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Studies on the acetonuria produced by diets high in fat.

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The following ratio was suggested to express the ketogenic balance of any diet:

$$100 \times \frac{1.5\ (\text{weight carbohydrate} + 25\ \text{per cent. weight protein})}{95\ \text{per cent. weight fat}}.$$

This ratio is based on the relative molecular weights of glucose and the higher fatty acids—stearic, palmitic, and oleic; in it it is