tion after three to four doses. In no experiment did a conspicuous increase in the refractory period occur.

Since the length of the refractory period is greatly influenced by the heart rate, it was, of course, necessary while the tests were being made to maintain artificially a constant rate considerably above the natural level. Between tests the heart was allowed to beat naturally. It was found that when the heart rate was suddenly raised, the refractory period did not at once drop to the level that it afterwards reached and maintained. In our first experiments the influence of the previous rate of beating upon the determination made immediately after a change in heart rate, was not realized. Confusing variations in the refractory period were, therefore, encountered.

In these experiments the possibility of variation in the threshold of excitability was eliminated by determining the value of the threshold stimulus from time to time.

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Effect of pilocarpin upon the cardiac mechanism in circus rhythm with ventricular tachycardia.

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Pilocarpine is known to stimulate the cardiac fibers of the vagus. The site of its action is in the neighborhood of the myoneural junction. Since vagus stimulation tends to increase the rate of the circus rhythm in auricular flutter and fibrillation and also to reduce the ventricular rate, pilocarpin might be expected to produce these effects.

We have given pilocarpin hydrochloride intravenously, in doses of one-sixteenth to one-eighth grain, to a number of patients with auricular fibrillation or flutter. In the majority of cases no definite effect was produced either upon the ventricular rate or upon the auricular mechanism. In one instance short attacks of auricular flutter were immediately abolished. In two cases of extreme ventricular tachycardia which followed the administration

of quinidin, pilocarpin produced a sudden and abrupt fall of ventricular rate. In one of these cases, in which the auricles were fluttering, this was associated with a slight rise in the rate of the circus rhythm.

The action of pilocarpin, upon the vagus, in the dosage referred to, is very feeble. Nevertheless, the drug may occasionally prove useful in the suppression of post-quinidin tachycardia. The tachycardia which follows the administration of quinidin in cases of auricular fibrillation and auricular flutter is due to a depression of the rate of the circus rhythm, combined with partial vagus paralysis. It may be extreme and may cause the patient serious discomfort. The action of pilocarpin is peripheral to that of quinidin, and although its action is feeble it may increase vagal tone sufficiently to greatly reduce the ventricular rate.

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Nature of abnormal ventricular complexes during quinidin treatment of auricular fibrillation.

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When quinidin is given to patients with auricular fibrillation there is almost invariably a considerable increase in the ventricular rate. This is accompanied in one-third to one-half of the cases by the appearance of groups of abnormal ventricular complexes. These abnormal complexes must be due either to abnormal impulse formation in the ventricular muscle or to defective intraventricular conduction. The former explanation has been advanced by Cohn,[1] Levy,[2] and Lewis[3]; the latter by White,[4] and others.

[1] Cohn, A. E., Personal Communication.

[2] Levy, *Arch. Int. Med.*, 1922, xxx, 474.

[3] Lewis, T., Drury, A. N., Wedd, A. M., and Iliescu, C. C., *Heart*, 1922, ix, 254.

[4] White, P. D., Marvin, H. M., and Burwell, C. S., *Boston M. and S. J.*,