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.*,

We have studied two cases in which these abnormal ventricular complexes were unquestionably due to defective intraventricular conduction. In the first case, runs of abnormal ventricular complexes occurred after eighteen grains of quinidin. After twenty-four grains all of the ventricular complexes were of the abnormal type. The administration of one-eighth grain of pilocarpin, intravenously, slowed the ventricular rate, and the longer pauses were followed by relatively normal complexes. There was, however, no close relation between the length of the pause and the form of the complex which followed. About two hours later, the auricular fibrillation gave place to normal rhythm but the ventricular complexes were still of the abnormal type. The normal type of complex returned, however, as soon as the quinidin effect wore off. After the rhythm had been normal for 3 days, eighteen grains of quinidin failed to produce abnormal complexes. The patient was then given three grains of quinidin twice a day to prevent a return of fibrillation but without success. When fibrillation returned the ventricular rate was rapid and runs of abnormal complexes again occurred. These disappeared when quinidin was discontinued and could not be produced by the intravenous administration of one-fiftieth grain atropin, which greatly increased the ventricular rate.

In a second patient quinidin converted auricular fibrillation into auricular flutter, and runs of abnormal complexes bearing a definite and constant relation to the flutter waves occurred. A similar relation between the flutter waves and the abnormal complexes produced by quinidin may be seen in a curve published by Levy.[5]

We have given single twelve grain doses of quinidin to twenty patients with heart disease, but without arrhythmia. Several other patients of the same type have received larger doses. In none of these patients did either single ventricular extrasystoles or runs of ventricular extrasystoles occur. In many instances there was a slight increase in the ventricular rate; in four instances inverted T waves became less inverted or upright.

We attribute the majority of the abnormal ventricular complexes, produced by quinidin in auricular fibrillation, to a combination of two factors: depression of intraventricular conductivity, and, accelaration of the ventricular rate. We do not deny

[5] *Archiv. Int. Med.*, 1922, xxx, 474, Fig. 7.

that the drug may occasionally induce abnormal impulse formation in the ventricular muscle, but satisfactory proof that it does so is as yet wanting.

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Changes in the electrocardiogram following the arsphenamine treatment of cardiac and aortic syphilis.

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We have recently observed conspicuous changes in the electrocardiogram following the administration of arsphenamine in four cases of cardiac and aortic syphilis.

A patient with syphilitic myocarditis and complete right bundle branch block developed an abnormal idioventricular rhythm following the administration of two-tenths gram of arsphenamine and died a few days later. For several weeks preceding the treatment his condition had been stationary.

Two patients with syphilitic aortitis, but without definite signs of cardiac syphilis, and with practically normal electrocardiograms, developed diphasic complexes suggesting incomplete bundle branch block, following intensive arsphenamine therapy. In one of these patients the T-wave changes gradually disappeared but the QRS changes persisted. The other patient could not be followed. Similar but less conspicuous changes occurred in a third patient with syphilitic aortitis who showed great enlargement of the heart and left ventricular preponderance before treatment.

These observations indicate that the administration of aspenamine in cases of cardiac syphilis may sometimes be followed by myocardial changes of an undesirable kind. The slow development and persistence of the electrocardiographic changes suggest that they are not due to a local Herxheimer reaction, although this possibility cannot be excluded. The rapid destruction of the luetic lesions and their replacement by scar tissue with