7764 P

## Effect of Atropine on Impaired Auriculoventricular Conduction in Rheumatic Fever.

HOWARD G. BRUENN. (Introduced by Robert L. Levy.)

*From the Department of Medicine, College of Physicians and Surgeons, Columbia University, and the Medical Clinic, Presbyterian Hospital.*

That auriculoventricular conduction is frequently impaired in rheumatic fever is now well known. It is generally assumed that this disturbance is due to the presence of rheumatic lesions in the region of the auriculoventricular node or bundle.

Atropine sulphate, in amounts varying from 1.5 to 3.0 mg., was injected intravenously in 12 patients suffering from acute rheumatic fever. In each instance the electrocardiogram showed impairment of A-V conduction which varied in degree from lengthening of the P-R interval above 0.20 second to complete heart block. None of these patients had received digitalis or quinidine. A control electrocardiogram was taken, and further records were made at intervals of 1 to 4 minutes during the half hour following the injection. The maximum effect usually occurred during the first 15 minutes. The action of the drug always disappeared completely in the course of 24 hours, when the conduction time resumed its former level or occasionally one slightly above it. No undue subjective discomfort was induced, although dryness of the mouth was usual; pupillary dilatation and flushing of the skin sometimes were noted.

Eighteen observations were made on the 12 rheumatic subjects and 6 observations were taken on 3 individuals who showed no evidence of heart disease.

*Rheumatic Group.* In 9 of the 12 patients, shortening of the conduction time was noted after atropine injection. In one case of incomplete block, dropped beats disappeared and the P-R interval was 0.23 second in each cycle. An example is given in Table I. The

TABLE I

Female, aged 38. Fever, polyarthritis, mitral stenosis, and insufficiency. Leucocytes, 11,000. Sedimentation rate of erythrocytes, 130.

| Time | P-R Interval | Heart Rate |
|---|---|---|
| 10:43 A. M. | 0.24 | 90 |
| 10:47 | *2 mg. atropine, intravenously* | |
| 10:48 | 0.19 | 100 |
| 10:50 | 0.18 | 105 |
| 10:52 | 0.19 | 100 |
| 10:57 | 0.20 | 98 |
| 11:02 | 0.20 | 98 |
| 3:00 P. M. | 0.21 | 85 |

increase in cardiac rate varied from 5 to 50 beats per minute. The decrease in conduction time ranged from 0.03 to 0.08 second. There was no direct relationship between the degree of acceleration of rate and the extent of shortening of conduction. In one case, an increase in heart rate of 5 beats per minute was associated with a decrease of 0.06 second in conduction time. In another instance, an increase of 50 beats per minute was accompanied by a decrease of 0.04 second in conduction time.

Approximately half of these patients were receiving aspirin at the time the observations were made. In a number of cases atropine was injected first when no aspirin was being given and then again after its administration, in amounts up to 7.5 gm. daily. The decrease in the duration of conduction was of the same order of magnitude whether aspirin was being given or not. The increase in cardiac rate following the injection of atropine tended to be less when aspirin was being given than when it was omitted.

In 3 patients the degree of block was unaffected by atropine. Two of these were instances of complete heart block. Atropine produced a marked acceleration in auricular rate in both, but complete A-V dissociation persisted. In one of these patients complete block has been present constantly for 4 months since the atropine study was made. In the third case, prolongation of the P-R interval was not affected by atropine. It seems likely that in these cases there was permanent fibrosis of the junctional tissues as the result of rheumatic lesions.

Three cases, in addition to impairment of A-V conduction, showed a delay in intraventricular conduction time. Atropine had no effect on this disturbance.

*Normal Group.* In 6 patients without heart disease, injection of atropine was followed by an acceleration in rate varying from 20 to 50 beats per minute. The decrease in conduction time ranged from 0.01 to 0.03 second.

*Summary.* Impairment of A-V conduction in rheumatic fever often, though not invariably, can be diminished or abolished by atropine. Inasmuch as acceleration in rate and decrease in conduction time do not always parallel one another in degree or duration, it appears that these 2 effects are not necessarily directly related. The exact mechanism involved is a matter for speculation. It is conceivable either that the release of the vagus lowers the threshhold for conduction through the junctional tissues, possibly through some chemical action; or that the strength of the excitatory impulse is augmented to such an extent that it can pass an area which offers

increased resistance. Further observations along these lines are in progress.

## 7765 P

## Diastase in Milk.

MARIANNE KUTTNER AND MICHAEL SOMOGYI.

*From the Laboratory of the Jewish Hospital, St. Louis.*

Béchamp in 1883 was the first to recognize the presence of diastase in human milk; at the same time in cow's milk he found no trace of this enzyme.[1] Bouchut[2] and Moro[3–5] confirmed the findings of Béchamp.

During the last decade a number of investigators have attempted to establish a quantitative diastase test as a means of detecting whether or not a milk had been pasteurized. Namely, diastase would be entirely or partly inactivated during pasteurization, the extent of its destruction depending on the temperature and the duration of heating. The methods used by these workers, while claiming to yield quantitative results, are quite crude in comparison to the qualitative methods of Béchamp and Bouchut. The more recent workers find diastase in the milk of practically all the mammals[6] examined and are able to determine diastatic activity in the presence of lead[7–9] and even mercury salts.[10] The latter fact is characteristic of the unreliability of these methods.

We approached the problem with analytical procedures, which in the instance of blood and urine proved to be adequate for the determination of very low as well as of high diastase values.

The method is in brief as follows: a 1.5% starch paste is prepared of pure commercial corn- or rice-starch (but not of soluble starch); 10 cc. of this starch paste and 4 cc. of a 1% NaCl solution

1 Béchamp, A., *Compt. rend.*, 1883, **96**, 1508.

2 Bouchut, E., *Hygiène de la première enfance*, Paris, 1885, 102. (Quoted in Moro's work.)

3 Moro, E., *Jahrb. f. Kinderheilkunde*, 1898, **47**, 342.

4 Moro, E., *Jahrb. f. Kinderheilkunde*, 1900, **52**, 524.

5 Moro, E., *Jahrb. f. Kinderheilkunde*, 1902, **56**, 391.

6 Chrzaszcz, T., and Goralowna, C., *Biochem. Z.*, 1925, **166**, 172.

7 Rothenfusser, S., *Z. Untersuch. Lebensm.*, 1930, **60**, 94.

8 Gould, B. S., *J. Dairy Sci.*, 1932, **15**, 230.

9 Weinstein, P., *Z. Untersuch. Lebensm.*, 1934, **68**, 73.

10 Kluge, H., *Z. Untersuch. Lebensm.*, 1933, **65**, 71.