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Combined Effect of Ephedrin and Atropin on Complete Heart Block.

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The use of ephedrin in complete heart block was first reported by Miller and later by Stecher. But the combined action of ephedrin and atropin on the human heart according to Chen and Schmidt[1] has never been investigated.

A case of complete heart block without Adams-Stokes' attacks was available to study its reaction to ephedrin alone. The patient was kept quiet in bed and undisturbed. Electrocardiograms were taken before and at frequent intervals after the administration by mouth of ephedrin. Blood pressure readings were made at frequent intervals and usually before the electrocardiograms, from which both auricular and ventricular rates were obtained. Results are shown in Table I. It will be seen that after the administration of 150 mg.

TABLE I.

Effect of Ephedrin on Complete Heart Block.

(Ephedrin 0.09 gm. at 10:30 a.m., and 0.06 gm. at 10:50 a.m., a total of 0.15 gm. in 20 minutes.)

Time. a.m.	Vent. rate per min.	Aur. rate per min.	B. P. mm. Hg.
10:28	38	91	110/70
10:40	38	82	110/70
11:00	38	80	110/75
11:20	37	80	115/75
11:50	38	79	115/75
12:10	38	78	115/75

of ephedrin in 20 minutes, there was no increase in ventricular or auricular rate, but an increase of 5 mm. Hg. in systolic and diastolic blood pressure.

Different explanations have been offered for the negligible pressor effect occasionally encountered as in this case. Chen and Schmidt attributed the depressant effect of large doses of ephedrin either to direct depression of heart muscle, or to a difference in the quantity of "receptors" present in the individual. Kreitmair explained the difference on the basis that small doses of ephedrin stimulate the sympathetic nerve endings, while large doses stimulate the para-

[1] For literature, see references of Chen & Schmidt's review of Ephedrin and Related Substances in *Medicine*, 1930, **9**, 94.

sympathetic. However in animal experiments according to Fujii and Kreitmair this depressant action of ephedrin on the heart is not antagonized by atropin, while on the other hand Mehes and Kokas believed that the depressant effect was partly removed by atropin. To test the validity of these hypotheses in the clinical case, atropin was given after the patient had been ephedrinized. The results are shown in Table II. It will be seen that atropin hastens and intensi-

TABLE II.

Combined Effect of Ephedrin and Atropin on Complete Heart Block.

Time a.m.	Vent. rate per min.	Aur. rate per min.	B. P. mm. Hg.	Remarks
9:08	37	94	110/70	Before ephedrin
9:10				Ephedrin 0.09 gm.
9:30	37	77	110/70	
9:32				" 0.06 "
9:40	37	74	110/75	
10:00	38	69	115/80	
10:02				Atropin 0.0009 gm. hypodermically
10:05			150/100	
10:20	39	94	160/100	
10:45	40	115	160/95	
11:45	39	115	120/70	

fies the pressor effect but shortens the duration. Such combined action of ephedrin with atropin suggests that atropin neutralizes the parasympathetic effect that results either from the individual's vagotony or from the stimulating action of ephedrin on parasympathetic nerves. If the lack of pressor effect after therapeutic doses of ephedrin is due to either limited quantity of "receptors" or depressant action of ephedrin as suggested by Chen, then atropin must increase if not in amount at least in sensitivity these "receptors" or abolish the depressant action of ephedrin on human heart. Two individuals with normal hearts were studied and found to have the same reaction as the patient.

In order to exclude a possible abnormal reaction of this patient to atropin 2 mg. of atropin alone were given hypodermically. The results showed an increase of 30 beats in auricular rate, and a decrease in ventricular rate of 2 beats per minute. Both systolic and diastolic pressure showed a fall of 10 mm. Hg. at the height of the effect. Evidently this patient reacted to atropin as usual.