THE AURICULAR RHYTHM IN PATIENTS WITH THE MORGAGNI-ADAMS-STOKES SYNDROME BEFORE AND AFTER ELECTRICAL CARDIAC STIMULATION

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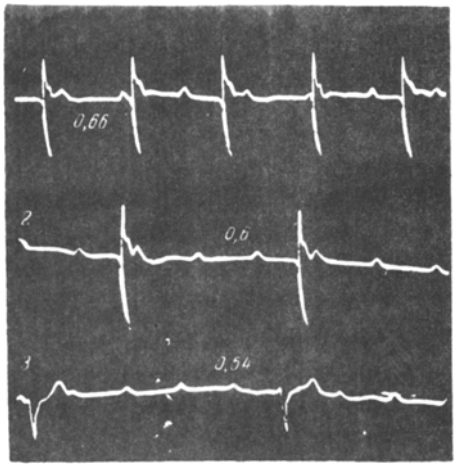
For the correction of a faulty slow rhythm in atrioventricular block, the method of electrical cardiac stimulation may be used; in cases of the Morgagni-Adams-Stokes syndrome it enables the characteristic attacks in which circulation is arrested to be suppressed. Electrical stimulation has been investigated many times; certain points concerning the frequency of change of auricular automatism during ventricular stimulation still remain unexplained.

In our clinic a study of cardiac electrical stimulation has been carried out since 1961. We have had under observation 17 patients with complete or incomplete atrio-ventricular block complicated by attacks of the Morgagni-Adams-Stokes syndrome. In these patients the ventricular rate varied from 18 to 44 contractions per minute; the auricular rate was from 66 to 90, and the highest rates occurred in cases of cardio-vascular insufficiency. In 10 patients we implanted myocardial electrodes and an artificial ventricular rhythm at 55-75/min was established.

In 10 of the patients we were able several times to record the time of onset and the development of the attack of the E.C.G. In two cases the ventricles failed to contract, in six there was a fibrillation or prefibrillar ventricular tachyarrhythmia, and in two cases there was fibrillation and failure of systole. In two patients, in the period preceding the attack the auricular rhythm rose from 60-80 to 110-125/min: in one the attack took the form of asystole, and in the other of ventricular fibrillation. Like other authors [1, 2, 3] we have observed an auricular tachycardia in the period of asystole, but it occurred only when the asystolic period lasted for 25 sec or more. Thus the longest asystole of 70 or 90 sec was recorded from the patient N. Initially the auricular contractions increased from 72 to 100/min, and then towards the end of the attack they fell to 48-46/min. In the same patient, during repeated periods of asystole lasting for 6-17 sec (on 6 occasions) the auricular rhythm showed no change. Unlike the authors to which we have referred, in our patients we found no pathological changes of the P wave; sometimes there was an auricular arrhythmia.

The Erlanger-Blackman phenomenon, which develops in complete atrio-ventricular block, is the effect whereby the P – P interval including the QRS-complex is a few tenths of a second shorter than the P – P interval, excluding the ventricular complex. The phenomenon occurs only when the atrioventricular node is the source of ventricular automatism; in the idioventricular rhythm the difference between the P – P intervals disappears. The effect is explained by supposing that in a ventricular contraction originating from the atrioventricular node the inhibitory influence of the vagi on the sinus bundle is temporarily absent. An injection of atropine eliminates the difference between the P – P intervals. We have observed the effect only in six patients in which the source of the ventricular automatism was the atrioventricular bundle. The difference in the P – P intervals was 0.06-0.09 sec. In three other patients with an idioventricular rhythm the Erlanger-Blackman phenomenon was not shown; the source of the ventricular automatism was to be found in various parts of the right and the left branch bundles; the ventricular complexes were extended in time and discoordinated. For example, in patient B the center of automatism was displaced from the atrioventricular bundle into the right bundle branch, and the effect under discussion was not present.

In patients in which prolonged electrical stimulation to the ventricles is maintained the ECG shows auricular waves which follow the innate rhythm, and ventricular complexes resembling right or left ventricular extrasystoles which followed the set rhythm (the electrodes were attached to the left or right ventricular myocardium). In two patients the P – P interval, including the artificially induced ventricular complex, was 0.06 sec shorter than the cor-



ECG of patient M during electrical stimulation of the ventricles at 67/min (1), and with an artificial rhythm of 34/min (2), and with a spontaneous rhythm of 24/min (3).

Duration of the P – P Interval for Different Ventricular Rhythms (in seconds)

Name of patient	Asystole	Rhythm: Spontaneous rhythm at 23-25/min	Rhythm: Artificial rhythm at 33-35/min	Rhythm: Artificial rhythm at 60-75/min
M-v.		0.54	0.56	0.6
		0.56	0.62	0.66
		0.58	0.60	0.69
Shch.	0.64	–	–	0.8
M.	0.56	–	–	0.68
K.	–	0.48	–	0.62
N.	0.66	–	–	0.84

Note. The patient M was studied three times, on: 2/15, 3/6, and 3/10.

responding interval without the ventricular complex. This effect differs from the Erlanger-Blackman phenomenon only in that here the role of spontaneous ventricular contraction is filled by the QRS-complexes elicited by the stimulator. Apparently the contraction of the ventricles influences the auricular rhythm quite independently of the way it originates.

During the course of the work we were required to switch off the stimulator (to measure the resistance of the heart, to change the batteries in the apparatus, etc.). Then in many cases in a patient with complete atrioventricular block a phenomenon occurred which we called suppression of the spontaneous automatism of the ventricles. The effect showed up as state of asystole, or a marked reduction in the rate of spontaneous activity of the ventricles, and it was brought on by sudden cessation of the artificial rhythm. To avoid this effect, before switching off the stimulator we reduced the rate of the artificial rhythm until spontaneous contractions occurred. It was found that when the stimulus rhythm was reduced the P–P interval was shortened, and consequently the auricular rhythm was accelerated; on the other hand stimulation at the optimal rate of 60-75/min led to a reduction of the auricular tachycardia, as observed before stimulation. We frequently observed this phenomenon in five patients during a short asystole of the ventricles, or during a reduction of the stimulus rate. Similar observations have been made by other authors [4, 5]. We must point out that the change of auricular automatism occurs simultaneously with an alteration in the frequency of the artificial rhythm of the ventricles. As in patient M the P – P interval at the spontaneous rhythm of 24/min was 0,54 sec, and at an artificial rhythm of 34/min it was 0.6 sec, while at an artificial rate of 67/min it was 0,66 sec (see Figure). The results for other patients are given in the Table.

The fact that the auricular rate of contraction is reduced by stimulation of the ventricles at a rate greater than the spontaneous may be used to enable the optimal stimulus frequency to be selected.

We must emphasize once more that the full phenomenon was only observed in patients with complete atrioventricular block: the rate of stimulation was (90/min, but the preceding spontaneous rhythm was 75/min. Then the P – P interval including the spontaneous complex was 0.8 sec, while the corresponding interval including the artificial complex was 0.72 sec; i.e., under the influence of a stimulus rhythm given at a higher rate than the spontaneous one the sinus automatism was increased and so raised the frequency of the auricular contractions.

The facts we have reported indicate that there is a direct relationship between auricular automatism and ventricular stimulation. The reason for the change of sinus automatism in ventricular stimulation at the optimal rhythm is to be found in the hemodynamic and metabolic changes related to changes of the minute volume of the heart, rate of blood flow, venous pressure, etc. The fact that this phenomenon occurs immediately indicates the reflex nature of the auricular automatism (enhanced by sympathetic innervation and by the Bainbridge reflex). These ideas, however, require experimental confirmation.

SUMMARY

Observations were made on 17 patients with complete or incomplete atrioventricular block complicated by attacks of the Morgagni-Adams-Stokes syndrome. In ten cases the attacks were recorded electrocardiographically. In two patients an enhanced rate of auricular contraction was observed to occur before the attack. During the attacks, in which ventricular asystole lasting for 25 sec or more occurred, the auricular contraction rate was at first increased, and then decreased and arrhythmic. The Erlanger-Blackman phenomenon was found in six patients in which the automatism was initiated in the atrioventricular node.

When the rate of electrical stimulation of the ventricles was increased to above the spontaneous rhythm the frequency of the auricular contractions was reduced. In our opinion these changes are reflex. The fact we have described, that a change of sinus automatism may be induced by electrical stimulation of the ventricles, may help in the choice of the optimal stimulus rhythm.

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