Stokes-Adams Attacks with Simultaneous Auricular and Ventricular Standstill

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This is a report of a case of Stokes-Adams seizures in a patient with partial auriculoventricular block. Simultaneous auricular and ventricular standstill in about half the seizures of which we have electrocardiographic record, and complete relief after atropine, leads us to think the seizures were due to vagal effect.

Since it was first described in the literature,1, 2 much has been written about the Stokes-Adams syndrome. The ventricular mechanism and the changes in auriculoventricular conduction have been investigated extensively. The usual mechanism of these attacks is prolonged ventricular asystole, or ventricular tachycardia, flutter or fibrillation of long duration.

Many observers have described similar attacks resulting from reflexes arising from the carotid sinus or from other sources, such as Brandenburger's three patients with Mikulicz's syndrome,5 Gluck's case of neoplasm of a bronchus causing pressure on the vagus,6 Faun and Klima's case of acute tonsillitis,7 Weiss and Ferris's patient with esophageal diverticulum8 and Scott and Sancetta's patient with Stokes-Adams syndrome who had these attacks when straining at stool or from digital irritation of the anal sphincter.9 In most of these instances there was total cardiac standstill during the attack without underlying heart block.

Such attacks have been termed the "neurogenic" Stokes-Adams syndrome.10 Many have considered them as merely syncope and not true Stokes-Adams attacks. Although lesions of the myocardium or temporary ischemia may have a definite influence upon the occurrence of such attacks, an additional neurogenic factor such as vagal stimulation may play an important role in their production.11 This factor seems not to have been emphasized sufficiently. The case to be reported shows the additive effect of vagal stimulation in the production of such an attack in a patient with arteriosclerotic heart disease and auriculoventricular and intraventricular block.

Case Report

A 75 year old white man was admitted to the hospital on May 14, 1953, complaining of attacks of unconsciousness of two or three minutes duration. For periods of approximately 20 seconds the patient's son, a physician, was unable to feel the pulse during these attacks. For three months he had as many as 20 to 50 attacks each day.

For 15 years he had had dizzy spells during sudden movement, for seven years dyspnea on exertion and in nocturnal paroxysms and for four years known hypertension. The family history was not significant. He was well developed and fairly well nourished. The blood pressure was 165/90; the pulse was 76 per minute. The palpable arteries were tortuous and sclerotic; the ocular fundi showed moderate arteriosclerosis. The heart was not enlarged and the rhythm was regular. There was a harsh systolic murmur of moderate intensity at the apex and a blowing systolic murmur at the aortic area. The prostate showed moderate benign hypertrophy. There were no other significant abnormalities.

Urinalysis was negative. The blood count showed erythrocytes 3,500,000, hemoglobin 9 Gm., leukocytes 8,000 with normal differential count. The erythrocyte sedimentation rate was 17 mm. in one hour (Cutler). Roentgenogram of the chest showed no cardiac enlargement. The electrocardiogram on admission (fig. 1 A) showed first degree A-V heart block and right bundle branch block; the duration of the P-R interval was 0.28 second, of the QRS complex 0.14 second. There was regular sinus mechanism and the rate was 64 per minute.

Shortly after admission seizures of two kinds began to occur; these recurred every 5 or 10 minutes. In the milder ones there were generalized muscular twizzings and flushing of the face, without impairment of consciousness. In the more severe seizures there was loss of consciousness with generalized clonic convulsions. Serial electrocardiograms were obtained. In addition to bundle branch block these

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records showed two types of conduction disturbance: (1) A-V block with ventricular asystole, the auricular rate increasing from 93 to 110 per minute during the asystole (fig. 1 B), and (2) simultaneous auricular and ventricular asystole (fig. 1 C). The latter occurred in about half the major attacks of which we have record. Loss of consciousness occurred when the ventricular asystole was prolonged. Immediately after the attacks the auricular rate returned to its initial level.

The duration of the longest ventricular asystole with normal auricular mechanism was six seconds. When auricular asystole was also present the longest duration was 5.6 seconds. Each auricular standstill was preceded by two or three ventricular complexes of incomplete 2:1 or 3:1 A-V block; in no instance did it follow ventricular asystole of longer duration. Except for these two or three cycles of high grade block, the basic mechanism preceding auricular asystole was first degree A-V block with long P-R intervals.

On the day of admission he had attacks every 5 or 10 minutes. Ephedrine sulfate was given in 25 mg. doses every three hours without benefit. (It was learned later that the drug had been used previously without effect.) Because of A-V block and frequent auricular standstill it occurred to us that vagal stimulation might be the cause of the attacks. Accordingly, atropine sulfate, 0.9 mg., every six hours, was ordered. The attacks ceased before the second dose was given and did not recur before he left the hospital 60 hours later. Urinary retention developed the second day after atropine was begun.

After leaving the hospital he was not under our observation. It was reported that he had used varying amounts of atropine. The attacks were relieved when the doses were sufficiently large and recurred on smaller doses. Six weeks after discharge from the hospital suprapubic cystostomy was done elsewhere. Following this he is said to have had atropine sulfate, 0.9 mg. three times daily, with complete relief from seizures.

On September 25, when he was seen last, it was found that he had gradually reduced the atropine to 0.9 mg. once daily and had experienced no seizure between June 27 and that date. At that time an electrocardiogram showed complete A-V block (fig. 1 D).

Discussion

In the case reported here ventricular standstill was the cause of Stokes-Adams attacks. The auricular rate increased during ventricular standstill, as reported in similar cases.12-15

In our case, in approximately half of the
seizures of which we have electrocardiographic record, standstill of auricles and ventricles occurred simultaneously. Heretofore, 20 seconds has been the shortest recorded duration of ventricular standstill producing auricular asystole.\textsuperscript{4} Shorter duration of ventricular standstill is usually followed by acceleration of the auricular rate. For this reason it would seem that auricular asystole occurring, as it did in our case, without the asphyctic effect of prolonged ventricular standstill, must be explained by vagal effect. This hypothesis is supported by the fact that before each episode of complete asystole there was a brief period of 2:1 or 3:1 A-V block. The idea is supported further by relief of the seizures by atropine. It is thought that our case should not be included among the rare instances of Stokes-Adams attacks in which atropine has been effective by unexplained mechanism.\textsuperscript{15}

It is notable that during the several months of our observation the electrocardiogram has changed to permanent complete auriculo-ventricular block. It seems likely that the development of an idioventricular mechanism has stabilized the rhythm and is a factor in freedom from attacks on smaller doses of atropine such as the patient was receiving.

This patient with arteriosclerotic heart disease and conduction disturbances and with Stokes-Adams attacks apparently had suffered myocardial damage and was repeatedly under exaggerated vagal effect which at times produced ventricular standstill with accelerated auricular rate and, with about equal frequency, simultaneous auricular and ventricular standstill.

**Summary**

1. A patient with Stokes-Adams attacks has been reported in whom simultaneous auricular and ventricular standstill has been shown to be the underlying disturbance of mechanism in about half of the attacks of which we have electrocardiographic record.

2. We have not been able to find elsewhere the record of auricular standstill in Stokes-Adams attacks unless it was preceded by prolonged ventricular asystole.

3. Atropine abolished the attacks completely and vagal stimulation is believed to have played a major role in their production.

4. The development of complete A-V block is thought to have stabilized the mechanism, enabling the attacks to be abolished by a smaller amount of atropine.

**Sumario Español**

1. Se ha reportado un paciente con ataques de Stokes-Adams en el cual pausa auricular y ventricular demostró ser el disturbo en el mecanismo en aproximadamente la mitad de los ataques en los cuales se obtuvieron trazados electrocardiográficos.

2. No hemos podido encontrar en ningún sitio trazados de pausa auricular en ataques de Stokes-Adams a menos que no fuera precedida por una prolongada asistolia ventricular.

3. La atropina abolió los ataques completamente y el estímulo vagal se cree juegue un papel importante en su producción.

4. El desarrollo de bloqueo A-V completo se cree que establece el mecanismo, permitiendo que los ataques puedan ser abolidos por una cantidad menor de atropina.

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