Stokes-Adams Attacks Induced by Rectal Stimulation in a Patient with Complete Heart Block

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This paper reports a case of complete heart block whose Stokes-Adams attacks were induced by straining at stool and invariably by digital stimulation of the rectum. Electrocardiograms during attacks exhibited high ventricular tachycardia or fibrillation and chaotic heart action.

In 1941, Parkinson, Papp and Evans reported their observations on the electrocardiogram of the Stokes-Adams attack. Contrary to the prevailing view at the time that ventricular standstill is the only common disturbance in the cardiac mechanism which, occurring in patients with complete heart block, causes the loss of consciousness, they found that in one-third of the reported cases, including 8 of their own, unconsciousness was ushered in by a high ventricular tachycardia and/or fibrillation. Following is the report of such a case observed during spontaneous attacks and in whom the attacks were often induced by straining at stool and always by rectal stimulation with the gloved finger.

Report of Case

A white woman aged 62 was admitted to the hospital on August 3, 1944, and died during sleep nine days later. She had been in good health until two weeks prior to admission, when she suddenly collapsed and remained unconscious for several minutes. Many attacks of faintness with a few followed by complete loss of consciousness occurred daily until death.

Physical examination as well as the usual laboratory procedures were within the limits of normal other than the cardiovascular system. Between attacks the patient did not appear ill. The ocular fundi were the seat of grade 1 sclerosis. The heart sounds were normal and the rate about 42 with a slight irregularity in rhythm. The peripheral vessels were sclerotic, and the patient's general demeanor suggested some degree of cerebral arteriosclerosis. The blood pressure was 140/85. The electrocardiogram on admission showed almost complete auriculo-ventricular block, auricular rate 104, ventricular rate 52 (fig. 1).

Attacks of complete unconsciousness would be ushered in by a sudden irregularity and increase in rate of the pulse. The patient would then complain of shortness of breath and faintness. Suddenly the radial pulse would disappear and the heart sounds become inaudible. Breathing became stertorous, there were clonic convulsions together with dilatation of the pupils, followed by twitching of face muscles and spasmodic movements of the arms and legs. The face became cyanotic. The entire episode varied in duration from one-half to three minutes. With a return of the pulse, the attack ended abruptly, and the patient appeared little the worse for the experience.

After several days the patient observed that most of her attacks occurred while using the bedpan. To check this observation, a finger was inserted into the anal orifice and within a few seconds the patient became unconscious, exhibiting the picture just described. This attack lasted approximately two minutes.

The patient was moved to the electrocardiograph laboratory and a control record (fig. 2) was taken, which showed almost complete auriculoventricular block, auricular rate 92, ventricular rate variable, with ventricular beats multifocal in origin. In spite of the disturbance in the cardiac mechanism exhibited in figure 2, the patient during this time was conscious and unaware of the cardiac disorder.

A finger was now inserted into the anal orifice and the pulse stopped, but instead of ventricular standstill as we had anticipated, there ensued a rapid ventricular activity suggestive of an impure form of ventricular tachycardia or a coarse ventricular fibrillation with a ventricular rate in some instances over 300 per minute, as shown in strip 3 of figure 3. The onset and offset of a spontaneous attack associated with a brief period of unconsciousness is shown in figure 4.

After this observation was made, ephedrine was discontinued and quinidine started. Although spontaneous Stokes-Adams attacks lessened somewhat

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FIG. 1. Admission electrocardiogram showing 3 standard limb leads. Auricular rate is fairly constant at 104, ventricular rate is slightly inconstant at approximately 52. Record is interpreted as representing almost complete heart block, with the large, broad QRS complexes representing the dominant idioventricular pacemaker. The second QRS in Lead I is probably conducted. The second QRS in Lead III is nodal with retrograde P. Patient was asymptomatic at this time.

in severity and duration, the patient died 12 hours later.

At postmortem examination the heart weighed 300 grams. Grossly there was only slight coronary artery sclerosis and no areas of fibrosis were seen. On microscopic examination of the region of the common bundle a few foci of round cell infiltration were present. There was slight atrophy, fibrosis and vacuolization of the myocardial cells, and moderate arteriolar sclerosis, the latter commensurate with similar arteriolar changes in the other organs. We did not attempt to study serial sections of the entire bundle region. Permission was not granted to examine the brain.

DISCUSSION

Since high ventricular tachycardia and ventricular fibrillation are clinically indistinguishable from cardiac standstill, the electrocardio-

FIG. 2. Consecutive recordings of standard Lead II again demonstrating almost complete heart block and also chaotic heart action. There is a sinus rhythm, rate 92, as demonstrated by the regular appearance of P waves. The second beat in strip 5 is probably normally conducted. Beats such as the fourth in strip 1 and the first in strip 3 are interpreted as conducted with intermittent intraventricular block. Otherwise most of the beats are idioventricular and multifocal. In strip 4 there is a short burst of ventricular fibrillation. Again during this period the patient felt well and was not aware of a cardiac disorder. (A portion of this record has been reproduced in Katz, L.: Electrocardiography, ed. 2, Philadelphia, Lea & Febiger, 1946, p. 713.)
gram recorded during the Stokes-Adams seizure is the sole means of determining the exact disturbance in the cardiac mechanism respon-

cible for the seizure. Schwartz\(^2\) has studied extensively the problem of transient ventricular fibrillation, and offers evidence that a careful

_**Fig. 3.** Continuation of record shown in figure 2, standard limb Lead II. Two and two-tenths seconds following rectal dilatation there appears a multiform mechanism interpreted as ventricular tachycardia or coarse fibrillation. The characteristic sequence of events followed (see text) and within 9.2 seconds the patient became totally unconscious. Strips 1 and 2 are consecutively recorded; strip 3 was recorded later in the attack and represents ventricular tachycardia with a ventricular rate at times slightly over 300. Fifteen seconds following the last beat shown in strip 3 the attack ceased and there was a return of chaotic heart action interspersed with periods of almost complete block and near regularity (as in fig. 1). Consciousness returned within 2 or 3 seconds following cessation of fibrillation-tachycardia. Although P waves cannot be seen, regularly spaced a waves were noted in the jugular vein, indicating continuation of sinus discharge. This entire attack lasted 57.8 seconds and was reproduced repeatedly by the same maneuver. (A portion of this record has been reproduced in Katz, L.: Electrocardiography, ed. 2, Philadelphia, Lea & Febiger, 1946, p. 713)._}

_**Fig. 4.** Onset and offset of spontaneous episode of paroxysmal rapid ventricular action, continuous recording, standard limb Lead II. Total duration 11.8 seconds, patient becoming briefly unconscious towards the end. The patient's subjective complaints of faintness and apprehension just prior to loss of consciousness were correlated with the first few seconds of rapid ventricular activity and clinical asystole.

clinical study of the patient may aid in differen-
tiating between syncope due to sudden ventricular asystole or an excessively slow and irregular idioventricular pacemaker on the one hand, and paroxysmal ventricular action on the other. To know whether the syncopal attack is due to cardiac standstill or to a high ventricular tachycardia or fibrillation is obviously important from a therapeutic standpoint.

The frequent attacks of syncope caused by straining at stool and the constant induction of attacks by gentle digital stimulation of the rectum force one to consider the possible nervous path by which impulses from the rectum may reach the heart. It was suggested to us by Professor Albert Kuntz\(^3\) of St. Louis University that through an intersegmental viscerovisceral reflex, the stimulation from distention of rectal or pararectal sympathetic endings might transmit impulses into the spinal cord, and via the cardiac accelerator nerves increase sufficiently the irritability of a ventricular focus as to
cause a paroxysm of rapid ventricular action as was observed in our patient during a Stokes-Adams seizure.

Our survey of the literature has not disclosed a similar case. Wilson\textsuperscript{4} mentions an instance of a 31 year old white man subject to numerous episodes of rapid ventricular action and syncope precipitated by exertion. He suffered one episode while straining on the bedpan, but a tracing was not made at this time. Schwartz\textsuperscript{5} refers to a patient subject to seizures of "transient ventricular fibrillation following the type of exertion associated with straining at stool or when walking rapidly up an incline". The attacks in our patient however were uniformly induced by simple insertion of the gloved finger into the rectum.

**Summary**

A case of auriculoventricular block with Stokes-Adams attacks is presented. Electrocardiograms taken during attacks showed high ventricular tachycardia and fibrillation. The case is unique in that syncopal attacks were often caused by straining at stool and unfailingly induced by gentle digital stimulation.

**REFERENCES**

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