Stokes-Adams Seizures

Case Report of "Paroxysmal Ventricular Standstill" and Its Production by Carotid Sinus Pressure

By Ford K. Hick, M.D.

A patient presenting syncope and convulsions was shown to have spontaneous attacks of ventricular standstill without preceding or subsequent partial heart block and with the auricles beating regularly throughout. Carotid sinus pressure reproduced one such attack. This is a demonstration of nervous control in the heart below the auriculoventricular node and perhaps in the ventricle itself. Cookson's term "paroxysmal ventricular standstill" is uniquely fitting. The presence of right bundle branch block raises the question of a lesion affecting both the left and right branches of the bundle of His.

This case report presents the electrocardiographic findings in a man suffering many spontaneous periods of ventricular standstill which could be reproduced by carotid sinus pressure. Between the attacks there was no partial heart block. As his Electrocardiogram showed right bundle branch block, there may have been disease of both branches of the bundle of His.

Case Report

A man, 63 years old, entered West Suburban Hospital (44239) on Nov. 26, 1962, as a patient of Dr. Bertha Fisher. For one week he had had numerous fainting spells and had fallen several times but had not hurt himself. He had continued working until admitted to the hospital. In the 24 hours before entrance, several attacks had occurred while he was at his office. Until this complaint started, he was well and active, walking several blocks without dyspnea or chest pain. He had no knowledge of previous heart disease.

Soon after admission, he was observed in a generalized convulsion in which he developed cyanosis. He denied any aura. The attacks came repeatedly, every few minutes. The radial pulse and heart tones disappeared before each convulsion and often disappeared for up to 10 seconds with no subsequent convulsion, in fact with no subjective sensation whatsoever. The attacks were not observed to be related to eating, straining at stool or posture. They appeared during sleep as well as while awake.

The patient was a small, slender man of 63; he was alert and cooperative. He did not appear to be sick save for the convulsions. The pulse rate was usually 88, blood pressure 170/80. He had no fever. There was no evidence of cardiac enlargement. No murmurs were heard. There were no findings of congestive heart failure. Neurologic examination added nothing. An initial white cell count was 13.600 per cubic millimeter; this fell to 11.000 in three days. Later a chest x-ray film showed the heart to be of normal size and contour.

The electrocardiogram showed a sinus rhythm with right bundle-branch block. A-V conduction time was 0.18 second. No evidence could be found of a fresh myocardial infarct. Figure 1 A, B, and C show the phenomenon of ventricular standstill and its termination, sometimes with reappearance of the usual ventricular complex or a ventricular ectopic beat, or ending in a convulsion, if asystole lasted 11 seconds. Note that the P-R interval was normal immediately before and after the period of asystole.

The patient was given atropine sulfate, 0.8 mg., subcutaneously at four-hour and later six-hour intervals. This appeared to reduce the frequency of the attacks. On December 3, the convulsive attacks ceased and the pulseless periods were less frequent and brief, their duration not exceeding five seconds. He was allowed progressively more activity and discharged on December 14 with the continuance of oral atropine. He had no more convulsions but had a few brief periods of faintness. He has since moved away and has not been under medical supervision. He is still living.

While on atropine and having frequent periods of spontaneous ventricular standstill carotid sinus pressure was attempted once with no effect on the rhythm of the ventricle. On Jan. 5, 1953, about three hours after his regular oral dose of atropine (0.4 mg.), an electrocardiogram still showed normal P-R interval and right bundle-branch block. At this time right carotid sinus pressure induced the ven-
Fig. 1. (A) This section of lead III shows two short periods of ventricular standstill. Note the normal P-R interval before and after the pause. (B) Continuous record showing a spontaneous ventricular standstill ending in a convulsion. (C) Ventricular standstill is shown terminating after a ventricular ectopic beat. Note P-R intervals are normal immediately before and after the asystole (lead III). (D) Continuous record (lead III) made when carotid sinus pressure was applied (arrow) showing slowing of the auricles and ventricular standstill of 10 seconds duration. (E) Lead I showing appearance of an idioventricular rhythm during ventricular standstill. This occurred after adrenalin. The different pattern of the QRS complexes is perhaps significant.

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Discussion

The report is believed warranted because of the rarity of evidence of responses of the ventricles to nerve impulses and because of the rarity of ventricular standstill without preceding or subsequent partial A-V block. Cookson reported three cases of "paroxysmal..."
ventricular standstill” to which he gave this term. Two of his cases had normal rhythm and conduction immediately before and following the standstill. In two of his cases the attacks could be precipitated by various procedures including carotid sinus pressure. Schwartz and Eichna\(^3\) presented records in which carotid sinus pressure produced ventricular standstill lasting long enough to induce a convulsion while the auricle kept beating. A-V conduction was initially normal, but the patient later developed heart block. Scott and Sancetta\(^4\) described the case of a woman with complete heart block having similar convulsive episodes induced by straining at stool and reproduced by digital rectal examination. These attacks proved to be high ventricular tachycardia and fibrillation. That reflexes of visceral origin have an effect on the bundle of His or on the ventricular muscle can be inferred from the case of Scott and Sancetta and from the one here reported. The possibility exists that such reflex effects may cause sudden death. Perhaps hearts susceptible to such exaggerated reflex responsiveness were not normal and the abnormalities present may have rendered them particularly susceptible to such reflexes.

Perhaps our patient really has disease of both branches of the bundle of His. Figure 1E shows the pattern of altered intraventricular conduction during idioventricular rhythm. This tracing was made early in his illness after the administration of adrenaline, 0.5 cc. subcutaneously. The pattern of the ventricular complex is quite different at the slow rate. Hein and Sanazaro\(^5\) reported a case of left bundle-branch block which consistently reverted to normal intraventricular conduction when slowed by carotid sinus pressure. They reviewed the literature on vagus action on intraventricular conduction.

**Summary**

A patient presenting syncope and convulsions was shown to have spontaneous attacks of ventricular standstill without preceding or subsequent partial heart block and with the auricles beating regularly throughout. Carotid sinus pressure reproduced one such attack. This is a demonstration of nervous control in the heart below the auriculoventricular node and perhaps in the ventricle itself. Cookson’s term “paroxysmal ventricular standstill” is uniquely fitting. The presence of right bundle branch block raises the question of a lesion affecting both the left and right branches of the bundle of His.

**Sumario Español**

Un paciente presentando el cuadro de síncope y convulsiones demostró tener ataques espontáneos de pausa ventricular sin ser precedidos o seguidos de bloqueo parcial cardíaco y con los aurículos contraíéndose regularmente durante el ataque. Esto es una demostración de control nervioso en el corazón inferior al nódulo atrioventricular y quizás en el ventrículo propio. El término de Cookson “pausa paroxística ventricular” es singularmente apropiado. La presencia de un bloqueo de ramo derecho suscita la posibilidad de una lesión lesión afectando ambos ramos izquierdo y derecho del haz de His.

**References**

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