Complete Heart Block with Stokes Adams Attack from Transient Ventricular Fibrillation

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SUMMARY

A patient with complete heart block is described who developed a Stokes Adams attack due to transient ventricular tachycardia and fibrillation. The probable mechanism of the ventricular arrhythmia is discussed.

Additional Indexing Words:
Arrhythmia Syncope Cardiac arrest

STOKES Adams attacks with loss of consciousness due to a sudden decline in cardiac output usually occurs in patients with complete heart block. The precipitating event is ventricular asystole, ventricular flutter-fibrillation or a combination of both arrhythmias. Ventricular fibrillation may follow a period of asystole, or ventricular fibrillation may precede a period of asystole. Infrequently the Stokes Adams attack is due exclusively to ventricular fibrillation. Transient ventricular fibrillation was recorded electrocardiographically in a patient with complete heart block and is the subject of this report.

CASE REPORT

A 78-year-old white male was admitted to the Bronx Veterans Administration Hospital because of shortness of breath and swelling of the legs of 2 weeks duration. On the morning of admission, the patient experienced for the first time a brief episode of dizziness. Prior to the current admission he was completely asymptomatic, and was not aware of any cardiac abnormality. At this time the blood pressure was 200/90 mm.Hg, pulse rate 45/min. and regular. The neck veins were moderately distended. Examination of the lungs revealed rales at both bases. The heart was enlarged with the point of maximal impulse at the 6th intercostal space. A grade III/VI systolic ejection murmur was heard at the aortic area and radiated to the neck. There was no palpable thrill and the aortic component of the second

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sound was well preserved. The liver was not palpable and there was 2+-bilateral pretibial edema. The admission electrocardiogram revealed second degree heart block with a 5:2 ventricular response. There was no evidence of ventricular hypertrophy or a myocardial infarction. A chest roentgenogram revealed a moderate degree of cardiomegaly and mild pulmonary congestion. The urinalysis, complete blood count, blood urea nitrogen, SGOT and SGPT were within normal limits.

The clinical impression was that the murmur was of little hemodynamic significance, and the diagnosis of anatomic bilateral bundle branch block was entertained. The patient's heart failure responded to one injection of 2 ml of Mercuhydrin. Over the ensuing 4 days the rhythm was very unstable alternating between complete heart block and second degree heart block with an atrial rate of 90 and a ventricular rate of 45. The electrocardiogram did not reveal changes compatible with an acute myocardial infarction and the serum enzymes were never elevated. On the 5th day, the patient suddenly had a Stokes Adams attack. The electrocardiogram taken just prior and during this episode revealed complete heart block followed by ventricular tachycardia, flutter and fibrillation. Spontaneous return to complete heart block was then observed (Fig. 1). The patient recovered immediately. A temporary and then a permanent pacemaker was inserted in order to prevent future seizures. When the cardiac rate was increased the systolic basal murmur was barely audible.

The patient was subsequently discharged, and remains well 2 months after the pacemaker insertion.

Fig. 1.
DISCUSSION

The underlying lesion in the great majority of patients with the Stokes Adams syndrome is anatomic bilateral bundle branch block due to degenerative fibrosis of both bundle branches or less often the bifurcation. These degenerative changes are believed to be due to the cumulative effects of mechanical wear and tear on these structures, for they increase with age and occur earlier and more severely in the presence of hypertension. They do not appear to be related to ischemia.\(^2\)

In our patient, this degenerative change was the most probable cardiac lesion. Coronary artery disease was of little consequence in view of the lack of historical, laboratory or electrocardiographic evidence to support this diagnosis.

Schwartz and Schwartz\(^3\) have recently analyzed 250 cases of Adams Stokes syndrome. They observed that most Adams Stokes seizures occur in patients with non-surgically acquired complete heart block. In approximately two thirds of the patients, the cardiac mechanism underlying the syncopal attack is slowing or standstill of the ventricles in the presence of persistent atrial activity. In about one-fifth of the patients, transient ventricular fibrillation is the cardiac mechanism responsible for the attacks.

Ventricular premature beats are the usual forerunner of ventricular fibrillation. With the slow heart rate of complete heart block, the formation of ventricular ectopic foci are favored. If a ventricular premature contraction falls into the vulnerable phase of the previous beat, ventricular tachycardia or fibrillation can ensue. Fortunately most attacks of ventricular tachycardia or fibrillation preceded by complete heart block, revert back to heart block within a few minutes. This was well documented in our patient. However, if ventricular fibrillation occurs in a patient who was previously in sinus rhythm, then spontaneous reversion to sinus rhythm is distinctly unusual.

REFERENCES