PERIODIC ASYSTOLE FOLLOWING MYOCARDIAL INFARCTION AND CARDIAC DEPRESSANT DRUGS

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The main use of quinidine and of procaine amide lies in the treatment of cardiac arrhythmias. As their action is depressant they are potentially dangerous. The following case showed an unusual type of arrhythmia, which developed after myocardial infarction and the administration of these agents.

Case Report

A man, aged 74 years, was admitted to hospital as an emergency after an attack of praecordial pain lasting 12 hours. He had had two similar episodes in the preceding two weeks.

On admission the pulse rate was 180 a minute and regular, the blood pressure was 90/70. The jugular venous pressure was raised but there was no oedema. The heart sounds were soft and an apical triple rhythm was present. An electrocardiogram showed a posterior myocardial infarct pattern and also an arrhythmia interpreted as ventricular tachycardia (Fig. 1). Anticoagulant therapy was started with intravenous dextran sulphate and phenindione by mouth. A single dose of quinidine sulphate, 0·3 g., was given orally; two hours later the pulse was irregular and the electrocardiogram showed irregularly occurring periods of asystole lasting one second (Fig. 2). No further quinidine was given, but as the tachycardia and hypotension were persisting and the condition of the patient was grave, a further attempt to restore normal rhythm was considered advisable. Accordingly, procaine amide, 250 mg. two-hourly by mouth, was substituted. However, repeated electrocardiographic control showed that the periods of asystole were recurring.

After four doses of procaine amide (total 1 g.) the radial pulse rate was noted to have fallen to 20 a minute. The general condition remained unchanged but the heart sounds were irregular and scarcely audible. The electrocardiogram showed that periods of asystole were recurring regularly every three seconds and lasting for one second each (Fig. 3). Between each of the asystolic periods there were five or six complexes, the first of which was always preceded by a P wave. The first beat of this cycle represented the pulse that was felt at the wrist and was responsible for the rate of 20 a minute. Careful observation revealed that the asystolic intervals were coincident with expiration. The P wave recorded at the beginning of the cycle coincided approximately with the beginning of inspiration. The procaine amide was stopped immediately these features were noted. The use of atropine was considered, but in view of the previous tachycardia this was not given. The regularly occurring asystolic periods continued for about eleven hours, after which there was a spontaneous reversion to normal rhythm interspersed with ventricular ectopic beats.

The patient's condition improved but three days later he again had a paroxysm of ventricular tachycardia. Under electrocardiographic control, intravenous procaine amide was administered and after 400 mg. had been given normal rhythm abruptly returned. Oral procaine amide 250 mg. six-hourly, was given for one week, and 125 mg. six-hourly for a further five days.

Five days after admission there was slight sacral and ankle oedema, which responded rapidly to mersalyl and chlorothiazide. Ten days later, while he was still on procaine amide, 125 mg. six-hourly, a further episode of tachycardia occurred, this time found to be supraventricular; carotid
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Fig. 1.—Electrocardiogram recorded at the time of admission, indicating posterior myocardial infarction and also auricular flutter with a 2:1 response or ventricular tachycardia.

Fig. 2.—Lead II, showing a period of asystole lasting 1.4 second.

Fig. 3.—Lead II, showing periods of asystole lasting 1.4 second and occurring at regular intervals.
sinus pressure reduced the rate abruptly by half and a cardiogram showed auricular flutter with 2 to 1 heart block. Release of pressure caused the tachycardia to resume. Digoxin, 0.5 mg. six-hourly by mouth, was given and the tachycardia settled down during the next 48 hours to 80 a minute. Auricular flutter persisted throughout the stay in hospital.

On a further occasion, carotid sinus pressure was applied in an attempt to convert the auricular flutter to normal rhythm. The right carotid sinus was compressed under electrocardiographic control and after five seconds ventricular standstill suddenly developed. Pressure was at once released and after six seconds ventricular activity returned. The auricular flutter persisted and was unaffected by the carotid sinus pressure.

This patient was discharged from hospital five weeks after admission. Out-patient follow-up for five weeks showed that the auricular flutter persisted.

Five weeks after discharge from hospital he suddenly fell in the street, fracturing his skull. He died a few hours later. At necropsy considerable cardiac enlargement was found. There was fibrosis of both ventricles and a recent infarct in the right ventricle. In the right coronary artery there was a brown organizing thrombus 2–3 cm. in length. The left coronary artery was atheromatous.

Discussion

Cardiac arrhythmia following myocardial infarction is a grave sign. Both quinidine and procaine amide are used to restore normal rhythm and both may cause myocardial depression of such degree that ventricular failure may occur, due to either asystole or fibrillation. Intravenous procaine amide is particularly dangerous in this respect (Epstein, 1953). Cookson (1952) reviewed the published work on ventricular standstill as a cause of Stokes-Adams attacks and reported three cases he had observed. In cases of this type and in some healthy subjects, altered A-V conduction can be induced by carotid sinus stimulation (Holmes and Weill, 1945; Hick, 1954). In those with already damaged conducting tissue, the ventricle may be so depressed by such stimulation that fibrillation or standstill follows.

The present case exhibited a particularly unusual arrhythmia in which three main factors seem to have been involved. First, the septal damage was presumably the primary cause of the arrhythmia. Secondly, the administration of quinidine and procaine amide caused sufficient myocardial depression to produce periods of complete cardiac asystole. Thirdly, the increased vagal tone occurring regularly during expiration was responsible for the regular pattern of the arrhythmia, which may be called periodic asystole.

Summary

A case of myocardial infarction complicated by ventricular tachycardia has been described. Following the administration of quinidine sulphate and procaine amide, an unusual arrhythmia occurred, which has been called periodic asystole, and which was found to be associated with the phase of respiration. This arrhythmia has been attributed to the action of the cardiac depressant drugs used, and to the increased vagal activity occurring during expiration.

I wish to thank Professor G. M. Wilson, under whose care the patient was admitted, for permission to publish this report and for helpful advice and criticism in its preparation; and Mr. H. Blacow Yates for the details of the ultimate fatal termination.

References

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Br Heart J 1960 22: 734-736
doi: 10.1136/hrt.22.5.734

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