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AURICULAR FIBRILLATION CONVERTED BY QUINIDINE AFTER FAILURE WITH DIRECT CURRENT DEFIBRILLATION

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The d.c. defibrillator offers a simple and effective method of converting patients with chronic atrial fibrillation to normal sinus rhythm. Previous reports have shown the procedure to be successful in about 90 per cent of the cases. It is quick and easy and produces relatively little discomfort to the patient.[1]

Cardioversion by the use of electric countershock is more often successful and much less time consuming than conversion with large doses of quinidine. We have been able to convert electrically a number of quinidine failures with the use of the d.c. defibrillator. This has been reported by others.[2, 3, 4]

We recently had a patient whom we could not convert with the d.c. defibrillator, using up to 400 watt/sec.[5] The day after the failure of attempted defibrillation, the patient was placed on quinidine in rather moderate doses. In two days the patient converted to normal sinus rhythm.

SUMMARY OF CASE

The patient is a 46-year-old white male, who appeared to be in excellent physical condition. During a visit for routine checkup in the spring of 1964 it was noted that he had an irregular heart beat, which proved to be atrial fibrillation. Rather large doses of digitalis failed to keep the ventricular rate below 100 per min.

He was hospitalized with the intent of cardioversion by the use of electric countershock. His only complaints were slight fatigue and episodes of profuse perspiration. There was no history of rheumatic fever. He had a history of murmur for the first 18 months of life. His only previous hospitalization was for herniated nucleus pulposus, for which he was treated successfully by traction and bedrest. He had hepatitis in 1943 with an uneventful recovery. Family and social history were noncontributory.

Physical examination disclosed a robust individual 6 ft., 2 in. tall, weighing 250 lb. The blood pressure was 170/102. The heart rate was grossly irregular at a rate of 110 per min. There were no audible murmurs. The heart was not clinically enlarged. A2 was slightly greater than P2. The remainder of the physical examination was normal.

Laboratory work, including PBI, was normal. The x-ray of the chest showed normal heart size and a healed Ghon complex. The EKG showed atrial fibrillation and digitalis effect.

The patient was thought to have atrial disease on the basis of hypertensive and

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arteriosclerotic heart disease. He was placed on anticoagulants and scheduled for d.c. defibrillation. Quinidine (3 grains) every 4 hr. was started the evening before defibrillation. During his hospital stay he had been receiving digoxin (0.5 mg) once a day. This was not given on the day of defibrillation. He was anesthetized with sodium Pentothal after premedication with Nembutal (100 mg) and Demerol (100 mg). The first attempt at defibrillation at 250 watt/second was unsuccessful. A second attempt at 300 watt/second was unsuccessful. Because of the patient's size, extra care was taken in application of the electrode jelly. A final attempt at 400 watt/sec. was nevertheless unsuccessful.

The following morning, November 21, 1964, the patient was given Lanoxin (0.75 mg) daily and quinidine (0.4 gm) every 2 hr. for five doses. On November 22, 1964, he also received 0.4 gm of quinidine at 8 a.m. and 10 a.m. before the dosage schedule could be changed. At 11 a.m. he was noted to have an irregular rhythm. He was then placed on 0.6 gm of quinidine at noon, 2 p.m. and 4 p.m. He was reexamined at 6 p.m. and was in normal sinus rhythm. This finding was corroborated by EKG. Careful auscultation at this time disclosed a rough grade II to VI systolic murmur at the apex transmitted to the anterior axillary line. The patient was given Quinaglute b.i.d. and digoxin (0.75 mg) daily, and a regular sinus rhythm has persisted to the present time.

DISCUSSION

Ninety to 95 per cent of individuals with chronic atrial fibrillation are effectively reverted by the use of synchronized d.c. precordial shock. The use of quinidine takes longer, is associated with toxic reactions and a 20 per cent risk of fatal outcome and is not nearly so effective as electrical methods. We felt that our patient, who had a therapeutic classification of I-A and a short duration of his arrhythmia, was an ideal candidate for conversion from auricular fibrillation to sinus rhythm. We were quite surprised to find that electrical conversion failed whereas rather small doses of quinidine were quite effective in converting to regular sinus rhythm and maintaining this rhythm. Before his conversion he was noted to have an irregular rhythm on all examinations during the six month period that the arrhythmia existed. Subsequent to conversion, he has been in normal sinus on all examinations. We therefore feel that this was not a coincidental change from auricular fibrillation to normal sinus rhythm that can be seen in patients with paroxysmal atrial fibrillation.

SUMMARY

A 46-year-old white male with atrial fibrillation was converted to normal sinus rhythm with small doses of quinidine after d.c. defibrillation failed.

The authors are not aware of this finding being previously reported.

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