Control of Ventricular Tachycardia by Direct Cardiac Electric Shock


VENTRICULAR TACHYCARDIA is a not uncommon abnormality of cardiac rhythm and is usually associated with some form of organic heart disease. It may follow myocardial infarction or be associated with heart failure occurring as a result of systemic hypertension. In a small percentage of cases no obvious cause can be found.

Conversion of the abnormal rhythm by simple measures such as breathing, carotid pressure, and pharyngeal stimulation, is much less likely than in atrial tachycardia. The usual treatment is by drug therapy of which quinidine and procaine amide are the most commonly employed.

This case report concerns a patient who developed ventricular tachycardia following a probable myocardial infarct. The abnormal rhythm could not be converted by any of the customary medical measures. When peripheral circulatory failure had occurred and death was imminent, surgically induced cardiac arrest converted the rhythm to normal with full recovery.

Case Report

M. H., a 67-year-old invalid pensioner, gave a history that he had sustained a sharppel wound to the chest in 1918 but no serious consequences developed. In the same year he had an operation for urethral stricture. In 1955, following an abdominal injury, he developed a perineal cellulitis, and a permanent suprapubic cystostomy was established. Two attacks of cardiac syncope are recorded as having occurred during the hospital admission. His blood pressure was noted as 180/80.

On December 16, 1958, the patient was admitted with a 12-hour history of substernal pain radiating to the left shoulder. The pulse was 140, with extrasystoles, and the blood pressure was 135/85. The electrocardiogram showed evidence of myocardial ischemia (fig. 1). Despite an episode of mild cardiac failure, the patient made an uneventful recovery and was discharged on quinidine and chlorothiazide.

In July 1959, the patient was re-admitted with nausea and vomiting for 12 hours. There had been no chest pain. The pulse rate was 204 per minute, the blood pressure was 105/70, and the jugular venous pulse was elevated to 3 cm. above the sternal angle when the patient was semi-recumbent at 45°. The electrocardiogram showed ventricular tachycardia (fig. 2). The serum glutamic oxaloacetic transaminase (SGOT) following admission was 250 units.

Carotid pressure, breathing, and pharyngeal stimulation failed to convert the arrhythmia. Pro- caine amide, 1 Gm., was given intravenously in 5 minutes without effect and was followed immediately by rapid digitalization because of the heart failure. On the day following admission quinidine sulfate was commenced. The dosage of the drug was increased over the ensuing 7 days until the eighth day when 6 Gm. were given over 12 hours in divided doses. This was combined with atropine sulfate, 1.2 mg.; for three doses. Some slowing of the pulse was achieved with spreading of the QRS complexes but no conversion of the rhythm (fig. 3).

Digitalis was then stopped and 1.5 Gm. of procaine amide was given intravenously in 5 minutes followed by 0.25 Gm. orally every 4 hours. The arrhythmia persisted, and by the next day the patient’s general condition had deteriorated considerably. There was marked peripheral cyanosis, and the jugular venous pressure was raised 5 cm. above the sternal angle. The blood pressure varied between 80 and 100 mm. Hg systolic. The patient was cold, sweating, and in peripheral circulatory failure. Considerable sacral edema was present, and the urinary output had decreased considerably over the last 24 hours.

It was considered at this stage that unless the rhythm could be converted the patient would be dead within a few hours. It was decided to attempt external conversion by electric shock. The patient was taken to the operating theater and given light anesthetic with pentobarbital and fluothane. Electrodes were applied in varying positions over the left chest, and electric shocks at 180 volts for 1 second were applied without effect. A higher
Electrocardiogram done in December 1958, showing evidence of myocardial ischemia.

Voltage was not obtainable with the equipment available at that time. At this stage the blood pressure was unrecordable. A small left anterolateral thoracotomy was performed and the pericardium was opened. The heart was considerably dilated and beating weakly at a rate of 140 to 160 beats per minute. The electrodes of the standard, internal defibrillator were then applied across the base of the left ventricle. Two shocks of 110 volts each for 0.1 second were applied. The heart immediately stopped. Massage was commenced and cardiac contraction quickly recommenced, with rapid improvement in ventricular tone. The rhythm varied from normal to some
degree of heart block (fig. 4). As cardiac rhythm and tone remained satisfactory, the chest was closed with drainage.

Despite a period of marked oliguria in the immediate postoperative period, the patient made a satisfactory recovery and was discharged from the hospital well, on the tenth postoperative day. Varying degrees of heart block persisted for the first week but normal rhythm with a few extrasystoles became established and has persisted ever since. The patient is now well 6 months after operation. He is maintained on 0.2 Gm. of quinidine daily and 500 mg. of chlorothiazide twice daily.

Discussion

In view of the previous history of myocardial ischemia and the high SGOT on admission it is reasonable to assume that an episode of infarction had precipitated the onset of tachycardia in this man. In attempting to convert the rhythm by medical means quinidine was given to the limit of tolerance as wide spreading of the QRS complexes occurred on the dosage reached. When oral quinidine fails, intravenous quinidine has been used but it was thought in this case that success with this would be unlikely. The dosage of procaine amide was high but recent experience suggests that it should be carried to a higher dosage than was used here.

External control of ventricular standstill and paroxysmal tachycardia by electric shock has been reported in many cases. Zoll reported 532 episodes of abnormal rhythms and included in these are eight cases of paroxysmal tachycardia controlled by external countershock. He found that voltages up to 400 volts were required in some cases.

In this patient external control by electrical countershock seemed the next step after failure of medical measures. It illustrates the fact that apparatus delivering only up to 200 volts is insufficient for external countershock. It further shows that when such equipment is not available, the standard internal defibrillator delivering 100 volts applied directly to the heart may control ventricular tachycardia with successful recovery.

Now that more adequate types of apparatus for electric countershock have become avail-

Figure 2

Electrocardiogram on admission showing ventricular tachycardia with a rate of 200 beats per minute.
able, external control of paroxysmal ventricular tachycardia should be attempted as soon as it is clear that medical management is not securing control of the abnormal rhythm and failure of the circulation is ensuing. In the event that the standard internal defibrillator is the only apparatus available, or external shock fails, direct application of the electrodes to the heart may be necessary to terminate the abnormal rhythm.

Summary

A case is presented of ventricular tachycardia occurring following myocardial infarction. When medical measures failed to convert the rhythm to normal, direct cardiac electric shock was employed, with return to normal of the cardiac rhythm and recovery of the patient.

References

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