Intractable Ventricular Tachycardia Treated with Massive Countershock and Large Doses of Procainamide

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SUMMARY

Ventricular tachycardia complicating myocardial infarction is a dangerous condition requiring urgent therapy. A case report describes the most challenging therapeutic problem of ventricular tachycardia which we have encountered over the past five years in the Coronary Intensive Care Unit.

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CASE REPORT

The patient, a 65-year-old White male, was admitted to the Coronary Intensive Care Unit a few hours after an acute myocardial infarction. His first stay in the unit, lasting 6 days, was complicated by sinus bradycardia, ventricular extrasystoles, complete heart block (for which a temporary transvenous pacemaker was inserted), cardiac failure requiring treatment with digitalis and diuretics, and the development of a systolic murmur compatible with papillary muscle dysfunction. His heart block regressed via a Wenckebach second-degree block, and by the twelfth day after infarction he was in sinus bradycardia with a normal P-R interval and his temporary pacemaker was removed.

Nineteen days after infarction, in a general medical ward, he developed ventricular tachycardia at a rate of 118/min. He suffered no distress, although his blood pressure fell from 140 to 90 mmHg systolic. He was readmitted to the Coronary Intensive Care Unit and his digoxin therapy was discontinued immediately.

During the next 11 days, the following drugs were used intravenously: lignocaine 200 mg over 5 min; Isoptin 10 mg over 5 min; procainamide 1 g over 50 min; Eraldin 25 mg over 25 min; potassium 80 mEq/litre over 2 hours at a time when his serum potassium was 4,4 mEq/litre; also quinidine 1 800 mg over 4 hours orally, which produced QRS widening of 20%; bretylium tosylate up to 800 mg intramuscularly every 12 hours, which produced postural symptoms and nausea, despite his being kept at bed rest; Epanutin 1 g intravenously over 6 hours, droperidol 5 mg intramuscularly and magnesium sulphate 20 ml of 50% solution intravenously over 5 min; and combinations of quinidine or procainamide with Eraldin.

In an attempt to increase the sinus node rate and achieve sinus capture, atropine was given intravenously without significant effect. Intravenous isoprenaline achieved a fast sinus rate with sinus capture, but ventricular tachycardia broke through as soon as the infusion rate was reduced.

During this period also, cardioversion using energies up to 400 Wsec was unsuccessfully tried, both alone, and immediately after 200 mg lignocaine intravenously. A transvenous pacemaker was inserted into the right ventricle, where overdrive ventricular pacing was successfully achieved. The arrhythmia, however, broke through whenever the pacing rate was lowered below that of the tachycardia. Paired ventricular pacing was then tried, and the arrhythmia was controlled with a paced ventricular rate of 180/min. Again, however, the arrhythmia broke through whenever the paired pacing was discontinued. After paired pacing for approximately 36 hours, the patient went into ventricular fibrillation and was successfully defibrillated into his original ventricular tachycardia rhythm, and paired pacing was discontinued.

With a resectable left ventricular aneurysm in mind, cardiac catheterisation was performed, which showed an akinetic inferior left ventricular wall, with an otherwise adequately contracting left ventricular muscle, but no frank aneurysm. Left ventricular diastolic pressure during the investigation was 26 - 43 mmHg. Coronary arteriography showed complete occlusion of the left circumflex artery with severe patchy disease of both the left anterior descending and the right coronary arteries.

By now the ventricular rate had gradually risen from 120 to 180/min at times (Fig. 1), and cardiac failure had developed. The blood urea had risen to 70 mg/100 ml with a serum creatinine of 2,4 mg/100 ml. On the advice of Dr Bernard Lown, 2 standard cardioverters were linked in series and an 800 Wsec DC shock was administered under 90 mg of Valium sedation. The patient reverted to sinus bradycardia with a current of injury persisting for about 30 sec after the shock (Fig. 2).

Twelve hours later the ventricular tachycardia broke through again, and it was thought that this was facilitated by the underlying sinus bradycardia. Further, transiently successful cardioversion was now achieved at lower ener-

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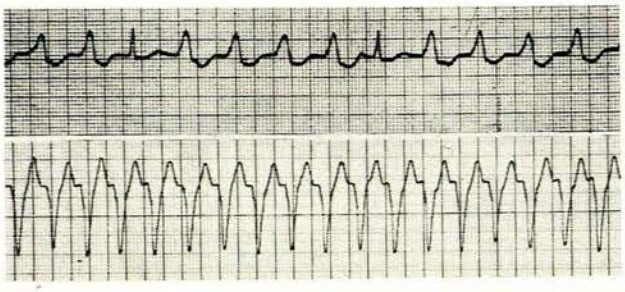

Fig. 1. Upper monitor lead ECG illustrates ventricular tachycardia at onset at rate of 118/min. The third and eighth complexes are sinus captures. Lower monitor lead ECG strip (different lead system) illustrates the faster ventricular rate which gradually developed.

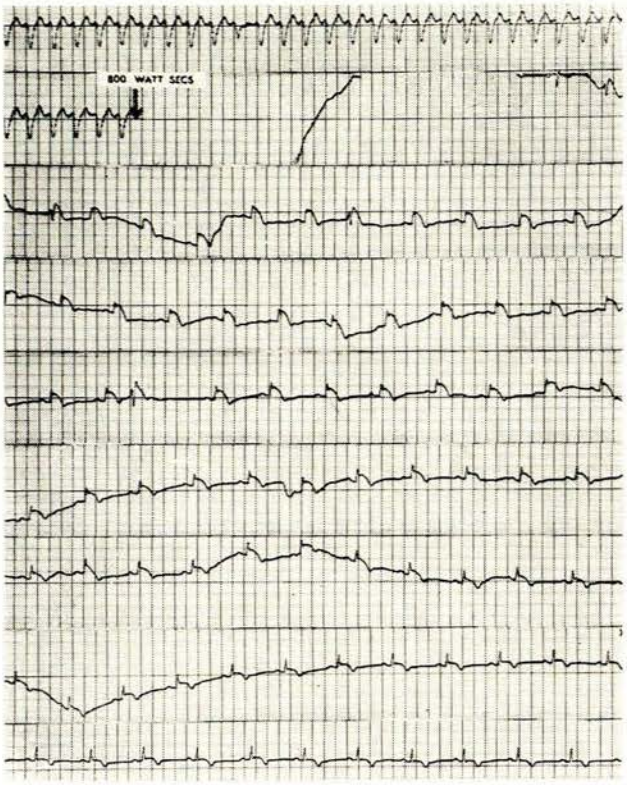

Fig. 2. Monitor lead ECG illustrating effect of first 800 Wsec DC countershock on S-T segments.

ies, usually in excess of 600 Wsec but, on occasion, equiring as little as 400 Wsec. Despite maintaining a ast sinus rate with a combination of atropine and iso-renaline, and subsequently with ventricular pacing, only nort-lived sinus capture resulted after each cardioversion efore ventricular tachycardia broke through again.

Eighteen days after the onset of ventricular tachycardia, ie patient was digitalised and given procainamide via a ontinuous intravenous infusion at the rate of 1 g/h. fter 5,15 mg of procainamide, sinus rhythm at a rate f 60/min resulted. A heart rate of 88/min was then maintained by ventricular pacing and procainamide, 1 g every 4 hours orally. The patient, however, vomited, and remained hypotensive and in cardiac failure with poor urine output and a rising blood urea. The dose was reduced, and finally discontinued in the hope that the faster paced rate alone would prevent a recurrence of the arrhythmia. Ventricular tachycardia, however, recurred, and it required a further 6 g of procainamide over 6 hours intravenously to convert the patient to sinus rhythm. Procainamide was now given via a constant intravenous infusion, and gastro-intestinal side-effects no longer presented a problem.

By now it was evident that the arrhythmia could be converted with large doses of procainamide but that breakthrough occurred at a slow intrinsic sinus rate. A permanent atrial pacemaker was therefore advised, as preservation of appropriately timed atrial systole was considered advantageous in the face of persistently poor cardiac function. Twenty-seven days after the onset of the ventricular tachycardia, a permanent atrial pacemaker was implanted at thoracotomy. During surgery ventricular tachycardia recurred and was again controlled with large doses of intravenous procainamide. Paced atrial rhythm with 2:1 A-V block was present postoperatively, but on withdrawing the digitalis, this changed to paced atrial rhythm with a normal P-R interval and 1:1 conduction. The patient's postoperative course was one of steady improvement. His systolic blood pressure rose to between 100 - 140 mmHg, cardiac failure regressed, blood urea fell to 20 mg/100 ml and serum creatinine to 0,9 mg/100 ml. His mental state returned to normal and his procainamide was reduced to a maintenance dose of 500 mg every 6 hours which he was able to take orally without ill effects.

Thirty-nine days after the arrhythmia had first appeared, the patient was intermittently in sinus rhythm above the reserve rate of the pacemaker of 90/min, and he was discharged from hospital with only slightly reduced effort tolerance. In the course of the following 12 months of follow-up, procainamide was withdrawn under careful monitoring control without recurrence of arrhythmia. On exercise he can accelerate his sinus rate above the pre-set rate of 90/min of the pacemaker, suggesting that his sinus node has recovered sufficiently to make his pacemaker redundant.

DISCUSSION

The incidence of ventricular tachycardia following acute myocardial infarction has been variously reported as from 6%[1] to 30%[2] with a mortality of between 30%[3,4] and 67%[1]; with prolonged ventricular tachycardia having a mortality as high as 72%.[5] Ventricular tachycardia occurs most commonly within 48 hours of infarction,[6-8] although ventricular tachycardia of late onset is a well-recognised occurrence.[3,9] Bouvrain[3] reported 20% of ventricular tachycardias occurring more than 3 weeks after infarction. Factors such as fresh infarction, increased sensitivity to catecholamines, hypoxia, acidosis, hypokalaemia, digitalis toxicity and increased free fatty acids, have been implicated in late onset arrhythmias. No obvious cause was operative in the patient described, although he had shown a

complicated initial course with A-V block and left ventricular failure, as is frequently seen in patients suffering late-onset arrhythmias.[9-10]

Therapy of ventricular tachycardia is fairly standardised, and although control of paroxysmal tachycardia has presented problems in some of our patients previously, we have not experienced undue difficulty in breaking attacks of ventricular tachycardia. In view of the failure of seemingly acceptable doses of current anti-arrhythmics, cardioversion, overdrive pacing and paired pacing, we undertook cardiac catheterisation, including coronary and left ventricular angiography, to look for a left ventricular aneurysm, resection of which may have aborted the arrhythmia. Recent reports by DeSanctis *et al.*[11] and Ecker *et al.*[12] describe the successful use of ventricular aneurysmectomy with or without saphenous aortocoronary bypass grafts, in the cure of drug-resistant late-onset ventricular tachycardias after myocardial infarction. No clear aneurysm was seen in our patient and no vessels were suitable for bypass grafting.

Desperation, therefore, led to the use of high-energy (800 Wsec) countershock on the advice of Dr Eernard Lown who had previously reported its successful use in a case of resistant ventricular tachycardia.[13] With the use of liberal amounts of electrode paste only slight erythema over the site of application of the cardioverter paddles was produced, although a striking transient current of injury was evident on the electrocardiogram which is not believed to result in any permanent cardiac damage.[14] Procainamide in doses similar to those used initially (100 mg every 5 min up to a total of 1 g) is a well-established and effective therapy for ventricular tachycardia in most patients. In the patient described, however, it was clearly inadequate and massive doses were required (5 - 6 g over 5 - 6 hours). Cohn *et al.*[5] made this point quite strongly, pointing out that many so-called drug-resistant cases of ventricular tachycardia have received insufficient procainamide, and recommended 50 - 100 mg/min intravenously for a minimum of 15 - 20 min, or until toxic manifestations occur. If necessary, they advocated continuing the procainamide with concomitant use of vasopressors if hypotension becomes a problem. Other authors [13,15-17] have also recommended very large doses of procainamide to break resistant ventricular tachycardia.

Finally, as has been shown by other workers,[18] the arrhythmia was eventually suppressed by the combination of an anti-arrhythmic agent, plus artificial pacing to overcome the persistent sinus bradycardia. With time, the bradycardia has subsided and procainamide has been successfully withdrawn. The reason for the prolonged sinus node depression in this patient is unknown, but may have been related to vascular interference at the time of myocardial infarction, with subsequent delayed recovery.

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