REFERENCES

1. Dowell VR Jr, Hawkins TM: Laboratory Methods in Anaerobic Bacteriology. Washington, DC, Government Office, 1968 (PHS Publication No 1803)
2. Cato EP, Cummins CS, Holdeman LV, et al: Outline of Clinical Methods in Anaerobic Bacteriology. Blacksburg, Virginia, Virginia Polytechnic Institute, Anaerobe Laboratory, 1969
3. Janbon, Labraque-Bordenave, Gordon-Martins: Septicémie subaiguë à *B. perfringens*, d'allure primitive, avec anémie et endomyocardite. Arch Soc d sc Med Biol de Montpellier 16:484-488, 1934-35
4. Cates JE, Christie RV: Subacute bacterial endocarditis. Quart J Med 20:93-130, 1951
5. Morgan WL, Bland EF: Bacterial endocarditis in the antibiotic era. Circulation 19:753-765, 1959
6. Vogler WR, Dorney ER, Bridges HA: Bacterial endocarditis. Amer J Med 32:910-921, 1962
7. Pankey GA: Subacute bacterial endocarditis at the University of Minnesota Hospital, 1939 through 1959. Ann Intern Med 55:550-561, 1961
8. Janbon, Chaptal, Labraque-Bordenave: Deux observations de septicémie à *B. perfringens* avec anémie, endocardite et phlébites. Arch Soc de sc Med biol de Montpellier 16:475-484, 1934-35
9. More RH: Bacterial endocarditis due to *Clostridium Welchii*. Amer J Path 19:413-420, 1943
10. Watanabe Y, Ueno K: Case of subacute bacterial endocarditis due to *Eubacterium*. J Jap Ass Infect Dis 42:78-83, 1968
11. Loewe L, Rosenblatt P, Alture-Werber E: A refractory case of subacute bacterial endocarditis due to *Veillonella gazogenes* clinically arrested by a combination of penicillin, sodium para-aminohippurate and heparin. Amer Heart J 32:327-338, 1946
12. Jones M: Subacute bacterial endocarditis of nonstreptococcic etiology. Amer Heart J 40:106-116, 1950
13. Kaplan K, Weinstein L: Diphtheroid infections of man. Ann Intern Med 70:919-929, 1969
14. Kourilsky R, Piéron R, Demay C, et al: À propos d'un cas de septicémie à Corynebacterium anaérobium. Sem Hop Paris 41: 1408-1412, 1965
15. Child JA, Darrell JH, Davies NR, et al: Mixed infective endocarditis in a heroin addict. J Med Microbiol 2:293-299, 1969
16. Fisher AM, McKusick VA: Bacteroides infections: clinical, bacteriological and therapeutic features of 14 cases. Amer J Med Sci 225:253-273, 1953
17. Tumulty PA, Harvey AM: Experiences in the management of subacute bacterial endocarditis treated with penicillin. Amer J Med 4:37-54, 1948
18. Cressy NL, Lahey WJ, Kunkel P: Streptomycin in the treatment of bacterial endocarditis: report of two cases. New Eng J Med 239:497-500, 1948
19. Rantz LA: Bacteroides endocarditis: report of a case successfully treated with terramycin. JAMA 147:124, 1951
20. Loesche WJ: Oxygen sensitivity of various anaerobic bacteria. Appl Microbiol 18:723-727, 1969
21. Zierdt CH, Wertlake PT: Transitional forms of *Corynebacterium acnes* in disease. J Bact 97:799-805, 1969
22. Wolfe MW, Amsterdam D: Addition of a hypotonic medium to blood culture routine for detection of aberrant microorganisms. Bact Proc:97, 1970
23. Felner JM, Dowell VR Jr: "Bacteroides" bacteremia. Amer J Med (in press)
24. Alpern RA, Dowell VR Jr: Non-histotoxic clostridial bacteremias. Amer J Clin Path (in press)
25. Gesner BM, Jenkin CR: Production of heparinase by bacteroides. J Bact 81: 595-604, 1961
26. Bjornson HS, Hill EO, Altemeier WA: Role of L-forms, *Bacteroides* species, and *Sphaerophorus* species in acute and recurrent thromboembolic disease. Bact Proc:87, 1970
27. Finland M: Current status of therapy in bacterial endocarditis. JAMA 166:364-373, 1958
28. Harvey WP, Capone MA: Bacterial endocarditis related to cleaning and filling of teeth: with particular reference to the inadequacy of present day knowledge and practice of antibiotic prophylaxis for all dental procedures. Amer J Cardiol 7:793-798, 1961
29. Drasar BS, Hughes WH, Williams REO, et al: Bacterial flora of the normal intestine. Proc Roy Soc Med 59:1243, 1966
30. Friedberg C: Diseases of the Heart. Third edition. Philadelphia, WB Saunders Company, 1966
31. Finegold SM, Sutter VL, Sugihara PT: Susceptibility of anaerobic bacteria to rifampicin. Bact Proc:73, 1969

CHEST THUMP FOR REVERTING VENTRICULAR TACHYCARDIA*

JAMES E. PENNINGTON, M.D., JACK TAYLOR, M.D., AND BERNARD LOWN, M.D.

Abstract A sharp precordial thump was employed to terminate 12 episodes of ventricular tachycardia (VT) in five patients with coronary heart disease. A precordial thump, through electromechanical transduction, provides a low energy current that depolarizes a re-entry pathway, the probable basis for most cases of clinical VT. Indeed, analysis of cardioversion energies for VT has demonstrated that 93 per cent of episodes are reversed with energies of 10 w sec or less. Chest thump is an important maneuver for treating cardiac arrest initiated by VT.

IN 1920, Schott reported that a blow to the chest restored a palpable pulse in a patient with a Stokes–Adams attack.[1] Sherf and Bornemann, in 1960, described 11 patients with asystole in whom a precordial blow stimulated a single electrical response and occasional brief repetitive ventricular arrhythmias.[2] Today, many textbooks and articles recommend a precordial blow for asystole. None, however, suggest that the maneuver is effective in ventricular arrhythmias.

Sustained ventricular tachycardia (VT), especially when it develops in the course of acute myocardial infarction, constitutes a medical emergency.[3] If the treatment of first choice, intravenous lidocaine, is not successful, cardioversion is employed.[4] The energies advised for electrical reversion range from 50 to 300 watt seconds (w sec).[5-9] It is not appreciated that a sharp blow to the chest or a discharge of distinctly lower energy content is frequently sufficient to restore a normal rhythm. The present report details some clinical observations on the use of chest thump for reverting VT to sinus rhythm. Experience with the use of low energy cardioversion is reviewed and is related to pertinent animal experiments. These findings shed light on the mechanism of VT in the patient with ischemic heart disease and provide an effective, immediate ap-

*From the Cardiovascular Research Laboratories, Department of Nutrition, Harvard School of Public Health, and the Medical Clinics of the Peter Bent Brigham Hospital (address reprint requests to Dr. Lown at the Department of Nutrition, Harvard School of Public Health, 665 Huntington Ave., Boston, Mass. 02115).

Supported in part by grants (HE-07776, 5T1-HE-5242, and PO1-HE-11306) from the National Institutes of Health, U. S. Public Health Service.

Abbreviations Used	
CCU:	coronary-care unit
VT:	ventricular tachycardia
VT(vp):	ventricular tachycardia at vulnerable period
w sec:	watt seconds

proach for dealing with life-threatening ventricular tachyarrhythmia.

Clinical Findings

Chest Thump

During the past year, 12 episodes of VT in five patients were terminated with a sharp blow to the lower chest. All five patients were men with ischemic heart disease. The following two case histories are illustrative:

Case 1. G.F. (P.B.B.H. 10-34-33), a 49-year-old man, was admitted to the Peter Bent Brigham Hospital because of recurring VT. At the age of 38 he had suffered an apical myocardial infarction. Four years later paroxysmal VT occurring at increasingly frequent intervals had developed. Quinidine, 2.4 g per day, propranolol, 160 mg per day, diphenylhydantoin, 400 mg per day, procaine amide, 1 g per day, lidocaine, 2 mg per minute intravenously, and digitalization with digoxin had failed to prevent recurrences. The tachycardia could not be reverted by the intravenous use of procaine amide (1 g), diphenylhydantoin (900 mg, with a blood level of 35 mg per liter) and lidocaine (in boluses of 0.50 mg). Atrial pacing, at rates faster than ventricular, failed to capture the ventricle during a paroxysm. Five weeks before transfer from another hospital he suffered 9 episodes of tachycardia resistant to drugs and requiring cardioversion. After the last 4 episodes, VT recurred within minutes after sinus rhythm was restored.

When he entered the Peter Bent Brigham Hospital, an electrocardiogram showed a tachycardia at a rate of 135 with a dissociated atrial rate of 79 clearly demonstrable in an intra-atrial lead (Fig. 1). Sinus rhythm was restored after a 1 w sec synchronized precordial discharge (Fig. 2). The electrocardiogram after reversion showed a pattern consistent with extensive anterior transmural infarction of uncertain duration.

Four days later a bout of ventricular tachycardia was terminated by a precordial blow with the fist after sedation with pentobarbital and diazepam (Fig. 3). Eight days later, while he was taking 5 g of procaine amide daily, VT recurred when the patient was recumbent after graded exercise. Two self-administered precordial blows and a single blow by the physician were without effect. After sedation with diazepam in preparation for cardioversion, a single blow restored sinus rhythm. The patient complained of soreness over the precordium for the ensuing 3 days. A subsequent recurrence of VT was reverted with 1 w sec without sedation. The arrhythmia, thereafter, was controlled with a combination of procaine amide, 4 g per day, propranolol, 30 mg per day, and acetyl digitoxin, 0.2 mg per day.

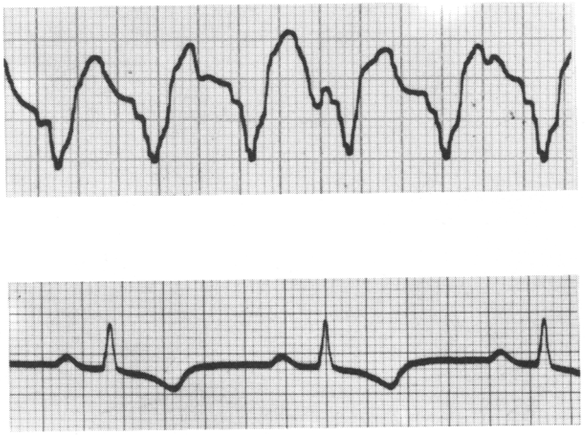

Figure 2. Two Lead 2 Strips Taken Three Seconds Apart in Case 1.

The top strip shows ventricular tachycardia, and the bottom strip was recorded immediately after cardioversion discharge of 1 w sec.

Case 2. R.M. (P.B.B.H. 6-00-89), a 46-year-old man, was in good health until the day of admission, when crushing precordial pain accompanied by nausea, vomiting and diaphoresis suddenly occurred. The electrocardiographic pattern was consistent with an evolving acute anterior myocardial infarction with transient right-bundle-branch block. He was hypotensive and in pulmonary edema. Within 10 minutes after arrival in the coronary-care unit (CCU) sustained VT developed. The house officer at the bedside immediately gave the patient a brisk blow to the chest, converting the mechanism instantly to sinus rhythm. In several minutes the patient again went into VT, and again a blow to the chest was successful. When the next episodes occurred, cardioversion was employed four times to restore sinus rhythm. Cardiogenic shock developed, and he died within 24 hours.

In three of the five patients, acute myocardial infarction was the basis for the VT. In each case a brisk blow to the midsternum or left precordial area was required to terminate the arrhythmia. One of these patients, after resuscitation from ventricular fibrillation, experienced 18 episodes of VT unre-

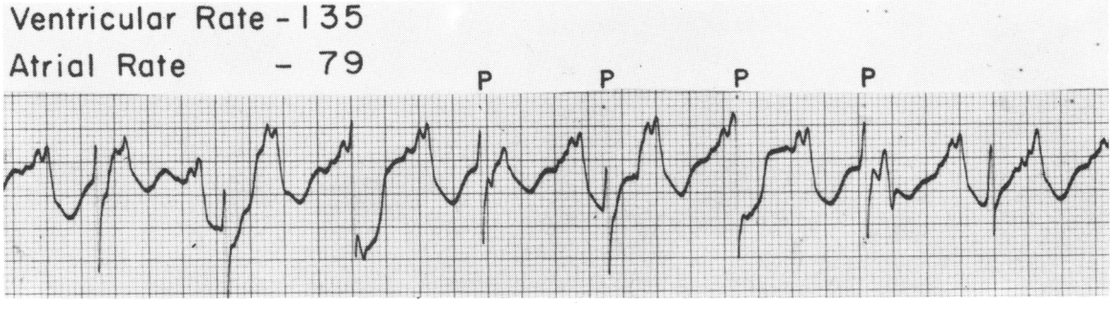

Figure 1. Intra-atrial Lead, in Case 1, Demonstrating Complete Atrioventricular Dissociation and Consistent with the Diagnosis of VT.

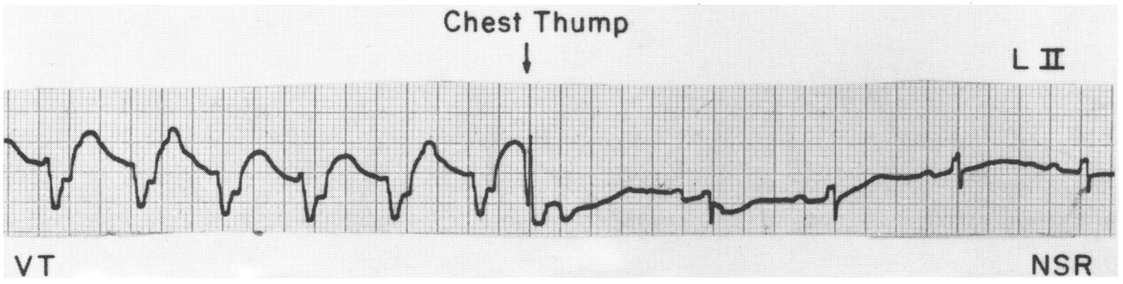

Figure 3. VT Terminated with a Single Sharp Blow Delivered to Lower Sternum in Case 1.

sponsive to lidocaine given as a 100-mg bolus followed by infusion of 4 mg per minute. In the first six VT was reverted by a thump on the chest; the ensuing 12 bouts were restored by means of cardioversion employing low energy discharge. The mechanical stimulus evoked a premature depolarization that at times was identical in morphology to the QRS complex of the VT. A brief pause ensued before sinus rhythm was resumed. On several occasions a few abnormal complexes intervened before sinus dominance was established. In one case VT at a more rapid rate resulted from the initial thump; a second precordial blow then restored sinus rhythm (Fig. 4).

at 10 w sec, in only three was cardioversion initiated with an energy setting of 5 w sec or less. If the energy titration in these nine episodes had been started at 1 w sec, a number might have responded to discharges of such lower energy content. Thus, VT in the patient with severe ischemic heart disease can be reverted with low energy shock.

Discussion

A thump to the chest or low energy shock can revert VT arising in patients with ischemic heart disease. In dogs with acute myocardial infarction it is possible to induce a prefibrillatory arrhythmia, designated as ventricular tachycardia of the vulnera-

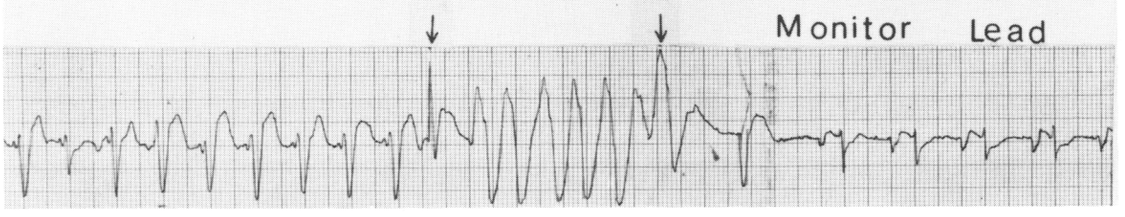

Figure 4. Ventricular Tachycardia at a Rate of 150 (Note That the Second Complex Is a Captured Beat).
A chest thump (↓) evokes an ectopic beat of different morphology and results in a more rapid tachycardia at a rate of 220 per minute. Immediate repetition of thump reverts arrhythmia to sinus rhythm. Note the single ectopic beat before normal mechanism supervenes.

Cardioversion with Low Energy

During the period of January 1, 1968, to June 30, 1970, 14 patients, exhibiting 32 episodes of ventricular tachycardia, were subjected to cardioversion. In 27 episodes in 11 patients, the initial energy employed was 10 w sec or less. Cardioversion was attempted when intravenous lidocaine proved unsuccessful. The following analysis is based on the 11 patients to whom reversion was initiated with low energy shock. Six were experiencing acute myocardial infarction; the remaining five had sustained infarctions in the past and had findings suggestive of ventricular aneurysm. Nine of the patients were male. Table 1 indicates the effective discharge energy for restoring sinus rhythm.

In 93 per cent of episodes of VT, 10 w sec or less was the effective energy. Of the 12 episodes reverted

ble period, or VT(vp), which can be terminated with transthoracic shocks of less than 1 w sec.[10] It was this observation that led to the use of low energy shock in human VT. This, in turn, suggested chest thump as an immediately available low energy source.

Reversion of atrial fibrillation to sinus rhythm requires cardioversion energies of about 100 w

Table 1. Effective Energy Discharge for Restoring Sinus Rhythm.

Energy *w sec*	No. Reverted
1	8
5	5
10	12
>10	2

sec.[11] Termination of ventricular fibrillation in man requires even higher energies. Cardioversion of atrial flutter can be accomplished with low energy discharge. All these arrhythmias are believed to be due to re-entrant mechanisms. The rhythm disorder is sustained by the continuous traverse of a depolarization wave front over fixed single or multiple and variable pathways. At any one instant part of the circuit is refractory, and part recovered. Premature depolarization of the recovered portion blocks conduction for the circulating wave front, thereby extinguishing the arrhythmia. A possible explanation for the divergence of energy for terminating re-entrant mechanisms is that depolarization of multiple pathways at differing stages of recovery requires a large electric discharge. However, when the arrhythmia is sustained by conduction over a single circuit, threshold energies for depolarizing the heart suffice. Indeed, recent observations in man indicate that pacemaker-induced extrasystoles suffice to abolish re-entrant arrhythmias.[12-14]

In the heart with asystole or with the re-entrant type of tachycardia, an effective blow to the chest induces focal excitation, which propagates as a single ectopic systole. Thus, the chest blow serves as an external pacemaker to produce ectopic depolarizations of a fraction of the re-entrant circuity, thereby terminating such arrhythmias.

The deliberate use of a blow to the chest for terminating ventricular tachyarrhythmia has not hitherto been reported. Semple[15] has indicated that a chest thump abolished a paroxysm of supraventricular tachycardia with bundle-branch block resistant to other measures. It has been assumed that if cardiac arrest responded to a precordial blow, the mechanism was asystole rather than tachyarrhythmia. The use of a blow to the chest is now a widely accepted practice in the treatment of cardiac arrest. When standstill develops in the ischemic heart a mechanical stimulus evokes but a single ectopic beat. As the heart grows increasingly hypoxic during arrest, a thump may activate salvos of bizarre complexes, which can degenerate into ventricular fibrillation.[2] In the CCU, where precise determination of the mechanism of cardiac arrest is possible, when arrest is due to asystole, a precordial blow results in but one depolarization. A single thump fails to restore sustained cardiac activity, though continuous precordial percussion has been shown to maintain an effective heartbeat for 90 minutes.[16] It is likely that the wide acceptance of precordial blow for cardiac resuscitation is based on the effectiveness of this treatment in terminating prefibrillatory VT rather than asystole.

Chest thump is an important maneuver in cardiac resuscitation. When arrest occurs, prompt action is essential. The longer the delay in restoring sinus rhythm, the less likely is immediate success and the slighter the possibility of late survival. A defibrillator or cardioverter is usually not available at the scene of such an emergency. It is likely that a majority of episodes of cardiac arrest are due to ventricular fibrillation. The arrhythmia is preceded by VT(vp), a disorder that can probably be terminated by a single ectopic depolarization of the heart. This requires small energies and can be provided by a mechanical blow to the chest wall. Once ventricular fibrillation develops, there is a steep rise in electrical energy necessary for restoring sinus rhythm. The method of chest thump affords the advantage of immediate availability. It will do no harm in cases of cardiac asystole and may prove lifesaving when the mechanism is prefibrillatory VT. When, however, VT is well tolerated and a cardioverter is available, cardioversion using low energies is more predictable, less traumatic and the procedure of choice.

REFERENCES

1. Schott E: Über Ventrikelstillstand (Adams-Stokes'sche Anfälle) nebst Bemerkungen über andersartige Arhythmien passagerer Natur. Deutsches Arch Klin Med 131:211-229, 1920
2. Scherf D, Bornemann C: Thumping of the precordium in ventricular standstill. Amer J Cardiol 5:30-40, 1960
3. Lown B, Klein MD, Hershberg PI: Coronary and precoronary care. Amer J Med 46:705-724, 1969
4. Lown B, Fakhro AM, Hood WB, et al: The coronary care unit: new perspectives and directions. JAMA 199:188-198, 1967
5. Harrison TR, Reeves TJ: Principles and Problems of Ischemic Heart Disease, Chicago, Year Book Medical Publishers, 1968, pp 176-177, and 192-193
6. Rubin IL, Gross H, Arbeit SR: Treatment of Heart Disease in the Adult. Philadelphia, Lea and Febiger, 1968, pp 202-204, 223 and 225
7. Schrire V: Clinical Cardiology. Second edition. London, Staples Press, 1966, pp 135 and 141
8. Friedberg CK: Diseases of the Heart. Third edition. Philadelphia, WB Saunders Company, 1966, pp 567-572
9. Weens HS, Gay BB Jr: Radiologic examination of the heart, The Heart: Arteries and veins. Second edition. Edited by JW Hurst, RB Logue. New York, McGraw-Hill Book Company, 1966, pp 316-330
10. Wolff GA, Vieth F, Lown B: A vulnerable period for ventricular tachycardia following myocardial infarction. Cardiovasc Res 2: 111-121, 1968
11. Lown B: Electrical reversion of arrhythmias. Brit Heart J 29:469-489, 1967
12. Durrer D, Schoo L, Schuilenburg RM, et al: The role of premature beats in the initiation and the termination of supraventricular tachycardia in the Wolff-Parkinson-White syndrome. Circulation 36:644-662, 1967
13. Massumi RA, Tawakkol AA, Kistin AD: Reevaluation of electrocardiographic and bedside criteria for diagnosis of ventricular tachycardia. Circulation 36:628-636, 1967
14. Zeft HJ, McGowan RL: Termination of paroxysmal junctional tachycardia by right ventricular stimulation. Circulation 40:919-926, 1969
15. Semple T: Discussion, Acute Myocardial Infarction: Proceedings of a symposium sponsored by the University of Edinburgh September 1967. Edited by DG Julian, MF Oliver. Edinburgh, E and S Livingstone, 1968, pp 82-83
16. Don Michael TA: Precordial percussion in cardiac resuscitation. Amer Heart J 69:721-722, 1965