

# Termination of Refractory Tachycardia by External Countershock

By PAUL M. ZOLL, M.D., AND ARTHUR J. LINENTHAL, M.D.

**V**ENTRICULAR and supraventricular tachycardias are occasionally resistant to drug therapy and may culminate in congestive heart failure, circulatory collapse, ventricular fibrillation, and death. In this paper are presented recent experiences with eight patients in whom tachycardias were unresponsive to drug therapy and were terminated by external electric countershock, usually under general anesthesia.

The technic of external electric countershock was originally developed for the termination of ventricular fibrillation without opening the chest.<sup>1</sup> It has become established as a safe, effective, and clinically practicable procedure for the emergency resuscitation of patients from this otherwise fatal arrhythmia.<sup>2</sup> Using this technic in unconscious subjects, we have also terminated other arrhythmias, namely, ventricular tachycardia causing Stokes-Adams attacks, and, in the experimental animal, ventricular tachycardia, supraventricular tachycardia, and atrial fibrillation.<sup>3</sup> These successes in terminating arrhythmias other than ventricular fibrillation suggested the applicability of this technic to desperate, refractory tachycardias.<sup>1-3</sup>

## Apparatus and Technic

The external countershock instrument\* contains a special step-up transformer that isolates the output from the line current and ground, and provides an output of 60-cycle alternating current in steps from 0 to 750 volts. The optimum electric

---

From the Medical Research Department, Beth Israel Hospital, and the Department of Medicine, Harvard Medical School, Boston, Massachusetts.

Aided by grants from the U. S. Public Health Service, National Heart Institute (H-5108, H-2208, HTS-5235), and the J. A. and Bessie Slosberg Charitable Foundation, Inc.

\*"The External Defibrillator" (D-72) is manufactured by the Electrodyne Company, Norwood, Massachusetts.

features of external countershock for the termination of ventricular fibrillation or tachycardia were carefully worked out in the laboratory.<sup>3</sup> The duration of the countershock is fixed at 0.15 second by a suitable condenser in a relay circuit. This duration is at the beginning of the plateau of the strength-duration curve for defibrillation by direct and by external countershock. Shorter countershocks are less efficient in terminating fibrillation, so that higher voltages are required; longer countershocks are no more effective in terminating fibrillation and merely increase the risk of cardiac damage.

Resistances between the electrodes have been found to average 50 ohms. Accordingly, the voltage output of the instrument is calibrated across a 50-ohm resistance; voltages measured across higher resistances may not be correct for the actual conditions of use. Countershocks up to 750 volts have been found necessary to terminate ventricular fibrillation externally; the current flow, therefore, may reach 15 amperes. For a wide margin of extra capacity the transformer, the power relay contacts, and the other components are designed to deliver 12,000 watts (15 amperes and 750 volts) for 0.15 second, repeated at intervals of 1 second.

Since external countershock would be extremely painful, conscious patients were anesthetized briefly. For convenience of anesthesia the countershocks were usually applied in the operating room. The small electrodes of a cardiac pacemaker-monitor,\* to which an electrocardiograph was usually connected for recording, were attached to the chest and the patient was anesthetized with intravenous sodium thiopental. At times, nitrous oxide and succinylcholine chloride were also given and intratracheal intubation was performed (table 1). The large electrodes (8.5 cm. diameter) of the countershock instrument were smeared liberally with electrode paste and held firmly on opposite sites of the precordium, usually by two persons, with care to avoid the monitor-pacemaker electrodes. With repeated countershocks, the chest was wiped dry between the electrodes to prevent short circuit of the current by previously applied electrode paste on the skin.

After the patient was anesthetized, an initial

---

\*Pacemaker-Monitor (PM-65) manufactured by the Electrodyne Company, Norwood, Massachusetts.

countershock of 150 to 450 volts was applied. If the tachycardia persisted or fibrillation supervened, additional, usually larger shocks were given promptly. If ventricular standstill occurred and persisted, external electric stimulation was applied. Again, at this point, it is important to wipe the chest clean and dry between the stimulating electrodes. If cardiac arrest persists, additional resuscitative measures should be undertaken even to the extent of thoracotomy and massage, if they are considered appropriate.

### Results

Table 1 summarizes in chronologic order recent experiences with termination of supraventricular and ventricular tachycardia and atrial fibrillation by external electric countershock.\*

Six of the patients were men and two were women. Their ages ranged from 42 to 79 years. Seven had had myocardial infarctions; in three, the infarct was recent, having occurred within a few weeks before the tachycardia. The eighth patient had mitral stenosis.

All the patients had also had previous arrhythmias, including atrial and ventricular premature beats, atrial fibrillation, ventricular tachycardia, and atrioventricular nodal tachycardia. Five patients were receiving maintenance doses of a digitalis glycoside at the time the tachycardia started, but in no case was digitalis toxicity apparent.

The rates of the tachycardias ranged from 140 to 220 beats per minute. As a consequence, the first seven patients were desperately ill with congestive failure and shock. In many, congestive failure progressed and the blood pressure could not be satisfactorily maintained by vasopressor agents. The tachycardias could not be terminated even though anti-arrhythmic drugs were given, often in heroic doses to the point of toxicity. The deteriorating clinical condition of these seven patients with the threat of imminent death impelled us to this new clinical application of external countershock.

\*The first patient was treated by Alexander, Kleiger, and Lown.<sup>4</sup> We wish to thank Dr. B. Lown for permission to include his case here and Drs. M. D. Phelps, Jr., C. Williams, A. L. Friedlich, H. Bauer, and J. L. Gouaux for the opportunity to treat patients 2, 3, 4, 6, and 8.

The unequivocal identification of tachycardias is often difficult. In two cases their origins could not be determined with certainty but ventricular tachycardia was clearly present in four, atrioventricular nodal tachycardia in one, and atrial fibrillation in one. Fortunately, this question is only of academic interest here, since external countershock can terminate supraventricular as well as ventricular tachycardia and fibrillation.

At the time of termination by external countershock, the tachycardias had lasted for 9 hours to 6 weeks. Patient 5, in severe pulmonary edema, was comatose. The other seven patients were conscious, and therefore were anesthetized briefly with sodium thiopental. In some cases nitrous oxide, intravenous succinylcholine chloride, and intratracheal intubation were also used.

The countershocks ranged in intensity from 150 to 650 volts. In four patients, only one countershock was required to terminate the tachycardia and restore persistent normal sinus rhythm. In patient 1, immediately after termination of the tachycardia, there was a short interval of ventricular standstill before normal sinus rhythm reappeared. In patients 2 and 6, there were intervening periods of slower atrioventricular nodal rhythm for 2 days and for 6 minutes. In patient 4, the single countershock resulted in the immediate return of normal sinoatrial activity.

In four patients, repeated countershocks were necessary. In patient 3, ventricular flutter, fibrillation, and tachycardia followed the first three countershocks. Finally, after 350 volts, there was a brief period of ventricular standstill and then normal sinus rhythm, even though atrial fibrillation had been present for 2 weeks before the tachycardia began. It appears likely that the countershocks also terminated the atrial fibrillation as well as the ventricular tachycardia in this patient.

Patient 8, with mitral stenosis, had uncontrollably rapid atrial fibrillation for 6 weeks with resultant congestive heart failure. Although she was not desperately ill, her persisting discomfort and disability impelled us

Table 1

*Summary of Eight Cases*

Case no.	Hospital	Age (yr.), sex	Before tachycardia			Tachycardia			Congestive failure and shock
			Myocardial infarction	Arrhythmia	Digitalis	Type	Rate (beats/min.)	Duration	
1	PBBH	59 M	Years	Atrial and ventricular premature beats	Months	Ventricular	190	2 days	Yes
2	NEBH	64 M	Years	Ventricular premature beats	1 week	Supraventricular or ventricular	175	22 days	Yes
3	MGH	64 M	2 weeks	Atrial fibrillation	2 weeks	Supraventricular or ventricular	193	14 days	Yes
4	MGH	42 M	7 months	Ventricular tachycardia	None	Ventricular	220	6 days	Yes
5	BIH	74 F	Years	Recurrent atrioventricular nodal tachycardia	Years	Atrioventricular nodal	150	2 days	Yes
6	BIH	64 M	4 weeks	Ventricular tachycardia	2 weeks	Ventricular	118-140	4 days	Yes
7	BIH	79 M	2½ weeks	Long P-R; ventricular premature beats	None	Ventricular	208	9 hours	Yes
8	BIH	51 F	None	Atrial fibrillation 3 times	None	Atrial fibrillation	160-200	6 weeks	Yes†

PBBH, Peter Bent Brigham Hospital; NEBH, New England Baptist Hospital; MGH, Massachusetts General Hospital; BIH, Beth Israel Hospital.

\*Comatose from pulmonary edema and intravenous morphine.

†Countershock was required four times in 30 minutes because of recurrent tachycardia.

‡Congestive failure only.

to use countershock before her condition should deteriorate further. Six shocks with voltages up to 650 volts were necessary to restore normal sinus rhythm.\*

Patient 5 was moribund, comatose, and in pulmonary edema on admission, so that there

\*In this connection, Dr. M. H. Paul, who worked with us in the development of the technic of external countershock defibrillation,<sup>1-3</sup> has recently used this procedure at the Children's Memorial Hospital in Chicago to terminate atrial tachycardia and flutter in three children.

was no time for extensive drug treatment, nor was anesthesia needed. The initial countershock terminated the atrioventricular nodal tachycardia immediately but it started again in a few minutes. Three more countershocks were required in a 30-minute period to terminate recurrent tachycardia, and intravenous procaine amide was given concomitantly before normal sinus rhythm finally persisted. Then, consciousness returned and pulmonary edema cleared rapidly.

In patient 7, the first countershock con-

Date	Countershocks				Results of countershocks			
	Anesthesia				Voltages	Rhythm	Blood pressure rise	Outcome
	Sodium thiopental	Nitrous oxide	Succinylcholine chloride	Intubation				
12-2-60	Yes	Yes	No	No	250	Ventricular standstill (10 sec.), normal sinus rhythm	Slow	Complete recovery
12-9-60	Yes	Yes	Yes	No	275	Atrioventricular nodal rhythm (2 days), normal sinus rhythm	Slow	Initial improvement; died in 7 days of renal and hepatic failure
1-11-61	Yes	Yes	Yes	Yes	150 250 250 350	Ventricular flutter Ventricular fibrillation Ventricular tachycardia Ventricular standstill (6 sec.), normal sinus rhythm	Prompt	Complete recovery
3-31-61	Yes	No	Yes	Yes	450	Normal sinus rhythm	Prompt	Complete recovery
6-15-61	No*	No	No	No	250 350 350 350 450	Normal sinus rhythm and recurrent atrioventricular nodal tachycardia each time† Persistent normal sinus rhythm	Prompt	Prompt subsidence of pulmonary edema and complete recovery
6-19-61	Yes	Yes	No	No	250	Atrioventricular nodal rhythm (6 min.), normal sinus rhythm	Slow	Complete recovery
9-26-61	Yes	No	No	No	350 450	Ventricular tachycardia-fibrillation Ventricular standstill	None	Died in ventricular standstill
10-24-61	Yes	No	No	No	250 350 450 550 550 650	Persistent atrial fibrillation, transient ventricular slowing, occasional ventricular premature beats Normal sinus rhythm and atrial premature beats	None	Complete recovery

verted the ventricular tachycardia to fibrillation. The second countershock terminated the fibrillation but ventricular standstill ensued, from which the patient could not be resuscitated by external cardiac massage or by external electric stimulation. Thoracotomy for direct cardiac massage was considered inappropriate in this patient. Even before the tachycardia, he had been desperately ill with congestive failure, severe renal disease, oliguria, and cerebrovascular disease.

In seven of these eight patients external

countershock terminated the tachycardia and restored normal sinus rhythm. There was a striking rise in blood pressure in six of the seven patients with hypotension due to the tachycardia (fig. 1). Six patients recovered completely from the effects of the tachycardia and were able to leave the hospital. The tachycardia recurred in the first patient after 3 months and was terminated again by external countershock;<sup>4</sup> recurrence has not been observed in the other patients in the follow-up periods of 2 to 12 months. The second patient

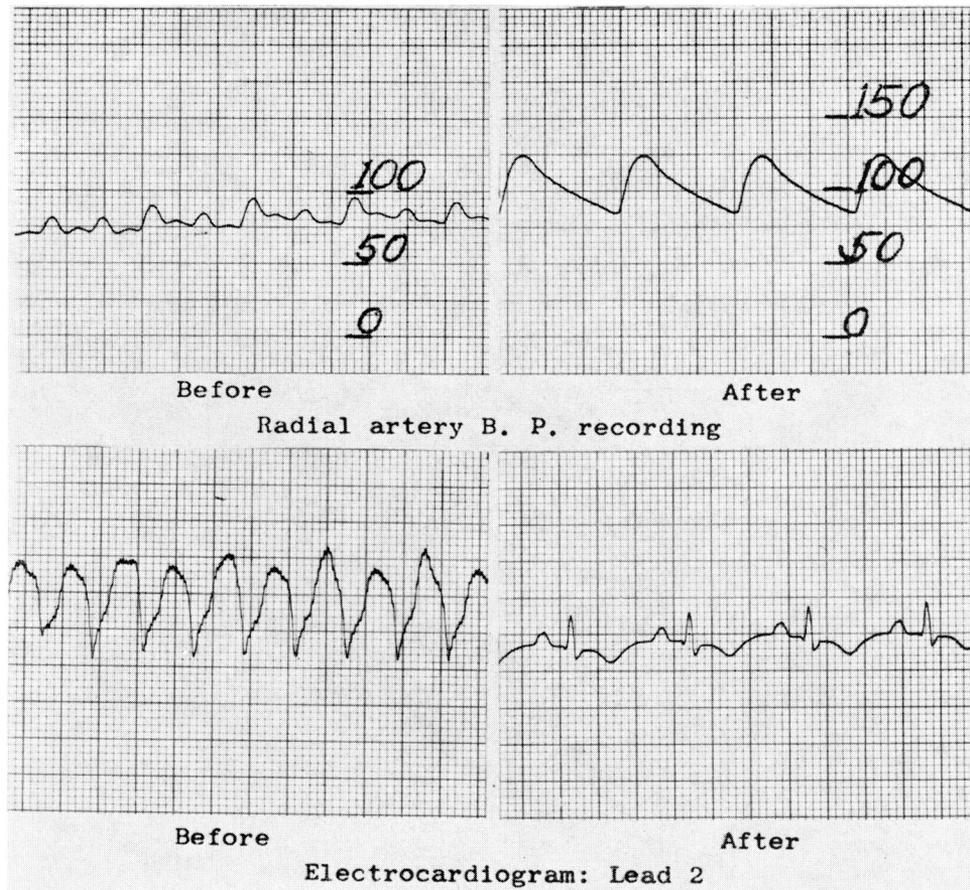


Figure 1

Case 4. Intra-arterial pressure curve (above) and electrocardiogram, lead II, (below) immediately before and after a 450-volt countershock. This figure was provided by Dr. A. L. Friedlich of the Massachusetts General Hospital.

improved at first but died 7 days after termination of the tachycardia because of renal and hepatic failure resulting from the prolonged congestive failure and circulatory collapse.

#### Case Reports

Two illustrative cases are reported in detail.

##### Case 6

A 64-year-old man was admitted to another hospital on May 19, 1961, because of an acute myocardial infarction. Ventricular tachycardia at 150 beats per minute began on the day of admission and was associated with hypotension. The arrhythmia stopped after 1 day of treatment with quinidine sulfate. In the next 3 weeks he suffered transitory oliguria and azotemia, severe pulmonary

edema treated by digitalization, low-grade pulmonary infection treated with tetracycline, and massive pedal edema treated with chlorothiazide without supplementary potassium.

On June 15, after recovery from these complications and 4 weeks after the infarction, ventricular tachycardia recurred at a rate of 122 per minute, and the systolic blood pressure dropped from 120 to 80 mm. Hg. Digitalis was stopped, and during the next few days he received intravenous potassium chloride (80 mEq.), oral quinidine sulfate (for 24 hours) until the blood concentration was 12 mg. per cent (0.3 Gm. and then 0.4 Gm. every 2 hours), oral procaine amide (1 Gm. and then 0.5 Gm. every 4 hours for 24 hours), and finally intravenous procaine amide until irregular multifocal ventricular beats appeared (1.8 Gm. in 100 minutes). Despite these measures the tachycardia persisted between 118 and 140 per minute as did

a low blood pressure that required vasopressor therapy.

After 4 days of tachycardia the patient was transferred to the Beth Israel Hospital for external countershock. On admission the heart rate was 128 (fig. 2A) and the blood pressure was 90/70. He appeared ashen but was alert and oriented. There were a systolic gallop rhythm and pulmonary congestion.

The patient was taken promptly to the operating room, the systolic pressure was raised to 110 mm. Hg by the intravenous infusion of norepinephrine, and he was anesthetized for a few minutes with intravenous sodium thiopental. An external countershock of 250 volts was given whereupon the tachycardia stopped. An atrioventricular nodal pacemaker appeared promptly at a rate of 90 per minute (fig. 2B); in 5 minutes sinoatrial rhythm returned at a rate of 78 per minute (fig. 2C).

Following termination of the tachycardia the patient did well. Occasional ventricular premature beats occurred, which had the same configuration as the complexes during the tachycardia, confirming the diagnosis of ventricular tachycardia (fig. 2D). Prophylactic oral quinidine sulfate was continued. For a few days the systolic blood pressure occasionally dropped below 90 mm. Hg but it then stabilized between 100 and 120. Persistent slight congestive failure cleared and the patient was ambulatory and feeling well at the time of discharge. The tachycardia has not recurred in the subsequent 5 months.

#### Case 8

A 51-year-old woman with mitral stenosis due to rheumatic heart disease had had three episodes of atrial fibrillation with rapid ventricular rates in the previous 4 years. The attacks were all terminated within 2 weeks by quinidine and digitalis, and she had no other cardiac symptoms.

On October 24, 1961, she entered the Beth Israel Hospital because of another episode of atrial fibrillation with ventricular rates between 160 and 200 per minute, which had persisted for 6 weeks despite massive doses of quinidine and digitoxin. She had been largely confined to bed because of dyspnea, fatigue, and palpitation and there were roentgenologic signs of cardiac dilatation and pulmonary congestion, but the blood pressure was normal.

Although the patient was not desperately ill, her disability and discomfort from the rapid rates over so long a time impelled us to use countershock for this refractory arrhythmia before her condition deteriorated further. She was taken promptly to the operating room and anesthetized with intravenous sodium thiopental. An initial countershock of 250 volts failed to stop the atrial fibrillation

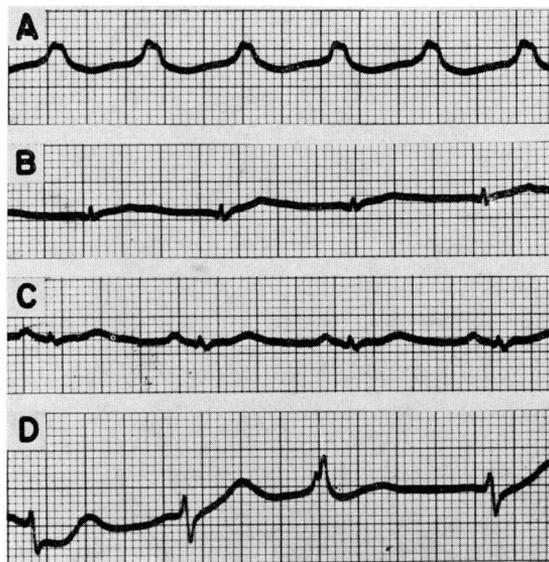


Figure 2

Case 6. Electrocardiograms, lead II, showing (A) ventricular tachycardia at 128 per minute; (B) atrioventricular nodal rhythm immediately after countershock of 250 volts; and (C) normal sinus rhythm 5 minutes later. (D), lead  $V_E$  3 hours later, shows normal sinus rhythm and a ventricular premature beat. This beat is of the same configuration as the complexes in this lead during the tachycardia, thereby confirming the diagnosis of ventricular tachycardia.

although it temporarily slowed the ventricular rate. Progressively larger countershocks were applied over the next 7 minutes until the sixth one, of 650 volts, was followed by normal sinus rhythm (fig. 3). For several seconds after some of the shocks, ectopic ventricular beats were noted; occasionally they were multifocal and there was one short bigeminal run and one burst of two ectopic beats in succession. Frequent atrial premature beats were also observed after termination of the atrial fibrillation, but these persisted throughout her hospitalization.

Following recovery from anesthesia the patient felt completely well. Studies of thyroid function were normal. Quinidine and digitoxin were given prophylactically and the patient was discharged within 2 days. Atrial fibrillation has not recurred in the subsequent 2 months.

#### Discussion

These successful experiences demonstrate the value of external electric countershock in terminating supraventricular and ventricular tachycardia and atrial fibrillation. External

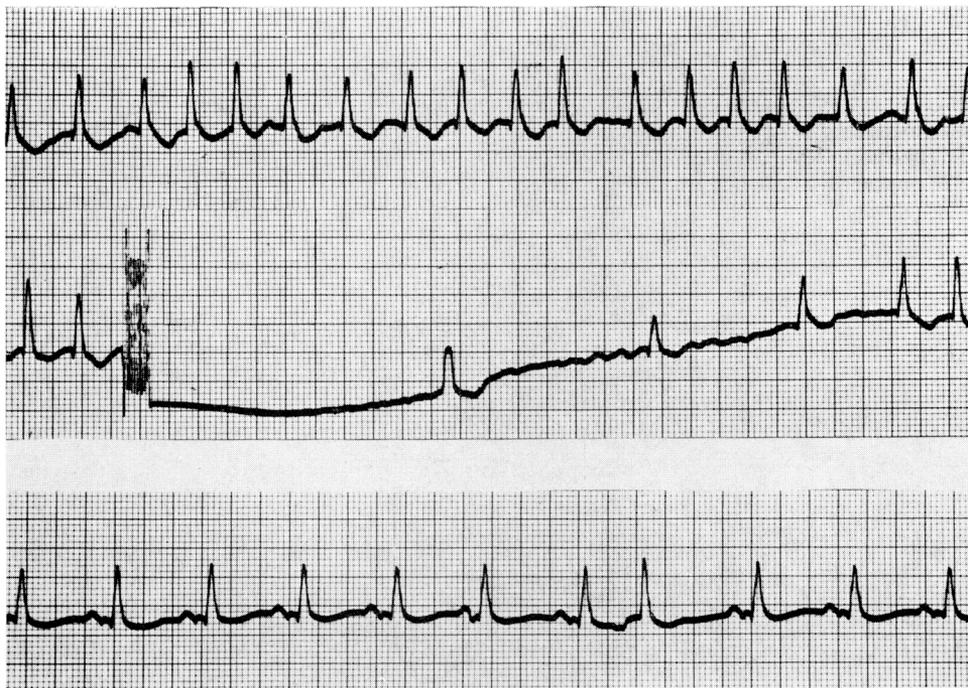


Figure 3

Case 8. Electrocardiogram, lead III. The two top strips are continuous; they show rapid atrial fibrillation, a 350-volt countershock, and subsequent transient ventricular slowing but persistent atrial fibrillation. The bottom strip, a few minutes later, immediately after a 650-volt countershock, shows normal sinus rhythm and an atrial premature beat.

countershock has already become established as a safe and effective emergency measure for resuscitating the unconscious patient in cardiac arrest from ventricular fibrillation. The present experiences demonstrate its wider usefulness, combined with anesthesia when necessary, in the deliberate termination of tachycardias of any kind. This technic is indicated when the patient's condition is desperate or intolerable and drug therapy is ineffective, too slow, or otherwise inadvisable. Application of this immediately effective procedure may well be safer at times than the administration of large doses of anti-arrhythmic drugs.

It should be emphasized that the application of external countershock carries definite risks. In the first place, general anesthesia, although brief, entails a significant risk in desperately ill cardiac patients. Secondly, ventricular fibrillation or standstill may follow countershock: fibrillation was produced

in patients 3 and 7, prodromal multifocal ventricular premature beats in patient 8, and persistent ventricular standstill from which the patient died (case 7). To be sure, ventricular fibrillation and standstill can ordinarily be handled effectively with further external countershocks or with external stimulation if the heart is not too severely damaged or anoxic.

Another problem in the management of refractory tachycardia with countershock is the possibility of immediate recurrence, as in case 5. To prevent recurrent tachycardia anti-arrhythmic drugs, such as intravenous procaine amide, may be given concomitantly. Furthermore, countershocks may be repeated as necessary so long as the patient is unconscious. Repeated countershocks, even in rapid succession, have been found to be well tolerated in these patients, as they were in many patients with repeated Stokes-Adams attacks due to ventricular fibrillation. Indeed, one

patient received over 300 countershocks in a 2-month period without ill effects.<sup>2</sup>

#### Summary

External electric countershock has been used to terminate refractory supraventricular and ventricular tachycardia and atrial fibrillation in eight patients. General anesthesia is necessary when the patient is conscious. This technic is indicated when the patient's condition is desperate or intolerable and drug therapy is ineffective, too slow, or otherwise inadvisable.

#### References

1. ZOLL, P. M., LINENTHAL, A. J., GIBSON, W., PAUL, M. H., AND NORMAN, L. R.: Termination of ventricular fibrillation in man by externally applied electric countershock. *New England J. Med.* **254**: 727, 1956.
2. ZOLL, P. M., LINENTHAL, A. J., AND ZARSKY, L. R. N.: Ventricular fibrillation: Treatment and prevention by external electric currents. *New England J. Med.* **262**: 105, 1960.
3. ZOLL, P. M., PAUL, M. H., LINENTHAL, A. J., NORMAN, L. R., AND GIBSON, W.: Effects of external electric currents on the heart. Control of cardiac rhythm and induction and termination of cardiac arrhythmias. *Circulation* **14**: 745, 1956.
4. ALEXANDER, S., KLEIGER, R., AND LOWN, B.: Use of external electric countershock in the treatment of ventricular tachycardia. *J.A.M.A.* **177**: 916, 1961.



It even happens that a fact or an observation stays a very long time under the eyes of a man of science without in any way inspiring him; then suddenly there comes a ray of light, and the mind interprets the fact quite differently and finds for it wholly new relations. The new idea appears, then, with the rapidity of lightning, as a kind of sudden revelation; which surely proves that in this case the discovery inheres in a feeling about things which is not only individual, but which is even connected with a transient condition of the mind. The experimental method, then, cannot give new and fruitful ideas to men who have none; it can serve only to guide the ideas of men who have them, to direct their ideas and to develop them so as to get the best possible results. The idea is a seed; the method is the earth furnishing the conditions in which it may develop, flourish and give the best of fruit according to its nature. But as only what has been sown in the ground will ever grow in it, so nothing will be developed by the experimental method except the ideas submitted to it. The method itself gives birth to nothing.—CLAUDE BERNARD. *An Introduction to the Study of Experimental Medicine*. New York, The Macmillan Company, 1927, p. 34.