VENTRICULAR fibrillation is the most serious of all arrhythmias. It was first described in 1849 by Ludwig and Hoffa. Its presence even for a short interval is usually incompatible with life. With the onset of ventricular fibrillation there is an immediate cessation of circulation with a series of rapid irregular undulations that replace the coordinated contractions of the ventricle. These oscillations become gradually less marked until they cease altogether.

Ventricular fibrillation may occur in patients during anesthesia; in cases of drowning and electrocution; in Stokes-Adams attacks; in toxicity states due to drugs such as digitalis, quinidine, and procaine amide; during cardiac catheterization; terminally in ventricular tachycardia; and in coronary artery disease.

Sudden death due to coronary occlusion is by no means always in direct proportion to the extent of the infarction. Stroud and Feil reported that in 50 per cent of the patients dying suddenly with coronary disease, death was due to a sudden paroxysm of ventricular fibrillation. Spontaneous recovery from ventricular fibrillation is rare although several cases have been reported including one in acute myocardial infarction.

The authors were fortunate enough to be in attendance at the bedside in the following case.

**Case Report**

A 45-year-old housewife and librarian was admitted to the Bryn Mawr Hospital on April 26, 1958, complaining of severe substernal chest pain. She was described as an apprehensive and tense individual with a history of transient labile hypertension. Her husband had died of a myocardial infarction several years prior to her admission.

On the afternoon of admission, while weeding in her garden at approximately 1:30 p.m., she suddenly experienced severe crushing anterior chest pain. Her physician arrived within 15 minutes and noted that the patient was pale, sweaty, slightly cyanotic, and pulseless. Heart sounds were barely audible and the blood pressure was 60/20. An injection of morphine sulfate 15 mg. and atropine sulfate 0.4 mg. relieved the pain somewhat and her pulse improved. She vomited copiously.

At 2:15 p.m. she arrived at the Bryn Mawr Hospital and still experienced anterior chest pain with some right arm pain; the blood pressure was 120/70. At 2:24 p.m. leads I, II, III, aVR, aVL, and aVF of the recording electrocardiogram had shown changes diagnostic of a recent posterior myocardial infarction and chest lead V1 was being recorded. At that moment the patient said, "Oh my, something's happening!" Within a few seconds she became unconscious, developed opisthotonus, and ceased to breathe. The heart sounds were inaudible. Clinically the patient appeared dead. The recording electrocardiogram showed ventricular fibrillation (fig. 1).

Nasal oxygen was started, the chest was pounded, and artificial respiration was attempted while the cardiac pacemaker and defibrillator were obtained from another floor. The electrocardiogram continued to reveal ventricular fibrillation during the 6-minute interval required to obtain the defibrillator and attach the electrodes externally to the patient's chest. Actually 4 minutes of ventricular fibrillation were recorded by the electrocardiogram. Short intervals during the 6 minutes were visually monitored but were not recorded (fig. 2). Three electric countershocks of 350 volts of 60-cycle alternating current of 0.15-second duration each were given at 10-second intervals at 2:30 p.m. with the Zoll-Electrodyne external defibrillator.

Manufactured by the Electrodyne Company, Norwood, Mass.
The electrocardiogram about 1 minute later showed complete atrioventricular block and an idioventricular rhythm. At 2:32 p.m. the patient was in regular sinus rhythm (fig. 3) and had regained consciousness.

She was very apprehensive and at 2:38 p.m. vomited copious amounts of fluid. By 2:50 p.m. the apprehension had increased and numerous premature ventricular contractions were noted. She was given sodium amytal 250 mg. and procaine

**Figure 1**

The 6-limb leads of the electrocardiogram were diagnostic of an acute posterior myocardial infarction. Lead V₁ taken at 2:24 p.m. revealed ventricular fibrillation.

**Figure 2**

During the 6 minutes that ventricular fibrillation persisted, it was recorded on lead II. Representative segments of the tracing during this interval are shown.
Figure 3

The heart was externally defibrillated with countershock at 2:30 p.m. Idioventricular rhythm was noted at 2:31 p.m., when the electrocardiogram was reconnected and regular sinus rhythm, was reestablished by 2:32 p.m.

amide 200 mg. intravenously at that time. The blood pressure was 152/110. The apprehension and premature contractions continued, and at 2:55 p.m. she was given morphine sulfate, 10 mg. hypodermically. At 3:05 p.m. atropine sulfate, 0.4 mg. hypodermically, and quinidine gluconate, 200 mg. intramuscularly, were administered, the blood pressure then being 114/90. Soon the apprehension and premature contractions diminished and the patient’s condition became less critical. The pulse, which was 120 immediately following restoration of regular sinus rhythm, fell to levels of 88 within 1 hour. The patient was quite talkative during the few hours after regular sinus rhythm had been restored.

The atropine was continued every 6 hours for the first day only, and quinidine 200 mg. by mouth every 6 hours was continued for weeks. The patient vomited small amounts frequently during the first few hours and the temperature rose to 101 F. She was given intramuscular penicillin. The temperature was normal within 3 days. Serial tracings were diagnostic of a posterior myocardial infarction (fig. 4). Except for a leukocytosis of 14,400 with a normal differential count, a sedimentation rate of 22 mm. per hour, and a serum cholesterol of 292 mg. per cent the routine laboratory studies were normal.

Anticoagulant therapy was delayed 48 hours lest a hemorrhagic pericarditis develop secondary to electrical burns from the shocks. Her skin continued to be pale gray for about 4 days, but gradually improved. Although she spoke coherently during the first 4 days of her hospitalization, she has an amnesia for events that occurred during that time. Two circular first-degree burns outlining the area where the electrodes were applied were noted; these healed by the sixth day.

The patient was discharged on May 28, 1958, 32 days after admission. The remainder of her convalescence was uneventful. There have been no motor or sensory defects noted in this patient, either while in the hospital or since her discharge. The patient’s ventricular fibrillation continued for 6 minutes and her unconsciousness continued for 8 minutes. She returned to her employment as a school librarian in September 1958 and except for some anterior chest discomfort that occurs with excitement is otherwise well (fig. 5).

Discussion

It is important to be aware that ventricular fibrillation may be the cause of so-called “sudden death” in patients with acute myocardial infarction. Promptly distinguishing this arrhythmia from cardiac standstill by means of the electrocardiogram and immediate treatment may result in successful resuscitation. Delay constitutes the major limitation in achieving success. External defibrillation seems more feasible in the treatment of ventricular fibrillation in coronary occlusion, since thoracotomy with cardiac massage in this age group could of itself prove fatal. At
least 5 cases of ventricular fibrillation in acute myocardial infarction are reported, however, that were treated by thoracotomy and cardiac massage and then by direct defibrillation with survival. Our own experience in treating 8 such patients by thoracotomy, cardiac massage, and direct defibrillation of the heart did not result in the survival of any patient for more than 13 hours; although in a few instances regular sinus rhythm was restored, shock and unconsciousness persisted in all to death.

Historically the first successful defibrillation by countershock was performed by Prevost and Batelli on the exposed dog heart in 1899. Hooker et al., Wiggers, and Guyton and Satterfield showed that currents if strong enough could successfully defibrillate the heart through the unopened chest and that 60-cycle alternating current was the most satisfactory. Beck, Pritchard, and Feil were the first to terminate ventricular fibrillation in man by the direct application of countershock current to the heart through the opened chest. Zoll and his group were the first to terminate ventricular fibrillation in man by the external application of countershock current across the unopened chest. Zoll and Kouwenhoven and his group have devised instruments to be utilized externally in the treatment of patients with this arrhythmia.
Experimentally, high voltage, rapid successive shocks without adequate rest periods have produced cardiac damage as manifested by abnormal QRS complexes, decreased blood pressure, spontaneous recurrent ventricular fibrillation, and actual thermal destruction of muscle.\'8\' Zoll, however, reported no untoward effects with countershock in numerous animals and no evidence of damage to the hearts in 3 patients autopsied who had been defibrillated multiple times.\'8, 19\n
There are at least 2 cases in children of ventricular fibrillation lasting as long as 24 and 45 minutes that were successfully defibrillated with survival. Both these cases, however, occurred in the operating room where immediate massage maintained the circulation until regular sinus rhythm was restored.\'15, 20\' Levine and Maton reported a case of ventricular fibrillation followed by asystole lasting 5 minutes in a patient with Stokes-Adams disease who recovered after the intracardiac injection of epinephrine.\'21\n
Experimentally, Weinberger et al. showed that if circulation was interrupted in cats for over 3 minutes 25 seconds that some permanent damage to the central nervous system occurred. No cat survived when circulation was interrupted over 8 minutes 45 seconds.\'22\' Our case had no detectable defect after 6 minutes of interrupted circulation except for the persistent amnesia for events of the first 4 days of hospitalization. The amnesia may at least in part be due to the sedation as the memory loss began with the initial injection of morphine prior to admission.

External defibrillation has been successful with patients surviving in cases of ventricular fibrillation occurring during cardiac catheterization, in drug toxicity, and in Stokes-Adams attacks.\'16, 17, 19, 23, 24\' Patients with acute myocardial infarction and ventricular fibrillation have been externally defibrillated a number of times but defibrillation resulted in either ventricular standstill that did not respond to external cardiac pacemakers or recurrent ventricular fibrillation and death.\'16

We have been unable to find another case of acute myocardial infarction with ventricular fibrillation that has been restored to regular sinus rhythm with external defibrillation and survived. It is unlikely that a patient with this catastrophe will often be observed within the time permitting resuscitation. We were fortunate that circumstances prevailed permitting success in this case.

**Summary**

A patient with an acute myocardial infarction was observed who gave all the appearances of being dead; the electrocardiogram attached at the time showed ventricular fibrillation, which persisted for at least 6 minutes.

Regular sinus rhythm was restored within 2 minutes after external defibrillation with the Zoll-Electrodyne External Defibrillator with use of 60-cycle alternating current at 350 volts every 10 seconds for 3 shocks.

No other similar case that survived has been found in the literature.

The necessity of immediate recognition of ventricular fibrillation in acute myocardial infarction is pointed out in cases of "sudden death."

The preference of external electrical defibrillation over direct heart defibrillation is discussed.

**Summario in Interlingua**

Esseva observate un patiente con acute infarcimento myocardial qui exhibita omne signos de esser morte. Le electrocardiogramma in progresso a ille tempore monstrava fibrillation ventricular que persisteva durante al minus 6 minutas.

Un regular rhythmus sinusalis esseva restaurate intra 2 minutas per medio de disfibrillazione externe effec-tute con le apparato Zoll del compania Electrodyne, utilisante un currente alterante de 60 cyclos a 350 volt e applicante un triple choc a intervallas de 10 secundas.

Nule simile caso, con superviventia del patiente, se trova in le litteratura.

Es signalate le necessitate de recognoscer fibrillation ventricular in acute infarcimento myocardial in cases de "morte substantee."

Le avantages del externe disfibrillazione electri es discutite in comparation con le direete disfibrillazione del corde.

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Successful External Electrical Defibrillation in Acute Myocardial Infarction:
Report of a Case

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