Treatment of Stokes-Adams Disease by External Electric Stimulation of the Heart

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An external cardiac pacemaker was developed and was used to stimulate the heart electrically in a series of patients with recent Stokes-Adams syncope. It resuscitated patients from attacks due to ventricular standstill; it maintained an adequate circulation during persistent ventricular standstill; and it prevented the recurrence of irregular ventricular tachycardia. Several patients have now survived for many months without recurrent syncope. Such long survivals suggest that the periods of cardiac disturbance which cause syncope may subside if the patient is kept alive during the crucial period.

The syncopal attacks of Stokes-Adams disease are unpredictable and often present desperate therapeutic problems. These episodes of circulatory arrest due to ventricular standstill, tachycardia or fibrillation may become very frequent and severe. A fatal attack is always imminent. Furthermore, the available therapy with drugs and cardiac puncture is dangerous and may be ineffective in resuscitating patients from individual attacks or in maintaining adequate ventricular rhythms.

We have developed a new therapeutic approach to this serious problem. This consists of electric stimulation of the heart by means of an externally-applied cardiac pacemaker which terminates ventricular standstill and maintains regular externally-paced ventricular beats until an adequate, spontaneous ventricular rhythm reappears. After experimental studies in animals established the efficacy and safety of this procedure, it was used in the treatment of patients with recent Stokes-Adams attacks.

Apparatus

The cardiac pacemaker* is a modification of existing physiologic stimulators. It produces monophasic, rounded electric impulses with an average duration of 2 to 3 milliseconds and with the entire wave form lying above the baseline. A variety of other wave forms (monophasic and biphasic spike, monophasic and biphasic rectilinear, and sinusoidal) were found to be less effective. The apparatus is light, portable and simple to use, with two controls permitting variation of frequency from 30 to 180 stimuli per minute and of amplitude from 0 to 150 volts. The low internal impedance of the instrument (approximately 50 ohms) permits adequate power output even across low body resistances. In the design of the instrument, the line voltage is carefully isolated to prevent its accidental transfer to the output circuit.

The pacemaker is attached to the patient by two output wires connected to 3 cm. circular chest electrodes. The electrodes may be placed in any positions on the chest that provide current flow across the heart. For convenience, the negative electrode is placed at the point of maximum cardiac impulse, and the positive electrode symmetrically, on the right anterior chest. Good electric contact is made with electrode paste and the electrodes are held in place by a rubber strap encircling the chest.

Satisfactory recordings of the electric stimuli from the cardiac pacemaker and of the activity of the heart can be obtained electrocardiographically. The electric stimuli often displace the base line markedly and distort the electrocardiogram or mask it completely. However, recording difficulties are avoided with late model electrocardiographs that do not have

* The Cardiac Pacemaker is manufactured by the Electrodyne Company, Norwood, Mass.
condensers in the input circuit. The patient, the pacemaker and the electrocardiograph must have a common ground. This is conveniently provided by wires between the electrocardiograph and the pacemaker, and between the pacemaker and an external ground. The patient is grounded by the negative output wire from the pacemaker. It is preferable not to use the conventional ground wire from the electrocardiograph to the right leg. The most satisfactory recordings are obtained with lead aVF at half-normal standardization in order to diminish the amplitude of the deflections produced by the stimuli. Occasionally, a fuse in the patient circuit of the electrocardiograph may be blown and should be replaced.

In every patient the threshold of effective stimulation is first determined. With the frequency of stimulation set between 60 and 90 per minute, the amplitude control is increased from 0 until cardiac responses are obtained. Thereafter, effective cardiac stimulation is maintained by amplitudes slightly above the threshold level. The current and voltage of effective stimuli, measured with an oscilloscope at the chest electrodes, ranged from 75 to 150 milliamperes and from 45 to 100 volts in our patients.

Case Reports

The cardiac pacemaker has been used in 14 patients with recent Stokes-Adams attacks. Eleven patients were treated with short or prolonged periods of stimulation for resuscitation from syncope or for maintenance of an adequate ventricular rhythm. In two additional patients trial stimulation produced effective beats, but treatment did not become necessary. In the fourteenth patient the efficacy of stimulation was not determined.

Case 1.* E. B. (B.I.H. No. M39623), a 63 year old man, had complete heart block and occasional syncopal attacks for two years. He entered the Beth Israel Hospital because of increasingly frequent episodes of syncope, culminating in one attack lasting 20 minutes.

On admission, external electric stimulation was found to be effective in producing cardiac responses during slow idioventricular rhythm (fig. 1a). Shortly after this trial, ventricular standstill and syncope occurred. The external pacemaker was started immediately and the patient revived at once. Each stimulus produced a ventricular response in the electrocardiogram and a pulse beat with a pressure of 130/80. The dependence of the electrocardiographic response, the peripheral pulse and consciousness upon the electric stimuli was repeatedly demonstrated by varying the rate of stimulation. Whenever the external pacemaker was stopped for short test intervals, syncope due to ventricular standstill recurred (fig. 1b). External stimulation was necessary for the next 90 hours to maintain an effective circulation. Initially the patient was very restless and complained bitterly of the electric shocks and the associated twitches of the pectoral muscles. This discomfort was relieved by meperidine hydrochloride (Demerol); with continued stimulation it became much less severe.

On the third day, stimulation was inadvertently interrupted for two and one-half minutes. A prolonged Stokes-Adams attack occurred, manifest by circulatory and respiratory arrest, convulsions and syncope. Resumption of external stimulation revived the patient immediately, but it was more than 18 hours before complete mental clearing occurred.

Various medications were given in an effort to arouse a spontaneous, sustained idioventricular rhythm. Sustained idioventricular beats finally appeared when the rate of intravenous administration of epinephrine was raised to 8 micrograms per minute. When these beats persisted at an effective rate (fig. 1c), both the external stimulation and the administration of epinephrine were stopped.

The slow spontaneous rhythm persisted for the next nine days. Occasionally the pacemaker was tested and was found to be effective. During this time, there were no signs of circulatory or cerebral damage and the patient felt well.

Suddenly, on the thirteenth hospital day, the patient had another Stokes-Adams attack; respiration stopped, heart sounds and motor activity disappeared, and the patient seemed dead. Because of a lapse in readiness, there was a delay of five minutes before stimuli were reapplied. Resumption of external stimulation resulted in immediate ventricular responses and return of peripheral circulation. Thereafter, the heart rate was maintained by the pacemaker, but coma, restlessness, coarse tremor and high fever indicated progressive cerebral damage. Repeated attempts to arouse spontaneous ventricular beats with intravenous epinephrine and norepinephrine failed. After 108 hours of stimulation, the blood pressure dropped despite increasing rates of norepinephrine infusion; respirations and ventricular responses to the stimuli ceased, and the patient died.

Case 2. I. L. (B.I.H. No. M37752), an 80 year old woman with complete heart block for 30 years, was admitted to the Beth Israel Hospital because of recent, repeated syncopal attacks. These were due to ventricular standstill, rapid ventricular tachycardia or both (fig. 2a).

During complete heart block, external electric stimulation produced synchronous electrocardiographic responses and pulse beats. Accordingly, stimulation was applied, at first intermittently to
resuscitate the patient from syncope, and then almost continuously for 24 hours to prevent syncope. The electric stimuli produced moderate contractions of the thoracic muscles, particularly the left pectorals. Initially these movements startled the patient and the stimuli were somewhat painful. After moderate sedation with meperidine hydrochloride and continued stimulation, the patient tolerated the procedure with little discomfort.

The efficacy of external stimulation in preventing

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Fig. 2. Case 2. Lead aVF; standardization half normal (1 mv. equals 5 mm.). (a) Stokes-Adams attack due to rapid ventricular tachycardia and ventricular standstill: Paroxysm of rapid ventricular tachycardia (rate approximately 230 per minute) followed by ventricular standstill for 3.6 seconds before resumption of idioventricular rhythm. Syncope occurred during the tachycardia and standstill. (b) Effective stimulation at varying rates: Each stimulus (E) produced a ventricular response (V). Each electrocardiographic ventricular response depended upon an electric stimulus as shown by the constant relationship at widely varying rates of stimulation. (c) Ventricular standstill during temporary interruption of stimulation: During a test period of 6.6 seconds no stimuli were applied. No spontaneous ventricular contractions occurred during this interval and the patient lost consciousness. The independent auricular rhythm continued. Resumption of stimuli immediately gave ventricular responses and the patient revived at once. (d) Rapid ventricular tachycardia during temporary interruption of stimulation: The paroxysm of ventricular tachycardia lasted 5.4 seconds, produced syncope, and terminated spontaneously. After a pause of one second spontaneous multifocal ventricular beats occurred. (e) Ineffective stimulation during paroxysm of rapid ventricular tachycardia: This paroxysm occurred shortly after the one shown in figure 2d during the same test period. The two paroxysms are almost identical. Stimuli (E) applied during the second half of this paroxysm produced no cardiac effect. After the paroxysm had stopped, apparently spontaneously, stimuli evoked ventricular responses (V).
FIG. 2.
Stokes-Adams attacks was repeatedly demonstrated during the 24 hour period. During stimulation syncope never occurred. The electric stimuli produced uninterrupted, effective ventricular beats at rates which varied from 50 to 100 per minute (fig. 2b). The spontaneous, irregular ventricular activity did not break into the slower, externally-paced rhythm. When stimulation was interrupted for short test periods, syncope occurred due to the absence of spontaneous ventricular contractions (fig. 2c) or to rapid ventricular tachycardia (fig. 2d). Although stimulation was effective in preventing rapid ventricular activity, it failed to terminate such paroxysms (fig. 2e).

After 24 hours of external stimulation, the periods of ventricular standstill and rapid ventricular activity gradually disappeared. The external pacemaker was then stopped completely and a slow, idioventricular rhythm continued. The patient has now survived 12 months without further syncope.

Case 3. L. W. (B.C.H. No. 1481070), a 71 year old woman with occasional Stokes-Adams attacks for two years, was admitted to the Boston City Hospital because of their increasing frequency. On admission, electrocardiograms showed complete heart block and short paroxysms of rapid, irregular ventricular activity. Soon thereafter, the patient had two major syncopal attacks lasting four minutes and one minute. They were due to prolonged ventricular fibrillation followed by short periods of ventricular standstill.

Throughout her hospitalization, atrioventricular conduction changed frequently, ranging from normal P-R intervals with regular sinus rhythm to complete heart block. The external cardiac pacemaker produced electrocardiographic ventricular

![Figure 3](http://circ.ahajournals.org/)

**Fig. 3.** Case 3. Lead aVF; standardization half-normal (1 mv. equals 5 mm.). (a) *Interruption of idio-ventricular rhythm by faster externally-paced beats:* The intensity of the electric stimuli (E) was progressively increased until ventricular responses (V) were produced. Three spontaneous idioventricular complexes (cycle length 2.04 seconds) are seen during ineffective stimulation; during effective stimulation at a faster rate (cycle length 1.11 seconds) they are suppressed. (b) *Resumption of idioventricular rhythm following external stimulation:* When stimulation was stopped, the spontaneous ventricular pacemaker reappeared after a pause of 4.64 seconds and regained its previous rate within three beats. (c) *Effective stimulation at varying rates:* Each stimulus (E) produced a ventricular response (V). Each electrocardiographic ventricular response depended upon an electric stimulus as shown by the constant relationship at widely varying rates of stimulation. (d) *Ventricular standstill during temporary interruption of stimulation:* During a test period of 5.5 seconds no stimuli were applied. No spontaneous ventricular contractions occurred during this interval and the patient lost consciousness. The independent atrial rhythm continued. Resumption of stimuli immediately gave ventricular responses and the patient revived at once.
responses and synchronous pulse beats during complete heart block (fig. 3), partial heart block and normal sinus rhythm (fig. 4).

Frequent and severe syncopal attacks occurred during complete heart block. Although rapid irregular ventricular activity occasionally recurred, the predominant mechanism of syncope was ventricular standstill. On eight occasions in two months prolonged stimulation was necessary for periods ranging from 6 to 48 hours to maintain an adequate circulation until a spontaneous sustained pacemaker reappeared. The intervals of stable rhythms between periods of stimulation varied from 2 hours to 19 days. Heavy sedation with narcotics, barbiturates and paraldehyde was required to control severe discomfort produced by the stimulation.

The patient's clinical course was complicated by severe, transient pulmonary infections. She has now survived for seven months since the initial resuscitation by the cardiac pacemaker and she has had no syncope during the past five months. Her present condition in a convalescent hospital is satisfactory, and she shows no deleterious effects on her mental or cardiac function as a result of either the prolonged stimulation or syncope.

Case 4. R. A.* (B.I.H. No. M34234), a 65 year old man with complete heart block, was admitted to the Beth Israel Hospital because of congestive heart failure and angina pectoris. Repeated Stokes-Adams attacks due to ventricular standstill and rapid irregular ventricular activity occurred in the hospital.

External electric stimulation was effective and stopped syncopal attacks. For five days, because of recurrent ventricular standstill, repeated electric stimulation was necessary until an adequate sustained idioventricular rhythm reappeared.

The patient lived for 10 months, free of syncope and without increase in his cardiac disability. He then died suddenly.

**Fig. 4.** Case 3. Lead aV1; standardization half-normal (1 mv. equals 5 mm.) External electric stimulation during normal sinus rhythm. The three strips were taken during a period of continuous stimulation. (a) After three sinoatrial beats (R) stimulation at a similar rate was started and immediately produced ventricular responses (V). (b) During the externally-paced rhythm at threshold voltage the fourth stimulus was ineffective and was followed by an escape beat (R) from the sinoatrial pacemaker. (c) After 25 seconds of stimulation at the same rate (E–E = 1.02 sec.) the sinoatrial rate increased (R–R shortened from 1.03 to 0.95 sec.) and competition is observed between the sinoatrial and external pacemakers. The normal pacemaker maintained control of the ventricles for seven beats (R). The first sinoatrial beat is partially masked by a stimulus. During this sinoatrial rhythm the electric stimuli were ineffective because they fell in the refractory phase of the ventricles. As soon as a stimulus (the seventh E) arrived during the responsive phase of the ventricles it was effective and the external pacemaker resumed control of the heart.
Case 5. D. S.* (B.I.H. No. M33443), a 75 year old man with complete heart block, was admitted to the Beth Israel Hospital because of repeated Stokes-Adams attacks due to ventricular standstill and rapid irregular ventricular tachycardia. Over a four hour period 34 cardiac injections of epinephrine were given.

External electric stimulation was effective and stopped syncopal attacks during a 25 minute period. Stimulation then became ineffective and the patient died because of cardiac tamponade which resulted from the cardiac punctures.

Case 6. P. G. (B.C.H. No. 1485391), a 78 year old man, was admitted to the Boston City Hospital because of frequent dizzy spells for two weeks. Electrocardiograms showed varying degrees of partial atrioventricular block, and then complete block with idioventricular rates as slow as 16 beats per minute. Initially he had numerous dizzy spells, then he became unconscious and never fully recovered.

External electric stimulation was effective: an adequate circulation was maintained with a blood pressure of 118/60. Continued stimulation was necessary for 36 hours because ventricular standstill was observed whenever stimulation was interrupted. An adequate idioventricular rhythm reappeared following the intravenous administration of epinephrine at a rate of 4 micrograms per minute and stimulation was then stopped.

Ten hours later, however, the ventricular rate slowed markedly and shock supervened. At this time, stimulation again produced electrocardiographic ventricular complexes, but there were no associated pulse beats. Finally, the electrocardiographic ventricular responses to stimulation also failed and the patient died.

Case 7. B. S. (P.B.B.H. No. C9205), an 81 year old woman, entered the Peter Bent Brigham Hospital because of three syncopal attacks. After admission, cardiac arrest, requiring cardiac puncture in order to restore the beat and resuscitate the patient, occurred approximately 30 times. Electrocardiograms showed regular sinus rhythm with normal P-R intervals, 2:1 atrioventricular block and variable ectopic supraventricular pacemakers. Paroxysms of rapid, irregular ventricular tachycardia followed by ventricular standstill were observed to cause syncope.

Accordingly, on the second hospital day, external electric stimulation was applied. The stimuli produced synchronous ventricular complexes and pulse beats with the patient's usual pressure of 120/80. The electric stimuli produced only slight pectoral twitch and little discomfort.

Immediately upon effective stimulation the syncopal attacks stopped. Stimulation was discontinued after five hours and syncope did not recur until 19 hours later. The attacks again ceased when effective stimuli were reapplied. Stimulation was continued for eight hours and the patient then had no further attacks for eight days.

Finally on the tenth hospital day the patient had another Stokes-Adams attack and expired. Necropsy showed a small bloody pericardial effusion and pericarditis due to the cardiac punctures.

Case 8. A. R. (B.I.H. No. M42953), a 76 year old woman, entered the Beth Israel Hospital because of chronic lymphatic leukemia and congestive heart failure. She was known to have had complete heart block and occasional syncopal attacks for one year.

On the sixth hospital day, following a prolonged Stokes-Adams attack, external electric stimulation was applied during slow idioventricular rhythm. Electrocardiographic ventricular responses and pulse beats were observed synchronous with the electric stimuli. The patient was disturbed by the muscle contractions and the pain associated with stimulation. Electric stimulation was reapplied three times on this day for recurrent Stokes-Adams attacks due to rapid ventricular tachycardia. Although stimulation was ineffective during the paroxysms of tachycardia, it became effective immediately when they stopped.

Two days later, another prolonged Stokes-Adams attack occurred and consciousness never completely returned. During the next hour, multifocal ventricular beats and paroxysms of irregular ventricular tachycardia predominated. Periods of ventricular standstill which frequently followed ventricular tachycardia were repeatedly terminated by external stimulation. Electric stimulation was applied almost continuously for the next 10 hours. At first an effective ventricular rhythm was maintained with only infrequent interruptions by spontaneous ventricular beats. Near the end of this period of stimulation, however, spontaneous irregular ventricular activity increased until persistent ventricular tachycardia supervened and stimulation became ineffective. The blood pressure then fell, the temperature rose to 105.7° F. and the patient died.

Necropsy showed no evidence of cardiac damage attributable to electric stimulation.

Case 9. A. S. (B.C.H. No. 1481716), an 82 year old woman with a past history of complete heart block and Stokes-Adams attacks, was readmitted to the Boston City Hospital because of recurrent syncope. Shortly thereafter she became unconscious. Repeated convulsive seizures were observed due to ventricular fibrillation followed by ventricular standstill. In one episode, recorded electrocardiographically, ventricular fibrillation lasted for three minutes.

External electrical stimulation was applied effectively for 20 hours. Although the patient improved and became somewhat responsive, convulsive

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* Cases 4 and 5, the first two patients treated, have been reported in detail.1
seizures continued. Spontaneous, multifocal ventricular beats frequently broke through the externally-paced rhythm. Finally, during a test interruption of effective stimulation, a convulsion occurred, the stimuli were then ineffective and the patient died.

Case 10. A. L. (B.I.H. No. M42806), a 72 year old woman, entered the Beth Israel Hospital because of increasing cardiac pain and congestive failure. Although her rhythm had been normal and digitalis had not been given, after hospitalization she was found to have complete heart block with multifocal ectopic ventricular beats and paroxysms of irregular ventricular tachycardia. Two severe Stokes-Adams episodes occurred which were treated with intracardiac injections of hydroxymethylamphetamine hydrobromide (Paredrine).

External electric stimulation was effective during slow idioventricular rhythm. However, the patient found it too painful to permit continuous application. Over a six-day period, external stimulation was applied during eight syncopal attacks, and consciousness returned promptly on each occasion. Satisfactory electrocardiographic observations of the mechanisms of these attacks were not obtained.

Electric stimulation was ineffective during the last 50 seconds of another syncopal attack which was due to rapid irregular ventricular tachycardia. As soon as the tachycardia stopped, apparently spontaneously, the stimuli immediately evoked ventricular responses and pulse beats. Finally, the patient died during a Stokes-Adams attack in which electric stimulation, though applied promptly, was ineffective.

Necropsy showed no evidence of cardiac damage attributable to electric stimulation.

Case 11. M. W. (B.C.H. No. 1458085), an 83 year old woman with complete heart block and auricular fibrillation for over one year, was admitted to the Boston City Hospital because of congestive heart failure. Following a debilitating pneumonia, her idioventricular rhythm slowed to 15 beats per minute and she suffered repeated syncopal attacks due to ventricular standstill. These increased in frequency despite the administration of ephedrine and epinephrine in oil.

External electric stimulation during complete heart block was effective, but could not be continued, because it was too painful. Therefore, it was decided to apply the stimuli as a resuscitative measure at the onset of syncope. During a subsequent syncopal attack, external stimulation immediately produced synchronous pulse beats and consciousness returned at once. One hour later, however, the patient was found dead.

Case 12. B. B. (B.I.H. No. M42598), an 84 year old man, entered the Beth Israel Hospital because of repeated Stokes-Adams attacks. Electrocardiograms showed normal sinus rhythm with prolonged and variable P-R intervals, and atrioventricular nodal rhythm.

External electric stimuli were applied for several short periods and the threshold voltage for effective cardiac stimulation was determined. The stimuli were observed to be effective during both normal sinus rhythm and atrioventricular nodal rhythm: they replaced the spontaneous rhythms with externally-paced ventricular complexes and synchronous pulse beats.

The pacemaker was kept ready for use but syncope did not recur. The patient was discharged on the fourteenth hospital day without further stimulation. Major syncopal attacks have not recurred for seven months.

Case 13. J. K. (B.I.H. No. M43138), a 73 year old man with complete heart block for one month, entered the Beth Israel Hospital because of repeated Stokes-Adams attacks over a four-day period.

External stimuli were applied only for a few minutes; effective cardiac stimulation was observed during complete heart block and the threshold voltage of the stimuli was determined. The electrodes were then left in place on the chest for several days with the pacemaker in readiness for use in case of recurrent syncope. However, the patient had no more attacks and was finally discharged on the thirty-first hospital day without further stimulation. Major syncopal attacks have not recurred for eight months.

Case 14. J. C. (Mt. A.H. No. 104138), a 65 year old man with complete heart block, entered the Mt. Auburn Hospital twice in one month because of repeated syncopal attacks. Each time, external electric stimulation was tested during idioventricular rhythm. Excessive disturbance from these stimuli prevented satisfactory pulse observations, and the electrocardiograph was unsuitable. Therefore, stimulation was not continued; whether it produced cardiac responses was not determined. The patient died of cerebral damage eight hours after a prolonged episode of circulatory arrest.

The clinical course of this patient was uninfluenced by the attempts at external stimulation of the heart. He represents the only failure in this series to demonstrate effective cardiac stimulation. In this patient, early in our experience, the excessive disturbance from the stimuli was not adequately controlled by sedation. Furthermore, the electrocardiograph had a condenser-coupled input circuit which was found subsequently to be the cause of the difficulty in recording.

**Discussion**

All 14 of these patients with syncope had Stokes-Adams disease*: complete or partial

* We have followed the suggestion of Parkinson, Papp and Evans in calling this condition a disease rather than a syndrome.²
atrioventricular block was present in each case; syncopal attacks were due to ventricular standstill, very slow idioventricular beats, ventricular tachycardia, ventricular fibrillation or combinations of these mechanisms.

External electric stimulation was demonstrated electrocardiographically to be effective in 13 of these 14 patients; its efficacy in the fourteenth patient was not determined. Each effective stimulus produced a ventricular complex; variations in rate and short interruptions of stimulation, as well as stimulation at sub-threshold voltages, demonstrated that this relationship was not coincidental. Effective stimulation was applied during normal sinus rhythm, atrioventricular nodal rhythm, partial heart block and complete heart block with idioventricular rhythm. The interruption of these rhythms by the externally-paced ventricular complexes was additional evidence of the effectiveness of external stimulation.

External electric stimulation was applied in all 13 patients with Stokes-Adams disease because they had suffered recent syncopal attacks. In every instance the threshold voltage of effective stimuli was determined. Then, continuation of stimulation and its duration depended upon several factors: the frequency and severity of syncope, the nature of the spontaneous ventricular activity, and the tolerance of stimulation by the patient. In two of these patients stimulation was not continued after the threshold was determined; the remaining 11 patients were treated with short or prolonged periods of further stimulation.

Untreated Group. In two patients (cases 12, 13) the syncopal attacks were relatively infrequent and mild; the ventricular rhythms were regular and persisted at adequate rates; and stimulation produced moderate discomfort. Accordingly, after the thresholds of effective stimulation were determined, it was decided to keep the cardiac pacemaker in readiness for immediate use in case of recurrent syncope. Treatment with the pacemaker was not necessary thereafter, since syncope did not recur.

Short Treatment for Individual Stokes-Adams Attacks. In five additional patients it was also decided to reserve the pacemaker for their resuscitation from syncopal attacks. The disturbance produced by stimulation (cases 2, 8, 10, 11) and our limited experience (case 4) led to this decision even though the syncopal attacks were frequent or severe and the ventricular rhythms unstable.

Syncpe due to ventricular standstill or a very slow idioventricular rate was terminated immediately by stimulation. This striking response was observed countless times over a three-day period in case 4.

Paroxysms of irregular ventricular tachycardia or ventricular fibrillation were not stopped by stimulation (fig. 2e, cases 2, 8, 10). Whenever the irregular tachycardia stopped spontaneously, however, stimulation was immediately effective and shortened the period of ventricular standstill which occasionally followed.

For two reasons, the decision to treat individual Stokes-Adams attacks with short periods of stimulation involves the risk of fatality. First, the state of unfailing readiness for emergency stimulation that is necessary to avoid fatal delay in case of persistent ventricular standstill is impractical at the present time even in a hospital. Second, an attack of irregular ventricular tachycardia which is unaffected by stimulation may persist and be fatal. In view of the changing mechanisms of syncope often seen in these patients, the risk of persistent tachycardia is present even when standstill has been the predominant cause of syncope.

Prolonged Treatment. Because of the risk of fatality inherent in the short treatment of individual Stokes-Adams attacks, it is preferable to continue treatment beyond the time of resuscitation when syncope is frequent or severe and the ventricular rhythms are unstable. Furthermore, after resuscitation, continued stimulation is mandatory if an adequate spontaneous ventricular rhythm fails to appear when stimulation is stopped. Prolonged stimulation was applied in nine patients for periods of 25 minutes to 108 hours; in five patients (cases 1, 3, 4, 5, 6) because of ventricular standstill or very slow idioventricular beats; in three (cases 7, 8, 9) because of irregular ventricular tachycardia; and in one
(case 2) because of combinations of these mechanisms.

During persistent ventricular standstill or very slow idioventricular beats, continued stimulation after resuscitation maintained an adequate circulation and consciousness. Although slight transient depression of ventricular pacemakers was frequently observed following brief stimulation during idioventricular rhythm (fig. 3b), prolonged stimulation during ventricular standstill did not prevent the reappearance of spontaneous sustained pacemakers. In all patients but one (case 5), adequate sustained beats finally reappeared and stimulation was then stopped. An improved myocardial status resulting from the more adequate externally-paced rate may favor this recovery. On the other hand, the conditions producing standstill may be reversible and, when the patient is kept alive by the external pacemaker, recovery may occur either spontaneously or as a result of drug therapy.

Drug therapy may be useful at times in arousing and maintaining spontaneous ventricular pacemakers at adequate rates. We have occasionally observed these effects following the administration of ephedrine intramuscularly, and epinephrine and norepinephrine intravenously. Multifocal ventricular activity was produced by the intravenous administration of both epinephrine and norepinephrine; they must therefore be used cautiously.

The ability to sustain life in patients with Stokes-Adams disease by means of the external cardiac pacemaker has provided a heretofore unavailable opportunity to study quantitatively the effects of drugs on the ventricular pacemakers and to clarify some of the mechanisms of the disorder.3

Although stimulation did not resuscitate patients from paroxysms of irregular ventricular tachycardia, its continued application maintained a regular, externally-paced rhythm for long periods without interruption by ectopic ventricular activity. In two patients, (cases 2, 7) ventricular irritability finally subsided and stimulation was then stopped. One of these patients (case 7) died from a subsequent Stokes-Adams attack. In the two other patients (cases 8, 9) control of the ventricular irritability was never complete and both died.

Untoward Effects of Stimulation. No untoward cardiac effects of external electric stimulation were observed in these 13 patients. External electric stimulation did not produce multiple ectopic ventricular beats or ventricular fibrillation, effects that are seen experimentally in direct electric stimulation of the heart.4 Necropsies in five patients showed no evidence of damage from stimulation to the heart or to neighboring structures. In two of these patients (cases 5, 7) there was damage resulting from intracardiac punctures; in one (case 5) cardiac tamponade was the immediate cause of death. The only tissue damage from stimulation consisted of superficial ulcerations under the chest electrodes in patients treated for a day or more. This problem has been minimized by frequent small changes in the positions of the electrodes.

The main untoward effects were chest pain and muscular twitch. The intensity of the pain and of the muscular contraction varied in different patients; in some it was negligible, in others it made continued stimulation difficult. Meperidine hydrochloride or paraldehyde usually made the discomfort tolerable and permitted continued stimulation. The administration of a curare-like drug (case 3) and local infiltration with procaine hydrochloride under the electrodes were ineffective. With prolonged stimulation the severity of the pain usually diminished and less medication was required.

On one occasion (case 8) an unusual effect on respiration was observed. When both electrodes were placed lower than usual on the chest, on the seventh or eighth intercostal spaces in the anterior axillary lines, stimulation produced apnea. When the electrodes were moved higher, to the fourth or fifth intercostal spaces, this interference with respiration stopped.

Resuscitation from Unexpected Circulatory Arrest. Circulatory arrest may occur unexpectedly during anesthesia. Like Stokes-Adams attacks, these episodes also represent desperate emergencies for which present therapy is often dangerous and ineffective. The mechanisms of unexpected circulatory arrest are ventricular
standstill or fibrillation; standstill is the more frequent cause. 5, 6, 7

The success of external electric stimulation in arousing the heart from ventricular standstill in patients with Stokes-Adams disease suggested its value in resuscitating patients from unexpected circulatory arrest. We have established the effectiveness of external cardiac stimulation during experimentally-produced ventricular standstill. 3 Marked cardiac slowing and hypotension were produced by vagal stimulation; during asystole lasting as long as 37 seconds, the external cardiac pacemaker evoked heart beats adequate to restore the blood pressure to normal.

Because of its efficacy, safety and ready applicability, external cardiac stimulation appears to be the method of choice for the immediate treatment of a patient with unexpected circulatory arrest. If the cardiac mechanism is ventricular standstill, as is usually the case, and if the myocardium is still responsive, resuscitation should be successful. If the cardiac pacemaker fails to resuscitate the patient, the chest must be opened at once, the heart massaged, and appropriate drugs administered. If ventricular fibrillation is present and persists, electrical defibrillation across the exposed heart should be attempted. On the occasions when ventricular standstill follows successful defibrillation, the external pacemaker may be used.

In addition to electric stimulation of the heart, electric defibrillation of the ventricles has been accomplished experimentally across the unopened chest. 3, 8, 9 At present, however, external defibrillation is not feasible clinically because of the size of the equipment and the large current required. Studies of this problem are in progress.

**Summary**

External electric stimulation was applied in 14 patients with Stokes-Adams disease; it was effective in 13. It was used in the treatment of recurrent syncope in 11; in two patients syncope did not recur. Six of the 11 treated patients were resuscitated from one or more Stokes-Adams attacks; 9 were treated for prolonged periods. Five patients survived following treatment for intervals of 9 days, 9 days, 7 months, 10 months and 12 months, respectively. Such long survivals without recurrent syncope suggest that the periods of cardiac disturbance which cause syncope may subside if the patient is kept alive during the crucial period.

Ten of these 14 patients have now died. In eight, death was clearly due to a Stokes-Adams attack: external stimulation was not applied terminally in three; it was applied after irreversible cerebral damage had occurred in two; and it was ineffective in three, presumably because the attack was due to persistent irregular ventricular tachycardia.

External electric stimulation resuscitated patients from attacks due to ventricular standstill; it maintained an adequate circulation during persistent ventricular standstill; and it prevented the recurrence of irregular ventricular tachycardia.

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**SUMARIO ESPAÑOL**

Estímulo eléctrico externo fue aplicado a 14 pacientes con la enfermedad de Stokes-Adams; fue efectivo en 13. Fue usado en el tratamiento del síncopé recurrente en 11; en 2 pacientes el síncopé no recurrió. Seis de los 11 pacientes tratados fueron resucitados de uno o más ataques Stokes-Adams; 9 fueron tratados por tiempo prolongado. Cinco pacientes sobrevivieron luego del tratamiento por intervalos de 9 días, 9 días, 7 meses, 10 meses y 12 meses respectivamente. Estas supervivencias prolongadas sin síncopé recurrente sugieren que los periodos de disturbio cardáco que causan el síncopé pueden apaciguarse si el paciente se mantiene vivo durante el período crucial.
Diez de estos 14 pacientes han muerto. En 8, la muerte se debió claramente a un ataque Stokes-Adams: estimulación externa no se aplicó terminalmente en 3: se aplicó luego de daño irreversible cerebral haber ocurrido en 2: y fue efectivo en 3, posiblemente debido a que el ataque fue causado por una taquicardia irregular ventricular persistente.

Estimulo eléctrico externo resucitó pacientes de ataques debidos a pausas ventriculares: mantuvo una circulación adecuada durante la pausa ventricular persistente; y evitó la repetición de taquicardia irregular ventricular.

REFERENCES
3 Unpublished observations.
Treatment of Stokes-Adams Disease by External Electric Stimulation of the Heart
PAUL M. ZOLL, ARTHUR J. LINENTHAL, LEONA R. NORMAN and ALAN H. BELGARD

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