

Electrical injury causing ventricular arrhythmias

PETER JØRN JENSEN,*† POUL ERIK BLOCH THOMSEN,*
JENS PETER BAGGER,* AAGE NØRGAARD,* ULRIK BAANDRUP‡

*From the *Department of Cardiology, Aarhus Kommunehospital, Aarhus; the †Department of Internal Medicine, Silkeborg Centralsygehus, Silkeborg; and the ‡Department of Pathology, Aarhus Kommunehospital, Aarhus, Denmark*

SUMMARY Dangerous or long lasting ventricular arrhythmias developed in three patients who had sustained an electrical injury in which current passed through the thorax. In all three cases there was a delay of 8–12 hours between the injury and the onset of symptoms. The ventricular arrhythmias were severe and long lasting. In two of the three patients, ventricular tachycardia or ventricular fibrillation or both occurred and in one patient ventricular parasystole developed. No enzymatic evidence of myocardial necrosis was found but the results of an endomyocardial biopsy carried out in two of the three patients showed focal myocardial fibrosis and increased numbers of Na, K-pumps. The two patients with ventricular tachycardia became symptom free after appropriate antiarrhythmic treatment and in the third patient ventricular parasystole disappeared spontaneously within two years.

Patients sustaining electrical injury in which current passes through the thorax should be monitored electrocardiographically for at least 24 hours, and patients with unexpected arrhythmias should be questioned about previous electrical injury.

A lightning strike is a recognised cause of sudden death. When the mechanism of such death was investigated in 1775 it was found that an electrical shock applied to a head stunned hen whereas another shock to the sternum resuscitated it.¹ In 1850 it was shown in a dog that application of a current to the exposed heart was followed by very rapid and irregular movements of the ventricles—that is, probably ventricular fibrillation.²

Nowadays electrical injuries are common. Several factors such as voltage, tissue resistance, tissue susceptibility, type of current, current pathway, site, and duration of electrical contact determine the severity and distribution of the injury.³ The heart in particular is liable to damage by electrical injury. Thus high voltage (> 500 V, direct or alternating current) and lightning can cause sudden death from cardiac arrest, but the low voltage (110–380 V, alternating current 50–60 Hz) used in household electrical equipment can also cause sudden death, usually from ventricular fibrillation.^{3,4} Less serious

transient electrocardiographic changes have also been described after electrical injury.⁵ Furthermore, electrical injury has been diagnosed on the basis of enzyme changes indicating myocardial necrosis.^{6,7} Finally, countershock applied for cardioversion can be followed by serious arrhythmias, either immediately or within a few hours,^{8,9} or by transient ST segment elevation.¹⁰ The effect of electrical injury on the heart has been demonstrated at necropsy in fatal cases and non-invasively in survivors by electrocardiography, enzyme activity, and echocardiographic examination.⁷

We report three cases of non-fatal electrical injury that were followed by delayed serious ventricular arrhythmias. Endomyocardial biopsy specimens were taken from two of the patients. Microscopy showed myocardial fibrosis.

The membrane bound enzyme, the Na, K-ATPase or Na, K-pump, performs the transport of sodium ions out of the cell and potassium ions into the cell and is essential for the specific properties of muscle and nerve tissue such as contractility and excitability.¹¹ We estimated the concentration of this pump in the endomyocardial biopsy specimens by measuring binding of [³H] ouabain.¹²

Requests for reprints to Dr Peter Jørn Jensen, Department of Cardiology, Aarhus Kommunehospital, DK-8000 Aarhus C, Denmark.

Patients and methods

ELECTROPHYSIOLOGICAL EXAMINATION

Electrophysiological provocation was performed as described elsewhere.¹³ Electrode catheters were introduced percutaneously and positioned high in the right atrium, the His bundle region, and in the right ventricle. Programmed ventricular stimulation was carried out in the right ventricular apex or outlet tract and consisted of pacing with a current of twice diastolic threshold with an impulse duration of 2 ms, and a basic cycle length of 500 ms for eight beats followed by single or double ventricular extrastimuli with varying coupling intervals. If this failed to induce ventricular tachycardia the basic cycle length was reduced to 460 ms and eventually to 400 ms. Finally, if this stimulation procedure failed to induce ventricular tachycardia we used burst pacing with bursts of eight beats with a cycle length of from 300 ms to 200 ms.

ENDOMYOCARDIAL BIOPSY SAMPLING

The left heart was catheterised from the femoral approach. After coronary angiography five endomyocardial biopsy specimens were taken from the left ventricle with Cordis biopsy forceps. Three of the biopsy samples were taken for microscopy and two were frozen (-20°C) for assay of the Na, K-pump.

ASSAY OF THE NA, K-PUMP

Binding of [^3H] ouabain was measured as described elsewhere.¹² The biopsy specimens were incubated twice in a TRIS sucrose buffer containing 10 mmol/l TRIS chloride, 3 mmol/l magnesium sulphate, 250 mmol/l sucrose, 1 mmol/l vanadate, and 1×10^{-6} mol/l [^3H] ouabain at 37°C for 60 min each. This was followed by four washes at 0°C for another 30 minutes each to remove [^3H] ouabain which was not bound to its receptor. After blotting, the samples were weighed and 5% trichloroacetic acid was added. After overnight extraction and liquid scintillation counting, the amount of [^3H] ouabain bound to the biopsy specimens was calculated on the basis of the specific activity of the incubation medium. After correction for non-specific uptake determined in the presence of surplus of unlabelled ouabain, results were expressed as pmol of [^3H] ouabain per g wet weight of muscle tissue.

Results

PATIENT 1

A 43 year old previously healthy male electrician sustained an electrical injury (3000 V, direct current) from one hand to the other. Twelve hours later while he was playing football he fainted suddenly with cardiac arrest. Resuscitation was started immediately. When he arrived at the local hospital ventricu-

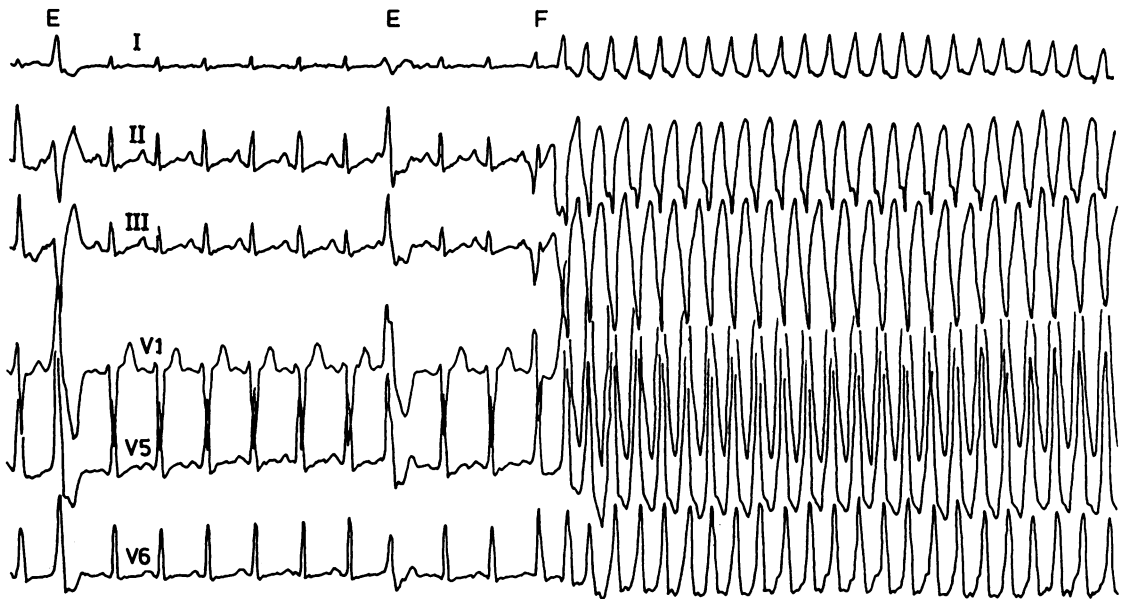


Fig 1 Electrocardiogram showing the onset of ventricular tachycardia with a frequency of 210 beats per minute just after exercise test in patient 1. The QRS configuration shows right bundle branch block suggesting a left ventricular focus. There were two ventricular extrasystoles (E) of different QRS configuration immediately before ventricular tachycardia started with a fusion beat (F) (paper speed 25 mm/s).

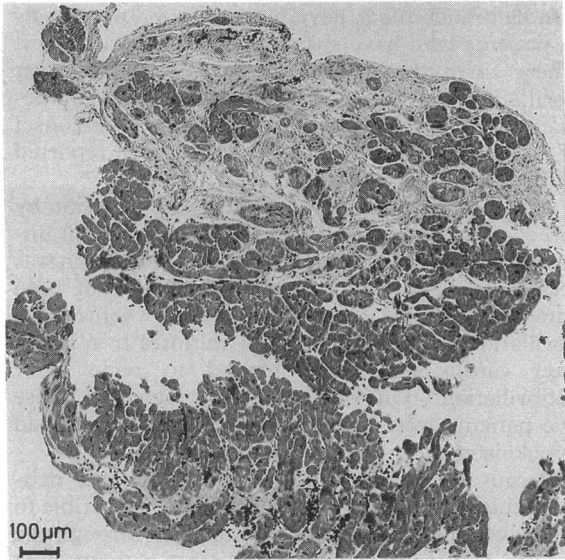


Fig 2 Left ventricular endomyocardial biopsy specimen from patient 1 showing endocardial and subendocardial fibrosis. Single myocytes are encased by the dense collagen tissue (elastic Van Gieson).

lar fibrillation was present and he was successfully treated with a direct current countershock. Electrocardiograms recorded half an hour later and in the next few days were normal. Furthermore, activities of coronary enzymes, including creatine kinase B, and serum potassium were normal. A new attack of ventricular fibrillation occurred six days after the accident while he was not being monitored. Con-

version to sinus rhythm by direct current countershock was successful.

Six months later he fainted suddenly while bicycling; again there was a cardiac arrest. He was resuscitated. On his admission to the local hospital the electrocardiogram showed ventricular fibrillation and the rhythm was again converted by a direct current countershock. The resting electrocardiogram, echocardiographic examination, and coronary arteriography were normal. Exercise testing showed a working capacity of 300 W for 18 minutes before general exhaustion. Just after the test, however, ventricular tachycardia developed (fig 1). This lasted for 54 s with a frequency of 210 beats per minute (cycle length 290 ms). The QRS configuration during this tachycardia showed right bundle branch block consistent with a left ventricular focus. An identical ventricular tachycardia could be induced by burst pacing at a cycle length of 230 ms in the right ventricular outlet tract. The induced ventricular tachycardia degenerated within a minute to ventricular flutter and fibrillation and the patient was given a direct current countershock. Left ventricular endomyocardial biopsy specimens showed focal myocardial fibrosis (fig 2) and a Na, K-pump concentration of 600 pmol/g wet weight. After administration of sotalol 80 mg three times a day exercise testing was normal and it was no longer possible to induce ventricular tachycardia by electrophysiological provocation.

PATIENT 2

A 45 year old previously healthy woman sustained an electrical injury in which current (380 V, 50 Hz)

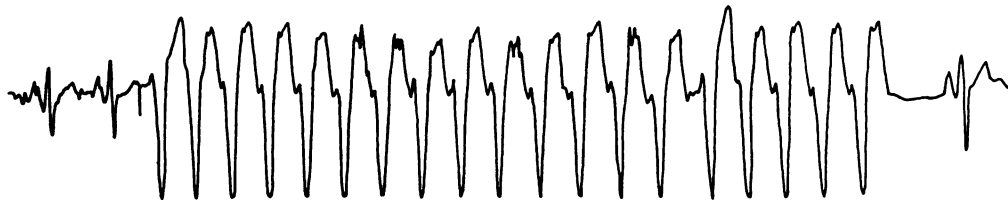


Fig 3 Electrocardiographic monitoring in patient 2 on CM showing a run of ventricular tachycardia with a frequency of 170 beats per minute lasting 7.4 seconds (paper speed 25 mm/s).



Fig 4 Electrocardiographic monitoring in patient 3 on CM showing ventricular parasystole with fusion beat (F), variable coupling interval, and a minimum manifest interectopic cycle length of 760 (720-780) ms (paper speed 25 mm/s).

passed from one arm to the other. Eight hours later she had dizzy spells and palpitation. Despite daily attacks she did not consult her physician until two months later. Ambulatory Holter monitoring showed frequent runs of ventricular tachycardia (170 beats per minute) that lasted up to two minutes. These were associated with dizzy spells (fig 3). At admission to hospital the patient mentioned the previous electrical injury. Resting electrocardiogram, coronary enzyme concentration, and serum potassium concentration were normal. Exercise testing and coronary arteriography were also normal. Left ventricular endomyocardial biopsy specimens showed focal myocardial fibrosis and a Na, K-pump concentration of 463 pmol/g wet weight. She has been treated with mexiletine for six months with excellent effect on the ventricular tachycardia and its symptoms.

PATIENT 3

A 34 year old previously healthy man sustained an electrical injury when current (220 V, 50 Hz) passed from his left hand to his right shoulder. Twelve hours later he experienced chest pain and palpitation. He was admitted to the hospital and physical examination was normal except for a pericardial friction rub. Electrocardiographic monitoring showed ventricular parasystole with highly variable coupling intervals, fusion beats, and a minimum manifest interectopic cycle length of 760 (720–780) msec (fig 4). During the next few days the electrocardiogram showed transient ST segment depression and negative T waves in leads I, II, III, and V4–6. All laboratory tests were normal including creatine kinase B, serum potassium, and virus titres. The exercise electrocardiogram was normal and parasystole disappeared completely when the heart rate exceeded 120 beats per minute. Thallium-201 myocardial exercise testing and cross sectional echocardiography were normal. Coronary arteriography, electrophysiological provocation, and endomyocardial biopsy were not considered to be justified.

Holter monitoring at two weeks and three, six, 12, 24, and 48 months after the injury showed a gradually decreasing number of ventricular parasystolic beats with unchanged minimum manifest interectopic cycle length. Within two years the ventricular parasystole had completely disappeared.

Discussion

In the present study there was a delay between electrical injury and the onset of arrhythmias. Cardiac arrhythmias and conduction disturbances such as ventricular fibrillation, ectopic focal arrhythmia, and

bundle branch block have been reported previously in patients who have sustained an electrical injury. These arrhythmias and especially ventricular fibrillation, however, seem to have developed immediately after injury. A delay between injury and onset of arrhythmias does not seem to have been reported before.^{3 4 7}

Arrhythmias are often seen after cardioversion by direct current countershock. These can be either immediate or delayed. Thus ventricular fibrillation was seen immediately after cardioversion in 26 of 1046 patients whereas in five patients ventricular fibrillation was delayed from 5 minutes to 6 hours after cardioversion and tended to recur after defibrillation.⁹ Unlike our patients, however, these five patients were heavily digitalised and four had hypokalaemia.

Because current tends to course along the neurovascular bundles, the heart is highly susceptible to injury when an electrical current passes through the thorax. Generally, however, in most patients who suffered electrical injury there were no major arrhythmias.^{3 4} This may be because evidence about whether the current had passed through the thorax or not was not specifically sought.

Several theories have been advanced to explain the effects of electricity on the heart. The development of patchy necrosis after electrical injury has been suggested.⁷ This theory is supported by our findings in the two patients in whom endomyocardial biopsy was performed. To our knowledge abnormal findings in the endomyocardial biopsy specimens of survivors of electrical injury have not been reported before. The biopsy specimens were obtained two and six months after the injury and the patchy fibrosis that we found at this time could explain the development of early arrhythmias. Furthermore, the fibrotic tissue found in the myocardium might be a potential chronic arrhythmogenic focus.

We found Na, K-pump concentrations of 463 and 600 pmol/g wet weight in two endomyocardial biopsy specimens assayed for [³H] ouabain binding capacity. In 15 patients in permanent sinus rhythm the mean (SE) concentration was 413 (26) pmol/g wet weight.¹² Although normal perfusion prevents permanent changes in the extracellular potassium concentration, an increased Na, K-pump concentration may be associated with transient and localised changes in sodium and potassium transport, potassium concentration, and membrane potential. This could be important in the development of arrhythmias.¹⁴

None of our patients had ischaemic heart disease and there was no electrocardiographic or serological evidence of pericarditis or myocarditis. The pericardial friction rub heard in patient 3 may have been

caused by a small pericardial haemorrhage, a feature which has already been found at necropsy in a person who died after being struck by lightning.⁵ The histological appearance of the endomyocardial biopsy specimens, however, does not exclude myocarditis.

We believe that patients sustaining an electrical injury in which the current passes through the thorax should be monitored electrocardiographically for at least 24 hours because the onset of arrhythmias may be delayed. Because long lasting and severe arrhythmias can develop under these circumstances, patients with unexpected arrhythmias should be questioned about previous electrical injury.

References

- 1 Scherf D, Schott A. *Extrasystoles and allied arrhythmias*. 2nd ed. London: William Heinemann Medical Books, 1973:898.
- 2 Hoffa M, Ludwig C. Einige neue Versuche über Herzbewegung. *Zeitschrift für klinische Medizin* 1850; 9:107-44.
- 3 Solem L, Fischer RP, Strate RG. The natural history of electrical injury. *J Trauma* 1977;17:487-92.
- 4 Di Vincenti FC, Moncrief JA, Pruitt BA. Electrical injuries: a review of 65 cases. *J Trauma* 1969; 9:497-507.
- 5 Burda CD. Electrocardiographic changes in lightning stroke. *Am Heart J* 1966;72:521-4.
- 6 Jackson SHD, Parry DJ. Lightning and the heart. *Br Heart J* 1980;43:454-7.
- 7 Lewin RF, Arditti A, Sclarovsky S. Non-invasive evaluation of electrical cardiac injury. *Br Heart J* 1983;49:190-2.
- 8 Lown B, Neuman J, Amarasingham R, Berkovits BV. Comparison of alternating current with direct current electroshock across the closed chest. *Am J Cardiol* 1962;10:223-33.
- 9 Faivre G, Cherrier F, Polu JM. Traitement de la fibrillation auriculaire par choc électrique externe. IV. Complications. *Coeur Med Interne* 1969;8: 487-94.
- 10 Chun PKC, Davia JE, Donohue DJ. ST-segment elevation with elective DC cardioversion. *Circulation*. 1981;63:220-4.
- 11 Skou JC. Enzymatic basis for active transport of Na⁺ and K⁺ across cell membrane. *Physiol Rev* 1965;45:596-617.
- 12 Nørgaard A, Kjeldsen K, Hansen O, Clausen T, Larsen CG, Larsen FG. Quantification of the ³H-ouabain binding site concentration in human myocardium: a post mortem study. *Cardiovasc Res* 1986;20:428-35.
- 13 Thomsen PEB, Nielsen TT, Albrechtsen O, Mortensen PT, Hansen OKH. Ventricular tachycardia. Electrophysiological principles, medical and surgical treatment. *Ugeskr Laeger* 1984;146:3727-32.
- 14 Opie LH, Nathan D, Lubbe WF. Biochemical aspects of arrhythmogenesis and ventricular fibrillation. *Am J Cardiol* 1979;43:131-48.