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ORIGINAL ARTICLE

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Cardiac pathology in death from electrocution

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Abstract To better characterize the morphologic changes in electrocution, morphologic changes in the hearts of 21 subjects, who died instantaneously of electrocution, were compared to the hearts of decedents with different types of death. Sixteen myocardial samples per heart were processed for histological examination, and sections were prepared with a variety of specific stains. The frequency, location and extent of myocellular segmentation (stretching and/or rupture) of intercalated discs and associated changes of myocardial bundles and single myocells [myofibre break-up (MFB)] were recorded, quantitatively analysed and statistically evaluated. The frequency of MFB was maximal in cases of electrocution (90%). The findings show that MFB is an ante-mortem change and may be a distinct finding in electrocution.

Keywords Electrocution · Electric shock · Ventricular fibrillation · Heart · Histology

Introduction

Electrical shock may result in death with variable degrees of damage to different organs. The final picture is determined by the type, voltage and intensity of the current [1]. Except for cutaneous electrical and heat-related effects, the other direct effects of electricity essentially leave no trace [2–5]. The principal cause of death is usually said to be a disturbance in cardiac electric conduction leading to ventricular fibrillation. Ventricular fibrillation is defined as a "chaotic, random, asynchronous electrical activity of the ventricles due to repetitive re-entrant excitation and/or rapid focal discharge" [6], but no morphologic changes has ever specifically been linked to electrocution.

In a previous study, we reported that a typical lesions, characterized by a break-up of myocardial fibres (MFB), could provide the structural substrate necessary to initiate chaotic, electrical asynchronous activity and could be induced by the passage of abnormal electrical currents [7].

The purpose of this study was to characterize the types and frequency of the structural myocardial injury observed in cases of fatal electrocution and to determine whether MFB lesions were more common in death from electrical shock.

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Materials and methods

Study population

Morphologic changes in three different control populations were compared to those found in victims of electrocution. The study groups were as follows: (1) 21 individuals died instantaneously of electrocution; (2) 26 individuals died from cocaine intoxication, 18 out of hospital and 8 in hospital [8]; (3) 45 individuals died of head trauma, 26 instantly and the others within a few hours [9] and (4) 26 healthy subjects died at home of carbon monoxide (CO) intoxication [10]. Complete autopsies were performed in every case within 24 h of the death. The main characteristics of the different groups are reported in Table 1.

Definition

The term myofibre break-up includes the following histologic patterns: (1) bundles of distended myocardial cells alternating with hyper-contracted cells. In the latter group of cells, there is also widening or rupture (segmentation) of the intercalated discs (Fig. 1). Myocardial nuclei in the

Table 1 Main characteristics of the groups investigated in this study

Groups	Cases	Gender		Age	Heart weight	Mode of death	Resuscitation	
		Male	Female	(years)	(g)		No	Yes
Electrocution	21	21	–	36±4	347±9	Accidental	21	0
Cocaine	26	20	6	35±1	351±10	In hospital (18) and out of hospital (8)	8	18
Head trauma	45	37	8	42±3	364±10	Accidental	45	0
CO	26	20	6	48±3	375±14	Accidental	26	0

hyper-contracted cells have a "square" aspect rather than the ovoid morphology seen in distended myocytes (Fig. 2); (b) hyper-contracted myocytes alternated with hyper-distended cells that are often divided by a widened disc; (c) non-eosinophilic bands of hyper-contracted sarcomeres alternating with stretched, often apparently separated sarcomeres (Fig. 3) [7].

Heart examination

The method of heart examination has been published previously [11]. According to a fixed protocol, 16 samples of myocardium were processed from each heart, and sections from each block were stained with haematoxylin and eosin. Additional specific stains (Mallory, Weigert elastic, Movat pentachrome, acid fuschin orange) were performed when indicated. Immunohistochemical staining was performed using monoclonal anti-complement C9 antibodies in all hearts [12, 13].

Quantitative analysis

The area occupied by tissue of each slide was measured in square millimeters using a colour video camera connected to a light microscope. The image was then digitalized by an image analysis system (Leica, Q500 Plus, UK). The number of foci and of necrotic myocytes were counted and normalised to 100 mm^2 [14].

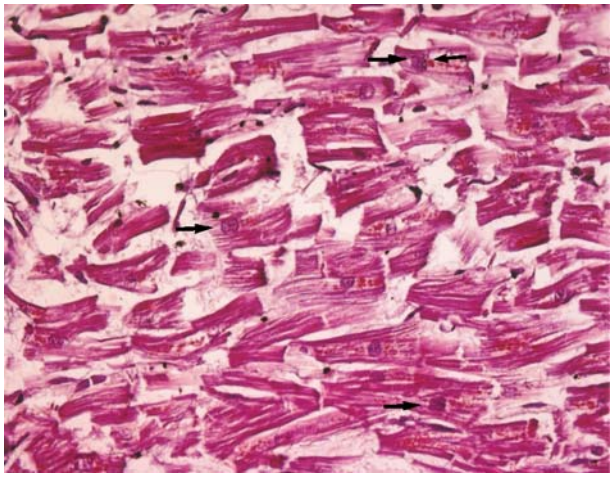

Fig. 1 Bundles of hyper-contracted myocytes (*arrows*) alternated with bundles of hyper-distended myocardial cells (trichrome stain ×100)

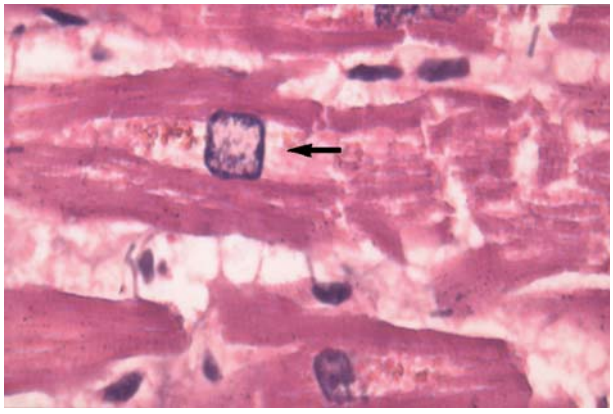

Fig. 2 Square nucleus expression of contraction (*arrow*) (H&E ×630)

Statistical evaluation

Data are expressed as mean value ± one standard error. To assess statistical significance, Student's *t* test for paired or unpaired data or one-way analysis of variance and post hoc Scheffe's test for continuous variables and χ^2 test for discrete variables were used where appropriate. Linear regression analysis was used to assess for the presence of correlation between continuous variables. A probability measured value <0.05 was considered significant.

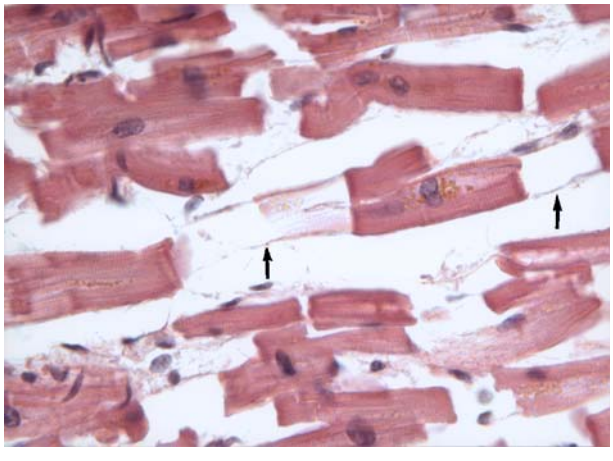

Fig. 3 Separation of sarcomeres (*arrows*) in myofibres connected with contracted ones (H&E ×250)

Table 2 Frequency, location and extent of myofibre break-up vs contraction band necrosis

Myofibre break-up in anterior left ventricle							
Groups	Cases	Present	CBN		Absent	CBN	
			F	M		F	M
Electrocution	21	19	8	46	2	–	–
Cocaine	26	11	8±2	25±7	15	1±0.3	2±0.3
Head trauma	45	29	13±7	28±13	16	1±0.4	4±0.4
CO	26	5	1±0.6	4±2	21	2	9

CBN Contraction band necrosis, *F* foci, *M* myocytes (×100 mm^2)

Results

Examination of the conduction system performed according to Davies [15] was unremarkable. The frequency of MFB was maximal in cases of electrocution (90%), followed by the head trauma group (64%).

There was no correlation between gender, age, heart weight, degree of coronary atherosclerosis and MFB. In the control groups, MFB was observed only in a few CO intoxication cases (19%) and in 64% of subjects with head trauma (Table 2).

No relationship was found between the frequency and extent of MFB and the frequency or extent of contraction band necrosis. However, there was a trend for the frequency of contraction band lesions to be greater than that of MFB in most groups (Table 2). This trend was significant in the cocaine group (group 2 $p<0.01$).

Discussion

It is assumed that death from electrocution is often the result of ventricular fibrillation [16], but myocardial alterations in such cases are poorly documented [1, 17, 18]. Lichtenberg et al. [19] examined the hearts of 19 individuals struck by lightning and concluded that the pattern of cardiovascular damage depended on the type of lightning strike (i.e. direct, splash and ground strike). The direct type of lightning injury can result in "myocardial injury" (evidenced by a significant increase in CPKmb), depression of the ST-T tract, alterations in ventricular kinetics visible with echocardiography and also lengthening of the QT interval [20–32].

The morphologic changes we observed ranged from small foci in one area to a diffuse involvement of most or all cardiac regions. This raises the issue of whether the observed changes were the substrate for the fatal arrhythmias or whether their occurrence was merely an epiphenomenon, occurring at the same time, but causally unrelated.

Two aspects of the process need further consideration: (1) whether the changes are real or artifactual and (2) whether the morphologic changes actually played a role in the initiation of the fatal arrhythmia.

The answer to the first question is that the MFB changes we have described appear to be vital. Myofibre fragmentation due to knife motion (sometimes referred to as "chatter") in cutting histological sections is easily distinguished from MFB. Similar changes have never been described as a consequence of rigor mortis [33]. Furthermore, if rigor mortis were the cause, MFB should be found in all cases autopsied after 1–24 h, and it was not. In a previous study, we demonstrated the absence of MFB in all hearts excised at transplantation [7]; this fact also argues against the possibility that MFB is an artefact, occurring secondary to histological processing.

It is generally accepted that any intervention that increases spatial inhomogeneity of heart muscle also facilitates the induction of certain cardiac arrhythmias. Ischemia is the most likely culprit for this inhomogeneity, but the findings of focal myocardial necrosis and/or fibrosis are common in cases of sudden arrhythmic death and are often assumed to be the cause of re-entry and electrical instability [7, 34, 35]. Whether MFB falls into this same category remains to be proved, but it certainly is a possibility that must be considered.

The documented clinical cardiovascular effects of an electrical shock include immediate heart failure, acute myocardial necrosis, myocardial ischemia with or without necrosis, arrhythmia, hemorrhagic pericarditis, acute hypertension with peripheral vasospasm and anomalous but non-specific ECG changes [5]. In rapid death due to electrical shock, we found typical cardiac lesions, characterized by a break-up of myocardial fibres that could provide the structural substrate necessary to initiate chaotic, electrical asynchronous activity and could be induced by the passage of abnormal electrical currents.

The obvious limitation of the study is that autopsy findings can only rarely be correlated with terminal electrocardiographic recordings [36]. However, in a previous study, we demonstrated that all the ECG-monitored cases with fibrillation had extensive MFB [7]. Systematic studies to confirm whether MFB is pathognomonic for malignant arrhythmia are needed.

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