TOPICS IN REVIEW

The electrophysiology of electrocution

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Electrocution is a death caused by an application of electrical current to the human body. Our present understanding of electrocution—as the induction of ventricular fibrillation (VF)—followed a nearly century-long path of misunderstandings and speculation primarily focused on hypotheses of asphyxia as well as central nervous system trauma. It is hard for us today to appreciate the past mystery of an unexpected sudden death usually bereft of visible trauma. Even today, a false dogma exists that direct-current shocks can cause asystole instead of VF. A lightning discharge (up to 500 megavolts) is differentiated because it can cause substantial acute and chronic neural effects leading to other cardiac arrest rhythms. The human heart is exquisitely sensitive to alternating currents, and VF can be induced with currents of one-eighth that required for mere pacing. Because of these low currents, this effect obtains only in the TQ interval, and low-power electrocution does not involve the vulnerable period. If a current is strong enough to electrocute, generally it will do so in 1–2 seconds; longer shocks do not tend to be more dangerous. Regardless of concomitant drug dosing, the electrocution cardiac arrest rhythm is still VF, suggesting that electrocution is a stand-alone cause of death; the electrical current does not potentiate the effects of the drug. The experimental and clinical data supporting VF as the mechanism for electrocution are provided.

KEYWORDS Electrocution; Ventricular fibrillation; Asystole; Asphyxia; Cardiac arrest; Direct current; Alternating current

(Heart Rhythm O^2 2023;4:457–462)

Introduction

Electrocution is a death caused by an application of electrical current to the human body. In this article, we review salient historical research and describe the scientific advances that support ventricular fibrillation (VF) as the mechanism of electrocution. The lay and media usage of "electrocution" to include nonfatal injury is reflected in some dictionaries but does not represent the usage of specialists.

Our present understanding of electrocution—as the induction of VF—followed a nearly century-long path of misunderstandings and speculation. It is hard for us today to appreciate the past mystery of an unexpected sudden death bereft of visible trauma. Among several previous theories, the most popular were asphyxiation (electrically induced respiratory arrest) and central nervous system damage. A detailed history of several other theories can be found elsewhere.[1] The special case of lightning-associated deaths is discussed at the end of this article.

Alternating current (AC) is defined as a current that alternates in polarity, whereas direct current (DC) maintains a single polarity, usually at a steady amplitude. A pulse current may have a single polarity or multiple polarities (eg, biphasic defibrillation waveform) but is dominated by a long duration of zero current (Figure 1). In these experiments, both AC and DC applications to the heart were evaluated.

Experimental Models Pre-Einthoven

Electrically induced VF was first demonstrated by Hoffa and Ludwig[2] in 1849 at the University of Leipzig. Lacking the electrocardiogram (ECG), they recorded the DC-induced VF mechanically with their "kymograph" (Figure 2). Because of the mechanical low-pass filtering, the tracing appears more regular than an ECG tracing of VF. Hoffa was Ludwig's PhD student, and they were investigating vagal influences on cardiac activity using strong DC current. Hoffa meant to stimulate neurons but accidentally stimulated the epicardium and noted the bizarre unregulated actions of the ventricles after application of the current. The rhythm was noted to persist even after electrical stimulation ended and to result in loss of cardiac output.

The first use of the term "fibrillation" stems from canine work by the French neurologist Edmé G Vulpian[3]; he called it "mouvement fibrillaire" in his 1874 paper. John

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2666-5018/

https://doi.org/10.1016/j.hroo.2023.06.004

KEY FINDINGS

- Non-lightning electrocution is the induction of ventricular fibrillation (VF).
- This understanding followed many decades of speculation primarily focused on hypotheses of asphyxia as well as central nervous system trauma.
- A misunderstanding persists that direct-current shocks can cause asystole instead of VF.
- The vulnerable period is rarely involved in electrocution.

McWilliam (aka MacWilliam) studied medicine and then under Ludwig performed postgraduate studies involving fibrillation experiments with eel, fish, and frogs before returning to Scotland to earn his MD. McWilliam[4] showed that the induction of VF was accompanied by a prompt fall in blood pressure and dilation of the left ventricle. McWilliam (1899) rejected the widely accepted theory that the electrocution cardiac arrest was due to primary asystole. He concluded that the arrest was due to VF and explained that the heart[5]:

> …. assumes, on the contrary, the form of violent, though irregular and uncoordinated manifestation of ventricular energy. Instead of quiescence there is tumultuous activity, irregular his character and wholly ineffective as regards its results… The normal beat is at once abolished, and the ventricles are thrown into a tumultuous state of quick, irregular, twitching action; at the same time there is a great fall of blood-pressure.

This earlier work was largely ignored until an American physician, Tatum,[6] published the first version of VF threshold measurements in 1890 based on dog studies. He may have been the first to describe electrocution as a result of paralysis of the heart.[7] Tatum's work also supported primary VF as the mechanism—not asystole—as he showed that vagal sectioning, atropine, and curare did not prevent death from electrical current. Tatum found that the placement of electrodes in the region of the heart was more lethal than in any other location and argued that this clearly refuted the competing central nervous system theory of involvement of the nervous system.

Experimental Data Post-Einthoven

The scientific evidence supporting VF as the mechanism and rejecting asphyxiation comes from the work of multiple investigators in Europe and the United States.

In 1899, Prevost and Batelli[8] published their landmark paper confirming that electrocution was due to VF as well as performing the first known defibrillation. They applied AC to different animal species using various shock pathways. Delivering current via a low-resistance pathway using electrodes in the mouth and rectum, they were able to fibrillate dogs with 20- to 40-V AC and then defibrillate them with 4800-V AC (Figure 3). They also showed that weaker currents (below the VF threshold) would cause respiratory arrest.[9] (Much later, it was shown that AC currents of 20 mA across the human chest will cause respiratory arrest compared to the VF threshold of approximately 100 mA.[10])

That same year, Cunningham[11] published a large dog study using both AC and DC with various pathways. He also demonstrated the theoretical possibility of death from respiratory arrest but argued that accidental human electrocutions were due to the induction of VF as the shock duration was too short for an asphyxial death. Sufficient current with a cardiac-involved pathway led to fibrillation and death within seconds. Currents involving the brain, spine, and upper chest caused temporary respiratory arrest but not death unless they were prolonged for minutes. He also applied high currents of 1.6 A directly to the brain of anesthetized dogs without causing death.

Georges Weiss[12] investigated the issue of asphyxia vs VF induction and concluded that electrocution was due to VF induction as fatal respiratory arrest required 10-minute duration medium currents in dogs. This was among a flurry of papers demonstrating that electrocution was due to VF, including those by Crile and Macleod[13] (1905), Boruttau[14,15] (1917 and 1919), and Cluzet[16] (1921), who all presented supportive animal study evidence.

Basic electrophysiological research continued with models testing VF mechanisms and thresholds in both tissue and animal experiments.[17–21] Notably, in 1936 Ferris[22] published a summary of his animal studies (~500 sheep) giving the currents required for VF as a function of shock duration. It is used to this day as the fundamental basis for international electrical safety limits.[23,24] Ferris noted zero deaths from asphyxia but acknowledged that this was a theoretical possibility if someone were unable to escape from a continuous lower current. Subsequently, throughout a century of electrocution research not a single human death from electrically induced asphyxia has been documented.

One of the earliest documented series of induction of VF in the electrophysiology laboratory was described in 1978 utilizing double ventricular extrastimulus administration.[25]

In 1999. Swerdlow et al[26] published their study showing how exquisitely sensitive the human heart is to AC. With a bipolar endocardial catheter, 60-Hz AC currents of 60 μA caused a continuous "capture" leading to hemodynamic collapse. VF was induced with 120 μA of AC; this was only 12% of the pacing threshold of 1-mA pulse current. The next year, Voroshilovsky et al[27] used an animal mapping study to show that AC can induce VF by generating a fast ventricular rate coupled with a steep restitution curve and nonuniform recovery of excitability of the myocardium. Because of the low AC currents, this effect obtains only in the TQ interval, thus refuting the present common belief that induction of VF requires interaction during the vulnerable period.

Thanks to the implantable cardioverter-defibrillator (ICD), we now have electrogram recordings of human beings receiving fatal and nonfatal shocks out of the hospital.

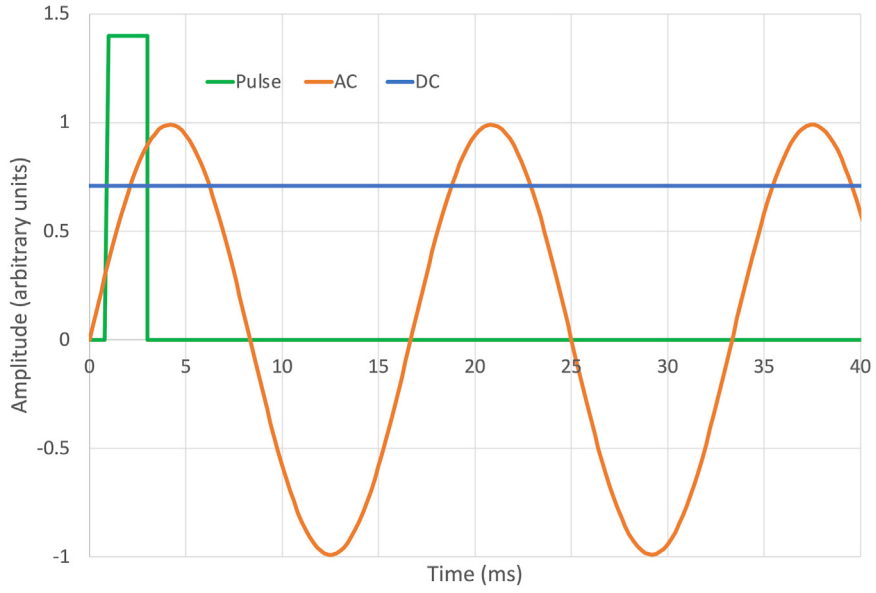

Figure 1 Examples of alternating current (AC) (60 Hz), direct current (DC), and pulse current.

In 1997, Mehdirad et al[28] described a patient who accidently grasped a 60-V AC line while kneeling in a damp ground utility tunnel. The patient remained conscious but was unable to release the power line. The 60-Hz interference was detected as VF causing him to receive a shock from his ICD, throwing him back and releasing the power line from his hand. Davis et al[29] later described a similar ICD–patient incident.

In 2008, Kondur et al[30] reported on a 75-year-old man who suffered a lightning side-flash strike, while adjusting his roof antenna, before he was rescued by his ICD. In 2010, Ginwalla et al[31] described a severely depressed

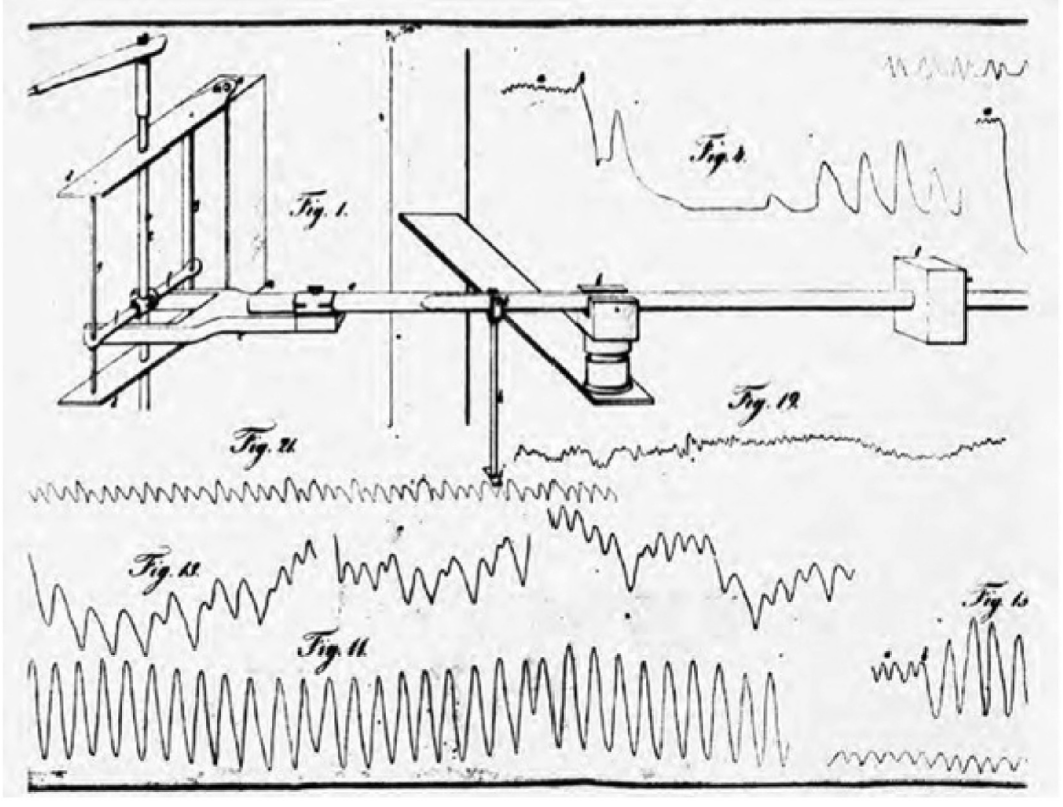

Figure 2 Hoffa and Ludwig's demonstration of electrically induced fibrillation. (From Hoffa and Ludwig.[2])

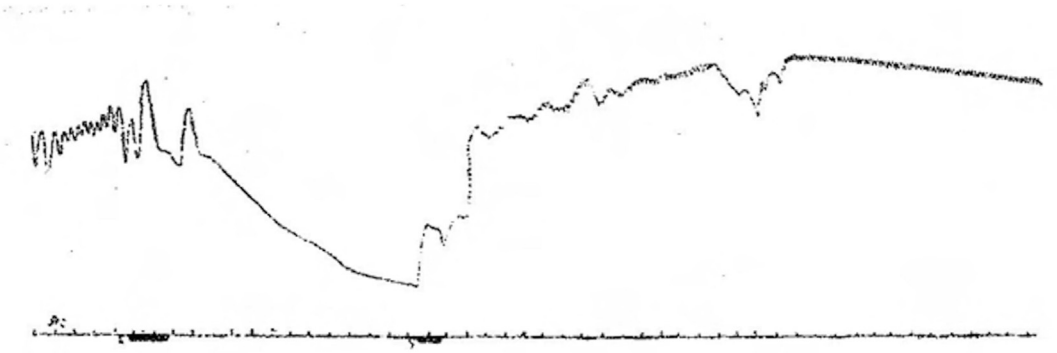

Figure 3 Canine arterial pressure (vertical axis) after fibrillation by 20-V alternating current (AC) and defibrillation by 4800-V AC. (From Prevost and Batelli.[8])

electrician who intentionally contacted a reported 240-V 3-phase panel in a suicide attempt. VF was induced, detected, and successfully treated with his ICD (Figure 4).

The Curious Dogma of DC and Asystole

A curious medical dogma holds that administration of DC current to the heart causes asystole.[32] In fact, neither asystole nor pulseless electrical activity is inducible with electrical stimulation.[33] Sharma et al[34] delivered DC directly to the hearts of 37 patients. All the patients developed VF, whereas no patient developed asystole. Lim et al[35] had the same results with 132 patients. The dogma may have been initiated with the 1962 article by Lown et al[36] comparing capacitor pulse shocks to AC for defibrillation. Two dogs remained in VF after many AC shocks but then developed asystole after a capacitator-pulse rescue shock. The title included the term "direct electroshock," which then apparently was confused with steady DC. The authors shortly followed with an article more appropriately entitled "alternating current and capacitor discharge," but the damage was done.[37] The usage of AC utility power (for the AC defibrillator) contrasted to DC battery power (for pulse defibrillation) added more fuel to the misunderstanding.[38]

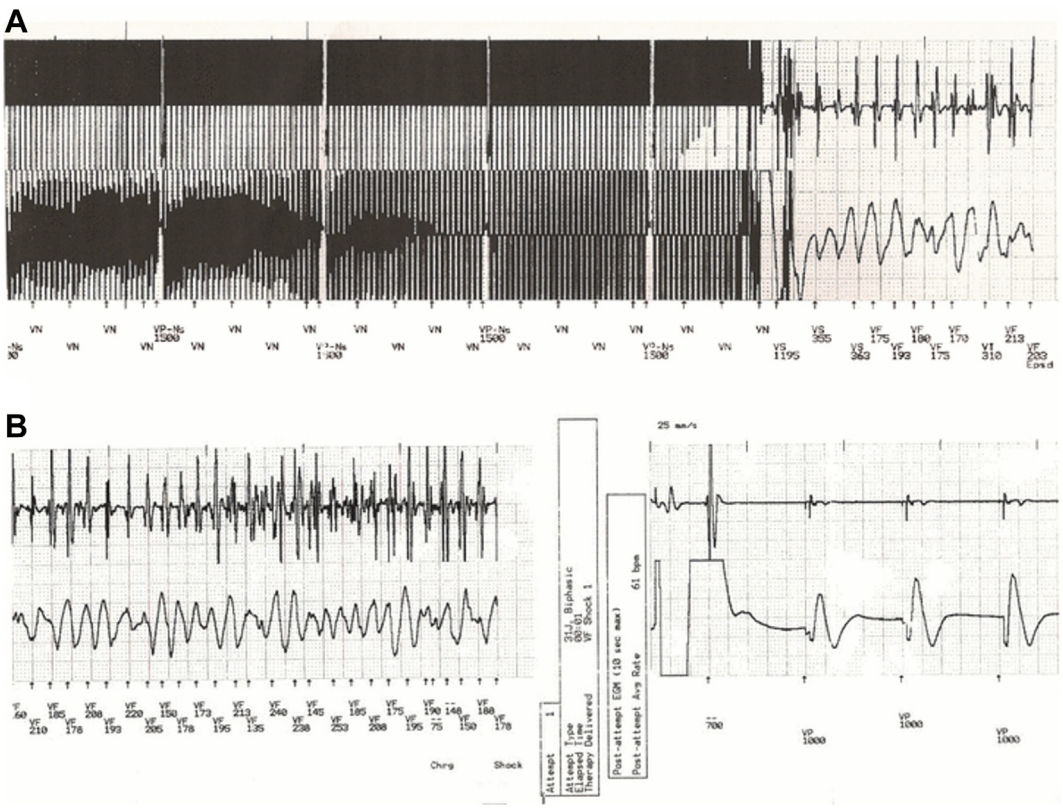

Figure 4 **A:** Electrogram showing 60-Hz interference followed by ventricular fibrillation (VF) detection after electrical source is released. **B**: VF is terminated by implantable cardioverter-defibrillator shock. (From Ginwalla et al[31] and used under STM permission guidelines.)

Lightning

Electrocution—from manmade sources—is presently understood to be synonymous with the induction of VF. On the other extreme, a lightning discharge (up to 500 megavolts) typically will cause substantial acute and chronic neural effects leading to temporary asystole and respiratory arrest.[39–42] Eventually, cardiac activity and spontaneous circulation typically return. In some cases, respiratory arrest persists, and the hypoxia will lead to asystole or pulseless electrical activity.[39] Andrews[43] used a sheep lightning model and found initial asystole followed by perfusing arrhythmias and ending with permanent asystole (presumably from electroporation neural damage). Karobath et al[40] reported both VF and asystole in their swine model of lightning strikes. Both asystole and VF have been reported in humans experiencing lightning strikes, with a typical progression of temporary asystole to VF.[41] The VF then finally deteriorates into asystole after approximately 30 minutes.[42] For primary VF, a side-flash is thought to be required in which a tree absorbs the vast majority of the strike energy, but a small arc emerges to strike a person standing close by.[30,44]

Conclusion

In this review paper, the experimental and clinical data supporting VF as the mechanism for electrocution was provided. Asphyxia as a mechanism is documented primarily in animal experiments.

Funding Sources: The authors have no funding sources to disclose.

Disclosures: Mark W. Kroll, Richard M. Luceri, and Hugh Calkins have been expert witnesses in electrocution cases and are members of the Axon Enterprises, Inc., scientific advisory board. Mark W. Kroll is an electrical safety consultant to Amarok, LLC, and a member of the Axon corporate board. Igor R. Efimov has no conflicts of interest to disclose.

Authorship: All authors attest they meet the current ICMJE criteria for authorship.

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