

Essentials of Low-Power Electrocutation: Established and Speculated Mechanisms

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Abstract – Even though electrocution has been recognized — and studied — for over a century, there remain several common misconceptions among medical professional as well as lay persons. This review focuses on “low-power” electrocutions rather than on the “high-power” electrocutions such as from lightning and power lines. Low-power electrocution induces ventricular fibrillation (VF). We review the 3 established mechanisms for electrocution: (1) shock on cardiac T-wave, (2) direct induction of VF, and (3) long-term high-rate cardiac capture reducing the VF threshold until VF is induced. There are several electrocution myths addressed, including the concept — often taught in medical school — that direct current causes asystole instead of VF and that electrical exposure can lead to a delayed cardiac arrest by inducing a subclinical ventricular tachycardia (VT). Other misunderstandings are also discussed.

1. INTRODUCTION

Electrical accidents are commonly misclassified as either low or high voltage with an arbitrary cutoff usually set at 1000 V. The voltage cutoff leads to some diagnostic errors. The 1,000,000-25,000,000 V Van de Graff generator does not cause injury as the power and current are almost zero. Nor does the 8 kV static shock (30 A peak) outlined in the IEC (International Electrotechnical Commission) standard.¹ A better classification is by power with a 1000 W cutoff.

The so-called “high-voltage” electrocutions include lightning and power line sources. A 7600 V power line can easily deliver about 60 kW of power to someone standing on the ground (or on an aluminum ladder) and touching the power line with a tool. Injuries can include arrhythmias, burns, superficial or deeper nerve damage, muscle damage with rhabdomyolysis and subsequent renal problems, and paralysis. Actual myocardial damage is often demonstrated.²⁻⁹

Such levels of systemic damage lead to numerous mechanisms of death and go beyond the scope of this review. Hence, this review will focus on low-power elec-

trocutations which cause death without causing systemic damage.

2. ESTABLISHED MECHANISMS

Arrhythmia Induction

The electrical injury of greatest concern is a lethal ventricular arrhythmia. The signature rhythm of electrocution is ventricular fibrillation (VF). It was long thought that there were only 2 means of inducing VF in the healthy heart with electrical currents. The first is the “shock on T” which involves delivering a single strong electrical pulse during the time of the T-wave to instantly cause VF.^{10, 11} The second method requires causing extremely rapid cardiac capture — typically > 450 BPM (beats per minute) — which induces VF within a few seconds in a normal heart.¹²⁻¹⁴ This is classical “electrocution.” This electrically-induced VF mechanism takes far less current than “T-shock” induction but also several pulses (typically at least 6 pulses.)¹⁵ It has recently been recognized that there is a 3rd method of inducing VF, namely with long-term high-rate cardiac capture causing sufficient ischemia to lower the VF threshold (VFT) to allow for the induction of VF.¹⁶ See Table 1 for a summary.

Single Pulse into the T-wave

The T-wave represents the time when the myocytes are returning back to their “resting” state. Some cells are absolute refractory while some are relatively refractory to electrical stimulation. Still others are depolarized and amenable to stimulation. A shock of appropriate charge during the T-wave leads instantly to VF from reentry.¹⁷ That is why the T-wave is referred to as the “vulnerable” portion of the heartbeat. For blunt trauma, mechanical energy delivered into the T-wave can also induce VF with a condition referred to as “commotio cordis.”^{18, 19}

Dorian, et al reported that delivering electrical charge into the T-wave sufficient to induce VF took a mean of 19 J (joules) with external patches.²⁰ One can calculate that this corresponds to an electrical charge of about 100,000 μC (microcoulombs) assuming typical external defibrillator capacitances.²⁰ Swerdlow had a patient (unpublished) that he induced with only 1 J which (assuming typical capacitances) corresponds to about 20,000 μC of delivered electrical charge.²¹ The value of 5,000 μC is what the IEC considers to be at the 50% probability of VF risk with unidirectional impulse currents of short durations delivered into the T-wave.²²

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Multiple Pulses And “Direct” VF Induction

Sufficiently strong repetitive external currents delivered to the heart will capture epicardial cells. According to the “multiple wavelet” hypothesis of VF, formation of new wavelets occurs through the process of wave break (or wave splitting), in which a wavelet breaks into new (daughter) wavelets.²³ Wave break occurs at sites of electrophysiological inhomogeneity, where regions of refractoriness provide opportunities for reentry to form. Epicardial cells are intrinsically heterogeneous in their repolarization properties.²⁴ This intrinsic heterogeneity provides a substrate for reentry formation during rapid (~ 450 BPM) capture. When a portion of the incoming wave front encounters refractory tissue while other portions continue to propagate, wave break occurs leading to VF.

The VFT is the amount of current required to induce VF in a particular subject with a particular electrode location. Reilly, in his text “Applied Bioelectricity” compiled all published studies on the effects of a pulse-train duration on the direct electrical induction of VF.²⁵ The VFT went down, with increasing pulse-train durations, until the exposure duration reached 1-5 s. This is reflected in recognized standards, as shown in Figure 1 taken from Reilly.²⁶ In other words, if an electrical current does not fibrillate within about 5 s it will not fibrillate with longer durations, (except as shown below by the 3rd mechanism of extended high-rate capture ischemically lowering the VFT).

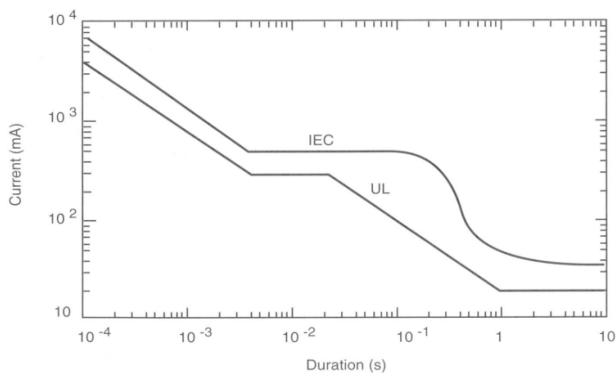


Figure 1. UL (Underwriters Laboratories) and IEC standards for VF risk suggest that VF is either induced or not in the first few seconds.

Long-Exposure High-Rate Cardiac Capture Ischemically Lowering VFT

High-rate cardiac capture with current densities of about 40% of the VFT will cause hypotensive collapse and will lead to VF after 90 seconds.²⁷ The current densities for this hypotensive response are above the threshold for continuous hypotensive capture.^{28, 29} Continuous cardiac capture at rates of >220 BPM, in swine, can eventually lead to VF. The required durations for this are on the order of minutes rather than seconds.

Prolonged rapid capture reduces cardiac output at the same time that the heart muscle continues to need

blood. This causes ischemia sufficient to lower the VFT in about 90 seconds in swine. In large mammals, with ischemia the VFT is cut to about 40% (of the direct-induction VFT).

The ability of rapid short pulses to induce VF is approximately equal to a 60 Hz AC current with RMS current of 7.4 times the aggregate current of the rapid short pulses.³⁰ For example the aggregate current of a nerve stimulator with 100 μ C pulses with a rate of 20 PPS is 2 mA. This has the VF-inducing capability of an AC source of 14.8 mA RMS. Note: The US FDA refers to this aggregate current value (charge • pulse rate) as the “average” current.³¹

Table 1. Mechanism of electrocution for various shock durations.

Duration	Mechanism	Qualitative Current Levels
1-10 ms	Single Pulse Shock on T	Requires very strong current.
1-5 s (train)	Direct induction of VF	Possible with strong electric current in any part of a ventricle.
5-80 s* (train)	<i>No known mechanism</i>	Unlikely with electrical current unless current is at the edge of the VFT.
90-300 s (train)	High-rate cardiac capture leading to ischemia lowering VFT.	Possible with weaker current in any part of a ventricle.

*There are some controversial data with 80 s exposures. However, this was in unventilated anesthetized swine.^{32, 33}

Figure 2 shows the 3 distinct time scales for the induction of VF by electrical current. The T-shock induction occurs instantly and is shown by the vertical line at 0 seconds. Direct (multiple pulse) induction of VF occurs typically in 0.1 - 5 seconds with the current required decreasing rapidly. Long-duration continuous high-rate capture (with current densities ~40% of the VFT) leads to an ischemically reduced VFT after ~ 90 s, or longer. It is important to note — and clearly shown by Scott’s canine data — that there is no known mechanism for VF induction taking 5 – 60 s.³⁴ The Nimunkar swine results required a median 150 s (300 s with 50% duty cycle of 5 s on and 5 s off) to induce VF with high-rate capture.³⁵ Finally, Roy showed that a cardiac arrest would always occur within 300 s with hypotensive capture in canines.³⁶ In swine current durations between 5 and 80 seconds are not shown to cause VF.²⁷

3. SPECULATED MECHANISMS

There are 4 speculated additional mechanisms for low-power electrocution. These are: respiratory arrest, asystole from direct current, induction of an intermediate ventricular tachycardia (VT), and accommodation of the VERP (ventricular effective refractory period).

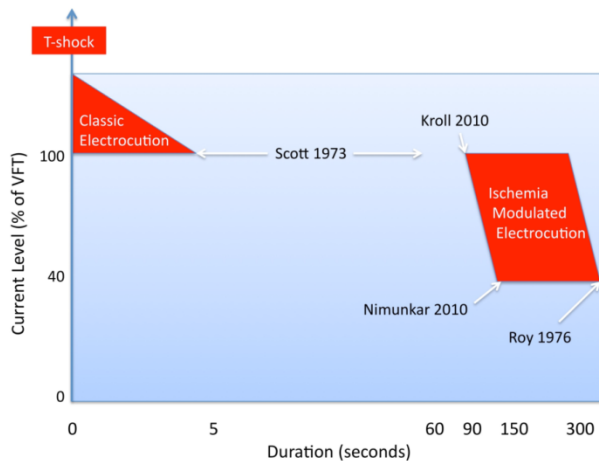


Figure 2. There are 3 distinct shock duration periods with which VF can be electrically induced.

Respiratory Arrest

Sufficient thoracic current can temporarily induce respiratory arrest. This current can be lower than the threshold for VF especially if it is conducted hand-to-hand as that can provide a pathway via the trapezius muscles capturing the phrenic nerves near the neck. There is even a case of respiratory compromise from a TENS (Transcutaneous Electronic Nerve Stimulator) delivering maximum current across the chest for angina pectoris.³⁷ Lightning can certainly cause respiratory arrest by first causing permanent damage to the brain.⁸

However, the speculated mechanism of an electrically-induced respiratory arrest raises several questions. The most likely scenario would be someone grabbing an exposed source with the total pathway resistance limiting the current to a level between 20-50 mA AC. Note that this is a very narrow band of currents. The current must be greater than both the “let-go” levels and the minimum for respiratory arrest.^{38, 39} However, it must be $< \sim 150$ mA (depending on the current pathway, and the subject’s weight) or VF would be instantly induced.⁴⁰ And, it must be less than 40% of the VFT or high-rate cardiac capture will lead to VF in 90 seconds which is sooner than a death from respiratory arrest.¹⁶ AC currents of 50-80% of the VF threshold will also cause temporary cardiovascular collapse due to rapid regular excitation.^{28, 41}

Electrical bilateral stimulation of the forearm muscles will quickly cause fatigue as electrical muscle stimulation is very fatiguing, per se, and the bilateral stimulation is tiring.⁴²⁻⁴⁴ Thus, the subject would release the connection. No documented case of low-power electrocution via respiratory arrest has been identified. Therefore it is most likely that persons with apparent respiratory arrest due to transthoracic current flow will die from VF rather than from asphyxia itself.

Asystole from Direct Current

It is a common misunderstanding in the medical fields that AC causes VF but that DC (or a battery) causes asystole. This is reinforced by the US Medical Licensing Examination which sometimes has a question related to the myth that batteries can cause asystole.⁴⁵ This is, of course false as batteries can be touched directly to the epicardium and will only produce VF.^{17, 46} It is hard to pinpoint the source of this myth. Portable external now-obsolete monophasic defibrillators would sometimes cause post-shock asystole. (Modern biphasic defibrillators can also cause post-shock PEA and asystole.) They were classically referred to as “DC” defibrillators to differentiate them from the utility-powered AC defibrillators. Also, lightning can certainly cause asystole through nerve or cardiac nodal damage and lightning is, indeed, a DC shock.^{47, 48}

VT and Delayed Cardiac Arrest

A speculation — that has been raised to attempt to argue for a long gap between an electrical exposure and VF — is that the electrical current induced an intermediate VT. Important definitions:

1. Sustained VT: a VT that lasts for more than 30 seconds.
2. Unstable VT: a VT that causes symptoms — most commonly passing out (syncope). Note that the “unstable” refers to the blood pressure being unstable — not the VT.

The intermediate VT induction speculation is scientifically unsupportable for several reasons:

1. A sustained VT cannot be induced in the absence of a myocardial reentrant substrate like nonhomogenous scar caused by a prior myocardial insult (myocardial infarction, myocarditis, etc.).⁴⁹⁻⁵² By definition, only a sustained VT would possibly allow a delay to cardiac arrest of more than 30 seconds. A small exception is HCM (hypertrophic cardiomyopathy). In some HCM patients a sustained VT can be induced with specialized pulse sequences but this is not expected.⁴⁹ In a swine study, a VT was induced in a normal heart (probably by a large infusion of epinephrine) but it only persisted for 7 seconds.⁵³ Meanwhile the blood pressure was 40 mm suggestive of the unstable nature of the rhythm with ensuing hemodynamic compromise. (The subject would have lost consciousness had it not been already anesthetized).
2. A VT that will lead to VF is almost always an “unstable” VT. An unstable VT will degenerate into VF within 34 ± 7 seconds in humans.⁵⁴ Hence, a VF delayed by, say, 60 seconds is extremely unlikely.
3. Any VT that leads to VF has such a rapid heart rate that it almost always leads to immediate syncope (loss of consciousness).^{54, 55}
4. VT induction generally requires specialized pulse timings and is generally not inducible with steady rate currents such as those from utility power, DC, or

an ECD.^{52, 56} Using specialized pulse timings, Cua was able to induce monomorphic VT in patients with a history of VT; however, steady AC stimulation universally induced only VF.⁵⁷

5. VT has never been documented in the literature as a cardiac rhythm in ARDs (arrest-related deaths) where an electronic control device (ECD) was temporally used.^{58, 59} In many cases of deaths considered temporal with ECD usage, the suspect was being cardiac monitored *before* any arrhythmias developed. And in these incidents VT was also not seen.
6. There are rare cases of VT following electrical injury, but none progressed to a cardiac arrest. Haim reported a VT in a 17 yo male electrocution who was hypotensive yet conscious.⁶⁰ Jensen reported a 45 yo woman that had VT after an alleged exposure to 430 VAC.⁶¹ This case is suspicious as she did not present until 60 days after the alleged exposure. Neither of these cases led to VF or any type of cardiac arrest and hence are not directly relevant.

There are some very uncommon VTs that do not require a diseased heart. These include:

1. Idiopathic normal heart VTs (originating in the RV and LV outflow tracts, His Purkinje system, aortic cusps, and sub valvular regions)
2. Catecholaminergic Polymorphic VT (CPVT)
3. Torsades de Pointes (TdP) is a polymorphic VT spontaneously induced by metabolic (low K, low Ca or low Mg) or drug (dofetilide, sotalol, some psychotropics, and methadone) related effects.

These are not relevant as they are either nonsustained, difficult to electrically induce, not associated with cardiac arrest, or very symptomatic and associated with syncope.^{56 62 63}

Several literature reviews have cast doubt on the possibility of a delayed cardiac arrest from low power electrical stimulation.⁶⁴⁻⁶⁶

There is a controversial example of a swine with an electrically-induced VT lasting 3 minutes which appears — at first blush — to contradict the human data that a sustained VT generally requires an infarct scar.³³

1. The pig had received 80 seconds of high-rate cardiac capture which would cause severe myocardial ischemia. In addition, the anesthetized pig was not allowed to breath during the 80-second ECD application (40 s on, 10 s break, and then 40 s on), which would have exacerbated the ischemic acidosis. This pig may have started out with a normal heart but it was far from normal when this VT was documented.
2. Not only was this seen in only 1 of the 6 swine tested, a sustained VT has never been documented in numerous swine studies where the animals were allowed to breath.^{27, 53, 67-71}
3. The high rate of the VT would have resulted in a loss of pulse and consciousness and thus is not applicable

to the typical human ARD case where delayed VF is speculated.

VERP Accommodation

The Ventricular Effective Refractory Period (VERP) accommodation theory suggests a new mechanism for electrical stimulation inducing VF in, say, 37 seconds after failing to do so in the initial 5 seconds. This contradicts all published literature which shows that an electrical current insufficient to induce VF, in a normal heart, in 5-seconds cannot do so until 90 seconds of exposure.^{12-14, 26, 28, 34, 36, 72-76} This VERP theory is based on the idea that the high-rate continuous capture would cause the VERP to gradually shorten and this would lead to VF.

The VERP is the heart's own "governor" that safely limits the rate at which the heart can beat. For a healthy human at rest, the VERP normally ranges around 200-250 ms, even though the heart may be beating at a much slower rate. This means that, with a VERP of 250 ms, the maximum heart rate, with continuous electrical stimulation below the VFT, is about 4 times a second or 240 BPM. Catecholamine (adrenaline) release in the body from agitation and stress might also shorten the VERP. However, the theory is that increased heart rate, along with the catecholamine increase caused by the agitation shortens the VERP to something like 200 ms, allowing the capture rate to increase to 300 BPM. That new rate further shortens the VERP, which increases the heart rate still further, perhaps triggering irregular captures (the capture ratio does not necessarily remain constant) that adds to the disorganization of the heart beat, until a rate of 450 BPM results which produces VF.

The VERP theory suggests that over a century of published research on VF induction and electrical safety standards is wrong.

The argument behind the VERP theory appears to be that intermittent stimulation changes everything. E.g. the movement of the chest wall changes the spacing between a chest contact and the heart and thus cardiac capture is irregular. There are 3 major problems with this VERP theory speculation:

1. Electrical safety studies used ventilated animals so their chests were being inflated regularly by the ventilator. None of these researchers reported intermittent capture even though the heart's thoracic position was changing.
2. The animal VF safety studies would slowly increase the AC current until VF was finally induced. For example, Scott used steady currents for 60 seconds at each current level.³⁴ As he would get close to the VF threshold, he would have certainly had some intermittent capture. This never led to any surprisingly low VF inductions being reported.
3. Human endocardial pacing studies show that even 20 μ C pulses — subthreshold since delivered during the refractory period — *extend rather than shorten* the VERP.⁷⁷ This directly contradicts any speculation

— that intermittent capture from a rapid source would facilitate VERP accommodation — as the non-capturing pulses would be subthreshold during the refractory period and would extend it.

The VERP accommodation theory apparently rests on an animal study.⁷⁸ Unfortunately, for this VERP theory, human studies are not supportive. The Zipes group paced human ventricles at 150 BPM for 30 minutes.⁷⁹ They concluded (abstract), “In contrast to traditional concepts of refractoriness, after the termination of sustained rapid ventricular rates, VERP prolonged.”

Even if the VERP does shorten, it does not adjust fast enough or far enough. Morady et al published a human study of 23 patients.⁸⁰ The study found that the VERP adjustment took 93 ± 34 seconds (233 ± 85 beats) of continuous rapid cardiac capture (150 BPM) to get to a 200 ms VERP. And none of the patients ever had VF. The same study also looked at the effects of catecholamine in 6 of the patients. They found that the minimum VERP was 202 ± 7 ms vs. 206 ± 12 ms (NS) with and without catecholamine influence.

Finally, the Morady study maintained the rapid cardiac capture for 10 minutes ($n=12$ patients) and the VERP never decreased below 200 ms. And, VF was never seen. Thus the scientifically unsupported speculation that this VERP theory mechanism would eventually lead to VF is unfounded and contrary to the published human literature.

The Morady study, used 100 and 150 BPM pacing and then used Methods “A” and “B” to determine the VERP. Only the 150 BPM Method B represents relevant human data since it alone achieved a 200 ms VERP as required for the VERP theory. For the relevant 150 BPM Method B results the fastest responding patient achieved his lowest VERP after 76 beats or 30 seconds. This was the extreme case and VF was still not induced.

Also, none of the recent published animal or human data shows any increase in the capture rate with time from rapid stimulation at 1120 BPM. Figure 2 of Nanthakumar shows a steady rate of capture over a 15-second ECD application.⁵³ The Cao pacemaker capture case shows steady capture measured at 281-290 ms cycle length.⁸¹ The Ho human capture case showed a steady capture rate of 240 BPM (CL = 250 ms).⁸² Lakkireddy never saw the capture rate increase during his multiple animal tests.^{70, 71}

4. CONCLUSIONS

There are the 3 established mechanisms for low-power electrocution: (1) shock on cardiac T-wave, (2) direct induction of VF, and (3) induction of VF from long-term high-rate cardiac capture ischemically lowering the VFT. We examined 4 other proposed mechanisms: (1) respiratory arrest, (2) asystole from direct current, (3) induction of an intermediate ventricular tachycardia, and (4) ac-

commodation of the VERP (ventricular effective refractory period). None of the speculated mechanisms are scientifically supportable.

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