Electronic Control Devices: Science, Law, and Social Responsibility

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The use of deadly force by law enforcement personnel is a hot-button issue in contemporary society. Their responsibility is to handle life-threatening situations while so far as possible avoiding fatalities or serious injuries of suspects, bystanders, or themselves. In these settings, the use of lethal weapons must be weighed against reactions to circumstances that do not pose an imminent threat of a fatal outcome. Moreover, use of intense physical restraint instead of lethal weapons in less ominous situations is not without risk either, in that fatal events due to asphyxia have occurred as an unintended consequence of this “non-lethal” control strategy. Moreover, the decision whether deployment of a lethal weapon, as opposed to physical restraint, is the proper action in a specific confrontation often must be made quickly, leaving little time for assessing options.

The development of electronic control devices (ECDs), or stun-guns, emerged as an alternative strategy, allegedly effective for controlling a threatening confrontation without resorting to needless serious injuries or fatalities. In principle, the ECD is more forgiving of errors of judgment than a firearm. The ECD is designed to deliver a painful and physically disabling electric shock, temporarily incapacitating a threatening individual. Although a significant number of deaths in association with, or following the use of, ECDs have been cited in a lay publication,1 peer-reviewed literature on the topic has been limited to several case reports, largely in the form of letters to the editors of journals.2 However, associations or correlations, in the absence of extremely strong population-based hazard ratios,3 are inadequate to support a scientific conclusion of causation, without additional information supporting the likelihood of a direct causal relation in an individual case. Data limited to a few case reports makes it difficult to provide informed statements about safety and appropriate precautions.

New Information
In this issue of Circulation, Zipes provides observational data from a series of 8 cases in which a reasonable accumulation of information on surrounding circumstances, clinical and pathologic data, and documented arrhythmias is provided. The source of his data is unique, as it is derived from information provided to him in his disclosed role as an expert during litigation of these cases. Based upon his analysis of the data, Zipes opines that the data from the cases he analyzed, in conjunction with previously published clinical and experimental observations, suffices to support a conclusion that ECD shocks, in some circumstances, can cause electrical capture of the heart and initiate cardiac arrest. He does not conclude that these cases reveal a fundamental flaw in the design of the devices.

**Analysis of the Data**

The most compelling elements in the data provided are that in each of the reported cases, loss of consciousness (LOC) occurred during the application of an initial or subsequent shock, and that in at least 6 cases the initial rhythm recorded was ventricular tachycardia/fibrillation. Since LOC under the reported conditions does not necessarily equate with cardiac arrest, the implications of these observations are diluted to some degree by the inability of the device to record the rhythm at the time of LOC, and by the lack of data that uniformly establishes the absence of a pulse immediately upon LOC. In 3 cases, the subjects were observed to be breathing after LOC, and two of these had a pulse initially. The observation of “breathing” does not, however, exclude cardiac arrest, since “gasping” respirations can occur during cardiac arrest and be confused with true breathing by lay observers. In addition, blood alcohol levels >0.30 gm/100 ml were recorded in 2 subjects; such levels themselves may be associated with LOC and, occasionally, death.

The source of the data leads to some concerns about distortions and biases that can
develop during the adversarial litigation process; but overall there is enough objective data to support reasonable judgments in the individual cases, if not definitive conclusions generalizable to all cases. Based on the circumstances, timing, and rhythm strips provided, and the pathologic data provided, it seems reasonable to conclude that some finite number of these cases, greater than zero, but likely less than all 8, demonstrates a direct association between delivery of an ECD shock and the onset of cardiac arrest in an individual in whom other possible causes are not present. One of the problems in interpreting the data is that there were undisputed pathologic findings of a normal heart in only 2 of the 7 autopsied fatal cases, with a mildly elevated heart weight and a blood alcohol level of 0.25 gm/100 ml in one of these 2. In both of these cases, the descriptions of the incidents and supporting data lend credence to the likelihood of an association that is strong enough to demonstrate a cause-and-effect relationship.

In the remaining cases, various levels of uncertainty result from the description of associated pathological findings; and in some cases, disparate opinions were offered by pathologists on opposing sides of the cases. In addition, in 2 cases, the delay between collapse and loss of pulse is consistent with LOC due to causes other than direct induction of ventricular fibrillation or pulseless ventricular tachycardia by the ECD shock, since a direct induction of a pulseless arrhythmia would likely be immediate. Certainly, an initial hemodynamically-stable ventricular tachycardia that subsequently deteriorated to a pulseless ventricular arrhythmia could not be excluded. These considerations limit the ability to establish independent causation in each case, but they are not sufficient to exclude an ECD shock as a possible cause or significant contributing factor to the induction of ventricular fibrillation consistently. Within the limits of the data provided for each of the cases, it seems fair to conclude that Zipes has provided sufficient information to amount to a proof of concept for a potential causal relationship between
an ECD discharge and the initiation of ventricular fibrillation. The likelihood of a causal relationship in any individual case would have to be judged on the basis of the combined information available. This can be challenging, because it is often difficult to distinguish between etiologic pathology and bystander pathology when the circumstances of a death are complex.

**Stress Physiology and Causation**

The interpretation of causation is made more complex by the stressful nature of the circumstances in which an ECD is usually used. The stress response may be mediated by the confrontation itself, by pain from the delivered shock, or by a combination of the two. A stressor resulting in epinephrine release is associated with greater vulnerability to electrical induction of ventricular fibrillation, in both normal and abnormal hearts. However, a stressor may also have adverse effects independent of its influence on responses to direct electrical stimulation of the heart. If epinephrine release is assumed to interact with a pre-existing pathologic abnormality in the causation of a cardiac arrest, it is difficult to determine whether the pathophysiological cascade leading to cardiac arrest is initiated by the confrontation or pain from the shock, as opposed to direct electrical stimulation of the heart. In 3 of the cases, there are contested diagnoses of hypertrophic cardiomyopathy, right ventricular dysplasia, and lymphocytic myocarditis, each of which, if present, can be associated with cardiac arrest during stress.

Transient stressors, other than ECD shocks, can be triggers for cardiac arrest. For example, on the day of the 1994 Los Angeles earthquake, the incidence of sudden cardiac death in the Northridge area was >5 times higher than the expected rate, as calculated from multi-year historical controls, and the incidence during the week following the earthquake was lower than expected. This led to the conclusion that the stress of the earthquake advanced events about to
happen among people with coronary disease to a single point in time when a common stressor was shared by the population. Observations such as this further complicate the identification of causation in disparate, stress-associated cases. Accordingly, the cumulative experience and data available in the Zipes report, and in other reports, cannot be generalized to a conclusion of a fundamental flaw in the concept, design or properties of ECD devices that can be generally applied to all cases. Each case must be evaluated in light of all its facts and conditions, including proper use of the device, in order to arrive at an opinion on specific causation.

**Pathophysiology and Biological Plausibility**

There are arguments supporting the claim that, under appropriate circumstances, it is biologically plausible that an ECD discharge can be a direct trigger for ventricular fibrillation. Both clinical concepts and experimental data support this, and are well-summarized in Zipes’ discussion.² The most salient points are that the energy delivered by the device is sufficient to achieve transthoracic capture when delivered to the anterior chest, analogous to clinical transthoracic pacing,⁷ in combination with a rate of stimulation that is sufficient to induce ventricular fibrillation. The notion that epinephrine release enhances the ability to achieve capture is supported by experimental studies, but its significance is confounded by the presence of pre-existing disease which can also be influenced by epinephrine release. Published experimental studies support these clinical notions, and therefore the proof-of-concept conclusion. Clinical and experimental data indicating biological plausibility is among the factors supporting a transition from an observation of an association to acceptance of a conclusion of causation.³

**Random Selection Bias versus Population Effect**

The absence of any form of control group, such as a denominator providing numbers of ECD shocks delivered without potentially fatal consequences, and their corresponding circumstances,
complicates efforts to determine the magnitude of risk of adverse events. Unfortunately, the accumulation of such data, absent a registry of ECD discharges, is virtually impossible to collect retrospectively. Local or regional registries, if established in the future, could both provide denominators and correct any under-reporting of adverse events. It would be very helpful to be able to estimate the frequency of LOC without cardiac arrests under circumstances similar to these 8 cases, as well as the incidence of confrontation-associated cardiac arrests in these circumstances, absent the deployment of ECDs.

**Transition from Population Science to Rules of Evidence in Law**

Causation arguments in civil law cases employ the dual concepts of *general* and *specific* causation. The former refers to the claim that a substance, device or action has properties that *can* cause the adverse outcome in question, while the latter asks the question whether it *did so* in the specific case under consideration. The U.S. legal process usually requires demonstration of general causation before a specific causation claim can be argued.\(^8\) Under the Federal Rules of Evidence,\(^9\) as elucidated by the U.S. Supreme Court in *Daubert v. Merrell Dow*,\(^10\) scientific reliability and relevance, as determined by judges and based on expert testimony during formal hearings on evidence, defines what can be placed before a jury. In some rulings, the absence of statistically significant population data has been held to be grounds for excluding general causation claims.\(^11\) Questions of specific causation can become even more complex. A number of jurisdictions have accepted the notion that a relative risk greater than 2.0 is necessary and/or sufficient for proof of specific causation, most notably in toxic tort cases.\(^12\) There are theoretical and practical reasons why this standard is flawed, not the least of which is the fact that it shifts *individual* specific causation to a *cumulative* metric of causation, which conceptually is more aligned with general causation than specific causation. Accordingly, the absence of any
population data on the ECD/cardiac arrest issue is a limiting factor for general causation claims, as well as specific causation based on the relative risk standard. This relegates specific causation theories to associations supported, but not necessarily determined, by factors such as temporal relationships, biological plausibility, and experimental evidence. In addition, rulings prohibiting specific causation arguments, in the absence of accepted evidence for general causation, have not been uniform across all jurisdictions.

**Lessons Learned From the ECD Data**

Practical questions pertaining to the use of these devices by law enforcement officers derive from observations such as those reported. In the absence of information that undermines the rationale for, and general usefulness of, these devices as an alternative to lethal weapons or potentially dangerous physical force, the use of these devices by law enforcement officers should not be stopped on the basis of the information provided in the report, as Zipes also notes.

However, two policy and procedure considerations emerge very strongly from the report. One is that there has to be appropriate education of law enforcement officers as to the potential, however remote, for an adverse outcome, either directly or as a contributing effect in the presence of pre-existing cardiac abnormalities. This calls for educational efforts that emphasize the potential concerns about factors that law enforcement officers can control, such as prolonged and repetitive shocks and avoiding shocks to the anterior chest, when conditions permit. The second precaution is that officers should be educated to consider that any subject exposed to such a shock who loses consciousness should be assumed to be in cardiac arrest until proven otherwise. Responses to these circumstances must not be delayed. When AEDs are available in police vehicles, they should be immediately deployed following LOC.

There remains an overarching ethical question about the proper uses of these devices.
If one assumes a very low risk of induction of ventricular fibrillation by ECDs, even with proper deployment and use according to instructions, then a distinction must be made between the use of these devices as an alternative to potentially lethal force and their use arbitrarily under circumstances in which physical interventions would be sufficient. Despite a low risk of the possibility of ventricular fibrillation as an unintended consequence of ECD use, their use seems appropriate to avoid the more likely fatal outcome with a lethal weapon. However, if ECDs are used indiscriminately under circumstances in which lethal weapons or intense physical force would not be necessary, then any risk of ventricular fibrillation induction would be unjustified. As with any lethal risk, the question whether use of ECDs is appropriate must be considered in light of the difficult and rapid judgments that must be made about the circumstances of individual cases. This ethical and practical challenge should be reflected in policies and procedures guiding law enforcement activities, and woven into the education of law enforcement officers who live with this responsibility daily.

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**References:**


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