Electronic Control Devices and the Clinical Milieu

In a 2006 issue of the Journal, Nanthakumar et al. (1) presented a study of Taser ECDs (electronic control devices) using 6 pigs averaging 50 kg (110 lb). Research on these devices is important as they are rapidly changing the practices of law enforcement in North America and the United Kingdom with over 620 uses per day. The ECD delivers its short, motor-neuron capturing pulses through a pair of small darts (or barbs) attached by very fine wires. Darts were positioned subcutaneously to maximize current through the heart, in a vector not possible in humans due to the differences in thoracic geometry. Some ventricular capture was seen but no arrhythmias induced during 94 full-strength applications. This is impressive as swine are more sensitive to electrical induction of arrhythmias than are other mammals (2), possibly due to the transmural penetration of the Purkinje fibers (3).

The researchers then infused epinephrine and delivered 16 ECD applications—again in the worst-case position. They had one case of ventricular fibrillation (VF) from these 16 applications. The investigators concluded that these devices may have cardiac risks. Although we recognize the solid reputations of the researchers and the quality review process of JACC, we have concerns about the applicability of this conclusion to the clinical setting. The first concern lies with the implicit assumption that the rhythm with a police “in-custody death” is VF. The ECDs are involved in 30% to 32% of in-custody deaths (J. Ho, unpublished data, 2006) (4,5). However, studies have not reported a single case in which the presenting rhythm was VF when an ECD was used. A study of 162 consecutive in-custody deaths found that, whereas there was a significant association of impact weapons with sudden death, the ECDs were never (0 of 50; p = 0.001) associated with a sudden collapse. This would seem to eliminate electrically induced VF as the cause of death.

The one anecdote, cited in Nanthakumar et al. (1), of possible electrically induced VF was misreported with material omissions (6). A violent subject exhibiting all the signs of excited delirium was briefly subdued with a short ECD discharge. Paramedics were present and found a normal pulse and respiration after the ECD discharge. After a 14-min delay, the subject collapsed and probably had an ideoventricular rhythm. After an aggressive therapy of 3 defibrillation shocks along with atropine and epinephrine, the subject finally had the VF strip shown in the published anecdote. A total of 23 min elapsed between the ECD application and the published VF strip.

Our second concern has to do with the timing of the shocks with the epinephrine infusion. The study does not mention any delay between the time of infusion and the ECD application. Although perhaps counterintuitive, epinephrine reduces the VF threshold only for the first few minutes (7). After that there is a supraventricular blockade, and the VF threshold is increased significantly above the baseline. This timing is concordant with the clinical scenario. Typically, an individual exhibits violent agitation with hyperactivity (8) for several minutes, and third parties call for help. The police require 5 to 15 min to arrive before they can apply any restraint device. At this point, the adrenergic tone has been elevated for several minutes. An epinephrine infusion minutes before an ECD shock would appear to give results opposite of those seen with the clinical scenario.

Finally, we would draw attention to the results of the Cleveland Clinic study published in the same issue of the Journal (9). This study used significantly higher exposures (total shock charge of approximately 2,000 five-second weapon discharges per pig versus about 50 for the Nanthakumar et al. [1]) and also evaluated the risk of the induction of ventricular arrhythmias. The investigators found no induction of arrhythmias except at a high multiple of the device output and that cocaine increased this safety margin even further. That would appear to be more consistent with the clinical results in which no objectively documented VF has occurred in 610,000 police uses of these devices.

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REFERENCES


Reply

We thank Drs. Pippin and Kroll et al. for their interest in our work (1). We disagree with the notion that experimental models are never of any use in understanding and in studying arrhythmias. Most of the work on mechanisms of fibrillation and defibrillation, including studies in cardiac arrest, have been conducted in guinea pigs, rats, pigs, and dogs. Readers of JACC are quite aware of the
limitation of experimental models. We do agree that extrapolation to humans has to be done cautiously, as discussed in our study.

The main point of our study was to demonstrate that electrical capture of myocardium, under specific circumstances, can occur after neuromuscular incapacitating device (NID) discharge. Unless the recording system is shielded from electromechanical interference, accurate assessment of cardiac effects of discharges is not possible, and conclusions about the safety of discharges are unreliable. We did not draw conclusions about the possible consequences of NID discharges reported in the media, but we wanted to highlight the potential for myocardial capture and potential risks of high-frequency cardiac stimulation under specific (and likely very uncommon in usual use) circumstances. We agree that it is not possible to directly extrapolate our results to NID use in humans.

The letter by Kroll et al. highlights the same difficulties, with regard to coming to safety conclusions based on the absence of objectively documented arrhythmias predicated on interviews and making surface recordings in humans. This line of argument regarding safety in humans can be misleading; as we have shown in our experiments, immediately before and after the NID discharge there was no observable cardiac stimulation. However, if one is able to "see through" the electrical artifact during the discharge, cardiac stimulation was seen. Until intracardiac recordings can be made in humans with shielding to obliterate the electrical artifact that obscures possible intracardiac events, making safety conclusions in humans is premature. We did not state that NIDs cause ventricular fibrillation in humans, and we agree that we cannot conclude from our study that NID discharges cause arrhythmias in typical use.

We hope that readers agree that our study does suggest the possibility that NIDs may, in some circumstances, cause cardiac capture, and that this possibility should at least be considered in future research in humans. We hope that our work stimulates such research, using similar methods, in this area in humans.

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Although it is always best to test a device or a drug in humans, such testing is not necessarily feasible in all situations. Extensive tests, such as those performed to assess the effects of electrode positions and drug effects, are all but impossible except in an anesthetized animal model. Animal models have been used in many studies and have contributed to our understanding of many physiologic processes directly applicable to human beings. The pig model, for example, has been extensively used in studying defibrillation in internal or external defibrillators. Those findings have extrapolated well to human beings.

However, human studies have limitations. Thus, we (1) would differ from Dr. Pippin’s broad indictment of animal studies as not being useful. Controversy exists in field reports of whether electrical stun guns have the potential, although infrequent, of directly causing ventricular arrhythmias. Human studies are limited in nature. Multiple exposures to the stun gun, prolonged exposure, seeking the most vulnerable thoracic sites, or safety margin of stun gun exposure would be impossible to perform in a human population. Monitoring of heart beats with transvenous electrodes would also be difficult to assess in a healthy group of human volunteers. The relationship of ventricular capture to induction of ventricular fibrillation (VF), for example, would have been difficult to assess in humans. The margin of safety and its relationship to thoracic position of the darts would also be difficult to assess. Although animals clearly do have a different anatomy, factors related to anatomic differences such as distance from the chest wall to the heart can be assessed.

We would also differ with Dr. Pippin’s assessment that the cocaine findings are in contradiction to the common impression that cocaine causes arrhythmias. Cocaine may cause coronary spasm and an increase in sympathetic tone. Both of these effects can lead to arrhythmias. Our findings are clearly stated (1). Cocaine reduces the ability of a stun gun to induce VF. We were careful to note that the sedated state may minimize some of the sympathetic responses associated with cocaine ingestion. However, given the manner by which VF is induced with the stun gun impulses, via ventricular capture, it is quite physiologic to expect that cocaine would actually increase VF induction thresholds. This finding would have been difficult to confirm in a human study.

Finally, although one always has to be careful in extending results of animal studies to humans, such findings are extremely helpful in understanding mechanisms and in directing potentially acceptable studies in humans that would further our understanding and facilitate increased safety or efficacy of the tested object.

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