

## Cardiovascular Consequences of Stun Gun

# Cardiac Electrophysiological Consequences of Neuromuscular Incapacitating Device Discharges

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| <b>OBJECTIVES</b>  | The purpose of this study was to evaluate the cardiac consequences of neuromuscular incapacitating device (NID)/stun gun discharge in an experimental model.   |
| <b>BACKGROUND</b>  | The large-voltage electrical discharges from NIDs have been suggested to pose a risk for triggering cardiac arrhythmias.   |
| <b>METHODS</b>     | Intracardiac catheters and blood pressure transducers were inserted before the application of NID discharges in six anesthetized pigs. Two different commercially available models (NID-1 and NID-2), two different vectors of discharges (thoracic: parallel to the long axis of the heart on the chest wall, and nonthoracic: away from the chest, across the abdomen), and two different durations of discharge (5 and 15 s) were tested. The effect of simulated adrenergic stress using epinephrine was also evaluated.   |
| <b>RESULTS</b>     | We studied a total of 150 discharges to 6 pigs; 74 of these discharges resulted in stimulation of the myocardium, as documented by electrical capture (mean ventricular rate during stimulation and capture, $324 \pm 66$ beats/min). Of the 94 thoracic discharges, 74 stimulated the myocardium, compared with none from 56 nonthoracic discharges ( $p < 0.0001$ ). During 16 discharges with epinephrine, there were 13 episodes of stimulation of the myocardium, of which 1 induced ventricular fibrillation and 1 caused ventricular tachycardia. Thoracic discharges from NID-1 were more likely to stimulate the myocardium than those from NID-2 (98% vs. 54%, $p = 0.0007$ ). |
| <b>CONCLUSIONS</b> | In an experimental model, NID discharges across the chest can produce cardiac stimulation at high rates. This study suggests that NIDs may have cardiac risks that require further investigation in humans. (J Am Coll Cardiol 2006;48:798–804) © 2006 by the American College of Cardiology Foundation  |

Neuromuscular incapacitating devices (NIDs), also known as stun guns, are used by law enforcement officers worldwide (1). Pulses of 50,000 V, 11  $\mu$ s to 50  $\mu$ s in duration, delivered at a rate of 16 to 20 pulses/s between the 2 darts fired by these devices, are intended to produce muscular incapacitation (2). Sudden deaths in association with the use of these devices have been reported, but a causal link between the device use and sudden death has not been established (1). Cardiac arrhythmia after NID device discharge has been documented (3), and concerns have been raised regarding the cardiac safety of these devices (4–6).

The short pulse duration of stimulation would be expected to have a small chance of stimulating myocardium, as opposed to nerve and skeletal muscle cells. However, if the device discharges caused a voltage gradient across the heart, cardiac stimulation could potentially ensue. The electromagnetic interference (EMI) with electronic recording equipment caused by the high-voltage discharge (7) has in part limited electrophysiological characterization of NID

discharges. There are reports of surface electrocardiographic monitoring studies of healthy volunteers before and after NID discharges (8); however, none of these examined the cardiac electrophysiological consequences during the NID discharges with the aid of intracardiac monitoring. We devised an experimental model shielded from EMI to characterize the cardiac electrophysiological consequences of NID discharges in a porcine model.

## METHODS

**Laboratory animals.** Farm pigs weighing 45 to 55 kg were used in this study. The pig was selected for this study because previous studies of NIDs had also used a porcine model (9,10). Approval of the research protocol was given by the Animal Research Committee of the University Health Network, and the study was conducted in accordance with the regulations of the Canadian Council on Animal Care. In our experimental model we tested two different vectors of discharges (thoracic: darts placed across the chest/heart, and nonthoracic: across the abdomen; see later text). We also tested two different commercially available models with different electrical specifications (NID-1 and -2) and two different durations of discharge (5 and 15 s).

Seven animals were studied. In the pilot animal the recordings were corrupted by EMI, and attention was paid

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#### Abbreviations and Acronyms

|     |                                       |
|-----|---------------------------------------|
| NID | = neuromuscular incapacitating device |
| EMI | = electromagnetic interference        |
| VF  | = ventricular fibrillation            |
| VT  | = ventricular tachycardia             |

to shielding the EMI effects during that study. All electrical cabling was shielded using either their built-in shielding sheath connected to a reference ground electrode via an ultra-low impedance path, or when individual wires were used, by channeling them as much as possible into metal conduits grounded in the same fashion. Thus the data reported here are from the six subsequent animals. In animal #1 we tested one vector and the two durations; in animal #2, different vectors, NIDs, and durations were tested, and we noticed differential effects between the two different guns and vector axis. Thus, in the subsequent animals (animals #3 to #6), we block-randomized the two different models of NIDs, vectors, and duration in random order. Every combination of device, stimulation vector, and duration was tested three times. Sixteen discharges were delivered during epinephrine infusion. In each animal, 4 additional discharges in the thoracic configuration, with the 2 NIDs, for 5 and 15 s, were delivered after removal of the intracardiac catheters with only a femoral arterial line for monitoring, to rule out a potential confounding effect of intracardiac catheters.

Over the course of study in six animals, a total of 150 NID discharges were analyzed. Discharges delivered without intracardiac catheters were not included in the analysis. **NID discharge.** Two NID devices were studied during the experiment. The X26 Advanced Taser (NID-1) and the M26 Taser (NID-2) (Taser International, Scottsdale, Arizona) were the devices used in the study. The two NID devices (X26 and M26) are quite different; the M26, as described in its patent application, has a power of 26 W and delivers 1.76 J of energy during each pulse (7). The NID-1 has a power of 6 W and delivers 0.36 J per pulse. The NID-1 sends a big pulse first for a short period (1.5  $\mu$ s) and then follows with a longer (50  $\mu$ s) but smaller wave. This is in contrast with the NID-2, which produces a single short impulse.

The guns were initially fired into a cardboard box; the barbs were removed and inserted subcutaneously in two different configurations on the pigs. These barbs that were shot into a cardboard box in this experiment are actually shot into the intended victim's clothing or skin from a distance of 5 to 15 ft and remain attached with a wire that delivers the electrical shocks. It is difficult to fire at a supine ventilated pig and achieve a dependable vector of discharge repeatedly. To control for this, we initially fired the gun into a box without the electrical discharge, removed the distal end of the barbs from the box while the proximal end was still attached to the gun via the wires, and reinserted the

barbs into the animal (in a configuration and inter-barb distance designed to estimate the barb location if the gun had been fired at a target about 7 to 10 ft away) and proceeded with the electrical discharge. This achieved the two different vectors without variation within and among animals.

An independent safety committee reviewing filed reports of skin penetration found that of the darts fired, a significant proportion (65%) of the deployments in the field did indeed penetrate the skin (11). Therefore, we decided to implement a subcutaneous insertion of the darts in our model. The skin was pulled up, and 1 cm of the barb was inserted parallel to the plane of the skin. Particular attention was paid to ensure that the darts were in the subcutaneous region and were not any deeper to avoid the possibility of direct stimulation through the pericardium.

The first configuration (thoracic) was designed to achieve the largest cardiac potential gradient of stimulation; one of the darts was placed in the right parasternal region, 5 cm away from the midline, and another dart was placed in the left lateral border of the thorax. The inter-dart distance was 26 to 30 cm. This is the average distance between the darts when fired at an object from 7 ft away (7). In the second nonthoracic configuration, the darts were affixed in an abdominal position with the darts attached below the lowermost ribs on the right and left, such that the vector was oriented across the abdomen.

The NID-1 and -2 were connected with the lead and dart systems according to a randomized sequence for each pig studied. Because it is illegal for civilians to operate NID devices in Ontario, Canada, the NID discharges were delivered by a weapons specialist, in the presence of the Deputy Chief Coroner, for durations of 5 or 15 s according to a randomized sequence for each pig studied.

**Pharmacologic stress.** Because individuals who need to be restrained are usually in a heightened sympathetic state, we also tested NID device discharge during simulated stress by infusing epinephrine. The epinephrine was administered as a continuous intravenous infusion at a dose of 0.1  $\mu$ g/kg/min to 0.7  $\mu$ g/kg/min titrated to increase the animal's heart rate to a 50% increase from the baseline before discharges.

**Experimental protocol.** The pigs were sedated with 12 mg intramuscular ketamine per kilogram of body weight and inhaled isoflurane. The pig was then intubated, and anesthesia was maintained with an inhaled mixture of 1.0% to 2.0% isoflurane and oxygen. A 6-lead surface electrocardiogram system was attached. Two 7-F venous sheaths were inserted into the right femoral vein, and one 8-F arterial sheath was placed into the right femoral artery. Electrocardiograms, blood pressure, and oxygen saturations were continuously monitored.

**Instrumentation and electrophysiological recording system.** Bipolar recording catheters were positioned in the right ventricle and coronary sinus under fluoroscopic guidance, approximately 4.5 cm apart. A high-fidelity micromanometer pressure transducer catheter (Millar Instruments, Houston,

Texas) was introduced into the arterial line and positioned in the descending aorta. All intracardiac signals were also connected to dedicated high-level channels on the recording system to quantify the voltage produced during NID discharge measured from the catheters (referred to as maximum intracardiac voltage, defined as the maximum voltage difference between the two electrograms at any point during discharge). The signals were sampled at 1 kHz, with a low pass of 200 Hz and a high pass filter of 0.05 Hz. We also performed a calibration study in which a capture threshold was determined for a 10- $\mu$ s pulse (Grass Telefactor, model S88, Astro-Med Inc., West Warwick, Rhode Island). Voltage was measured by both the mapping system and an oscilloscope with a sampling frequency of 1 million samples/s and a frequency bandwidth of 250 MHz. Our calibration showed that because of the sampling and bandwidth limitations in measuring a 10- $\mu$ s pulse, the mapping system measurements underestimated the true intracardiac voltage by 100-fold.

**Analysis.** The data were analyzed off line by two blinded independent observers (S.M. and I.B.) and verified by a third observer (K.N.). Myocardial stimulation caused by the NID discharge was defined as change in electrogram morphology and rate during the discharge and perturbation of arterial blood pressure. Episodes of arrhythmia and maximal intracardiac voltage were also analyzed. To test for differences in myocardial stimulation based on vector of discharge, a repeated-measures logistic regression was performed using the SAS system (SAS Institute, Cary, North Carolina). Vector of stimulation was treated as a fixed effect in these models, and an animal identifier was included as a repeated-measure effect in the model to control for the lack of independence between observations taken on the same experimental unit. We also created an alternate statistical model in which we treated the observations as independent and performed chi-square tests. To test for NID model effects and duration of discharge effects on stimulation of the myocardium within the thoracic vector, a series of repeated-measures logistic regression analyses were performed. The NID model and duration of discharge were treated as fixed effects in these models, and an animal identifier was included as a repeated-measure effect to control for the relatedness of multiple observations performed on the same experimental unit. To test for an association between maximal intracardiac voltage and vector of discharge, and to assess the effects of NIDs and duration differences in the discharges in the thoracic vector, a series of repeated-measures analysis of variance was performed on the natural-logarithm transformed absolute value of the voltages (because the voltage data were highly right-skewed and this transformation results in the data following a more Gaussian distribution). All effects of interest (i.e., vector of discharge, NID, and duration) were treated as fixed effects in these models, and animal identifiers were included as repeated effects to control for the relatedness of observations performed on the same subjects.

## RESULTS

### Myocardial stimulation: effect of the vector of discharge.

The mean weight of the pigs was  $49.9 \pm 1.2$  kg. We studied a total 150 discharges to six pigs; of these, 94 discharges were thoracic and 56 discharges were nonthoracic (Table 1). In the thoracic vector, 79% resulted in stimulation of the myocardium, compared with 0% in the nonthoracic vector ( $z = 22.24, p < 0.0001$ ) (chi-square = 77.87,  $p < 0.0001$ ). Figure 1 is a typical episode of NID discharge with a nonthoracic vector across the abdomen and illustrates the lack of myocardial stimulation. Figure 2 is a typical episode of NID discharge with a thoracic vector and illustrates myocardial stimulation and capture. In segment B during the NID device discharge, the surface electrocardiograph leads are corrupted by the EMI. However, the consequences of the NID are evident on intracardiac recordings and blood pressure recordings. The mean ventricular rate during stimulation was  $324 \pm 66$  beats/min.

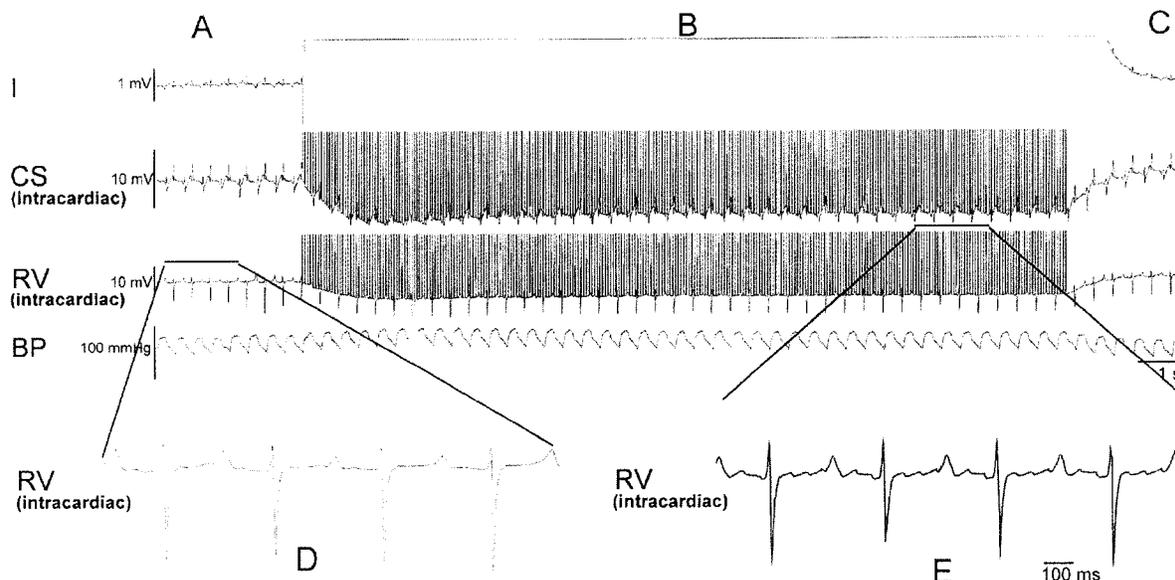
### Myocardial stimulation: effect of NID and duration.

Table 1 shows the effect of the two different NID models and the effect of duration. There was a significant effect on myocardial stimulation caused by NID model, with discharges from NID-1 more likely than those from NID-2 (98% vs. 54%) to stimulate the myocardium ( $z = 3.38, p = 0.0007$ ). Significant duration effects were also detected, with the longer duration more likely to result in stimulation of the myocardium than the shorter duration (Table 1) ( $z = 2.35, p = 0.0187$ ).

**Simulated stress.** All discharges during epinephrine infusion were delivered across the chest. There were a total of 16 discharges during epinephrine administration in four animals, resulting in 13 episodes of stimulation of the myocardium. The mean dose of epinephrine delivered to achieve the 50% increase in heart rate response was  $0.5 \mu\text{g}/\text{kg}/\text{min}$ . One episode resulted in ventricular fibrillation (VF), as shown in Figure 3A. In another animal, an episode of stimulation of the myocardium resulted in nonsustained

**Table 1.** Summary of Neuromuscular Incapacitating Device Discharges

|                | Capture |    |        |
|----------------|---------|----|--------|
|                | Total   | n  | %      |
| n discharge    | 150     | 74 | 49.33  |
| Vector effect  |         |    |        |
| Chest          | 94      | 74 | 78.72  |
| Abdomen        | 56      | 0  | 0.00   |
| Device effect  |         |    |        |
| X26 Chest      | 53      | 52 | 98.11  |
| M26 Chest      | 41      | 22 | 53.66  |
| M26 Abdomen    | 29      | 0  | 0.00   |
| X26 Abdomen    | 27      | 0  | 0.00   |
| Time effect    |         |    |        |
| X26 Chest 15 s | 28      | 28 | 100.00 |
| X26 Chest 5 s  | 25      | 24 | 96.00  |
| M26 Chest 15 s | 20      | 12 | 60.00  |
| M26 Chest 5 s  | 21      | 10 | 47.62  |

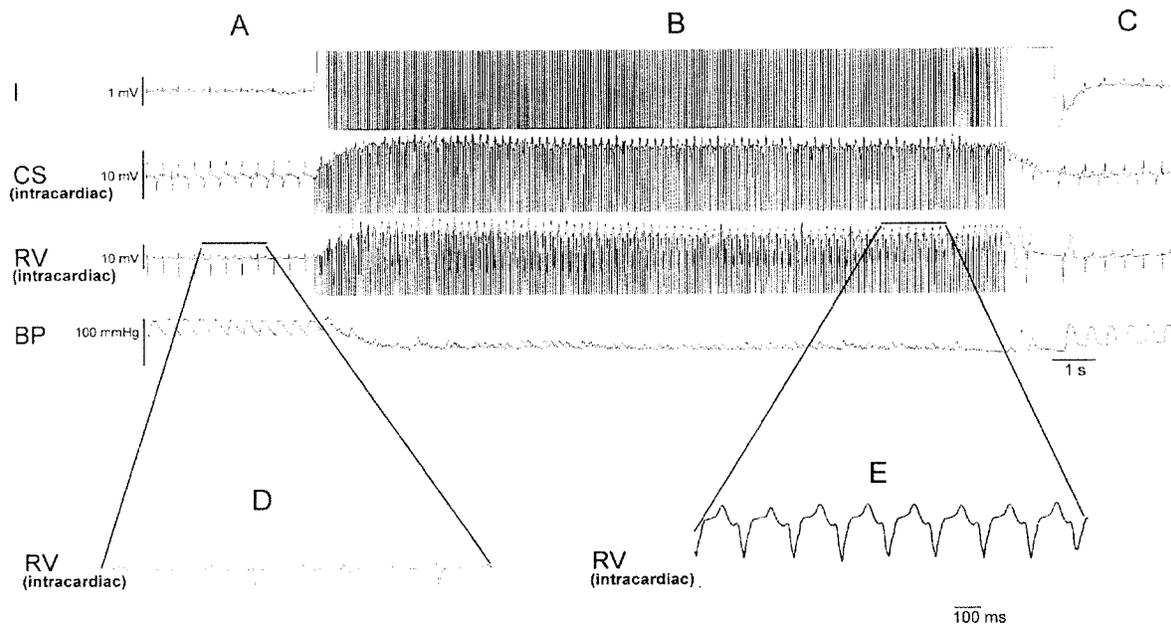


**Figure 1.** Nonthoracic neuromuscular incapacitating device (NID) discharge. This figure illustrates a typical episode of NID discharge in the nonthoracic vector configuration, which does not result in the stimulation of the myocardium. The surface electrocardiogram lead I, intracardiac electrograms from the coronary sinus (CS) and the right ventricular (RV) apex, and blood pressure (BP) recording from the MILLAR catheter in the descending aorta are shown. (A) The rhythm before the NID discharge. This shows regular rhythm. It is very similar to the rhythm and rate in (C). Interestingly, in B, the surface electrocardiograms are corrupted by the high voltage discharge. However, the intracardiac electrograms, as shown in D and E, do not show any significant change in rate morphology and are not phase locked (no temporal relationship between stimuli and the electrogram) with the NID discharge. Note also the lack of perturbation of blood pressure during the discharge. The rate and morphology are not significantly different between D and E, further illustrating the lack of myocardial stimulation.

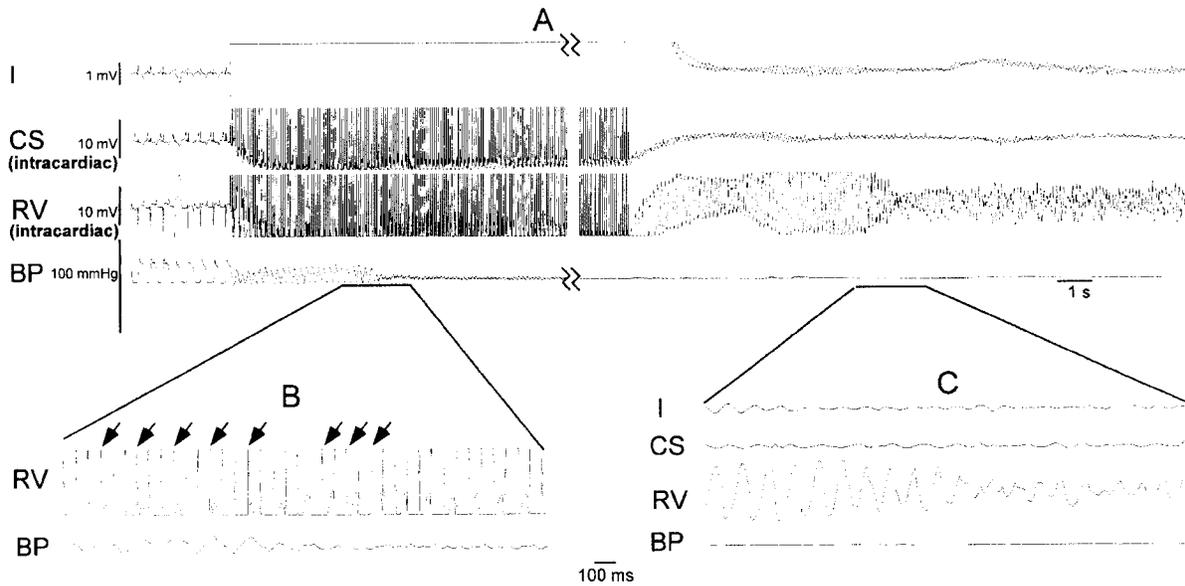
ventricular tachycardia (VT)/VF that spontaneously terminated (Fig. 4).

**Intracardiac voltages.** The test for an association between voltage and location found a significant effect

caused by location ( $F = 184.72$ , degrees of freedom [df] = 1.3,  $p = 0.0009$ ) with voltages in the chest being significantly larger than those in the abdomen ( $144 \pm 77$  mV vs.  $35 \pm 32$  mV). The tests to assess the effects of gun model



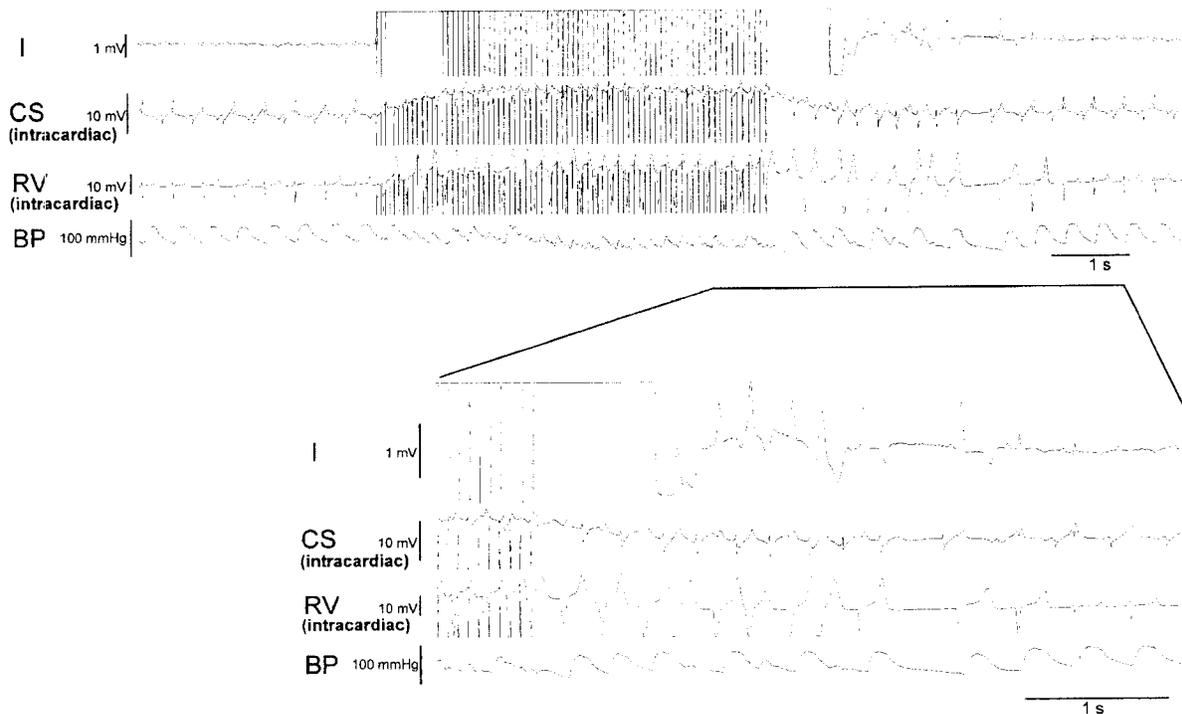
**Figure 2.** Thoracic neuromuscular incapacitating device (NID) discharge. This figure shows a typical example of an NID (model X26) discharge in the thoracic vector configuration. This shows again the corruption of surface electrocardiographic leads in B; however, in the intracardiac electrograms, electrical activity is noted. In C, after the NID discharge, spontaneous return of regular sinus rhythm and blood pressure are shown. Note the immediate return of the rhythm, similar to A. In D and E, the intracardiac electrograms have been magnified and the same duration is shown in both. It is evident in E that the rate is much faster and the rhythm is wider compared with D. The morphology of the tachycardia in E is wider than the morphology in D. There is a constant NID stimulus artifact to electrogram duration as shown in E, with every third NID discharge resulting in stimulation of the heart. Note the loss of blood pressure during the stimulation and the recovery of blood pressure once the discharge is completed. Abbreviations as in Figure 1.



**Figure 3.** Ventricular fibrillation during simulated stress and neuromuscular incapacitating device (NID) discharge. This shows an NID discharge in the thoracic configuration that resulted in ventricular fibrillation during epinephrine infusion (A). In B, as noted by the **arrowheads**, during the discharge there was a 3:1 phase lock of NID discharge that progressed to a 2:1 phase lock resulting in rapid ventricular tachycardia (VT), and in C this tachycardia degenerates into polymorphic VT that results in ventricular fibrillation. Abbreviations as in Figure 1.

and discharge duration were performed for thoracic discharges only. There were significant gun effects, with higher absolute voltages being associated with the NID-1 ( $195 \pm$

$64 \text{ mV}$ ) compared with NID-2 ( $77 \pm 18 \text{ mV}$ ) ( $F = 134.48$ ,  $df = 1,3$ ,  $p < 0.0001$ ). No significant duration effects were detected ( $F = 0.03$ ,  $df = 1,5$ ,  $p = 0.8711$ ).



**Figure 4.** Ventricular tachycardia during simulated stress and neuromuscular incapacitating device (NID) discharge. This shows an NID discharge in the thoracic configuration that resulted nonsustained ventricular tachycardia that spontaneously reverted back to sinus rhythm. In the enlargement, the time of onset of tachycardia shows the possibility that the penultimate NMI discharge was delivered at the vulnerable period of the T-wave, resulting in the arrhythmia. Abbreviations as in Figure 1.

## DISCUSSION

This report describes the first experimental model to assess the cardiac electrophysiological consequences of NID discharge. When the discharge was vectored across the chest, electrical and mechanical capture of the heart ensued. The cardiac stimulation at high rates persisted during the discharge, and as soon as the discharge ceased there was resumption of normal electrical rhythm. The stimulation was dependent on the model of the NID device used. We also found that discharges away from the chest did not stimulate the heart or trigger arrhythmias. During simulated stress with epinephrine infusion, presumably because of the shortening of ventricular refraction, some discharges resulted in VF and VT. The mechanism of VF induction may indeed have been caused by a discharge during the T wave (stimulation during the vulnerable period [12]). In the enlargement of the time of onset of tachycardia in Figure 4, it is very suggestive that the penultimate NMI discharge was delivered at the vulnerable period of the T-wave, resulting in nonsustained VT; however, we are unable to make definitive statements regarding this because in Figure 3 the intracardiac signals were partially corrupted by the EMI.

These findings suggest that there exists the possibility of serious ventricular arrhythmia during NID discharges in structurally normal hearts during intense catecholamine stress. In patients with structural heart disease, in which electrophysiological inhomogeneities are present, rapid ventricular stimulation is known to produce catastrophic ventricular arrhythmias (13). Our findings of rapid ventricular stimulation with NID discharge across the chest suggest a particular risk in individuals with pre-existing inhomogeneities caused by structural heart disease.

The NIDs have been deployed in an estimated 5,000 law enforcement agencies in North America among some 130,000 officers (14). Contemporary discussions around the safety of NID devices focus on the often-observed delay between NID discharge and in-custody death and the presence of other attributable factors, including cocaine or phencyclidine intoxication. States of excited delirium and associated metabolic derangement are often present in subjects subdued with NMI discharges, and may also increase the risk of ventricular arrhythmias (15–17). Although it may be difficult to determine the cause of death in such situations (in the absence of continuous cardiac monitoring at the time of discharge), our data suggest that the safety of NIDs and their arrhythmogenic potential during discharge needs to be assessed in humans and with intracardiac monitoring shielded from EMI.

The greater voltages during discharge across the chest seen in our model support the hypothesis that it is indeed the maximum voltage vector across the heart that results in stimulation of the myocardium. The 2 NID devices tested are quite different. We have seen higher intracardiac voltage generated by the NID-1 than by the NID-2; 195 mV versus 77 mV while discharged across the chest. Our calibration

data suggest that the absolute voltages are higher than those recorded by the mapping system. It is unknown whether darts positioned across the chest in humans would produce a discharge vector that results in the same voltages seen in a 50-kg pig. However, our findings may have relevance to smaller individuals who have died after NID discharges (18). The large, short initial impulse produced by the NID-1 is thought to be less injurious for internal organs because a high-frequency pulse, while traveling into conducting material, is thought to travel preferentially in the periphery of the conductor and thus in the skin and muscle on the torso. Our data suggest that this effect is negligible at these frequencies, and indeed that a substantial amount of voltage is seen across the heart when the discharge is vectored across the chest.

The NID-2 delivers greater power compared with the NID-1. However, the pulse duration of the NID-1 is longer, thus theoretically allowing a longer time for the membrane to be charged, producing cellular depolarization. The time constant for membrane depolarization is on the order of 2 to 5  $\mu$ s (19); the strength duration curve for cardiac stimulation suggests that for very large local voltage gradients effective stimulation may occur, especially if stimulation is repetitive, allowing charge to build up on the cardiac cell membranes. This may be another potential explanation for greater stimulation with the NID-1.

Studies that assessed safety before and after NID discharge with surface electrocardiographic monitoring (8) may have not been able to record cardiac stimulation because of the EMI during discharge. For example, in our study, Figures 2A and 2C do not show the intense stimulation that is seen in segment Figure 2B, in which the surface lead 1 is corrupted because of the EMI. In this situation, we would have arrived at the same conclusion as previous investigators (8) if not for the intracardiac catheters and the shielding. Our study findings are concordant with the early testing of NID discharges; on direct contact with the pericardium without the barb system, Roy *et al.* (9) showed that the pig heart would fibrillate. In our model we ensured that there was no direct stimulation of the heart through the pericardium, yet were able to observe stimulation of the myocardium. Our data are discordant with work on cardiac safety performed by simulated NID discharge testing (10); however, it is difficult to compare a study that tested a simulated device that had a similar waveform morphology but a different set of charge specifications with our study, which tested a commercial model NID.

A study comparing mortality among subjects subdued by police with handguns compared with those subdued with an early NID with a barb and lead system reported 3 deaths among 218 subjects (1.4%) subdued with the NID device (20). An independent safety committee reviewing filed reports of dart locations found that of the darts fired facing a victim, most commonly landed in the upper sternal position, with the second dart landing in the epigastric location (11). Keeping in mind the limitations of a reporting

system, the configuration we adopted was probably the third most common when fired from directly in front of the victim. We designed this study as described in the Methods section to portray the worst-case scenario: one of the explanations for not seeing greater incidence of harm in real life may be because the darts commonly do not attach in the worst-case scenario vectoring the discharge across the heart.

It is possible that NID deployment in the field rarely results in a barb position that results in discharge vector that is oriented across the heart, and also that the human chest anatomy results in a different current distribution than in our experimental model. Thus our model describes the worst-case scenario when the discharge is vectored across the heart and is applicable only to such situations. Our model is valuable in ruling out arrhythmias as the cause of death when the vector of discharge was not across the heart and was long after the discharges.

The general anesthesia used may have increased the threshold for arrhythmia induction in this model. The fact that these pigs had structurally normal hearts may have decreased the amount of ventricular arrhythmia that could have been sustained. The episode of sustained VF and of nonsustained VT observed during the epinephrine infusion suggests that shortened refractoriness may have had a part to play. The additional discharges that were delivered at the end of the study after the intracardiac catheters had been removed reproduced the catastrophic hemodynamic consequences, ruling out the possibility that these findings were caused by intracardiac catheters channeling the electricity and provoking the arrhythmias.

**Study limitations.** Using epinephrine to simulate stress may not be physiological and may not reconstruct exactly the states of excited delirium, a situation in which adverse outcomes have been reported. Other methods, such as direct sympathetic nerve stimulation (such as via stellate ganglion stimulation), with its nonuniform distribution in the myocardium, may simulate stress better, but would still be invasive and nonphysiological. Our observations on triggered arrhythmias are few, and statements made regarding the mechanisms of them are essentially hypotheses. The threshold for induction of VF in pigs may be lower than in humans, and the structural variation in the chest wall anatomy is another limitation with regard to extrapolating our model to humans. The possibility of an independent interaction between the isoflurane and the epinephrine is unlikely because we did not observe spontaneous VF with either the epinephrine infusion or catheter manipulation.

**Conclusions.** In an experimental model, NID discharges across the chest can produce cardiac stimulation at high rates. This study suggests that NIDs may have cardiac risks that require further investigation in humans.

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#### REFERENCES

1. Fish RM, Geddes LA. Effects of stun guns and TASERS. *Lancet* 2001;358:687–8.
2. Murray J, Resnick B. *A Guide to TASER Technology*. Whitewater, CO: Whitewater Press, 1997.
3. Kim PJ, James WH. Ventricular fibrillation after stun-gun discharge. *N Engl J Med* 2005;353:958–9.
4. Kornblum RN, Reddy SK. Effects of the TASER in fatalities involving police confrontation. *J Forensic Sci* 1991;36:434–8.
5. Koscove EM. The TASER weapon: a new emergency medicine problem. *Ann Emerg Med* 1985;14:1205–8.
6. O'Brien DJ. Electronic weaponry—a question of safety. *Ann Emerg Med* 1991;583:163–7.
7. Wilkinson D. Scientific Development Branch Further Evaluation of TASER Devices. St. Albans, UK: Home Office Police Scientific Development Branch, 2005.
8. Levine SD, Sloane C, Chan T, Vilke G, Dunford J. Cardiac monitoring of subjects exposed to the TASER. *Acad Emerg Med* 2005;12:S71.
9. Roy OZ, Podgorski AS. Tests on a shocking device—the stun gun. *Med Biol Eng Comput* 1989;27:445–8.
10. McDaniel WC, Strathbucker RA, Nerheim M, Brewer J. Cardiac safety of neuromuscular incapacitating defensive devices. *Pacing Clin Electrophysiol* 2005;28:S284–7.
11. The Joint Non-Lethal Weapons Human Effects Centre of Excellence. Human effectiveness and risk characterization of the electromuscular incapacitating device: a limited analysis of TASER: part 11. Available at: <http://www.opcc.bc.ca/Reports/2004/Interim%20Taser%20Report%20and%20Recommendations.pdf>. Accessed January 11, 2006.
12. Link MS, Wang PJ, Pandian NG, et al. An experimental model of sudden death due to low-energy chest-wall impact (commotio cordis). *N Engl J Med* 1998;338:1805–11.
13. Buxton AE, Marchlinski FE, Flores BT, Miller JM, Doherty JU, Josephson ME. Nonsustained ventricular tachycardia in patients with coronary artery disease: role of electrophysiologic study. *Circulation* 1987;75:1178–85.
14. Amnesty International. USA/Canada: pattern of abuse—suspend use of Taser guns. Available at: <http://web.amnesty.org/library/index/ENGAMR511682004>. Accessed September 20, 2005.
15. British Columbia Office of the Police Complaints Commissioner. Taser technology review and interim recommendations. Victoria, Canada: 2004. Available at: <http://www.opcc.bc.ca/Reports/2004/Interim%20Taser%20Report%20and%20Recommendations.pdf>. Accessed September 20, 2005.
16. Rutenber AJ, Lawler-Heavner J, Yin M, Wetli CV, Hearn WL, Mash DC. Fatal excited delirium following cocaine use: epidemiologic findings provide new evidence for mechanisms of cocaine toxicity. *J Forensic Sci* 1997;42:25–31.
17. Wang RY. pH-dependent cocaine-induced cardiotoxicity. *Am J Emerg Med* 1999;17:364–9.
18. Turner MS, Jumbelic MK. Stun gun injuries in the abuse and death of a seven-month-old infant. *J Forensic Sci* 2003;48:180–2.
19. Mouchawar G, Kroll M, Val-Mejas JE, et al. ICD waveform optimization: a randomized, prospective, pair-sampled multicenter study. *Pacing Clin Electrophysiol* 2000;23:1992–5.
20. Ordog GJ, Wasserberger J, Schlarter T, Balasubramanian S. Electronic gun (Taser) injuries. *Ann Emerg Med* 1987;16:73–8.