

Effects of Cocaine Intoxication on the Threshold for Stun Gun Induction of Ventricular Fibrillation

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OBJECTIVES	This study sought to assess cocaine's effects on Taser-induced ventricular fibrillation (VF) threshold in a pig model.
BACKGROUND	Stun guns are increasingly used by law enforcement officials to restrain violent subjects, who are frequently intoxicated with cocaine and other drugs of abuse. The interaction of cocaine and the stun gun on VF induction is unknown.
METHODS	We tested five adult pigs using a custom device built to deliver multiples of standard neuromuscular incapacitating (NMI) discharge that matched the waveform of a commercially available electrical stun gun (Taser X-26, Taser International, Scottsdale, Arizona). The NMI discharges were applied in a step-up and step-down fashion at 5 body locations. End points included determination of maximum safe multiple, minimum VF-inducing multiple, and ventricular fibrillation threshold (VFT) before and after cocaine infusion.
RESULTS	Standard NMI discharges ($\times 1$) did not cause VF at any of the 5 locations before or after cocaine infusion. The maximum safe multiple, minimum VF-inducing multiple, and VFT of NMI application increased with increasing electrode distance from the heart. There was a 1.5- to 2-fold increase in these values at each position after cocaine infusion, suggesting decreased cardiac vulnerability for VF. Cocaine increased the required strength of NMI discharge that caused 2:1 or 3:1 ventricular capture ratios at all of the positions. No significant changes in creatine kinase-MB and troponin-I were seen.
CONCLUSIONS	Cocaine increased the VFT of NMI discharges at all dart locations tested and reduced cardiac vulnerability to VF. The application of cocaine increased the safety margin by 50% to 100% above the baseline safety margin. (J Am Coll Cardiol 2006;48:805-11) © 2006 by the American College of Cardiology Foundation

The neuromuscular incapacitating (NMI) effects of the electrical stun gun have made it a device that is increasingly used among law enforcement authorities (1). Recent reports have suggested a potential connection between some in-custody deaths and electrical stun gun (Taser [a trademarked name that was originally an acronym for Timothy A. Swift electrical rifle]) application (2). Kornblum et al. (3) reported the presence of at least 1 of 3 illegal drugs (phencyclidine, cocaine, amphetamines) in 13 of 16 cases in which a Taser was used. Violent subjects who pose a threat to law enforcement officers are often intoxicated from illicit drugs such as cocaine, phencyclidine, and amphetamines (4). Cocaine has a variety of cardiac effects, including potential proarrhythmic effects (5-13).

The ventricular fibrillation (VF) threshold of Taser shocks has been reported to be relatively high and directly proportional to body mass (14). The interplay of cocaine and NMI current on the induction of arrhythmias is not known. This article examines cocaine's effects on VF induc-

ibility by NMI current when applied to various body locations in a pig model.

METHODS

Electrical stun device. The Taser X-26 shoots two tethered darts and delivers 19 pulses/s at up to 1,200 V over 5 s. The pulse characteristics are shown in Figure 1. A custom device was built to deliver NMI electrical discharges that matched the waveform characteristics of the Taser X-26 (Taser International, Scottsdale, Arizona). The experimental device allowed output capacitance to vary as a multiple of standard capacitance for a regular NMI device.

Laboratory animals. The research protocol was approved by the Animal Research Committee of the Cleveland Clinic Foundation and conducted in accordance with the regulations of the Association for Assessment and Accreditation of Laboratory Animal Care. Five adult pigs (4 male, 1 female) with a length of 103 ± 8 cm, a weight of 34 ± 8.7 kg, and a chest circumference of 65.8 ± 4 cm were studied. Pigs were chosen because they have a heart-body weight ratio and cardiac anatomy similar to that of humans, with relatively low VF thresholds (15). Previous studies on electrical stun gun effects also used pigs (14). The animals were sedated with intramuscular ketamine (12 mg/kg) and intubated. Anesthesia was maintained with 1% to 2% isoflurane mixed with oxygen and nitrous oxide. At the end

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Abbreviations and Acronyms

CK	= creatine kinase
ECG	= electrocardiogram
maxSM	= maximum safe multiple
minVFIM	= minimum ventricular fibrillation-inducing multiple
NMI	= neuromuscular incapacitating
PMI	= point of maximum impulse
SN	= sternal notch
VF	= ventricular fibrillation
VFT	= ventricular fibrillation threshold

of the experiment, a mid-sternotomy was performed, VF was induced with 9 V direct current, and the heart was harvested and processed for histologic examination.

Position of the darts. Human field experience has shown that the posterior and anterior upper trunk regions were the most common dart attachment sites (2, personal communication, Taser International, Scottsdale, Arizona). Figure 2 identifies the five different paired-dart positions tested on

the pig body, labeled Positions A through E. Because we hypothesized that current application nearest the heart and along its axis would be the most arrhythmogenic, we tested Position A at the beginnings of the two series. The point of maximum impulse (PMI), typically located slightly left of the xyphoid process, was palpated and confirmed with auscultation and echocardiography. The sequence of testing the remaining four sites was randomized. Two darts were inserted to full depth at the mentioned sites. The mean distance of the PMI dart tip from the epicardial surface measured by echocardiography was 18 ± 4 mm.

Cocaine infusion. After baseline testing, high-dose cocaine was infused intravenously at 8 mg/kg over 30 min (8,9). Plasma cocaine and benzoylecgonine levels 30 min after infusion were 557 ± 280 U/l and 462 ± 123 U/l, respectively.

Determination of VF threshold and ventricular capture. Standard NMI discharge is a 5-s application equivalent to a single Taser X-26 application. Testing was started with a standard discharge ($\times 1$) followed by discharges of increas-

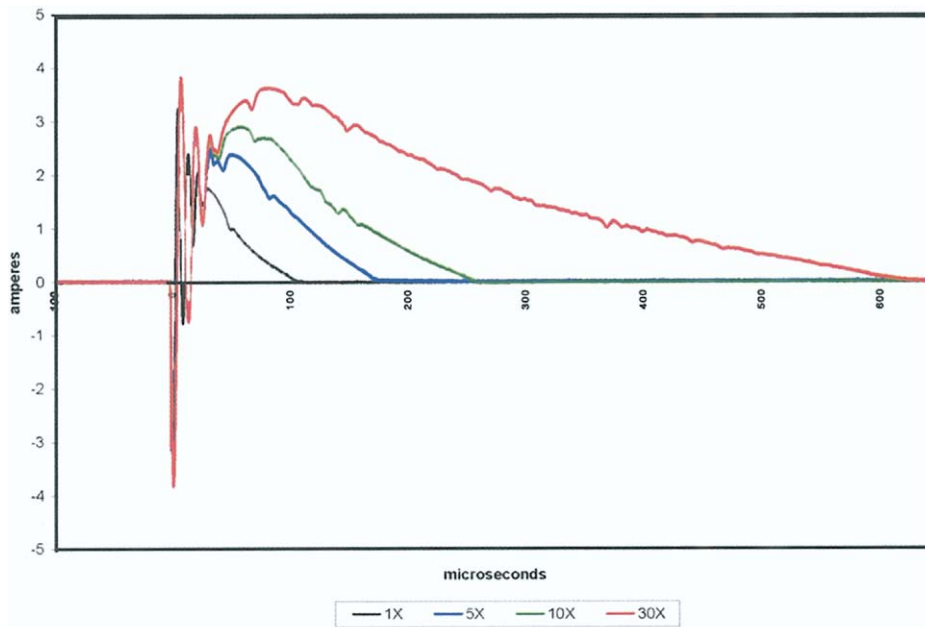


Figure 1. The prototype Taser X-26 stun gun and the darts. The electrical current waveform characteristics at $\times 1$, $\times 5$, $\times 10$, and $\times 30$ of the standard discharge from the Taser X-26. The waveform of the standard pulse has a duration of about 100 μ s and a net delivered charge of about 100 μ C. Variations of the current waveform with increased output are shown. Because of the output stage transformer effects in front of the capacitors, there is an increase in both pulse duration and peak current. The gun and darts (9 mm in length) with insulated wires that carry the charge from the gun to the darts are shown in the lower panel.

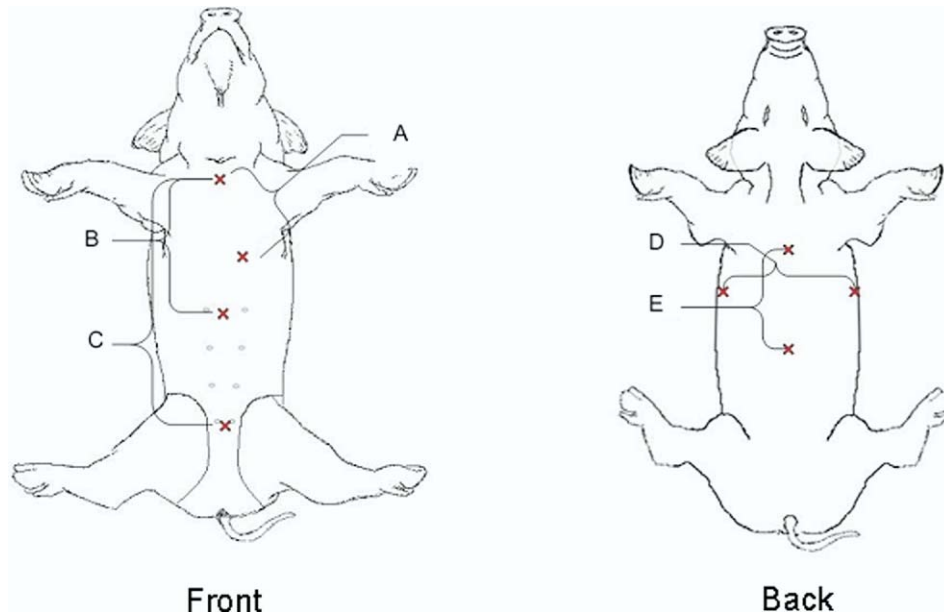


Figure 2. Dart positions on the front and back of the pig. Position A = sternal notch (SN) to point of maximum cardiac impulse (PMI); Position B = SN to supraumbilical region; Position C = SN to infraumbilical region; Position D = side-to-side across the chest; Position E = upper to lower midline posterior torso.

ing stored charge in a step-up fashion until VF was induced. The stored charge was increased for each step by multiples of the standard capacitor ($\times 5$, $\times 10$, and multiples of $\times 10$ up to $\times 100$). After the first VF induction, the capacitances were decreased in reversed sequence with the addition of $\times 7$ and $\times 2$ when needed until 3 sequential discharges of equal stored charge did not induce VF. Surface electrocardiogram (ECG) leads II and V_1 were recorded. A right ventricular intracardiac bipolar electrogram (EP Technologies, Boston Scientific Ltd., Sunnyvale, California) was monitored to assess ventricular capture during NMI application. When ventricular capture occurred, it usually had a fixed ratio to the delivered pulses. Thus, the frequency of ventricular capture was quantified as the ratio of NMI pulses to each ventricular capture beat. Pigs were defibrillated on VF induction with a 300-J direct current biphasic shock. The animals were rested 3 min between each discharge without VF and 15 min with VF induction.

Blood samples. Arterial and venous blood samples were obtained before starting NMI application, after the first NMI application at Position A (to assess effects of standard NMI applications), before and 30 min after the infusion of cocaine, and at the end of the entire experiment.

Definitions of variables. Minimum VF-inducing multiple (minVFIM) was defined as the lowest NMI discharge multiple that induced VF at least once in 3 tries. Maximum safe multiple (maxSM) was defined as the highest discharge multiple that could be applied 3 times without VF induction. Ventricular fibrillation threshold (VFT) was defined as the average of these 2 values. The NMI discharge multiples at which 2:1 and 3:1 ventricular captures were seen are reported here. These 2 ratios were chosen because 3:1

capture was the highest capture frequency that did not induce VF, whereas 2:1 capture always induced VF.

Data analysis. All continuous variables were summarized by their means and standard deviations. The effect of cocaine on maxSM, minVFIM, and VFT was tested using the paired *t* test. A general linear model for repeated measures with a difference contrast was used to compare the Taser and cocaine effects on hemodynamic and metabolic data, cardiac markers, and ECG data. The Bonferroni adjustment was used to correct for between- and within-subjects factors. A level of $p < 0.05$ was considered statistically significant.

RESULTS

Induction of VF. Table 1 shows the maxSM, minVFIM, and VFT data. The lowest mean maxSM, minVFIM, and VFT were seen at Position A, whereas the highest were seen at Position E. These variables increased 1.5- to 2-fold after cocaine infusion at all positions. The increases were statistically significant in 4 of the 5 positions.

Differences in ventricular capture ratios. Ventricular capture increased with progressive increase in current application strength and ranged from no capture to $\leq 2:1$. The VF was consistently inducible whenever the ventricular capture ratio was $\leq 2:1$. No VF induction was noted when the ventricular capture was $\geq 3:1$. A greater degree of ventricular capture at lower strengths was seen at Position A than at other locations. This correlated with our finding that VF was induced with the lowest minVFIM at this location. Application strength multiples of $\times 40$ and higher were needed on the back (Position E) to accomplish similar ventricular capture ratios. Standard NMI discharge at Po-

Table 1. Thresholds for Ventricular Fibrillation Induction at Different Positions Before and After Cocaine Infusion

Location	B-maxSM	C-maxSM	p Value	B-minVFIM	C-minVFIM	p Value	B-VFT	C-VFT	p Value
Position A	4.2 ± 1.10	8.6 ± 6.88	0.192	8.0 ± 2.74	15.0 ± 10.00	0.135	6.1 ± 1.92	11.3 ± 8.79	0.260
Position B	12.0 ± 7.58	28.0 ± 4.47	0.030	20.0 ± 10.0	38.0 ± 4.47	0.037	14.5 ± 9.59	33.0 ± 4.47	0.032
Position C	22.0 ± 8.37	50.0 ± 18.71	0.009	32.0 ± 8.37	60.0 ± 18.71	0.009	27.0 ± 8.37	55.0 ± 18.71	0.009
Position D	30.0 ± 7.07	48.0 ± 17.89	0.070	40.0 ± 7.07	58.0 ± 17.89	0.070	35.0 ± 7.07	53.0 ± 17.89	0.070
Position E	38.0 ± 4.47	60.0 ± 14.14	0.011	48.0 ± 4.47	70.0 ± 14.14	0.011	43.0 ± 4.47	65.0 ± 14.14	0.011

Bold values are statistically significant.

B = before cocaine infusion; C = after cocaine infusion; maxSM = maximum safe multiple; minVFIM = minimum ventricular fibrillation induction multiple; VFT = ventricular fibrillation threshold.

sition A did not induce VF in any animal despite ventricular capture ratios ranging from 6:1 through 3:1, nor was VF induced with standard ×1 NMI application at any of the other four locations with or without cocaine. The NMI output multiples at which 2:1 and 3:1 ventricular capture ratios were seen for the various dart positions are shown in Figure 3. Cocaine increased the required strength of NMI discharge that caused a 2:1 or 3:1 ventricular capture ratio at all positions.

Other observations. There was no significant change in the ECG variables before or after cocaine infusion. No

ST-segment or T-wave changes suggestive of myocardial ischemia were seen. There were no significant changes in blood pressure, electrolytes, arterial pH, or blood gases throughout the experiment.

Serum creatine kinase (CK) levels increased from 553 ± 72 U/l at baseline to 2,699 ± 828 U/l after the baseline precocaine VF induction testing (p = 0.04). This increase reflects cumulative delivery of more than 1,000 times the standard NMI discharge and is most likely related to multiple intense muscular contractions. There was a milder increase in CK levels after cocaine infusion before further NMI application (3,058 ± 984 U/l vs. 2,699 ± 828 U/l, p = 0.21). These CK values reached a level of 13,273 ± 6,163 U/l (p = 0.02) at the end of the experiment, when a cumulative NMI application of more than 2,000 times the standard strength had been delivered. No significant changes were seen in CK-MB throughout the experiment.

Pathologic and histopathologic findings. The mean weight of these hearts was 215 ± 25 g. There was no gross evidence of myocardial necrosis or damage. Detailed histologic analysis showed no structural changes in 1 animal and some myocardium, conduction system, or endocardium changes in 4 animals (Table 2). The findings seen in these animals were limited to the ventricular subendocardial region, localized to focal areas not exceeding 5% of the total myocardium.

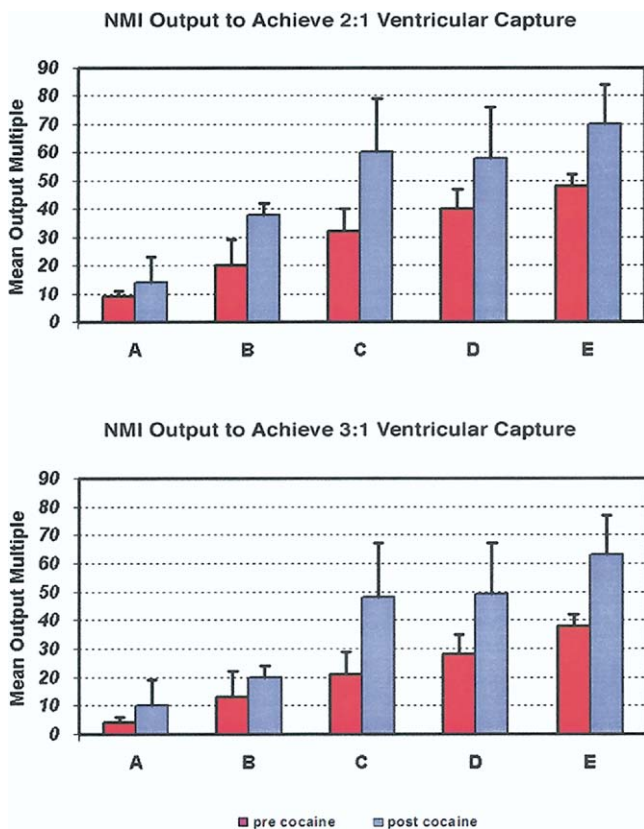


Figure 3. Differences in ventricular captures rates before and after cocaine infusion at the 5 tested positions using varying multiples of standard current strength. These 2 graphs show the mean neuromuscular incapacitating (NMI) output multiples needed to achieve 2:1 (top) and 3:1 (bottom) ventricular capture at Positions A through E. **Error bars** indicate 1 standard deviation. Higher output multiples were needed after cocaine infusion (blue bars) to achieve the same degree of ventricular capture as during baseline stimulation (red bars).

DISCUSSION

Several types of cardiac rhythm disturbances may occur with cocaine use in humans (5). A number of investigators have reported on the arrhythmogenic effects of cocaine in animals (10–12). Despite the widely-held belief in an arrhythmogenic effect of cocaine, spontaneous or inducible arrhythmias were produced in none or few conscious or anesthetized animals with normal intact hearts. In the majority of cases, the reported arrhythmias associated with cocaine have occurred in the setting of significant hemodynamic or metabolic disturbances, such as hypotension, hypoxemia, seizures, or myocardial ischemia/infarction (5,16). In experimental models, cocaine was found to provoke arrhythmias only in the presence of induced myocardial ischemia or infarction, sometimes during concomitant infusions of epinephrine or norepinephrine (13,17).

Table 2. Histopathological Findings in the Pig Hearts

Animal	Myocardium	Conduction System	Endocardium	Epicardial Arteries
Pig 1	None	None	None	None
Pig 2	Minimal focal myocyte damage (LV)	None	Acute subendocardial mural thrombus (LV)	None
Pig 3	Minimal focal myocyte damage (LV) and focal subepicardial interstitial hemorrhage	None	Subendocardial thrombus	None
Pig 4	Focal subendocardial ischemic damage (LV)	None	Subendocardial mural thrombus (RV)	None
Pig 5	Focal subendocardial ischemic damage (LV)	None	None	None

LV = left ventricle; RV = right ventricle.

How NMI discharge would interact with the effects of cocaine on the inducibility of cardiac arrhythmias has not been reported. Given the increased incidence of cocaine intoxication in subjects being restrained with Taser weapons by law enforcement officers, it is important to assess the effect of cocaine on ventricular arrhythmia induction by NMI devices. Our study showed that VF could not be induced using the standard 5-s Taser discharge applied to a pig's body surface even at the most sensitive area tested. Clearly, increasing the delivered charge beyond the standard NMI application can induce VF. The sensitivity to VF induction at increasing strengths of application varied depending on the dart locations over the body. As might be expected, the most sensitive dart location seemed to be the PMI-to-sternal notch (SN) axis (Position A). This is most likely because of the proximity of one electrode to the heart (PMI) and bracketing of the heart by the other electrode (SN). The tip of the electrode at the PMI position was only 1 to 2 cm from the myocardium. With increasing distance of these electrodes from the heart, VF thresholds increased precipitously. Ventricular fibrillation could be induced only at very high multiples of NMI discharge when applied to the back of the trunk. Thus, it is likely that induction of VF is directly related to current density at the myocardium and pulse width of the delivered impulses.

Our study is the first to describe capture of ventricular myocardium during application of NMI pulses. Although it is difficult to appreciate myocardial capture on the surface ECG, the recording from the right ventricular bipolar electrogram can show myocardial capture. When rapid capture of the myocardium occurred, the blood pressure recordings also showed a precipitous decrease. An example of such observations is shown in Figure 4. Our observations indicate that rapidity of myocardial capture may play a significant role in the induction of VF. In our experiments, occurrences of 2:1 or more frequent capture were associated with induction of VF. Such capture was not seen even at Position A during delivery of the standard discharge. This observation probably explains the lack of VF induction with the standard discharge at all positions.

Our data suggest that the presence of cocaine decreases the likelihood of NMI-induced VF. Cocaine increases the safety margin approximately 1.5 to 2 times from baseline. Our study also showed less myocardial capture after cocaine

infusion. This observation is consistent with our hypothesis that rapid capture is the mechanism of VF induction. The sodium channel blocking effects of cocaine along with its ability to create a hypersympathetic state have been postulated as potential mechanisms behind its arrhythmogenicity (5,18,19). However, it is not clear whether these properties in the absence of an appropriate substrate would increase vulnerability to VF.

The very limited histopathologic findings reported here suggest that damage to the heart by the applied NMI current is minimal even after cumulative doses of over 2,000 times standard NMI discharges. Cardiac pathologies associated with high-voltage and high-current exposures have been well documented in multiple prior reports (20–24). Focal changes affecting the myocardium are described as extensively dispersed throughout the ventricles and atria. These injuries are seen with much higher current than that delivered by NMI devices. Thus, it is not surprising that we did not observe such injury to the heart in our histopathological analysis. Toxic effects of cocaine on the myocardium can result in scattered foci of necrosis, contraction band necrosis, myocarditis, and foci of myocardial fibrosis (8,25,26). It is impossible to separate the cocaine effect from that of NMI application when assessing the minor histologic findings reported here. Given the large number of NMI applications made during these experiments, it would be difficult to extrapolate any of our histologic findings to a clinically relevant scenario.

Extending animal data to human beings should always be done with caution. However, pigs frequently have been used in fibrillation and defibrillation threshold studies with the results generalized to humans. The results of our study and the few prior animal studies (14) would suggest that NMI discharge at the standard 5-s application is unlikely to cause life-threatening arrhythmias, at least in the normal heart. Our data regarding myocardial capture, however, suggest the potential for induction of ventricular tachycardia in subjects with substrate for ventricular tachycardia, especially if one of the electrodes were to come within a few centimeters of the myocardium, with the other positioned to direct the current toward the heart. In humans, the anterior apical right ventricular myocardium is closest to the chest wall. Positioning of an electrode in a small, thin human in the region of the left nipple with the other electrode near

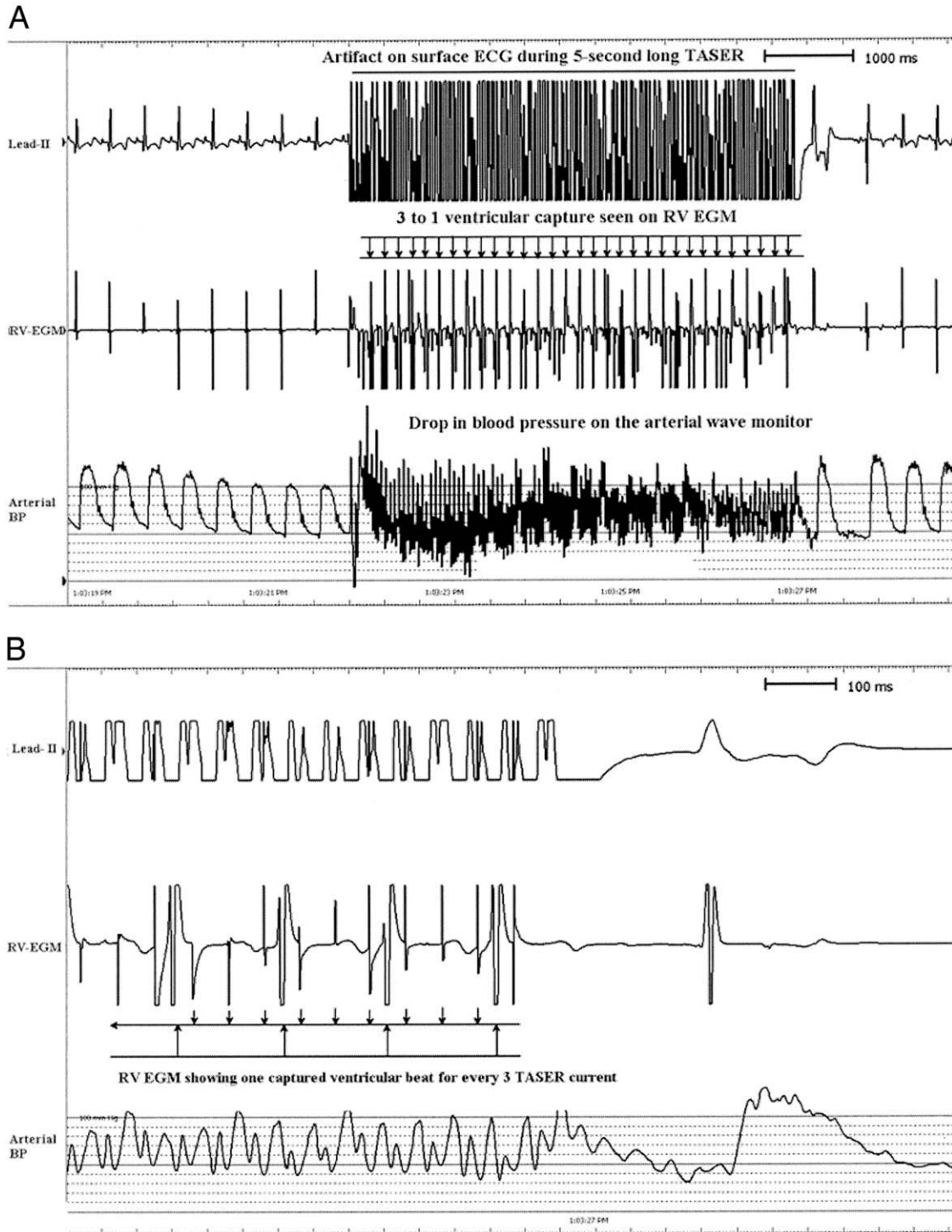


Figure 4. Example of 3:1 ventricular capture during neuromuscular incapacitating (NMI) application. The tracings from top to bottom in each panel are surface electrocardiogram (ECG) lead II, intracardiac right ventricular bipolar electrogram, and blood pressure recording. (A) The entire 5-s burst of NMI delivered at $\times 5$ output, Position A. The stimulus artifact overwhelms the surface ECG recordings. The time scale (1,000 ms, upper right of panel) does not allow appreciation of ventricular capture on this panel. There is also a stimulus artifact on the blood pressure tracing, but an overall decrease in blood pressure can be appreciated through the artifacts. (B) The end of NMI application at an expanded time scale (100 ms). Ventricular activation on the right ventricular bipolar recording at a 3:1 ratio to the stimulus artifacts can be readily appreciated. Lower arrows point to the stimulus artifact, and upper arrows point to the right ventricular bipolar electrogram. After termination of the NMI application, normal rhythm resumes with a normal blood pressure pulse.

the sternal notch may simulate our Position A and could potentially achieve comparable proximities of electrodes to the heart. Avoidance of this position would greatly reduce

any concern for induction of ventricular arrhythmias. Our data also indicate that cocaine decreases the vulnerability to ventricular arrhythmias with electrical stun gun use.

Study limitations. Our study was performed in anesthetized pigs for obvious ethical reasons. It is conceivable that application of a Taser in a nonanesthetized state, similar to a real-life situation, could activate a higher sympathetic tone, which may have a different effect. However, the extent of such an increase in sympathetic tone is hard to estimate because human studies have shown a minimal increase of heart rate (mean 20 beats/min) caused by the NMI application (27). The pigs used in this study also had no particular cardiac abnormality. It is possible that structural heart disease may affect the inducibility of arrhythmias by NMI devices. Such inducibility may interact differently with the presence of cocaine. Lastly, although the findings were quite consistent from animal to animal, the total number of animals used in this study was small.

Conclusions. Cocaine increased the VFT of NMI applications in all dart locations tested and actually reduced vulnerability to VF. Induction of VF at higher output than standard NMI applications seems to be related to myocardial capture.

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