

**Submission to:**  
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**The use of Neuromuscular Incapacitating Devices (Taser)  
and the Risk of Sudden Death.**

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By way of introduction I am a cardiac electrophysiologist. I am fully trained as a cardiologist and have spent 2 extra years of training in the management of people with abnormalities of the electrical system of the heart, which lead to various rhythm disturbances or arrhythmias. I did my training in electrophysiology at Duke University, Durham, North Carolina and returned to practice cardiology and electrophysiology in 1981. I initiated the field of invasive electrophysiology in Western Canada and have worked in that field since that time. I am currently Head of the Arrhythmia Management Program, St. Paul's Hospital and the University of British Columbia.

### **Basic Electrical Function of the Heart**

Every time the heart beats, it is a result of an electrical impulse spreading through the heart and activating the heart muscle cells to contract. Electrical signals are stimulated by the naturally occurring pacemakers in the human heart. Abnormal rhythms can arise due to abnormal circuits where electricity moves around in circular motion or in circumstances where extra pacemakers in the heart fire off at excessive rates. Of most concern in the issue of Taser use would be the electrical function of the ventricles, or the main pumping chambers of the heart. As electricity flows through the heart the muscle cells are activated by a wave of electrical current as it flows through the electrical conduction system of the heart. This leads to a contraction of the heart.

Immediately following this activation there is a short period of time during which the cells cannot be activated again; that is, they are “refractory” to further activation. The activation of the muscle of the heart produces a characteristic electrical signal on an electrocardiogram (ECG) called the QRS complex that can be easily identified. Following this period where the muscle is refractory, there is gradual recovery of muscle cells that occurs at slightly different times in various areas of the heart. This inscribes another electrical signal on the ECG that is labeled the T wave. Following the T wave the heart cells are then able, once again, to be activated by an electrical signal.

Ventricular fibrillation is an extremely rapid rhythm in the lower chambers of the heart, where electrical signals are conducted in circles around scars in the muscle or around circles of normal cells. This leads to ineffective contractions of the heart, where the heart simply quivers. In this state, the heart cannot pump blood and, unless it is interrupted quickly, sudden cardiac death will follow. Ventricular tachycardia is a more organized, but slower, arrhythmia that can also lead to death, but may be tolerated to some degree for longer periods.

There is a vulnerable period during the cardiac cycle, right on the peak of the T wave, when an extra electrical signal (“R on T”) can result in the initiation of dangerous heart rhythms, most notably ventricular tachycardia and ventricular fibrillation. The vulnerable part of the cardiac cycle is felt to be about 3 – 5 % of this overall cardiac cycle. For example, if a patient had a heart rate of 60 beats per minute, the cardiac cycle would be one second or 1,000 milliseconds in duration. Therefore, there is a vulnerable period of approximately 30 – 50 milliseconds, where an extra signal may result in the initiation of ventricular tachycardia or ventricular fibrillation.

This “R on T” phenomenon can occur in a number of settings. It can occur with coincidental trauma during the vulnerable period. This is felt to be the mode of sudden cardiac death that has been reported in young athletes, particularly if they receive a blow with an object such as a hockey puck, stick, or ball in the mid chest area. The trauma right over the heart will result in a physical stimulation of the heart which, in turn, can result in an electrical stimulus. This is called “Commotio Cordis” and, unfortunately, is reported intermittently in the press when such an unfortunate event occurs. This has been one of the impetuses to have automatic external defibrillators placed in sporting arenas.

A second circumstance where the R on T phenomena can occur is what we do almost every day in the practice of cardiac electrophysiology. We can introduce electrical signals on the T wave to intentionally induce arrhythmias. This can be done in a laboratory setting to see if a patient is prone to the development of malignant arrhythmias in order to be pro-active in preventing that arrhythmia in that patient. We know that if we place an electrical wire into the heart through the veins, we can pace the heart. If we then put extra beats in at the appropriate time this can result in the initiation of malignant arrhythmias, particularly in individuals who have disease of their ventricles such as a previous heart attack or a primary heart muscle disease (cardiomyopathy).

We also induce ventricular fibrillation as part of an implantation of an ICD (implantable cardioverter defibrillator). This is a device that is implanted permanently in a patient. It acts as a pacemaker to support slow heart rhythms but also shocks a patient’s heart automatically back to normal should they develop what would be a fatal arrhythmia. We have put in approximately

650 ICDs in British Columbia last year, 450 through the Heart Programs in Vancouver. During implantation we test these devices to make sure they are capable of defibrillating the heart. We do this by an appropriately timed shock of very low energy delivered inside the heart muscle through the lead that is permanently implanted in the right ventricle. These extra shocks are applied during the vulnerable period and very easily induce ventricular fibrillation with energy of one Joule or less. The defibrillator then senses the very rapid abnormal rhythm in the ventricle and shocks the heart through the same electrical wires with a much higher energy and returns the patient's rhythm to normal. We know that fibrillation can be easily induced with these techniques, particularly in patients with other structural heart disease.

We also know that hearts that are under stimulation of the sympathetic nervous system are also more prone to developing abnormal heart rhythms. Again, in a model in the electrophysiology laboratory, where we induce arrhythmias on a regular basis, we often use an adrenaline-like drug to simulate stressful situations. Many people will only develop their abnormal rhythms when they are under a situation of considerable stress such as intense physical activity, pain, or emotional upset. Discharge of the central nervous system and an increase of adrenaline-like products circulating through the body accelerate the heart rate and alter the electrical characteristics of heart cells, making them more prone to the development of heart arrhythmias.

### **Relationship of Taser Use to the Induction of Arrhythmias**

First, it must be clear that I am not an expert on Tasers. From what I have read, I understand that they shoot 2 barbs that can penetrate into the body of a targeted individual. The barbs are

approximately 9 mm long and .5 mm in diameter. The voltage between the 2 electrodes is 50,000 volts. However, because of the very high resistance to the small barbs and the human tissue, the current delivered is relatively low (36 milliamps as reported in a paper by Levine in Journal of Emergency Medicine). The pulse durations are very short, 100 to 150 micro-seconds, making the total current and energy delivered relatively small. The energies delivered by 2 Taser models are reported to be 1.76 Joules for Model M26 and 0.36 Joules for Model X26. The devices deliver 15 to 19 such pulses per second for a total duration of 5 seconds, for a total of 75 to 95 pulses.

Theoretically, the very short pulse durations will make it less likely to deliver sufficient current to result in electrical capture of the heart and potential induction of ventricular fibrillation.

### **Animal Studies Using Taser Application**

There have been several animal and theoretical studies performed. Several have suggested that the energies delivered by a Taser would not be able to stimulate the heart and produce ventricular fibrillation. Some have suggested that there is a wide “safety margin” between the energies delivered with a Taser and those required to induce ventricular fibrillation.

However, independent studies have been performed by Dr. Nanthakumar and colleagues in Toronto and Dr. John Webster and his group from Wisconsin demonstrating the ability not only to capture the ventricle, but to induce ventricular fibrillation. Dr Nanthakumar used a pig model in which discharges from 2 models of Taser were applied with the barbs across the chest or

across the abdomen. He reports electrical capture of the heart in approximately 75% of discharges across the chest and none across the abdomen. When they added an infusion of adrenaline, to simulate stress, 13 of 16 discharges resulted in ventricular capture. One discharge caused ventricular fibrillation and a second caused a run of ventricular tachycardia. Webster and colleagues also utilized a pig model. They turned back the fat and muscle and inserted the barbs between the ribs. They found that they could induce ventricular fibrillation in all 10 pigs. They found that the distance of the barb to the heart was critical, with the average distance that induced ventricular fibrillation being 17 mm, ranging from less than 10 to 24 mm. It did not appear to be important how far the 2 barbs were apart but the location of the barb delivering the current to the heart was critical. It is argued that this experimental model used conditions that do not replicate what would happen in a human. On the other hand, these pigs were anesthetized and, therefore, would not have been subject to a high sympathetic stimulation state, as a human would likely be in a situation where a Taser weapon would be used.

Dr. Webster's group also looked at echocardiography data to evaluate the distances from the human skin to the heart. This was done with humans in the standing position and the distance from the skin to the heart was found to be from 10 to 57 mm. Furthermore, the dart would penetrate up to one centimetre below the skin, making it possible that the dart could be found within the distance as suggested in the pig model. The same group utilized some modeling to look at the probabilities that a barb could land in a spot and cause ventricular fibrillation and estimated it to be low. However, these experiments were in anesthetized animals and did not take into account the probable enhancement of the sympathetic nervous system in an excited state.

I believe these studies have been well designed and certainly suggest that a Taser discharge could activate the heart and cause ventricular arrhythmias.

### **Can Taser Discharges Cause Ventricular Fibrillation in Man**

To be honest, we don't know. I could not find a report of an actual electrocardiographic recording of ventricular fibrillation during delivery of a Taser discharge. There have been case reports of documented ventricular fibrillation associated with incidents where Tasers had been discharged. However, it is unclear whether the Taser itself caused the ventricular fibrillation or whether it is primarily the extreme agitated state with very high sympathetic tone, possibly made worse by other ingested chemicals. Anyone with underlying structural heart disease such as a previous heart attack or a primary heart muscle disease would be more prone to developing these arrhythmias. There is one report of an individual who had a pacemaker when he received a Taser discharge. The memory capacity of the pacemaker showed that, at the time of the delivery of the shock, there was rapid pacing that captured the ventricle, suggesting that, in that particular circumstance, the Taser shock did result in transmission of electrical signals to the heart and excitation of the heart. Whether this was related to the transfer of current to the pacemaker leads or whether this was direct stimulation of the heart muscle is unclear. It does raise the issue of safety of these devices in patients with pacemaker devices and, even more so, in patients with implantable cardioverter defibrillators.

An interesting observation was made in a study of electrical recordings during Taser shocks given to volunteer police officers. The shocks were all delivered to the back using the X26 device. They did not cause any arrhythmias, as one might expect by the long distance from the dart to the heart. However, it is interesting that the average heart rate of the 115 officers was 122 beats per minute (bpm) before the shock and 137 bpm after the shock. Eight had heart rates of greater than 150 bpm prior to the Taser discharge and 15 had such fast heart rates after the Taser discharge. I think that this exemplifies the extraordinary stress that individuals can be under, even trained police officers, who are used to working in tense situations. One can imagine that an individual with psychiatric disturbance or an individual with ingestion of drugs like amphetamines could have even higher stimulation of the heart, making them more prone to ventricular fibrillation. Furthermore, the pain inflicted by the Taser discharge would likely further increase the adrenaline-like chemicals and increase sympathetic nerve discharge.

It is my opinion that there is a small possibility that an electrical discharge from a Taser dart could directly induce ventricular fibrillation. The probability is likely relatively low for any given discharge, given the necessity of having the dart penetrate within a specific distance from the heart. I cannot underscore the potential importance of the extreme agitated state and very high circulating adrenaline levels and high sympathetic nervous system discharge. Therefore, based on animal studies and the extremely agitated state of most people receiving a Taser shock, I believe that induction of ventricular fibrillation in man is possible.

Taser discharges also cause intense muscle contraction. This coupled with subsequent physical restraint of the individual could also result in the inability to breathe adequately and possibly a

drop in oxygen levels and changes in the acid balance in the blood, which would make the patient more prone to ventricular arrhythmias.

Whatever the cause of death in patients receiving Taser discharges, there does appear to be the potential of a cardiac arrest situation, as has been documented on a number of occasions. My opinion is that this risk needs to be recognized when Taser weapons are used. The perception that one gets from reports is that the police officers do not seem to recognize that situations in which a Taser is used could lead to death. A Taser certainly may be much safer than other weapons for both the victims and our police force members, but I think there needs to be an understanding that there is a potential for harm in the situation where Tasers are used, whether the Taser directly causes ventricular fibrillation or not. If this risk is acknowledged, then all people in such situations should have a firm understanding of cardio-pulmonary resuscitation and the use of automatic external defibrillators. It would seem reasonable to recommend that an automatic external defibrillator be readily available in such circumstances.

More and more British Columbians have implantable cardioverter defibrillators placed because of the risk of malignant and dangerous arrhythmias. There appears to be no evidence as to the potential harm that a Taser discharge could evoke in such a patient. The high frequency of high voltage signals certainly could be sensed by the defibrillating electrodes, and fool the device to believe that the patient was in a malignant ventricular arrhythmia, resulting in delivery of shocks inappropriately. I am unaware of any data as to the effects of Taser discharge on defibrillators but this is something that may be more important in the future.