Can the Direct Cardiac Effects of the Electric Pulses Generated by the TASER X26 Cause Immediate or Delayed Sudden Cardiac Arrest in Normal Adults?

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Abstract: There is only a small amount of experimental data about whether the TASER X26, a nonlethal weapon that delivers a series of brief electrical pulses to cause involuntary muscular contraction to temporarily incapacitate an individual, can initiate ventricular fibrillation to cause sudden cardiac arrest either immediately or sometime after its use. Therefore, this paper uses the fundamental law of electrostimulation and experimental data from the literature to estimate the likelihood of such events. Because of the short duration of the TASER pulses, the large duration of the cardiac cell membrane time constant, the small fraction of current from electrodes on the body surface that passes through the heart, and the resultant high pacing threshold from the body surface, the fundamental law of electrostimulation predicts that the TASER pulses will not stimulate an ectopic beat in the large majority of normal adults. Since the immediate initiation of ventricular fibrillation in a normal heart requires a very premature stimulated ectopic beat and the threshold for such premature beats is higher than less premature beats, it is unlikely that TASER pulses can immediately initiate ventricular fibrillation in such individuals through the direct effect of the electric field generated through the heart by the TASER. In the absence of preexisting heart disease, the delayed development of ventricular fibrillation requires the electrical stimuli to cause electroporation or myocardial necrosis. However, the electrical thresholds for electroporation and necrosis are many times higher than that required to stimulate an ectopic beat. Therefore, it is highly unlikely that the TASER X26 can cause ventricular fibrillation minutes to hours after its use through direct cardiac effects of the electric field generated by the TASER.

Key Words: TASER, ventricular fibrillation, sudden cardiac arrest, fundamental law of electrostimulation

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It has been known for over 100 years that large electrical stimuli can cause sudden cardiac arrest.[1,2] If sufficiently strong, an electrical stimulus can induce ventricular fibrillation, which is an uncoordinated, tremulous contraction of the ventricles that causes a loss of blood flow to the brain and the rest of the body, rapid loss of consciousness, and death if defibrillation is not performed. Because of the concern that electrical devices might, under certain circumstances, cause arrhythmias, many studies were performed a number of years ago to determine the range of the strengths of different types of electrical stimuli that could be delivered by these devices and to determine the conditions under which these stimuli could induce arrhythmias.[3]

The recent use of the TASER as a nonlethal weapon has led to a renewed interest in this question. Individuals have been reported to have suddenly collapsed and died after being subdued with a TASER, raising the possibility that the TASER induced an arrhythmia that led to sudden cardiac arrest. Very few studies have been published dealing with this question. An animal study by McDaniel et al[4] that used the stimulus waveform of the TASER X26, the most popular currently available TASER device, provides evidence that the device does not induce an arrhythmia, nor does it even stimulate an ectopic heart beat. While this study dealt with the question of whether the TASER can immediately cause sudden cardiac arrest, no studies have yet been published investigating whether use of the TASER can cause sudden cardiac arrest minutes to hours after its use, which is the time period in which many of the reported deaths have occurred. This paper reviews biologic and physical findings to examine the likelihood that the use of a TASER could lead to immediate sudden cardiac arrest or to delayed sudden cardiac arrest minutes to hours after its use via the effect of the electric field generated by the TASER pulses directly on the heart. This paper is limited to consideration of normal adult humans, which is a prerequisite to dealing with the added complexities of the effects of the smaller body size of children, cardiac disease, and drugs.

Electrical Stimulation of an Ectopic Heart Beat

Under certain conditions, electrical pulses can initiate an ectopic activation.[5] The implantable cardiac pacemaker uses this effect to pace the heart in patients in whom it is medically needed. Such a pacemaker initiates a heart beat by passing an electrical pulse through an electrode that is in

contact with the heart muscle. The pacing pulse must be of sufficient strength and duration to alter the transmembrane potential of the cardiac cells near the pacing electrode so that it is above the activation threshold, initiating a new action potential.[6] Activation then propagates away from this tissue near the stimulating electrode to activate the remainder of the myocardium.

Because the membrane of the cardiac cell has a capacitance as well as a resistance, the change in the transmembrane potential in response to a pacing stimulus does not instantaneously reach its full extent.[7] Rather, the effect increases with time (Fig. 1). As long as the transmembrane potential is below the activation threshold, the time that it takes for the change in transmembrane potential in a cell to reach approximately 63% of the change in transmembrane potential that would occur if the stimulating pulse were infinitely long is called the time constant. When the electrical pulse is turned off, if the change in transmembrane potential has not been sufficient to stimulate an action potential, the transmembrane potential decays back to its starting level with a similar time constant (Fig. 1).

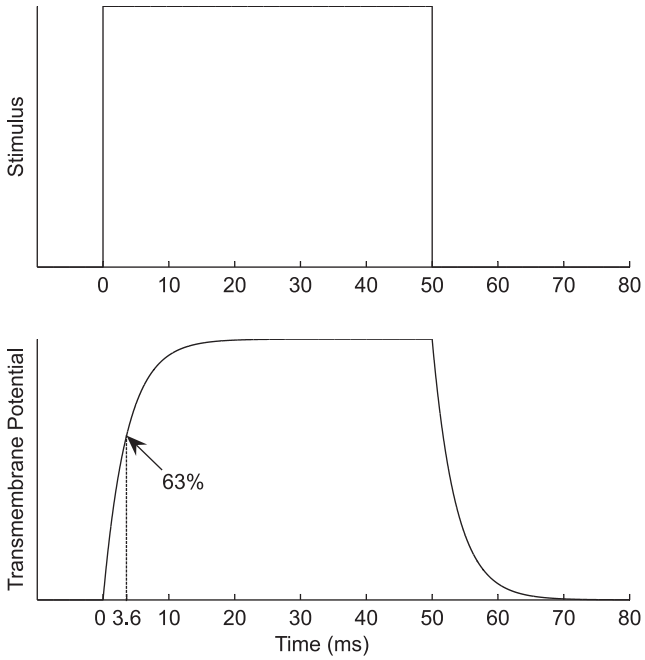

FIGURE 1. Calculated effect of a subthreshold extracellular electric stimulus (top) on the transmembrane potential of a cell modeled as a resistor-capacitor network. The change in transmembrane potential does not immediately occur to its maximum effect at the onset of the stimulus. Rather, it exponentially changes as the stimulus continues. The time for the change in transmembrane potential to reach 63% of the maximum change that would occur if the stimulus were infinitely long is called the time constant. The duration of the time constant depends upon the values of the resistance and capacitance and is different for cardiac cells and nerve fibers. When the stimulus is turned off, the transmembrane potential behaves similarly and exponentially decreases back to its original level.

Because of the time constant, there is a strength-duration relationship in the requirements for a minimum electrical pulse to stimulate a new action potential: the shorter the pulse, the higher the pulse strength needed for stimulation (Fig. 2). The effective time constant for stimulation of an action potential increases as the distance between the excitable tissue and the electrode increases.[8] While the time constant for stimulation of an ectopic beat with an electrode in direct contact with the heart has been experimentally determined in humans,[9,10] we are unaware of any publications giving the time constant for cardiac muscle with stimuli from the chest wall. However, this time constant can be estimated from experimental data for transchest pacing of

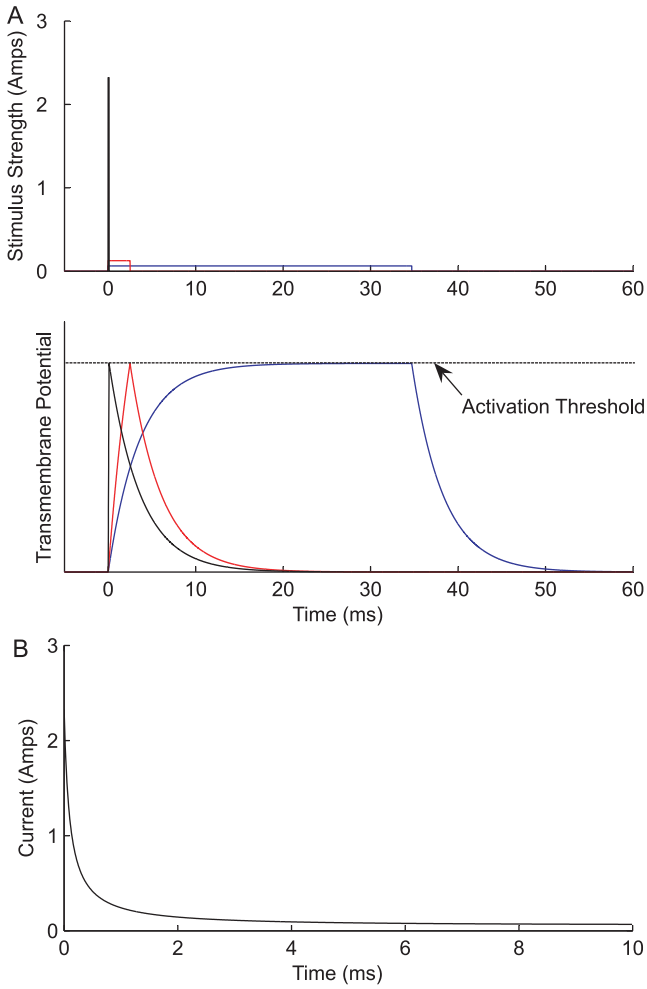

FIGURE 2. The effect of different stimulus durations on the change in transmembrane potential. The top of A shows the strengths of 3 durations of electrical stimuli, 0.1 ms, 2.5 ms, and 34.7 ms, to raise the transmembrane potential to the activation threshold according to the fundamental law of electrical stimulation when the time constant is 3.6 ms. The 0.1-ms stimulus must be 36.5 times higher than the 34.7-ms stimulus to achieve threshold. B, The complete strength-duration curve indicating the stimulation threshold for all stimulus durations between 0.1 ms and 10 ms.

dogs given in 2 papers.[11,12] These time constants are 2.6 ms and 4.6 ms, for an average value of 3.6 ms.

Application of the Fundamental Law of Electrostimulation to TASER X26 Pulses

A century ago, physiologists began to develop what is known as the fundamental law of electrostimulation. In 1901, Weiss[13] described the strength-duration requirements for excitable tissue in terms of charge and energy delivered. In 1909, Lapicque[14] developed strength-duration relationships for excitation in terms of current. In 1932, Blair[15] proposed a simplified resistor-capacitor model of the subthreshold transmembrane potential in response to a square wave stimulus. The fundamental law of electrostimulation has been shown to be sufficiently accurate to optimize defibrillation and pacing waveforms.[16–18]

As shown in the paper by McDaniel et al,[4] the pulse generated by the TASER X26 is not a simple square wave but is complex in shape (Fig. 3). However, according to the fundamental law of electrostimulation, the stimulatory effect of a waveform is the same as a square wave whose duration is equal to the duration of the actual waveform and whose current strength is equal to the average current of the actual waveform. Therefore, in the following calculations we will approximate the TASER X26 pulse as a 1 A square wave lasting 0.1 ms (Fig. 3). We also will assume that the time constant for transchest stimulation in humans is the same as in the dog (ie, 3.6 ms).

We are unaware of any published data about the stimulus strength needed in humans to stimulate the heart from electrodes on the body surface with a stimulus lasting 0.1 ms. However, we were able to find 10 papers in which the mean and standard deviation of the current strength needed to pace humans from the body surface were given for stimuli that were between 20 and 40 ms long and were delivered through electrodes on the anterior chest where the pacing threshold is lowest.[19–28] We calculated the mean and standard deviation for all 196 individuals in the 10 studies combined. The mean current was 63.7 mA, with a standard deviation of 13.8 mA, for an average stimulus duration of 34.7 ms. Because of the strength-duration relationship, for a time constant of 3.6 ms, a square-wave stimulus 0.1 ms in duration requires a stimulus strength 36.5 times greater than that for a 34.7 ms long stimulus to change the transmembrane potential the same amount (Fig. 2). Thus, according to the fundamental law of electrostimulation, the minimum strength of the TASER pulse required to stimulate the heart should be 36.5 times 63.7 mA, or 2.33 $\pm$ 0.50 A. The 1 A current of the TASER pulse is 2.63 standard deviations less than this value. In a normally distributed population, 99.6% of individuals will have a pacing threshold greater than 2.63 standard deviations below the mean pacing threshold. Therefore, 0.4% of individuals could experience an ectopically paced beat stimulated by a TASER X26 pulse if the TASER electrodes are located precisely in those positions on the left and right anterior chest where the pacing threshold is lowest.

However, if the electrodes are located elsewhere on the body, the pacing threshold will be higher. The pacing threshold rapidly increases as the electrodes are located progressively farther away from these precise locations. Geddes et al[29] found in dogs that moving the electrode on the left anterior chest only about 4 cm away from the location with the lowest pacing threshold doubled the threshold and moving it about 8 cm away tripled it (Fig. 4). Therefore, unless the TASER electrodes are located with one electrode on a small region of the left anterior chest and the other on the right anterior chest, it is highly unlikely that an ectopic beat will be stimulated.

The fact that the electrical pulses generated by the TASER X26 are too small and too short to stimulate the heart raises the question of why these same pulses are able to cause skeletal muscle contraction, the desired effect of the device. It is likely that the electrical pulses stimulate the motor nerves, which in turn cause the skeletal muscle to contract.[30] The time constant for stimulation of motor neurons from elec-

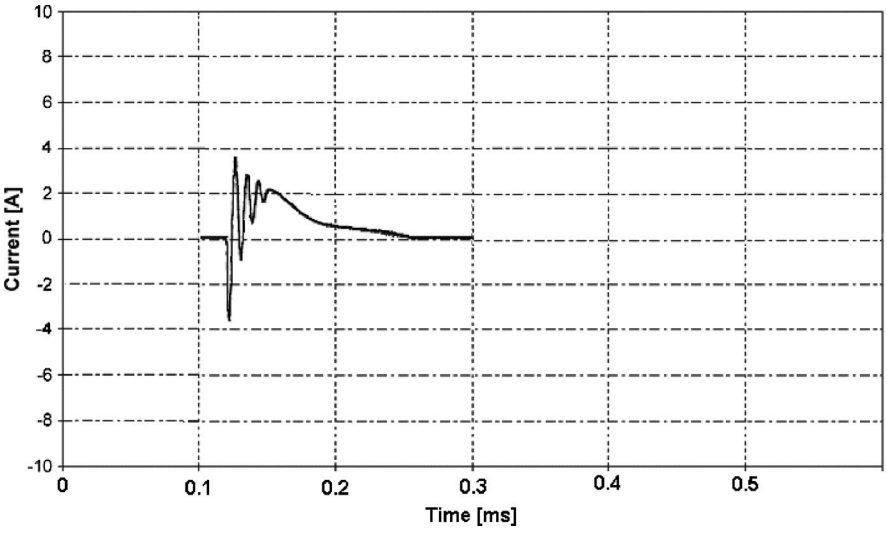

FIGURE 3. The electrical pulse delivered by the TASER X26 according to McDaniel et al.[4]

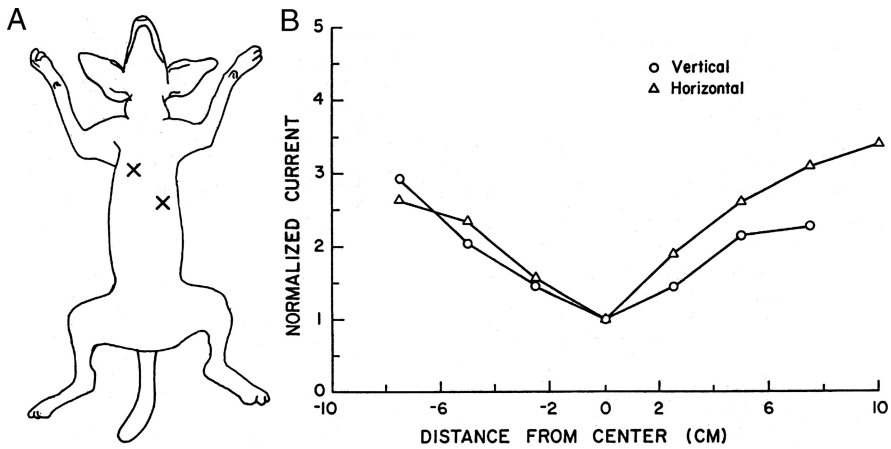

FIGURE 4. The sites on the anterior chest of the dog where the pacing threshold is lowest (Xs in A) and the increase in pacing threshold with distance of the left chest electrode away from this optimum site (B). Modified from Geddes et al,[29] with permission.

trodes on the chest of dogs is approximately 0.24 ms, much shorter than the 3.6 ms for cardiac muscle.[11] Therefore, the change in transmembrane potential in response to the electrical stimulus is much more rapid in the motor nerve than in the cardiac cell (Fig. 5). By the end of a 0.1-ms TASER pulse, the motor neuron transmembrane potential will have changed over 34% of the amount it would change with an infinitely long pulse, while the cardiac cell transmembrane potential will have changed less than 3%.

The second reason that the motor neurons are excited by the TASER pulse while the heart muscle is not is that the excited motor neurons are much closer to the electrodes that deliver the pulses than is the heart. The electrical field generated by the pulses decreases quickly with distance away from the electrodes, just as the strength of illumination from a lamp decreases as one moves farther away from the light.[31] This fact explains why the muscles that are most markedly affected are those nearest to the electrodes of the TASER device. Besides the decrease in strength of the electric field with distance away from the electrodes, the different conductivities of the various tissues in the body, such as the very low conductivity of the air-filled lungs, also cause the electric field to be many times smaller in the heart than in the subcutaneous tissues near the electrodes. Indeed, several studies have reported that the amount of current that passes through the heart from electrical pulses delivered to the chest wall is only about 4% to 10% of the total current delivered through the electrodes.[32,33] Most of the current is thought to flow around the chest between the 2 electrodes in the intercostal muscles. Also, the 4% to 10% value is for electrodes optimally placed on the chest to pace with the lowest stimulation threshold. When the electrodes are elsewhere on the body, as they are in the large majority of cases when the TASER is used, the percentage of applied current that traverses the heart would be expected to be even less.

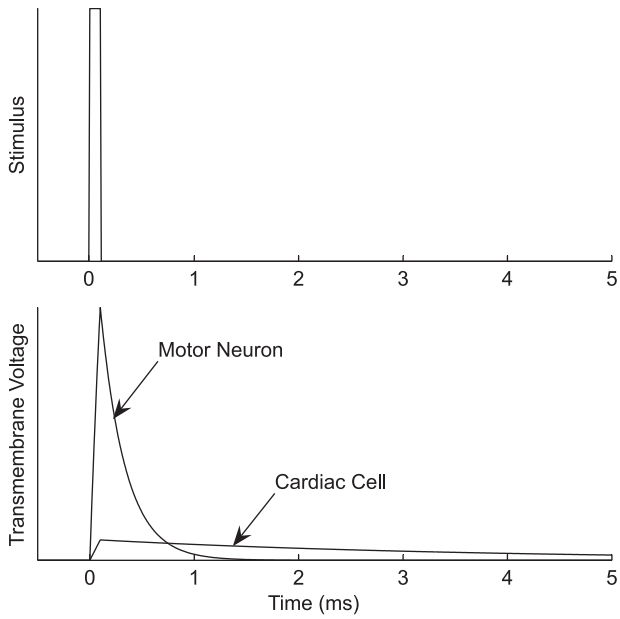

FIGURE 5. Effect of the same electrical stimulus, shown at the top, on the transmembrane potential of a motor neuron with a 0.24-ms time constant and a cardiac cell with a 3.6-ms time constant as described by Blair's[15] model.

If a series of pulses is delivered quickly in succession, it is possible that their effects could summate to change the transmembrane potential more than that caused by a single pulse (Fig. 6A). The TASER X26 delivers 19 pulses per second, which means that the onsets of successive pulses are approximately 53 ms apart.[5] If the time constant of the cardiac membrane is 3.6 ms,[11,12] the time between pulses is almost 15 time constants. Therefore, any change caused in the cardiac transmembrane potential by a pulse will have returned to within 0.0001% (63% reduction 15 sequential times) of the initial resting value before the onset of the next pulse (Fig. 6B). Thus, there should be almost no additive effect of the pulses.[34]

Electrical Stimulation of Immediate Ventricular Fibrillation

The fact that the estimated pacing threshold for an adult is 2.33 times the size of the TASER pulses and that for most positions of the electrodes on the body surface the pacing

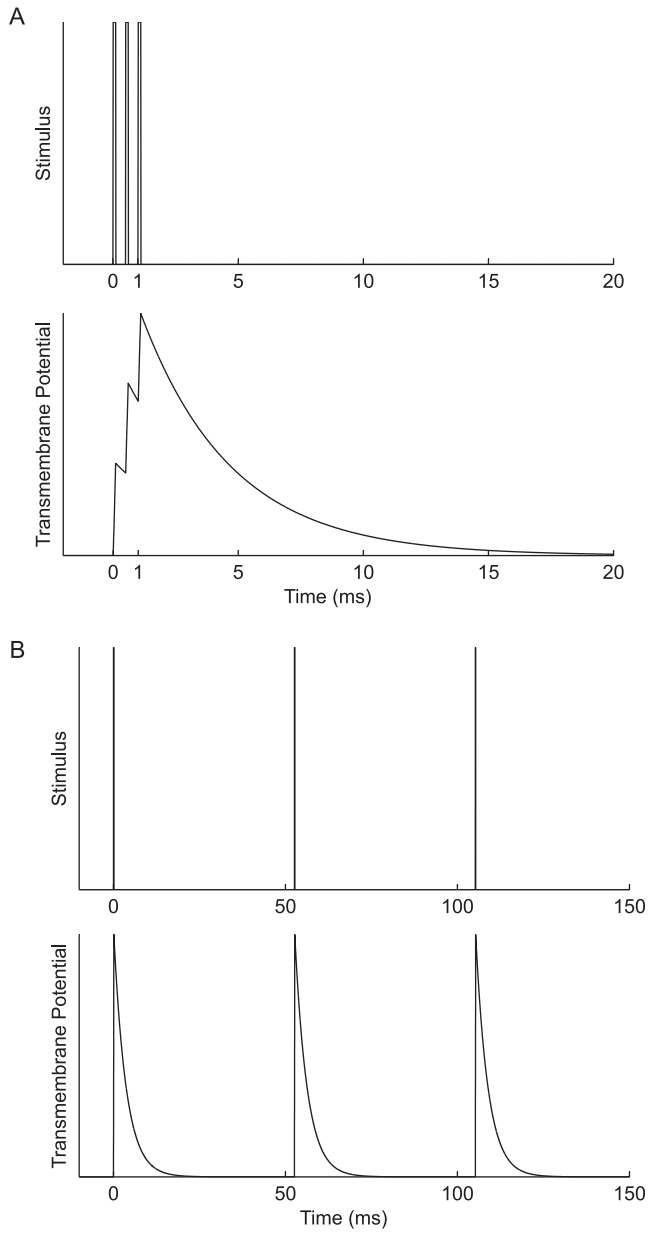

FIGURE 6. Effects of multiple electrical stimuli on the transmembrane potential according to Blair's[15] model. The time constant is 3.6 ms and the stimuli are 0.1 ms in duration. When the pulses are 0.5 ms apart (A), the membrane has not fully recovered when the next pulse occurs, so that the transmembrane potential increases with each pulse. However, when the pulses are 53 ms apart (B), as in the TASER X26, the transmembrane potential has returned almost to its initial value before the next pulse is given, so that summation does not occur.

threshold will be much higher suggests that the pulses should not stimulate an ectopic heartbeat in the large majority of individuals. Also, the estimated pacing threshold used in this calculation is the minimum excitation threshold, which means that any stimulated beat will not be very premature, since beats that are very premature have an elevated excitation threshold and require a stronger electrical stimulus.[35] In a normal heart, it is extremely unlikely that a single ectopic beat can induce ventricular fibrillation unless it is very premature.[36] Therefore, because of the larger stimulus required, the TASER X26 is highly unlikely to induced very premature stimuli that could induce ventricular fibrillation. In animals, the strength of a stimulus given during the vulnerable period of the cardiac cycle required to induce ventricular fibrillation has been found to be approximately 12.6 times the minimum pacing threshold.[12] Since the fundamental law of electrostimulation estimates that the average minimum pacing threshold is 2.33 times the size of the TASER X26 pulse, the ventricular fibrillation threshold should be approximately 29 times the magnitude of the TASER pulse. This estimate is in good agreement with the experimental study of McDaniel et al,[4] who found that the size of the pulses needed to induce ventricular fibrillation in pigs is a mean of 28 times the size of the TASER pulse. Again, these results are for electrodes located in small regions on the anterior chest; the stimulus strength required to initiate ventricular fibrillation with electrodes at other sites on the body surface should be much higher. Thus, it is unlikely that the TASER X26 will immediately induce ventricular fibrillation.

A hallmark of ventricular fibrillation is sudden collapse and loss of consciousness of the subject. Because of lack of blood flow to the brain, an individual will experience loss of consciousness within a few seconds of the onset of ventricular fibrillation. There have been reports of individuals who remain active immediately after being subdued by a TASER devise but who collapse and die minutes to hours later. One possibility is that the electrical stimuli initiated sustained ventricular tachycardia, which some minutes later degenerated into ventricular fibrillation. However, this can only occur if the electrical stimuli induced 1 or more premature beats to induce the tachycardia. As discussed above, it is unlikely that the electrical pulses generated by the TASER X26 can stimulate premature beats in the large majority of individuals. Second, ventricular tachycardia is not a stable, sustained arrhythmia in normal hearts; arrhythmias induced electrically usually either stop spontaneously after a few beats or degenerate quickly into ventricular fibrillation.[37,38] Sustained ventricular tachycardia occurs in hearts with an anatomic obstacle such as a myocardial infarction scar or a surgical scar, which can supply the milieu needed for the maintenance of a stable reentrant circuit that can maintain ventricular tachycardia. Thus, if an individual without heart disease collapses minutes after being subdued with a TASER, it is extremely unlikely that the collapse was caused by ventricular tachycardia induced at the time of the TASER stimuli.

Electrical Stimulation of Delayed Ventricular Fibrillation

Another possibility is that the electrical stimuli do not immediately induce an arrhythmia but do so minutes to hours later. The most obvious way for this to occur is if the electrical stimuli damage the heart. Electric shocks can cause

myocardial necrosis,[39,40] and myocardial necrosis can cause ventricular arrhythmias minutes to hours after it occurs.[41] However, the shocks must be very large to cause myocardial necrosis. For example, evidence suggests that shocks of 150 J or even higher used for defibrillation do not cause myocardial necrosis.[42] A typical 150-J defibrillation shock has a mean current of over 5 A and lasts approximately 10 ms. This shock delivers over 50 mC of electric charge, which is approximately 500 times the charge of a single TASER X26 pulse and over 5 times the total charge delivered by a 5-second TASER application. Thus, it is extremely unlikely that the TASER X26 causes myocardial necrosis.

The mechanism by which a large shock causes myocardial necrosis is thought to be by electroporation.[43] During electroporation, the shock electric field creates such a large change in the transmembrane potential that the membrane breaks down and pores form in it that allow the free flow of ions across the membrane.[44] If the electroporation is severe, the cell dies and becomes necrotic. If the electroporation is less severe, the cell's repair mechanisms can close the pores before the ion perturbations are so great that the cell dies. During this time of repair, which can last for several minutes, the electrical activity of the cell is abnormal and arrhythmias can occur.[45] However, the change in transmembrane potential needed to cause electroporation is greater than that needed to ectopically stimulate a single beat during diastole.[46] Thus, since TASER stimulation is unlikely to pace the heart, it is even less likely to induce electroporation.

A second way in which an electric stimulus could cause myocardial necrosis is by generating enough heat to damage the myocardium. The energy in joules expended by an electric pulse is I^2Rt, where I is the current in amperes, R is the resistance in Ω, and t is the time in seconds. If we assume that the resistance to the TASER X26 pulse is 1000 Ω, which is probably an overestimate, then the energy of a single pulse is (1 A) raised to the second power times 1000 Ω times 0.1 ms, which equals 0.1 joules. Since the TASER X26 delivers 19 pulses per second for 5 seconds, there are 95 pulses which together generate 9.5 joules of energy. One joule of energy generates 0.24 calories of heat. Therefore, 2.3 calories are generated during the 5-second period. One calorie of heat is sufficient to raise the temperature of 1 mL of water by 1°C. Thus, while there might be a slight temperature increase immediately adjacent to the TASER electrodes near the body surface, the effect on the temperature on the heart would be insignificant.

CONCLUSION

In summary, this review of the scientific literature suggests that the immediate induction of ventricular fibrillation by the direct electrical effects of the TASER X26 on the normal adult heart is unlikely and that the induction of delayed cardiac arrest by this mechanism is extremely unlikely. This conclusion is partially based on several assumptions, eg, that the depiction of the TASER X26 pulse in the paper by McDaniel et al[4] is accurate and that Blair's[15] method and the fundamental law of electrostimulation accurately predict the stimulatory effects of 0.1-ms pulses. However, unless these assumptions are grossly in error, the large safety factors for the induction of immediate or delayed ventricular fibrillation suggest that this conclusion is still true even if some of the assumptions, such as the effective time constant of the heart for electrical stimulation from the body surface, are not precisely correct. In addition, this conclusion is bolstered by the limited amount of experimental evidence in animals.[4]

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