I, Douglas P. Zipes, M.D., declare under penalty of perjury pursuant to the laws of the United States as follows:

Introduction

1) I am an electrophysiologist, a sub-specialist within cardiology who focuses on the electrical impulses that regulate the heart rhythm. I am submitting this expert report pursuant to Federal Rule of Civil Procedure 26(a)(2)(B) in the matter of *Rich v. TASER International, Inc.* After setting forth my credentials, I explain my opinion that, to a high degree of medical certainty, the electrical impulses from a Model X26 electrical control device (ECD) manufactured by defendant TASER International, Inc., (TASER) caused the cardiac arrest, and therefore the death, of 33-year-old Ryan Rich, M.D., on January 4, 2008.

2) I also address the inadequacies of TASER's pre-release testing, and therefore the recklessness with which it marketed products for law enforcement officials to use on human beings.

3) Finally, I explain my opinion that TASER's representations of safety made to the involved police agency and officer prior to this incident were not correct, that the risk of causing cardiac arrest was well known prior to this incident, and that the risk could have been minimized had TASER issued proper warnings and training materials rather than false and exaggerated representations of cardiac safety.

4) Note that many items of fact about TASER products and discussions of many of the publications were presented to two other courts. The first was in a

declaration I signed January 29, 2010, filed with a successful opposition to a motion for summary judgment in the matter of *Butler v. TASER International, Inc.*, California Superior Court for the County of Santa Cruz Case No. CV161436, and the second on October 28, 2010 filed in the matter of *Fontenot v. TASER International, Inc.*, W.D.N.C. Case No. 10-CV-125, and have not been changed substantially in this report. I also filed a report raising many of these same issues in *Williams v. TASER International, Inc.*, N.D. Ga. Case No. 06-CV-0051. Of course the unique facts of this case are addressed.

My Credentials for Offering This Expert Report

5) I received my Bachelor of Arts degree cum laude from Dartmouth College in 1961, and my medical degree (M.D.) cum laude from Harvard Medical School in 1964. I performed post-graduate training in internal medicine (1964-1966) and cardiology (1966-1968) at Duke University Medical Center, Durham, North Carolina. From 1968-1970, I was in the United States Navy and discharged with a Letter of Commendation at the rank of Lieutenant Commander. I joined Indiana University School of Medicine as an Assistant Professor of Medicine in 1970, and became Professor of Medicine in 1976. I also became Professor of Pharmacology and Toxicology in 1993. In 1994, I became Distinguished Professor, the university's highest professorial category for academic accomplishment. In 1995, I became Director of the Division of Cardiology at the Krannert Institute of Cardiology, a post I held until 2004, when I became Emeritus. I continue to see patients and consult

with many physicians on difficult patient problems. I attend conferences, teach and interact with members of the Division, and house staff. (Attached hereto, as Exhibit A, is a copy of my *curriculum vitae*.)

6) My principal areas of research and clinical activities focus on cardiac electrophysiology (heart rhythm problems). However, I also take care of patients with the entire spectrum of cardiac diseases, including hypertension, heart failure, atherosclerosis, acute and chronic myocardial infarction (MI), lipid abnormalities, heart muscle abnormalities, thromboembolic problems, and adult congenital heart disease.

7) I have published over 800 medical articles and 21 textbooks. I am the co-editor of *Zipes/Jalife Cardiac Electrophysiology, From Cell to Bedside* (2009, fifth edition) and a co-editor of *Braunwald's Heart Disease, a Textbook of Cardiovascular Medicine* (2011, ninth edition). Each is regarded as the authoritative text in heart rhythm disorders and in general cardiology, respectively. I am co-author of *Clinical Arrhythmology and Electrophysiology* that was published in November 2008.

8) I am a member of numerous societies, including the American Society for Clinical Investigation and the Association of American Physicians, both of which have very stringent acceptance qualifications. I am past president of the Association of University Cardiologists and of the Cardiac Electrophysiology Society. I have consulted for, and been on review committees of, the Veterans Administration, the American Heart Association (AHA), and the National Heart, Lung, and Blood

Institute (NHLBI) of the National Institutes of Health (NIH).

9) For the Heart Rhythm Society (HRS), which is the largest group of heart rhythm experts in the world, I was a founding member in 1980, served in many roles, and became President 1989-1990. I am the founding Editor-in-Chief of the HRS journal (see below).

10) For the American Board of Internal Medicine (ABIM) (the organization that writes all of the examinations for medicine and the sub-specialties which a physician must pass to call himself/herself Board Certified), I was Chair of the Clinical Cardiac Electrophysiology Test Committee which wrote the first (and subsequent two) examinations in clinical cardiac electrophysiology; Chair of the Subspecialty Board on Cardiovascular Disease, which wrote the examinations dealing with all of cardiology; Chair of the Committee on Subspecialty Internal Medicine (dealing with issues of all of internal medicine); and ultimately Chair of the entire ABIM (2002-2003).

11) For the American College of Cardiology (ACC) (35,000 cardiology members worldwide), I have held multiple roles since 1975, becoming a member of the Board of Trustees (1992-1997 and again 1999-2005); Chair of the Nominating Committee on two separate occasions; Chair of the Development Committee; Vice President, President-Elect; and then President (2001-2002). I became a Master of the ACC in 2002 (highest membership category). I was Co-Chair of the ACC/AHA/ European Society of Cardiology (ESC)/HRS Ventricular Arrhythmia and Sudden

Cardiac Death Guideline Committee, which wrote the guidelines on how to care for patients with heart rhythm problems. I was also a member of the Ethics Committee and chaired the Task Force on Legal Expert Testimony. In 2009, I received an ACC Presidential Citation for organizing and chairing the first International Cardiovascular Conference: Focus on the Middle East, which brought together cardiologists from each of the Middle Eastern countries for two days of education and interaction. The second one was held in March 2010, and was attended by over 500 cardiologists. The third symposium was held April 2, 2011, attended by over 350 cardiologists. I received an award from the Iran Cardiac Society when I lectured in Tehran November 2008, and I gave the Plenary Lecture for the Saudi Cardiac Society in February 2010 and for the Qatar Cardiac Society in April 2010.

12) I am a member of the editorial boards of more than 15 cardiology journals; and have reviewed articles for other general medical journals, such as the *New England Journal of Medicine* (NEJM) and *Journal of the American Medical Association*. I have been Editor-in-Chief of *Progress in Cardiology*, and Founding Editor-in-Chief of *Contemporary Treatments in Cardiovascular Disease and Cardiology in Review*. I was the Founding Editor-in Chief of the *Journal of Cardiovascular Electrophysiology* (1989-2004), and in 2004, I became Founding Editor-in-Chief of the journal, Heart*Rhythm*, which is the official journal of the HRS and has become the number one specialty cardiology journal in the world.

13) I have received many awards, but will mention only a few. From the

AHA I received the Distinguished Achievement Award (1989), the Herrick Award (1997), and the Cor Vitae Award (2004). These awards have been for distinguished contributions to our knowledge base of clinical cardiology and patient care. I have received the Distinguished Scientist Award from the HRS (1995) and from the ACC (1996), for research contributions to both basic and clinical cardiology. On June 2, 2004, the Honorable Baron P. Hill, United States House of Representatives, read a tribute about me into the Congressional Record. After my tenure as Division Chief, the following were endowed: the Medtronic Zipes Chair in Cardiology, and the Joan and Douglas Zipes Visiting Professorship at Indiana University School of Medicine; the Douglas P. Zipes, M.D., Lectureship given annually at the HRS Sessions; and the Douglas P. Zipes, M.D., Distinguished Young Scientist Award given annually at the ACC Scientific Sessions. In 2007 I was made an honorary foreign member of the Argentine Society of Cardiology, and received the Distinguished Alumnus Award from the Duke University Medical Center. In May, 2010, I was inducted as an honorary member in the Hungarian Society of Cardiology. In September, 2010, I received the President's Medal from Indiana University, the University's highest award. In April 2011, I became a Master of the American College of Physicians (membership 130,000 internists, internal medicine subspecialists, medical students, residents, and fellows), which according to their website, is given to *"highly distinguished physicians, selected from among Fellows, who have achieved recognition in medicine by exhibiting preeminence in practice or medical research, holding positions of high honor, or making*

significant contributions to medical science or the art of medicine."

14) I am a Fellow of the: AHA, HRS, European Society of Cardiology, Master of the American College of Physicians and of the American College of Cardiology, and honorary fellow of the: Argentine Society of Cardiology, the Hungarian Society of Cardiology, and of the Cardiac Society of Australia and New Zealand. I am Board Certified in Internal Medicine and Cardiovascular Diseases and was Board Certified (until 2005) in Clinical Cardiac Electrophysiology, which I let lapse because I no longer perform invasive procedures.

15) I was a consultant for Medtronic from 1975 to 2010, and am the inventor of the implantable cardioverter, which is part of the implantable cardioverter defibrillator manufactured by Medtronic and other device companies, the usual device implanted in high risk patients. All royalties for that invention were assigned to Medtronic and I have received none. I have written on warnings about side effects of implantable devices and drugs and their ability to cause cardiac arrhythmias. Warnings are included in a number of manuscripts I have published.

16) I have been retained as an expert witness and have testified in deposition and court on multiple occasions both for plaintiffs and defendants. A list of my testimony is attached as Exhibit B, and my fee schedule as Exhibit C. Three of those cases involved a cardiac arrest, in my opinion, caused by electronic shocks administered by a TASER product: *Williams v. TASER International, Inc., Butler v. TASER International, Inc,* and *Fontenot v. TASER International, Inc.* I prepared an expert

report or declaration and sat for a deposition in all cases. I understand that *Williams* was resolved, confidentially and *Butler* for $2.85 million. *Fontenot* is pending.

17) Based on my work in *Williams* (later reaffirmed in *Butler* and *Fontenot*), I became concerned that TASER was misrepresenting that there are no cardiac risks posed by its ECDs. Accordingly, I delivered a PowerPoint presentation on those risks at the May 2009 HRS meeting in Boston. A copy is attached as Exhibit D.

18) The PowerPoint presentation was delivered as part of a formal, invited, scheduled debate with a distinguished colleague, Patrick J. Tchou, M.D., who headed the Section of Electrophysiology and Cardiac Pacing in the Robert and Suzanne Tomsich Department of Cardiovascular Medicine at Cleveland Clinic in Cleveland, Ohio, from 1994 to 2006, and is currently a practicing staff physician there concentrating on treatment of cardiac arrhythmias, especially those of complex origins. At the end of my presentation, Mark W. Kroll, Ph.D., an electrical engineer and the head of the TASER Scientific and Medical Advisory Board, who was in the audience -- Dr. Kroll sold TASER stock options for $4 million and makes more than $100,000 annually for part-time TASER work, according to his deposition testimony -- spoke in opposition to my remarks. Further, I told an official of TASER, I now understand him to be Tom Smith, who was also in the audience, that TASER needed to issue a warning to indicate its products might cause cardiac effects. We had sharp disagreements, and I left more concerned than ever about the undisclosed cardiac dangers of ECDs. After the debate finished, the moderator polled the

audience and found that more than 90 percent of the audience supported my side of the debate that TASER ECD shocks could produce ventricular fibrillation. (TASER issued its first warnings to avoid chest shocks about four months later, on September 30, 2009.)

19) I exchanged emails with Dr. Tchou after the debate to arrange for us to continue our debate in the pages of Heart*Rhythm*. The exchange is attached hereto as Exhibit E as an email string, and should be read from the bottom. I suggested to Dr. Tchou that "I would write 'Taser shocks can cause ventricular fibrillation,' while you would write 'Taser shocks do not cause ventricular fibrillation.'" Dr. Tchou responded, "I appreciate the invitation. But, the line is drawn too far to one side for me to truly defend that position very well. From the very beginning, as we obtained our data from our animal study, I had advised TASER that the possibility of inducing ventricular arrhythmias is there and at the minimum we cannot categorically say that this is not possible."

20) The list of materials I reviewed in connection with this case, including some of those I reviewed for *Williams, Butler* and *Fontenot*, are attached as an addendum to this report. In addition to the materials listed, as an ongoing part of my practice, I continually review scientific literature about materials potentially relevant to my experience in the areas of cardiology, medicine, and electrophysiology. Being essentially infinitely voluminous, I obviously cannot identify all such materials in this case. For practical reasons, I referenced herein those materials which most directly

support my opinions.

21) This information forms the basis of my findings and analyses and permits me to arrive at my opinions. I understand that discovery is ongoing, and should additional information become available, I reserve the right to review that information as well as my analysis and findings. I just received the important deposition transcripts of Trooper Lazoff and Dr. Morris. I expect to receive TASER's expert witness reports shortly and address the issues stated in them in my deposition testimony and, if necessary, prepare a supplemental report.

Summary of the Incident Involving Ryan Rich, MD

22) I have reviewed available statements, interviews and medical records, as listed in this report, and understand the general facts of this incident to be as follows.

23) On January 4, 2008, at about 1:00 p.m., Nevada State Trooper Loren Lazoff observed a black pickup truck traveling southbound on I-15 strike the rear of a semi-trailer, then hit a van, crossing three lanes of traffic, and then crash into the center barrier wall twice and stop with its left front wheels halfway up the barrier. The trooper approached the passenger door and knocked. The driver, Dr. Ryan Rich, just stared at him, then turned and looked straight ahead. He was wearing hospital scrubs. The trooper knocked again, and saw no response. The trooper knocked again and commanded Dr. Rich to open the door. Dr. Rich turned and stared at him. Trooper Lazoff went back to his car and retrieved his baton. He knocked again, asking that Dr. Rich open the door, and again Dr. Rich just turned his head and

stared. Trooper Lazoff used the baton to break the passenger window, reached in, unlocked the door, turned off the ignition and tried to put the gear in park. He was unable to remove the key. He noted an empty pill bottle on the seat. (It had contained gabapentin, an anti-seizure medication.) There was no smell of alcohol. Dr. Rich was not saying anything.

24) Responding to the commands of Trooper Lazoff, Dr. Rich crawled to the passenger side and Trooper Lazoff cuffed his left wrist. Dr. Rich pulled back in response to the cuffing, pulling Trooper Lazoff, who was holding the handcuffs, into the cab. Trooper Lazoff then pulled Dr. Rich out of the cab. Dr. Morris describes Dr. Rich as being in a "stupor." According to Dr. Morris, Dr. Rich was standing by his truck for some time, but kept trying to wander away, potentially into traffic. According to Trooper Lazoff, however, while Dr. Rich was standing up, "crab walking" according to the deposition testimony, Trooper Lazoff drew his TASER Model X-26 and shot Dr. Rich in the chest at a range of 4 to 5 feet. Trooper Lazoff, as TASER instructed, aimed for "center body mass." Trooper Lazoff noted that one probe was near the heart. He did not see the other one. The shock seemed to have the intended effect and Dr. Rich fell backwards. The dataport shows the cycle to have lasted 9 seconds. Trooper Lazoff was able to pull Dr. Rich by the right arm to a safer position on the shoulder, towards the rear of the pickup truck. As Trooper Lazoff was trying to complete handcuffing, he thought Dr. Rich might be reaching for the probes, so he shocked Dr. Rich with a second cycle. The dataport shows 9 seconds

between the first two cycles. Dr. Rich stood up and was shocked a third time. The trooper felt the shocks from the wires and turned off the device. That apparently happened twice. The Trooper then cycled the device a fifth time. Apparently Dr. Rich was lying on his chest at this point. According to the dataport and Trooper Lazoff's deposition testimony, he cycled the device another six times in probe mode. The discharges appeared to be constricting Dr. Rich's body.

25) During the initial tasings, a passerby, Dr. Craig Morris, a facial surgeon, stopped to help. Trooper Lazoff drive-stunned Dr. Rich, *but with the cartridge still in place.* Trooper Lazoff believes that the first drive stun was for the full five seconds, but the second one was pulled off Dr. Rich prematurely, meaning that most of the current went through the probes. Trooper Lazoff testified that on this last discharge, the X26 continued to cycle while out of contact with Dr. Rich. With Dr. Morris' assistance, Trooper Lazoff was able to handcuff Dr. Rich within a few seconds of the final discharge. As the trooper stood up, Dr. Rich was on his chest, with his face turned away. Neither Trooper Lazoff nor Dr. Morris checked Dr. Rich's eyes, whether he was breathing or checked his pulse.

26) Because of ongoing transmission problems with his hand-held radio, Trooper Lazoff had to walk back to his vehicle to use the radio there to inform dispatch what had happened and to advise them to send the medical team, routine for TASER uses.

27) Dr. Morris indicates he looked back at Dr. Rich about 30 seconds later and

noted that the suspect was turning blue (cyanotic). He told the trooper and they ran back to find that Dr. Rich was not breathing. They turned Dr. Rich on his back and began CPR, with his hands still cuffed behind him. They state that Dr. Rich might have intermittently breathed on his own, but they felt no pulse and continued CPR until emergency personnel arrived.

28) In his statement at the Coroner's Inquest, Dr. Morris states initially that he thinks Dr. Rich was tased four times but later states he thinks it was five times over a period of four or five minutes (we know it was 13 times over 3 minutes from the dataport, see below). In his voluntary statement in an interview with M. Weidmann on 1/4/08, Dr. Morris states that after cuffing Dr. Rich's breathing appeared normal (specifically, not "unbelievably fast") despite his having actively resisting being handcuffed at the time they left him. Dr. Morris says he followed the officer to his vehicle, turned to look at the driver about 20-30 seconds later and saw that Dr. Rich wasn't moving. Dr. Morris walked back to the driver who looked pale, asked him if he was okay. Dr. Rich didn't respond and his eyes didn't focus. Dr. Morris went back to the officer, told him about the driver, and they both walked back to the Dr. Rich and his face was "pretty much gray".

29) Dr. Morris listened to the man's chest but states, "It was so loud, I couldn't really hear whether he was breathing or not because of the…the traffic." He then states, "I could hear that his heart was beating but I could not hear any air going in and out…" And then he says that Trooper Lazoff could not feel a pulse and so they

initiated CPR, Dr. Morris providing breathing support and Trooper Lazoff pumping on his chest.

30) The discrepancies are obvious. The traffic was making so much noise Dr. Morris couldn't hear breath sounds but he reports he could hear heart beats with his ear on Dr. Rich's chest through the scrub shirt Dr. Rich was wearing (since there is no indication he took off Dr. Rich's shirt), during a tumultuous time period. Further, he heard heart beats at a time when the trooper could feel no pulse. Finally, the events indicate that Dr. Rich was having cardiac arrest and not likely to have an audible heart beat. We know that Dr. Morris was mistaken in his recollection of the number of TASER shocks during the press of the events (noted above), and he was likely also mistaken about his recollection of Dr. Rich's normal breathing (when he left him) and hearing heart beats (when he returned), despite being a physician, albeit a facial surgeon. In is deposition, Dr. Morris states that, "...the noise from the traffic was so heavy I couldn't hear anything, I couldn't hear whether his heart was beating or whether he was breathing..."

31) More likely than not, Dr. Rich developed cardiac arrest at the time he no longer resisted being handcuffed, when they left him to walk to the trooper's vehicle. The ECG recorded from the defibrillator pads at Spring Valley Hospital January 4, 2008, shows electrical activity consistent with very fine ventricular fibrillation at the end of the strip labeled 14:49:35 and in the strip labeled 14:49:55. Probable monitor strips labeled Acuity begin at 14:02:42 are consistent with asystole. Since he is

pronounced dead at 13:53:00, the timing of the strips recorded from the defibrillator pads are most likely not synchronized with the timing of the other recordings and were probably recorded earlier. So, it is likely he developed ventricular fibrillation as the rhythm causing cardiac arrest, which progressed to low amplitude VF (called fine VF) and then asystole as the lack of cardiac perfusion progressed.

32) The time line given is that Trooper Lazoff initiates a traffic stop of Dr. Rich at 13:00:50. At 13:09:25, he requests EMTs, which is presumably after he has just applied the ECD. At 13:10:51 Lazoff advises dispatch Rich is unresponsive, presumably after Dr. Morris brought Dr. Rich's unresponsive state to his attention. At 13:16:36, the fire truck arrives. At 13:21:00, American Medical Response arrives. At 13:32:00 Dr. Rich is transported to the hospital, arriving at 13:40:00. He is pronounced dead at 13:53:00.

33) The dataport of the TASER indicated that the trigger was pulled 13 times, 1 time for 9 sec, 1 time for 2 sec, 4 times for 4 sec and 7 times for 5 seconds, for a total activation time of 62 seconds delivered over slightly less than 3 minutes. The first 52 seconds were delivered solely in the probe mode (by the report from the trooper), with 13:03:38 being the end of first cycle, and a portion of the last 5 second deployment was delivered through the probes and in the drive stun mode to Dr. Rich's right leg, with the last cycle ending at 13:06:29.

34) While the LVMP Homicide Report states there is no way to determine how many electrical cycles Dr. Rich actually received, the LVMP Homicide Report

also indicates that, "Upon arrival on scene Ell's crew witnessed an NHP officer attempting chest compressions and another attempting to provide respiration's via a BVM on a male pt that was in the supine position handcuffed with the pt's hands to his back. Pt also appeared to be extremely cyanotic to his face and head. *Ell's crew also noticed what appeared to be 2 sets of tazer barbs attached to the pt.*" (italics mine). **Therefore, it is apparent that the TASER barbs were attached to Dr. Rich's chest for all 13 discharges, for a total of 62 seconds delivered over a period of less than 180 seconds**. As I understand Trooper Lazoff's testimony, probably no more than 6 or 7 seconds were delivered by drive stun.

35) Autopsy revealed cause of death to be "due to seizure disorder with other conditions including restraining procedures" by Piotr A. Kubiczek, M.D.Medical Examiner. There was abnormal brain anatomy with neuronal dropout, diffuse neuronal heterotopias, and other changes consistent with a lifelong seizure disorder. Many blunt force injuries and abrasions were noted, including burn marks on the buttocks consistent with the drive stuns. One punctate wound was noted in the left upper and another left lower chest, consistent with chest barbs from the TASER X-26. The depth of penetration was not determined. Heart weighed 470 gms, stated by the medical examiner to be mildly enlarged. I disagree. According to Kitzman et al. (Mayo Clin Proc 63:137, 1988) normal heart weight for an adult male weighing 220 pounds ranges between 296 and 516 grams. Based on height of 6'2", the normal range is 247-492 grams, but Kitzman et al. indicate that heart size correlates better

with body weight, which should be used, rather than height, when available. The RV wall measured 0.3 cm thick and the LV wall 1.9 cm. According to Kitzman et al, normal RV wall thickness is .38-.4 cm and LV between 1.23 and 1.5 cm, so the LV wall could be considered slightly thickened. There was 10-20% narrowing of the left anterior descending coronary artery. Cardiac histology was normal. Gabapentin 31micrograms/ml (Neurontin, non-narcotic medication being taken for a seizure disorder, and for back pain) was noted in the blood, which was negative for alcohol and other drugs. In patients with normal renal function, the maximum serum concentration is 25 micrograms/ml with the FDA approved dosing (Blum Clin Pharmacol Thera 56:154, 1994; Bookwalter Pharmacotherapy 25:1817, 2005). An average starting dose is 300 mg three times daily with doses increasing to 600 or 800 mg three times daily. Dr. Rich was prescribed 800 mg four times daily.

36) At the coroner's inquest April 18, 2008, Dr. Kubiczek indicated the cause of death was a seizure disorder with other contributing factors including restraining procedure and manner of death was homicide. He said there was no scientific evidence that an electronic control device can contribute to death, which is not correct. Such evidence exists in the peer-reviewed literature. He did not know whether a TASER application over the heart had any bearing. It is well established in the peer-reviewed literature that trans-cardiac vectors over the chest pose the highest risk of cardiac capture. He stated the evidence indicated Dr. Rich was tased four or five times, when the dataport shows 13 cycles. In his deposition, he states that Dr.

Rich developed an arrhythmia from the seizure disorder and cannot state that it was the TASER because there is no scientific basis to support that conclusion. Dr. Kubiczek's deposition on 3/24/11 affirms that he knows nothing about the scientific literature that demonstrates how ECD devices can produce sudden death.

Mechanisms Producing Ventricular Fibrillation

37) It is important to understand the cardiac mechanisms involved in this incident. Ventricular fibrillation (VF) is a highly disorganized heart rhythm (fibrillation) of the bottom chambers (ventricles) discharging at rates of 400-600 times/min. Such a rate is too fast for the ventricles to pump blood effectively to the brain and other organs. VF results in unconsciousness in 10-20 seconds and irreversible brain damage or death in 5-8 minutes unless the VF is terminated by an electrical shock from a defibrillator. VF is the mechanism of Rich's cardiac arrest and subsequent death. Ventricular tachycardia (VT) is a more organized heart rhythm at slower rates (100-250/min) that can progress to VF. There are many causes of VT/VF but in the present context, I will consider two: ischemic and electrical.

38) Ischemia, or reduced/lack of blood flow, can result when the blood pressure falls to low levels, such as can occur during very rapid ventricular rates. Cardiac ischemia, when the heart muscle is deprived of normal blood flow, creates an electrophysiologically unstable ventricle prone to developing VF. In the TASER studies on pigs by Lakkireddy and Tchou et al., and Nanthakumar et al, the rapid ventricular rates following "capture" (see below) of the heart by the TASER ECD

pulses caused very low blood pressures that undoubtedly resulted in degrees of cardiac ischemia during the duration of the ECD cycle. Long ECD cycles or repeated short cycles with little recovery time in between could result in cardiac ischemia sufficient to cause VF.

39) Recently, Kroll et al. published (32nd Annual International Conference of the IEEE EMBS Buenos Aires, Argentina, August 31-September 4, 2010) "A Novel Mechanism for Electrical Currents Inducing Ventricular Fibrillation: The Three-Fold Way to Fibrillation," claiming to "present new data showing a 3rd mechanism of inducing VF which involves the steps of delivering sufficient current to cause high-rate cardiac capture, causing cardiac output collapse, leading to ischemia, for sufficiently long duration, which then lowers the VFT (VF threshold) to the level of the current, which finally results in VF." I had indicated this mechanism in my February 19, 2009 expert report on *Butler v. TASER International*, as explained above, based on well-established physiological principles known for some time. Kroll et al. state that "There is some existing support for this hypothesis in scattered data in the existing literature." This is not a new mechanism anymore than adding an intervention such as a drug or coronary occlusion to the rapid pacing would constitute a new mechanism. It is pacing induced VF modulated by another event, in this instance, ischemia.

40) The paper by Kroll et al. hypothesizes that rapid pacing-induced ischemia leading to VF takes a minimum of 90 seconds to occur. Therefore, the

authors claim that rapid pacing-induced VF, such as might be delivered from a TASER ECD shock, occurs either in the first 4-5 seconds or after 90 seconds, with no VF possible in between the two times. I disagree. My conclusion is that VF can occur at virtually any time during an ECD cycle if there is cardiac capture.

41) Besides resting on faulty reasoning, the Kroll et al. study on which that conclusion is based has multiple, serious scientific flaws that invalidate that conclusion. First, the only figure purporting to show data from the study (figure 4) exhibits results from only 5 seconds of rapid pacing (not differing periods up to 90 seconds); second, the blood pressure channel has no calibration and shows a return of blood pressure midway, which the authors explain as a skeletal muscle contraction effect, which would be unlikely in an anesthetized animal and could represent transient loss of pacing capture that would blunt the ischemia by allowing transient coronary blood flow; third, they state that heart contraction and capture by TASER was monitored by echo but give no examples, so there is no proof of cardiac capture at the asserted rates; fourth, there is no example of 90 seconds of pacing-induced ischemia leading to VF, their main conclusion; fifth, there are no endpoints showing actual attainment of ischemia, such as reduced pH or elevated potassium, known ischemic end products, important since there can be degrees of ischemia (it is not a binary end point, present or not), and the cycle length chosen may have been insufficient to provoke a significant degree of ischemia; sixth, there are no statistics to prove that the results were statistically significant, i.e., $p<.05$; and finally, the only

"data" given are not data at all but merely statements labeled "new data" at the bottom of table 3, without actually showing any data. In that regard, it would be highly unlikely that 4 of 6 animals all developed VF at 90 sec of pacing exposure, as stated. The paper was published as part of an IEEE conference in Buenos Aires and whether it was subjected to the usual review process is not indicated.

42) Further, even if the authors' conclusions were supported by data and tenable, the alleged ischemia attained would be induced in supine, anesthetized, ventilated pigs with presumably normal hearts at alleged paced rates of 228 beats per minute. Anesthesia can act as an antiarrhythmic intervention. This animal experiment is a far cry from rapid pacing in an upright, agitated human being under severe sympathetic stress, such as Ryan Rich, when the effects of ischemia would be far more pronounced. Further, the heart might be captured at a faster rate due to the sympathetic effects that shorten ventricular refractoriness (see below). Finally, some individuals have underlying heart disease such as coronary obstructions, left ventricular hypertrophy, or other problems, which would exaggerate the effects of rapid pacing and shorten the time to VF. (I see no indication of any of these conditions in Ryan Rich, however.)

43) Direct electrical stimulation of the ventricles can cause VF in at least two ways. First, electrical stimulation during the vulnerable period of the T-wave (a 30-40 ms time interval during ventricular recovery from the preceding beat when the heart is potentially unstable – its timing is during the peak of the T-wave in the ECG) can

provoke VF. The electrical stimulus required to do this is fairly large – about 1 joule – requiring current 3-4 times that necessary for "capture." Such T-wave stimulation is done routinely in the electrophysiology lab to provoke VF in patients and test the ability of an implanted cardioverter-defibrillator (ICD) to recognize the VF and defibrillate it.

44) The second mechanism is by ventricular capture at rapid rates. The ventricles can be stimulated directly by an electrical pulse of 0.5 milliseconds (msec) duration and 0.8-1.0 volts, or about 2 mA, delivered over an electrode on or in the heart. When the heart depolarizes in response to that electrical stimulus, it is said to be "captured" by the stimulus. The heart also can be captured by an electrical pulse delivered across the chest wall, which requires more electricity, in the range of 50-80 mA. However, that threshold is modulated by the stimulation site and type of electrodes, type of shock, sex, torso and body mass, cardiac and non-cardiac diseases, drugs such as cocaine and alcohol, medications, and the adrenergic (excited) state of the individual. An adrenergic state means more catecholamines (naturally produced stimulant chemicals like adrenaline and noradrenaline) are in the blood, which can facilitate the initiation of VF by electrical stimuli. In fact, in the evaluation of patients with VT in whom we are unable to induce the VT during an electrophysiologic study (that involves electrically stimulating the heart at fast or premature intervals), we routinely infuse a catecholamine IV to facilitate induction of the VT. With electrical stimuli delivered over electrodes on/in the heart or on the chest wall, when the rate of

capture of the heart by the stimuli exceeds around 250-300/min, the organized ventricular electrical activity can become disrupted, the contractions can become disorganized and VT/VF can result, causing a cardiac arrest such as that suffered by Ryan Rich.

45) Electrical current from a TASER X26 application that captures the heart could result in VF at virtually **any time** during the pulsing shock delivery. The shocks just need to produce a sufficiently rapid ventricular rate to disorganize the electrical activity of the ventricles, which can happen at any time during the ventricular capture. It does not have to be within the first 5 seconds or after 90 seconds for the following reasons. The X26 delivers 19 pulses per second (52.6 msec intervals), which is a rate of 1140 beats per minute (BPM) with each pulse 100 microseconds in duration, delivering a peak amperage of 3-4 amps and a total charge of about 100 microcoulombs. The ventricles of the heart have a period after being stimulated called the ventricular effective refractory period (VERP) during which they will not respond to another stimulus at all (absolute VERP) or only respond to a larger stimulus (relative VERP). It is a "rest period" during which the heart prepares for the next contraction and is one of nature's ways to protect the heart from beating too rapidly. The VERP protects quite well most of the time, as, for example, with ordinary pacemakers. However, extraordinary pacing exposures such as with a TASER ECD shock can disrupt that safety feature. Human VERP normally ranges between 200-250 msec. So, assuming a VERP of 250 ms, the maximum rate of

pacing capture will be 4/sec or 240 BPM (60,000 msec in one minute divided by 250 ms = 240BPM). Accordingly, a TASER ECD shock stimulating at 1140 BPM will result in roughly a 5:1 capture, i.e., one ventricular response to every 5 stimuli, which is what Kroll et al. report in the pig study noted above and is close to what Cao et al. found in the patient with a pacemaker receiving a TASER ECD shock. Importantly, however, heart rate modulates the VERP, so the faster the heart rate, the shorter the VERP. Catecholamines from sympathetic discharge also shorten the VERP. I would expect that Dr. Rich had a significant sympathetic discharge of catecholamines from the pain of the probe deployments and drive stunning.

46) Therefore, the following scenario can occur: an individual receives shocks from a TASER X26 which, depending on his VERP, can result in an initial heart rate of 240 BPM. However, that increased heart rate, along with sympathetic discharge from the pain and excitement that causes the release of catecholamines, shortens the VERP to 200 ms so the capture rate can increase to, say, 300 BPM. That new rate can further shorten the VERP, which will increase the heart rate still further, often with irregular captures (the capture ratio does not necessarily remain constant) that add to the disorganization of the heart beat, until a rate results that is sufficient to produce VF. A ventricular capture can also occur during the T wave to cause VF due to a ventricular beat falling in the vulnerable period of the T wave. Clearly, the onset of VF can occur any time along the continuum of capture from the TASER shock.

47) The process described above is well illustrated in the Nanthakumar et al

paper, cited below. Figure 2 shows cardiac capture by the TASER X26 in a 3:1 ratio, at a rate of about 375 BPM, but no VF. The rhythm and blood pressure return to normal when the TASER shock is stopped. In figure 3, after epinephrine (adrenaline, a catecholamine) given at 0.5microgm/kg/IV (the anesthetized animal feels no pain and is not agitated so the clinical scenario of a human being feeling pain in the field is replicated by administration of a catecholamine IV), the capture ratio spontaneously decreases from 3:1 to 2:1 (rate about 550 BPM) that results in VF. Importantly, in that figure, the VF starts **more than 20 seconds after the beginning of the TASER ECD shock**, clearly in contrast to the statements by Kroll et al. In addition, the TASER ECD shock initiated rapid VT, **which degenerated to VF about 7 seconds after the TASER ECD shock was stopped**. Thus, VF can occur many seconds after the beginning of the TASER ECD shock and even after cessation of the TASER ECD shock, if the latter has induced VT. Here, that helps explain any breathing by Dr. Rich shortly after the final ECD discharge.

48) A final important point to emphasize is that the TASER ECD shock can initially induce polymorphic ventricular tachycardia (VT) that can gradually transform to VF, as seen in fig 3. Depending on its rate, it is possible for the polymorphic VT to generate some organized cardiac contractions that can maintain some blood flow (and a palpable pulse) for a variable period of time before total collapse of the circulation. That too helps explain any apparent breathing of Dr. Rich after the final ECD discharge. Moreover, continued or repeated application of the TASER ECD

shock during the polymorphic VT can increase the ventricular disorganization and help transform the VT into VF.

49) A fundamental problem with the conclusions from the paper by Kroll et al is that they fail to account for the complexities and variables of human heart function. We start here in Ryan Rich with a cardiac collapse that is virtually simultaneous with the electrical discharge in an individual with no known heart disease. The mechanisms for the ECD shocks to have caused this cardiac arrest are well established in the medical literature, and there is no other plausible explanation for the cardiac arrest at that moment. Without an actual ECG recording, there may be some question about the precise time and manner in which Ryan Rich's heart rhythm went from a normal sinus rhythm to deadly VF under the effect of the ECD shocks of January 4, 2008; however, there is no doubt that is what occurred.

50) Therefore, to a high degree of medical certainty, the shocks from the TASER X26 through these mechanisms caused Ryan Rich's heart to go into VF, either directly or through a transformation of VT into VF, and therefore caused the tragic, untimely death of this 33-year-old physician on January 4, 2008.

TASER ECDs

51) TASER's documents and the peer reviewed literature indicate that ECD technology was developed in the 1970s as a law enforcement tool. The original systems were lower power, generally around seven watts. They were nevertheless associated with a number of in-custody deaths according to a retrospective study by

Kornblum (cited in the addendum). The mechanisms of death in those cases were unclear, but at least one was attributed in part to the ECD current.

52) According to "Medical Safety Information" issued in approximately 2000 with the initial sales of TASER's first high-power ECD, the 26-watt Model M26 ADVANCED TASER, TASER CEO Rick Smith developed a stronger current to replace the 7-watt system by shocking an anesthetized animal with increasing power, until at four times the previous level "the muscles of the body went into a complete, uncontrollable contraction."

53) The "Medical Safety Information" document describes TASER's pre-release animal testing as follows:

> "During these tests, two leading experts in cardiac safety tested the ADVANCED TASER under extreme circumstances to evaluate if the system could pose a medical threat. Under none of the applications simulating potential real world use of the weapon was a dangerous interaction found. Not only did the researchers test the ADVANCED TASER by placing the probes on the surface of the chest in the locations which are known to have the greatest probability of cardiac interference, they used hypodermic needles inserted into the chest to directly stimulate the surface of the heart. They used drugs such as epinephrine, Ketamine, and isoproterenol to see if the ADVANCED TASER would have an effect on a person under the influence of drugs

known to sensitize the heart to stimulation. They even simultaneously applied the shock from two ADVANCED TASERS (over 52 Watts of power) directly to the chest regions where the cardiac affect [*sic*] would be greatest. Even under these extreme circumstances, they were unable to cause a dangerous cardiac fibrillation. Over the course of three days of testing, in 192 discharges of the ADVANCED TASER, these researchers administered over 14,000 of the 26 Watt ADVANCED TASER Wave pulses to five animals all of which are significantly smaller (and hence more susceptible to electrical fibrillation) than humans. **Two leading experts in cardiac safety, purposefully attempting to cause fibrillation by using drugs, implanted needles to the surface of the heart, and even simultaneously applying two ADVANCED TASERS to the chest were unable to cause fibrillation with the ADVANCED TASER.**"

(Bold in original.) No such studies were peer-reviewed and published at that time, however. (The first peer-reviewed "ADVANCED TASER" (26-Watt) study would not be published until five years later.) Moreover, these claims were contradicted by later independent studies demonstrating consistent cardiac capture and episodic fibrillation in test animals, some of which were published prior to the Ryan Rich incident in this report.

54) TASER'S initial animal studies to determine the safety of its higher

power ECDs were inadequate. The design of the Model M26 waveform, according to Dr. Stratbucker's deposition taken June 10, 2010, was based on the concept that a short pulse or train of short pulses, despite high amplitude, cannot capture the heart because of the long cardiac refractory period, and the electrical energy transmitted to the heart is therefore safe. Apparently, on January 11, 1996, he used a custom device on one anesthetized 18.2 kg Hampshire shoat pig more than 48 times to establish safety and efficacy of custom designed ECD delivering current via darts in the skin at the suprasternal notch and umbilicus. The pig was not intubated and he did not monitor metabolic response, blood gases, etc. He only studied strain gauges for muscle responses in the extremities. He allowed the pig to wake up, recover, and the pig was re-tested a few days later. He states that the pig did not show any cardiac ectopy (i.e., premature beats) or myocardial injury. The experiments resulted in increases in the capacitor that caused more muscle disruption and culminated in electrical characteristics of the waveform for the M26 in beginning of 1998, which went to market late 1999. VF never occurred in the one pig, and capture was not measured. Importantly, Dr. Stratbucker tested an M26 strength current at only 2-2 ½ pulses/sec (pps; 400 msec intervals) with a duration of 13 microseconds. In fact, the M26 delivers 15-19 pps (66.7-52.6 msec intervals) of 40 microseconds duration with peak current of 15-17 amps.

55) Later, Dr. Stratbucker produced VF in one dog using an off-the-shelf Model M26 TASER delivering the stimuli over a catheter in the heart. This result,

which contradicted his hypothesis that the ECD cannot capture the heart because of the long cardiac refractory period, was never reported.

56) These results are troubling from several points. First, studying one pig shocked 48 times is very different than studying 48 pigs shocked one time, since that one pig may not be representative of all pigs, and other pigs may not have been equally resistant to the shocks. Second, the electrical characteristics of the device, particularly in terms of pulses per second and duration, were not similar to the actual M26. Finally, not reporting that one dog developed VF when exposed to the Model M26 discharge, albeit via a catheter in the heart, was unacceptable because it showed that under certain circumstances the Model M26 can cause VF in a mammalian heart. This is especially true because that finding squarely contradicts the statement in the "Medical Information" at page 9 that "the short pulse duration of stun guns have very little effect on heart operation which uses much longer electrical pulses." Regardless, the statement that trains of short duration pulses would not cause VF is erroneous.

57) Dr. Stratbucker et al. performed tests with a device capable of increased current he called a "Super-TASER." (PACE 2005; Suppl 1:S284) Rick Smith refers to the devise as a "scalable TASER," a more accurate description. 9 pigs were subjected to five second discharges. The results suggested a safety index for stored charge ranging from 15X to 42X as weight increased from 30 to 117Kg. He stated that the typical TASER product therefore had a safety margin of at least 100 times and that the short pulse has little effects on the heart. In a January 6, 2000 letter Dr.

Stratbucker stated he did studies with Dr. McDaniel on an off-the-shelf M26 after the M26 was being marketed. He attached TASER darts to 5 dogs and fired the M26 for 10 seconds, he thinks. This was published as an abstract (ref 9 in the PACE article). He states that these devices do not cause serious cardiac rhythm abnormalities in the otherwise normal heart.

58) Dr. Stratbucker states in his deposition of June 10, 2010, that the observation of TASER capture in the Lakkireddy study is a "trivial artifact" and that "everybody's known since time began that you can put a hundred amps in there on a defibrillator pulse and – and – and not make somebody fibrillate. You get plenty of capture, but – but it doesn't fibrillate you." For reasons I explain above, this is an erroneous conclusion.

59) The TASER "Medical Safety Information" document also refers to "human subject studies," but these are simply anecdotal accounts of various volunteers being subjected to short duration (.5- to a few full 5-second) exposures, many through alligator clips and other forms of electrodes. There was no physiological monitoring or data reported. Referring to these as "human subject studies" is highly misleading.

60) In 2003 TASER developed the X26, which is smaller and lighter than the M26. While similar in most other respects, the X26 uses less power and pulses a lower current (3-5 amp versus 15-17 amp) but longer (100 microseconds versus 40 microseconds) "waveform." The total charge delivered per pulse is about the same,

100 microculombs. This is the device that was used on Ryan Rich, January 4, 2008.

61) The Model X26 "Operating Manual" makes the following representations regarding safety:

(a) "Aim for the center of the back or the chest of the subject."

(b) "In animal testing, the X26 was found to have a safety margin of 20 times (the X26 was 1/20th of the danger level)."

(c) "The TASER X26 was tested extensively on both animals and human volunteers and has been found to cause no dangerous cardiac or other effects.

(d) "Further, the TASER output will not damage an implanted pacemaker." Pacemakers are designed to withstand the pulses of electrical defibrillators hundreds of times stronger than TASER pulses."

23) In its original owner's manual and in the training versions I reviewed, TASER relied on research performed for the M26 to apply to the X26 as well. Independent testing, however, revealed that the X26's longer waveform translates into a higher capacity for cardiac capture than the M26. (Nanthankumar, 2006).

Police Department Training

62) Training Version 10 (issued June 2003) and Version 11 (issued January 2004) contain PowerPoint presentations for the instructor courses which made the following representations regarding cardiac safety:

(a) Slide 19 (Ver. 10) and Slide 21 (Ver. 11) state "It's not the volts, it's the amps that are dangerous," and then represents the devices to be "Low

Amperage," stating "Both the M26 and X26 are less than 0.004 amps (very low amperage)." "High Voltage + High Power + Low Amperage = Safe & Effective weapon." In fact the peak amperage (a relevant measure for cardiac safety) of the M26 is 15-17 amps, and the peak amperage of the X26 is 3-5 amps. The amount of total current delivered is similar because the X26 pulse lasts longer. (See Braidwood Testimony of J. Patrick Reilly, May 5, 2008).

(b) Slide 22 (Ver. 10) states "TASER tests have found: - No effect on heart rhythms" when "tested on animals."

(c) Slide 23 (Ver. 10) states "Heart rate unchanged during TASER X26 stimulation directly through [the] chest, across the heart." The "Instructor's Note" states that the representation is based on "a blood pressure reading from an anesthetized pig. The X26 was applied across the chest with the two probes in a 'worst case' scenario (the points most likely to stimulate the heart). Note that the heart beat continues normally. The small fluctuations in blood pressure are the result of skeletal muscle contractions that add fluctuations to blood pressure. It is important to note that the heart rate does not change at all. This is important because it shows that the level of the X26 stimulation is below the threshold to pace the heart."

(d) Slide 25 (Ver. 10) and Slide 31 (Ver. 11) state, "Using 'worst case' scenarios, two cardiac safety experts found no interference by the M26 with the heart rhythms."

(e) Slide 29 (Ver. 11) states, "Extensive animal testing has shown no effect on heart rhythms or blood pressure."

Instructing police officers with such information presents the conclusion that TASER ECD administration cannot cause cardiac arrest. The Ryan Rich case, among other incidents and studies – some of which occurred prior to the Ryan Rich incident – show that not to be the case.

63) Further, the TASER "Command Demonstration" PowerPoint Version 12 (released November 2004) included the following representations relating to cardiac safety:

(a) Slide 3 states "X/M26 will not cause heart or pacemaker failure."

(b) Slide 14 states, "Extensive animal testing has shown effect on heart rhythms or blood pressure to be insignificant."

(c) Slide 18 states, "The ADVANCED TASER M26 was applied directly to the chest of experimental animals without causing heart failure during tests at the University of Missouri," "Using 'worst case' scenarios, cardiac safety experts found no induction by the M26 weapon of abnormal heart rhythms," and "No arrhythmia provocation occurred even when the animals were given the stimulant drugs epinephrine and isoproterenol, agents that make the heart more susceptible to electrical stimulation."

64) Version 13 (released May 1, 2006), which Trooper Lazoff was most likely trained on, contains the following representations in its "X26 User's Course":

(a) Slide 18 states, "Low average current: M26 & X26 < 0.004 A."

(b) Slide 21 states, "TASER devices operate at low average currents (0.0021 - 0.0036 A)." For the reasons stated above, these numbers are very misleading as the peak current is the relevant measure for cardiac safety, and the peaks range from 3 to 17 amps. (See Braidwood Testimony of J. Patrick Reilly, May 5, 2008)

(c) Slide 26 states, "TASER Devices are among the most extensively studied non-lethal weapons," clearly implying that the devices have been determined to be cardiac safe. At the time, however, there were only a few peer-reviewed publications available (including a case report published in the *New England Journal of Medicine* warning that the product appeared to have caused ventricular fibrillation in a 14-year-old youth), and several warned about cardiac risks, two specifying increased risk of cardiac capture specifically from shots to the chest. TASER's own study had warned that chest shots should be avoided. Yet the Version 13 User Course illustrated shots to the chest, Slides 11 and 68, and instructed in Slide 67 to "Aim like a standard firearm at center of mass" without warning that chest shots significantly increase the risk of cardiac arrest.

(d) Slide 29 states, "Animal testing has shown insignificant effects on heart rhythms or blood pressure."

(e) Slide 30 states, "The ADVANCED TASER M26 was applied directly to the chest of experimental animals without causing heart failure during tests at

the University of Missouri," and "Using "worst case" scenarios, cardiac safety experts found no induction by the M26 of abnormal heart rhythms." These were based on a TASER funded study, but by the time Version 13 was released, a more recent TASER-funded study had demonstrated that the X26 captured heart rhythms when probes were stuck into the chests of test animals, and an electrophysiologist involved in that study had warned TASER that chest shots should be avoided.

65) TASER Training Version 14, issued during 2007, which Trooper Lazoff also was trained on, is very similar to Version 13, and has misrepresentations about the power of the TASER current, for example Slide 33 claiming it to be many times smaller than the amount of current required to power a Christmas tree light, when in fact it is many times higher. Slide 33 states that "Animal testing has shown insignificant effects on heart rhythms or blood pressure," when in fact TASER had the results of the study by Nanthakumar, et al., showing the induction of ventricular fibrillation. Slide 35 refers to test results from the Model M26 that "No arrhythmia provocation occurred even when animals were given stimulant drugs epinephrine and isoproterenol, which make the heart more susceptible to electrical stimulation." At the time TASER had test results from Nanthakumar, et al., and Dennis, et al., demonstrating the induction of VF with off the shelf Model X26s. Slide 144 instructs users to aim at "center of mass," in other words, the chest, slide 145 illustrates the whole body as "effective target zones," and Slide 147 instructs to aim at the "open

front of unzipped jacket." Because he was TASER certified instructor, I understand that Trooper Lazoff would be familiar with Versions 13 and 14, and would therefore believe based on TASER's instructions that TASER ECDs posed no increased risk of cardiac arrest when shot directly into the chest of individuals. As I explain, however, this belief is not supported by the medical and scientific evidence available to TASER at the time it issued Training Versions 13 and 14.

Documented Risks of TASER ECDs Prior to the Rich Incident

66) I have not seen evidence that was peer-reviewed and published prior to 2005 to substantiate TASER's representations of cardiac safety, and based on my knowledge of electrophysiology these representations are inconsistent with what one might expect when electric shocks of the magnitude delivered by TASER ECDs are delivered over darts to the chest. Starting in 2005, however, documentation of the TASER ECD's cardiac risks began appearing in reputable medical journals.

67) Kim and Franklin reported in *The New England Journal of Medicine* (353:958, September 1, 2005), Ventricular Fibrillation After Stun Gun Discharge, that an adolescent subdued with a TASER ECD collapsed. "Paramedics found the adolescent to be in ventricular fibrillation" and began performing cardiopulmonary resuscitation within two minutes after the collapse. Four shocks and drug administration restored a perfusing rhythm and the adolescent made a nearly complete recovery, discharged from the hospital several days later. (The authors published the electrocardiogram showing ventricular fibrillation being terminated after

a 360 joule defibrillation shock. Although that happened, the strip published was not the final shock, but one depicting an earlier shock converting VF into an idioventricular rhythm. That mix-up has no effect on the conclusions.) TASER criticized this publication, claiming in an open letter (around October 2005) signed by Drs. Rick Luceri, Hugh Calkins, and Mark Kroll, and a published letter to the editor of the J Amer Coll Cardiol (49:732, 2007), that the paramedics did not in fact find the adolescent collapsed or in ventricular fibrillation, as reported, but that the collapse occurred in an ambulance some 23 minutes after the TASER administration. Deposition testimony by a paramedic on the scene, Jill Hutchinson, however, substantiates the conclusions of the article. Specifically, the 14-year-old individual immediately lost consciousness at the time of ECD shock, initially had a pulse and appeared to be breathing, but two minutes after collapse had an ECG recorded by the paramedics that showed VF, thus refuting the arguments made by TASER. In his deposition, Dr. Kroll blames the difference between the time recorded by the TASER and actual time for the mistake. The ECGs published in the NEJM were the first two recorded, showing VF and termination of VF by a 360 J shock (cropped, and actually 200 J). The VF recurred and required three further defibrillations and the young boy required multiple medications to be resuscitated. Fellow TASER Scientific and Medical Advisory Board member Dr. Charles Swerdlow refused to sign the letter, according to the deposition of Dr. Kroll September 29, 2010, because he was not being paid a high enough retainer to allow TASER to publically use his name. Dr.

Swerdlow, when I told him of that statement, disagreed with Dr. Kroll's explanation (see attached email, Exhibit F).

68) TASER funded a pig study by Lakkireddy, et al., accepted March 20, 2006, entitled Effects of Cocaine Intoxication on the Threshold for Stun Gun Induction of Ventricular Fibrillation (*Journal of the American College of Cardiology* Vol. 48, No. 4, 2006). Dr. Tchou participated in this study, and it is referenced in Exhibit E, our email exchange. Darts were placed in various positions on anesthetized pigs and five-second shocks were delivered using a "Super-TASER" modified to deliver both a standard Model X26 charge as well as increased charges. The study described the relationship between cardiac capture and the location of the darts on the chest, describing darts to the chest as more likely to cause capture, with one dart at the sternal notch and the other on the left side at the "point of maximum cardiac impulse (PMI)," referred to in the study as "Position A," being most likely of all the positions tested to capture the heart.

69) The authors found that standard X26 discharges at "Position A" resulted in "ventricular capture ratios ranging from 6:1 through 3:1," and that "VF was consistently inducible whenever the ventricular capture ratio was [less than or equal to] 2:1. The authors write that their "study is the first to describe capture of ventricular myocardium during application of [ECD] pulses." The authors state the following in their discussion:

"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 would suggest that [ECD] 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 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."

70) Although TASER funded this study and therefore must have known of this finding in early 2006, at the latest, (one participant, Dr. Tchou, told me: "From the very beginning, as we obtained our data from our animal study, I had advised TASER that the possibility of inducing ventricular arrhythmias is there." Exhibit E), TASER took no steps to warn its users that avoiding shots to the chest "would greatly

reduce any concern for induction of ventricular arrhythmias." In fact, no such warning was issued by TASER until September 30, 2009, 21 months after the incident involving Ryan Rich. Instead, TASER continued to misrepresent the cardiac safety of its products in its training and promotional literature.

71) As noted above, Ryan Rich had one dart embedded just above and one dart just below the left nipple, thus encompassing the heart in an electrical vector similar to the ideal position established by the Lakkireddy study. There are three important limitations to the Lakkireddy study, which suggest that the study may have understated the cardiac risks to human beings. First, the test animals were anesthetized, which as I mentioned above can suppress development of arrhythmias. Second, also because the test animals were anesthetized, they did not experience the pain felt by human subjects when shocked by X26s. Pain causes the body to release catecholamines (substances like adrenaline) and, as noted above, create an adrenergic state and stimulate the heart, making it more subject to arrhythmia. Third, shocks were administered for only one five-second cycle, yet the X26 is designed to deliver multiple and prolonged cycles. The probability of capture degenerating into VT or VF increases with increased duration of capture time. As mentioned above, Dr. Rich received multiple cycles over a 3-minute period.

72) An animal study by Nanthakumar, et al. (*Journal of American College of Cardiology*, accepted February 7, 2006), Cardiac Electrophysiological Consequences of Neuromuscular Incapacitating Device Discharges, reported on 150 discharges in six

pigs using both the Model X26 and the Model M26. The results were first reported as a poster abstract on May 19, 2006 at the Heart Rhythm Society meeting in Boston (Heart Rhythm vol 3: (1S) p. S237, 2006). The first copies of the abstract article were mailed early that May. Dr. Nanthakumar told me that Dr. Kroll, the head of TASER's Scientific and Medical Advisory Board, attended the presentation and spoke against it, much as he did with me following my presentation three years later. This appears to me, however, to be a thorough and well designed study by independent researchers, and its findings are entitled to great weight.

73) The authors note that, while surface ECG monitoring has been done in healthy human volunteers before and after delivery of energy from ECDs, there had been no intracardiac monitoring to eliminate electromagnetic interference produced by the ECD, causing electrical artifact to be recorded by the ECG. Such artifact would make interpretation of true cardiac activity difficult or impossible from a standard ECG. They used an intracardiac recording (electrode recording from inside a chamber of the heart) approach in this study, a technique too invasive to use on human volunteer test subjects.

74) Out of 41 Model M26 discharges delivered to the chest with an interdart distance of 26-30 cm, 22, or 53.66% resulted in stimulation of the myocardium (cardiac capture). All but one of 53 Model X26 discharges (98.11%) did so, suggesting that the longer Model X26 waveform is significantly more likely to result in cardiac capture, even at about one-fourth the peak amperage of the M26. None of 56

non-thoracic discharges from either device stimulated the myocardium, again demonstrating the high correlation between darts in the chest, close to the heart, and potential arrhythmias.

75) The cardiac capture from electrical stimulation at high rates persisted during the discharge, and as soon as the discharge ceased there was resumption of normal electrical rhythm. Importantly, however, the blood pressure fell to very low values during the rapid stimulation. During epinephrine (adrenaline) infusion (a catecholamine) to simulate the agitated stress state of an individual experiencing pain or resisting restraint, one ECD administration resulted in non-sustained ventricular tachycardia that spontaneously reverted to sinus rhythm, while another produced ventricular fibrillation and cardiac arrest. These findings demonstrate that the possibility exists of TASER ECDs – the Model X26 more than the Model M26 – inducing serious ventricular arrhythmias during discharge in structurally normal hearts, especially during the intense catecholamine release that accompanies the stress of the situation and the pain of the ECD discharge.

76) The authors designed the study to portray the worst case scenario and noted that general anesthesia – a requirement for the humane treatment of test animals – may have increased the threshold for arrhythmia induction, in other words made it more difficult to induce an arrhythmia. While objections have been raised about using the pig as an animal model, in the Lakkireddy study noted above and financed by TASER, they state, "However, pigs frequently have been used in

fibrillation and defibrillation threshold studies with the results generalized to humans." It is important to note that all the pre-release TASER ECD testing that could be remotely considered "scientific" rather than anecdotal was performed on pigs and dogs, rather than human beings.

77) The results of this study also support my opinion that VF can occur at any time during a TASER shock that captures the myocardium, as elaborated above.

78) I have been provided a written Version 13 certification test and answer key often administered to officers for TASER certification. The test was written after TASER knew the results of the Lakkireddy and Nanthakumar studies, almost two years before Ryan Rich's death. The supposedly correct answer given for Question 24 is that the TASER X26 affects the "sensory and motor nervous systems," but not the "cardiac system." For Question No. 1, "When deploying probes, the TASER should generally be aimed at?" the answer given is "center of body mass," in other words the chest. Yet TASER knew at that time Model X26 administrations near the heart risked cardiac capture and therefore cardiac arrest.

79) Additional animal studies continued to confirm cardiac capture and incidents of VF. Of particular interest were a series of tests performed by independent researchers at a Chicago institution. Dennis, et al (*Journal of Trauma* 2007; 63:581) Acute Effects of Taser X26 Discharges in a Swine Model), performed a study in which anesthetized pigs were exposed to two 40-second discharges from a TASER Model X26 separated by a 10-second pause. One of the pigs in the test group

developed ventricular tachycardia progressing to ventricular fibrillation after the ECD discharge. Three pigs showed capture with rapid (approximate rate 300/min) contractions seen on echo during the shock, and stopping when the shock stopped. Two pigs in the control group prior to being euthanized underwent a left anterior thoracotomy to view the heart during ECD administration to the chest. They both showed visual cardiac capture by the ECD shock. Immediately after exposure, one pig developed ventricular tachycardia progressing to fatal ventricular fibrillation (a video clip of the heart of the open chest pig clearly shows the onset of ventricular fibrillation). The surviving animals in the test group showed a significant increase in heart rate and significant hypotension. In their second study, published August 2007, the researchers injected test animals with a paralyzing agent and were still able to induce cardiac capture with a standard Model X26 in all eight animals tested, causing one to experience ventricular fibrillation. In their third study, published December 2008, the researchers induced VF in two of four animals with a 10-second Model X26 discharge to a spot on the left upper chest, again near one of Ryan Rich dart wounds. They found the cardiac effects of the Model X26 highly dependent on transcardiac vectors of the probes as well as the configuration of the positive and negative darts.

80) In a study funded by TASER (two of the authors, Dr. Dawes and Dr. Ho, serve as medical consultants to TASER and are stockholders, and Dr. Ho serves as the Medical Director of TASER) Dawes et al (Effect of an Electronic Control Device Exposure *Academic Emergency Medicine* 2010; 17:436-443) studied 16 Dorset

sheep exposed to none or incremental doses of methamphetamine and, 30 min later, to 5, 15, 30, or 40 sec of TASER Model X26 intermittent shocks with darts inserted 9mm deep at the sternal notch and cardiac apex. Cardiac motion was determined by thoracotomy and echocardiography. Certain animals had supraventricular dysrhythmias after ECD exposure and one had 6-8 beat multifocal VT, while the larger animals had only sinus tachycardia. Three of the smaller animals demonstrated cardiac capture during ECD exposure, while two of the larger control animals did also. No animal developed VF. Once again, in a study funded by TASER and conducted by TASER consultants, the Model X26 demonstrated the capability to capture the heart at rapid rates, which, in a field situation, could lead to VF, and certainly did in the Ryan Rich case.

81) In a modeling study (Sun, et al., Estimating the probability that the Taser directly causes human ventricular fibrillation *J Med Engineering and Technology* 2010,1-14), the authors "estimated mean probability of human VF was 0.001 for data from a pig having a chest wall resected to the ribs and 0.000006 for data from a pig with no resection when inserting a blunt probe. The VF probability for a given dart location decreased with the dart-to-heart horizontal distance (radius) on the skin surface." While these data are interesting, there is no way a modeling study can replicate the clinical scenario that occurred to Ryan Rich while being shocked for 62 seconds. The authors themselves cite many limitations to their intellectual exercise. And even if that estimate were possible, the first estimate of 0.001 means that one

individual in a thousand would develop VF while being tasered, which may be consistent with what is actually experienced, though numbers of total TASER applications and the incidence of VF, particularly the latter, may not be completely accurate.

82) On September 30, 2009, TASER issued new warnings about the risk of VF and the importance of avoiding shocks to the chest. Recent TASER training materials have cited a risk of ventricular fibrillation of 1:100,000 TASER ECD applications. In fact, if one considers only TASER ECD applications where the darts were impaled in the anterior chest, since darts in other positions would not be expected to produce VF, the odds of a TASER shock inducing VF with that dart configuration are probably significantly higher, as Dr. Kroll testified at his deposition.

83) There have been some human tests, mostly TASER funded, and I do not cite these. They do not, in my professional opinion, eliminate the concerns raised by the animal studies. The basic limitation with the human studies is ethical. Any human testing must be designed with safety parameters to avoid VF induction, which eliminates the sort of testing done on pigs, where fibrillation thresholds can be determined. Moreover, human testing on volunteers cannot replicate the "real life" situation experienced by individuals involuntarily receiving repeated Model X26 shocks in the chaos of a field setting.

84) A recent TASER-funded human study (with two authors, Drs. Ho and Dawes, serving as expert medical consultants to TASER, International for public

speaking purposes and litigation involving corporate product, and who own personal shares of stock in the company), by Ho, et al. (Human Cardiovascular Effects of a New Conducted Electrical Weapon *Forensic Science International* (2010, doi:10.1016/j.forsciint.2010.05.003) on normal human volunteers tested a new generation TASER ECD with different circuit and multiple cartridges that can be discharged simultaneously. They used echo monitoring and demonstrated "an apparent brief episode of cardiac capture" at a rate of 240 beats/min during the 10 second TASER ECD shock. It was "assumed to be electrical capture by the device," according to the authors, thus substantiating the capability of TASER-induced cardiac capture, albeit with a newer device, **in humans.** One probe was in the center of the chest and the second on the right groin area. The study was stopped and a re-designed TASER ECD was allegedly substituted, which showed sinus rhythm in 27 of 42 subjects, while "the rhythms of the remaining subjects were unable to be determined due to subject movement during exposure, however, the maximal rate in any of the subjects exposed to the (new) device was 162 bpm." Naturally, that does NOT exclude TASER-induced capture at a rate slower than 240/min. While neither of these devices was used on Ryan Rich, the importance of this study is that, even with the new (presumably, improved) TASER ECD, cardiac capture was verified by experts in TASER's own company. If it is true that, "The cardiac safety profile of the NGCEW (new electronic control) device appears similar to previous CEW devices when used in multiple probe application formats as intended," this is hardly

reassurance that TASER ECDs do not produce VF.

85) However, there are human data that support the conclusion that TASER ECDs cause cardiac capture and even VF. Cao, et al *(Journal of Cardiovascular Electrophysiology* 18:876, 2007), published "Taser-Induced Rapid Ventricular Myocardial Capture Demonstrated by Pacemaker Intracardiac Electrograms." This is a very important case report of a 53-year old male with a dual-chamber pacemaker implanted subcutaneously beneath the left clavicle (Medtronic Kappa) who received ECD shocks while in a prison, with two barbs delivered with a Model X26. The man was struck on the right chest and did not suffer any immediate observable adverse events. During pacemaker evaluation, however, there were two ventricular high rate episodes that corresponded to the exact time of the Model X26 shocks. I am aware that the pacemaker leads may have provided a pathway for the Model X26 shocks to reach the heart, but nevertheless, the study shows clear cardiac capture from Model X26 shocks in the field.

Selected Clinical Cases of TASER Related Sudden Cardiac Death

86) I personally examined the case history of Greshmond Gray, a 25-year old male who was shocked three times – sixteen seconds, five seconds and five seconds – in La Grange, Georgia, on November 2, 2004, by a TASER ECD. He became non-responsive coincident with the TASER ECD application. An ambulance was called and CPR initiated. There was an automatic external defibrillator (AED) available. Prompt application of the AED showed an initial rhythm of ventricular

fibrillation. 200 joules were delivered followed by asystole and then a wide-complex rhythm followed by redevelopment of ventricular fibrillation. A second AED shock of 200 joules was followed by asystole and another wide-complex rhythm. Mr. Gray could not be resuscitated. The officers testified that it was a very short period of time between the last ECD shock, recognizing that Mr. Gray was unresponsive, obtaining an AED from the police car, and giving the first AED shock. One officer believes this occurred in minutes and certainly less than five minutes. The AED was analyzing for a second shock when the EMTs first arrived.

87) The autopsy indicated that Mr. Gray had two puncture sites on the medial left upper chest at the base of the neck and midline lower chest/upper abdomen, similar to the ideal "Position A" noted in the Lakkireddy study. Heart weight was 410 grams (normal) with normal coronary arteries, normal cardiac chambers and normal cardiac valves. Toxicology found marijuana metabolite in the blood and 0.145 percent blood alcohol. I concluded to a reasonable degree of medical certainty that because Mr. Gray developed ventricular fibrillation closely following the ECD administrations directly to the chest, and there was no other explanation for the cardiac arrest at that moment of TASER ECD application, the TASER ECD caused his sudden death due to ventricular fibrillation.

88) I used this case to support my opinion in *Williams v. TASER International, Inc.* Charles D. Swerdlow, M.D., a colleague who was a defense expert for TASER at the time as well as a member of TASER's Scientific and Medical Advisory Board

(now no longer consulting for TASER to my understanding), subsequently presented on this case, along with others, at our 2009 Heart Rhythm Society meeting in Boston, and then published "Presenting Rhythm in Sudden Deaths Temporally Proximate to Discharge of TASER Conducted Electrical Weapons" (May 2009). Although he did not identify the case by name, he described the Greshmond Gray case as follows: "For subject 1, who collapsed immediately . . . , neither drugs nor cardiac disease can be implicated; both the time course and the electrode location are consistent with electrically induced VF," and "To the best of our knowledge, this is the first reported fatality suggestive of [ECD]- induced VF."

89) Additional cases exist which demonstrate that shocks to the chest by a TASER ECD can cause sudden death due to ventricular fibrillation. The case of *Butler v. TASER International, Inc.*, for which I gave a deposition on February 22, 2009, involved a 48-year-old man who received 3 shocks from a Model X26 TASER for 5, 8, and 5 seconds over about 30 seconds, and became limp without pulse or respirations. An ECG recorded VF shortly thereafter. He was resuscitated but with brain damage. Darryl Turner was a 17-year old who, on March 20, 2008, received a 37-second shock from a TASER X-26 directly to his chest that produced simultaneous cardiac arrest and death. I prepared an expert report very similar to this one and gave a deposition in that matter as well. Robert Mitchell was a 16-year-old boy who received a single TASER shock in the chest, collapsed more or less immediately and presented with VF, according to the reports available to me. I have seen the autopsy reports and EMS records, including rhythm strips, for this incident. While the autopsy indicates he had arrhythmogenic right ventricular cardiomyopathy, if indeed he did have that, it does not exclude a TASER ECD shock from inducing VT-VF. The ECG strips during resuscitation show an irregular VT. He was not resuscitated and died. Rory McKenzie was a 25-year-old male who collapsed about 2 min after receiving TASER shocks from two TASERS simultaneously, was found to be in VF 10-12 minutes later and could not be resuscitated. Autopsy showed no significant heart disease. Derek Jones was a 20 year-old male who collapsed due to VF immediately after a TASER shock to his chest and could not be defibrillated. He

had a normal heart at autopsy. Toxicology showed ethanol 0.22%, THC carboxylic acid, but no other drugs. Additional cases no doubt exist, but to my knowledge havenot been systematically collected and analyzed.

Statement of Opinions

90) I was asked to evaluate the available records and information concerning the cardiac arrest of Ryan Rich following thirteen cycles of TASER Model X26 electronic control device shocks for a total of 62 seconds in about 3 minutes, to determine whether such data were sufficient to make a finding of causality, whether TASER provided appropriate warnings to law enforcement about the cardiac risks of the product, and whether TASER's representations about its product's safety were truthful in light of the known or knowable scientific evidence. My opinions stated above and that follow are expressed to a reasonable degree of medical certainty, based on my education, clinical practice, research, training, experience, literature review, document review, and generally accepted principles of medicine and clinical science.

91) A TASER Model X26 discharge can cause cardiac arrest by capturing the cardiac rhythm at very rapid rates and precipitating ventricular tachycardia or ventricular fibrillation, as shown in animal testing and human reports. The temporal relationship of Ryan Rich's collapse to the Model X26 shocks in the absence of any equally plausible alternative explanation for his heart to develop cardiac arrest at that precise moment demonstrates that the ECD's electrical current directly caused Dr. Rich's cardiac arrest. His only health problem was epilepsy; there was no family

history of heart disease, nor were there confounding issues such as drugs. I do not think he had an enlarged heart, based on the data from the Mayo Clinic paper and his body weight of 220 pounds. The LV wall measurement is probably in error and included papillary muscle. It is therefore my opinion to a high degree of medical certainty that Ryan Rich developed ventricular fibrillation as a result of the X26 shocks that he received, which led directly to his death, and that there was no other cause of death.

92) Because the TASER barbs remained attached to his chest after the initial ECD applications in the probe mode (see paragraph 34 above) and the TASER cartridge remained in place, according to the statement given by Trooper Lazoff, current delivered during the drive stun mode also was distributed to his heart. While the exact amount of current obviously was not measured, and some may have "leaked" off, particularly during the first drive-stun application to his legs, the almost "perfect" probe position for cardiac capture on Dr. Rich's chest, based on the animal work, may have facilitated pacing of the heart even with somewhat reduced current during the drive stun applications

93) In my opinion, the cardiac arrest began when Ryan Rich no longer resisted handcuffing. The trooper and physician left Dr. Rich to walk to the trooper's vehicle at that time. I find it hard to believe Dr. Morris's statements that Dr. Rich's breathing was normal at that time, i.e., not even accelerated due to the fighting. In fact, "normal breathing" under such conditions would itself be abnormal. I suspect Dr. Morris did

not take accurate notice, since he believed there was no reason to do so. Further, if Dr. Rich was breathing, it may have appeared more or less "normal" in the early throes of a ventricular tachycardia. The lethal rhythm can remain somewhat organized and produce a blood pressure and pulse for several seconds early on, as I have explained above (see paragraphs 45-48), so there may have been no noticeable respiratory effects while they were taking leave of him. The 20-30 second walk to the trooper's vehicle would be the minimum length of time for someone in cardiac arrest to become obviously cyanotic, which is around when Dr. Morris turns and notices this change in Dr. Rich's appearance.

94) While Ryan Rich apparently did have epilepsy, and may have been in a post ictal state from a seizure at the time of his erratic driving, that event played no direct role in causing his death, as I understand Dr. Engel explains in his report. It is just as likely that the gabapentin at high doses contributed to a confusional state and the erratic driving, but as with the epilepsy, played no direct role in causing death.

95) Alternative causes of Ryan Rich's cardiac arrest are excluded, including excited delirium and sudden unexplained death in epilepsy (SUDEP). The former has been reported as death associated with an extremely agitated and irrational state, usually compounded by physical restraint. Many of the individuals dying with this alleged diagnosis have taken stimulant drugs such as PCP, methamphetamine and cocaine, or suffered from severe mental illness, and were restrained with hands bound behind them, legs shackled, and held down on the floor in a prone position (on their

chests). Drug toxicity and/or postural hypoxia/anoxia have been appropriately suggested as contributing to death in many of these individuals. The elevated catecholamines resulting from such restraint and attempts to break free facilitate the ability of a TASER shock, such as that from an ECD, to precipitate ventricular tachycardia or fibrillation, as found by Nanthakumar (above). Ryan Rich did not exhibit these characteristics, and cannot be said to have been suffering from "excited delirium." The diagnosis of "excited delirium" is not recognized by the American Medical Association as a medical or psychiatric condition but is recognized by National Association of Medical Examiners. It is possible that "excited delirium" is a form of takotsubo syndrome (Wittstein et al: Neurohumoral features of myocardial stunning due to sudden emotional stress. *N Engl J Med.* 2005 Feb 10;352(6):539-48), which might be a cause of some in-custody deaths. However, it would be extremely unlikely for Ryan Rich's cardiac arrest to occur virtually immediately during/following the X26 shock and be due to takotsubo syndrome.

96) SUDEP usually occurs during sleep or rest, is often associated with hypoventilation, and excessive seizure activity (Langan J Neurol Neurosurg Psychiatry 68:211, 2000; Lhatoo Ann Neurol 68:787, 2010; Bateman Epilepsia 51:916, 2010; Nilsson Lancet 13:353, 1999; Opeskin Seizure 12:456, 2003; Tomson Epilepsia 46 suppl 11:54, 2005). Most importantly, Ryan Rich was alive and resisting being handcuffed prior to application of the TASER shocks and long after what might have been a seizure. I understand that Dr. Engel addresses this issue in more detail in his

report.

97) The medical hazard of ECD shocks resulting in cardiac arrest was foreseeable prior to January 2008 and appropriate testing should have been done to investigate this possibility before placing these products on the market. Delivering 1- or 2-second shocks in areas remote from the heart (for example, the back) in humans while recording an ECG recording is totally inappropriate and in no way would exclude the possibility of ECD-induced ventricular tachycardia or ventricular fibrillation from occurring. Even testing 5-,10- or 15- second cycles over the anterior chest of volunteers does not replicate real-life field conditions. At the least, testing should have been done in an appropriate animal model with infused catecholamines to simulate an agitated state, drugs, and various cardiac pathologies such as coronary artery disease and old myocardial infarction. Further, since the devices are designed to deliver repeated and prolonged cycles, such animal testing should have employed multiple, repeated and prolonged shocks. However, noted above, recent testing in humans has demonstrated that a TASER shock can capture the heart at fast heart rates of 240 BPM.

98) It is not open and obvious to the lay or average law enforcement user that such adverse effects as ventricular tachycardia and fibrillation could occur from ECD use. TASER has trained and instructed law enforcement individuals that its device was safe and posed no cardiac risk, even when shot into the chest and cycled multiple times. TASER's representations and claims that are detailed above were not,

and are not, scientifically or medically supported. The representations which TASER made were either false or misleading in that TASER improperly downplayed potential dangers that were not adequately understood and overplayed its safety claims and studies.

99) It was not until September 30, 2009, that TASER first warned law enforcement to avoid shocking individuals like Ryan Rich in the chest. Based on the medical evidence that TASER should have had from the outset, and that it in fact did have no later than the spring of 2006 – almost two years before this incident – this warning should have been provided to law enforcement long before Trooper Lazoff fired darts from his Model X26 into Ryan Rich's chest. I note that Trooper Lazoff testified that now knowing of the cardiac risk he would never use a TASER except in the most extreme circumstances: "I'd have to, you know, really be getting my ass kicked to even, you know, think about it again."

100) Prior to the incident involving Ryan Rich, the failure of TASER to conduct reasonable testing regarding the cardiac risks of its electronic control device, the failure to adequately warn of the known and knowable cardiac risks of the product, and the misrepresentations and misleading statements it made to law enforcement regarding medical and cardiac safety of the device, were actions in conscious disregard of the safety of the persons on whom this device was intended by TASER to be used, and were a substantial factor in the ventricular fibrillation, cardiac arrest and ultimate death which Ryan Rich suffered.

101) This product is not regulated by the FDA or Bureau of Alcohol, Tobacco and Firearms. There is no similar regulatory process to determine the accurate warning regarding known and unknown risks. Accordingly, it is particularly incumbent upon the manufacturer of this device to perform adequate testing and issue appropriate warnings.

I declare under penalty of perjury pursuant to the laws of the United States that the foregoing is true and correct. Executed this 25th day of April 2011, at Carmel, Indiana.

Douglas P. Zipes, M.D