

Report to the Braidwood Commission

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I have been asked by the Braidwood Commissions of Inquiry to determine the cause of Mr. Robert Dziekanski's death and the role of the Taser in his death.

Taser Discharges:

Mr. Dziekanski received the first two 5-second Taser probe discharges over the central anterior chest. The third probe discharge may not have been delivered, as the wires appear to have disconnected when he rolled on the ground during the second discharge. He received an additional two drive-stun discharges for approximately 5-6 and 8-9 seconds.

In the postmortem report by Dr. Charles Lee, a "dark punctuate abrasion on the central chest... consistent with an electrode from a Taser" is evident. Dr. Lee reports further that "the other electrode mark is not apparent, but a couple of punctuate abrasions are present on the chest and abdomen, and one of these might be the other Taser mark." If one of the other punctuate abrasions on the chest found by Dr. Lee represents the second electrode mark, the first two Taser probe discharges were delivered over the cardiac axis with probe penetration; the first of these discharges resulted in cardiac capture and induction of sustained ventricular tachycardia.

The vector of Taser electrodes over the cardiac axis is critical for cardiac capture and induction of sustained ventricular tachycardia in animal studies.[1-3] Furthermore, the electrode-to-heart distance is important for cardiac capture, with penetration of Taser electrodes to an average distance of 6 mm from the heart necessary for induction of malignant ventricular arrhythmias.[4] Although one study of Taser applications in resting human volunteers over the cardiac axis did not demonstrate any ventricular arrhythmias, Taser electrodes in the study were placed manually on the skin surface rather than allowing the electrodes to penetrate the skin as they do in field use.[5] Since the typical skin-to-heart distance in humans is between 10-57 mm,[6] electrode placement on the skin surface would result in an electrode-to-heart distance too great to result in cardiac capture and induction of ventricular arrhythmias. The punctuate abrasions evident on Mr. Dziekanski's anterior chest are consistent with electrode penetration to the distance necessary for cardiac capture.

Simulated stress in experimental models with pharmacologic agents increases vulnerability to malignant ventricular arrhythmias.[1] Thus, the agitated state of stress and high levels of adrenaline during Mr. Dziekanski's encounter further increased his vulnerability to ventricular tachycardia as a result of direct cardiac capture by the Taser.

If the punctuate abrasion over the abdomen was the second electrode mark, the vector was not over the cardiac axis and the Taser discharge did not directly induce ventricular tachycardia by cardiac capture. In this case, the adverse physiologic effects, including stress, pain, and adrenaline surge, resulting from the initial and subsequent Taser discharges triggered a sustained ventricular tachycardia in Mr. Dziekanski.

Alcoholic Cardiomyopathy

Conflicting conclusions exist regarding whether Mr. Dziekanski had alcoholic cardiomyopathy. Dr. Lee found that Mr. Dziekanski's heart mass (370 g) was beyond

normal range with ventricular dilation and microscopic evidence of "mildly increased, patchy interstitial and perivascular fibrosis." In conjunction with cerebellar atrophy and a fatty, inflamed liver, Mr. Dziekanski had findings consistent with alcoholic cardiomyopathy. Myocardial scarring from such a condition places individuals at risk for ventricular tachycardia and ventricular fibrillation. Although Dr. Butt did not find microscopic evidence meeting his criteria for alcoholic cardiomyopathy, perhaps the limited sampling of cardiac sections missed more evident areas. Nevertheless, even in the setting of normal hearts in animal experiments, sustained ventricular tachycardia and fatal ventricular fibrillation were directly induced by Taser discharges.[1-4, 6]

Ventricular Tachycardia Leading to Cardiac Arrest and Death

Mr. Dziekanski died as a result of a fatal arrhythmia in the setting of alcoholic cardiomyopathy which made him more vulnerable to ventricular tachycardia. Thus cardiac capture by the Taser resulted in a sustained ventricular tachycardia even after the Taser discharge. Due to alcoholic cardiomyopathy, his cardiac function was already compromised and ventricular tachycardia eventually resulted in hemodynamic instability and collapse, followed by degeneration to ventricular fibrillation and asystole.

Mr. Dziekanski was observed to struggle for approximately 90 seconds following the first Taser discharge which induced sustained ventricular tachycardia either directly or indirectly depending on the vector. In situations of stress and high levels of circulating adrenaline, subjects are able to maintain an adequate blood pressure for several minutes, even in the setting of ventricular tachycardia and alcoholic cardiomyopathy. However, the low cardiac output due to ventricular tachycardia eventually results in a blood pressure too low to maintain consciousness or blood flow to the brain or vital organs. Thus, at this point Mr. Dziekanski became unconscious and unresponsive and was handcuffed.

Approximately 1 minute later, Mr. Dziekanski was observed to become cyanotic (turn blue) because of the continued low cardiac output during ventricular tachycardia. From this point on, several observers described Mr. Dziekanski to have faint and shallow breathing, consistent with the typical agonal breathing pattern observed just prior to death.

Mr. Enchelmaier, a layperson, assessed Mr. Dziekanski's carotid pulse three times in the period between initial cyanosis and approximately 2 minutes before the arrival of firefighters. Firefighters found no pulse or breathing. During Mr. Enchelmaier's first assessment of carotid pulse, he detected a "strong and fast" pulse. However, Mr. Enchelmaier obviously could not compare the strength of the pulse he felt during this period to Mr. Dziekanski's pulse prior to the encounter; thus, the pulse was very likely weaker than during normal rhythm. Moreover, a study of a blinded anesthesiologist's assessment of the presence of carotid pulse in patients in shock as compared to invasive blood pressure monitoring resulted in systolic blood pressures only between 35 and 55 mmHg.[7] Thus, Mr. Enchelmaier's observations are consistent with the low cardiac output resulting from a rapid ventricular tachycardia.

During the second and third assessments Mr. Enchelmaier describes a slower pulse. Heart failure and ventricular tachycardia are two important causes of pulsus alternans, a condition where electrical rhythm results in alternating strong and weak pulses.[8]

Because Mr. Dziekanski's systolic blood pressure was already very low during ventricular tachycardia (between 35-55 mmHg), Mr. Enchelmaier described a slower pulse because he only felt every other pulse, resulting in a perceived heart rate half of the ventricular tachycardia rate. Since Mr. Enchelmaier did not time the pulse with a watch, it is difficult to assess the exact rate of these pulse assessments. During this time Mr. Enchelmaier describes respirations consistent with agonal breathing.

The low cardiac output due to ventricular tachycardia in the setting of alcoholic cardiomyopathy continues to the point when the heart itself receives too little blood flow, thus ventricular tachycardia degenerates to ventricular fibrillation, which when untreated almost universally results in asystole and death. Mr. Enchelmaier last assessed pulse and agonal breathing 2 minutes before firefighters arrived. Since they found no breathing or pulse, at this point Mr. Dziekanski was either in ventricular fibrillation or asystole. Another 90 seconds elapsed before an AED was connected by ambulance personnel showing asystole. Thus in the span of 3½ minutes, Mr. Dziekanski's rhythm degenerated from ventricular tachycardia to ventricular fibrillation to asystole.

Following this, advanced resuscitative measures were undertaken without success and Mr. Dziekanski was pronounced deceased.

In-Custody Sudden Death

Although there is little doubt Mr. Dziekanski's rhythm was ventricular tachycardia leading to cardiac arrest, if the Taser vector was over the abdomen rather than over the chest, another possible cause of ventricular tachycardia and cardiac arrest is in-custody sudden death, which others have termed sudden death during restraint or "excited delirium." In this case, the Taser discharges were contributory to the in-custody sudden death of Mr. Dziekanski. This is supported by the results of my own research in 50 California cities which demonstrated a 6-fold increase in rates of in-custody sudden death after deployment of the Taser.[9]

Conclusions:

1. Mr. Dziekanski died as a result of hemodynamically unstable ventricular tachycardia leading to cardiac arrest and death by one of three mechanisms:
 - a. Direct induction of hemodynamically unstable ventricular tachycardia by Taser discharge over the anterior chest
 - b. Triggering of hemodynamically unstable ventricular tachycardia by the adverse physiologic effects (stress, pain, and adrenaline surge) of Taser discharge over the chest-abdomen
 - c. Contribution of the adverse physiologic effects resulting from Taser discharges to the syndrome of in-custody sudden death
2. A diagnosis of alcoholic cardiomyopathy which has been made by several forensic pathologists would have made Mr. Dziekanski even more vulnerable to ventricular tachycardia, either by direct induction by Taser discharge or indirectly via the adverse physiologic effects of Taser discharge.

3. A number of scientific and medical studies support the conclusion that the Taser either directly caused or contributed to Mr. Dziekanski's death.
4. Without exposure to Taser discharges, Mr. Dziekanski would very likely not have experienced sudden death.

References

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