Abstract (Summary)

Purpose - Less lethal weapons have become a critical tool for law enforcement when confronting dangerous, combative individuals in the field. The purpose of this paper is to review the medical aspects and implications of three different types of less lethal weapons.  
Design/methodology/approach - The paper conducted a comprehensive medical literature review on blunt projectiles, irritant sprays including oleoresin capsicum (OC), and conducted energy devices such as the Taser(TM). It reviews the history, mechanisms of action, intended and other physiologic effects, and medical safety risks and precautions of these devices. In particular, the paper focuses on the issue of sudden in-custody death and less lethal weapons, reviewing case reports, animal research and human investigative studies on this topic. Findings - In general, these three different types of less lethal weapons have been effective for their intended use. Each type of less lethal weapon has a number of physiologic effects and specific medical issues that must be considered when the weapon is used. There is no clear evidence that these devices are inherently lethal, nor is there good evidence to suggest a causal link between sudden in-custody death and the use of irritant sprays or conducted energy devices. Originality/value - While further research on the physiologic effects of these devices is needed, this paper provides law enforcement with a medical review of less lethal weapons including blunt projectiles, irritant sprays such as OC, and conducted energy devices such as the Taser.
Impact or blunt projectiles include bean bags and rubber bullets. These weapons are less lethal than firearms and allow a safe distance between the officer and subject. Irritant sprays and riot control agents, such as tear gas, mace (CN) and oleoresin capsicum (OC), have been used by law enforcement to facilitate compliance and temporarily incapacitate violent individuals or crowds. CEDs such as the Taser have become increasingly popular in law enforcement as a less lethal technology to temporarily subdue individuals. In this chapter, we review the different types of less lethal weapons, including their history, mechanisms of action, intended and other physiologic effects, and medical safety risks and precautions.

In addition, we review the research regarding the potential association of these devices with sudden in-custody deaths. This report includes a review of case reports, animal research and human research investigating this issue. In general, case reports of sudden deaths associated with these weapons cannot determine specific causality. Findings from animal research are limited in their applicability to humans. Even human studies investigating the physiologic effects of these devices may be limited depending on the specific conditions of the study subjects and how closely field conditions are replicated.

Impact projectiles

Impact projectiles are used as an alternative to standard firearm rounds when trying to disperse a crowd from a distance or subdue a combative, dangerous individual without the use of lethal force. Modern day impact projectile weapons were first used during the Hong Kong Riots of the 1950s and 1960s, utilizing projectiles made of wood. Similar weapons were used during conflicts in the Northern Ireland, Israel and Palestine in the 1970s and 1980s. Early devices included hard rubber missile-shaped projectiles that were difficult to direct, resulting in injuries to the head, face and chest. These projectiles have evolved into PVC-type bullets, modern-day blunt rubber bullets, and bean bag type rounds, which are currently in use by law enforcement agencies.

The action of the blunt impact projectile is to induce pain, irritation and minimal injury to the subject without causing any life-threatening injuries. In general, all involve a blunt type impact that can impart energies on the order of 100-200 Joules depending on the type of round and the distance from firing. The physiologic effects of blunt impact projectiles are directly related to anatomic location where the blunt impact projectile strikes the subject and induces blunt force trauma to the individual.

Case reports, reviews and research studies

The majority of the medical literature on this topic is based on case reports and case series. Both injuries and deaths have been reported with blunt impact projectiles which have caused injury by direct penetration into the body. [72] Wawro and Hardy (2002) and Hardy report of a 56-year-old man who survived after his chest wall was penetrated by bean bag rounds fired from a 12 gauge shell at an unknown distance. Similar cases of intrathoracic bean bag penetration have also been reported by others, including penetration with intact bean bags as well as bean bag pellets when the bag fails ([10] Charles et al., 2002; [22] Grange et al., 2002). Suyama described 25 patients evaluated for injuries related to less lethal weapons implemented during a period of civil unrest in Cincinnati, Ohio. There were no deaths, but three patients required admission, including one with a pulmonary contusion, one with a liver laceration and one with
an Achilles tendon rupture ([66] Suyama et al., 2003). [14] de Brito et al. (2001) retrospectively reviewed five years of bean bag injuries in Los Angeles County Hospital and reported on 40 patients with one death from a massive hemothorax caused by chest penetration of the projectile.

In addition to penetrating trauma, blunt impact projectiles can also cause significant injury from blunt trauma. Chute and Smialak reported a case of a 61-year-old woman shot in the chest with a plastic bullet (AR-1 baton round) who subsequently collapsed, suffered cardiac arrest and died. Autopsy showed she had sustained multiple rib fractures to the left chest, an underlying lung laceration, and heart lacerations that led to significant bleeding into the chest cavity. The cause of death was reported as blunt force injuries of chest due to plastic bullet wound ([11] Chute and Smialek, 1998).

Several studies have looked at the injury patterns from the use of plastic and rubber bullets. Their conclusions all tend to show that while generally regarded as less lethal weapons, significant injuries including death can occur when the weapons strike the chest, abdomen, or head. Millar et al. reviewed 90 patients who had sustained injuries to various parts of their bodies, concluding that the eyes, face, skull, bones, and brain are at greatest risk of injury from rubber bullets. The distance at which the rubber bullets resulted in serious injury ranged from 17 to 25 meters ([45] Millar et al., 2005). Hughes reviewed 29 cases of injuries from a new plastic baton round in Northern Ireland. There were no fatalities, but seven patients required admission ([31] Hughes et al., 2005). Steele retrospectively reviewed patients presenting to six hospitals during a one-week period of civil unrest in Northern Ireland who were injured by plastic bullets. He reported a total of 155 patients with 172 injuries, no fatalities, but 42 admissions, with three to intensive care. Of those in intensive care, one had globe rupture and multiple facial fractures, one had a laparotomy for three perforations of the small bowel and the third required splenectomy for a splenic laceration ([62] Steele et al., 1999). Ritchie and Gibbons reported on 80 subjects injured by rubber bullets, with four who died, three from ventricular dysrhythmias secondary to cardiac contusion and one from a hemopneumothorax. An additional 19 patients required hospitalization for significant chest wounds ([56] Ritchie and Gibbons, 1990).

Commotion cordis is a rare occurrence when a direct blow to the chest causes a sudden fatal disturbance of cardiac rhythm in the absence of demonstrable signs of significant mechanical injury to the heart. Although, no in-custody death cases associated with impact projectiles have been specifically attributed to commotion cordis, blunt impact weapons generate energies that are on the order of those that have induced commotion cordis in other situations (such as an individual struck to the chest with a baseball). Therefore, one could speculate that these devices would have the same risk of causing ventricular fibrillation (VF) and sudden death.

Overall, impact projectiles have been used widely and effectively as less lethal weapons. From the medical perspective, injuries and rare deaths have been directly related to the blunt traumatic force delivered by the projectile onto the individual. While efforts continue to focus on reducing this risk, it is unlikely that such injuries can be completely eliminated given that these devices are designed to deliver pain and irritation through blunt force.

**Irritant sprays**

Irritant sprays include agents like CN, CS, and OS (pepper spray), which can be used to disperse large gatherings or to temporarily incapacitate individuals. These agents are commonly dispersed
as gases, smoke or aerosols, and therefore, may affect users as well as subjects.

CN was first synthesized in 1871. It was used in World War I as well as served as the primary tear gas used by law enforcement and the military up through the 1950s. It is a colorless crystalline substance that can be disseminated in a smoke form from an explosive device, such as a grenade, or propelled as a liquid or powder. It acts as an irritant smoke when in contact with skin or mucous membrane tissues such as the eyes, nasal passages, oral cavity and airway. Symptoms of exposure include sneezing, rhinorrhea, coughing and increased airway secretions, as well as burning sensations of the nasal passages and airways. Oral cavity and gastrointestinal exposure can result in the sensation of burning in the mouth, increased salivation, gagging, nausea, and vomiting. Ocular exposure to CN causes a burning sensation in the eye, injection of the conjunctiva, eye irritation, photophobia, and tearing. Similarly, skin contact can result in burning, irritation, and erythema.

CS is an irritant agent first synthesized in 1928, and replaced CN as the standard riot control irritant agent in the US Army in 1959. Because of its perceived improved effectiveness, it had replaced CN in most law enforcement agencies in the USA by the late 1950s as well. CS is typically disseminated by dispersion of the powder or solution by explosion, spray or smoke. Because of its insoluble nature, decontamination of buildings or other items after exposure can be challenging. CS also has a high-flammability rating and has been noted to have caused some structure fires ([13] Danto, 1987).

The clinical effects that may be seen with the use of CS are similar to those of CN, resulting in irritation and inflammation of the skin, airways, and mucous membrane tissues on exposure. The effects typically start within minutes of exposure and continue as long as the person is exposed to the material. After minutes of exposure, a sensation of skin burning will typically occur, particularly over moistened or freshly shaven areas. The degree of symptoms tends to worsen based on concentration and duration of exposure. Increased exposure can have symptoms progress to gagging and vomiting, more skin and mucus membrane burning and subjective tightness in the chest. These symptoms improve after removal of the exposure and gradually resolve over 30-60 minutes, but skin erythema may last up to several hours. However, if the exposure is with a high concentration of CS, under high temperature or humid conditions, severe erythema along with edema and skin vesication can occur, typically occurring within the first hour. Tolerance to CS has been demonstrated from prolonged or repeated exposures ([52] Punte et al., 1963; [2] Bestwick et al., 1972).

During the riots in Washington DC in 1968, firefighters were often exposed to CS when they entered buildings in which the agent had been previously used. Movement around the building and use of water hoses re-aerosolized the material, causing erythema and edema on the skin of a number of firefighters ([54] Rengstorff and Mershon, 1969a). In workers with repeated exposure and sensitization to CS, acquired contact dermatitis has occurred, confirmed by skin testing. Symptoms ranged from simple erythema to large vesicles and bullae. No pulmonary symptoms were reported ([60] Shmunes and Taylor, 1973).

OC or pepper sprays are derived from the natural oily extract of pepper plants in the genus capsicum. The use of OC spray by law enforcement agencies increased in the 1980s as the use of CS was on the decline, and by the 1990s, the majority of states had legalized OC spray use by
the public ([61] Smith and Greaves, 2002). Concentrations of OC may range from 1 to 15 percent, with the commercially available OC typically being about 1 percent in concentration. Delivery modes include liquid stream spray, aerosol spray, and powder delivered as a projectile.

OC spray can cause direct irritation to the eyes, skin and mucous membranes. The onset of symptoms is almost instantaneous, causing burning and tearing of the eyes, as well as eye spasm, ranging from involuntary blinking to sustained closure of the eyelids. Cutaneous symptoms may include flushing, tingling and intense burning sensation of the skin, particularly over recently shaved areas. Mucous membrane exposure, especially of the nasal passages, will cause irritation, rhinorrhea and congestion, with some subjects reporting nausea. Exposure of the airway and respiratory tract to aerosolized OC causes tingling, coughing, gagging and shortness of breath, as well as a transient laryngeal paralysis and a temporary inability to speak ([63] Steffee et al., 1995).

Case reports and reviews

CN and CS: A few cases of severe allergic reactions have been reported with CN, particularly following a previous exposure. One case was reported in a military recruit who had been previously exposed to CN 17 years earlier which manifested as minimal itching at that time. Following a repeated exposure, he developed generalized itching, which progressed over the next several hours to generalized erythema all parts of his body except the face portion which had been covered by a mask. He developed a fever to 103 and by 48 hours had diffuse vesication and edema, followed ultimately by sloughing of much of his skin, but recovered ([53] Queen and Standler, 1941). Other cutaneous reactions have been reported as well ([40] Madden, 1951).

Thorburn reported on the medical complications associated with prolonged exposure to CN in a prison where there were recurrent and prolonged exposures in closed spaces with limited ventilation. Cutaneous complications included first and second degree burns. Treatment with steroids and bronchodilators for laryngotracheo-bronchitis, inflammation of the airway, was needed in several patients, but none reported any permanent damage. All eye complaints were transient and required no specialized treatment, resulting in no corneal injuries or permanent damage ([69] Thorburn, 1982).

Chapman and White reported on a prisoner who was found dead under his bunk 46 hours after a reported prolonged CN gassing of inmates in cells with no ventilation. The deceased inmate was found in rigor mortis and on autopsy was noted to have evidence of inflammation and damage to his airway and lungs ([9] Chapman and White, 1978). Another reported death occurred after closed room exposure with an estimated ten times the lethal concentration. On autopsy, there were similar findings as with the previous case ([52] Punte et al., 1963).

Park and Giammona report a case in which CS tear gas canisters were fired into a house resulting in a four-month-old infant being exposed for two-to-three hours. The infant required hospital admission for frequent suctioning of upper airway secretions and was treated with steroids and antibiotics as well as positive pressure ventilation for respiratory distress and wheezing. He was ultimately discharged home fully recovered after 28 days ([50] Park and Giammona, 1972). Thomas et al. reported on nine marines involved with strenuous exercise and exposure to CS in field training who developed a transient pulmonary syndrome. They presented with cough, shortness of breath, hemoptysis and hypoxia, with some requiring close monitoring
and treatment for hypoxia, but all nine recovered and demonstrated normal lung function within a week after the exposure ([68] Thomas et al., 2002). Hu reported a case of exposure in an asthmatic who developed semi-chronic symptoms of cough and shortness of breath for up to two years after the exposure. Her FEV1 (Forced Expiratory Volume in 1 second) at four weeks post-exposure was 62 percent of predicted and her forced vital capacity was 78 percent. At one and a half years after exposure, her FEV1 was 128 percent of predicted, with a 16 percent drop with brisk exercise in cool air ([30] Hu and Christiani, 1992). Based on the report, it is difficult to determine if her subjective symptoms of dyspnea were related to her underlying chronic asthma rather than the CS exposure.

The use of CS has resulted in reports of eye injuries, particularly when a tear gas cartridge is discharged at close range. In some cases, particles of agglomerated CS were driven into the eye tissue by the force of the dispersion device, typically a blast. In these cases, chemical reaction damage of the cornea was noted over the course of months to year, which are characteristically different than blast injuries from particles other than CS ([36] Levine and Stahl, 1968).

OC: Since, OC spray is commonly used by many law enforcement agencies, there are many case reports and case series of deaths and injuries following OC use ([63] Steffee et al., 1995; [21] Granfield et al., 1994; [51] Pollanen et al., 1998; [47] O'Halloran and Frank, 2000). Amnesty International claims that over 90 persons have died after exposure to pepper spray in the USA since the early 1990s ([1] Amnesty International, 2005). [21] Granfield et al. (1994) reported 30 cases of in-custody death following OC exposure, in which drugs and underlying natural diseases were a significant factor in a majority of these cases. [47] O'Halloran and Frank (2000) reported of 21 cases of restraint in-custody death, of which ten of the restraint episodes were preceded by use of OC spray, and [51] Pollanen et al. (1998) reported 21 in-custody restraint deaths of which four had been sprayed with OC.

However, a causal connection between OC exposure and death remains controversial. There is no definitive evidence that OC is inherently lethal. In almost all of these cases of reported deaths associated with OC, the OC spray was determined not to have been the cause of death, with the exception of only one case. In that patient, Steffee et al. reported that a person who had a history of asthma and was sprayed with OC spray 10-15 times suffered a sudden cardio-respiratory arrest. Autopsy revealed severe epithelial lung damage with the cause of death attributed to severe bronchospasm probably precipitated by the use of pepper spray ([63] Steffee et al., 1995).

Billmire described a four-week old healthy infant who was sprayed in the face with a 5 percent OC spray when a key chain self-defense canister accidentally discharged. The child had sudden onset of gasping respirations, epistaxis, apnea, and cyanosis. The child required mechanical ventilation and extracorporeal membrane oxygen support. The child was discharged home after a 13-day hospitalization ([3] Billmire et al., 1996). An 11-year-old boy required intubation and ventilation four hours after exposure for severe croup (upper airway inflammation) that resulted from intentional inhalation of OC spray. He was extubated two days later and recovered uneventfully ([73] Winograd, 1997).

Since, OC is typically directed towards the face, symptoms often involve the eyes. Corneal abrasions have been reported in up to 7 percent by [71] Watson et al. (1996) and 8.6 percent of
cases by [7] Brown et al. (2000). These findings have been noted as transient and do not require any additional treatment beyond decontamination with water irrigation. These temporary ocular injuries were also reported by [70] Vesaluoma et al. (2000).

Research studies

CN and CS: There is limited human research on the risks of CN in terms of inducing disability or death. Although permanent eye damage has been reported associate with the use of CN at close range, it is challenging to separate out whether the damage is from the CN or the actual weapon. However, at harassing or standard field concentrations, there is no evidence that CN causes permanent eye injury. Holland performed human studies in which 0.5 milligram of CN placed on subjects' skin for 60 minutes caused irritation and erythema, as compared with CS which had no effects when used in amounts of less than 20 milligram. Skin vesication was seen with the same dose of CN when the skin was moist, whereas no vesication occurred with CS at levels of 30 milligram or less ([29] Holland and White, 1972).

There is little evidence that CS results in any permanent lung damage even after several exposures to field concentrations ([4] Blain, 2003). In 36 subjects exposed to CS, [2] Bestwick et al. (1972) found no change in tidal volume, peak flow or vital capacity when comparing pre-exposure values to those measured immediately afterward and at 24 hours post-exposure. In another study on human subjects, [52] Punte et al. (1963) reported that individuals subjected to daily exposures to CS showed no changes in airway resistance immediately following, as four or ten days after CS exposure.

In terms of other types if injuries, human studies have been performed to assess the effects of CS on skin using different concentrations and assessing the effects of various ambient temperatures and humidity levels. Subjects developed first and second degree burns at different levels and the authors concluded that many variables affect the likelihood of blistering, making risk assessment difficult to predict ([26], [27] Hellreich et al., 1967, 1969). Human ocular exposures of 0.1 or 0.25 percent CS carried in different solutions caused the inability to open the eyes for 10-135 seconds. Evaluation after the exposure via slit lamp examination noted a transient conjunctivitis, but no corneal damage ([54], [55] Rengstorff and Mershon, 1969a, b).

OC: Because of its ability to block pain sensation and itching, capsaicin has been studied in many different clinical conditions including treatment of psoriasis, osteoarthritis, post-herpetic neuralgia, and diabetic neuropathy. These capsaicin-related pharmacotherapies have typically been associated with topical application of the agent. Given its ability to induce cough, capsaicin has also been utilized to study the cough reflex and the pulmonary system, as well as to assess the efficacy of various cough suppressants ([18] Foster et al., 1991).

Some animal and in-vitro human tissue studies have suggested that capsaicin increases airway resistance and bronchoconstriction ([38] Lundberg et al., 1983; [24] Hansson et al., 1992). However, clinical studies in humans with nebulized capsaicin are less definitive. Fuller reported that inhaled nebulized capsaicin resulted in a temporary increase in airway resistance that was dose-dependent, maximal at 20 seconds, and lasting less than 60 seconds ([19] Fuller et al., 1985). [5] Blanc et al. (1991) and [12] Collier and Fuller (1984) both reported no significant decrease in FEV1 in subjects who inhaled nebulized capsaicin at concentrations sufficient to induce cough. However, direct bronchoconstriction caused by capsaicin may be masked by
cough and deep inhalation as both have bronchodilatory effects. In fact, doses of inhaled capsaicin low enough to not induce coughing have been shown to cause changes in airway resistance and pulmonary function ([20] Fuller, 1991; [41] Maxwell et al., 1987; [25] Hathaway et al., 1993).

Unlike capsaicin, data on the human effects of OC spray are limited, particularly any interventional data ([58] Ross and Siddle, 1996). A number of observational reports have been published assessing safety of OC spray use, including a two-year joint study by the FBI and US Army that reported that OC spray was not associated with any long-term health risks ([48] Onnen, 1993).

Chan et al. conducted a randomized, cross-over controlled trial in 35 volunteer human subjects who were exposed to either OC spray or placebo propellant without OC, followed by a ten minute period of being placed in either the sitting or prone maximal restraint position. Pulmonary function testing was performed and arterial blood gases sampled during this time. OC exposure did not result in abnormal pulmonary dysfunction, hypoxemia or hypoventilation when compared to placebo in either the sitting or restraint positions. However, there was an increase in mean heart rate and blood pressure in subjects exposed to OC that did not occur in the placebo group. The investigators concluded that OC spray did not result in any evidence of respiratory compromise with and without restraint that would make place subjects at risk for asphyxiation from OC exposure. The changes in cardiovascular parameters, however, indicated the need for additional study ([8] Chan et al., 2002).

Beyond clinical research in the laboratory setting, OC spray use has been widespread and a number of epidemiologic studies have reported on its use and safety. The California State Attorney General reported that no fatal consequences occurred in over 23,000 exposures to OC spray. Watson et al. reviewed 908 exposures to OC spray that had occurred locally and found that fewer than 10 percent of subjects required any medical attention, and more specifically less than 1 percent had respiratory complaints requiring medical treatment. None of these patients were determined to have any significant injuries. Additionally, no fatalities were reported in either of these studies ([71] Watson et al., 1996; [39] Lundgren, 1996).

Overall, OC spray has been used hundreds of thousands of times with no long-term health effects reported. Although there are case reports of death following use, in the large majority of cases other causes such as drug intoxication, excited delirium or underlying medical condition, have been implicated as the primary cause of death in the large majority of these cases. Moreover, clinical and epidemiologic studies on OC have yet to report any compelling evidence that OC is inherently dangerous or lethal.

Conductive energy devices

CEDs were introduced into the law enforcement force continuum in the late 1970s. CED is a generic term referring to any device to subdue and control an individual by delivering electrical energy to the subject. The most well-known CED is the Taser® (Thomas A. Swift Electric Rifle) energy device, but others on the market include the Stinger stun gun and the remote activated custody control (RACC) belt®. There are other electronic belts, shields, and a host of hand-held contact stun guns available to law enforcement. Many of these products are also available to the general public.
In the past decade, the Taser has become the most popular incapacitating neuromuscular device on the market with an estimated 10 percent of all police officers in this country currently carrying the device ([23] Hamilton, 2005). According to Taser International®, Tasers have been purchased by over 9,000 police departments in the USA and abroad. The manufacturer asserts that the device helps officers avoid the use of deadly force while lowering the risk of injury to users. It has been reported that the device has been used on over 150,000 volunteers during training sessions and on over 100,000 subjects by law enforcement officers in actual field confrontations, though the true total number of uses is unknown ([67] Taser International, 2006).

The Taser X26 is a handheld device resembling a handgun intended to be used on subjects up to 21 feet away. The energy output of the device is 26 watts total, 1.76 joules per pulse, at 1.62 milliamps, and 50,000 volts. It utilizes an automatic timing mechanism to apply the electric charge for 5 seconds. The device initially propels two probes at a velocity of 180 feet per second. The electrical energy is discharged through a sequence of dampened sine-wave current pulses each lasting about 11 microseconds. This energy is neither pure AC nor pure DC, but probably akin to rapid fire, low amplitude DC shocks.

CEDs work by incapacitating volitional control of the body. These weapons create intense involuntary contractions of skeletal muscle, causing subjects to lose the ability to directly control the actions of their voluntary muscles. CEDs directly stimulate motor nerve and muscle tissue, overriding central nervous system control and causing incapacitation regardless of the subject's mental focus, training, size, or drug intoxication state. Subjects report painful shock-like sensations and the feeling that all of their muscles are contracting at once. During the CED discharge, subjects are unable to voluntarily perform motor tasks, however they remain conscious with full memory recall.

This effect terminates as soon as the electrical discharge is halted. Immediately after the taser shock, subjects are usually able to perform at their physical baseline. There is no known permanent lasting effect on the muscular system aside from any injuries that may result from an associated fall. There is a large experience of police trainees who have been tasered as part of their training. Most reported that the experience was unpleasant and declined to be re-tasered. A few subjects described a tingling sensation in the area under the probe sites lasting a few minutes after being tasered ([34] Koscove, 1985). There is some residual muscle soreness reported by some who have been tasered.

CED effects vary depending on the particular device used, body location of and distance between the probes, and the condition of subject. For example, probes spread apart over a larger distance on the subject's body will have a greater effect because it allows for the electrical discharge to affect a larger portion of the body ([16] Fish and Geddes, 2001). The effects of these devices have been reported to increase with the duration of application such that prolonged exposures may result in some sensation of fatigue and weakness even after the discharge is halted ([57] Robinson et al., 1990). On the other hand, CEDs may fail to have their intended effect if the probes do not make adequate contact with the body, the probe spread is not wide enough thereby only affecting local muscle groups, or if the device fails to discharge.

**Case reports and reviews**
There has been a great deal of publicity in the lay press recently regarding in-custody deaths in subjects following use of the Taser ([23] Hamilton, 2005). Amnesty International claims that more than 70 persons have died after Taser deployment by law enforcement. Some have postulated that the electrical discharge of CEDs on the body can induce life-threatening heart conduction abnormalities or cardiac dysrhythmias, disrupt normal respiration or cause metabolic derangements that could lead to death. However, there is limited research on the direct physiologic effects of CEDs and a direct causal connection between CEDs and the reported fatalities remains controversial.

Kornblum and Reddy examined 16 deaths that were associated with Taser use over a five-year period. All of these cases involved young men with a history of drug abuse who were behaving in a bizarre or unusual fashion drawing police attention. The ultimate cause of death was determined to be drug overdose in the majority of cases. The authors suggest that most of the subjects died after being in a manic, agitated, combative state, known as agitated delirium. Drug intoxication itself caused or predisposed the subjects to have increased risk for sudden death, and that the taser was not likely the causative factor. There was one case, however, in which Taser was felt to be contributory. In this case, the subject had a history of cardiac disease, for which he had been told to get a pacemaker, but had not done so. On autopsy he had a diseased heart and lethal levels of PCP in his system, but the cause of death was listed as cardiac arrhythmia due to sick sinus syndrome, mitral valve prolapse, and electrical (Taser) stimulation while under the influence of PCP ([33] Kornblum and Reddy, 1991). Overall, the authors of the report concluded that the Taser in and of itself did not cause death, but may have contributed in this one case.

In a prospective case review conducted by Ordog in Los Angeles in the mid-1980s, 218 patients who presented to the emergency department after being shot with a taser were evaluated. These patients were then compared with 22 similar patients who were shot by police with 0.38 caliber handguns during the same time period. In 76 percent of the cases in which the Taser was utilized, subjects displaying bizarre and uncontrollable behavior. Ninety-five percent were men and 86 percent had a history of recent phencyclidine (PCP) use. The mortality rate in the taser group in this study was 1.4 percent (3 of 218 patients) and the morbidity rate was 0 percent. All three patients who died arrived to the emergency department in asystole, had high levels of PCP in their system and went into cardiac arrest shortly after being tasered, ranging anywhere from 5 to 25 minutes after taser deployment. The medical examiner’s reports on all three cases listed PCP toxicity as the cause of death, with no signs of myocardial damage, airway obstruction, or other fatal pathologic findings. Of the 22 patients shot with the 0.38 special, 50 percent of died and 50 percent had varying degrees of serious morbidity ([49] Ordog et al., 1987).

Strote et al. evaluated deaths associated with Taser use found via a search of Lexus-Nexus and Google. They identified 71 deaths associated with Taser use, with 28 (39 percent) having autopsy reports available. The average age was 34.8 years, all were male, and 39 percent were White, 46 percent were Black and 14 percent were Hispanic. No deaths were found to occur directly because of Taser use, but 21 percent reported a possible contributory component. Causes of death was felt to be directly drug related in 57 percent of cases, with 68 percent of the cases having cocaine or methamphetamine use. Excited delirium was either directly or indirectly responsible in 57 percent of cases and 46 percent of cases had significant pre-existing cardiac disease reported ([65] Strote et al., 2005).
Mehl reported a case of a miscarriage in a 32-year-old pregnant woman at approximately 8-10 weeks gestation one week after she had received a Taser activation. One probe lodged above the uterus in the abdomen, and the other in the left thigh. Reports of the duration of shock varied from 3 to 10 seconds. She fell to the ground and was reportedly unable to move for 5 minutes afterwards. One day later she began having vaginal spotting that continued for 7 days and was subsequently diagnosed with an incomplete miscarriage. Pathology analysis of the tissue from a uterine curettage revealed products of conception with extensive hemorrhage, necrosis, and inflammation. Though a temporal relationship is suggested between the Taser activation and miscarriage, no clear cause and effect relationship can be established ([44] Mehl, 1992).

Research

Human research on the effects and safety of CEDs is limited, with most physiologic investigations having been conducted in animal models. One of the reasons for the limited human studies is the requirement that such studies be approved by local human research protections committees, which are often wary of these devices because of preconceived notions based on media and press reports. In fact, the approval of the original devices were not based on actual human or animal studies, but rather theoretical calculations of the physical effects of dampened sinusoidal pulses, for which the US Consumer Product Safety Commission concluded that the taser should not be lethal to a normal healthy person ([46] Obrien, 1991).

One of the more common concerns regarding CEDs is whether these devices can cause cardiac dysrhythmias or cardiac standstill. The development of dysrhythmias or standstill would then cause the heart to not pump blood to the rest of the body, resulting in sudden death. The two main cardiac rhythm disturbances that are of greatest concern are VF, which is the lack of organized electrical activity and contraction of heart muscle cells, and asystole, which is the absence of any electrical activity.

For externally applied current, the fibrillatory current (the current that produces VF in human beings is believed to be a function of the duration, frequency, and magnitude of the current, as well as the patient's body weight and the timing in the cardiac cycle during which the current is applied ([34] Koscove, 1985; [15] Ferris et al., 1936; [35] Kouwenhoven et al., 1959). The threshold for VF in men for externally applied, 60 Hertz current has been proposed to be 500 milliamps for shocks of less than 200 microseconds duration, and 50 milliamps for shocks of more than two seconds ([34] Koscove, 1985). The longer a current flows, the greater the chance a shock will occur during the vulnerable part of the cardiac cycle (early ventricular repolarization which is approximately 10-20 percent of the cardiac cycle) ([17] Forrest et al., 1992). The Taser X26 carries a current of 2.1 milliamps for a duration of 0.0004 seconds ([67] Taser International, 2006).

Additionally, resistance is also going to play a role into how much current actually flows for a given voltage (voltage = current × resistance). The lower the resistance, the larger the current that will flow. The total resistance of the body is the sum of internal resistance plus twice the skin resistance as current enters and exits the body ([17] Forrest et al., 1992). A skin effect is known to exist when high-frequency electricity is used as these currents tend to stay near the surface of a conductor. Since, the Taser devices use very high-frequency electricity, the output of the Taser is believed to stay near the skin and muscle surface of the body and not penetrate
deeply to the internal organs, such as the heart ([6] Bleetman et al., 2004).

A porcine study published by Roy in 1989 used an older model stun gun that produced high voltages (>100,000 volts) and short duration pulses (<20 microseconds). The investigators compared five different models of stun gun with varying energies. The average value of the current applied during each shock was calculated to be 3.8 milliamps. When towels were placed between the skin and the electrodes to simulate clothing, the maximum current spike was 190 milliamps with a pulse length of 20 microseconds. Using two anesthetized normal healthy pigs, the investigators were able to induce VF when the leads of the stun gun were applied directly to the heart or to the chest of one of the animals in which a cardiac pacemaker had been implanted. Important to note was that these adverse effects were immediate, not delayed. The authors surmised that the mechanism of action inciting VF was not pacemaker inhibition, but rather fibrillatory current directly accessing the heart via the pacemaker leads. This device's shock also produced cardiac standstill when applied through layers of simulated clothing over a prolonged period. However, these findings only occurred with the two stun gun models delivering the highest energy. There were no cardiac effects seen with the lower energy units. This study demonstrated that VF was indeed possible, but only at very high-energy outputs and when the electrical discharge occurred directly over or with direct access to the heart ([59] Roy and Podgorski, 1989).

More recently, McDaniels and Stratbucker studied the Air Taser and Advanced Taser M26 in five anesthetized dogs with an average weight of 54 pounds. Over 200 electrical discharges of the devices placed directly over the chest failed to induce VF in any of the animals. The authors did note that when both probes were placed directly over the heart they were able to pace the heart similar to a pacemaker, but still did not induce VF ([43] McDaniel et al., 2000).

Stracbucker et al. studied 13 adult domestic pigs by applying Taser-like electrical discharge to the thorax similar to human use of the device, and then gradually increased the energy output above that level until VF was achieved. The investigators did not induce VF in the pigs until levels of energy that of the standard Taser level. When using energy levels below that threshold, 43/43 discharges did not induce VF ([64] Stracbucker et al., 2003).

In another animal study, McDaniel et al. evaluated the cardiac effects on nine pigs shocked using a device that delivered an electrical discharge identical in waveform and charge to the Taser X26 device. The electrodes were placed across the thorax of the animals using the barbs that matched the probes used by the standard device. The animals were shocked for 5 seconds, simulating field use of the device. The study used gradually increasing amounts of charge delivered to identify two levels. The first being the lowest amount of charge required to induce VF at least once, called the VF threshold. The second defined as the highest discharge that could be applied five times without inducing VF called the maximum safe level. The authors then compared this value to the standard device discharge and the ratio of the two values to determine the safety index. The study found that the electrical discharge required to induce VF was 15 to 42 times the energy output of a standard Taser discharge. This safety factor increased with the size and weight of the subject. The conclusion of the authors was that discharge levels output by fielded Taser devices have an extremely low probability of inducing VF ([42] McDaniel et al., 2005).

In one of very few studies in human subjects, Levine et al. conducted a study monitoring 67
subjects electrocardiographically immediately before and after Taser shock during police training sessions. The investigators reported no changes in cardiac rhythm, ECG morphology, or presence aberrantly conducted beats following the taser discharge. Mean heart rate increased by just over 19.4 beats/minute following the taser shock, but no abnormal cardiac dysrhythmias were identified ([37] Levine et al., 2005).

Recently, Ho et al. evaluated 66 volunteer subjects who received a standard five second Taser activation at a training course. The authors obtained venous blood samples before, immediately after, and 16 hours and 24 hours after activation. The blood samples were analyzed for troponin, myoglobin, lactate, potassium, glucose, blood urea nitrogen, creatinine, and creatine kinase levels. There were no significant changes from baseline values of the electrolyte or blood urea/creatinine ratio. There was an increase in the serum bicarbonate and creatinine kinase levels at 16 and 24 hours. Serum myoglobin levels were elevated at all three time intervals post-Taser activation, but the troponin levels all remained < 0.3 nanograms per millilitre except for a single 24 hour post exposure level. That subject was evaluated at a hospital by a cardiologist, with no evidence of myocardial infarction or cardiac disability found. The troponin level returned to normal eight hours later ([28] Ho et al., 2006).

The potential for life-threatening cardiac dysrhythmias or cardiac muscle damage to occur as a result of the electrical discharge from current Taser devices appears to be low based on the available studies. However, there may be theoretical risks to patients with pacemakers or underlying cardiac disease, and the effect of recurrent or prolonged taser discharges remains unclear.

To date, little research has been conducted on the non-cardiac effects of the Taser. An air force study published by Jauchem et al. investigated the metabolic effects of repeated taser activations on sedated swine that received five-second Taser activations alternating with five seconds of rest for three continuous minutes. The animals demonstrated transient, clinically insignificant increases in potassium and sodium, a significant decrease in blood pH (increase in acid level) that returned toward normal after 1 hour, a significant rise in blood lactate that returned to baseline after 2 hours, and a significant rise in whole blood pCO2 that returned to baseline after 1 hour. The correlation of these results to use in humans, where far fewer applications are utilized, is unknown ([32] Jauchem et al., 2005).

More recently, the respiratory effects of the Taser were studied in 32 human subjects who underwent a 5 second Taser discharge. In this study, pulmonary function, ventilation, oxygenation and carbon dioxide elimination were monitored in human volunteers up to 1 hour after the Taser shock. Overall, ventilation and respiratory rate actually increased during the first 10 minutes, then returned to baseline levels. The subjects did continue to breathe during the 5 second shock. There was no evidence of abnormally low oxygen or elevated carbon dioxide levels in the blood following the shock, suggesting the Taser had no detrimental impact on respiratory function (Chan, SAEM 2007). The effect of Taser discharges on neurologic function remain to be studied.

**Conclusion**

Impact projectiles, irritant spray agents, and CEDs are important in the use of force armamentarium for law enforcement when dealing with violent, combative individuals who
place themselves and the general public at risk. While associated with rare cases of sudden in-custody deaths, it is unclear what causal connection may exist between these less lethal technologies and reported fatalities. In many instances, individuals were in conditions which placed them at high risk for sudden death regardless of what force was utilized. In addition, a combination of force methods may have been utilized in these cases. Further, research is needed to study the impact of these weapons on human physiology, as well as the underlying condition of those individuals who come in contact with law enforcement and are at greatest risk.

American Journal of Forensic Medicine and Pathology

[Reference]


8. Chan, T.C., Vilke, G.M., Clausen, J., Clark, R.F., Schmidt, P., Snowden, T. and Neuman, T.


[Appendix]

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