

TECHNICAL NOTE**PATHOLOGY/BIOLOGY**

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Increased Hematocrit After Applications of Conducted Energy Weapons (Including TASER[®] Devices) to *Sus scrofa**

ABSTRACT: Conducted energy weapons (CEWs) are used by law enforcement personnel to incapacitate individuals quickly and effectively, without intending to cause lethality. CEWs have been deployed for relatively long or repeated exposures in some cases. In laboratory animal models, central venous hematocrit has increased significantly after CEW exposure. Even limited applications (e.g., three 5-sec applications) resulted in statistically significant increases in hematocrit. Preexposure hematocrit was significantly higher in nonsurvivors versus survivors after more extreme CEW applications. The purpose of this technical note is to address specific questions that may be generated when examining these results. Comparisons among results of CEW applications, other electrical muscle stimulation, and exercise/voluntary muscle contraction are included. The anesthetized swine appears to be an acceptable animal model for studying changes in hematocrit and associated red blood cell changes. Potential detrimental effects of increased hematocrit, and considerations during law enforcement use, are discussed.

KEYWORDS: forensic science, forensic pathophysiology, conducted energy weapon, electronic control device, TASER, electro-muscular disruption, hematocrit, animal model, swine

Conducted energy weapons (CEWs) (sometimes referred to as “electronic control devices”), including those manufactured by TASER International (Scottsdale, AZ), are used by law enforcement personnel to incapacitate individuals quickly and effectively, without intending to cause lethality. The devices have been deployed for relatively long or repeated exposures in some cases. The potential of CEWs to cause or contribute to death is a subject of intense debate (see [1] for discussion). Hypotheses for such outcomes include: (i) increased physiological stresses associated with law enforcement actions (such as those because of increased physical struggle) that can occur (and not simply the electrical discharge of the weapon), and (ii) increased susceptibility to cardiac arrhythmia in certain subjects.

Cardiac events, such as ventricular fibrillation, are unlikely to occur as a result of any direct effect of CEW pulses on the heart (2). Several other potentially detrimental physiological changes, however, have been noted in animal-model studies, including increased blood potassium, acidosis, and increased hematocrit (Hct; [3–8]).

On the basis of previous studies (summarized in [1]), there appears to be a wide margin of safety, relative to blood potassium concentration, for most CEW applications. Blood pH, although

significantly decreased following CEW exposures (3–8), was not different in terms of preexposure values for nonsurvivors versus survivors (6).

In one study, a significantly higher preexposure Hct occurred in nonsurvivors versus survivors after CEW applications (6). As the difference between groups was small, this aspect was originally considered unlikely to have played a role in survival. On the basis of a continuing consistent finding of increased Hct after CEW exposures (7,8), however, this hypothesis should be reconsidered. A substantially increased Hct can result in detrimental effects (details listed later).

The number of muscle movements over a given time period, and the rate of development of each movement, can differ between electrically induced versus voluntary muscle contraction. Both (i) multiple spinal reflexes and (ii) direct motor-neuron stimulation may play roles in muscle action during CEW exposure (9). Although muscle-contraction responses to CEW applications generally bypass volition, some aspects of such responses may be similar to changes during exercise (10). In spite of some differences in the details of recruitment patterns of muscle motor units, there are also many similarities between electrical stimulation at high levels and the voluntary muscle action occurring during exercise (11). Because of these similarities, knowledge of previous studies of exercise/muscle contraction may be relevant to responses during CEW applications. (In contrast with the beneficial aspects of regular endurance exercise, single episodes of intense muscle contraction can result in transient detrimental effects.)

In *all* previous CEW experiments in which Hct was measured (3–8,12), central venous Hct increased after exposure. The purpose of this technical note is to address specific questions that may be generated when examining these results.

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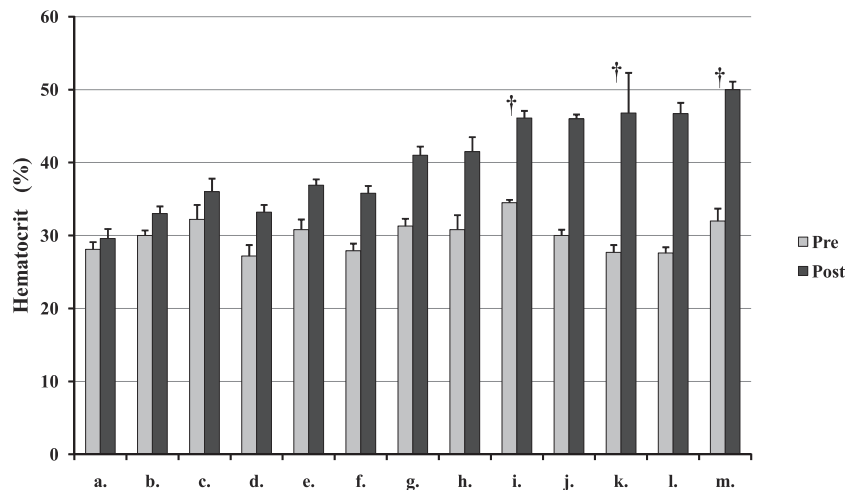


FIG. 1—Pre- and postexposure values of hematocrit (Hct mean \pm standard error of mean) in published studies of conducted energy weapon (CEW) applications in anesthetized *Sus scrofa*. Lowercase alphabet letters on the x-axis refer to portions of studies listed later (full citation information available in list of references). Studies are, in general, placed on x-axis in approximate order of increasing severity of CEW exposures. Pre- versus postexposure Hcts were statistically significant in each reference. †subsets of animals that did not survive exposures. (a) Single 5-sec X26 CEW application (N = 10) (ref. 7). (b) Three repeated 5-sec on/5-sec off cycles of X26 CEW (N = 10) (ref. 5). (c) 30-sec continuous C2 CEW application (N = 7) (ref. 8). (d) 30-sec continuous X26-like CEW application (N = 5) (ref. 7). (e) 60-sec continuous X26-like CEW application (N = 5) (ref. 7). (f) 1st series of 18 repeated 5-sec on/5-sec off cycles of X26 CEW (N = 7) (ref. 4). (g) 2nd series of 18 repeated 5-sec on/5-sec off cycles of X26 CEW (N = 7) (ref. 4). (h) Eighteen repeated 7-sec on/3-sec off cycles of X26 CEW; animals that survived (N = 7) (ref. 6). (i) Eighteen repeated 7-sec on/3-sec off cycles of X26 CEW; animals that died (N = 7) (ref. 6). (j) 30-sec continuous X26, followed by repeated 5-sec on/5-sec off cycles of X26 CEW for 12 min; animals that survived (N = 6) (ref. 12). (k) 30-sec continuous X26, followed by repeated 5-sec on/5-sec off cycles of X26 CEW for 12 min; animals that died (N = 3) (ref. 12). (l) 30-sec continuous X26, followed by repeated 5-sec on/5-sec off cycles of X26 CEW for 22.5 min; animals that survived (N = 6) (ref. 12). (m) 30-s continuous X26, followed by repeated 5-sec on/5-sec off cycles of X26 CEW until death (N = 5) (ref. 12). (“X26” and “C2” are trademarks of TASER International, Inc. TASER® is a registered trademark of TASER International.)

Changes in Hct After CEW Applications, Other Electrical Muscle Stimulation, and Exercise/Voluntary Muscle Contraction

Methods for previous CEW experiments have been discussed in detail (13). The equipment used in these studies (Gem Premier 3000 analyzer; Instrumentation Laboratory, Lexington, MA) allows highly reliable Hct measurements, compared with microcentrifugation (14). Results of all CEW experiments (in which Hct was measured) are summarized in Fig. 1.

The increases in Hct because of CEW applications, mentioned previously, were similar to increases reported by other investigators, in studies of (i) electrical muscle stimulation in pigs (15) and dogs (16), and (ii) submaximal or exhaustive exercise in pigs (17,18) and dogs (19). In most of these studies, Hct recovered relatively rapidly to baseline levels (e.g., 30 min after the end of exercise [19]).

Heavy exercise will induce significant acute increases in Hct and blood viscosity (20) in human subjects. These alterations are often accompanied by changes in several other hemorheological factors, including (but not limited to): (i) increased red blood cell (RBC) aggregation (caused, in part, by increased concentration of plasma proteins), (ii) increased RBC “aggregability” (intrinsic tendency of an RBC to aggregate), (iii) decreased ability of RBCs to disaggregate, and (iv) impaired RBC deformability. The effects are dependent on the intensity, duration, and type of exercise, and on the athletic capacity of an individual subject.

As exercise-induced hemolysis is essentially because of mechanical trauma directly on RBCs (e.g., from recurring impact of hands or feet with unyielding surfaces; [21]), such hemolysis might not be expected after CEW applications in controlled experiments. No gross hemolysis was generally observed in the spun plasma samples of previous CEW studies (3–8).

Specific Questions when Reviewing Results

Is Porcine Blood an Acceptable Model for Human Blood?

Studies of Hct changes in human subjects after CEW applications had not been accomplished at the time of this technical note. Although the Hct of physiological porcine blood is commonly lower than human blood (22), porcine blood (compared with, e.g., bovine and ovine blood) exhibits strongly non-Newtonian behavior at different Hcts. Thus, porcine blood, rheologically, is very comparable with human blood (23). The viscoelasticity of porcine blood was similar to that of human blood at a wide range of Hct values and shear rates (23). The compressibilities of human and porcine blood, as measured by a highly sensitive density measuring system (24), were also similar.

Brinkman et al. (25) classed animal species into four different types on the basis of their degrees of RBC aggregation. Humans and swine were listed in the same group (“moderate rouleaux formation”). Plasma concentrations of total protein and of fibrinogen (each of which can influence RBC aggregation) were similar in swine and humans (26). Weng et al. (27) found that, in addition to exhibiting aggregability similar to normal human RBCs, the shear rate necessary to disrupt aggregates was similar for porcine compared with human RBCs.

In terms of specific RBC deformability values, the swine was more comparable with the human than seven other species studied (28). Porcine RBCs have a normal biconcave shape and a cell diameter that is much more analogous to the human RBC than bovine or ovine RBCs (23). Although RBCs of the echinocyte type (which are poorly deformable) are often described in porcine blood, these are usually observed in peripheral blood smears *in vitro* (29).

What are the Potential Mechanisms of Changes in Hct During Muscle Contraction Owing to CEW Applications?

Potential mechanisms mentioned below may be applicable to both pigs and humans exposed to CEWs. As repeated or long-duration applications in humans would rarely be approved by institutional review boards, animal models are a potential source of useful information.

During exercise, increased blood pressure and increased action of muscle contraction on venules can cause increased capillary hydrostatic pressure, with plasma fluid being pushed into the extravascular space (30). In addition, muscle contraction can result in the release of metabolic waste products (e.g., lactate, phosphate, and potassium), which increase the osmolality of the interstitial space. The resulting osmotic force would pull plasma fluid from the vasculature into the extravascular space (31). A fluid shift because of sweating, such as during exercise, might not occur to the same extent during CEW applications.

One important mechanism of increased Hct during exercise may be splenic contraction. During exercise or stimulation with epinephrine, pigs can exhibit an increased Hct because of contraction of the spleen (32). As most information regarding this “reservoir function” of the spleen has been obtained from animal studies, the contribution of the organ to humans’ circulating blood volume has often been considered to be unimportant. During exhaustive exercise, however, such contraction can account for approximately 25% of any increase in Hct (33). During isometric handgrip exercise in humans, the splenic volume has been reduced as much as 18% during the first minute of exercise (34).

Repeated breath holds in humans can also cause increased Hct via contraction of the spleen (35). Hypoxia can play a role in splenic contraction during apnea, possibly via chemosensor-related sympathetic activity (36). In experiments of anesthetized pigs (4–8), there was interruption of breathing during CEW application. Moreover, oxygen saturation significantly decreased in some of those studies (4,8). Even without hypoxia, other factors may trigger spleen contraction (36). In addition to splenic contraction during exercise, there may also be redistribution of RBCs in other vascular beds (37).

Although the use of some anesthetics may lead to increased spleen size (at least in dogs; [38]), propofol, one of the anesthetics used in previous CEW experiments (3–8), can result in decreased spleen size (and, consequently, greater Hct; [38]). Notwithstanding this point, preexposure Hct values in the CEW experiments were within a normal range reported for swine (see, e.g., [8,22], for further discussion). It is conceivable that, if other anesthetics were used, increases in Hct may have been even greater (because of a potentially larger spleen preexposure, which could have been capable of ejecting more RBCs into the circulation).

What are the Potential Detrimental Effects of Acutely Increased Hct and Other Concurrent Factors?

Increased Hct and other hemorheological alterations may contribute to problems with tissue perfusion (39). Increased viscosity can reduce venous return and cardiac output (40). At higher Hcts, blood viscosity may play a greater role in cardiac-output regulation than other factors, such as vascular resistance (41). Although polycythemia increases blood oxygen content, this potential benefit is counterbalanced by a decrease in cardiac output, which will, in turn, adversely affect pulmonary diffusion and alveolar ventilation (42). Regardless of whether exercise is (i) short term and heavy versus (ii) longer duration but more moderate, there is a similar risk of

sudden cardiac death as a result of increased blood viscosity (43). Acute exercise resulted in an increased blood viscosity of as much as 81% (44) in human subjects.

Marini et al. (45) noted, in swine, “all other factors being the same, a Hct greater than 35% may cause a decrease in blood flow rate and change in blood flow characteristics as a consequence of increased blood viscosity, which may alter and compromise cellular oxygen transfer” (p. 108). In 10 of the 13 series reported in Fig. 1, post-CEW-exposure values of Hct were above 35%. At high Hcts, a large number of vessels in the microcirculation can become stagnant because of packed RBCs (46). Despite this, of the five groups in Fig. 1 with post-CEW-exposure values of Hct above 45%, two were groups of animals that survived.

In an *in vivo* dog-muscle preparation (47), an increase in Hct from 42% to 55% was associated with a 35% decrease in blood flow in nonexercising muscle. In working muscle, however, rhythmic muscle contractions appeared to counteract the disadvantageous effects of increased Hct (and increased blood viscosity) on blood flow. As mentioned previously (8), intermittent (as opposed to sustained long duration) CEW applications would allow reintroduction of blood flow between contractions, potentially minimizing detrimental effects. Propulsion of blood from skeletal muscle vasculature, however, would be expected to be less efficient during long-duration CEW applications than during exercise (particularly during locomotory exercise with rhythmic sequential lengthening and shortening contractions of different muscle groups [48]).

Excessive RBC aggregation (which often occurs concurrently with increased Hct), up to a certain point, can actually reduce viscosity and assist in ease of flow. This is because of the creation of a cell-free zone surrounding a central core of aggregates. With a more extreme increase in Hct, however, vascular obstruction can occur (49).

RBC deformability is important because even at high Hcts, such deformability permits blood to remain fluid (50) and allows RBCs to pass through capillaries. RBCs with impaired deformability may cause decreased oxygen diffusion capacity (51). Decreased RBC deformability can cause significant elevations in resistance to blood flow in the pulmonary system (37).

Exercise-induced Hct increases are often correlated with increased blood lactate levels (37). Blood lactate increased after CEW applications in both animal models and human volunteers (summarized by Jauchem [52]). Lactate can impair RBC deformability in untrained subjects (37).

Acute increases in Hct can have rapid major effects on other physiological factors (e.g., renin release [53]). A high Hct may be associated with disseminated intravascular coagulation (54). The time course required for this effect, however, may be relatively long compared with most potential CEW applications. In swine, higher baseline Hct predicted early death because of sepsis (55). Sepsis, however, will involve other factors different from those involved during muscle contraction caused by CEW applications.

Considerations During Law Enforcement Use of CEWs

As immediate “direct electrical effects” of CEWs (e.g., ventricular fibrillation) are unlikely (2), other more “indirect effects” (due, e.g., to repeated muscle contractions) on physiology are of interest. One of these effects is increased Hct. Unfortunately, when death occurs in conjunction with CEW applications during law enforcement operations, blood samples may not be obtained in a timely fashion. As Hct values of the blood increase rapidly after death (56), a forensic investigation into this phenomenon would be problematic.

Unlike transient increases in Hct identified by other investigators after exercise in human subjects (49,57) or dogs (19), Hct in some of the CEW studies remained elevated for at least 2.5- or 3-h post-exposure (5,7,8). Whether or not these differences were because of variations in the degree of muscle contraction from the different modes of stimulation is unknown. Experiments of extreme (i.e., long duration or repeated) exposures to CEWs cannot ethically be performed in human subjects under controlled conditions.

It is unknown whether CEW exposure would exacerbate any preexisting polycythemia (or associated changes) that could be present during apprehension of unlawful subjects (from, e.g., an endocrinopathy [58], psychological stress [59,60], diabetes [61], cocaine use [62], chronic alcohol abuse [63], or use of opiates [64]).

Conflict of interest: The authors have no relevant conflicts of interest to declare.

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