INTRODUCTION:

The TASER® electronic control device (ECD) is used to control violent/agitated behavior in two ways. The primary method is probe deployment. The secondary method is the “Drive Stun” (DS) which produces a painful stimulus. This project is the first to study the human effects of the DS. ECDs are scrutinized since individuals occasionally die unexpectedly following their use. Some deaths have occurred after a DS. There are cases of custodial sudden deaths when no ECD has been used, but a causal relationship is hypothesized.

METHODS:

Volunteers underwent a 24 hour monitoring process. After informed consent, a health history and baseline bloodwork was obtained. Subjects then received either a 15-second or two consecutive 5 second DS applications. Applications were to the neck/shoulder region using a TASER X-26 ECD. Bloodwork was obtained after exposure and again at 8 and 24 hours after exposure. Samples were analyzed for: BUN/Creatinine ratio, Potassium, CK-MB, Lactate, and Troponin I.

DISCUSSION:

Recent animal studies have raised the question of whether CEW application can capture underlying heart rates or induce arrhythmias.3,4 The limitations of these animal model studies are significant and include size limits, probe placement limits, use of deep sedation/anesthesia and other factors that may make their methodology unrealistic.

Our use of adult humans in an exhausted state is believed to be a better model in which to study this question.

RESULTS:

21 subjects enrolled (98.5% male, mean age 40.3 years + 6.8, range 29 to 55, mean body mass index 28.4 + 3.5, range 21.1 to 36.8), 11 had the single continuous exposure and 10 had the 2 shorter exposures. Repeated measure ANOVA showed no significant change from baseline at the four time points or between exposure types for BUN/ Creatinine ratio (mean value 14.8 + 3.7, range 6.6 to 23, p=0.40), serum potassium (mean value 4.0 mEq/L + 0.4, range 3.0 to 5.1, p=0.26), or serum CK-MB (mean baseline value 2.45 ng/ml + 2.89, range 0 to 20.3, p=0.32).

A significant decrease in serum lactate occurred from baseline at the 8 hour time point (p=0.005, baseline mean 1.87 mmol/L 95% CI 1.39 to 2.35, immediate post exposure mean 1.35 mmol/L 95% CI 1.04 to 1.65, 8 hour mean 1.06 mmol/L 95% CI 0.92 to 1.2, 24 hour 1.22 mmol/L 95% CI 1.1 to 1.4). All troponins were <0.2 mcg/L.

CONCLUSIONS:

There were no worrisome changes in the measured serum biomarkers. There was a significant decrease in serum lactate after exposure.

This data does not support a causal relationship between ECD DS exposure and worsening physiology.