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EXCITED DELIRIUM AND ITS CORRELATION TO SUDDEN AND UNEXPECTED DEATH PROXIMAL TO RESTRAINT

Date: 2004 - 09

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EXCITED DELIRIUM AND ITS CORRELATION TO SUDDEN AND UNEXPECTED DEATH PROXIMAL TO RESTRAINT

A Review Of The Current and Relevant Medical Literature

By Sgt Darren Laur

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Caveat:

The purpose of this paper is to shed some light, create meaningful dialogue, and encourage further scientific and medical research into the multi-factoral issues surrounding sudden and unexpected death proximal to restraint. Although a rare occurrence in Canada, sudden and unexpected death proximal to police use of force and restraint is a reality that is gaining more and more public attention not just in our country, but internationally as well. Often, the causes of these deaths are attributed by some in the media, special interest groups, and the general public to a specific force option used by police during arrest (Taser, pepper spray, physical restraint), due to the fact that their proximity to death make a cause and effect correlation easier to accept and understand, even when these same force options have been medically discounted as contributing to the cause of death later at autopsy. Looking for causation in sudden and unexpected death proximal to restraint is an emotionally charged subject, which up until very recently had very few answers. However, based upon both the scientific research and the medical literature reviewed for this paper, the writer will be

suggesting that in some cases it may not be the force option used that causes death, but rather a severe and some times fatal medical condition known as, "Excited Delirium".

I am not a medical professional, and as such, I make no medical assumptions or findings in this paper. I am, however, a trained police investigator and thus I have attempted to gather both the scientific and medical research that I believe to be relevant, specific to Excited Delirium, so that those with the expertise can review and hopefully begin to find answers to some of the questions that others and I have in this topic area. What I have learned thus far in my research of both the scientific and medical literature is that the causes of death attributed to Excited Delirium, proximal to restraint, are medically "multi-factoral". What appears to be missing in the literature, however, is a "synthesis" of the scientific and medical opinion from a variety of differing specialties into one document. Each medical discipline mentioned in this paper (Pathologists, Psychologists, Psychiatrists, Pharmacologists, Neurologists, Cardiac Electrical Physiologists, and Exercise Physiologists), appear to hold a piece of the puzzle to the understanding of Excited Delirium, but no one has gathered these opinions, or put the pieces of the puzzles together, into a clearer understanding of the phenomena of death associated with Excited Delirium, thus the reason for this paper.

The information contained in this paper should be shared with all first responders and medical care workers who may come into contact with those experiencing excited delirium. If your agency has experienced a sudden and unexpected death proximal to restraint, this paper should also be shared with investigators, coroners, medical examiners, pathologists, and lawyers or anyone else who is involved in the investigation of the death.

"Research is to see what everyone else has seen, and think what no one else has thought"

Albert Szent-Gyorgy (Nobelish 1927)

INTRODUCTION:

Police receive a call of a male, semi-clothed, yelling and screaming at the top of his lungs and acting very bizarre in the downtown core. Upon arrival, police attempt to communicate with this male unsuccessfully, due to the fact that he is totally incoherent, and appears to be suffering from either a psychosis, or drug induced delirium. As police continue their negotiation with this male, he begins to walk out into traffic, becoming a danger to himself and/or others, at which time the officers attempt to take this male into custody under the authority of the Mental Health Act. Upon physical contact with this male, he immediately begins to fight with police resulting in a protracted physical encounter at the conclusion of which, the subject is finally taken under control and handcuffed. While waiting transport, the suspect is still physically resisting his restraint and upon arrival of the wagon or Provincial Ambulance Service, it is noted that the male has stopped breathing and has no pulse. Attempts at resuscitation by both paramedics and emergency room staff are futile, and the suspect is pronounced dead at hospital. At autopsy, the pathologist finds there is insufficient evidence to establish a cause of death.

Although the above noted hypothetical is "believed" to be a rare occurrence, a very similar incident happened to the Victoria Police Department (Dawson, 1999). After reviewing the mountains of medical literature on the topic of "In-Custody Death", it is estimated that in the United States of America alone, there are between 50 and 125 in-custody deaths of similar circumstances every year (Conner, 2002). This is an estimate

as there are no official statistics available at this time. It should be noted, however, that since January 2003, federal legislation was passed in the United States requiring all law enforcement agencies to not only report, but also categorize all in-custody deaths. It is hoped that the Bureau of Justice Statistics will have a statistical report available, specific to this topic area, in the fall of 2005. In Canada, we too face a similar issue in that there is no central national repository that collects statistics on sudden and unexpected death, proximal to restraint, attributed to Excited Delirium. Although most provincial coroners do keep statistics on sudden deaths attributed to law enforcement, most do not break these deaths down into specific categories. As such, all deaths, including shootings and suicides while in police custody, are lumped into one category. Very recently, however, some provinces such as Ontario are now beginning to capture this data. It is because of this fact, that in Canada, it is believed that on average there are between 6-10 sudden and unexpected deaths, proximal to restraint by law enforcement, yearly across our country, a number that appears to be reflected in the Canadian literature and Provincial Coroner reports reviewed for this paper. It is also interesting to note that of those who die suddenly and unexpectedly proximal to restraint, 77% die at the scene of their arrest, or while being transported to cells or hospital. (Ross, 1998)

For the purpose of this paper, I am not going to refer to the commonly used term ***"In-Custody Death"***, due to the fact that it does traditionally group all deaths while in police custody, including shootings and suicides, into one category. Instead, I am going to

specifically look at the medical condition of Excited Delirium as it relates to ***"Sudden and Unexpected Death Proximal to Restraint."***

HISTORY:

The incidents of sudden and unexpected deaths proximal to restraint first came to the attention of law enforcement in the early 1980's, due to the increased empirical data of deaths associated with police use of force, especially where a suspect was placed into a maximal prone restraint which is better known as being hog-tied. In 1995, the terms "hog-tie and "hobble" became interchangeable (Stratton, 1995) and for the purpose of this paper, need to be differentiated. The maximal prone restraint method (hog-tie), involves securing both wrists and ankles together behind the back, while the "hobble" is the tactic of securing the ankles together (without connecting them to the wrists), to inhibit the subject from placing the soles of their feet in contact with the ground whether they're in a back down or face down prone position. (Lawrence, 2004)

It should also be noted that deaths similar in nature to those occurring in law enforcement are being experienced in psychiatric and geriatric care facilities where patients/residents are required to be restrained for their safety and security (Paterson et al., 2003). In fact, since 1995, there have been 20 reported deaths in U.S. medical facilities as a result of physical restraint being used by medical staff personnel (Joint Commission for Accreditation Of Health Care Organization, 1998)

In 1988 Dr. Reay, a King County medical examiner in Washington State, USA, began to hypothesize that sudden and unexpected deaths proximal to restraint appeared to be associated with something that he and others termed "Positional Asphyxiation". Positional Asphyxiation was associated with a suspect being hogtied after being physically restrained, hands and feet secured behind the back, and the suspect placed in the prone position (Maximal Prone Restraint). Dr. Reay in his research, concluded that such a restraint, associated with the prone position, was responsible for sudden and unexpected deaths proximal to restraint due to asphyxia (Reay, Flinger, Stillwell, & Arnold, 1992).

Due to Dr. Reay's research, many law enforcement agencies around the world began to prohibit the use of the hog-tie restraint, and provided training to their members on the issues surrounding "Positional Asphyxia", in an attempt to combat the incidence of sudden and unexpected deaths proximal to restraint. Although Dr. Reay's research was ground breaking, including his recommendations to law enforcement, subjects were still dying suddenly and unexpectedly in police custody in the United States and Canada, even though not placed in a face down hog-tied position.

In 1998, a lawsuit was decided in the state of California, Price v. the County of San Diego. Dr. T. C. Chan, Dr. G. M. Vilke and Dr. Tom Neuman, physicians from the Department of Emergency Medicine, University of California San Diego Medical Center replicated the Reay study (Chan, Vilke, Neuman, & Clausen, 1997) and came to

conclusions that did not support the original research conducted by Reay. The results of these research efforts were presented during this trial. The result can best be described by a quote from the court's decision:

> *After Dr. Reay's retraction, little evidence is left that suggests that the hog-tie restraint can cause asphyxia. All of the scientists who have sanctioned the concept of positional asphyxia have relied to some degree on Dr. Reay's work. The UCSD study has proven Dr. Reay's work to be faulty, which impugns the scientific articles that followed it. Like a house of cards, the evidence for positional asphyxia has fallen completely (Ann Price et al., Plaintiffs, v. County of San Diego et al., Defendants, 1998).*

The problem that exists is that subjects are still dying, suddenly and unexpectedly, in circumstances similar to the case involving Daniel Price. It should be noted that in the above noted court case, Dr Reay did not retract his belief that the prone hog-tie position was dangerous. What Dr Reay did admit to, was that his methodology in his studies may have been flawed.

A further 1998 retrospective study looked at 61 cases of sudden and unexpected deaths proximal to restraint (Ross, 1998). Dr. Ross found that only 38% of all subjects who had died had been placed in the prone hog-tied position, due to this fact, he reported that Excited Delirium was more related to the sudden deaths than to the restraint position, because apparent positional asphyxiation during restraint accounted in only 20% of the 61 deaths reviewed.

A further 1999 research study that looked at the effects of positional restraint on heart rate and oxygen levels (Schmidt & Snowden, 1999) found that healthy persons, even

after physical exertion, are at little risk when held in the hog-tie position and placed onto their side.

Most reported cases of sudden and unexpected death proximal to restraint involve young men in an "excited" state or one of "agitated delirium" as a result of psychiatric illness or intoxication from illicit drug use. These individuals were combative, violent, and often struggled or suffered traumatic injuries as a result of a confrontation with law enforcement before their placement in the restraint position (Chan, Vilke, & Neuman, 1998). Again, it is important to note in the review of the medical literature, that subjects used in testing, including those in the Reay, Chan, and Snowden studies, were in fact healthy adult volunteers who were not representative of restraint subjects in medical and law enforcement settings, that are experiencing an Excited Delirium because of psychosis or drug, and due to this fact, some medical professionals questioned the validity of these studies. As an example, in Dr Chan's research (Chan, Neuman, Clausen, Eisele & Vilke, 2004) the following test criteria were utilized:

- Test subjects were ten healthy male volunteers between 18 and 45 years of age.
- The weights utilized in testing, and placed separately between the shoulder blades of the volunteers, were 25lbs and 50lbs each.
- Subjects were not physically stressed maximally prior to the application of restraints and weight loads.
- Once restrained, test subjects did not physically resist their restraints, but rather passively submitted to testing.

In contrast to Dr. Chan's research criteria, in most cases of sudden and unexpected death proximal to restraint reviewed for this paper, the subjects restrained were:

- Experiencing an Excited Delirium due to psychosis or drug.
- Most had "maximally" resisted arrest and restraint procedures prior, during, and after restraint, thus placing increased loads on their ventilation/breathing process.
- On average, in policing circumstances, there are usually between 3-5 officers involved in the restraint process, with a total combined weight far greater than the 25lbs and 50lbs utilized in Dr. Chan's research.

Based upon the above noted research, it is no wonder that some in the medical field are of the opinion that the correlation of Dr. Chan's research to actual street application and conditions is somewhat questionable. In fact, Dr. Chan and his co-authors stated:

> *" Our study has limitations. First, as this was a laboratory physiology study, we could not reproduce all conditions encountered in the field setting with such cases. In particular, we did not simulate trauma, struggle, drug intoxication, and other physiologic and psychologic stresses that commonly occur with individuals who are being restrained in the field setting. Second the amount of weights selected for this study may not reproduce the actual amount of weight force used on individuals during the restraint process." (Chan, Neuman, Clausen, Eisele & Vilke, 2004 pg 118)*

THE MODERN EMERGENCE OF EXCITED DILIRIUM:

In 1998, the Ontario Coroner's Office published a retrospective study of 21 cases of unexpected death in people with Excited Delirium that occurred between 1988 and 1995

within the province of Ontario (Pollanen, A., Cairns, & Young, 1998). Of the cases reported, 18 deaths occurred while the subject was in police custody. In all 21 cases, Dr Pollanen found that "many deaths related to Excited Delirium are associated with restraint in the prone position"(p. 1607) and that all of the subjects who died had lapsed into "tranquility" shortly after being restrained. Other findings in Dr Pollanen's medical research included:

- Twelve (12) subjects (57%) experienced Excited Delirium caused by a psychiatric disorder
- Eight (8) subjects (38%) experienced cocaine induced psychosis
- Eighteen (18) of the deaths (86%) happened while in police custody and could not be resuscitated
- Eight (8) of the 18 (44%) people restrained in the prone position also suffered chest compression from the body weight of 1 to 5 people who were restraining them.
- Four (4) (19%), had been pepper sprayed
- Four (4) (19%) had heart disease at the time of death
- Two (2) (10%) of the deaths happened in hospital after being in a coma for several days
- Six (6) people with cocaine Excited Delirium had cocaine levels similar to recreational users and lower than those who actually died from cocaine intoxication

- Levels of cocaine associated with recreational use may be sufficient to cause Excited Delirium.
- Of the deaths, none were Taser related.

Another study, involving the examination of the sudden and unexpected deaths of 21 males, occurring between 1992 and 1996 (O'Halloran & Frank, 2000) reported:

- One death associated with Taser use, the rest with other force options;
- Best estimates for the time held in a prone position was 2-12 minutes;
- Eight (8) had a history of mental illness excluding substance abuse;
- Eight (8) had a history of substance abuse;
- Seventeen (17) appeared to be "acutely delirious";
- Eleven (11) had stimulant drugs in their system;
    - 8 cocaine (concentration ranged from 0.02 to 5.4 mg/L [mean, 1.4mg/L])
    - 2 methamphetamine
    - 1 had both cocaine and methamphetamine/amphetamine
- Six (6) could be considered obese while 6 were normal weight
- At postmortem, temperature was only taken in 3 cases with one reported to be hyperthermic (the relevance of hyperthermia will be discussed later in this report).
- Six (6) were noted to be sweaty prior to death

This was the first report the author found that mentioned the correlation between dopamine levels specific to cocaine, bi-polar disorder, and schizophrenia ***(this too will be mentioned later in this paper).***

A third research project related to the sudden and unexpected death of subjects requiring restraint for Excited Delirium (Stratton, Rogers, Brickett, & Gruzinski, 2001) reported the following factors:

- Study period between 1992-1998 in the Los Angeles area;
- Looked at 18 deaths resulting from 216 arrests made of subjects requiring restraint for Excited Delirium;
- 198 Excited Delirium subjects who were physically arrested and hobbled ***DID NOT*** die;
- Unique to these data is a description of the initial cardiopulmonary arrest rhythm in 72% (13) of the sudden death cases;
- Associated with the 18 deaths was a struggle by the victim which resulted in forced restraint;
- 78% had stimulant drugs in their system;
- 56% had chronic disease;
- 56% were classified as obese;
- All cardiopulmonary arrests were unanticipated and proceeded by a short period (estimates 5 minutes or less) during which the victim ceased in struggling against restraints and developed a labored or shallow breathing pattern.

- Of the 18 incidents, 5 were Taser related
- Remaining 13 deaths, other force options, excluding firearms, were used
- Report mentioned "severe metabolic acidosis" specific to Excited Delirium although the presence of metabolic acidosis was not determined in this study.

As a result of the above-published medical studies and research, as well as the questions being raised specific to the medical validity of positional asphyxia, the medical community now began to focus on research surrounding the biological and physiological effects of a new medical phenomena that they termed "Excited Delirium" specific to sudden and unexpected death proximal to restraint.

Although identified as a new medical phenomenon in law enforcement, as mentioned earlier, problems similar to Excited Delirium have been reported in the medical literature since the mid 19th Century (Bell, 1849). In 1849, Dr. Luther Bell, physician and Superintendent of the McLeon Asylum for the Insane in Somerville Ma, was the first medical professional to describe Excited Delirium stating, "Victims of this organic mental disorder may be apathetic or depressed, or excited with fear or rage accompanied by sympathetic nervous system arousal." Dr. Bell had spent more than twelve years treating those admitted to his hospital. From 1836 to 1848 Dr. Bell admitted over 1700 patients and among those, 40 cases manifested a "peculiar" form of delirium. At least three-quarters of these cases, according to Dr. Bell, terminated fatally, with the remainder recovering fully.

A further report from the UK (Paterson et al., 2003) outlined the following information specific to death proximal to restraint in medical institutions:

- In mental health, before effective treatment for the acute phase of mania or psychosis was available, death as a consequence of exhaustion in patients was not uncommon.
- In a South Carolina hospital from 1915-1937, there were 360 deaths in which the cause was listed to be, "Exhaustion due to mental excitement".
- In 1946, Dr. Shulack appears to be the first medical professional to describe this phenomenon as "sudden exhaustive death in excited manics".
- In a 1952 study by Bellak et al, they describe the onset and symptoms of this syndrome as:
    - Sustained motor and mental excitement with continued activity for a period of time
    - Rapid, thready, pulse
    - Profuse clammy perspiration
    - Fall in blood pressure
    - Hyperthermia
    - Delirium and death

(Note: very similar to what we are seeing now as will be mentioned later in this paper):

The literature review conducted to date confirms that Excited Delirium death, associated proximal to restraint, is not just a phenomena experienced by law enforcement, but also in psychiatric and geriatric care facilities (Joint Commission for Accreditation of Healthcare Organizations, 1998). In this published report, researchers found a total of 20 deaths associated with physical restraint in hospitals, psychiatric care facilities, as well as geriatric care facilities in the United States. A further report found in the Cormorant (Weis, 1988) reported about 145 deaths in chronic care facilities. As well, in a report authored by The Office of the Ombudsman for Mental Health and Mental Retardation in Minnesota (Office of The Ombudsman For Mental Health and Mental Retardation, 2004), they located 142 "reported" restraint associated deaths in mental health facilities between 1988 and 1999. As can be appreciated, sudden and unexpected death proximal to restraint is not just a policing concern, but rather an important factor for any occupation where the restraint of those experiencing Excited Delirium is a reality; some of those fields of work include; paramedics, fire first responders, emergency room staff, as well as psychiatric and geriatric care workers.

CURRENT RESEARCH INTO EXCITED DELIRIUM:

The reasons for sudden and unexpected Excited Delirium deaths proximal to restraint are very complex and multi-factoral and as such, new research is coming to light every few months. In Canada, Chris Lawrence (Ontario Police College) working with other medical experts such as Wanda Mohr (Associate Professor, Psychiatric Mental Health Nursing,

University of Medicine and Dentistry New Jersey) as well as physicians has been conducting groundbreaking research into the medical literature associated with Excited Delirium. These authors have also developed an investigator protocol for police relating to the types of death associated to Excited Delirium (Lawrence & Mohr, 2004). In the United States an overview of the situation relating Excited Delirium to cocaine is available (Barkley Burnett & Adler, 2004).

According to Lawrence, sudden and unexpected deaths proximal to restraint fall into two specific categories:

1. Subjects die from conditions that leave evidence readily apparent at autopsy, and;

2. Subjects die and there is insufficient evidence to establish a cause of death at autopsy.

According to the medical literature reviewed for this paper, there appears to be three specific groups of people who are most prone to sudden and unexpected death proximal to restraint attributed to Excited Delirium:

1. Those who are suffering from psychiatric illness (bipolar, schizophrenia). This is also noted in a study where both agitated and non-agitated subjects suffering from schizophrenia died suddenly and unexpectedly (Rosh, Sampson, & Hirsch, 2003).

2. Those who are chronic illicit stimulant substance abuse users (cocaine, methamphetamines) including marijuana (for those who are bipolar or schizophrenic) and alcohol.

3. Combination of mental illness and substance abuse.

The common outward presenting body autonomics related to excited delirium include:

- Unbelievable strength
- Impervious to pain
- Able to offer effective resistance against multiple officers over an extended period of time (one case in Calgary Alberta Canada, multiple officers wrestled with a person who was suffering an excited delirium for over fifteen minutes)
- Overheating (hyperthermia)
- Sweating
- Bizarre and violent behaviour
- Aggression
- Hyperactivity
- Extreme paranoia
- Incoherent shouting

When police officers are dealing with those experiencing Excited Delirium, both manias specific to psychiatric illness as well as drug induced psychosis, often present outwardly

in the same manner. Based on the basic level of medical training provided to police officers, it is unreasonable to expect them to make any medical assessment to differentiate between the three causes of Excited Delirium. In fact, Dr Lohrasbe (Victoria based Forensic Psychiatrist) stated to the writer that, "Excited Delirium is not one entity or a disease, but rather a symptom cluster which causes both paranoia and control over-ride."

Based on the literature reviewed, it seems reasonable that Excited Delirium causes a person's sympathetic nervous system to shift into hyper drive. Such a shift may in fact occur long before police arrival. It may be that the subject is near physiological exhaustion even though they may not present so upon physical arrest. When combined with the fact that these subjects may have to be physically restrained, police may be compounding the effects of the sympathetic nervous system to the point that the body, more specifically the heart, is unable to sustain the exertion.

As stated earlier, the causes of Excited Delirium are multifactoral. No one really knows for certain what is happening. Current medical research has been summarized well within the eMedicine article (Barkley Burnett & Adler, 2004). In the literature, current medical research indicates some of the following medical concerns play a contributory role, or synthesis, when it comes to Excited Delirium as it relates to sudden and unexpected deaths proximal to restraint:

Contributing Factor #1: Cocaine toxicity and Dopaminergic effect to heart muscle

It is widely accepted in the medical community that both illicit drugs, especially cocaine, as well as some psychotic prescription drugs, may cause the heart to be much more susceptible to an arrhythmia **(**Straus, Bleumink, Dieleman, 2004). Long term use of cocaine markedly increases norepinephrine raising the fact that long-term cocaine users, should they also accumulate excessive norepinephrine, may be primed for a malignant arrhythmia (Barkley Burnett & Adler, 2004). Other conditions that provide such an anatomic substrate include Wolfe-Parkinson-White syndrome and left ventricular enlargement. Even low levels of cocaine in a person's system can cause tachydysrhythmias. (Barkley Burnett & Adler, 2004)

Contributing Factor #2: Cocaine toxicity to brain (hyperthermia)

In the hypothalamus, chronic cocaine use causes issues in the brain preventing it from clearing dopamine from the synapses resulting in delirium (Barkley Burnett & Adler, 2004) . Due to the fact that dopamine also plays a role in the regulation of core body temperature, increased dopaminergic neurotransmission "may" contribute to psychostimulant-induced hyperthermia. It is hypothesized that hyperthermia "may" result from extensive muscular activity in the setting of warm ambient temperature and, perhaps, humidity (summer months or even hot rooms with poor ventilation) in combination with aberrant thermoregulation in the hypothalamus and mesolimbic system. Chronic cocaine use only multiplies this hyperthermic reaction to very dangerous levels.

This is one reason why in those who die from excited delirium, at autopsy, D1 receptors in the brain are not affected upon medical examination. (Barkley Burnett & Adler, 2004)

The above noted is also supported by the National Association Of medical Examiners (Stephens, Jentzen, Karch, Wetli, & Mash, 2004). In this medical paper, several doctors found that chronic drug use is necessary to induce the changes in the neurochemistry that lead to Excited Delirium. The presence of hyperthermia (core temp>103 degrees F, 41 degree C) is strongly supportive of cocaine induced excited delirium. This position paper also went onto to say, " a catecholamine – mediated excited delirium, similar to cocaine, is becoming increasingly recognized and had been detected in patients with mental disorders taking anti-depressant medications. In psychotic patients who have stopped taking their anti-depressant medications the neurochemistry is similar to the effects of cocaine." Again, this study draws a correlation of why bipolar/schizophrenia subjects, as well as those who are chronic users of cocaine, methamphetamine, and other illicit stimulants are prone to excited delirium.

To further support the above noted correlation between Excited Delirium and those suffering bipolar/schizophrenia or chronic cocaine and methamphetamine use, Dr Bayla Schecter, regional medical advisor mental health and addictions Vancouver Island, shared in a lecture called "Balancing the Brain: Mental Health, Medications and Substance Abuse" , those suffering bipolar/schizophrenia and or chronic users of cocaine or crystal methamphetamine have one important similarity; both groups may experience huge releases of Dopamine in the brain. Dr Schecter further stated that Dopamine not only

controls thermoregulation in the body, but it is also one of the main neurotransmitters for the fight/flight response. (Schecter, 2004). Again, Excited Delirium appears to be generated by increased intrasynaptic dopamine concentrations resulting from a defect in the regulation of the dopamine transporter. When compared to drug-free controls, cocaine recognition sites on the striatal dopamine transporter are increased in cocaine users without Excited Delirium. Persons dying of Excited Delirium have no such increase; thus, they may have problems clearing dopamine from the synapses, a condition that easily could result in agitation and delirium. (Barkley Burnett & Adler, 2004)

Another reported factor that may cause Excited Delirium that is mentioned in some of the medical literature reviewed, and briefly mentioned in the previous paragraph, is the fact that it is not just the levels of drugs ingested (illicit or prescribed), but it is also possible that the sudden, self-imposed cessation of prescribed medication (non-complaint) causes the subject's mental condition to deteriorate to a point where psychosis results. This deterioration can occur over a short or longer time period.

Contributing Factor #3: Cocaine Associated Rhabdomyolysis (CAR)

Rhabdomyolysis can be caused by severe over exertion of muscle (such as struggling with police or continued struggle against restraint once in custody) and can also be caused by many drugs of abuse including alcohol, as well as certain types of prescribed medications.(A.J. Ruttenber et al,1997, A.J. Ruttenber, McAnally & Wetli, 1999)

According to some of the medical literature reviewed, once rhabdomyolysis begins, muscle cells break down and allow the contents of the cell to leach their contents into the blood stream making the heart much more susceptible to arrhythmia due to alterations in the potassium and sodium levels of the blood.

It is hypothesized (Barkley, Burnett & Adler, 2004) that long-term cocaine use, rather than short-term use, is responsible for persistent changes in dopaminergic function that places users at risk for both Excited Delirium and CAR. Elevations in muscle enzymes levels are observed in asymptomatic chronic cocaine users and in untreated persons with schizophrenia; this evidence lends support to the hypothesis that chronic alterations in dopaminergic function can affect skeletal muscle physiology.

Contributing Factor #4: Metabolic Acidosis:

In 1999, Hick, J et al raised awareness of the relationship between Metabolic Acidosis and its effects possibly contributing to sudden death during restraint (Hicks, Smith, & Lynch 1999). Hick's Study found that there might be exacerbation of exercise induced lactic acidosis by sympathetic induced vasoconstriction, which could be enhanced by cocaine and other CNS stimulants. Due to the fact that the literature reports that delirium, both by psychosis or drug, may alter pain sensation, it allows for physical exertion far beyond normal physiological limits and may result in a severe acidosis with maximal sympathetic discharge.

Contributing Factor #5: Neuronal Catecholamine Release:

During violent activity there is going to be an abundant release of catecholamines into the blood stream that, according to the literature reviewed, can sensitize the heart and promote rhythm disturbances. It is also reported in the literature that catecholamines enhance the toxicity of cocaine, which can lead to seizures, respiratory arrest, and cardiac arrest (Mets, Jamdar, and Landry, 1996)

In a further research paper which looked at post exercise sudden deaths, (Dimsdale, Hartley, Guiney, Ruskin and Greenblatt, 1984), specific to Catecholamine release, reported the following:

> "These biochemical abnormalities, although present only transiently during the post exercise period may contribute to the vulnerability of the metabolically stressed myocardium to other arrhythmogenic factors, such as coronary insufficiency or ischemia. One well-documented effect of a reduction in plasma potassium concentrations that may affect cardiac vulnerability to arrhythmias is the increase in vascular resistance caused by reductions in potassium, especially in the presence of high levels of catecholamines. If the coronary arteries constrict in response to the sharp fall in potassium after exercise, the risk of arrhythmia would be elevated in subjects whose coronary perfusion already was limited by pre-existing disease."

Contributing Factor #6: Antipsychotic Drugs and Sudden Death:

Due to the fact that police are often called to assist with the restraint of those experiencing a psychotic event in hospital, investigators should know that there appears to be a link between Antipsychotic drugs used to "chemically restrain" violent patients, and sudden cardiac deaths (Straus SM, Bleumink GS, Dieleman JP, King JH & Stricker BH, 2004). It has been reported in the medical literature that three antipsychotic medications have an increased correlation to sudden and unexpected death in patients experiencing Excited Delirium type events; Haloperidol, Droperidol and Thioridazine. All three antipsychotic medications are reported to cause QTc interval prolongation and, on occasion, torsade de pointes and death. (Glassman AH, Bigger JT, 2001).

Another rare, but well documented, contra-indicator surrounding the use of antipsychotic drugs is a condition known as Neuroleptic Malignant Syndrome (NMS). NMS is a rare, but life-threatening, idiosyncratic reaction to a Neuroleptic medication such as prochlorperazine (Compazine), promethazine (Phenergan), clozapine (Clozaril), risperidone (Risperdal). The syndrome is characterized by fever, muscular rigidity, altered mental status, and autonomic dysfunction. The incidence of mortality, once reported at 20-30% is now estimated at 5-11.6%. Deaths usually results from respiratory failure, cardiovascular collapse, myoglobinuric renal failure, arrhythmias, or diffuse intravascular coagulation. (Benzer, 2002)

Contributing Factor #7: Genetic Susceptibility to Arrhythmia

Very recently, several studies (Lehnart et al., 2004: Priori & Napolitano, 2004: Vos & Paulussen, 2004) have reported that the identification of the molecular determinants of inherited arrhythmogenic disease has been pivotal to the understanding of several aspects of cardiac arrhythmias and sudden death. These researchers have found that there is a wide spectrum of clinical phenotypes caused by abnormal genes encoding for transmembrane cardiac ion channels that can cause sudden death. Here in Victoria during the coroner inquest of Anthony Dawson, a local medical geneticist, Dr Patrick MacLeod, found a rare gene in a specific First Nations family that makes one more susceptible to the negative effects of Excited Delirium.

Contributing Factor #8: Hypertrophic Cardiomyopathy:

"Hypertrophic Cardiomyopathy (HCM) is a complex but relatively common form of genetic heart muscle disease that occurs in 1 out of 500 people, but often goes undiagnosed in the community, and has caused some confusion to both patients and physicians periodically over the years. Hypertrophic Cardiomyopathy is the most common cause of heart-related sudden death in people under 30 years of age, and it can also be responsible for exercise disability at almost any age. HCM occurs equally in both sexes and has been reported in many races and is in fact the single most common cause of sudden death in otherwise healthy young people. (Maron, 2002)

Contributing Factor #9: Face Down Prone Restraint Proximal To Arrest:

It is interesting to note, from an empirical investigative standpoint, that in the majority, ***but not all***, of sudden and unexpected deaths proximal to restraint involving a subject experiencing Excited Delirium, that most subjects had been restrained and left in a prone position. Although Dr Reay's research, specific to positional asphyxia, has been put into question by Chan, Snowden, and Ross's, independent research, (remember that Dr. Chan's research has been medically questioned and challenged as well) there still appears to be some medical or physiological issues with restraining a subject who is experiencing Excited Delirium, for an extended period of time, in a prone position.

One keystone which was identified, and that appears periodically in the medical literature surrounding Excited Delirium, is the dangerous lowering of pH. If blood pH drops too low then death, attributed to cardiac arrest, is a certainty if pH is not corrected (Ortega-Carnieer, Bertos-Polo, & Gutierrez-Tirado, 2001). Some medical literature reviewed for this paper points to the fact that pH can be affected either metabolically through acidosis (very common in Excited Delirium) or through hypoventilation, a state in which a reduced amount of air enters the alveoli in the lungs, resulting in decreased levels of oxygen and increased levels of carbon dioxide in the blood. Causation of hypoventilation can be due to breathing that is too shallow hypopnoea or too slow bradypnea or to diminished lung function. (MedicineNet, 2004) Some medical literature also points to the fact that hypoventilation can be drug induced via dopamine (Zapata, 1980; Bisgard,

Forster, Klein, Manohar, and Bullard, 1980), cocaine use (Bauman JL, DiDomenico RJ, 2002; Kolecki PF, Curry SC, 1997; Wilson LD, Shelat C, 2003) or through body positioning especially after exercise (Haouzi, Chenuel, & Chalon, 2002). Again, it should be emphasized that according to the medical literature reviewed, hypoventilation can be caused by a number of factors and not just body positioning. As a police investigator this is an important point due to the fact that persons who die, suddenly and unexpectedly proximal to restraint, ***are not always kept in a face down prone position after control is obtained.***

Because we know that body position affects the control of breathing in exercise by altering the coupling between ventilation and pulmonary gas exchange, is it possible that the prone restraint interferes with this fundamental tenet of blood gas homeostasis for those in an Excited Delirium state who are kept in a face down prone position? In discussions with Dr Christine Hall, Program Director, FRCP Program in Emergency Medicine, during a seminar on Excited Delirium hosted by the Calgary Police Department, she hypothesized that hypoventilation may be contributing to a fatal shift in blood pH. In her hypothesis, individuals suffering from Excited Delirium who are restrained in a prone position may be unable to breathe rapidly enough to exchange carbon dioxide. Although these individuals have a clear airway and can speak, the restraint prevents them from breathing at a rate necessary and the excess carbon dioxide contributes to an academic state.

Given that a person who is experiencing Excited Delirium will likely have a sympathetic nervous system that will be kicked into hyper drive, coupled with the fact that there will likely be a protracted physical struggle (causing hyperventilation) with police resulting in some kind of physical restraint, would it not make sense that a prone restraint would likely affect the coupling between ventilation and pulmonary gas exchange. Could it be that a prone restraint position would cause a subject's hyperventilation state to be transitioned into a restraint induced hypoventilation state ***(rather than asphyxiation),*** which would then lead to acute hypercapnia, which could then lead to a dangerous lowering of pH levels, which would then lead to ventricular fibrillation, which is another keystone in most Excited Delirium deaths? The harder the physical exertion prior, during, or after restraint, the greater the risks of hypoventilation if breathing is impaired, especially if the subject is left in a prone position. Could this also explain why it is common for subjects to state that they cannot breathe after a protracted physical fight that ultimately results in a prone restraint? Could it be that even though a subject is restrained in a face down prone position yelling and screaming, thus indicating that breathing is taking place, is it possible that CO2 is not being dissipated efficiently due to hypoventilation, thus causing the chemoreceptors that regulate ventilation to register consciously that one is not breathing? The medical literature reviewed stated that dead space, or wasted ventilation, occurs when lung regions are "well ventilated" but underperfused or, conversely, when well-perfused alveoli are ventilated with gas that contains a high fraction of CO2. (Merck Manual, 2004). As noted in the Albany New York Regional Emergency Medical Organization protocol for overdose (REMO, 2004), "Most prehospital OD arrests are from ***hypoventilation and hypoxia***." This was also

echoed in the Vanderbilt University Medical Center Lifeflight protocol for general management of the toxicological emergency (Vanderbilt, 2004) where it was mentioned that, ***"hypoventilation and hypoxia often go unnoticed."***

To support the above noted hypothesis, in the paper " Metabolic Acidosis in Restraint-associated Cardiac Arrest: A Case Study" (Hick, Smith, & Lynch, 1999), researchers found that when severe metabolic acidosis combined with stimulant drug use (notably cocaine) and exertion are all combined, profound metabolic acidosis can have significant negative cardiovascular effects which may contribute to cardiovascular collapse. In this paper they looked at the five following cases studies:

Case #1:

> ***"****A 36 year old man was acting extremely agitated and belligerent on a downtown sidewalk. When approached, he attacked a police officer and ran. He was subsequently subdued by several officers. He was transported to the ED (emergency department), where he continued to fight vigorously while lying prone with his hands cuffed behind him. Breath analysis was negative for ethanol. Shortly thereafter, the patient had a witnessed respiratory arrest. He was intubated within 4 minutes of his apnea. Shortly after intubation, a 15-second episode of asystole was noted; he recovered a sinus rhythm at a rate of 140 beats/min after epinephrine, atropine, and hyperventilation.* ***His initial arterial blood gas (ABG) obtained 5 minutes after intubation was pH 6.46, pCO2 49mm Hg, p"O2 523 mm Hg, and a bicarbonate (HCO3) of 4 mEQ/L.*** *Aggressive fluid resuscitation was begun with crystalloid and 100 mEq of sodium bicarbonate was given. Within 30 minutes, the patient awoke and was able to follow commands, but had a bilateral lower-extremity paraplegia. The patient was admitted to the intensive care unit (ICU). His serum lactate level obtained one hour after admission was more than 24 mEq/L. he was extubated the following day, by which time his paraplegia had completely resolved. His urine toxicology screen was positive for cocaine and he admitted to being cocaine-toxicated during his arrest. He ruled out for a myocardial infraction by creatine kinase (CK) isoenzymes, but had a several-day course of rhabdomyolysis with peak CK levels higher than 40,000 IU/L. He was aggressively hydrated and maintained a good urine output. He developed renal insufficiency with a peak creatinine of 3.7 mg/dl. He also developed evidence of liver injury with elevated liver transaminases and increased prothrombin and partial thromboplastin times. These abnormalities resolved after several days. He was discharged on*

hospital day 5 with good urine output and a creatinine of 3.2 mg/dl. He failed to keep his follow-up appointments." (Hick, Smith, & Lynch, 1999 pg 239-40)

Case 2:

"A 39-year-old man with a history of unspecified psychiatric illness was brought to the emergency psychiatric area for evaluation of agitation and psychosis. The patient became violent and was restrained by several security guards. He was placed prone with his arms behind him. During the restraint process he became apneic and pulse less. He was moved across the hallway to the stabilization room in the ED and immediately intubated. He was initially in a nonperfusing bradycardia, which deteriorated into ventricular fibrillation, and then asystole. An external pacemaker was applied but failed to capture. Epinephrine, atropine, and bicarbonate were given without results. A thoracotomy was then performed, and after internal cardiac massage and defibrillation, he developed a perfusing rhythm. ***The ABG values obtained immediately after thoracotomy were pH 6.81, pCO2, 30 mm Hg, and po2 162 mm Hg. His initial anion gap was 37 mEq/L.*** *He was given a total of 450 mEq of bicarbonate during the case. His hemodynamic status stabilized and the acidosis reversed within 12 hours. Life support was subsequently withdrawn two days later due to a persistent vegetative state. Urine toxicology was positive for cocaine; serum levels were not available."* (Hick, Smith, & Lynch, 1999 pg 240)

Case 3:

"A 30-year-old man stole a purse, and after a long foot chase was apprehended by two witnesses who sat on the patient to restrain him. He lost consciousness, and when the paramedics arrived, he was in cardiac arrest with an idioventricular rhythm. He was intubated orally in the field, CPR was begun, and over the next 10 minutes, he was given a total of 4 mg of epinephrine, 1 mg of atropine, and 0.4 mg of naloxone. No bicarbonate was given. He was transported to the ED, where the idioventricular rhythm became asystolic. ***Initial ABG levels obtained at ED arrival showed pH less than 6.8, pCO2 18 mm hG, and pO2 255 mm Hg****. Serum bicarbonate was undetectable by standard assays. He received 150 mEq of sodium bicarbonate, escalating doses of epinephrine to 5 mg, and an additional 1 mg of atropine. An external pacer failed to capture. Transthoracic echocardiography showed a motionless heart, and resuscitative efforts were halted. Urine toxicology was positive for cocaine; serum levels were unavailable."* (Hick, Smith, & Lynch, 1999 pg 240)

Case 4:

"After firing a gun in an apartment, a 39-year-old man was apprehended and restrained by several police officers. He continued to struggle during transport in a prone position with his hands cuffed behind his back. Upon entering the ED, he violently kicked a door, and then had a sudden cardiopulmonary arrest. CPR was

started, and the patient was moved to the stabilization area. The presenting rhythm was idioventricular. Despite epinephrine, atropine, and standard advanced life support, he failed to respond, and died. ***Initial ABG levels, immediately upon ED arrival were pH less than 6, pCO2 more than 100 mm Hg, and pO2 30 mm Hg.*** *The anion gap was 24 mEq/L/Serum bicarbonate was undetectable by standard assays. A serum toxicology screen was positive for free cocaine. Autopsy revealed a nonthrombosed 75% left anterior descending coronary artery stenosis. Cause of death was attributed to "cocaine-induced excited delirium."* (Hick, Smith, & Lynch, 1999 pg 240)

Case 5:

"A 38-year-old man was observed standing in the middle of a local street, attempting to hit passing cars with his fists. Responding police personnel were not able to escort him from the street. The patient was wrestled to the ground, maced, and then carried to the median and placed on his side. He continued to struggle, and then had a sudden cardiorespiratory arrest. Paramedics were present and immediately intubated the patient. The initial rhythm was ventricular fibrillation, with defibrillation resulting in asystole. The patient received 1 mg of epinephrine, 1 mg of atropine, and 50 mEq of sodium bicarbonate with a transient return of pulses at a rate of 120 beats/min. He became pulse-less again. Five milligrams of epinephrine and an additional 50 mEq of sodium bicarbonate were given, again with return of pulses. ***The patient was transported to the ED where initial ABG levels were pH 6.25, pCO2 50 mm Hg, pO2 221 mm Hg, and bicarbonate 4 mEq/L.*** *He was admitted to the ICU, where he required aggressive fluid and pressor support. Refractory hypotension and disseminated intravascular coagulation led to an eventual bradysasystolic arrest ten hours after admission. Resuscitative efforts were futile. Autopsy showed no anatomic cause of death. Serum toxicology revealed rising levels of benzoylecgonine, a cocaine metabolite, during the time from ED presentation until the patient's death."* (Hick, Smith, & Lynch, 1999 pg 240-41)

Hick, Smith, & Lynch's paper also reported, "As our awareness of these cases (the above noted 5) has grown, we have treated at least five additional patients, who continued to struggle against restraints until Emergency Department presentation. All admitted crack cocaine use once their mental status normalized. None of these patients experienced cardiac arrest, although one was hypotensive, with systolic blood pressure of 70mm Hg at presentation. **Initial pH ranged from 6.76 to 7.16.** These patients were treated with

aggressive fluid resuscitation and those with pH<7.10 received sodium bicarbonate." All of these patients resolved their acidosis and were discharged at a later date.

To support Hick, Smith, and Lynch's observation and protocol, the following letter to the editor of the journal "Anaethesia" if offered (Allam, S., & Noble, J.S. 2001):

> ***"****We would like to report a case of cocaine-excited delirium in which the patient survived despite extreme acidosis.*
>
> *A 25-year-old male patient jumped from a first-floor window to escape his pursuers who were allegedly chasing him with swords. Whilst giving a statement to the police, he suddenly ran off and was apprehended. Although initially conversant with the paramedics, he became drowsy, and in the ambulance had a clonic seizure lasting 1 min.*
>
> *On arrival in casualty at 13:30 h, there was no eye opening, he was flexing to pain and making incomprehensible sounds. His pulse was 116 beats.min 1, blood pressure 100/40 mmHg, respiratory rate 28 breaths.min 1 and temperature 38.3 °C.* ***The initial blood gases revealed a hydrogen ion concentration of 292 nmol.l 1 (pH 6.53), P a co 2 of 13.13 kPa, base deficit of 35.6 mmol.l 1 and a P a o 2 of 25.61 kPa on 10 l of oxygen via a trauma mask.*** *Because the 12-lead ECG demonstrated tall, peaked T waves, he was assumed to be hyperkalaemic. He was intubated with a rapid sequence induction and manual in-line stabilisation of the head with 2 mg of alfentanil and 100 mg of propofol. Hyperventilation was instituted; he was given 10 ml of 10% calcium gluconate and 50 ml of 8.4% sodium bicarbonate. He had 1.5 l of 0.9% saline over 90 min. The initial plasma potassium level was 7 mmol.l 1, sodium 153 mmol.l 1, bicarbonate 12 mmol.l 1, anion gap 44 mmol.l 1, urea 8.4 mmol.l 1, creatinine 202 µmol.l 1.*
>
> *By 14:00 h, his temperature had risen to 39.7 °C. He was given a total of 2 mg.kg 1 of dantrolene in two separate aliquots within 20 min. Ice packs and a fan were employed as cooling measures. Blood gases taken at 14:47 h revealed a hydrogen ion concentration of 40.1 nmol.l 1 (pH 7.4), P a co 2 of 4.28 kPa, base deficit of 3.8 mmol.l 1 and a P a o 2 of 42.92 kPa on an F i o 2 of 0.6. Repeat electrolytes showed potassium of 5.1 mmol.l 1 and an anion gap of 30 mmol.l 1. By 15:00 h, his temperature had decreased to 37.6 °C. X-rays of pelvis, cervical spine, chest and CT scan of head were normal. Bacteriological and biochemical analysis of the CSF was unremarkable.*
>
> *He was admitted to the ICU and extubated at 20:00 h. Clotting studies were normal and the creatinine phosphokinase level peaked at 8460 µmol.l 1 the following day. He was discharged from the ICU the day after admission and from the hospital on the succeeding day with normal renal function. On further*

questioning, he admitted drinking heavily on the night preceding hospital admission, and to taking cocaine. Toxicological analysis of his urine showed no trace of opioids, benzodiazepines or amphetamines. Cocaine was omitted from the toxicological screen.

The paranoia, agitation and rapidly progressive pyrexia with which this patient presented are features of cocaine-excited delirium [1]. This condition occurs within 24 h of cocaine ingestion in habitual users. Coma and death result without intervention. ***The prompt administration of hyperventilation, passive cooling, sodium bicarbonate and dantrolene led to a remarkably swift correction of the acidosis and a successful outcome in this case. Survival after such a severe acidosis illustrates that the arterial hydrogen ion concentration gives a restricted view of what is happening at the intracellular and mitochondrial level.***

We are reporting this case to increase awareness of cocaine-excited delirium and to suggest one potential management strategy for this potentially fatal syndrome.

References

1 *Henry JA. Metabolic consequences of drug misuse. British Journal of Anaesthesia 2000; 85: 136-42.***"**(Allam, S., & Noble, J.S. 2001 pg 385)

As investigators, we cannot ignore the fact that there does appear to be an empirical correlation between restraint positions and death in Excited Delirium cases. If Dr. Reay was wrong about positional asphyxia, could the above noted paragraphs and case studies explain the nexus between restraint position and death? Only further medical research surrounding restraint positioning, hypoventilation, acute hypercapnia, hypoxia, and pH will answer these very important questions. Could it be that the hypoventilation, acute hypercapnia, hypoxia, and pH are the "tipping point", ***in combination with the other multifactoral issues mentioned in this paper,*** that may cause the cascading slide into tranquility that might result in sudden and unexpected death if not treated in some***, but not all,*** Excited Delirium cases no matter what the position of restraint (Park, Korn & Henderson, 2001)?

The writer does not suggest that there has never been a death associated with "Positional Asphyxia", where individuals who have been physically exhausted and placed into a hog-tie position, often with loads placed on their chests from several police officers holding them face down over an extended period of time, die from respiratory failure due to asphyxia. The writer is suggesting, however, that there may be a larger number of sudden and unexpected deaths proximal to restraint cases, which may be associated to ***hypoventilation and pH issues,*** rather than positional asphyxia alone, in combination with the other multi-factoral issues surrounding these deaths that have been mentioned in this paper. Although there appears to be an empirical correlation between Excited Delirium, physical restraint, and sudden and unexpected death, the exact causal mechanisms are still medically unknown and it is because of this fact, that the casual inference that physical restraint position, or certain types of holds, are an independent predictor of death during physical restraint cannot be made definitively at this time until further scientific and medical research has been conducted. (Day, 2002)

Veterinary Similarities to Excited Delirium In The Animal Kingdom:

As a result of having this paper peer reviewed by other officers, Sgt Chris Butler (Calgary Police Service Skills and Procedure Unit) contacted the writer and advised that when he was a Park Ranger, he came across a similar pattern of sudden and unexpected death in the animal kingdom after a chase and subsequent capture of deer, bear, cougar and Big

Horn Sheep. Known as "Capture Myopathy" or "Exertional Myopathy", this phenomenon has been studied by veterinarians for over thirty years (Deer Digest, 2001), and the similarities to sudden and unexpected death proximal to restraint of a human experiencing Excited Delirium are remarkable. According to the veterinary literature reviewed (Fyffe, 2004; Caulkett N., & Haigh JC, 2001; Caulkett N, 2001) deaths from Capture Myopathy exhibit the following conditions in animals:

- Hyperthermia
- Hypoventilation
- Rhabdomyolysis
- Acidosis
- Extreme lowering of pH
- If death is not immediate after capture, it occurs soon after restraint and usually within 2-3 days

Again, in the veterinary papers reviewed, if the writer did not know that he was infact reading about Capture Myopathy, he would have honestly believed he was reading a paper on Excited Delirium. Due to the fact that Capture Myopathy is identified as a medical emergency in veterinary medicine, intervention protocols included (ZCOG, 1995):

- Calming measures
- Cooling measures
- Intravenous fluids

- Vitamin E combined with Selenium
- Sodium bicarbonate to combat acidosis
- Calcium channel blockers

As in Excited Delirium cases, if not medically treated immediately, many of the pathologic changes mentioned above are irreversible and despite drastic medical intervention, animals suffering from Capture Myopathy will often die within minutes, not unlike those who are restrained suffering Excited Delirium.

Due to the uncanny similarities between Excited Delirium and Capture Myopathy, should the medical experts who are researching Excited Delirium be consulting with their brothers and sisters in veterinary medicine? Could the thirty years of studying Capture Myopathy by veterinarians, including their early intervention protocols, be of some benefit and assistance to those who are looking at developing protocols for Excited Delirium cases in humans?

It is interesting to note that the veterinarian intervention protocol is very similar to those mentioned in the Hick, Smith, & Lynch paper, the Allam & Noble letter to the Journal Anaethesia, as well as a proposed medical intervention located in a paper by Park, Korn, & Henderson (2000), specific to Excited Delirium in human subjects. Because of these similarities, could a possible medical intervention protocol for humans be:

1) Chemically restraint the patient (this is done by ALS paramedics and not police) to slow down the SNS (Fight/Flight) via the use of a drug from the benzodiazepine family such as "Midazolam".

2) Immediately get an IV into the client due to the fact that many who are experiencing ED are severely de-hydrated.

3) Immediately get a pH reading utilizing a new device called "istat", which allows paramedics in the field to get a pH reading (as well as other very important blood gases) within minutes.

4) If the pH is shown to be below 7.10 immediately push a sodium bicarb to bring the pH back to homeostasis

5) Immediately begin cooling procedures to deal with the hyperthermia via cooling packs/ blankets and if able, use a drug such as dantrolene which is used in some hospitals to treat malignant hypothermia

Only further medical research, study, and data collection will allow for such a medical intervention protocol "hypothesis" to be tested and applied in the field. A national or even international multi-site prospective study involving a multi-disciplinary team of medical experts, needs to be funded to make recommendations and informed changes (if any) to medical intervention protocols, based upon the data collected in the prospective study, so as to prevent some (not all unfortunately) sudden and unexpected deaths proximal to restraint, and for life retrieval protocol should a subject die during or after a physical altercation/ restraint that is associated with Excited Delirium.

CONCLUSION OF RESEARCH:

Excited Delirium is described as:

" A state of extreme mental and physiological excitement, characterized by extreme agitation, hyperthermia, epiphoria, hostility, exceptional strength and endurance without apparent fatigue" (Morrison & Sadler, 2001).

Throughout the medical literature reviewed, the one thing that all of the medical community can agree upon is that Excited Delirium is a "medical emergency" no matter what the cause (Barkley Burnett & Adler, 2004; Farnham & Kennedy, 1997; Lawrence & Cairns, 2001; Young, 1995).

Good practice guidance, specific to the use of force, suggests that restraint should be subject to risk assessment such that the risks involved are considered against the risks of alternatives (Paterson et al., 2003). There are going to be times in policing, as well as in health care, be it psychiatric or geriatric, where non-physical approaches to control will not be practicable or reasonable given the emergence of the situation, and physical restraint may represent the only intervention capable of protecting the subject or others from death or serious bodily injury. Physical restraint or control of those suffering Excited Delirium is intrinsically an option that always has with it risks, however, in some circumstances, these risks may be less dangerous than the alternatives available. As one

medical professional stated at a workshop on Excited Delirium in Calgary, Alberta Canada, ***"There can be no medical treatment of these individuals without restraint".***

It may be that deaths, associated to Excited Delirium, can never be completely eradicated. Hopefully by understanding the processes that may be involved and communicating the information to police officers, paramedics and emergency medical teams we may be able to optimize the emergency systems responses to incidents involving people with Excited Delirium. In fact, this is the theme of a research proposal Chris Lawrence, Team Leader of the Defensive Tactics Training Section at the Ontario Police College, will be forwarding through Royal Roads University as his major project for the completion of a graduate degree. Chris has stated that our investigation and recommendations specific to this issue will greatly assist him in this project. While work has already started on this project the expected completion date is August 2005. In the meantime Chris has developed an "Investigator Protocol: Sudden In-Custody Death" which all departments and health care agencies should follow when investigating such a death. As well, there are some specific protocols that should be followed by anyone who has taken into custody, through physical restraint, a person reasonably believed to be suffering from Excited Delirium:

- Due to the fact that Excited Delirium is a medical emergency, all subjects should be transported to hospital via ambulance (Advanced Life Support Paramedics) and monitored closely including heart rate, blood pressure, respirations, CO2 levels, pH levels, and temperature.

- Given the empirical correlation between the maximal restraint position (hog-tie) and sudden and unexpected death, such a restraint should be strongly reconsidered for use by all law enforcement agencies until further medical research has been conducted.

- Once in custody and awaiting transport via ambulance, or while being transported in the ambulance, the restrained subject should be placed in a supine position. If the subject must be maintained on their side it is recommended that they be placed resting on their left side if possible.

- If reasonable and able to do so, have Advanced Life Support Paramedics on standby prior to a physical interdiction of a person who appears to be experiencing Excited Delirium.

- From a control/restraint/safety perspective, the quicker control can be established the better. It may be that the longer the physical confrontation goes on with a subject experiencing Excited Delirium, the higher the risk of an in custody death.

- If the restrained subject suddenly becomes quiet and stops resistance, pulse rate, blood pressure, and temperature should be recorded continually. Advanced life support should be summoned where available, and preparation for CPR should be made.

Although protocols for police, specific to dealing with subjects experiencing Excited Delirium are important, others who also interact with these types of persons; including paramedic staff, emergency room staff, and even pathologists should be looking at developing protocols, specific to Excited Delirium patients, as well. Research, introduction, and the application of such protocols for most professions are lacking greatly, based upon the literature review and personal interviews that were conducted for this paper. Dr. Christine Hall, Calgary Health Region, who has conducted research into the issues surrounding Excited Delirium, and according to the medical literature that she reviewed specific to this topic area, found that there has never been one successful save once a person experiencing Excited Delirium has fallen to cardiac arrest prior to arrival at hospital. Could one of the goals of the medical research into Excited Delirium be to develop a standard protocol for after care with a relevant and needed data collection process, concurrent with developing an after care protocol for human life retrieval? This is a goal that I hope the medical community will take a serious look at in the very near future here in Victoria, and one that we are facilitating utilizing a medically multi-disciplinary approach. If we can start the ball rolling, a multi-center medical study would be enormously helpful in answering some of the questions raised in this paper.

When someone dies suddenly and unexpectedly during or following an altercation with police officers without a cogent explanation, the subject's family, the general public, and the media will be left to search for their own answers. The traditional approach of not

releasing any information pending the outcome of a thorough investigation can exacerbate speculation, which can generate mistrust. A major step could be realized if such a cogent explanation, based in lay terminology, could be derived and delivered to the public. Such an explanation would not fit into a sound bite on the evening news but rather provide the basis for an investigative report, the type usually seen during prime time viewing hours. Without such an explanation the concept of sudden and unexpected death due to Excited Delirium runs the risk of becoming a mere media buzzword. It should also be stated that despite the highly controversial and newsworthy profile of these types of sudden and unexpected deaths proximal to restraint, they remain a rare event compared with the overall prevalence of restraint use in law enforcement and health care. (Day, 2002)

Due to the type of situation that surrounds a sudden and unexpected death associated to what seems to be Excited Delirium, rigorous study is virtually impossible, due in large part to ethical issues that must be considered. An alternative is to formulate hypotheses, based on the collective, have objective assessment of a diverse group of experts, and then test the hypotheses to the extent ethically and practically possible (something that we will be doing with our medical review committee in Victoria). The progress of such an effort needs to be communicated to the groups involved in such an event as well as the general public.

I hope this review of the medical literature, specific to Excited Delirium as it relates to sudden and unexpected death proximal to restraint, was of some assistance. The research into this very important and sometimes emotionally charged topic is truly multi-disciplinary and as such, this paper should be considered a "living" document. There is no doubt that as further scientific and medical research on Excited Delirium, and its' relationship to sudden and unexpected death proximal to restraint becomes available, the information and hypothesis found in this paper will need to be updated as well.

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Special Note:

Special thanks go to Chris Lawrence who has dedicated the past five years of his professional career as a police educator to the issues surrounding Excited Delirium. Chris pointed me in all the right directions, thus allowing me to quickly come up to

speed on the medical research/literature mentioned in this paper. Thanks Chris, your passion and commitment to this issue is second to none, and will go along way in providing a possible protocol for "Human Life Retrieval" for those on the path to sudden and unexpected death proximal to restraint.

References

Allam, S., & Noble, J. S. (2001). Cocaine excited delirium and severe acidosis. *Anaesthesia, 56*(4), 385.

Ann Price et al., Plaintiffs, v. County of San Diego et al., Defendants. (1998). Retrieved August 15, 2004, from http://www.aele.org/Hot5.html

Barkley Burnett, L., & Adler, J. (2004). *Toxicity, Cocaine*. Retrieved August 16, 2004, from http://www.emedicine.com/emerg/topic102.htm

Bauman, J.L. & DiDomenico, R. J. (2002). Cocaine-induced channelopathies:emerging evidence on the multiple mechanisms of sudden death. *Journal of Cardiovascular Pharmacology and Therapeutics. 7(3),* 195-202.

Bell, L. (1849). On a form of disease resembling some advanced stages of mania and fever, but so contradistinguished from any ordinary observed or described combination of symptoms as to render it probable that it may be overlooked and hitherto unrecorded malady. *American Journal of Insanity, 1849*(6), 97 - 127.

Benzer, Theodore (2002). Neuroleptic Malignant Syndrome. Retrieved November 19, 2004, from http://www.emedicine.com/emerg/topic339.htm

Bisgard, G. E., Forster, H. V., Klein, J. P., Manohar, M., & Bullard, V. A. (1980). Depression of ventilation by dopamine in goats--effects of carotid body excision. *Respiration Physiology, 40*(3), 379-392.

Caulkett, N. & Haigh, J. C. (2001). Field anesthesia of deer and bison. *Large Animal Veterinary Rounds, 1(6)*.

Chan, T. C., Vilke, G. M., & Neuman, T. (1998). Reexamination of custody restraint position and positional asphyxia. *American Journal of Forensic Medicine and Pathology, 19*(3), 201 - 205.

Chan, T. C., Vilke, G. M., Neuman, T., & Clausen, J. L. (1997). Restraint position and positional asphyxia. *Annals of Emergency Medicine, 30*(5), 578 - 586.

Chan, T. C., Neuman, T., Clausen, J., Eisele, J., &Vilke, G. M. (2004). Weight force during prone restraint and respiratory function. American Journal Forensic Medicine and Pathology;25:185-189.

Conner, M. G. (2002). *In-custody death: excited delirium, restraint asphyxia, positional asphyxia and 'in-custody death" syndromes: controversial theories that may explain why some children in treatment programs die when restrained.* Retrieved February 3, 2003, from http://www.strugglingteens.com/mconnorart/incustodydeath.html

Davis, A. M., & Natelson, B. H. (1993). Brain-heart interactions: the neurocardiology of arrhythmia and sudden cardiac death. *Texas Heart Institute Journal, 20*(3), 158-169.

Day, P. (2002). *What evidence exists about the safety of physical restraint when used by law enforcement and medical staff to control individuals with acute behavioural disturbance?* (No. 1-877235-41-5). Christchurch: NZ: New Zealand Health Technology Assessment.

Deerfarmer.com (2003). Why deer die. Retrieved September 9, 2004 from http://www.deer-library.com/artman/publish/article_98.shtml.

Dimsdale, J. E., Hartley, L. H., Guiney, T., Ruskin, J. N., & Greenblatt, D. (1984). Postexercise peril: Plasma catecholamines and exercise. *Journal of the American Medical Association. 251(5)*. 630-632.

Farnham, F. R., & Kennedy, H. G. (1997). Acute excited states and sudden death: much journalism, little evidence. *British Medical Journal, 315*(7116), 1107-1108.

Fyffe, J. (n.d.). Capture myopathy. Retrieved September 9, 2004 from: http://www.dia.org/captmyop.htm.

Glassman AH, Bigger JT(2001). Antipsychotic drugs: prolonged QTc interval, toursade de pointes, and sudden death. American Journal of Psychiatry, 2001 Nov;158(11):1774-82

Hick, J. L., Smith, S. W., & Lynch, M. T. (1999). Metabolic acidosis in restraint-associated cardiac arrest: a case series. *Academy of Emergency Medicine, 6*(3), 239 - 243.

Haouzi, P., Chenuel, B., & Chalon, B. (2002). Effects of body position on the ventilatory response following an impulse exercise in humans. *Journal of Applied Physiology, 92*(4), 1423-1433.

Joint Commission for Accreditation of Healthcare Organizations. (1998). *Sentinel event alert: preventing restraint deaths,*. Retrieved January 8, 2004, from http://www.jcaho.org/edu_pub/sealer/sea8.html

Kolecki, P. F. & Curry, S. C. (1997). Poisoning by sodium channel blocking agents. *Critical Care Clinics. 3(4),* 829-848

Laitinen, P. J., Swan, H., Piippo, K., Viitasalo, M., Toivonen, L., & Kontula, K. (2004). Genes, exercise and sudden death: molecular basis of familial catecholaminergic polymorphic ventricular tachycardia. *Annals of Medicine, 36(Suppl 1),* 81-86.

Lawrence, C. W., & Cairns, J. T. (2001). Sudden custody death: the Ontario perspective. *RCMP Gazette, 63*(5), 33 - 35.

Lawrence, C. W., & Mohr, W. K. (2004, January). Investigator protocol: sudden in custody death. *The Police Chief, 71,* 44-52.

Lawrence, C. W. (2004). Prone position linked to sudden death in suspects. *Blue Line Magazine, June,* 24-25.

Lehnart, S. E., Wehrens, X. H., Laitinen, P. J., Reiken, S. R., Deng, S. X., Cheng, Z., et al. (2004). Sudden death in familial polymorphic ventricular tachycardia associated with calcium release channel (ryanodine receptor) leak. *Circulation, 109*(25), 3208-3214.

Maron,B.J.,HYPERTROPHIC CARDIOMYOPATHY,HURST'S THE HEART,10th Ed.,PP.1967-1983.

MedicineNet.com (2004) *Hypoventilation*. Retrieved September 6, 2004 from http://www.medterms.com/script/main/art.asp?

Merck Manual of Diagnosis and Therapy (2004). Respiratory failure. Retrieved September 12, 2004 from: http://www.merck.com/mrkshared/CVMHighLight?file=/mrkshared/mmanual/section6/chapter66/66a.jsp%3Fregion%3Dmerckcom&word=hypoventilation&domain=www.merck.com#hl_anchor articlekey=32266

Mets, B., Jamdar, S., & Landry, D. (1996). The role of catecholamines in cocaine toxicity: a model for cocaine "sudden death". *Life Sciences, 59*(24), 2021-2031.

Morrison, A., & Sadler, D. (2001). Death of a psychiatric patient during physical restraint. Excited delirium--a case report. *Medicine Science and Law, 41*(1), 46 - 50.

Office of the Ombudsman for Mental Health and Mental Retardation.*Current Issues in Seclusion and Restraint*. Retrieved September 6, 2004, from http://www.ombudmhmr.state.mn.us/alerts/currentissuesonseclusionandrestraint.html.

O'Halloran, R. L., & Frank, J. G. (2000). Asphyxia death during prone restraint revisited: a report of 21 cases. *American Journal of Forensic Medicine and Pathology, 21*(1), 39 - 52.

Ortega-Carnicer, J. n., Bertos-Polo, J., & Gutierrez-Tirado, C. (2001). Aborted sudden death, transient Brugada pattern, and wide QRS dysrrhythmias after massive cocaine ingestion. *Journal of Electrocardiology, 34*(4), 345-3249.

Park, K. S., Korn, C. S., & Henderson, S. O. (2001). Agitated delirium and sudden death: two case reports. *Prehospital Emergency Care, 5*(2), 214 - 216.

Paterson, B., Bradley, P., Stark, C., Saddler, D., Leadbetter, D., & Allen, D. (2003). Restraint related deaths in health and social care in the UK: learning the lessons. *Mental Health Practice Journal. 6(9)*, 10-17.

Pollanen, M. S., A., C. D., Cairns, J. T., & Young, J. G. (1998). Unexpected death related to restraint for excited delirium: a retrospective study of deaths in police custody and in the community. *Canadian Medical Association Journal, 158*(12), 1603-1607.

Priori, S. G., & Napolitano, C. (2004). Genetics of Cardiac Arrhythmias and Sudden Cardiac Death. *Annals of the New York Academy of Sciences, 1015*, 96-110.

Rashad Net University. (n.d.) Acute hypercapnia. Retrieved September 6, 2004 from http://www.rashaduniversity.com/mrashad/achy.html

Reay, D. T., Flinger, C. L., Stillwell, A. D., & Arnold, J. (1992). Positional asphyxia during law enforcement transport. *American Journal of Forensic Medicine and Pathology, 13*(2), 90 - 97.

Regional Emergency Medical Orgaization. (n.d.) Medical emergiences: M-6 overdose protocol. Retrieved September 8, 2004 from http://www.remo-ems.com/newoverdoseprotocol.doc.

Rosh, A., Sampson, B. A., & Hirsch, C. S. (2003). Schizophrenia as a cause of death. *Journal of Forensic Sciences, 48*(1), 164-167.

Ross, D. L. (1998). Factors associated with excited delirium deaths in police custody. *Modern Pathology, 11*(11), 1127 - 1137.

Ruttenber, A. J., Lawler-Heavner, J., Yin, M., Wetli, C. V., Hearn, W. L., & Mash, D. C. (1997). Fatal excited delirium following cocaine use: epidemiologic findings provide new evidence for mechanisms of cocaine toxicity. *Journal of Forensic Sciences, 42*(1), 25-31.

Ruttenber, A. J., McAnally, H. B., & Wetli, C. V. (1999). Cocaine-associated rhabdomyolysis and excited delirium: different stages of the same syndrome? *American Journal of Forensic Medicine and Pathology, 20*(2), 120 - 127.

Schecter, B (Nov 2004). Lecture: Balancing the Brain: Mental Health, Medications and Substance Abuse. Victoria British Columbia Canada

Schmidt, P., & Snowden, T. (1999). The effects of positional restraint on heart rate and oxygen saturation. *Journal of Emergency Medicine, 17*(5), 777-782.

Stephens, B. G., Jentzen, J. M., Karch, S. B., Wetli, C. V., & Mash, D. C. (2004). National Association of Medical Examiners position paper on the certification of cocaine-related deaths. *American Journal of Forensic Medicine and Pathology, 25*(1), 11-13.

Stratton, S. J. (1995). Agitated delirium versus positional asphyxia. *Annals of Emergency Medicine, 26*(6), 761.

Stratton, S. J., Rogers, C., Brickett, K., & Gruzinski, G. (2001). Factors associated with sudden death of individuals requiring restraint for excited delirium. *American Journal of Emergency Medicine, 19*(3), 187 - 191.

Straus, S. M. J. M., Bleumink, G. S., Dieleman, J. P., van der Lei, J., 't Jong, G. W., Kingma, J. H., et al. (2004). Antipsychotics and the risk of sudden cardiac death. *Archives of Internal Medicine, 164*(12), 1293-1297.

Vanderbilt University Medical Center Lifeflight (2004). General management of the toxicological emergency. Retrieved September 8, 2004, from: http://www.mc.vanderbilt.edu/lifeflight/web/protocol/9a.htm

Vos, M. A., & Paulussen, A. D. C. (2004). Genetic basis of drug-induced arrhythmias. *Annals of Medicine, 36*(Supplement 1), 35-40.

Weiss, E. M. (1998, 11-15 October). Deadly restraint: a nationwide pattern of death. *Hartford Courant*.

Wilson, L. D., & Shelat, C. (2003). Electrophysiologic and hemodynamic effects of sodium bicarbonate in a canine model of severe cocaine intoxication. *Journal of Toxicology: Clinical Toxicology, 41*(6), 777-788.

Young, J. G. (1995). Memorandum #636: Excited delirium and the use of restraint. Toronto: ON: Ministry of the Solicitor General & Correctional Services, Office of the Chief Coroner.

Zapata, P., & Zuazo, A. (1980). Respiratory effects of dopamine-induced inhibition of chemosensory inflow. *Respiration Physiology, 40*(1), 79-92.

Zoo Conservation Outreach Group (1995). Capture Myopathy. Retrieved September 9, 2004 from http://www.zcog.org/zcog%20frames/Capture%20Myopathy/Capture%20Myopathy.htm