

EXCITED DELIRIUM SYNDROME (EXDS), SUDDEN POLICE IN-CUSTODY DEATHS AND THE IMPLICATIONS OF VNR

2018 Version 3-8-2018

Glen Carpenter Use of Force Consulting

INTRODUCTION

- × 25 years in law enforcement.
- × 18-Years in Patrol – Graveyard.
- × Lead Defensive Tactics Instructor
- × Firearms Instructor
- × WSCJTC DT Master Instructor
- × Certified Force Science Analyst (2016)
- × Certified Institute for the Prevention of In-Custody Deaths Exctied Delirium & Agitated Chaotic Events™ Instructor (2017)
- × UOF Subject Matter Expert
- × WSCJTC Patrol Tactics Instructor
- × Graveyard Patrol Sergeant
- × SWAT Team Member / Trainer
- × Field Training Officer Advisor
- × Seattle Police Department Integrated Combat Control Instructor (2017)
- × WSCJTC BLEA TAC Officer and Patrol Procedures Core-Block Instructor
- × Special Investigations Unit Drug Investigator
- × Drug Enforcement Administration (DEA) Task Force Officer

SCOPE OF TRAINING

- × Implications of VNR
- × History of ExDS
- × Definitions
- × Symptoms
- × Causation
- × Response
- × Documentation

REFERENCES

EXCITED DELIRIUM SYNDROME

Cause of Death and Prevention

2007

Theresa G. DiMaio
Vincent J.M. DiMaio

Taylor & Francis

Sudden Deaths in Custody

Edited by
Darrell L. Ross, PhD
Theodore C. Chan, MD

2006

HUMAN PRESS

Handbook of FORENSIC PATHOLOGY

Vincent J.M. DiMaio and Suzanna E. Dana

2007

Taylor & Francis

American College of
Emergency Physicians®
ADVANCING EMERGENCY CARE

White Paper Report on Excited Delirium Syndrome

ACEP Excited Delirium Task Force

September 10, 2009

2009

Revised to the General and Panel of Directors and Joint Delirium at the Direction of American Society (2008)

IPICD

INSTITUTE FOR THE PREVENTION
OF IN-CUSTODY DEATHS, INC

TR - 02 - 2005

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

Date: 2004 - 09

By Sgt Darren Loe

Victoria Police Department

2009



Special Panel Review of Excited Delirium

October 2011

2011

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PENNSTATE

CURRENT PRACTICE IN Forensic Medicine

EDITORS
JOHN GALL
JASON FRYNE-JAMES

2016

WILEY-BLACKWELL

GUIDELINES FOR INVESTIGATING OFFICER-INVOLVED SHOOTINGS, ARREST-RELATED DEATHS, AND DEATHS IN CUSTODY

Darrell L. Ross • Gary M. Vilke

2017

VASCULAR NECK RESTRAINT

Vascular Restraint Demonstration

NECK HOLDS

Bar Arm Choke Holds / Strangles

Neck Holds i.e. “Choke” Holds, Carotid Sleeper Hold or Shime Waza.

Vascular Neck Restraint (VNR)

BAR ARM CHOKE HOLDS

Mechanism – Yoking Strangulation

Airway Compression

Deadly Force Technique

Used by the Military (Sentry Removal)

Can be used in Law Enforcement (Deadly Force)

NECK HOLDS HISTORY

Safely used in Sport Judo since it was founded in 1882 by Professor Jigoro Kano, derived from the martial art of Juijitsu.

No deaths attributed to “Choke” Holds or Shime Waza* used in Judo.

“Choke” is a fallacious term, as technique does not restrict breathing or affect the airway.

In 1981, class action suit bought against City of Los Angeles for fatalities from using “Choke Holds” and “Bar-Arm Holds” (see 16 deaths 1975-1982 and City of Los Angeles vs. Lyons 1981).

1987 - Journal of Forensic Sciences produced study by Dr. E. Karl Koiwai MD “Deaths Allegedly Caused by “Choke Holds” (Shime Waza), which examined the deaths. Training recommendations to continue it's use.

*Koiwai E.K. “How safe is Choking (Shime Waza) in Judo - 1968

LATERAL VASCULAR NECK RESTRAINT®

- Developed by Jim Lindell KCPD in 1970.
- LVNR System based on the study done by Dr. Eichi Koiwai – 1968
- Term “LVNR” adopted in 1984.
- No deaths or injuries in over 175,000 documented applications by Kansas City Police Officers alone.
- No serious injuries or deaths in over 137 years.
- Considerably less dangerous than “knockout” in boxing.

VASCULAR NECK RESTRAINTS

Mechanism: Bilateral Compression to
sides of neck

- Compression of Carotid Arteries

- Compression of Jugular Veins

- Compression of Carotid Sinus

- Stimulation of Vagus Nerve

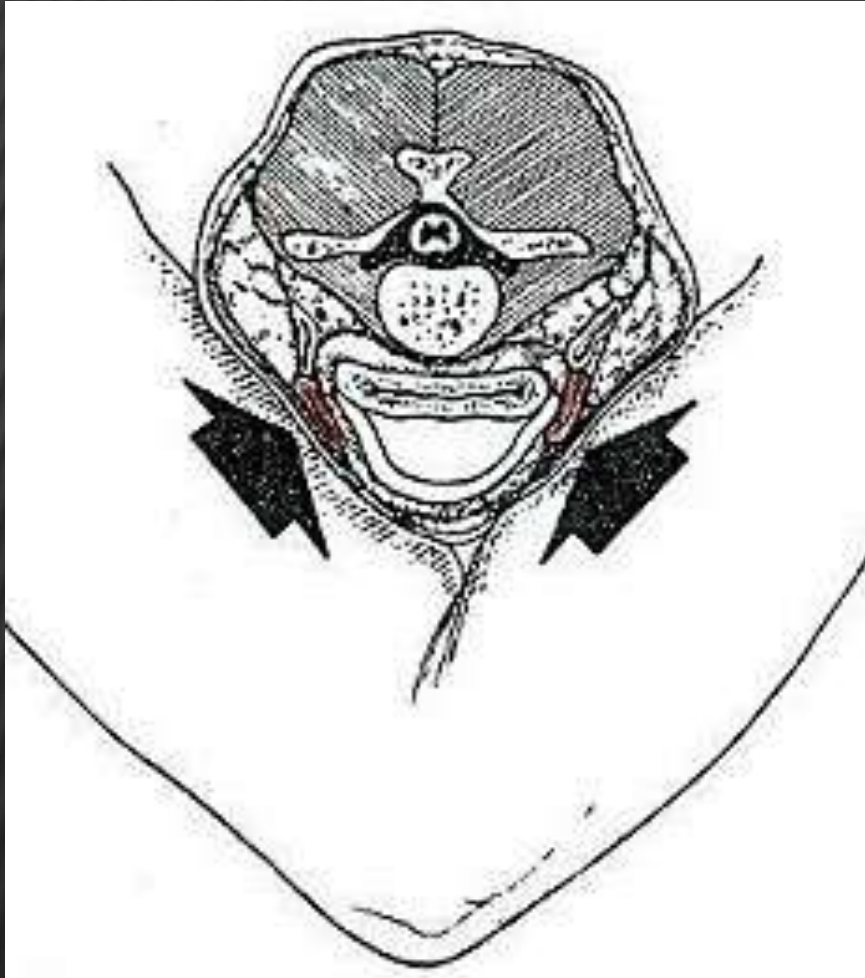
- The Valsalva Maneuver

No Airway Compression

No Forcible Neck Manipulation

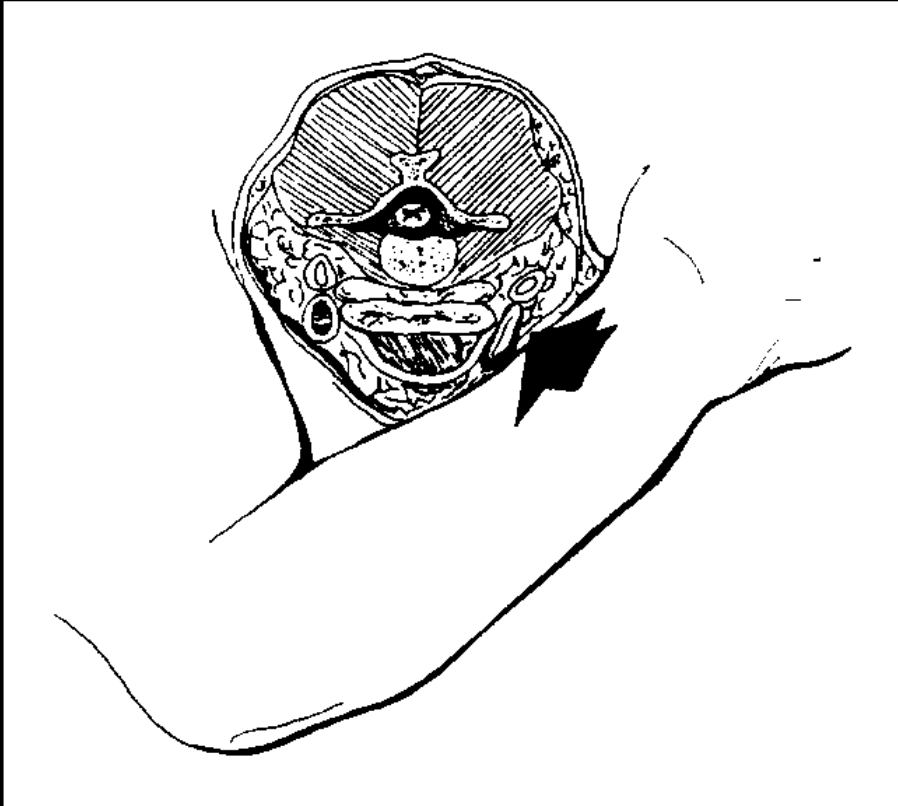
Not deadly force

BI-LATERAL VASCULAR NECK HOLDS



- ✖ Pressure is applied Bilaterally to the sides of the neck structure.
- ✖ Studies show it's almost impossible to apply “true” bilateral pressure.
- ✖ Uses “C” collar principal or Neck Brace Principal to avoid injury to neck.

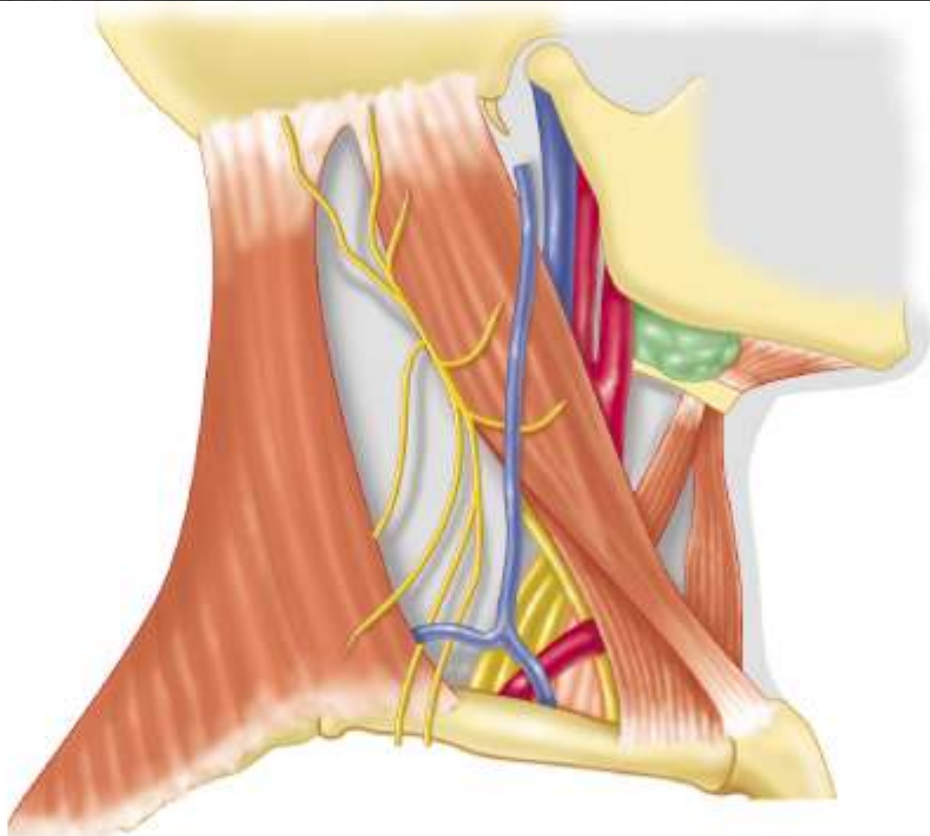
UNILATERAL VASCULAR NECK HOLDS



Unilateral neck holds will also cause unconsciousness, but manipulation to the neck can cause injurious or deleterious affects, which may be considered application of deadly force.

Unilateral holds may cause forcible neck rotation, which is suboptimal.

VNR – CAROTID TRIANGLE



(b)

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VNR produces bilateral Pressure to the Carotid Triangle.

Only 7lbs of pressure is required to occlude the arteries/veins in the Carotid Triangle.

Muscle Structures are minimally affected by this pressure.

VNR - APPLICATIONS

- Goal of a VNR is to gain compliance, not to necessarily render a person unconscious.
- There are three levels of control based on the suspects resistance level and allows the officer to de-escalate at any time.
- Uses “C” Collar or Neck brace Principal to avoid injury to both officers and offenders by restraining the neck minimizing forced rotary movement.

VNR - APPLICATIONS

- When resistance ceases or the desired level of unconsciousness is reached, technique is released to **“LEVEL I MINUS”, or, “ZERO COMPRESSION”**
- When maximum compression is applied, unconsciousness occurs within 4-7 seconds
- **SUBJECTS SHOULD REGAIN CONSCIOUSNESS IN 5-20 SECONDS.**
- Medical aid is immediately summoned.
- 2-hour observation period recommended by NLETC*

*Absolutely no medical data contained in the NLETC LVNR Manual

NECK HOLDS

Compression of the neck structures:

Jugular Veins	4.4 lbs (206 mmHg)
Carotid Arteries	7 lbs (362 mmHg)
Trachea	33 lbs (1706 mmHg)

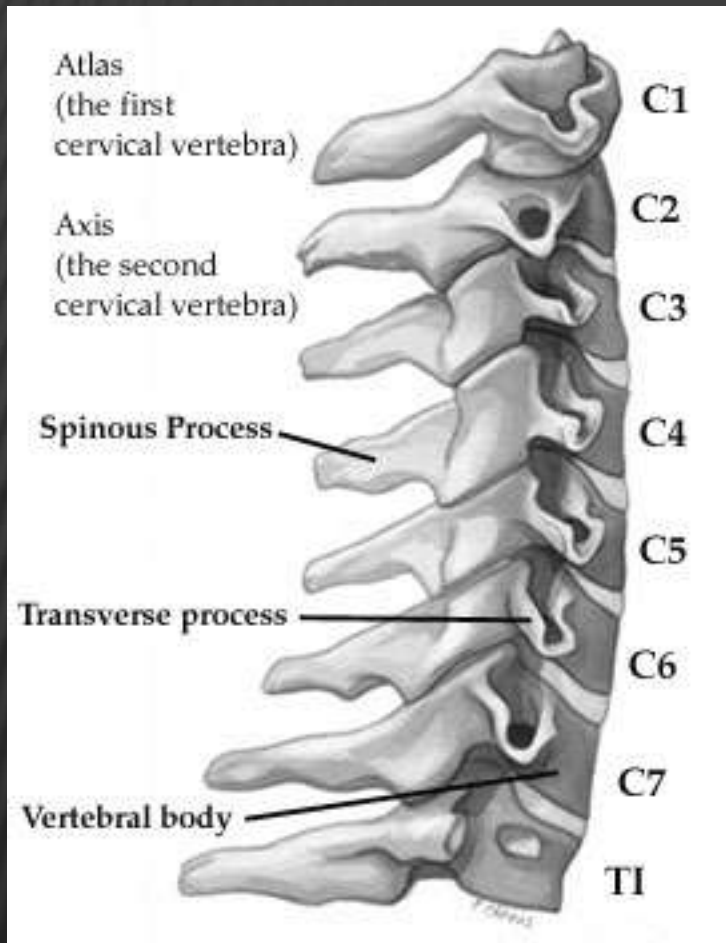
COMPRESSION OF THE NECK STRUCTURES

Compression of the carotid arteries or jugular veins—causes cerebral ischemia (not hypoxia).

Compression of the laryngopharynx, larynx, or trachea—causes asphyxia.

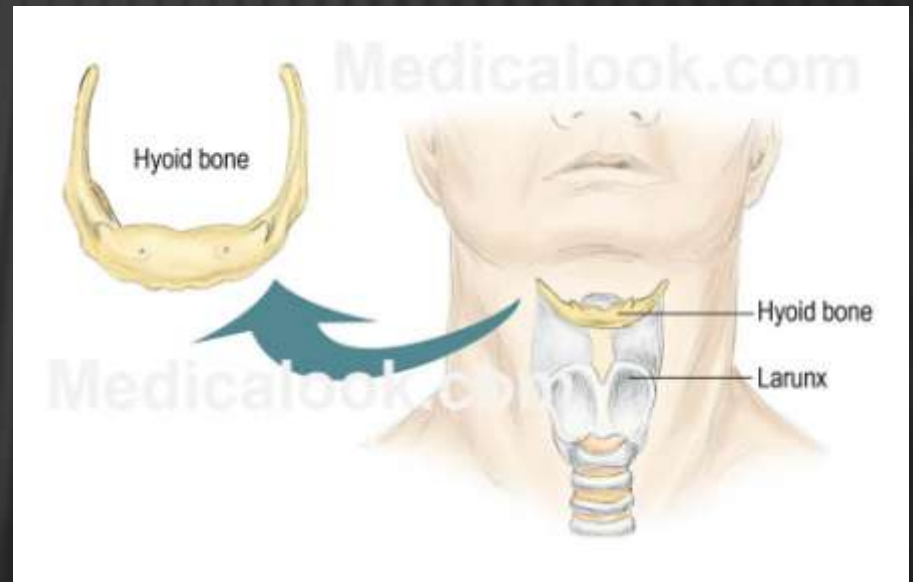
Stimulation of the carotid sinus reflex—causes bradycardia, hypotension, or both.

NECK ANATOMY - BONES



Seven Cervical Vertebrae

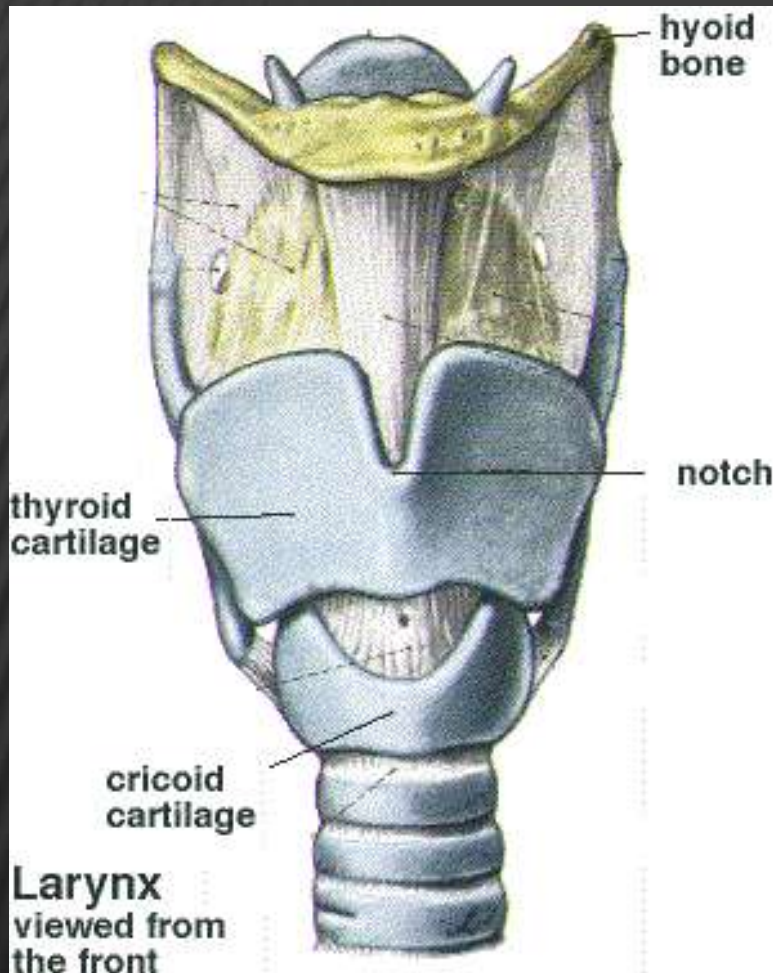
Hyoid Bone



VNR – BONY STRUCTURES

- A properly applied VNR does not adversely affect the hyoid bone as there is no compression of the subject's airway.
- The VNR Neck Brace Principal prevents any adverse affects to the Cervical Vertebrae by preventing forced rotary movement.
- Any acute neck soreness or abnormal mobility to this area after application of VNR warrants a trip to the ER.

NECK ANATOMY - CARTILAGE



Larynx

Thyroid Cartilage

Cricoid Cartilage

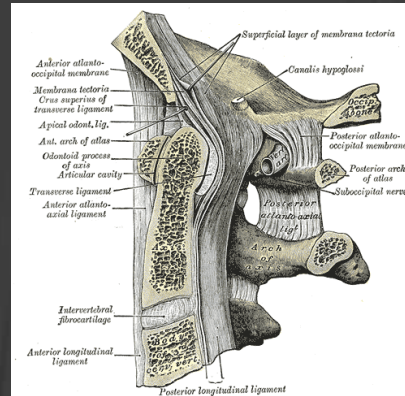
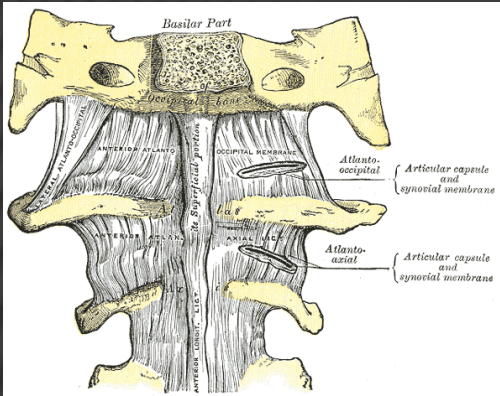
Trachea

VNR - CARTILAGE

- A properly applied vascular neck restraint will result in only minimal pressure on these structures; damage or injury is extremely rare.
- Damage to these areas takes approximately 33lbs of pressure, and it only takes 7lbs to occlude the arteries and veins in the neck to be effective.
- Apply the technique without pressure to this area and recognize improper application, ensuring proper alignment of the elbow and chin.
- Complaint of acute pain in this area or obvious difficulty breathing by any offender warrants a trip to the ER.
- Compression of the Larynx, or a strike to the Larynx, may cause swelling that causes suffocation or asphyxia. However, because there is little vascularity in the Larynx, the swelling may not occur for 3-4 hours after the application.

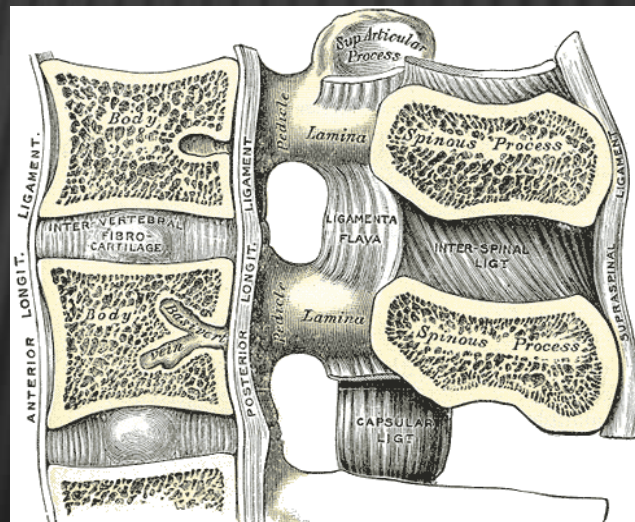
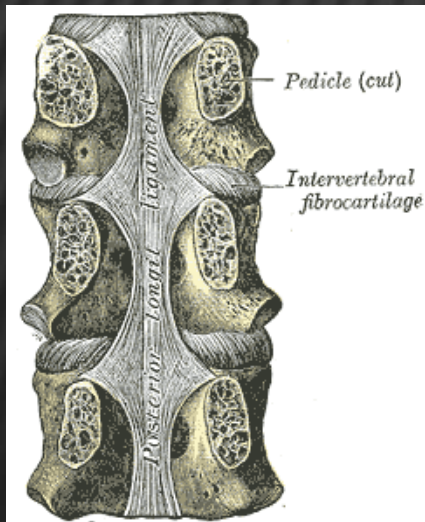
LIGAMENTS

Anterior Longitudinal Ligaments



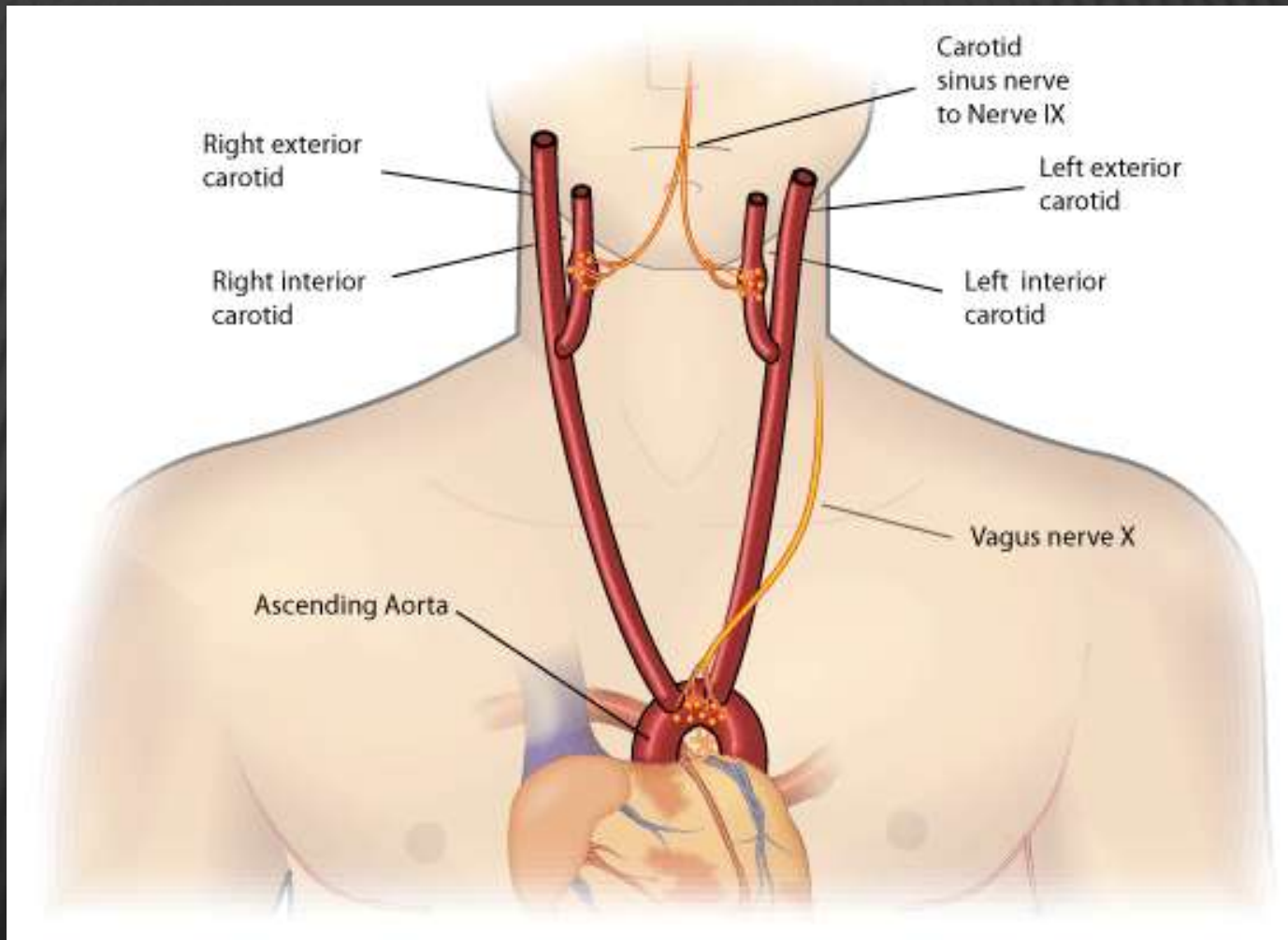
Any forced rotary movement during the struggle may cause a strain, sprain or possible rupture to the Ligaments in the neck.

Posterior Longitudinal Ligaments



Any acute stiffness, immobility or stasis in the neck after application may warrant a trip to the E.R.

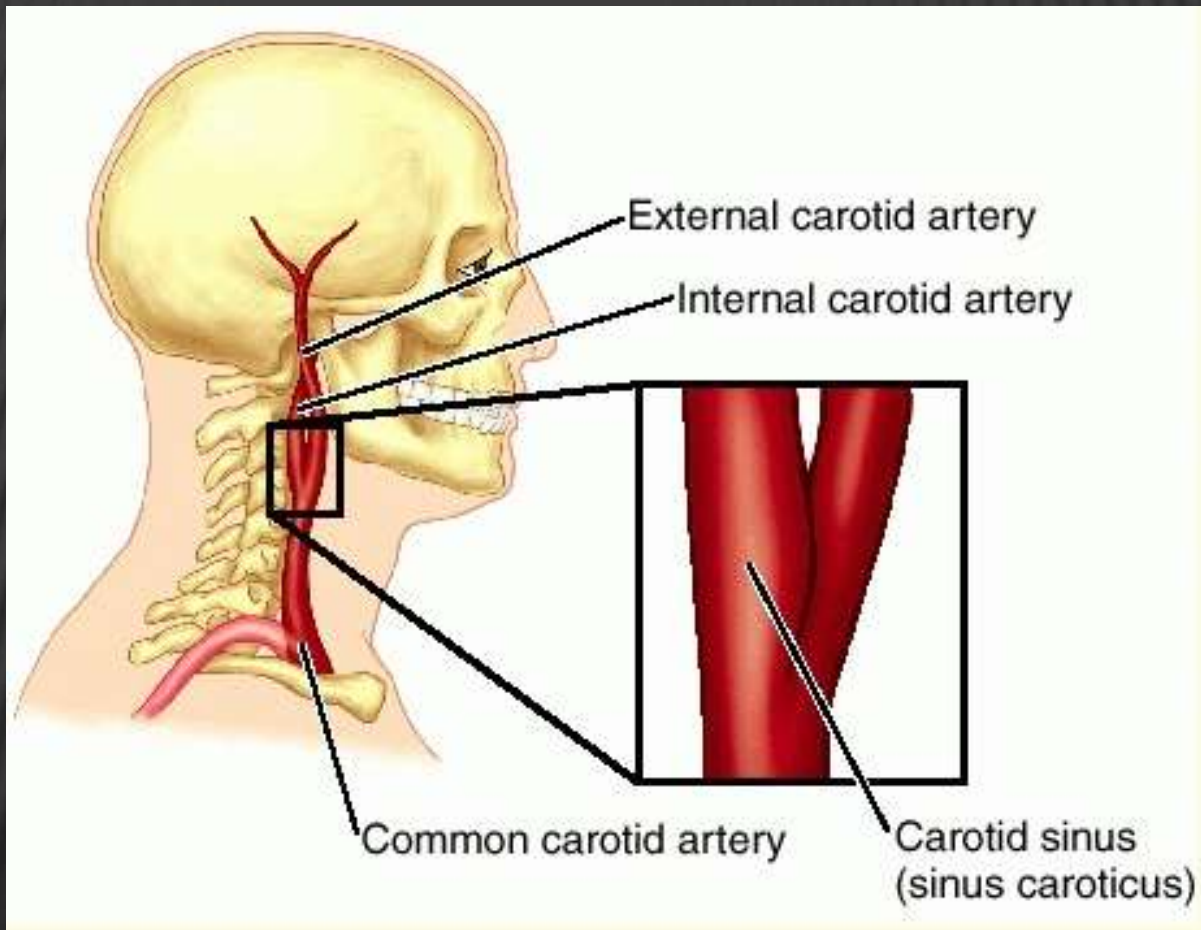
NECK ANATOMY – NERVES



VNR NERVOUS SYSTEMS

- VNR results in compression of the Carotid Arteries, Jugular Veins and compression of the Carotid Bulb, which stimulates the Vagus nerve (Carotid Sinus Massage).
- Artificial stimulation of the Vagus Nerve by external compression of the Carotid Bulb can result in a lowering of the heart rate.
- There is no evidence that over stimulation of the Vagus Nerve can create any significant injury or death; it is not a risk factor associated with VNR (VNR/CPRC 2007).

NECK ANATOMY – CAROTID ARTERIES



VNR – CAROTID ARTERIES

- Proper application of a VNR results in the occlusion of the Carotid Arteries, which results in the acute arrest of cerebral circulation.
- There are absolutely no deleterious affects from arrest of cerebral circulation to the brain for up to 100 seconds (see Rossen-Kabat-Anderson study – 1943)
- Officers are trained to release the technique to level 1 minus after a subject is rendered unconsciousness.
- Officers are trained that subjects will regain consciousness within 5-20 seconds.
- If subject does not regain consciousness within 30 seconds, they will immediately be transported to the ER for evaluation.

ROSSEN-KABAT ANDERSON (KRA) APPARATUS

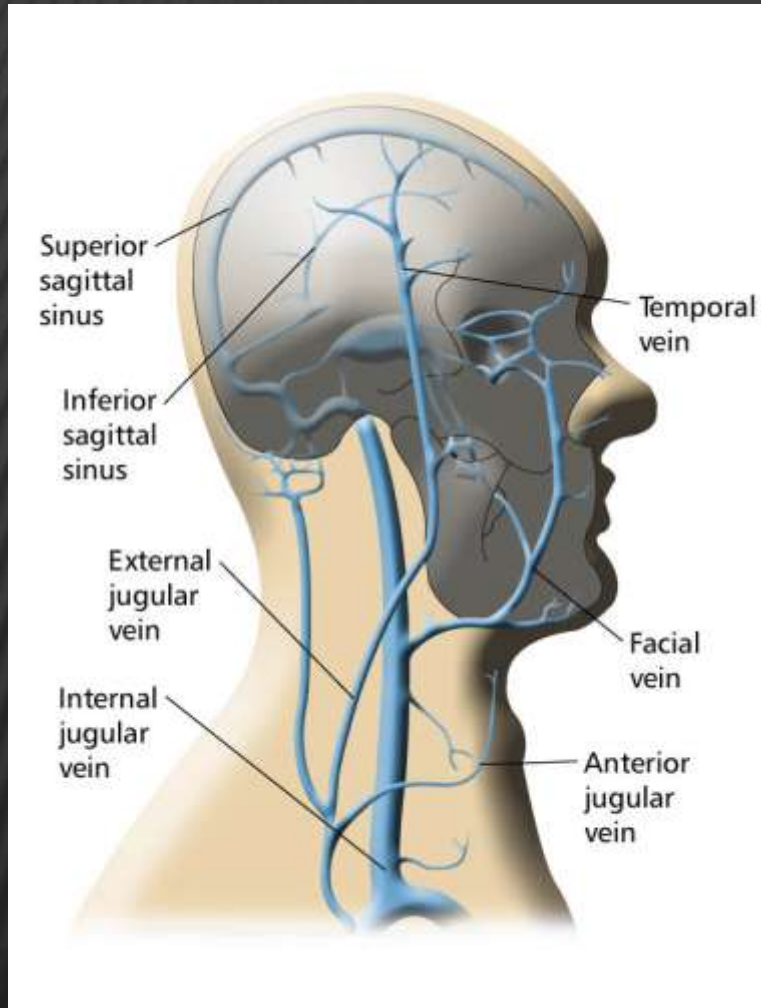


Lieutenant Ralph Rossen (MS) U.S.N. R.,
Herman Kabat, M.D., Ph.D.

120 SECONDS

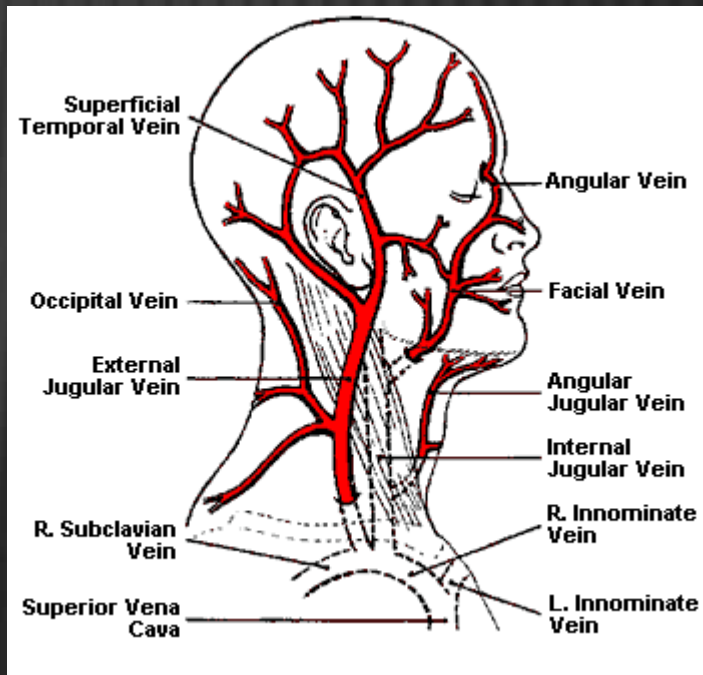


NECK ANATOMY – JUGULAR VEINS



External Jugular Vein

Internal Jugular Vein



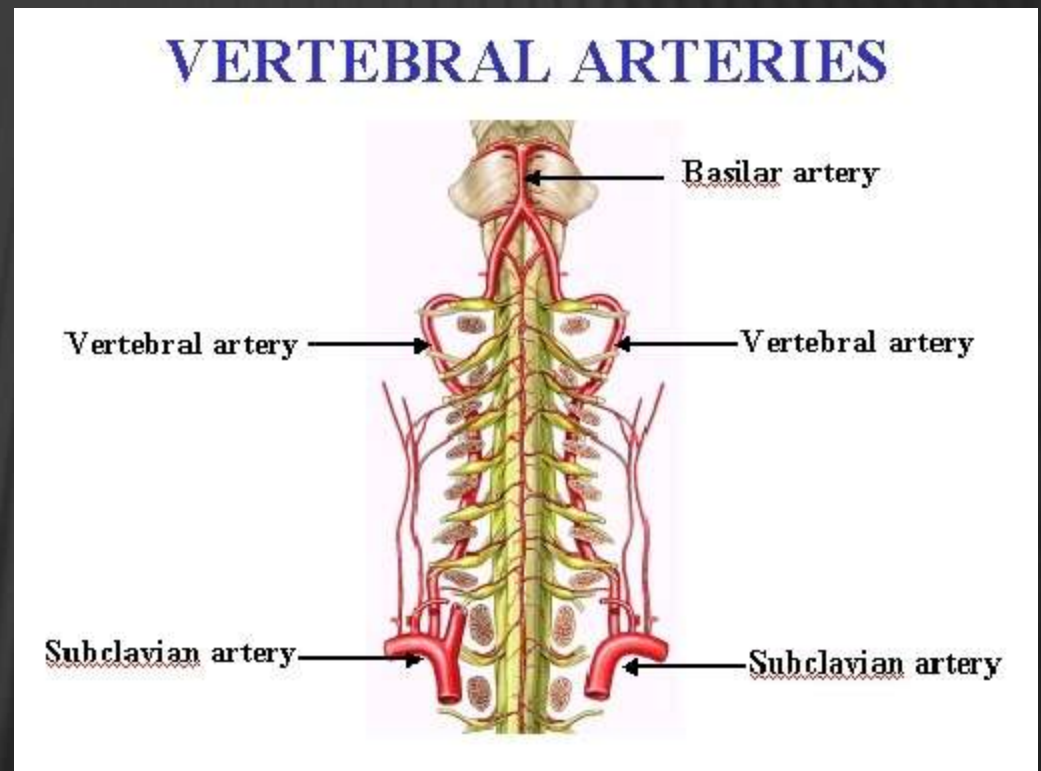
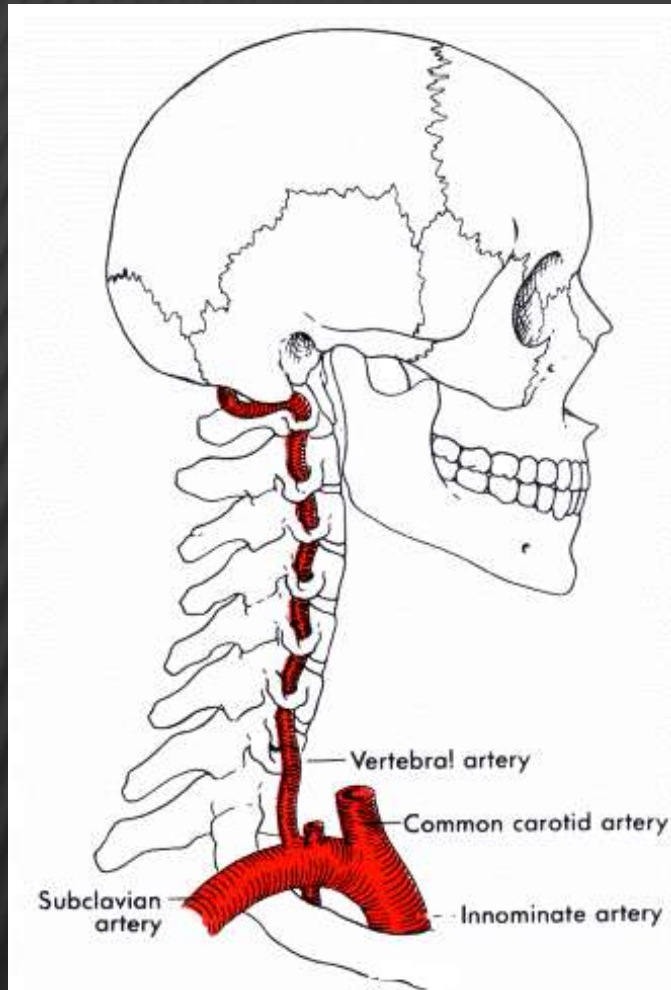
VNR - VEINS

Pre-engorgement of blood by partial occlusion of the veins prior to a VNR application created no deleterious affects.

In fact, studies show that pre-engorgement significantly reduced recovery times after unconsciousness (Rossen Et. Al 1943).

Properly applied VNR produces no deleterious affects to the Jugular Veins.

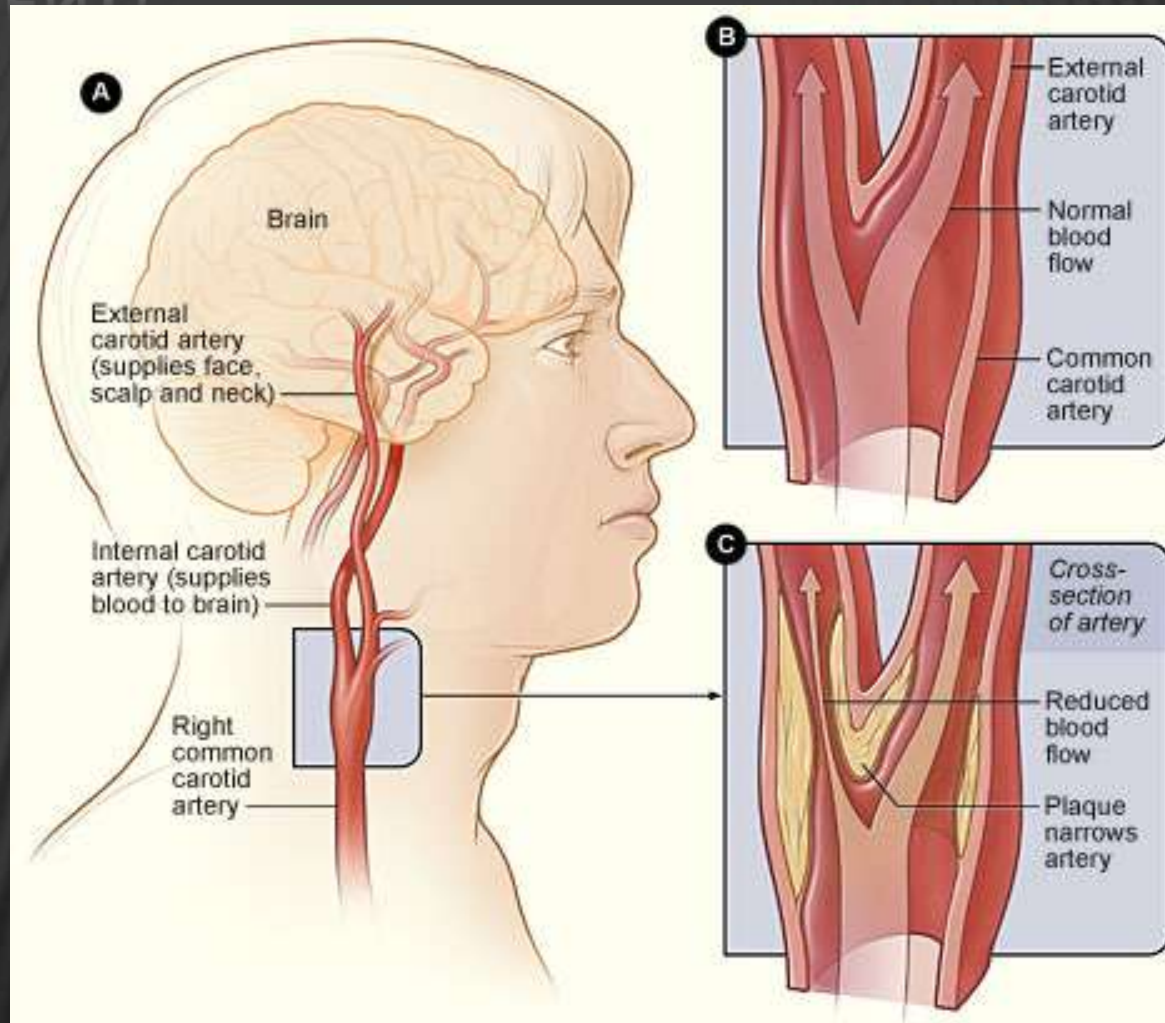
NECK ANATOMY – VERTEBRAL ARTERIES



VNR – VERTEBRAL ARTERIES.

- Properly applied VNR does not affect the Vertebral Arteries as they are deeply embedded within the bony portion of the neck.
- “Forced Rotary Movement” of the head and neck may induce dissection or tearing of the inner wall of one of the four major vertebral arteries.
- A VNR does not utilize “Forced Rotary Movement”
- Many people voluntarily adjust their necks and chiropractors frequently perform these kinds of neck manipulations. According to neurologists, adverse outcomes to these manipulations occur as infrequently as 1/5,800,000 (2007 CPMC)
- However, symptoms of carotid or vertebral artery dissection include a delayed presentation for medical assessment characterized by headache, localized sensory and motor neurological abnormalities (stroke-like symptoms).
- * It is due to this infinitesimal risk that VNR applications to unconsciousness require 2-hour observation period (VNR Manual/2007 CPMC).

ISCHEMIC STROKE (CEREBRAL VASCULAR ACCIDENT)



VNR – ISCHEMIC STROKE RISKS

- It is extremely difficult to induce an ischemic stroke from external sources (2007 CPRC).
- In a minority of elderly subject with preexisting carotid artery narrowing, carotid sinus massage further limit the already reduced blood flow inducing a transient ischemic attack or stroke (less than 0.1% likelihood (2007 CPRC)).
- There is one documented case of a police trainee receiving VNR and dying from Ischemic Stroke; however, the officer previously had a stroke and had previous carotid surgery to remove plaque from within his carotid artery. Neither his employer or trainer knew of the previous surgery. Such a history is uncommon for younger individuals.
- There are no documented cases of Ischemic Stroke caused by an application of VNR for Law Enforcement Purposes.

VNR CONSIDERATIONS

GENERALLY:

- Not applied to obviously elderly people (65 or over)
- Not applied to children under age of 11 years
- Not applied to women visibly pregnant
- Not applied to people with obvious neck injuries or known medical or heart problems.
- Not applied to persons with Down's Syndrome*

* (2007 CPRC)

MEDICAL CONSIDERATIONS

- VNR is shown to be almost 100% effective with no documented injuries or deaths resulting from its application in over 135 years.
- All studies show that an ER visit is not necessary for properly applied VNR techniques.
- Any likely medical risks from use of VNR can be adequately assessed by trained deputies or fire department personnel on scene at the time of VNR application.
- VNR has proven to be **SAFER** than Taser and OC Pepper Spray applications in the field (NLETC Article).

SPORT SCIENCE



POST VNR PROCEDURES

Handcuff subject whether conscious or not.

PRONE HANDCUFFING FOR ALL VNR APPLICATIONS

If unconscious he should recover in 5 to 20 seconds.

Summon medical aid or fire department personnel to evaluate subject before booking.

Begin mandatory 2-hour visual observation of any subject rendered unconscious.

Advise medical personnel that VNR was used and that subject was rendered unconscious.

QUESTIONS?



BREAK

EXCITED DELIRIUM SYNDROME



EXCITED DELIRIUM HISTORY

- × 1849 – The concept of sudden death due to “excited delirium” was introduced by Dr. Luther Bell –

“In examining the Registers or Case Books of the McLean Asylum, from the 30th of December, 1836, to January 1, 1849, a period of twelve years, and comprising an admission of over 1700 cases, I find some forty cases which, according to the diary and my recollections, have presented essentially the indications hereafter to be described. Three quarters at least, of them, terminated fatally”.

AMERICAN
JOURNAL OF INSANITY,
FOR OCTOBER, 1849.

ARTICLE I.

On a Form of Disease resembling some advanced stages of mania and fever, but so contradistinguished from any ordinarily observed or described combination of symptoms, as to render it probable that it may be an overlooked and hitherto unrecorded malady: by LUTHER V. BELL, M. D., Physician and Superintendent of the McLean Asylum for the Insane, Somerville, Mass.

[*Read at the meeting of the Association of the Medical Superintendents of American Institutions for the Insane, May, 1849.*]

Soon after it fell to my professional lot to treat the insane in considerable aggregations, now more than a dozen years since, my attention was culled to a class of cases, not unfrequently presented, and of the gravest moment, but of which I had had no previous experience, and the authorities offered no distinct recognition. Every year since succeeding, has presented more or less specimens of the form of disorder referred to, and I have found, on personal exchange of opinions with my collaborators in this speciality, that its existence has been verified by others, and it seems to have had its existence recognised in an annual report of one of our institutions, some time since.



DR. BELL'S OBSERVATIONS:

- ✕ Fever and Delirium
- ✕ Hands and Tongue Tremulous
- ✕ Pupils dilated or “Contracted”
– no intolerance of light
- ✕ Face pinched up, florid and greasy
- ✕ Anxious
- ✕ Tongue coated thickly, considerably reddened
- ✕ Delusions are infinite – confused – but partaking always of the distressful type
- ✕ Loathing of food – suspicions of it being filthy or poisoned (paranoia)
- ✕ No conveyance of thirst despite indications of dehydration
- ✕ Has to be restrained to bed or will stand until exhausted – scarcely employed in any other cases

DR. BELL'S RESEARCH

- ✘ “This sensation of danger will exhibit itself in the patients attacking anyone who approaches, with a blind fury.”
- ✘ “If held, he will struggle with the utmost desperation, irrespective of the number or strength of those who may be endeavoring to restrain him.”
- ✘ “There is no just calculations of the number supposed to be attacking, no disposition to yield to an overpowering force, noticeable in some degree in the blindest fury of the most intense forms of ordinary mania.”
- ✘ “At the expiration of two to three weeks, your patient will sink to death.”

EXCITED DELIRIUM HISTORY

- ✖ 1881 – “Excited Delirium” found in the Cyclopedia of Practice and Medicine.
- ✖ Between 1954 and 1975, the advent of the neuroleptic drugs like Thorazine transformed psychiatric practice and reduced the incident of exhaustive mania in institutionalized and unmediated patients. (Mash 2016)
- ✖ 1980’s – Acute form of Exhaustive Syndrome starts to occur due to the use and abuse of stimulant drugs such as Cocaine and PCP.
- ✖ 1985 – The term “excited delirium” was popularized in 1985 by Chuck Wetli and D. A. Fishbain in their publication, “Cocaine-induced psychosis and sudden death in recreational cocaine users.”

EXCITED DELIRIUM HISTORY

- ✖ Medical examiner review of these cases did not reveal a definite anatomic cause of death, although drug overdose, trauma and underlying cardiac disease were excluded (Wetli, 1987; Ruttenber et. Al., 1997)
- ✖ 1993 – National Association of Medical Examiners (NAME) recognize Excited Delirium Syndrome as a cause of death (Paquette M. Excited Delirium: does it exist? Perspectives in Psychiatric Care 2003).
- ✖ An estimated 250 patients die in the United States each year from ExDS, about eight to 14 percent of those who experience the syndrome, says Dr. Mark L. DeBard, a professor of emergency medicine at Ohio State University College of Medicine and chairman of the Excited Delirium Task Force. (2009 ACEP)

EXCITED DELIRIUM HISTORY

- ✖ 2003 Federal Legislation requiring all US agencies to report and categorize in-custody deaths.
- ✖ September 10, 2009, American College of Emergency Physicians (ACEP) Task Force RESOLVED the existence of “Excited Delirium Syndrome” or ExDS.
- ✖ December 2011 Weapons and Protective Systems Technologies Center (WPSTC) Special Panel Review of Excited Delirium (National Institute of Justice (NIJ) Report).

2009 ACEP TASK FORCE:

- × Pathologists
- × Neurologists
- × Psychologists
- × Cardiac Electrical Physiologists
- × Psychiatrists
- × Exercise Physiologists
- × Pharmacologists
- × Emergency Service Personnel

MEDICAL DEFINITIONS

- ✘ “Sudden Death” - Applied to the unexpected cardiac deaths of individuals who were in stable medical condition less than 24-hours previously with no evidence of non-cardiac cause. (Chan 2006)
- ✘ “Instantaneous death” - Death that occurs within 5-minutes of the onset of symptoms” (Engle, 1971)
- ✘ “Sudden Cardiac Death” (SCD) – A dramatic and/or spontaneous death that is thought to be (and usually is) caused by a heart condition and may have been caused by exercise such as fighting with law enforcement officers (Behr, 2003, pg. 2)
- ✘ “Sudden Arrhythmic Death Syndrome” (SADS)* - has been defined as having occurred when “no definite cause of death can be found, even after the heart has been examined by an expert cardiac pathologist (Behr, 2003, pg. 2)

*Frequency of SADS occurs in “about 1 in every 20 cases of sudden cardiac death”

1-YEAR IN CUSTODY / NO DRUGS



“EXCITED DELIRIUM” DEFINITIONS

- ✘ A state of extreme mental and physiological excitement, characterized by extreme agitation, hyperthermia, euphoria, exceptional strength and endurance without apparent fatigue.

(Morrison & Sadler, 2001)

- ✘ Excited Delirium is a “medical emergency” no matter what the cause.

(Barkley Burnett & Adler, 2004)

- ✘ “Excited Delirium” – Aggression or violence associated with a state of Delirium.

(DiMaio 2007)

- ✘ An altered mental state with impaired cognition and perception, and severe psycho-motor agitation.

(WPSTC Special Panel Review of ExDS 2011)

NOT A MEDICAL DIAGNOSIS

- ✖ Opponents of excited delirium syndrome note it is not a medical diagnosis, nor recognized by the American Medical Association.
- ✖ At this time, they are CORRECT, as Excited delirium is a “state” a person is in and is not a medical nor psychiatric diagnosis.
- ✖ Opponents of the term “Excited Delirium” have often said that this term is used to cover-up excessive force incidents. None of this is true nor accurate.

TYPES OF DELIRIUM

What opponents fail to understand is that there are nine diagnoses in the International Classification of Disease (ICD) that fit a person in a state of excited delirium:

1. (799.2X) Abnormal excitement
2. (296.00S) Manic Excitement
3. (799.2AM) Psychomotor Excitement
4. (307.9AD) Agitation
5. (799.2V) Psychomotor Agitation
6. (780.09E) Delirium
7. (293.1J) Delirium of mixed origin
8. (292.81Q) Delirium, Drug Induced
9. (292.81R) Delirium, Induced by drug

According to Dr. Deborah Mash Ph.D., excited delirium is at the end of the spectrum of adverse conditions due to drug abuse.

(Mash, 2007)

DIAGNOSIS

- ✖ Personnel are not being trained to make a diagnosis of the individual (this is only for qualified medical and mental health professionals), but rather they are being trained to recognize behavioral cues that may permit them to identify a high-risk individual for a sudden unexpected death.

▪ IPICD 2017

EXCITED DELIRIUM CUES

- × Dilated Pupils / Lid Lift
- × Unbelievable strength.
- × Impervious to pain.
- × Effective resistance against multiple police officers for protracted period.
- × Hyperthermia ($103^{\circ}\text{F} \leq$)
- × Sweating Profusely
- × Foaming at the mouth / white tongue
- × Bizarre violent behavior
- × Aggression
- × Hyperactivity
- × Extreme Paranoia (Terror)
- × Incoherent Shouting (Keening)
- × Skin discoloration (extremities)
- × Large Belly (alcoholism)
- × Rapid breathing or Respiratory Distress (Agonal breathing)

IMPERVIOUS TO PAIN



PRE-DISPOSITION FACTORS

- ✗ Chronic use or under the influence of illicit drugs (cocaine, meth, bath salts, synthetic drugs, Ecstasy, PCP, LSD, psilocybin).
- ✗ Psychiatric patients who are medication noncompliant
- ✗ Cardiomegaly (Myocardial hypertrophy)
- ✗ Under influence of alcohol
- ✗ Dehydration
- ✗ Hypoglycemic (low blood sugar)
- ✗ Underlying psychiatric disease (e.g. paranoid schizophrenia)
- ✗ Traumatic Brain Injury (TBI)
- ✗ Epilepsy
- ✗ Small vessel wall thickening
- ✗ Coronary atherosclerosis
- ✗ Fibrotic scarring
- ✗ Sickle cell anemia

CAUSATION TERMINOLOGY

- × Causation
- × Association
- × Necessary Causes
- × Contributing Causes
- × Proximal Causes

PROXIMAL CAUSES AND CAUSATION OF DEATH:

- × “Choke” Holds
- × Vascular Neck Restraints
- × “Positional Asphyxia” or “Hog-tying”
- × CEW’s
- × OC Spray
- × The tendency to confuse proximity with causality, become greater when the necropsy fails to disclose an anatomic cause of death.

S.B. Karch, B.G. Stephens, (1999).

MEDICAL EXAMINER FINDINGS

- × **Cause of death** – injury or disease that produces a physiological derangement in the body that results in an individual dying. i.e. gunshot wound to chest, Adenocarcinoma of the lung, or coronary atherosclerosis.
- × **Mechanism of death** – is the physiological derangement produced by the **cause of death** that results in death i.e. hemorrhage, septicemia, and cardiac arrhythmia.
- × **Manner of death** – explains how the cause of death came about. The manners of death are: natural, homicide, suicide, accident and undetermined.

A manner of death is ruled undetermined when there is insufficient information about the circumstances surrounding the death to make a ruling or the cause of death is unknown.

Legal Requirements for a Defensible Diagnosis

Law Enforcement Officer (actions must be based upon)	Federal Rules of Evidence Federal Rules of Civil Procedure	Medical Examiner's Report, Findings and Conclusions
OBJECTIVE Facts	FRE 702 and FRCP 26(a)(2)(B)	None required
OBJECTIVE Bases of actions	FRE 702 <ul style="list-style-type: none"> • qualifications; <ul style="list-style-type: none"> ✓ knowledge, ✓ skill, ✓ experience, ✓ training, or education • reliability; <ul style="list-style-type: none"> ✓ based on sufficient facts or data, ✓ product of reliable principles and methods, and ✓ applied the principles and methods reliably to the facts • Scientifically reliable foundations 	None required "ASSUMED" to be reliable "ASSUMED" based on science "ASSUMED" in most states "ASSUMED" by most courts "ASSUMED" by most decision makers NOT held accountable – in most states cannot be challenged
Definitive degree of certainty: <ul style="list-style-type: none"> • Reasonable suspicion (detention) • Reasonable suspicion (frisk) • Probable cause (arrest) • Probable cause (search) 	Reasonable degree of medical or scientific certainty based on reliable foundation, evidence, and scientific support	"ASSUMED" to reasonable degree of medical and scientific certainty

POSITIONAL ASPHYXIA AND EXDS

- ✖ 1988 “Positional Asphyxia” term coined by Dr. Reay, King County Medical Examiner, WA. Despite ban on the use of hog tying, deaths continued to occur. Further research was done. (Reay, Flinger, Stillwell and Arnold, 1992)
- ✖ However, in a 1998 study of 61 cases of unexpected deaths proximal to restraint found that only 20% of those subjects had been placed in the prone hog-tied position. Excited Delirium was determined to be the cause. (Ross 1998)
- ✖ Factors other than body positioning are more important determinants for the sudden, unexpected deaths that occur in individuals who are placed in the hog-tied or four-point restraint position.

REAY'S RESEARCH DISCREDITED

- × 1998 State of California, Price v. County of San Diego. Dr. Chan et. al., physicians from the Department of Emergency Medicine, UCSD, replicated the Reay Study and found the conclusions did not support the original research conducted by Reay. “Positional Asphyxia” was dismissed as a possible cause of death.
- × *“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.*

RESTRAINT METHODS

- ✕ Positional Asphyxia (discredited)
- ✕ Postural Asphyxia (airway position)
- ✕ Mechanical Asphyxia (strangulation)
- ✕ Compression Asphyxia (weight)
 - Adult male requires 4050 ± 320 N of dynamic force to cause flail chest.
Drs. Kroll, Nueman, and Graham – 2017
- ✕ Restraint Asphyxia (Restraint Induced Hypoventilation)

RESTRAINT “ASPHYXIA”

Variable	Death Rate
Rats injected with Saline Solution (n=36)	None
Rats injected with Saline and then restrained (n=12)	None
Rats injected with cocaine (n=12)	17%
Rats injected with cocaine and then restrained (n=12)	58%
Rats injected with cocaine and wrapped in porous plastic to prevent position reversals.	100%

NECK RESTRAINTS AND EXDS

- ✖ City of Los Angeles v. Lyons, (1981).
- ✖ 16 deaths associated to “neck holds”.
- ✖ 1984 Lawsuit appealed.
- ✖ 1987 - Journal of Forensic Sciences produced study by Dr. E. Karl Koiwai MD “Deaths Allegedly Caused by “Choke Holds”.
- ✖ Cerebral Ischemia vs. Asphyxia.
- ✖ According to Dr. Chris Hall, Force Science Institute, VNR is the preferred tool to use against patients suffering from ExDS. Also recommendation by WSCJTC Master Instructor Cadre.

VNR, EXDS OR HOMICIDE?

WARNING:

**THE FOLLOWING VIDEO CONTAINS IMAGES
THAT SOME VIEWERS MAY FIND UPSETTING**

TASER AND EXDS

- ✖ 2005 press release by ACLU claims Taser responsible for 148 deaths in the USA since 1999.
- ✖ Critics claimed the term “Excited Delirium” was coined by Taser International to cover-up their lethal device.
- ✖ Taser International have conducted studies that show three subsequent 5-second exposures (15-seconds) increased hematocrit, potassium, and sodium.*
- ✖ Blood pH was significantly decreased for one hour following exposure, but subsequently returned toward a normal level.*
- ✖ Recent case law has placed CEW’s at an intermediate Use of Force requiring both “Active Resistance” and an “Immediate Threat” to justify their use.

James R. Jauchem a,, Clifford J. Sherry b, David A. Fines b, Michael C. Cook (2006)

CEW'S CAN CONTRIBUTE TO SICD



OC-SPRAY AND EXDS

- ✘ According to a 2016 study by ACLU, 1 in 600 OC-spray applications result in death.

Lethal in Disguise, ACLU, 2016

- ✘ Despite numerous accusations, OC-Spray has never been deemed the “causation” of death.
- ✘ Nevertheless, there can be adverse effects from the use of OC-Spray to include Anaphylaxis.
- ✘ A study on the safety of pepper spray concluded that 2 of 63 people had died from being pepper sprayed, but both had asthma
 - NIJ, 2003
- ✘ There was NY case where OC-Spray was an associated cause.

ACCELERANT + IGNITION = FIRE



EXDS CAUSES

Causes of Excited Delirium fall into four categories:

1. Metabolic (e.g. low blood sugar)
2. Pharmacologic (e.g., cocaine etc.)
3. Infections (e.g. meningitis)
4. Psychological (underlying psychiatric illness)

EXDS CAUSES

- × Alcohol withdrawal
- × Autism
- × Bipolar Disorder
- × Chronic illicit drug use
- × Cessation antipsychotic medications
- × Diabetes (type 4)
- × Delirium
- × Dementia
- × Diabetes
- × Energy drinks
- × Epilepsy
- × Fatigue/exhaustion
- × Hyponatremia (water intoxication)
- × Mental Illness
- × Schizophrenia
- × Synthetic Drugs

NECESSARY CAUSE OF EXDS RELATED DEATHS

- ✖ The necessary cause of excited delirium related to sudden in-custody death proximal to restraint is undetermined. However, there are Contributing Causes, which the death is attributed to. The more of these factors present, the more likely the cause is Excited Delirium Syndrome.
(CPRC 2005)
- ✖ While the precise cause and mechanism of lethality remains controversial, the likely whys and wherefores of sudden death of ExDS victims are seen to be “biological”, since excessive dopamine in the brain triggers the manic excitement and delirium, which unabated, culminates in a loss of autonomic function that progresses to cardiorespiratory collapse.
(Mash 2016)

BREAK

CONTRIBUTING CAUSE #1

- ✗ Chronic stimulant drug use accounts for +80% of ExDS cases.
- ✗ **Stimulant drugs** can cause Arrhythmia.
- ✗ Long term use markedly increases norepinephrine causing malignant arrhythmia and fibrillations.
- ✗ Even low levels of stimulant drugs can cause Tachydysrhythmias (ventricular tachycardia).
- ✗ Left ventricular hypertrophy. (LVH)

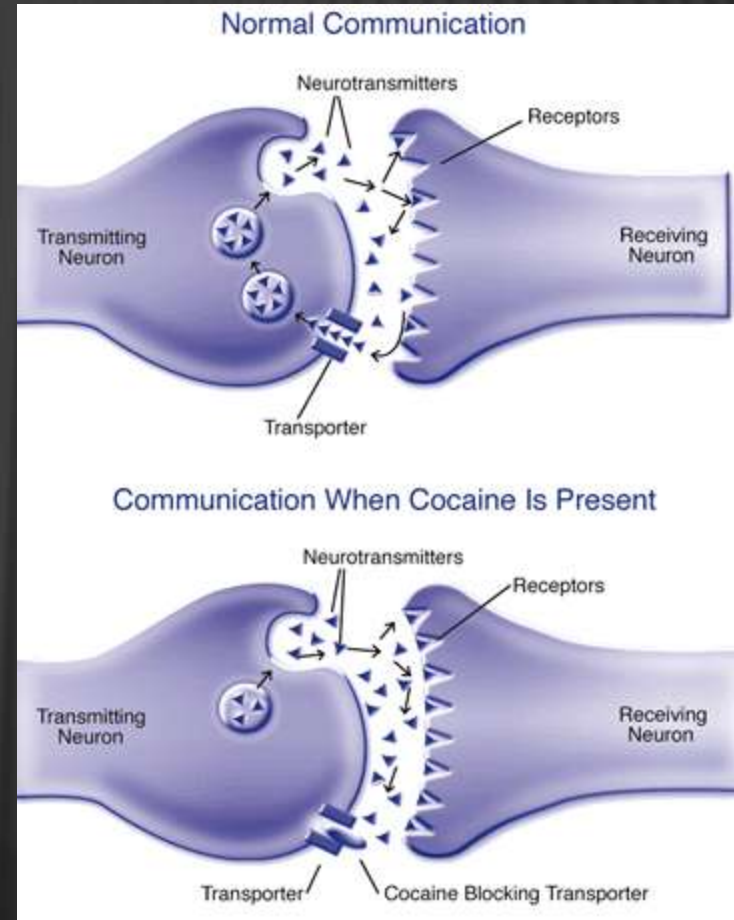


- ✗ LVH patients have 3.4-fold greater odds of supraventricular tachycardia.
- ✗ LVH patients have 2.8 times greater odds of ventricular fibrillation.

* IPICD 2017

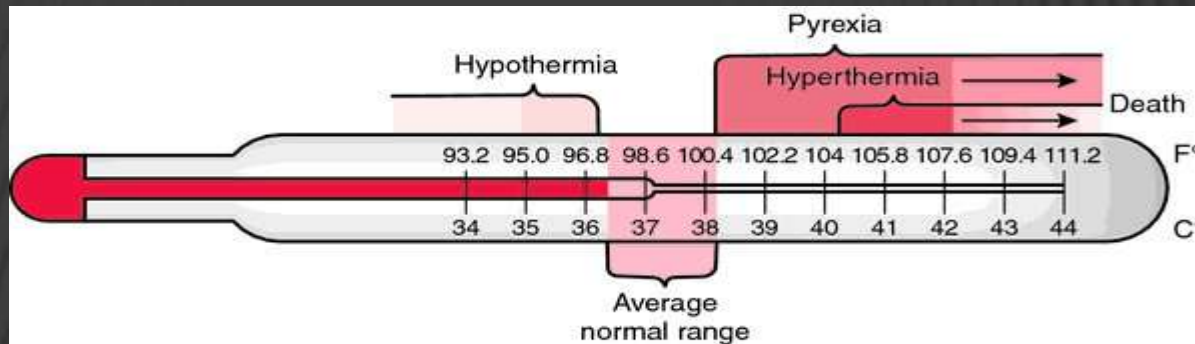
CONTRIBUTING CAUSE #2

- ✖ Stimulant Drug Toxicity to the Brain
 - + Chronic stimulant drug use causes issues in the brain preventing it from clearing dopamine from the synapses resulting in delirium.
 - + Chronic drug use induces the changes in the neurochemistry that lead to excited delirium.
(Stephens, Jentzen, Karch, Wetli, & Mash, 2004)
 - + Self Imposed cessation of prescribed anti-psychotic medications can cause similar effect.
 - + 34% of ExDS cases associated with chronic mental illness.



CONTRIBUTING CAUSE #3

✖ Hyperthermia



- + All four categories of ExDS have hyperthermia as a hallmark characteristic (Wetli, 2005, p. 278; Peters, 2007)
- + Core temperatures of $>103^{\circ}\text{F}$ strongly supports stimulant induced excited delirium. (*Stephens, Jentzen, Karch, Wetli, & Mash, 2004)
- + A loss of dopamine transporter regulation together with increases in heat shock protein 70 (hps 70) expression as a biomarker of hyperthermia.
- + A high body core temperature at the time of death is key in proving an excited delirium syndrome related death.

CONTRIBUTING CAUSE #4

× Stimulant Induced Rhabdomyolysis.

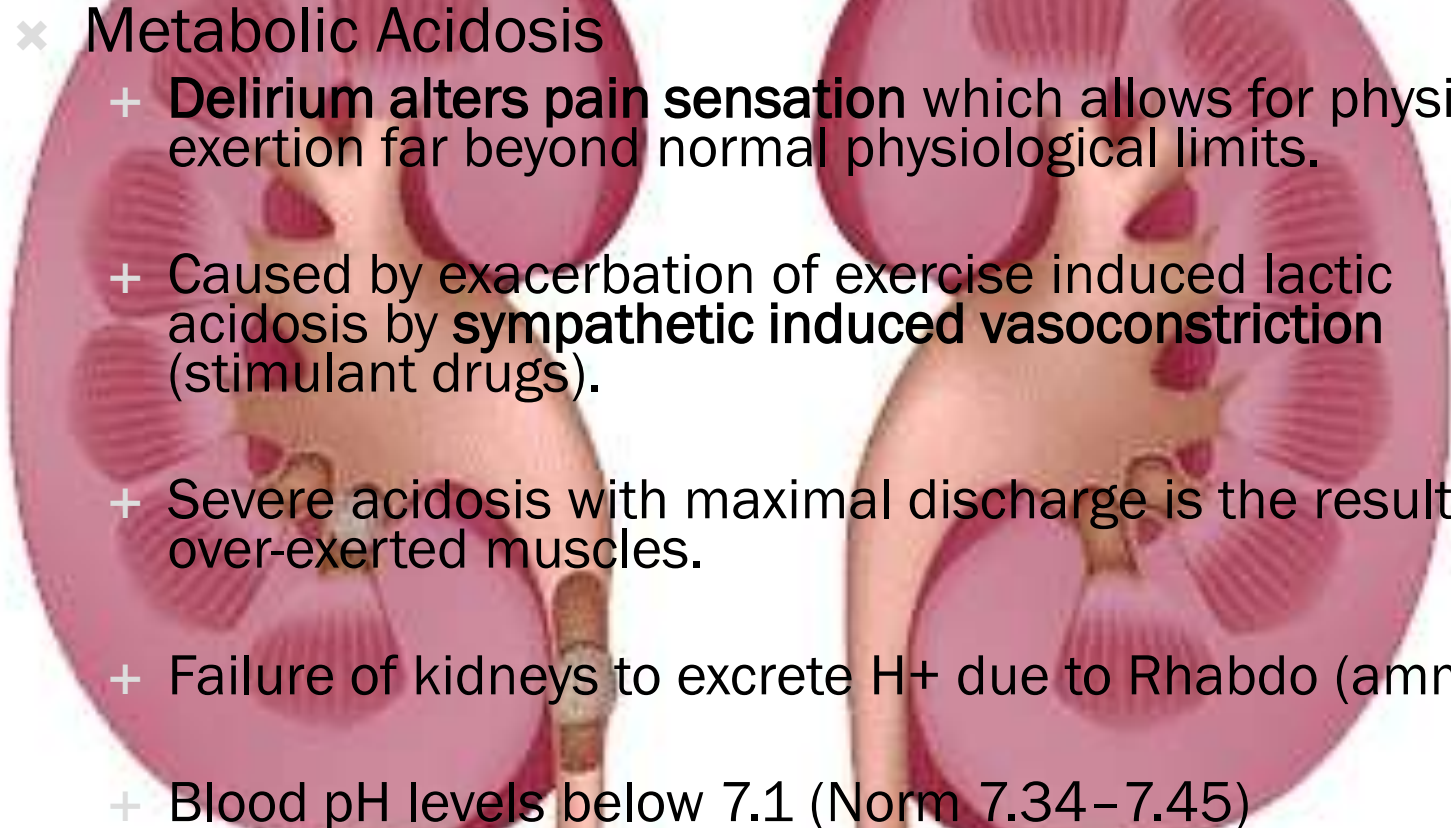
- + Once rhabdomyolysis begins, muscle cells break down and allow contents of the cell to leach their contents into the blood stream (Myoglobin, enzymes and electrolytes),
- + Hyperkalemia - increased levels of Potassium and Sodium make the heart more susceptible to cardiac arrhythmia (Potassium Chloride is used for lethal injections).
- + Peak CK levels in excess of 40,000 IU/L (normal is 60-400 IU/L).
- + CK concentrations rise steadily for 12 hours, and peak for 1-3 days.
- + Elevations in muscle enzymes levels are observed in asymptomatic cocaine users AND untreated schizophrenia suffers.
- + Kidneys fail due to stress or heart stops due to high levels of potassium in the blood due to workload on the kidneys.

RHABDOMYOLYSIS

- ✖ February 17, 2015
- ✖ Treated for contusion
- ✖ Rhabdo check positive @ 14,000 U/I
- ✖ Peaked at 300,000 U/I
- ✖ Dialysis for nine days saved his life



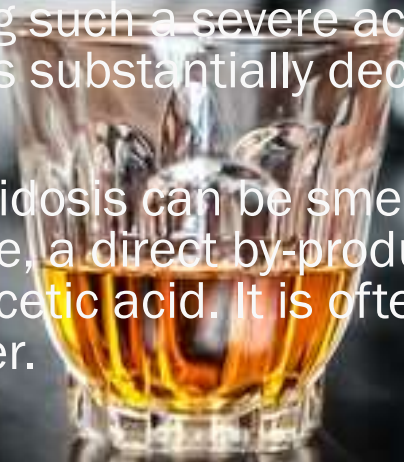
CONTRIBUTING CAUSE #5

- 
- Two anatomical illustrations of human kidneys, one on the left and one on the right, shown in a cross-section view. They are reddish-brown with visible internal structures like the renal cortex, medulla, and pelvis. The kidneys are positioned behind the main text list.
- × Metabolic Acidosis
 - + **Delirium alters pain sensation** which allows for physical exertion far beyond normal physiological limits.
 - + Caused by exacerbation of exercise induced lactic acidosis by **sympathetic induced vasoconstriction** (stimulant drugs).
 - + Severe acidosis with maximal discharge is the result of over-exerted muscles.
 - + Failure of kidneys to excrete H^+ due to Rhabdo (ammonia)
 - + Blood pH levels below 7.1 (Norm 7.34–7.45)

CONTRIBUTING CAUSE #6

× Ketoacidosis

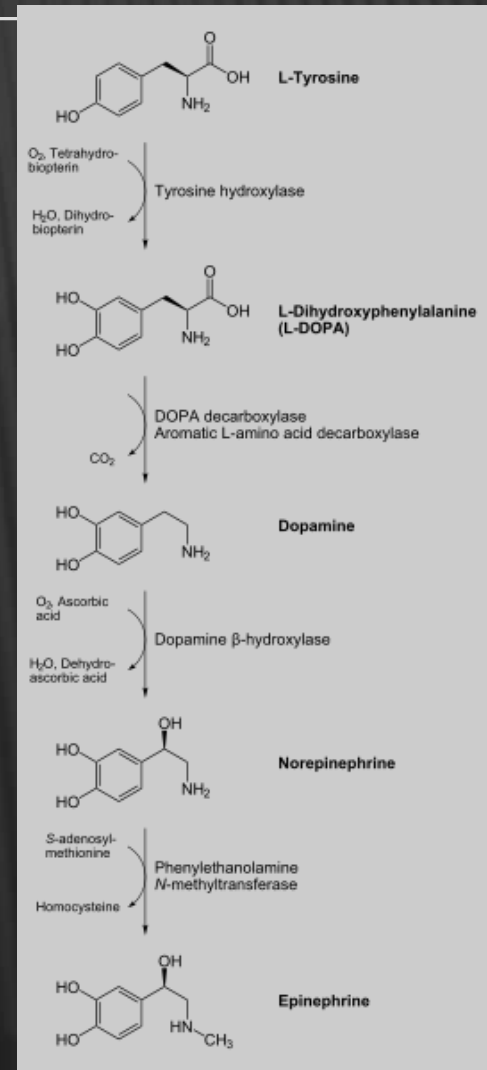
- + Three common causes of ketoacidosis are diabetes, starvation and alcoholism.
- + a metabolic state associated with high concentrations of ketone bodies, formed by the breakdown of fatty acids and the deamination of amino acids.
- + In ketoacidosis, the body fails to adequately regulate ketone production causing such a severe accumulation of keto acids that the pH of the blood is substantially decreased.
- + Ketoacidosis can be smelled on a person's breath. This is due to acetone, a direct by-product of the spontaneous decomposition of acetoacetic acid. It is often described as smelling like fruit or nail polish remover.



CONTRIBUTING CAUSE #7

✖ Neuronal Catecholamine Release

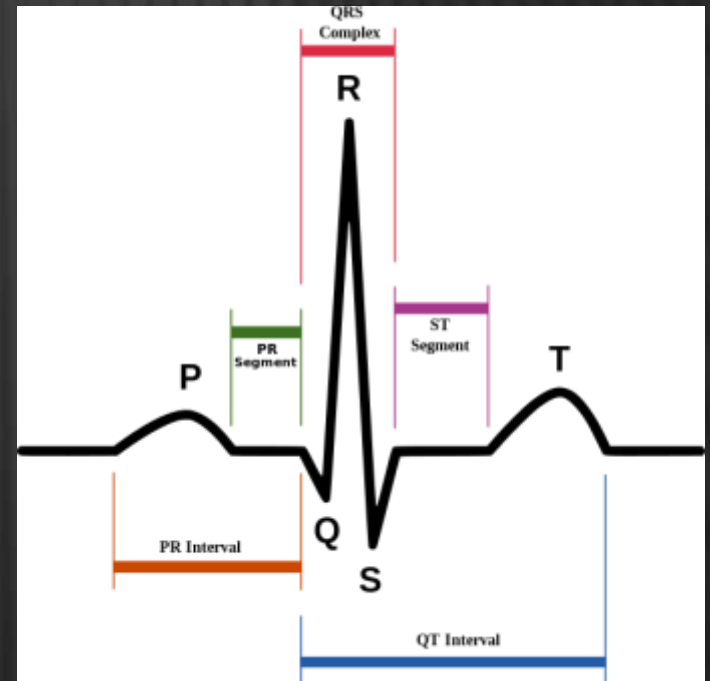
- + During violent activity, the body releases catecholamines into the blood, which can sensitize the heart and promote rhythm disturbances.
- + Epinephrine (adrenaline), Norepinephrine (fight or flight) and Dopamine (reward neurotransmitter) are released into the system.
- + Catecholamines also enhance the toxicity of stimulant drugs, which can lead to seizures, respiratory arrest and cardiac arrest.
- + Yet peak levels of catecholamines are reached not during the physical activity but in those 2 to 5 minutes after cessation of the activity and may reach ten times base levels.



CONTRIBUTING CAUSE #8

✗ Antipsychotic drugs

- + Voluntary cessation of bipolar/schizophrenia drugs can also induce the changes in the neurochemistry that lead to excited delirium.
- + Drugs such as Haloperidol, Droperidol and Thioridazine.
- + All three antipsychotic medications are reported to cause QTc interval prolongation and, on occasion, torsade de pointes and death (Ventricular fibrillation).



CONTRIBUTING CAUSE #9

- ✖ Genetic susceptibility to arrhythmia.
 - + Dr. Patrick MacLeod, Victoria BC medical geneticist, and one of the ACEP panel experts, found a rare gene that makes one more prone to the negative effects of Excited Delirium.

CONTRIBUTING CAUSE #10

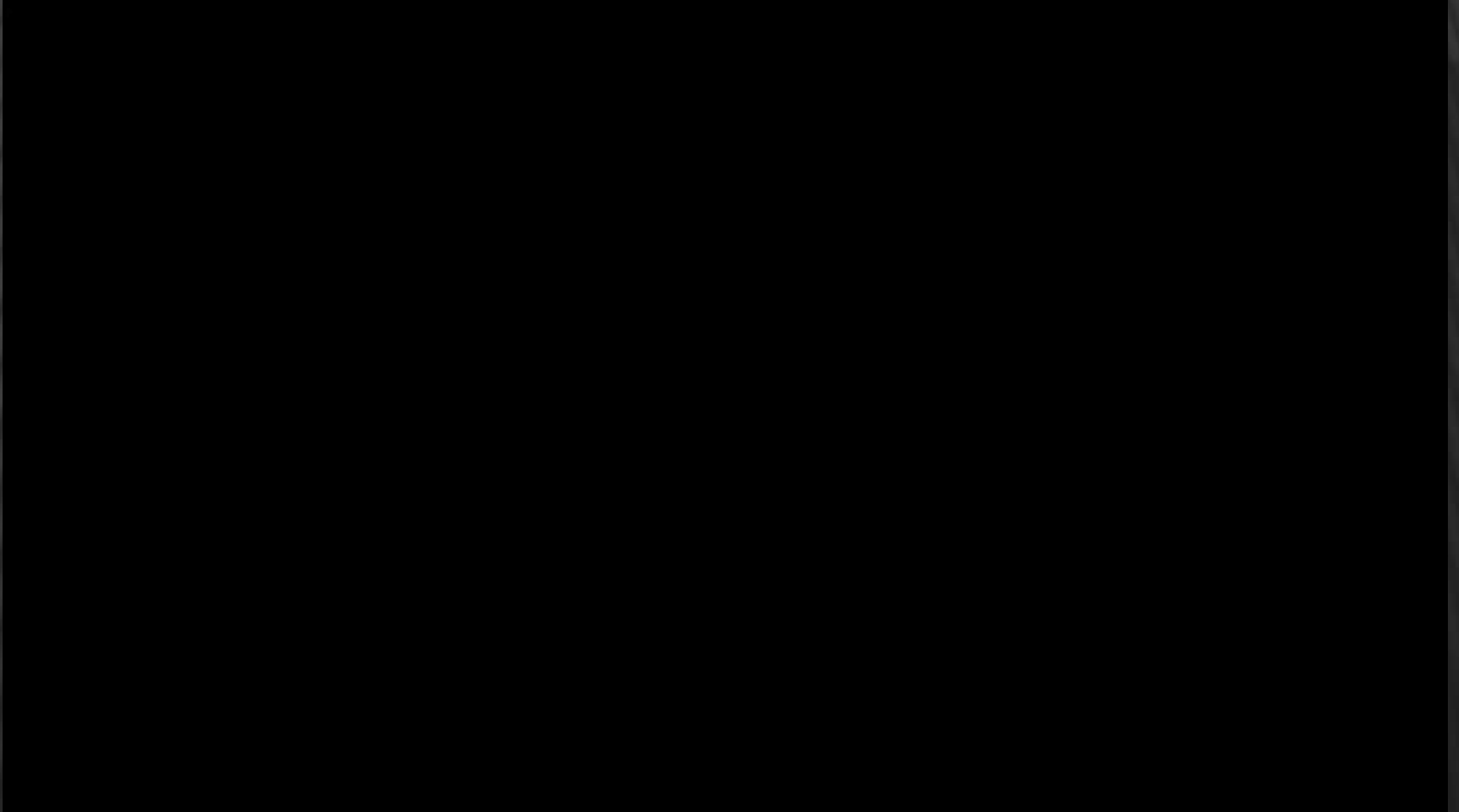
× Hypertrophic Cardiomyopathy

- + A complex but relatively common form of genetic heart muscle disease that occurs in 1:500 people, but often goes undiagnosed in the community.
- + It is the most common cause of heart related sudden death in people under 30 YOA.
- + A portion of the myocardium is hypertrophied (thickened) without any obvious cause.

CONTRIBUTING CAUSE #11

- × **Restraint Proximal to Arrest.**
 - + Blood pH lowers during Excited Delirium
 - + Blood pH affected by:
 1. Severe acidosis
 2. Hypoventilation
 - + Hypoventilation can be affected by:
 1. Drug induced via dopamine production
 2. Body Positioning
- × **Subjects who are restrained in a prone position may be unable to breathe rapidly enough to exchange carbon dioxide; hence, the excess carbon dioxide contributes to an acidotic state (restraint induced hypoventilation)**

RESTRAINT INDUCED HYPOVENTILATION



SUMMARY

1. Myocardial hypertrophy
2. Chronic Stimulant Drug Use or Psychiatric Disease
3. Hyperthermia
4. Stimulant induced rhabdomyolysis
5. Metabolic Acidosis pH >7.1
6. Ketoacidosis (starvation, diabetes, alcoholism)
7. Fight/Flight Response
8. Prolongation of QT Interval
9. Genetic Dispositions
10. Heart Disease
11. Restraint induced hypoventilation

CONCLUSION

- ✕ Death from Excited Delirium Syndrome results from a fatal cardiac arrhythmia due to **hyperadrenergic state** caused by:
 1. The excited delirium, which in itself triggers release of catecholamines
 2. Additional release of catecholamines due to the struggle
 3. A rapid and steep drop in blood potassium concentrations following cessation of the struggle in association with increasing levels of catecholamines (period of peril).

(Over)

CONCLUSION (CONTINUED)

- × The hyperadrenergic state is almost invariably aggravated by the effects of:
 1. Illegal stimulants, which directly and indirectly cause increased levels of catecholamines
 2. Medications that have either the same actions as the stimulants in causing increased concentrations of catecholamines and/or cause prolongation of the QT interval.
 3. The presence of natural disease of a degree insufficient in itself to cause death but when in combination with a hyperadrenergic state can do so.

BREAK

EXDS RESPONSE MEASURES

LAW ENFORCEMENT - MEDICAL

- × **IDENTIFY** – Observe, record, and communicate the indicators related to this syndrome – handle primarily as a MEDICAL EMERGENCY.
- × **CONTROL** – Control and/or restrain subject as soon as possible to reduce the risks related to a prolonged struggle
- × **SEDATE** – Administer sedation as soon as possible. Consider calming measures. Remove unnecessary stimuli where possible, including lights and sirens.
- × **TRANSPORT** – Take to hospital as soon as possible for full medical assessment and/or treatment.

WPSTC – Special Panel Review of ExDS (2011)

IDENTIFY –

1. Extremely aggressive or violent behavior
2. Constant or near constant physical activity
3. Does not respond to police presence
4. Attracted to/destructive of glass/reflective
5. Attracted to bright lights/loud sounds
6. Naked / inadequately clothed
7. Attempted “self cooling” or hot to touch
8. Rapid breathing
9. Profuse sweating
10. Keening (unintelligible animal-like noises)
11. Insensitive to/extreme tolerant of pain
12. Excessive Strength (out of proportion)
13. Does not tire despite heavy exertion.

CONTROL =

- ✖ Ensure Adequate Back-Up
 - + Recommended minimum of four officers, adequate police presence to handle the incident
 - + Remove civilians from the scene
 - + Stage Fire Department (medical)
 - + Isolate victim
 - + Try to verbally de-escalate
 - + Eliminate unnecessary audible and visual stimulus

DE-ESCALATION WORKS



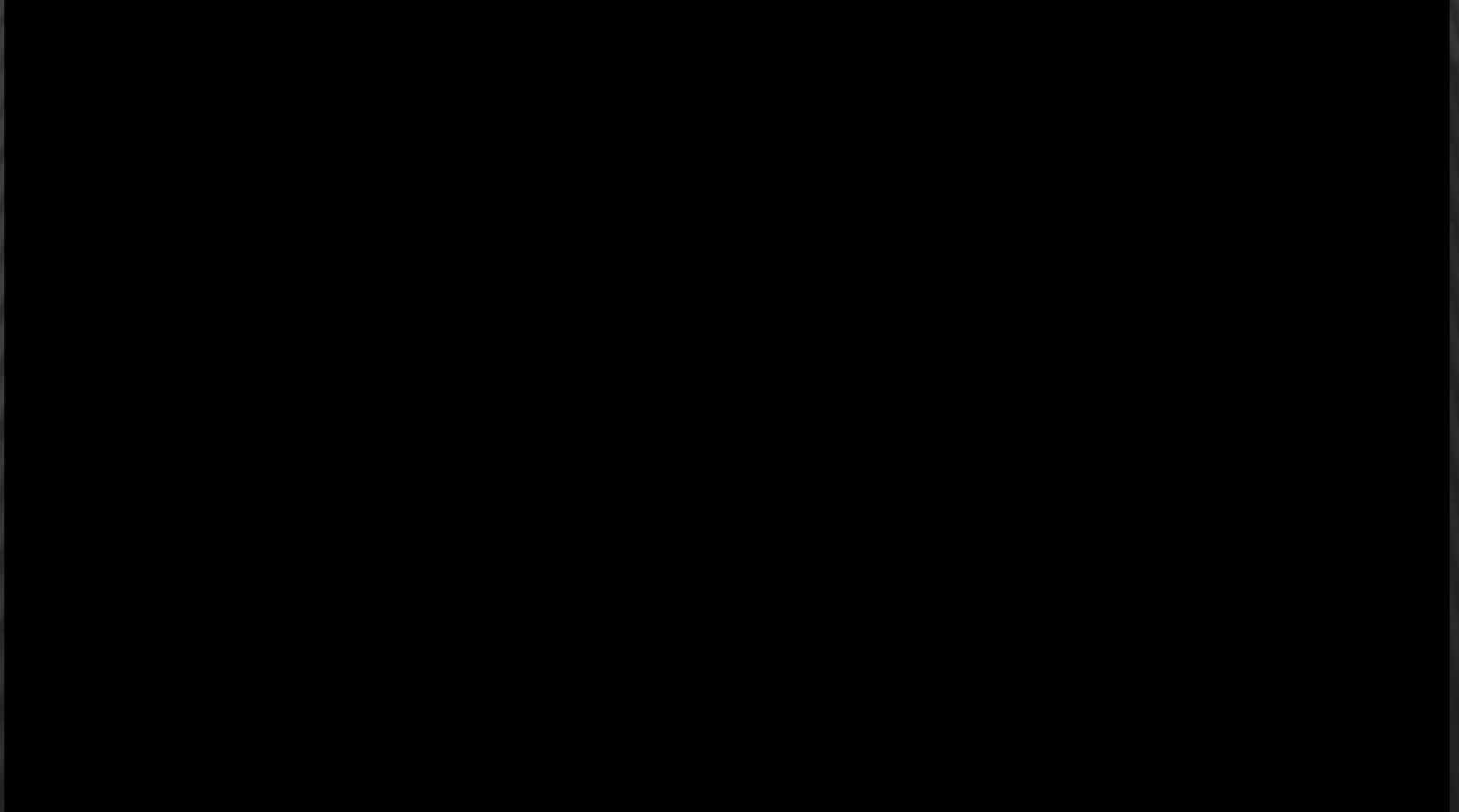
CONTROL - (CONTINUED)

- + To prevent further injury to subject or others, officers need to go in.
- + Use rapid and overwhelming tactics to gain control.
- + Restrain using the minimum amount of fight.
- + The longer they fight, the increased chance of a negative outcome (death).
- + VNR is the preferred intermediate tool, not Taser* or **pepper spray.
- + ExDS is a medical emergency AFTER THE CUFFS ARE ON

*Studies show Taser can increase both lactic acidosis and potassium levels after 15-second exposures

**Neuronal catecholamine levels have been shown to increase after OC Exposure

PREPARE TO FIGHT



CONTROL - (CONTINUED)

- ✖ Ensure Breathing
 - + DO NOT hinder the subject's ability to breathe!
 - + DO NOT subject their thorax to unnecessary weight.
 - + Place / secure subject into a recovery position.
 - + Subjects will be metabolically acidotic from the intense exertion.
 - + Exertion will often continue while in handcuffs and the tendency may be to keep subject pinned to the ground until EMS arrives.
 - + Subjects need to breathe at a faster and deeper rate to buffer the build-up of metabolic acid and prevent cardiopulmonary arrest.

AGONAL BREATHING



SEDATION -

- ✖ ExDS is a medical crisis, and subjects should be immediately transferred to the care of EMS personnel.
- ✖ These protocols often (not universally) allow emergency medical personnel to sedate if they determine an individual likely has Excited Delirium Syndrome.
- ✖ Law Enforcement may assist medical personnel with restraining the subject for sedation (Intranasal or intramuscular).

EMS TREATMENT OF EXDS

1. Chemical restraint to slow down SNS
(See Table 5)
2. IV Immediately (Rhabdomyolysis)
3. Body-core temperature should be part of life saving measures.
4. Blood pH reading in the field (if possible).
 1. If blood pH is below 7.10 immediately push sodium bicarbonate (Acidosis).
 2. Immediately begin cooling procedures via cooling packs, water mist, blankets, drugs or infusion of cold saline IV. Care taken not to “overshoot” (hypothermia).

Excited Delirium Task Force, white paper report on ExDS (2009)

SEDATION AGENTS FOR EXDS–TYPE SYMPTOMS

Class	Agent (Trade Name)	Available Routes	Dosing (mg)*	Onset (min)	Duration (min)
Midazolam (Versed)		IN	5	3-5	30-60
		IM	5	10-15	120-360
		IV	2 - 5	3-5	30-60
Lorazepam (Ativan)		IM	4	15-30	60-120
		IV	2 - 4	2-5	60-120
Diazepam (Valium)		IM	10	15-30	15-60
		IV	5 - 10	2-5	15-60
†Haloperidol (Haldol)		IM	10– 20	15	180-360
		††IV	5 – 10	10	180-360
†Droperidol (Inapsine)		IM	5	20	120-240
		IV	2.5	10	120-240
Ziprasidone (Geodon)		IM	10– 20	10	240
Olanzapine (Zyprexa)		IM	10	15-30	24 hrs
Ketamine (Ketaset, Ketalar)		IM	4-5 mg/kg	3-5	60-90
		IV	2 mg/kg)	1	20-30

IN: Intranasal; IM: Intramuscular; IV: Intravenous

* Typical adult dosing for severe agitation.

† The Food and Drug Administration has issued “Black Box” warnings regarding potential serious adverse effects (QT prolongation and torsades de points) with these agents. Clinicians should use their clinical judgment regarding the risk / benefit ratio on a case by case basis.

†† Though widely used in clinical practice, Haloperidol is not FDA approved for intravenous administration.

(For adequate control of ExDS, the above doses are conservative and describe a reasonable starting point. Clinical effect in ExDS may require doses greatly in excess of those for traditional medical use in other conditions).

TRANSPORT -

- ✖ Transport to a medical facility is accomplished only by qualified medical personnel as soon as possible.
- ✖ Law Enforcement should be required to follow or ride in the ambulance for UOF.
- ✖ “Many emergency room nurses will not know about excited delirium” (Theresa Di Maio)
Therefore, knowledge of ExDS is needed for evaluation and treatment at a medical facility.

DOCUMENT

- ✘ ExDS cases should be documented like any other critical incident, regardless of “outcome”.
- ✘ Include photos, video and audio, as well as detailed reports and supervisor response.
- ✘ Everyone write paper on all Use of Force.
- ✘ Guard against ExDS being the focus of the event – remember, you are not making a medical diagnosis.
- ✘ Slice and rebuild your observations of the subject.
- ✘ Don’t use subjective statements e.g. “He was impervious to pain, he had super-human strength, he was breathing...”

INVESTIGATOR CHECK LIST

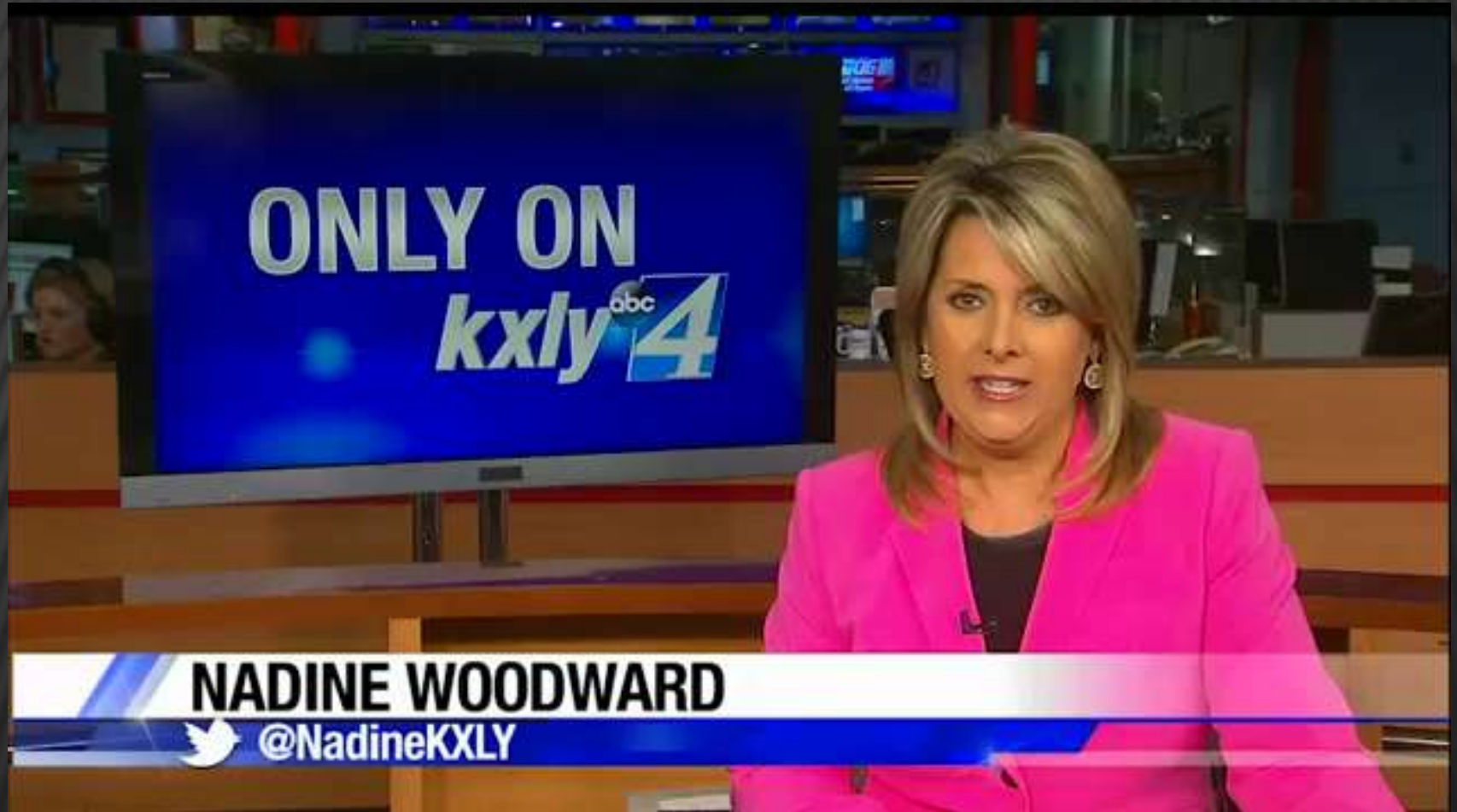
- × ID the suspect ASAP.
- × All specifics on UOF, length of struggle and methods that failed.
- × All specifics on method of restraint.
- × Obtain family / suspect history
- × Obtain medical history
- × Obtain criminal history
- × List current medications
- × Take the blood ASAP!!!
- × Obtain body core temperature
- × Obtain urine levels of cocaine and benzoylecgonine.
- × Obtain an analysis of the person's brain. (800-UM-BRAIN).
- × Obtain analysis of the hair (drug history).
- × Obtain heart size (myocardial hypertrophy).
- × Obtain analysis for arthrosclerosis, focal myoncrosis, myofirbrosis, small vessel wall thickening, and heat shock proteins.
- × Obtain the environmental factors such as weather, humidity and temperature at the time of the struggle.

POST-MORTEM CHECKLIST

- ✘ Pulmonary Edema (lung fluid).
- ✘ Cerebral Edema (brain swelling).
- ✘ Toxicological screening.
- ✘ The brain must be harvested within 24-hours, put on dry ice and sent for analysis (1-800-UM-BRAIN). Corneal slices of the anterior stratum and substantia-nigra need to be collected as soon as possible in relation to the time of death (IPICD 2017).
- ✘ Neurochemical examination revealing abnormal dopamine synaptic markers or receptors in the stratum and hypothalamus.
- ✘ Brain tissue analysis revealing elevated heat shock proteins.
- ✘ Lack of other causes of death (trauma, asphyxiation, overdose, etc.)

(Guidelines for investigating OIS, ARD and DIC.
Darrell L. Ross – Gary M. Vilke – 2018)

EXCITED DELIRIUM SYNDROME SAVE



QUESTIONS?

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