

RESTRAINT ASPHYXIA – SILENT KILLER

Parts One & Two, and References

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INFORMATION Related to TERMS Used Within This Article:

GENDER TERMINOLOGY:

Although I am a **woman**, it is easier to use male pronouns ("he," "him," and so on) than writing "she/he," "her/him," and the like. So, that's what I've done.

MEDICAL TERMINOLOGY:

Because people with little-to-no medical education require this information just as much as medical professionals, I have minimized use of medical terms and explained concepts as simply as possible. Any reader who is offended by the simplicity of my explanations, can go play in my **RESTRAINT ASPHYXIA LIBRARY**.

Within this article, the term, "**VICTIM**" means:

a "patient," "client," "consumer," "suspect," "subject," "criminal," "inmate," "student," *or the like!*

I've used the term, **VICTIM**, because – **without fail** – ALL individuals who have ever died due to restraint asphyxia were "Victims" of some form of problem that caused them to involuntarily suffer some form of "**ALTERED LEVEL OF CONSCIOUSNESS**" that ultimately caused them to require application of restraint.

Within this article, the terms "**RESPONDER**" or "**RESTRAINER**" refer to ANY PERSON who's job description includes the responsibility of responding to, and possibly restraining, ANY individual experiencing an "emergency" that requires restraint.

And, any **RESPONDER** who is responsible for ensuring the care and safety of a "patient," "client," "consumer," "suspect," "subject," "criminal," "inmate," *or the like*, becomes a **RESTRAINER** when they begin to apply restraint.

In other words: **RESPONDERS** and **RESTRAINERS** are Emergency Medical Services (EMS) or Fire Department personnel; Law Enforcement Officers or Correctional Facility personnel; Emergency Department (ED) personnel; General-Hospital or Psychiatric Facility personnel; **OR they are** Staff members of Homes, Schools, Vocational Training, or other Facilities established for the care and supervision of mentally ill or developmentally-disabled individuals – **AND THE LIKE!**

"PHYSICAL" Restraint is defined as one or more human beings applying their HANDS (and other of their BODY PARTS) to an individual's body, for the purpose of preventing that individual's freedom of movement.

"MECHANICAL" Restraint is defined as affixing any form of "DEVICE" to any part of an individual's body, for the purpose of preventing that individual's freedom of movement.

In the past, the term "MECHANICAL Restraint" was most often used only when referring to METAL restraint devices – handcuffs, ankle "shackles," and the like. However, *any* DEVICE used for

restraint (be it metal, or a roll of gauze, a sheet, a plastic or Velcro® strap, a leather or fabric cuff/strap, or the like) is, technically, a "mechanical" restraint.



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"PHYSICAL FORCEFUL-PRONE-RESTRAINT"

is defined as placing an individual's body FACE-DOWN ("prone") upon ANY surface (such as the ground, a long back board, an ambulance wheeled stretcher, a bed, or any other surface), and Restrainers physically applying pressure with their HANDS (and/or OTHER BODY PARTS) to the Victim's shoulders, posterior torso ("back"), hips, *and/or* upper legs – **physically** preventing the Victim from moving out of the PRONE position.

"MECHANICAL FORCEFUL-PRONE-RESTRAINT"

is defined as placing an individual's body FACE-DOWN upon a MOBILE surface (such as a long back board, an ambulance wheeled stretcher, a bed, or any similar device) and then using a MECHANICAL device, of any sort, to affix the Victim's body to the mobile surface – **mechanically** preventing the Victim from moving out of the PRONE position.



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Clearly, "PHYSICAL" and "MECHANICAL" forms of FORCEFUL-PRONE-RESTRAINT are relatively the same thing: Application of FORCE to an individual's body while he is in a PRONE position, and preventing his movement out of the forcefully-prone-restrained position.

Hence, within this article, "**FORCEFUL-PRONE-RESTRAINT**" refers to EITHER physical or mechanical means of forcefully maintaining someone in a prone-restraint position.



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The term
"**HOBBLE RESTRAINT**" has
previously been *inaccurately defined* (even
by ME – *GASP!*) as being simply another
term for "**HOGTIE**" restraint.



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Technically, however, "**Hobble**" refers *ONLY* to the binding of an individual's ANKLES together.
A hobbled individual is not hogtied. A hogtied individual is also hobbled.

"HOGTIE RESTRAINT" is universally (correctly!) defined as

- binding an individual's wrists together behind his back (usually via handcuffs),
- then binding his ankles together ("hobbling" him),
- then bending his knees and tying his hobbled-ankles to his bound-wrists.



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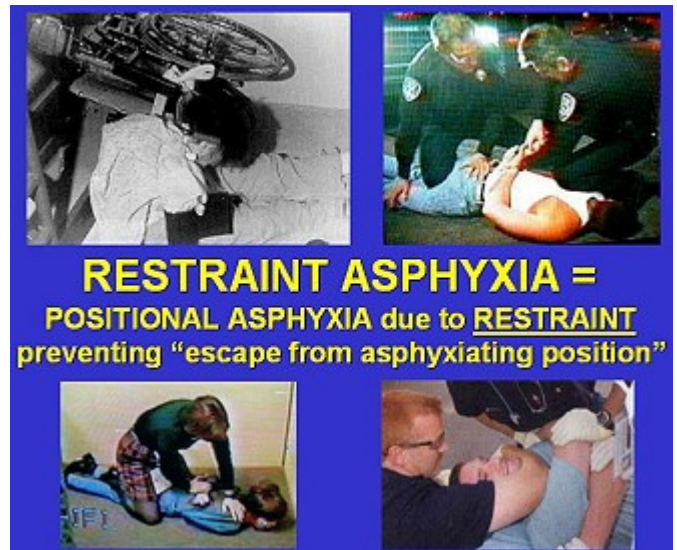
"POSITIONAL ASPHYXIA" is most simply defined as death that occurs because the **POSITION** of a person's body interferes with respiration (breathing), and the person cannot get **OUT** of that position. Death occurs due to the person's inability to **BREATHE** anymore.

ANY body position that **obstructs the AIRWAY**, **OR** that interferes with the **muscular or MECHANICAL MEANS** of getting air into and out of the body (the body's **"BELLOWS" function**) will result in a positional asphyxia death, if the person cannot get out of it.

"RESTRAINT ASPHYXIA" is a *form* of **POSITIONAL ASPHYXIA** death.

The factor that distinguishes a "restraint asphyxia" death from a "positional asphyxia" death, is that some form of **RESTRAINT** is the reason the Victim couldn't **ESCAPE** the asphyxiating position.

First proposed in 1993, the phrase "Restraint Asphyxia" has become more widely-adopted since 1998.^(1,2) Yet, several other phrases continue to be used when referring to incidents of restraint asphyxia: "Mechanical Asphyxiation," "Traumatic Asphyxiation," "Sudden In-Custody Death," and the like.



Why is this **TERMINOLOGY** "important?"

When unrelated to restraint, positional asphyxia deaths are caused by the "passive entrapment" of an individual – no "person" is responsible for the Victim being unable to escape the asphyxiating position. Thus, the "manner" of death historically attributed to "positional asphyxia" has always been **"ACCIDENTAL."**

Since 1994, however, leading medical examiners and forensic pathologists officially recognized restraint-related forms of positional asphyxia as being quite different. Restraint asphyxia deaths are due to **PEOPLE** having acted to physically or mechanically entrap someone.⁽³⁻⁵⁾ Thus, the "manner" of death for the majority of restraint asphyxia deaths is now recognized as **"HOMICIDE."**

RESTRAINT ASPHYXIA – SILENT KILLER

Responders sometimes encounter excited or agitated individuals who act-out in physically-exertional, violent and dangerous manners. To ensure Victim AND Responder safety – and to facilitate *thorough* medical evaluation and care of the Victim – violently-exertive, excited or agitated individuals **MUST** be RESTRAINED.

However, just as "choking" someone into unconscious submission is **NOT appropriate**, restraining a violently-exertive, excited or agitated individual in any OTHER manner that prevents them from being able to **BREATHE** is ALSO, emphatically, **NOT appropriate!**

The physical or mechanical restraint of an individual is *always* a "last resort." The "least-restrictive" means of restraint (or use of force) – such as verbal counsel and de-escalation techniques – should always be employed before Responders resort to using hands-on or mechanical restraint.⁽⁶⁻⁸⁾ Unfortunately, verbal restraint techniques are *entirely ineffective* when someone has become (due to any of a myriad of causes) the Victim of an **ALTERED LEVEL OF CONSCIOUSNESS**. Someone with an altered level of consciousness does not have the mental ability to comprehend verbal counsel or de-escalation techniques. Thus, some form of restraint application is **REQUIRED** to ensure the safety of the Victim and all around him.

HOWEVER. Even when restraint is **REQUIRED**, only the "**least restrictive**" means may be employed.⁽⁶⁻⁸⁾ In other words: only the *minimum* amount of force or restraint necessary to ensure everyone's "SAFETY" (and to afford thorough medical evaluation and treatment of the Victim) may be used. And, the SAFETY of both Restrainers *and* Victim must be ensured during the **initiation, employment, and maintenance** of ANY form of restraint.

Any form of restraint that interferes with someone's **AIRWAY**, or that interferes with someone's **MECHANICAL means of breathing**, can KILL the restrained Victim.



Thankfully, "CHOKE HOLDS" (aka: "Sleeper Holds" ... "Tactical Neck Holds" ... "Lateral Vascular Neck Restraints" ... and the like) have *rightly* been BANNED from use by many emergency services.

CHOKE HOLDS can cause death in two different ways:

1. Death due to Airway Obstruction *OR*
2. Death due to "CAROTID ARTERY COMPRESSION."^(2,9)

No one argues about the fact that choking can cause death due to Airway Obstruction.

But, anyone who promotes the idea that even a "carefully" applied form of choke hold can "SAFELY" be employed (by avoiding obstruction of the Victim's AIRWAY) is obviously entirely uneducated about the OTHER, equally-lethal, choke hold effects caused by Carotid Artery Compression.⁽¹⁰⁾

Carotid Artery Compression can cause stroke or death due to obstruction of blood-flow to the brain – or by dislodging a plaque embolus (a clump of the "crud" that builds up along the lining of some people's arteries), and sending it to obstruct an artery in the brain).

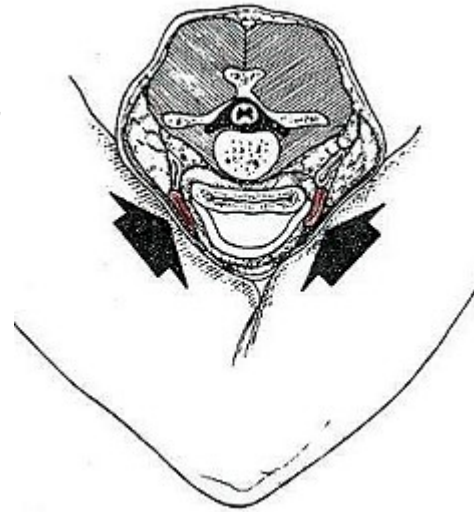
OR Carotid Artery Compression can cause death due to "CAROTID-SINUS-STIMULATION".

Stimulation from pressure placed on one or both carotid arteries triggers a lethally SLOW heart-rate – in fact, it can actually STOP the heart from beating.⁽²⁾

As you can see in the diagram at right:

A Restrainer thinking to avoid compressing the airway by using the "crook" of his arm to "cradle" the violently-struggling Victim's neck actually may *increase* the likelihood that CAROTID ARTERY OBSTRUCTION or STIMULATION will occur.

In other words: No matter how "carefully" any form of "CHOKE HOLD" is employed, any Victim subjected to a choke hold can rapidly DIE!



Because choke holds have rightly been banned from use for many years, **Restraint Asphyxia is most often caused by "Forceful-Prone-Restraint" application.**



Besides seriously interfering with thorough medical examination and care provision, forceful-prone-restraint application (with or *without* the simultaneous application of any kind of "hogtie" restraint) has been recognized as being a cause of restraint asphyxia deaths since 1988⁽¹¹⁾, and its use has long been **CONTRAINDICATED** by knowledgeable professionals.

Alternative methods of restraint are available to every Responder;

- SUPINE restraint methods easily employed and equally as immobilizing as use of forceful-prone-restraint ...
- SUPINE restraint methods that safely allow full access for the Victim's thorough medical examination and care provision ...
- SUPINE restraint methods that safely avoid *the threat of RESTRAINT ASPHYXIA DEATH*.

Furthermore, if access for thorough medical examination and care provision is not a "concern," SAFE methods of employing PRONE restraint ARE possible.

All that is required for ANY restraint method to be SAFE and EFFECTIVE is **EFFECTIVE (in-depth) EDUCATION**, practice, and team work.



In order for a Medical Examiner (or Forensic Pathologist) to pronounce someone dead *due to* **"POSITIONAL ASPHYXIA"** (whether *or not* the positional asphyxia death is restraint-related), ALL of the following THREE key elements **MUST BE SUBSTANTIATED** during an AUTOPSY INVESTIGATION:^(2,12-15)

- (1) ALL other causes of death – natural and unnatural, medical and traumatic – must be explored by autopsy and **EXCLUDED**, to within a reasonable degree of medical certainty. In other words; if the victim's autopsy identifies a strongly-probable natural or unnatural, medical or traumatic cause of death (apart from asphyxia), THAT pathological evidence should be considered indicative of the "cause" of death – not positional asphyxia.

In fact, it is only when there is no clearly apparent "cause of death" found at autopsy that a medical examiner is prompted (required) to more-closely examine the activities and events surrounding the actual TIME OF DEATH, in order to determine the factual CAUSE of death.^(2,16-18)

- (2) Upon investigation of the activities surrounding the time of death; **AT the Time Of Death** the victim must have been in a position readily-recognized as one that could have interfered with his ability to BREATHE.

BTW: The "**Time Of Death**" position that the medical examiner must investigate is the position the Victim was in at the moment when his breathing or pulse was **FIRST** noticed to have stopped – **NOT** the position the Victim was in at the time that all prehospital and/or in-hospital resuscitation efforts were determined to have failed, and he was "PRONOUNCED" dead.

"ASPHYXIATING" POSITIONS RANGE FROM

- a position that obstructs the mouth and nose ("upper airway"); or
- a position that causes hyperflexion (extreme bending-forward) of the neck, so as to obstruct the trachea (the largest "lower airway" passage); or
- a position that causes restriction of the chest or diaphragm – a position that impedes or prevents the MECHANICAL means of breathing;
- **OR** a combination of any of the above-described asphyxiating positions.

- (3) **LASTLY: The inability of the Victim to ESCAPE the asphyxiating position must be explained.**

Clinical asphyxia studies have shown that airway obstruction causes a variable period of physically-forceful muscular hyperactivity and reposition attempts, while the **CONSCIOUS** Victim struggles to alleviate or escape the position that is interfering with his breathing. If the victim is unable to regain the ability to breathe, he rapidly becomes UNCONSCIOUS.

Once UNCONSCIOUSNESS ensues, a portion of the Victim's brain **INVOLUNTARY** cues his body to continue performing muscular activity, in an unconscious effort to alleviate or escape the position that is interfering with breathing. Thus, the Victim's muscular "struggle" does not stop even after loss of consciousness occurs. In fact, struggle to alleviate or escape the position that is interfering with breathing **does not STOP until after the Victim's PULSE STOPS!**⁽¹⁹⁾

In positional asphyxia deaths *unrelated to RESTRAINT*, alcohol intoxication is the most frequent explanation of the Victim's inability to escape from the asphyxiating position.⁽¹⁴⁾

[Please Note: The aforementioned "alcohol intoxication" does *not* imply an alcohol "overdose" or "poisoning." If alcohol "overdose" or "poisoning" occurs, the Victim's respiratory center is deactivated. Thus, THAT would be the "cause of death" – **not positional asphyxia.**]

Only if **ALL** of the above **THREE ELEMENTS** are discovered during an accurate and effective autopsy investigation, can an individual's cause of death be identified as a form of "POSITIONAL ASPHYXIA" – whether due to restraint asphyxia, or not.

For Responders to develop an effective understanding of the deadly relationship between forceful-prone-restraint and restraint asphyxia, a basic understanding of **RESPIRATORY PHYSIOLOGY** (how NORMAL BREATHING occurs) is REQUIRED.

Effective Respiration is entirely dependent upon a combination of three critical elements⁽¹²⁾:

1. An open and "patent" airway (a passageway for air to travel from the atmosphere to the lungs, that is open and not threatened – in any way),
2. An adequate gas exchange between the lung's tissue (alveoli) and the body's smallest blood vessels (the capillaries), *and*
3. A functional respiratory muscular pump – or "bellows" system – the ability to change the interior size of the chest, so that airflow in and out of the lungs can be accomplished.

If any ONE of these elements is impaired or prevented from functioning, the act of breathing will be impaired or completely prevented.

If lung tissue or circulation is severely diseased or damaged by injury, oxygen and carbon dioxide are not exchanged adequately and breathing is ineffective.


Additionally, even with a completely open airway, perfectly healthy lungs and normal circulation, if the Victim's **mechanical component** of respiration fails (if his "bellows system" fails), effective respiration cannot be achieved.


An Effectively Functioning Muscular Pump or "Bellows System"

requires – at least – the FIRST and THIRD of the following three elements^(2,12):

Effective Bellows Function Requires:

- 1) A Brain-Respiratory-Center correctly "cuing" breathing to happen ...
- 2) Ribcage Expansion / Relaxation (which is **OPTIONAL!!!**)
- 3) A Functional DIAPHRAGM with FREEDOM to Move!





NORMAL Breathing Requires:

- 1) An OPEN AIRWAY ...
- 2) Reasonably healthy lungs & circulation for O₂ - CO₂ exchange
- 3) An EFFECTIVELY Functioning Mechanical "BELLOWS" System

1. An appropriately functioning central nervous system control of respiratory muscle activity: an unimpaired brain "respiratory center" sending appropriate "messages" to the respiratory muscles, causing them to act in a manner that effectively produces inspiration and expiration,

2. The ability of the ribcage to be expanded and relaxed by action of the intercostal muscles (muscles between the ribs), appropriately increasing and decreasing the size of the chest and interior lung-space.

[If the diaphragm is properly functioning, ribcage expansion is **not** required for "normal" breathing to effectively be accomplished.⁽¹²⁾]

AND

3. The ability of the diaphragm (the largest and most IMPORTANT respiratory muscle) to contract and displace abdominal contents downwards/outwards, increasing the size of the interior lung-space, so that inhalation can occur – and the ability of the diaphragm to relax and return to its normal position, decreasing the size of the interior lung-space, so that exhalation can occur.

NEXT: Normal breathing requires both effective inspiration and expiration; the movement of air in *and* out of the body. But, air will ONLY move from one place to another – from the atmosphere into the lungs, or from the lungs back out to the atmosphere – if the *air pressure* in one of those two places has been **CHANGED**.

If a person's airway was open, but his lung-size was in a fixed, unchanging position, the air-pressure **INSIDE** his lungs would be the **SAME** as the air-pressure **OUTSIDE** his body.


An **EQUAL** air-pressure inside and outside of the body results in

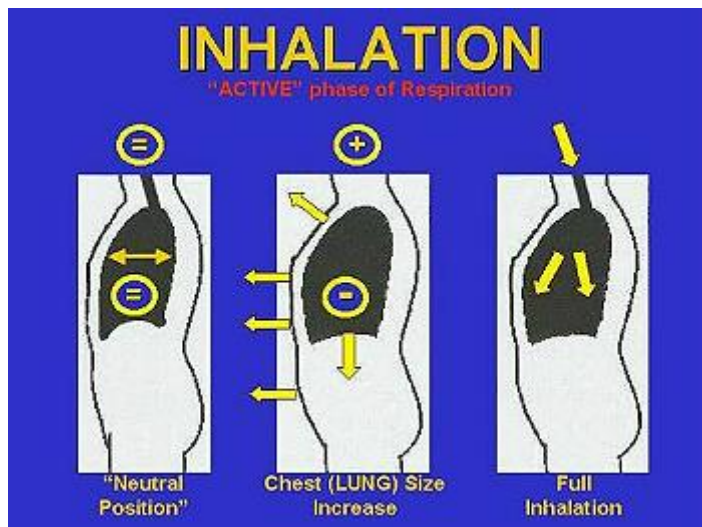
NO AIR MOVEMENT between these two spaces.

NO "breathing."

Air Movement REQUIRES Creation of "Air Pressure" DIFFERENCES!

If Atmospheric Pressure
EQUALS
Internal Lung Pressure,
NO AIR moves IN.
NO AIR moves OUT.





When respiratory muscles are appropriately activated by messages from the brain's respiratory center, the muscles between the ribs contract to make the ribcage expand, and the diaphragm contracts to descend into the abdomen (pushing the abdominal contents out of the way).

This **INCREASES** the **SIZE** of internal lung-space.

Ribcage expansion only slightly increases the size of the chest and internal lung-space. The greatest internal lung-space size increase occurs when the diaphragm contracts and travels down into the abdomen.⁽¹²⁾

When the internal lung-space size is increased, the air pressure inside the lungs becomes "stretched," **decreasing** the internal air pressure, and creating a "negative intrathoracic air pressure" (relative to atmospheric air pressure). In other words, increasing the space and size of the lungs creates a "**vacuum**" within the lungs.

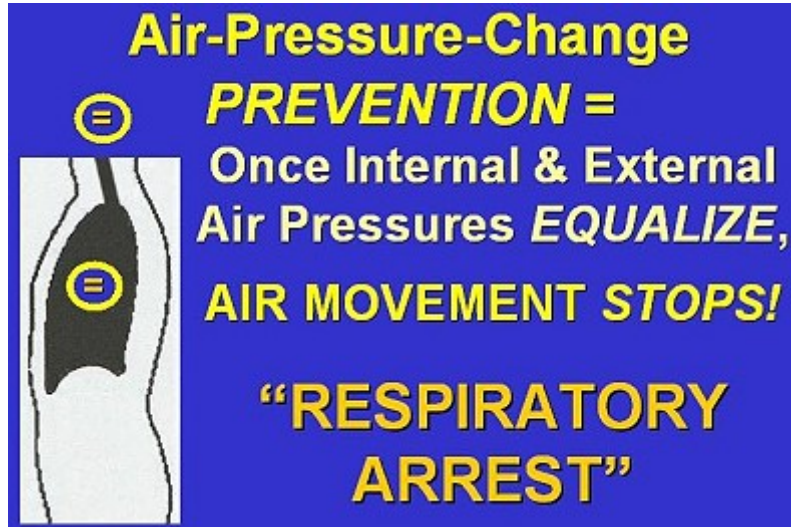
When the airway opens, air moves from the area of higher pressure (the atmosphere) to the area of lesser pressure (the lungs) = air rushes into the lungs, producing **INSPIRATION**. Air keeps moving into the lungs, continuing inspiration, until the air pressure inside the lungs becomes the **SAME** as the air pressure in the atmosphere. Once the internal and external air pressures **equalize**, all air movement stops, and all inspiration stops.

[Since "active" movement (**energy-expenditure**) is required to make the internal lung-space **LARGER**, inspiration is also called the "**Active Phase**" of respiration.]

When breathing becomes difficult (for whatever reason), "accessory" muscles are automatically employed to assist the intercostal muscles and diaphragm in creating the lung-space-size changes (the air pressure changes) required for inspiration and expiration to occur.^(11,19)

The muscles used to assist lung-space-size changes when breathing has become difficult include: the muscles between the ribs, neck and shoulder muscles, lateral abdominal muscles, and even the muscles of the arms and legs.

In fact, it is practically **impossible** for Restrainers to tell the **difference** between a Victim struggling to "escape restraint," and a Victim **desperately struggling to BREATHE!**



Once someone is entirely prevented from physically changing their internal lung-size and space, entirely prevented from changing the air pressure inside their lungs, breathing cannot occur.

When breathing cannot occur, that condition is called, **"RESPIRATORY ARREST."**

Thus ENDS the basic explanation of **RESPIRATORY PHYSIOLOGY** – how NORMAL BREATHING occurs!

Restraint asphyxia researchers have been warning Restrainers against the use of prone-hogtie and forceful-prone-restraint since the late 1980's.^(1,3,11,12,16,17,20) In recent years, however, restraint asphyxia researchers have come to the relatively-universal agreement that, restraint – ***by itself*** – is not solely responsible for asphyxia that occurs while a Victim is being actively restrained by others.^(1,4,5,12,17,18,21-25)

Restraint asphyxia only occurs when an asphyxiating form of restraint is ***preceded by*** and ***associated with*** lethally-exhausting and body-chemical-unbalancing activities.

But, the lethally-exhausting and body-chemical-unbalancing activities preceding restraint application – ***by themselves*** – do not cause death, ***either***.^(26,27,28)

THUS, ***in RESPONSE to*** these lethally-exhausting and body-chemical-unbalancing Victim-activities, IF the manner of restraint that is employed ***interferes with the Victim's ability to BREATHE***, the asphyxiating form of restraint employed **REMAINS the ultimate "cause" of the Victim's death.**

[Here begins PART 2 of the Restraint Asphyxia – Silent Killer article.]

To systematically describe and thoroughly explain how they place a Victim at risk for restraint asphyxia, I've separated these lethally-exhausting and body-chemical-unbalancing activities into **3 ACTIVITY "PHASES"** – phases that have consistently been associated with **EVERY** recognized restraint asphyxia death ever documented.



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PHASE 1 – The "Altered Level Of Consciousness" ONSET PHASE



Photo Credit = ?

PHASE 1 begins when an individual suddenly develops an **"altered level of consciousness"** that just happens to adversely effect the part of their brain that triggers them to ***involuntarily*** act-out in an unusually aggressive or violent, emotionally- and physically-exertive manner. Because it is caused by an altered level of consciousness (a state of "delirium") that produces violently "excited" or "agitated" behaviors, medical professionals call this state of extremely emotionally- and physically-exertive behavior, **"AGITATED DELIRIUM"** or **"EXCITED DELIRIUM."**

In simpler terms: some or all of the Victim's brain becomes "tweaked" by something. And, a PART of his brain that accidentally becomes seriously-tweaked begins **ORDERING** the Victim to act-out in an unusually aggressive or violent behavior.

Thus, the altered level of consciousness Victim is entirely incapable of volitionally "refusing" to respond to the ORDERS being issued by his tweaked-brain.

Anything that can cause someone to suffer an altered level of consciousness –

Anything that can "tweak" ANY part of someone's brain –

ALSO can "tweak" the part of the brain that triggers the ***INVOLUNTARILY*** performance of unusually aggressive or violent behaviors.

The mnemonic, **"AEIOU TIPS,"** serves to remind medical professionals of the many, many different emergencies or conditions that can cause someone to suffer an **Altered Level of Consciousness:**

A .. Acidosis (a body-chemistry imbalance) .. Alcohol intoxication

E .. Epilepsy (seizures ... "convulsions")

I .. Infection (especially when accompanied by fever)

O .. Overdose (of "medications," or "drugs," or alcohol)

U .. Uremia (a body-chemistry disorder)

T .. Trauma (to the head) ... Tumor (of the brain)

I .. "Insulin" (states of low or high blood sugar in diabetics ***or*** non-diabetics)

P .. Poisoning ... Psychosis (sudden onset)

S .. "Stroke" (TIA or CVA)

**Altered-Level-of-Consciousness Causes *NOT* Clearly Indicated
by the "AEIOU TIPS" Mnemonic:**

- Unusual and unexpected adverse reactions to non-overdose-levels of ***ANY*** medication or drug ("idiosyncratic reactions" to ***ANY*** medication or drug) ...
- ***ANY OTHER "body-chemistry imbalance" causes;*** such as "water-intoxication" ... deficiency ***or*** overdose of ***ANY*** "electrolyte" or "vitamin" (potassium, calcium, magnesium, and so on) ...
- Sleep deprivation ...
- Sudden onset of a "manic" phase in an individual with a "bipolar" brain disorder ...
- Abrupt withdrawal from a prescribed medication that requires "weaning" to safely discontinue
- ... ***Essentially, the list of these causes is almost ENDLESS!***

Obviously, it is vitally important for all Medical-Responders to understand the myriad of altered level of consciousness causes. But, why is it important for Non-Medical-Responders to understand them?

Because PERSONAL ATTITUDE *significantly influences* the amount of force and the manner of restraint employed by **ALL** Restrainers!

No matter where or for whom we work, we ALL are human beings. And, no matter how much "job experience" we have, we ALL *involuntarily* experience strong emotional responses, triggered by our personal interpretations of (our "gut reactions" to) EVERY "emergency" situation we face.

Thus, it is **vitally important** for ALL Restrainers to REMEMBER that:

States of "Agitated" or "Excited Delirium" are **NOT** experienced **ONLY** by "unpleasant" individuals who engage in drug or alcohol abuse!

In fact, almost ALL Restrainers are **related to** or **personally acquainted with** cherished individuals who have medical conditions (such as epilepsy or diabetes) that put them at greater risk than others for becoming an agitated delirium Victim!

And, ANY individual who – under "normal" circumstances – is a perfectly pleasant and respectable person, may suddenly become the **Victim** of an emergency that triggers an altered level of consciousness and induces a state of agitated delirium.

So, when we don't "know" the Victim, it is especially inappropriate for us to automatically assume that he is a drug or alcohol abuser, simply because he is acting-out in an aggressive or violent manner and "refusing" to follow our cues!

Because it is so vitally important to ALL Restrainers' attitudes that we remember the fact that violently-aggressive states of agitated delirium are *involuntarily* triggered by so many different *tweaked-brain* causes, I have decided to coin a **BRAND NEW ABBREVIATION** within this article's September 2004 update:

"Tweaked-Brain Involuntary Agitation" = "**T-BIA**"

This new abbreviation does NOT need to become universally-accepted or used elsewhere.

I am using it only so that every time YOU (my reader) see the abbreviation, "**T-BIA**" ("TEE-bee-ah") you will be REMINDED that

"Tweaked-Brain Involuntary Agitation" states are experienced by people who NORMALLY are perfectly wonderful, respectable, and pleasant individuals! AND, by the end of this article, I promise that you won't need any more reminding.

Time Frames Related to PHASE 1

Most causes of **T-BIA** – such as head injury, acute psychotic or manic episodes, strokes, intoxication, and the like – require 30 minutes to an hour (sometimes even longer) before the Victim's exertive behavior causes him to become *severely physically exhausted*, and **extremely susceptible to restraint asphyxia**.

However, two **T-BIA** causes produce SEVERE states of physical exhaustion QUITE QUICKLY.

Full-Body Seizure Activity – **all by itself** – represents significantly-extreme total-body physical exertion condensed into a very brief period of time (*merely three to five minutes!*), producing "prominent oxygen desaturations" (potentially lethal states of oxygen deprivation), and rapidly causing extreme physical exhaustion.^(29,30)

A full-body seizure is caused by an "electrical storm" in the brain that makes every muscle in the Victim's body rapidly and forcefully contract and relax, in a manner that is faster and more forceful than any consciously-controlled muscular activity. **EVERY** muscle in the Victim's body!

During a full-body seizure, even the body's "sphincters" (the little muscular rings responsible for things like keeping urine inside the bladder and feces inside the bowel) are rapidly and forcefully contracted and relaxed. Which means that, they aren't "working well" during the seizure. Thus, during full-body seizures, Victims almost always lose control of their bladder, and often lose control of their bowel.

Furthermore, while the diaphragm is "seizing" it is *not* traveling into and out of the abdomen in a manner that effectively changes the air pressure inside the Victim's lungs. Thus, the Victim isn't **breathing** in any meaningful manner during the seizure, and his brain rapidly becomes starved for oxygen.^(29,30)

Thankfully, once full-body seizures stop, MOST Victims are simply profoundly exhausted and confused. Most full-body seizure Victims simply require a few minutes to breathe and rest before their brain becomes able to function better.

BUT! If a seizure's "electrical storm" happens to *tweak* the part of the Victim's brain that triggers involuntary physically-exertive activity, and physically-exertive activity *continues AFTER* the seizure has stopped, the extreme physical exhaustion caused *during* the seizure becomes worsened, and the post-seizure **T-BIA** Victim rapidly becomes *lethally* exhausted.

Victims of a Sudden Onset of Hypoglycemia (**Low Blood Sugar**) also require very little time to suffer extreme physical exhaustion – whether or not their low blood sugar emergency is caused by a "diabetic" disorder. For instance: impressively healthy marathon runners who fail to adequately "carb-up" prior to a race will rapidly succumb to hypoglycemia, and fail to complete the race.

The brain is significantly sensitive to low blood sugar levels. In fact, the **very FIRST "SIGN" of low-blood-sugar** exhibited by a hypoglycemia Victim (whether or not he is a "diabetic") is an "altered level of consciousness," demonstrated by confusion or "abnormal" behavior.

Hypoglycemia also increases the brain's susceptibility to suffering damage due to "hypoxia" – a lack of adequate oxygen in the blood.⁽³¹⁻³⁶⁾ Thus, low blood sugar can also cause confusion or "abnormal" behavior due to the brain's inability to adequately obtain oxygen.

Additionally, because blood sugar is the vital form of "energy" required to fuel the body's mechanical (muscular) activity, a low blood sugar state – **all by itself** – rapidly produces **profound physical exhaustion**. After all, if adequate fuel is not available, but the muscles are forcefully-activated anyway, the muscles almost immediately become "exhausted."

Finally, the altered level of consciousness caused by a low-blood-sugar-brain-tweak often causes the Victim to act-out in a physically-exertive manner. Thus, just as fatiguing physical exercise can cause otherwise healthy and non-diabetic persons to suffer from low blood sugar^(37,38), hypoglycemic events that cause a Victim to act-out in a physically-exertive manner can rapidly cause lethally-extreme physical exhaustion.^(39,40)

PHASE 1 SUMMARY:

If a full-body seizure or low blood sugar is responsible for the Victim's **PHASE 1 "Altered Level Of Consciousness Onset,"** and a **T-BIA** response is triggered, the Victim may become *extremely physically exhausted* within as few as **3 to 5 minutes** after the ONSET of PHASE 1.

No matter WHAT kind of emergency condition *causes* the Victim's **PHASE 1 "Altered Level Of Consciousness Onset" T-BIA** response, the Victim is being *involuntarily*-cued to act-out in an unusually aggressive or violent, emotionally- and physically-exertive manner. The Victim is entirely unable to consciously control his behavior, and entirely unable to volitionally respond to verbal counsel or de-escalation techniques.

The **MOMENT** it starts, the **T-BIA** Victim's PHASE 1 emotionally- and physically-exertive behavior begins to physically exhaust ALL of the muscles in his body. His unusually aggressive or violent behavior presents a "threat" to the safety of himself or others. AND, the Victim requires some form of physical restraint.

PHASE 1's physically-exertive-activity continues until the individual's "out of control" **T-BIA** behavior is noticed by someone who either

- (A) begins some form of "intervention" (without training or authority); **OR**
- (B) notifies those legitimately responsible for "intervening" with the Victim's behavior, and the appropriate Responders arrive.



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PHASE 2 The INTERVENTION- INITIATION PHASE



Photo credit = ?

When Responders arrive they rapidly recognize that the Victim's altered level of consciousness makes verbal forms of "restraint" (the "least restrictive" forms of restraint) entirely ineffective. Thus, **PHASE 2** begins the moment Responders act to **physically INTERVENE** with the **T-BIA** Victim's behavior.

If initiated in an open space (out-of-doors), this phase often begins with a "chase." If Responders are law-enforcement, security, or correctional officers, the beginning of this phase is often accompanied by employment of things such as pepper spray, mace, Taser guns, or the like.

But, because the Victim is being involuntarily-cued to perform violent or aggressive exertional activity; no matter who the Responders are – no matter what initial setting or circumstances are involved – the Victim is **involuntarily compelled** to **RESIST** Restrainers' attempts to stop him from performing the violent or aggressive exertional activity.

Thus, this phase **ALWAYS** includes a period of "wrestling" (violent struggle) between Restrainers and the Victim.

During **PHASE 2**, the **T-BIA** Victim's excessively-exertive activity continues during his involuntary struggle with Restrainers. The physical exhaustion suffered by the Victim during **PHASE 1** is increased (worsened) by the physical exhaustion suffered during his **PHASE 2** struggle with Restrainers.

If a maximum, "full-body," form of restraint that **interferes with breathing** (such as forceful-prone-restraint) is the **first** form of restraint applied by Responders, restraint asphyxia death can occur during **PHASE 2**.

Time Frames Related to **PHASE 2**

Commonly, PHASE 2 is quite BRIEF. The moment Responders begin to employ "normally-effective," minimally-restrictive, techniques of hands-on restraint, they immediately discover that the **T-BIA** Victim is **INCREDIBLY STRONG!** In fact, Restrainers almost immediately realize that minimal-to-

moderate amounts of force, minimal-to-moderate amounts of restraint, are entirely unable to safely or effectively "contain" the individual's movements.

PLZ NOTE: **T-BIA** Victims are **rarely ever** "Arnold-Schwarzenegger"-types! Most often, they are "**White-Bread-Eating-Couch-Potatoes**." Essentially, what I mean by that terrifically flippant label is this:

T-BIA Victims usually are individuals with absolutely NO "weight-training" or "body-building" background.

Most often, **T-BIA** Victims are simply "regular people."

In fact, **T-BIA** Victims most frequently are overweight and "out of shape" people!

Contrary to the assumption commonly made by many medically-educated individuals; the incredible strength exhibited by ALL **T-BIA** Victims is NOT due to "adrenalin" stimulation.

Yes, **T-BIA** Victims are experiencing adrenalin ("fight/flight" system) stimulation. But, adrenalin stimulation automatically makes people "go faster" ... it does NOT automatically make people "stronger.")

So? WHY are **T-BIA** Victims so STRONG?

For some (still unclear) reason; when an altered-level-of-consciousness-cause happens to tweak the part of the brain that triggers a Victim to involuntarily act-out in a violent or agitated and exertive manner,

**it ALSO causes the Victim to NO LONGER
perceive PAIN STIMULUS in a "normal" manner!**

A person's ability to perform feats of "strength" is entirely based upon his ability to tolerate PAIN. When someone is bench-pressing a barbell loaded with steel weights, it is only when it "hurts" too much to "push" additional weight that the person reaches his bench-press weight limit. If he continues his weight-lifting regimen, his muscles become better and better developed, allowing him to push more and more weight before it starts to HURT too much, and he has to stop.

"PCP" (phencyclidine) is a drug used to tranquilize large animals (horses, elephants, and the like). It is an "anesthetic" with "analgesic" properties – in other words, PCP eliminates PAIN perception. When a human being takes PCP, the drug does NOT make him "stronger." It makes him unable to perceive PAIN. And, THAT is why PCP-abusers are so incredibly "strong!"

**If it doesn't ever HURT to exert strength, the ONLY limit to the amount of
STRENGTH that someone can exert is "STRUCTURAL" in nature.**

When a person doesn't perceive PAIN STIMULUS in a "normal" manner:

- "Pain-Control" techniques (such as "**Koga®**" maneuvers, "thumb locks" or "arm locks," and the like) do NOT bother the person.
- Pepper Spray or "Mace" products do NOT bother the person – no matter how many times he is sprayed.
- Even Taser guns (and the like) may have little to NO effect on the person.

AND, because the **T-BIA** Victim perceives **NO PAIN** – no matter how much he exerts his muscles – his ability to perform incredible feats of strength is limited only by things like bone-density and tendon-anchor-adherence-strength factors.

When forcefully-restrained by others, a **T-BIA** Victim (or PCP-abuser) actually may exert so much resistance-force and "strength" that he *fractures his own bones!*

He may exert so much resistance-force and "strength" that he **RIPS the tendons that anchor his muscles to his bones from their moorings!**

Clearly, such injuries will immediately cause the **T-BIA** Victim's injured limb to **WORK** less effectively (and more easily be restrained). However, since the Victim feels no pain, the parts of his body that remain uninjured continue to struggle with tremendous strength.

PHASE 2 SUMMARY:

PHASE 2 activity begins the moment Responders act to *physically INTERVENE* with the **T-BIA** Victim's behavior.

PHASE 2 is usually a **BRIEF** phase, because Restrainers rapidly realize that the **T-BIA** Victim is **INCREDIBLY STRONG**, and that minimal-to-moderate amounts of "normally-effective" restraint techniques are entirely unable to safely or effectively "contain" the individual's movements.

The physical exhaustion suffered by the Victim during PHASE 1 is increased (worsened) by the physical exhaustion suffered during PHASE 2 struggle with Restrainers.



Photo Credit = ?

PHASE 3 – The CONTINUED STRUGGLE PHASE:



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PHASE 3 begins when an increasing number of Restrainers begin applying an increasing amount of restraint (and restraint force) – rapidly progressing to a maximum, "full-body," form of restraint.

The physical exhaustion suffered by the **T-BIA** Victim during PHASES 1 and 2 continues to be increased (worsened) by the physical exhaustion that occurs during the involuntary struggle against maximum restraint in PHASE 3. But, by now, the **T-BIA** Victim is **ENTIRELY** physically exhausted. Thus, if the manner of maximum "full-body" restraint employed prevents the Victim from being able to adequately breathe, respiratory arrest rapidly occurs.



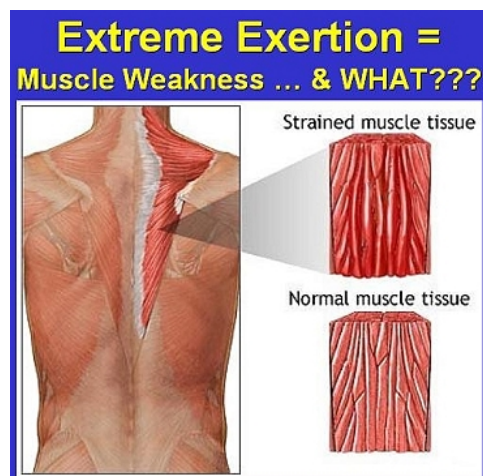
The "Pathophysiology" of The Exertional Phases of Activity Preceding & Associated with Restraint-Asphyxia

**(WHAT Makes The Effects Of These
PHASES So *LETHAL*?!)**

No matter what the original cause of the Victim's physically-exertive brain tweak, in addition to progressively exhausting all of the Victim's body muscles, each of the physically-exertive activity phases preceding and associated with restraint asphyxia cause **INTERNAL CHEMISTRY IMBALANCES** that adversely effect ALL of the individual's body functions.

Beginning in PHASE 1 and continuing throughout PHASES 2 & 3, the **T-BIA** Victim's **unusually-exertive** muscular activity causes excessive "lactic acid" production.⁽²⁶⁻²⁸⁾

Lactic acid production is a natural-occurring side effect of unusually-exertive muscular activity. The longer the unusually-exertive muscular activity continues, the more lactic acid is released into the Victim's system, and a lactic acid "overdose" can occur – something medical professionals call a "profound metabolic acidosis."⁽²⁶⁻²⁸⁾



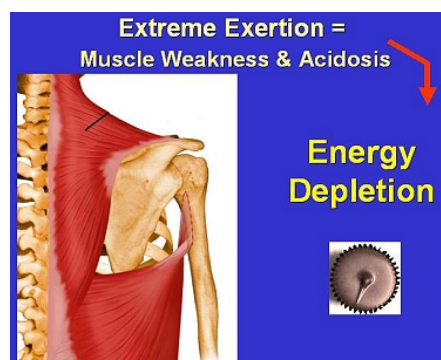
Metabolic acidosis is a serious and dangerous state of chemical imbalance. When suffering from a state of metabolic acidosis, the human body's natural (involuntary) response is to "correct" it by **hyperventilating**. "Hyperventilation" occurs when the depth and frequency of respirations are increased. Increasing the depth and frequency of respirations effectively "blows-off" the excess acid that is adversely affecting the body's systems. But, if the Victim's ability to **normally breathe** is impaired or prevented, hyperventilation clearly cannot occur, and the Victim is prevented from naturally-correcting the acidosis overdose he is suffering.⁽²⁶⁻²⁸⁾

Profound metabolic acidosis has consistently been noted in deaths "associated with cardiovascular collapse following exertion in a restrained position," and in other forms of asphyxial (suffocation) deaths.^(26-28,41)

However, profound metabolic acidosis is **not** a finding associated with victims of a "common" cardiopulmonary arrest – a suddenly-occurring "heart attack," caused by heart disease or heart circulation problems.^(26,42-48)

Thus, if the restraint asphyxia Victim is documented as being in a state of metabolic acidosis upon his emergency department arrival (especially in spite of rapidly-provided pre-hospital resuscitation measures), and no autopsy evidence clearly identifies another cause of his death; anyone who opines that his death was due to "cardiopulmonary arrest" does so without ANY form of scientific or forensic supportive evidence.

In fact, anyone who opines that the death of ANY victim with such a profound state of metabolic acidosis measured upon his arrival at the emergency department was solely due to "cardiopulmonary arrest," does so **in spite of** scientific and forensic evidence that the victim's death was much more likely due to some form of **asphyxia**.



Another side-effect of unusually-exertive muscular activity is "energy" depletion – **depletion of the body's sugar stores**; something that can lead to dangerous states of low blood sugar. Remember, muscles require oxygen and sugar to fuel their function. The more-forcefully muscles are required to work, the more sugar they use up to do so.^(37,38)

As more and more sugar is used up by unusually-exertive muscular activity, less and less sugar is available to the muscles, causing all the body's muscles to become less-well fueled, more and more fatigued.^(39,40)

The more sugar that is used up to fuel unusually-exertive **extremity** muscular activity, the less sugar is available to the Victim's **BRAIN** – which means that his brain-tweak gets worse, and brain damage can occur!⁽³¹⁻³⁶⁾

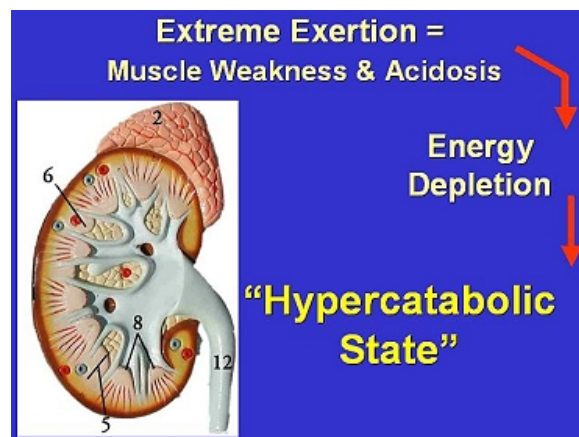
States of extreme emotional- and physical-exertion also generate excessive production of several other naturally-produced body chemicals; especially the chemicals released by the "Fight/Flight" nervous system, such as adrenalin and noradrenalin. Medical professionals call these naturally-produced "Fight/Flight" chemicals, "**catecholamines**."

During all of the emotionally- and physically-exertive activities that precede and are associated with restraint asphyxia deaths, progressively-increasing amounts of catecholamines are released into the Victim's system.

This creates what medical professionals call a, "**hypercatabolic state**" – an "overdose" of these naturally-produced chemicals. A hypercatabolic state is a chemical imbalance that weakens ALL of the body's muscles. But, it **especially weakens the respiratory muscles**.^(1,12)

Unless we have a respiratory disease, human beings never even *notice* the "effort" required to breathe. However, we certainly notice occasional extremity muscle "effort"-exertion (such as when having to walk up several flights of stairs). This means that our extremity muscles are accustomed to occasionally being called upon to perform extra exertion. But, our respiratory muscles are NOT accustomed to occasionally having to work harder!

So, because our respiratory muscles are entirely unaccustomed to exerting excessive effort to accomplish breathing, our respiratory muscles do not boast a significant amount of "muscle tone" or "strength." Thus, when faced with extreme-exertion requirements and body chemical imbalances, our respiratory muscles begin to "fail" long before our extremity muscles do.



A hypercatabolic state also adversely effects the HEART.^(1,12)

Catecholamines cause the heart to contract (beat) faster, and with greater FORCE of contraction – exerting greater "effort" to work. Since the heart is a muscle that (like the respiratory muscles) is entirely unaccustomed to having to work terrifically hard for a prolonged period of time, the heart rapidly becomes exhausted when required to work harder than it is used to working.

Furthermore; to work FASTER and more FORCEFULLY, the heart muscle requires more SUGAR and OXYGEN to fuel an increased functional-performance demand.

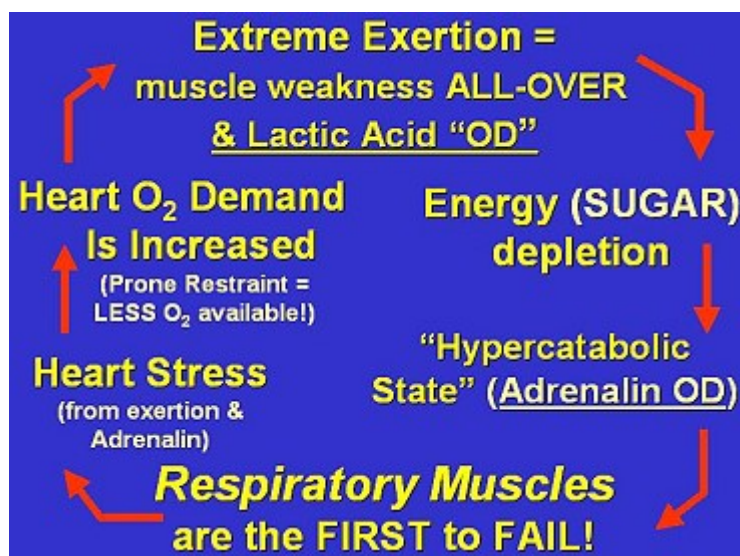
But, when struggling against Restrainers, the Victim's extremity muscles are using up the vast majority of the body's sugar stores. So, less and less sugar is available to fuel the heart's function.

AND, when struggling against Restrainers, the Victim's heart requires *greater than "normal"* amounts of oxygen to support the increased workload that is demanded of it. BUT, if the Victim is struggling against Restrainers who are employing a form of restraint that MAKES BREATHING DIFFICULT (such as forceful-prone-restraint), his heart receives *far less* than normal amounts of oxygen.^(1,17,26-28,40)

"Cause of Death" CONSIDERATIONS

Clearly: As the physically-exhausting and body-chemical-unbalancing effects of the Victim-activities preceding and associated with restraint application *continue*, the Victim's physical condition becomes increasingly worsened.

However (again), the physically-exhausting and body-chemical-unbalancing effects caused by Victim-activities preceding and associated with restraint application – by themselves – do NOT cause death.^(26,27,28)



As long as the **T-BIA** Victim is NOT restrained in a manner that interferes with his ability to breathe, all of these struggle-related physically-exhausting and body-chemical-unbalancing adverse effects are entirely SURVIVABLE.⁽²⁶⁻²⁸⁾

The prolonged muscle-exertion lactic acidosis overdose can naturally be "hyperventilated" away ... once the cause of his delirium is corrected, the Victim's catecholamine overdose will naturally begin to dissipate (being filtered and excreted by the his liver and kidneys) ... and so on.

Furthermore, physical exhaustion ONLY EVER results in "respiratory arrest" when the Victim's respiratory muscle function (particularly his diaphragm function) is interfered with.

This is why restraint asphyxia researchers' agreement with the concept that "restraint" – by itself – is probably not *solely* responsible for the vast majority of restraint asphyxia deaths" is a "**QUALIFIED**" agreement!

Two published scientific studies, performed under **controlled clinical situations**, have yielded information indicating that *minimal-to-moderate forms* of "full-body" prone restraint (**even with a tiny bit of "weight" applied to the Victim's shoulders**) do NOT produce *obviously-lethal* respiratory-interference effects in **entirely-healthy study subjects**.^(21,49)

However, both of these studies' findings have little-to-NOTHING to do with "real life" restraint situations!^(22,23)

Both of these clinically-controlled studies utilized **entirely-healthy study subjects** who were subjected to only *minimal-to-moderate forms* of "full-body" prone restraint. **NONE of either study's subjects were suffering from the physically-exhausting and body-chemical-unbalancing effects of the Victim-activities that consistently precede or are associated restraint asphyxia deaths.**

FURTHERMORE, not a *single* scientific experiment or case study has ever been performed or published (or anecdotally distributed), identifying even ONE incident wherein the adverse physical and chemical effects of delirium-triggered physically-exertive activities have EVER – by themselves – resulted in someone's DEATH.

In fact, in ALL documented accounts of deaths associated with individuals suffering a state of "excited delirium": if the Victims didn't die due to **physical trauma** (caused by jumping off of a building, or the like) or chemical overdose (drug or alcohol "overdose") – causes of death clearly evidenced at autopsy – the Victims ultimately died ONLY because they were subjected to a **body position or form of restraint that interfered with (or entirely prevented) their ability to BREATHE!**

THE BOTTOM LINE: States of "Excited" or "Agitated Delirium" have NEVER, ever, been demonstrated to – by themselves – result in someone's death. Thus, anyone who opines that "Excited" or

"Agitated Delirium" was the "cause" of someone's death, does so without ANY form of scientific or forensic support.



**When
RESTRAINT
Is
ADDED
TO
THE
"MIX"**



When a **T-BIA** Victim has suffered the adverse physical and chemical effects of one or more physically-exertive activity PHASES, and the manner of restraint employed to control him interferes – even minimally – with his ability to breathe, the Victim's **need for oxygen** progressively increases, but less and less oxygen is available.^(1,17,26,41,50)

Individuals with "a large abdominal panniculus" (a medically-polite phrase meaning people who are overweight and have "a big belly") are at extreme risk for rapid onset of restraint asphyxia from prone positioning.^(23,25,51)

"A large, bulbous abdomen (a beer belly) presents significant risks because it forces the contents of the abdomen upward within the abdominal cavity when the body is in a prone position. This puts pressure on the diaphragm, a critical muscle responsible for respiration, and restricts its movement. If the diaphragm cannot move properly, the person cannot breathe."⁽⁵¹⁾

In other words: Simply putting someone with a big belly in a prone position immediately "sandwiches" his abdominal contents between the ground and his spine. The bones of his pelvis prevent abdominal contents from shifting away from his chest. So, the only place for them to go is toward his chest. This pushes on the diaphragm and immediately interferes with his ability to change the internal-air-pressure of his lungs.

Putting someone with a big belly in a prone position causes **SOME** breathing interference, even **without** any **force** being applied to his posterior torso! If his shoulders and hips are held in place, breathing immediately becomes difficult.

And, remember; when faced with extreme-exertion requirements and body chemical imbalances, respiratory muscles begin to "fail" long before extremity muscles do. Once the diaphragm fails, the Victim's bellows system fails.

The moment a restrained Victim's respiratory bellows system is prevented from changing the internal air pressure within his lungs, the Victim can no longer breathe. Thus, the Victim suffers "**RESPIRATORY ARREST**" – even though **HE IS STILL "CONSCIOUS" (AWAKE)**.

In spite of his "altered" level of consciousness, while the respiratory arrest Victim is still awake, he will employ every muscle he possibly can in order to escape the asphyxiating position and regain the ability to breathe. In fact, when a manner of restraint has started to interfere with or entirely prevent breathing, there is absolutely **NO PERCEIVABLE DIFFERENCE** between:

- the muscular activity of someone consciously struggling to "escape restraint," and
- the muscular activity of someone consciously struggling to "regain the ability to breathe."

A conscious respiratory arrest Victim will employ his shoulders and arms, his legs and hips, his "accessory" chest and abdominal muscles, all in an effort to give his exhausted diaphragm room to

contract and move into his abdomen, so as to change the internal-lung air pressure and allow him to breathe again.

If diaphragmatic freedom to move (the ability to breathe) is not regained, a conscious respiratory arrest Victim rapidly becomes **unconscious**.⁽¹⁹⁾ When one or more physically-exertive activity PHASES has preceded application of full-body-restraint that interferes with breathing,

**RESPIRATORY ARREST and UNCONSCIOUSNESS has occurred
within less than 3 minutes**

after an asphyxiating-form of restraint was applied

– even when the Victim was a slender, entirely "healthy" young adult, who was not under the influence of any respiratory-center- or cardiovascular-system-altering drugs.⁽²⁸⁾

Once unconscious, the respiratory arrest Victim's brain will instinctively and *involuntarily* cue his "accessory" muscles to **continue to "struggle,"** in an effort to regain the ability to breathe.⁽¹⁹⁾

Accessory muscles most commonly include those of the neck, shoulders, and intercostal muscles (the muscles between the ribs). But, when being "trapped" or "restrained" in an asphyxiating position, arm and leg muscles may also be instinctively and involuntarily cued to move. Thus, several of the Victim's body muscles will continue to "move" after loss of consciousness – albeit in a less focused and forceful manner than that of consciously-controlled muscle movement.

Uneducated restrainers erroneously interpret this continued muscular activity as an indication that the individual is **"still breathing"** and/or still **"fighting"** with them. Consequently, restraint is continued.

In fact, restraint often is maintained until well after the Victim stops moving. When all movement stops, uneducated restrainers often assume that the restrained individual is "playing possum." Thus, they continue restraint application until they finally notice that the individual is no longer breathing ... is turning "blue" ... and/or has no pulse.

Unfortunately, the involuntary muscle movement of an unconscious respiratory arrest Victim does not stop until **after the Victim's heart stops effectively circulating blood**.⁽¹⁹⁾

Thus, body movement doesn't stop until after the Victim is in FULL cardiopulmonary arrest.

The pathophysiology of death caused by purposefully-induced-asphyxia ("What happens to a body after it cannot breathe anymore?") has been clinically-studied in both dogs and rats.^(19,41) "[Induced] Asphyxia led to cardiac arrest within approximately 4 minutes in all rats."⁽⁴¹⁾ Clearly, a human being might take that long, or longer, or less long to die after asphyxia occurs. But, the amount of time it takes for **a human being's** heart to stop after respiratory arrest occurs cannot be clinically explored by experiment! So, we must rely upon evaluation of **case studies** to determine a reasonable estimate of how long it takes asphyxia to cause a human's heart to stop beating.

I assessed the times documented in several published restraint asphyxia case studies, to determine the average time interval between first application of forceful-prone-restraint and when Restrainers finally realized that the Victim was not breathing **and did not have a pulse**.^(1,11,18, 20,52)

**The average time between first application of forceful-prone-restraint
and when full cardiopulmonary arrest was noticed
is only 5.6 minutes!**

Remember, however! Respiratory arrest and unconsciousness occurred long before that amount of time elapsed.^(19,28,41) And, the actual onset of cardiopulmonary arrest probably occurred **before** Restrainers finally discontinued restraint and recognized it.

Once a restraint asphyxia Victim is in FULL cardiopulmonary arrest
 – once his heart has finally stopped –
 successful resuscitation is virtually unheard of.^(20,25,26,41)

Even when the Victim is in the care of ACLS-providers who immediately notice the cardiopulmonary arrest, and immediately begin basic and advanced resuscitation measures, the Victim is not resuscitated.⁽²⁰⁾ The currently-popular scientific theories offered to explain this failure-to-resuscitate phenomenon revolve solely around the fact that:

the therapeutic effects of almost ALL medications administered during standard Advanced Cardiac Life Support (ACLS) cardiopulmonary resuscitation protocol-performances are *interfered with* ("inactivated")
 if the Victim's internal body-system is chemically-imbalanced – especially if
 the Victim has an excessively **ACIDOTIC** internal body-system.^(26,41,53-56)

At the time respiratory arrest occurs, the restraint asphyxia Victim already has a high systemic level of struggle-related lactic acidosis. Furthermore, both the conscious and unconscious muscle-exertion that is continued by the restraint asphyxia Victim, *after* respiratory arrest occurs, produces additional lactic acid.

BUT, when **respiratory arrest** occurs and exchange of oxygen and carbon dioxide **stops**, ANOTHER cause of acid-production begins – an **inadequate-ventilation** form of acid production, that is called, "**RESPIRATORY**" acidosis.

Thus, the degree of acidosis measured in restraint asphyxia Victims is *significantly greater* than the degree of acidosis measured in Victims of a "common" cardiopulmonary arrest (a suddenly-occurring "heart attack" caused by heart or heart circulation problems).^(26,41-47,57,58)

If you wish to read a brief discussion of "common" cardiopulmonary arrest acidosis levels as they compare to restraint asphyxia acidosis levels, go to:
<http://www.charlydmiller.com/RA/restrasphyxacid.html>

Current research suggests that rapidly-administered "aggressive ventilation and bicarbonate therapy" offers restraint asphyxia Victims the best possible chance of survival.^(26,41) Deep and rapid artificial ventilation ("aggressive" hyperventilation) has long been proven to reverse systemic acidosis caused by respiratory-failure.^(42-48,57,58) But, because restraint asphyxia Victims also suffer from such unusually-extreme muscle-exertion lactic-acid-production (prior to suffering from "common" respiratory-failure acid-production), they require more than merely "aggressive" hyperventilation to reverse their systemic accumulation of acid.

Since Sodium Bicarbonate ("Bicarb") is considered the chemical "ANTIDOTE" to excessive levels of acidosis (whether from muscle-released lactic acidosis, or from respiratory-failure acidosis), researchers have hypothesized that rapid administration of Bicarb to restraint asphyxia Victims should correct their acidosis. AND, researchers have hypothesized that; once the asphyxia-victim's acidosis is corrected, standard ACLS medications should then work as effectively as they normally do for "common" cardiopulmonary arrest Victims' resuscitation.

Unfortunately, the clinical studies exploring these hypotheses, as well as case studies of restraint asphyxia victims who received Bicarb, have failed to demonstrate that rapid Bicarb administration is successful for effectively resuscitating extremely acidotic asphyxial cardiopulmonary arrest Victims.^(26,27,41)

Thus, once a restraint asphyxia Victim's heart has finally stopped, successful resuscitation remains virtually unheard of.

Part Three of this article provides directions for accomplishing SUPINE total-body-restraint in a manner that is every bit as immobilizing as that of forceful-prone- or prone-hobble-restraint.

However, this supine total-body-restraint technique will **NOT** threaten the patient's life with restraint asphyxia!



Photo © 1994 Michal Heron

Additionally (perhaps even MORE importantly), supine total-body-restraint affords care providers **full access** to the patient for accomplishing a complete and thorough examination, affording **unimpeded performance** of any and all emergency care procedure required.

PLZ NOTE: I have **NOT** updated **Part Three** yet!

Thus, this PDF file does not include PART THREE.

The current version of Part Three is only “important” to individuals using MECHANICAL FORMS of restraint.

PLANS FOR THE *EVENTUAL* Part Three Update:

Discussion of using a long back board for all patients requiring restraint and medical care.

Discussion of A Few PHYSICAL RESTRAINT Techniques Used by NON-Medical Care Providers:

Those Who Care for Developmentally Disabled and Mentally Ill individuals.

AND, communication techniques for STOPPING OTHERS from performing forceful-prone-restraint!

To Read the currently-posted PART THREE of this article, go to:

<http://www.charlydmiller.com/RA/restrasphyx03.html>

About the Author: Charly is an internationally-known emergency care author, EMS Instructor, Consultant, and Restraint Asphyxia Expert Witness.

A paramedic since 1985 (nine years as a "Denver General" Paramedic), Charly is a seasoned prehospital emergency care provider. With his additional experience as a psychiatric medical technician and an Army National Guard helicopter medic, Charly is one of the country's most exciting and entertaining EMS educators.

For further information, go to:

Charly Miller's Restraint Asphyxia Newz Directory :

<http://www.charlydmiller.com/ranewz.html>

Charly Miller's Restraint Asphyxia Library:

<http://www.charlydmiller.com/RA/RAlibrary.html>

RESTRAINT ASPHYXIA – SILENT KILLER REFERENCES

For ALL THREE Parts

Updated (finally!) in SEPTEMBER, 2004.

Reference articles followed by a bold red asterisk (*)

are available in Charly's RESTRAINT ASPHYXIA LIBRARY:

<http://www.charlydmiller.com/RA/RAlibrary.html>

1. O'Halloran RL, Lewman LV; Restraint asphyxiation in excited delirium. *Am J Forensic Med Pathol*, 1993;14(4):289-295.*
2. Reay DT; Death in custody. *Clin Lab Med*, 1998 Mar;18(1):1-22.*
3. Hirsch CS: Letter to the editor RE: Restraint asphyxiation in excited delirium & the article authors' reply *Am J Forensic Med Pathol*, 1994;15:266.*
4. O'Halloran RL, Frank JG: Asphyxial death during prone restraint revisited: A report of 21 cases. *Am J Forensic Med Pathol*, 2000;21(1):39-52.*
5. O'Halloran RL, Frank JG: Authors' Reply to a Letter to the Editor regarding; Asphyxial death during prone restraint revisited: a report of 21 cases. *Am J Forensic Med Pathol*, 2000;21(4):420-422.*
6. Lavoie FW: Consent, Involuntary Treatment, and the Use of Force in an Urban Emergency Department. *Ann Emerg Med* January 1992;21:25-32.
7. Rice MM; Moore GP: Management of the Violent Patient, Therapeutic and Legal Considerations. *Emerg Med Clin North Am* February 1991;9(1):13-30.
8. Rund DA; Keller MD: To Restrain or Not to Restrain. *Emerg Med Serv* 1986 Jan/Feb;15:24,46-9.
9. Reay DT, Eisele JW: Law enforcement neck holds. *Am J Forensic Med Pathol* 1986 Jun;7(2):177.*
10. Dick T: Straight shot: use of restraints, part 2. *JEMS*, Jan 2003, 28(1) p98, 100-1.*
11. Reay DT, Howard JD, Fligner CL, Ward RJ; Effects of positional restraint on oxygen saturation and heart rate following exercise. *Am J Forensic Med Pathol*, 1988;9(1):16-18.*
12. Reay DT, Fligner CL, Stilwell AD, Arnold J: Positional asphyxia during law enforcement transport. *Am J Forensic Med Pathol*, 1992;13(2):90-97.*
13. **FORENSIC PATHOLOGY, Second Edition**, © 2001 by CRC Press LLC; DiMaio VJ, DiMaio D. Chapter 8: Asphyxia.*
14. Bell MD, Rao VJ, Wetli CV, Rodriguez RN. Positional asphyxia in adults: A series of 30 cases from the Dade and Broward County Florida medical examiner offices from 1982 to 1990. *Am J Forensic Med Pathol*, 1992;13(2):101-107, 1992.*
15. **FORENSIC PATHOLOGY, Second Edition**, © 2001 by CRC Press LLC; DiMaio VJ, DiMaio D. Chapter 22: Sudden Death During or Immediately after a Violent Struggle.*
16. Luke JL, Reay DT; The perils of investigating and certifying deaths in police custody. *Am J Forensic Med Pathol*, 13(2):98-100, 1992.*
17. Ross DL; Factors associated with excited delirium deaths in police custody. *Mod Pathol*, Nov 1998, 11(11) p1127-37.*
18. Pollanen MS, Chiasson DA, Cairns JT, et al.; Unexpected death related to restraint for excited delirium: a retrospective study of deaths in police custody and in the community. *CMAJ*, Jun 16 1998, 158(12) p1603-7.*

19. Swann HG and Brucer M. The cardiorespiratory and biochemical events during rapid anoxic death - V. Obstructive asphyxia. *The Texas Reports on Biology and Medicine*, 1949;Vol. 7; pgs 593-603.*
20. Stratton SJ, Rogers C, Green K; Sudden death in individuals in hobble restraints during paramedic transport. *Ann Emerg Med*, May 1995;25:710-712.*
21. Chan TC, Vilke GM, Neuman T, Clausen JL: Restraint position and positional asphyxia. *Ann Emerg Med*, November 1997;30:578-586.*
22. Howard JD, Reay DT. Letter To The Editor. *Ann Emerg Med*, July 1998; 32(1):116-117.*
23. Reay DT: Hog-tied revisited. C.D. Miller's Restraint Asphyxia Library; <http://www.charlydmiller.com/LIB/reaysdiego.html>; July 1st, 1998.*
24. Chan TC, Vilke GM, Neuman T; Reexamination of custody restraint position and positional asphyxia. *Am J Forensic Med Pathol*, 1998;19(3):201-205.*
25. Stratton SJ, Rogers C, Bricket K, Gruzinski G: Factors associated with sudden death of individuals requiring restraint for excited delirium. *Am J Emerg Med*, May, 2001;19:187-191.*
26. Hick JL, Smith SW, Lynch MR: Metabolic acidosis in restraint-associated cardiac arrest: a case series. *Academic Emerg Med*, March 1999, Volume 6, Number 3.*
27. Park KS, Korn CS, Henderson SO. Agitated delirium and sudden death: two case reports. *Prehosp Emerg Care*. 2001;5:214-216.*
28. Miller CD. Restraint-related positional asphyxia "near-death" – a case study. <http://www.charlydmiller.com/RA/neardeath01.html> August 30th, 1998.
29. Blum AS, Ives JR, Goldberger AL, Al-Aweel IC, Krishnamurthy KB, Drislane FW, Schomer DL. Oxygen desaturations triggered by partial seizures: implications for cardiopulmonary instability in epilepsy; Abstract. *Epilepsia*, 2000 May;41(5):536-41.
30. Langan Y, Nashef L, Sander JWAS. Sudden unexpected death in epilepsy: a series of witnessed deaths. *J Neurol Neurosurg Psychiatry*, 2000 Feb;68(2):211-3.
31. Tekkok SB, Godfraind JM, Krnjevic K. Moderate hypoglycemia aggravates effects of hypoxia in hippocampal slices from diabetic rats. *Neuroscience* (United States), 2002, 113(1) p11-21.
32. Ichord RN, Johnston MV, Traystman RJ. MK801 decreases glutamate release and oxidative metabolism during hypoglycemic coma in piglets. *Brain Res Dev Brain Res* (Netherlands), Jun 29 2001, 128(2) p139-48.
33. Derwen JJ, Linsenmeier RA. Hypoglycemia increases the sensitivity of the cat electroretinogram to hypoxemia. *Vis Neurosci* (England), Nov-Dec 2001, 18(6) p983-93.
34. Ichord RN, Northington FJ, van Wylen D, et al. Brain O2 consumption and glutamate release during hypoglycemic coma in piglets. *Am J Physiol* (United States), Jun 1999, 276(6 Pt 2) pH2053-62.
35. Strachan MW, Deary IJ, Ewing FM, et al. Acute hypoglycemia impairs the functioning of the central but not peripheral nervous system. *Physiol Behav* (United States), Jan 2001, 72(1-2) p83-92.
36. Rabinovici GD, Lukatch HS, MacIver MB. Hypoglycemic and hypoxic modulation of cortical micro-EEG activity in rat brain slices. *Clin Neurophysiol* (Netherlands), Jan 2000, 111(1) p112-21.
37. Brun JF, Dumortier M, Fedou C, et al. Exercise hypoglycemia in nondiabetic subjects. *Diabetes Metab* (France), Apr 2001, 27(2 Pt 1) p92-106.
38. Guagnano MT, Pace-Palitti V, Formisano S, et al. Does holiday hypoglycaemia exist? *Panminerva Med* (Italy), Mar 2000, 42(1) p23-6.
39. King P, Kong MF, Parkin H, et al. Well-being, cerebral function, and physical fatigue after nocturnal hypoglycemia in IDDM. *Diabetes Care* (United States), Mar 1998, 21(3) p341-5.
40. Dorchy H. Sports and type I diabetes: personal experience. *Rev Med Brux* (Belgium), Sep 2002, 23(4) pA211-7.
41. Katz LM, Wang Y, Rockoff S, et al. Low-dose Carbicarb improves cerebral outcome after asphyxial cardiac arrest in rats. *Ann Emerg Med*, Apr 2002, 39(4) p359-365.*

42. American Heart Association; Standards and guidelines for cardiopulmonary resuscitation and emergency cardiac care. *JAMA*, 1986;255:2843-2989.
43. **Textbook of Advanced Cardiac Life Support, Second Edition** © 1987 by The American Heart Association.
44. Dybvik T, Strand T, Steen PA. Buffer therapy during out-of-hospital cardiopulmonary resuscitation. *Resuscitation* (Ireland), Apr 1995, 29(2) p89-95.
45. Fillmore SJ, Shapiro M, Killip T. Serial blood gas studies during cardiopulmonary resuscitation. *Ann Intern Med*, 1970; 72:465-9.
46. Chazan JA, Stenson R, Kurland GS. The acidosis of cardiac arrest. *N Engl J Med*, 1968; 278:360-4.
47. Gazmuri RJ, Weil MH, von Planta M. Cardiopulmonary resuscitation: acid-base alterations and alkalizing therapy. *Rev Med Chil* (Chile), Mar 1989,117(3)p322-9.
48. Von Planta M, Bar-Joseph G, Wiklund L, et al. Pathophysiologic and therapeutic implications of acid-base changes during CPR. *Ann Emerg Med*, Feb 1993, 22(2 Pt 2) p404-10.
49. Chan TC; Neuman T; Clausen J; Eisele J; Vilke GM. Weight force during prone restraint and respiratory function. *Am J Med Pathol*, 25(3):185-189, September 2004.*
50. Roeggla M, Wagner A, Muellner M, Bur A, Roeggla H, Hirschl MM, Laggner AN, Roeggla G. Cardiorespiratory Consequences To Hobble Restraint. *Wien Klin Wochenschr*, 1997 May 23;109(10):359-61.*
51. Reay DT: Suspect restraint and sudden death. *FBI Law Enforcement Bulletin*, May, 1996.*
52. Ambulance transport death results in questioning of techniques. *EMS Professionals*, July-August 1997;6-8.*
53. **Prehospital Emergency Pharmacology**. Bledsoe BE, Clayden DE, Papa FJ: © 2001 by Prentice-Hall, Inc.; p.134.
54. Huang YG, Wong KC, Yip WH, et al. Cardiovascular responses to graded doses of three catecholamines during lactic and hydrochloric acidosis in dogs. *Br J Anaesth* (England), May 1995, 74(5) p583-90.
55. Modest VE, Butterworth JF. Effect of pH and lidocaine on beta-adrenergic receptor binding. Interaction during resuscitation? *Chest* (United States), Nov 1995, 108(5) p1373-9.
56. American Heart Association; Standards and guidelines for cardiopulmonary resuscitation and emergency cardiac care. *JAMA*, 1974;227:833-868.
57. **1997-99 Emergency Cardiovascular Care Programs, Advanced Cardiac Life Support** © 1997 by The American Heart Association.
58. **2000 Handbook of Emergency Cardiovascular Care for Healthcare Providers** © 2000 by The American Heart Association.