
RESTRAINT ASPHYXIA DEATHS vs. "COMMON CARDIOPULMONARY ARREST" DEATHS ACIDOSIS LEVELS DISCUSSION

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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**.

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).(26,41-48,57,58)

During "common" cardiopulmonary arrests, inadequate ventilation and blood circulation causes retention of carbon dioxide, producing a small degree of "respiratory" acidosis. If any involuntary

muscle movement occurs after the heart stops ("twitching), this activity *may* also generate a tiny production of lactic acid – a tiny amount of "metabolic" acidosis.(26,42-47,57,58)

Early Advanced Cardiac Life Support (ACLS) researchers erroneously assumed that ALL cardiopulmonary arrest victims would rapidly suffer from **significantly-high** levels of acidosis – acidosis levels that would be detrimental to successful resuscitation. Thus, the earliest ACLS protocols dictated an *automatic* initial administration of Sodium Bicarbonate (Bicarb), intended to chemically reverse this erroneously-presumed state of serious acidosis.

In fact, the 1974 American Heart Association (AHA) national standard protocols for ACLS-provision mandated the **automatic initial intravenous (IV) administration of**

- **ONE** 50mEq ampule of Bicarb, if the patient had been in cardiac arrest for "**less than 10 minutes**" prior to ACLS-providers' arrival; OR,
- **TWO** 50mEq ampules of Bicarb, if the patient had been in cardiac arrest for "**greater than 10 minutes**" prior to ACLS-providers' arrival.(56)

By **1986**, however, several scientific studies had clearly demonstrated that rapid provision of **effective ventilation** and artificial circulation were entirely adequate means of managing the **small** amount of respiratory- (or metabolic-) acidosis that accompanied common cardiac arrests. In fact, these studies determined that; if effective ventilation and artificial circulation were rapidly provided, *adverse side effects* were consistently associated with the "automatic" IV administration of even **ONE** 50mEq ampule of Bicarb.(42-47)

Consequently, in 1986, the AHA national standard protocols for ACLS Bicarb-administration were dramatically changed:

"Sodium bicarbonate should be used, if at all, only after application of more definitive and better substantiated interventions, such as prompt defibrillation, effective chest compressions, endotracheal intubation and hyperventilation with 100% oxygen, and the use of drugs such as epinephrine and lidocaine. These interventions will usually take approximately 10 minutes; thereafter, sodium bicarbonate therapy, although not recommended, can be considered in specific clinical circumstances such as documented preexisting metabolic acidosis with or without hyperkalemia."(42-47)

Because they continue to be proven effective and appropriate for treatment of "common" cardiac arrests, the 1986 ACLS Bicarb-administration protocol guidelines have remained unchanged and in effect since then; to include the most-recently updated (2000) ACLS protocol guidelines.(43,57,58):

"Adequate ventilation and CPR, not bicarbonate, are the major 'buffer agents' in cardiac arrest. [Bicarb is] Not recommended for routine use in cardiac arrest patients."(58)

Only in states of "known preexisting hyperkalemia" (when the patient is known to be a victim of a potassium "overdose" – such as a renal failure patient undergoing dialysis); states of "known preexisting ... diabetic ketoacidosis" (states of **TOO-HIGH** blood sugar – the opposite of the most common cause of a diabetic's altered level of consciousness, **LOW** blood sugar), or in cases where known overdoses of tricyclic antidepressants or aspirin have occurred; should

administration of Bicarb be considered for initial administration during cardiac arrest management.(42,43,48,57,58)

"pH" stands for "potential of hydrogen," and represents the measurement of a substance's degree of acid or alkali (the opposite of acid) content. To measure someone's internal body pH measurement, medical personnel must obtain a blood sample from an **artery**. (Most blood samples are obtained from a vein.) Then, the arterial blood sample is sent to have its "gas" content (measurements of oxygen, carbon dioxide, and the like) analyzed and obtained.

Thus, this form of blood test is called an "**Arterial Blood Gas**" – an "**ABG**."

The normal human ABG pH measurement ranges from **7.35 to 7.45**. If the ABG pH measurement number is less than 7.35, the person is suffering from a state of abnormal **acid** content; a state of "acidosis."

In studies of patients who suffered common out-of-hospital cardiopulmonary arrests and received "no buffer therapy, the average pH at hospital arrival was 7.23, with a range of 7.20 – 7.26."(26,44-47)

According to published studies of restraint asphyxia Victims, however, the average pH measured at hospital arrival was **6.99**, with a range of **6.76 – 7.16**."(26,27)

Unfortunately (for case-study-evaluation purposes), the vast majority of restraint asphyxia Victims are pronounced "dead" at the emergency department, without *ever* having their pH measurement obtained.

Of the 19 restraint asphyxia cases I have personally evaluated to date (as an "Expert Witness"), the pH measurement of only 3 Victims was obtained prior to discontinuation of resuscitative efforts – prior to them simply being pronounced "DEAD.":

- Baby Leavy, Personal Representative of the Estate of Rodell Braxton, and Freddie Braxton, vs. City of Janesville, Wisconsin; et al. CAUSE NO. pending.: **pH of 6.9**
- Kapanak vs. City of Phoenix; Superior Court of the State of Arizona; County of Maricopa; No. CV 2001-012136.: **pH of 7.24 (after administration of 2 amps of Bicarb!)**
- Giannetti vs. City of Stillwater Police Department, Cause No. Pending.: **pH of 6.7**

Because the pH reading obtained in the Kapanak case was *skewed* ("altered" by administration of two amps of Bicarb *prior* to ABG draw), it must be excluded from consideration. But, after averaging the other two cases' pH readings in with those of published case studies, the average pH at hospital arrival was 6.9, with a range of 6.7 – 7.16.(26,27)

Thus, the pH findings of restraint asphyxia cases I have worked are entirely consistent with those evaluated and published by others.

IN SUMMARY:

- The normal human ABG pH measurement ranges from **7.35 to 7.45**.
- Common out-of-hospital cardiopulmonary arrest average pH measurements upon hospital arrival are **7.23**, with a range of **7.20 – 7.26**.
- Common out-of-hospital restraint asphyxia average Victim pH measurements upon hospital arrival are **6.9**, with a range of **6.7 – 7.16**.

Clearly, "common" cardiopulmonary arrests are *not* accompanied by the degree of extreme acidosis consistently demonstrated by restraint asphyxia Victims. Thus, when an individual suffers a cardiopulmonary arrest while restrained in a manner that makes breathing difficult (or impossible), and the Victim's pH is significantly *more acidotic* than that of a "common" cardiopulmonary arrest victim, the Victim's death can ONLY be due to restraint asphyxia – and not, at all, be legitimately attributed to a "common" cardiopulmonary arrest.

REFERENCES for **THIS SEGMENT** (and ALL OTHER PARTS) of
RESTRAINT ASPHYXIA – SILENT KILLER
can be Accessed at:

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

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