

ORIGINAL PAPER

Pathology/Biology

Prone restraint cardiac arrest in in-custody and arrest-related deaths

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Abstract

We postulate that most atraumatic deaths during police restraint of subjects in the prone position are due to *prone restraint cardiac arrest* (PRCA), rather than from restraint asphyxia or a stress-induced cardiac condition, such as excited delirium. The prone position restricts ventilation and diminishes pulmonary perfusion. In the setting of a police encounter, metabolic demand will be high from anxiety, stress, excitement, physical struggle, and/or stimulant drugs, leading to metabolic acidosis and requiring significant hyperventilation. Although oxygen levels may be maintained, prolonged restraint in the prone position may result in an inability to adequately blow off CO₂, causing blood pCO₂ levels to rise rapidly. The uncompensated metabolic acidosis (low pH) will eventually result in loss of myocyte contractility. The initial electrocardiogram rhythm will generally be either pulseless electrical activity (PEA) or asystole, indicating a noncardiac etiology, more consistent with PRCA and inconsistent with a primary role of any underlying cardiac pathology or stress-induced cardiac etiology. We point to two animal models: in one model rats unable to breathe deeply due to an external restraint die when their metabolic demand is increased, and in the other model, pressure on the chest of rats results in decreased venous return and cardiac arrest rather than death from asphyxia. We present two cases of subjects restrained in the prone position who went into cardiac arrest and had low pHs and initial PEA cardiac rhythms. Our cases demonstrate the danger of prone restraint and serve as examples of PRCA.

KEYWORDS

prone restraint, in-custody deaths, arrest-related deaths (ARDs), police-involved deaths, restraint asphyxia, positional asphyxia, excited delirium, sudden cardiac death, metabolic acidosis, George Floyd, forensic pathology, autopsy

Highlights

- Prone restraint deaths result from metabolic acidosis, not hypoxic asphyxia.
- Metabolic demand is increased by stimulant drugs, physical exertion, and stress.
- Metabolic acidosis requires hyperventilation to “blow off” carbon dioxide (CO₂).
- Prolonged restraint in the prone position may prevent adequate elimination of CO₂.
- An initial ECG rhythm of PEA or asystole after a cardiac arrest is generally inconsistent with a primary cardiac etiology.

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1 | INTRODUCTION

Deaths occurring during police encounters garner significant attention (consider, for example, the trial of Derek Chauvin for the death of George Floyd [1]). Recently, it has been claimed that there were over 800 deaths from police encounters per year in the United States and over half went unreported in the National Vital Statistics System [2, 3].

However, not all deaths during police encounters or while in-custody are of a “George Floyd” type. Many such deaths occur from overt violence, such as shoot-outs or from drug overdoses that began prior to the arrival of the police, while others appear to be related to the police encounter and are not from trauma or obvious explanations. In attempting to identify cases of interest, various investigators have focused on deaths that are in-custody, occurring upon arrest, or when force is used and may variously exclude deaths occurring during incarceration, during pursuit, or while attempting to render emergency medical care. Here, we will discuss arrest-related deaths which involve restraint in a prone position and without other obvious explanation of death.

Social scientists and prosecutors have typically investigated deaths during police encounters by use-of-force analyses [4], but such analyses are not scientific or medical determinations of whether or not the police intervention caused the death. The Bureau of Justice Statistics (BJS) collects data on arrest-related deaths from law enforcement agencies that are more specific to cause of death determinations pursuant to the Death in Custody Reporting Act of 2013 (DCRA). BJS recently reported that of the 70% of the cases in which the manner of death was classified as homicide [5]:

- In 19.9%, the police fought or struggled with decedents.
- In 7.6%, the police physically restrained decedents.
- In 12.3%, the police restrained decedents with equipment.
- In 6.2%, decedents were placed in the prone position.

However, due to non-uniform, non-detailed, and possibly biased reporting, we do not have good statistics on the frequency, manner, or duration of prone positioning, use of restraints, the underlying health of the subjects in these police encounters, or other factors that should be analyzed to understand these deaths.

2 | BACKGROUND

Beginning in 1981, Wetli in Miami reported on fatal cocaine-induced *excited delirium* [6, 7], borrowing the psychiatric concept of fatal acute exhaustive mania (Bell’s mania) [8, 9]. He described subjects behaving bizarrely and then dying suddenly. Often the subjects would be hyperthermic and sometimes they would manifest super-human strength, requiring several officers to subdue them. Excited delirium has since been attributed to a broad array of stimulant drugs besides cocaine [10]. DiMaio declared that excited delirium describes a fatal syndrome involving an adrenergic storm that is

refractory to treatment [11]. The mechanism of death has not been well elucidated but has been attributed to stress-induced cardiac arrhythmia, myocardial infarction, channelopathy, or cardiomyopathy. Diagnostic criteria, defining the levels of excitement or delirium needed, have not been established and more recent literature suggests the term has been relaxed to include non-psychotic events.

Reay in Seattle had been studying deaths in custody since at least 1968 [12] and had published on asphyxial deaths from neck holds [13, 14]. In 1988, Reay et al. reported an experimental study on the deleterious physiological effects when a person is prone and hog-tied (handcuffs behind the subject’s back and tethered to leg shackles with knees bent; the term *prone maximal restraint position* (PMRP) may be used when additionally the back is flexed) [15]; however, the study was later recognized as flawed [16–18]. Then in 1992, he reported on three cases of deaths from *positional asphyxia* occurring when arrestees were placed hogtied in a prone position in the rear compartments of police cars [19]. Bell et al., later that year, reported 30 cases of positional asphyxia, in which the decedents were found in positions that suggested external airway obstruction or inadequate ventilatory function [20]. After the early reports of the dangers of hog-tying, police departments thereafter largely stopped using it as a control tactic (at least for a period of time).

In 1993 and in later papers, O’Halloran noted that cases of excited delirium occurred in subjects restrained in a prone position by police officers and suggested that the restraint rather than the excited state was responsible for the deaths. The restraint in most cases did not involve hogtying. He thought the deaths resulted from the restraint of the arms and legs and weight applied to the backs of victims rather than body posture and he suggested the use of the term *restraint asphyxia* instead of positional asphyxia [21–23].

After a lawsuit was launched against the County of San Diego for the death of Daniel Price in 1994 [24], the County funded a medical group at the University of California at San Diego to perform experimental studies involving weights placed on young healthy subjects in the prone position. That San Diego group and their colleagues published several reports concluding that the effect on ventilation was clinically insignificant, including weights up to 102 kg (225 lbs.) [17, 25–27]. Kroll has pointed out that the entire weight of an officer is not necessarily applied to the back of the chest [28] and that there is a difference between static and dynamic compressional forces; he developed a biomechanical model suggesting that an adult male can tolerate up to 572 lb. of static pressure for a short period before developing rib fractures and a flail chest [29].

Not only has the credibility of restraint asphyxia been questioned, but so too the credibility of excited delirium has been challenged as lacking a scientific foundation [30].

2.1 | Forensic pathologist community reaction

The National Association of Medical Examiners (NAME) promulgated a position paper in 2017 on in-custody deaths, but it does

not discuss the possible causes of death in these cases [31]. In the absence of a clear cause of death, the forensic pathology community has been offered two polarizing explanations for the cause and mechanism for this subset of in-custody deaths—excited delirium and restraint asphyxia. Both excited delirium, which tends to exonerate the police, and restraint asphyxia, which tends to implicate the police, have their proponents and detractors. Many forensic pathologists, wanting to avoid either term, have asserted, with little supporting evidence, that sudden deaths were due to a malignant cardiac arrhythmia or underlying coronary heart disease in the setting of a physical struggle. Some have pointed to stress-related cardiomyopathy (Takotsubo's). Takotsubo's cardiomyopathy has a high overall survival rate (>94%), with deaths being noted predominantly in older females (>82%); it has no association with the prone position [32, 33]. PRCA, on the other hand, is characterized by healthy young males and a high death rate. Still, other explanations for in-custody deaths have been espoused or used on death certificates [34].

Autopsy examinations in arrest-related deaths may be unrevealing. Luke and Reay stated that deaths in-custody often demonstrate little pathologic evidence of the cause of death and there are no diagnostic markers of asphyxial deaths [35]. Krexi found that more than half of sudden cardiac deaths during or after a stressful event, primarily restraint or altercation, in a predominantly young cohort, had negative autopsies and morphologically normal hearts [36]. Wolf described sixteen cases of atraumatic in-custody homicides and found:

“Causes of death statements in these cases are usually long and descriptive, reflecting the inability to ascribe a relative importance to one factor over another... The cause of death statements were descriptive in each and often included a listing of a presumed mechanism (e.g. sudden cardiac death or arrhythmia), underlying natural disease (e.g., hypertensive or atherosclerotic disease), the presence of intoxicant(s), and/or psychiatric conditions, along with an external factor associated with restraint or pursuit.” [37].

Karch was more blunt in his assessment, when he declared, “[Arrest-related deaths] go unexplained, at least in any acceptable scientific sense” [38].

2.2 | Recent developments

In 2020, Strömmer et al. comprehensively reviewed the literature and concluded that restraint was the common denominator for virtually all fatalities and found “no existing evidence that indicates that [excited delirium] is inherently lethal in the absence of aggressive restraint” [39]. This debate as to whether or not prone restraint is dangerous has attracted the attention of the public media [40, 41].

More recently in 2020, Steinberg, a cardiologist, postulated that atraumatic prone restraint arrest-related deaths are a result of acute metabolic acidosis, rather than restraint asphyxia or hypoxia [42]. In other words, the deaths are not a matter of hypoventilation causing hypoxia. Rather, the deaths result from an inability to adequately hyperventilate and perfuse the lungs, resulting in an inability to adequately eliminate CO₂, with consequent cardiac arrest, in a person who has been engaged in physical exertion and is restrained in a prone position. In 1999, Hick observed the profound metabolic acidosis and cardiac collapse in restrained patients, especially in the prone position [43], but Steinberg further developed the association with prone restraint and proffered the term *prone restraint cardiac arrest* (PRCA). His article sparked a lively discussion [44–48].

We now present two cases, both documented on video recordings, which demonstrate the danger of prone restraint and support the concept of PRCA. In both cases, stimulant drugs were involved, causing agitation and delusions, but without significant struggle or hostile aggression.

3 | CASE #1

An obese 51 year-old male subject (BMI 39.7), who used stimulant drugs, began “acting weird.” See Table 1 for detailed timeline. He cut his hand when he broke a ceramic plate on the kitchen

TABLE 1 Timeline for case #1

0 min	Police were called
~5 min	Subject taken to the ground supine by brother-in-law
~17 min	Police arrived at apartment
~20 min	Subject handcuffed
~24 min	Subject's ankles shackled
~27 min	EMS arrived at residence
~29 min	Subject rolled prone
~31 min	Subject strapped on stretcher in prone position
~33 min	Subject placed still prone on rollaway with more straps
~34 min	Subject became quiet
~35 min	Last movement, presumed cardiac arrest; subject loaded into ambulance
~38 min	All straps were removed
~39 min	Subject rolled supine & CPR began
~44 min	ECG revealed sinus bradycardia
~45 min	Carotid pulse was found to be absent
~66 min	Subject arrival at emergency room
~84 min	Blood drawn for chemistries & bicarbonate
~130 min	Arterial blood drawn for blood gases
~3.8 days	Pronounced dead after initial call

counter. This woke up his brother-in-law, who then controlled him by wrestling him to the ground supine and placing his foot on his left arm. When the police arrived, the brother-in-law was standing over the subject still with his foot on his arm. The subject was lying in place and repetitively shouting irrational thoughts. The police called emergency medical services (EMS) for his bleeding hand and then quickly handcuffed the subject with his hands behind his back and hobbled his ankles. Having secured him, they turned him on his side and attempted to calm him as he continued to shout. EMS arrived, placed him prone on a bedsheet and lifted him onto a flexible portable stretcher onto which they strapped him tightly around the lower chest and thighs in that face-down position. They carried him down the stairway out to a wheeled stretcher, onto which they placed him in the face-down position still strapped prone in the stretcher, and secured him with more straps pulled tightly around the chest and sides. He was wheeled and loaded onto the ambulance with a mechanical lift. During the first approximate 3 min while strapped, he forcefully raised his head and shoulders multiple times while constantly yelling out a two-word utterance. He then became silent and barely lifted his head for another approximate 48 seconds, after which there was no further movement or sound.

As he became unresponsive, he was loaded onto the ambulance. Bag-valve-mask ventilation was given. A 3-lead ECG was applied, and it revealed a sinus bradycardia at a rate of 42-44 bpm. The straps and restraints were finally removed, and he was turned onto his back. He had been in the prone position for approximately 10 min, during which time he had been singly and doubly tightly strapped in that prone position for about 7 of those 10 min. A carotid pulse was absent and shortly thereafter he became asystolic. Lactated Ringers and four doses of epinephrine were administered via intraosseous access. With further resuscitative measures, electrical activity was regained, but his pulse was inconsistent, and he arrived at the hospital in PEA. During transport, he received four doses of intraosseous epinephrine and two boluses of Ringers lactate. In the ambulance, his end tidal CO₂ averaged 65.6 mmHg (17-99; normal 35-45 mmHg).

Shortly after arrival at the hospital, he was successfully resuscitated, but never regained consciousness. His admission vital signs included a temperature of 101.1°F, blood pressure of 135/47 mmHg, pulse of 79 bpm, respiratory rate of 48 rpm, and a S_pO₂ of 95%. Arterial blood gases collected approximately 1½ hours after his cardiac arrest, revealed a profound acidosis and hypercarbia (see [Table 2](#)). An echocardiogram during his hospital stay revealed normal heart function. He was pronounced brain dead roughly 3½ days after admission.

Autopsy found only minor external trauma. Internal exam revealed an enlarged heart (650 gm), although it was recognized that he was found to have a normal echocardiographic heart function in the hospital prior to his death. Cardiovascular pathology consultation noted biventricular hypertrophy and dilatation, as well as multiple foci of punctate subendocardial hemorrhage of the left ventricle consistent with a reperfusion injury, but no gross coronary artery disease. Neuropathology revealed acute hypoxic-ischemic

encephalopathy and cerebral edema. Postmortem toxicology, performed on autopsy blood after 3½ days hospitalization, revealed phencyclidine (PCP), tenocyclidine (TCP), methadone, and cocaine metabolites (see [Table 2](#)).

4 | CASE #2

An obese 41 year-old male (BMI 32.7) was seen by police stepping in and out of a busy roadway, shouting and talking to an imaginary, threatening person; see [Table 3](#) for a detailed timeline. He cooperated when told to sit on the curb and was handcuffed after admitting to having used methamphetamine. While awaiting an ambulance for involuntary mental health detention, he was fidgety, breathing heavily, sweating profusely, and continued to react to imaginary threats. When he abruptly stood up, he was again asked to sit down, but did not comply this time. As it appeared he was about to flee, the three responding officers immediately grabbed him and took him to the ground, where he continued to physically resist by thrashing about and raising and kicking his legs. He was rolled over into a prone position on the pebble-laden ground, with his face and shoulders on the adjacent concrete sidewalk. When he began scraping his face, a towel was placed around and under his face to lift it up from the concrete. He continued to gasp and quietly cry, with occasional bursts of loud shouts. At one point, he twice uttered in a low voice "I can't breathe, I can't breathe." For a brief period, an attempt was made to hobble his ankles just when the ambulance finally arrived.

Then, while still being prone restrained by officers, paramedics instructed that a rigid spine board be placed on his back and an officer sit on it. At least 5 officers pushed down on the board and an officer, who weighed 190 lbs. and wore heavy tactical gear, sat on top while the handcuffs and ankle hobble were removed, and as they began to strap the wrists and ankles to the board on his back. Prior to the board being placed on his back, the subject had already been restrained in a prone position for approximately 10-3/4 min. The rigid board was on his back while in the prone position for approximately 3-3/4 min, with the officer sitting on it for approximately 1¼ min. The subject uttered his last sound 9 s after the officer sat on the board.

When the subject was finally rolled over to the supine position, strapped to the rigid board, after having been in prone restraint for approximately 14-1/2 min, cardiac arrest was recognized. He was placed on a wheeled stretcher, pushed into the ambulance, and ACLS was initiated. The first ECG strip displayed PEA and then deteriorated to asystole. During transport, two IV doses of epinephrine were administered, but no fluids. The EMS transport to the ED lasted 12 min. ACLS was unsuccessful and he was pronounced dead approximately 12 min after transfer to the emergency room. Hospital laboratory results are noted in [Table 4](#).

Autopsy confirmed multiple minor injuries and a large subcutaneous contusion across his back. The heart was enlarged (436 g) with

TABLE 2 Laboratory results in case #1

Antemortem clinical chemistries				
Specimen	Analyte	Result	Units	Reference range
~49 min post cardiac arrest				
Whole blood	Sodium	142	mmol/L	(135–146)
Whole blood	Potassium	4.3	mmol/L	(3.2–5.0)
Whole blood	Glucose	286	mg/dL	(65–140)
Whole blood	Creatinine	1.7	mg/dL	(0.5–1.5)
Whole blood	Troponin	0.485	ng/mL	(0.0–0.045)
Whole blood	HCO ₃ ⁻	18	mmol/L	(18–32)
~95 min post cardiac arrest				
Arterial blood	pH	7.015		(7.35–7.45)
Arterial blood	pO ₂	273.4	mmHg	(75–100)
Arterial blood	pCO ₂	70.6	mmHg	(35–45)
Arterial blood	HCO ₃ ⁻	15	mmol/L	(18–32)
~5 h post cardiac arrest				
Blood plasma	Lactate	0.9	mmol/L	(0.5–2.0)
Hospital toxicology screen				
Specimen	Analyte	Result		
~70 min post cardiac arrest				
Urine	Phencyclidine	Positive		
Urine	Cocaine (metabolites)	Positive		
Urine	Opiate	Negative		
Urine	Methadone	Positive		
Postmortem toxicology				
Specimen	Analyte	Result	Units	
~5 1/2 days post cardiac arrest				
Autopsy subclavian blood	Phencyclidine	0.1	mg/L	
Autopsy subclavian blood	Tenocyclidine	0.06	mg/L	
Autopsy Subclavian blood	Methadone	0.4	mg/L	
Autopsy bile	Cocaine metabolites	Positive		

[Correction added on 16 Aug 2022, after first online publication: Table 2 has been revised.]

left ventricular hypertrophy and focal severe (>80%) stenosis of the left anterior descending and right coronary arteries. Postmortem toxicology on autopsy blood revealed amphetamine and methamphetamine (see Table 4).

5 | DISCUSSION

We discuss here a subset of police arrest-related deaths with no obvious cause of death and involving prone restraint. It has been reported that the rate of arrest-related deaths is approximately one death per every 1000 arrests [28]. Prone restraint related deaths have been observed to occur two to three times per year in Ontario, England and Wales, Los Angeles and the Netherlands [49–52], each with populations exceeding 10 million. Thus, they are relatively rare, so it is not surprising that smaller prospective epidemiologic studies have failed to find such cases [53–55]. As

mentioned in the introduction, over half of arrest-related death cases have not been reported to vital statistics; good statistics on prone positioning do not exist.

5.1 | Stressful encounters and increased metabolic demand

Stress, agitation, and physical exertion are characteristic of the arrest-related deaths under discussion here. Police are called to situations involving violent or agitated behavior. Stimulant drugs with or without such behavior will increase metabolic demand. Arrival of the police will likely trigger a physiologic fight-or-flight response. Under the influence of drugs or in a psychotic or agitated state, the subject may not cooperate well with the commands of law enforcement and emergency medical personnel. The escalating confrontation eventuates in an arrest and restraint of the subject.

TABLE 3 Timeline for case #2

0min	Police observed subject in road
~7–9 min	Subject seated on curb & handcuffed
~11 min	Subject abruptly stood up
~12 min	Subject taken to the ground by police
~13 min	Subject rolled prone
~18 min	Towel used to raise head
~19 min	Subject exclaimed "I can't breathe!"
~22 min	Subject's ankles shackled
~22 min	Ambulance arrived
~23.7 min	Rigid board placed on face-down subject's back as cuffs & shackles began to be removed
~24 min	Officer sat on rigid board initially as subject's wrists & ankles were being strapped to board
~24.2 min	Last sound (presumed cardiac arrest)
~25.2 min	Officer seated on board got up
~27.5 min	Rolled supine, strapped to board, unresponsive
~28 min	Subject placed supine on rollaway
~30 min	Subject loaded into ambulance
~30 min	CPR began
~30 min	ECG revealed PEA
~41 min	Subject transferred to emergency room
~46 min	Venous blood gas tested
~53 min	Pronounced dead

The subject may physically struggle against the force used by police or against the physical restraints. In a study by Michalewicz et al., young healthy study subjects reached 84% of their maximal treadmill heart rate in a mere 60s from rest when verbally encouraged to struggle against their restraints [27]. Such physical exertion, agitation, emotional state, use of stimulant drugs, and stress of the encounter result in a heightened metabolic demand, not present at rest.

The increase in metabolic demand is manifested as both an increased oxygen consumption and an increased production of carbon dioxide, which in turn will result in increases in cardiac output and minute ventilation. The increase in both oxygen consumption and carbon dioxide production is dramatic, perhaps over ten-fold (see Table 5). Oxygen demand rises linearly with work rate (power) up to a threshold when oxygen consumption becomes maximal and rises no further. The oxygen demand and threshold will vary according to an individual's health, fitness, and athletic training. The increase in ventilation is much greater than in cardiac output [56] (see Table 5).

Under increased metabolic demand, the body conserves oxygen, but carbon dioxide increases remarkably. Due to compensatory mechanisms, including a right shift in the hemoglobin dissociation curve, arterial pO_2 will remain constant with exercise or even increase slightly initially. Emergency resuscitation procedures now emphasize circulation before airway and breathing due to the

oxygen reserves of the blood and lungs [57]. Moreover, pulse oximetry does not provide early detection of hypoventilation, apnea or airway obstruction. End-tidal CO_2 monitoring is now recommended by the American Society of Anesthesiologists for procedural sedation and anesthesia, as respiratory depression will be over 17 times more likely to be detected with capnography [58, 59].

The problem with most experimental studies of prone restraint, as Reay had warned, is that they were not conducted under real-world stress conditions or a state of high metabolic demand and thus, will not reveal the ultimate dangerousness posed by the prone restraint [16, 18].

5.2 | Metabolic acidosis and the need to hyperventilate

Metabolic demand will produce a metabolic acidosis (blood $pH < 7.35$) when compensatory mechanisms are overcome. Intense physical exercise may result in blood pH levels below 7. Above a certain threshold, called the anaerobic threshold or point of onset of blood lactate accumulation, anaerobic glycolysis predominates over aerobic cellular metabolism, and lactic acid, a product of glucose metabolism, rapidly accumulates; the concomitant lactate threshold is defined as blood lactate level of 4.0 mM [60]. Metabolic acidosis is caused by non-mitochondrial ATP turnover, although blood lactate accumulation is a good indicator of the acidosis caused by the resultant hydrogen proton release [61]. The lactic acid buildup occurs at lower levels in unfit individuals and occurs at higher levels with the fitness and training of the individual. At the threshold point, carbon dioxide production outpaces oxygen compensation; the respiratory exchange ratio (RER) of CO_2 output to O_2 consumption will increase from below 1 to greater than 1 with anaerobic metabolism.

Ho et al. found that significant physical exertion, simulating resistance to police, resulted in a reduction of blood pH from a normal value of 7.36–7.04 within 45s and it remained low for 10min [62]. Hick et al. reported profound acidosis in five individuals in agitated states due to recent cocaine use who went into cardiac arrest after a struggle and restraint (at least 4 prone) and had an average pH of <6.62 (6.25–6.81). Another five (unknown position) who did not have cardiac arrest had an average pH of 7.01 (6.76–7.16) [43]. He noted that the degree of acidosis that he observed exceeded that routinely seen in sudden death and referred to a series of 257 patients with out-of-hospital cardiac arrests without buffer therapy all of whom had a pH of 7.2 or above [63].

The body responds to metabolic acidosis and hypercarbia by hyperventilation [56, 64–68]. This is a respiratory alkalotic compensation in which ventilation (rate and volume of breathing) is increased and carbon dioxide is eliminated during exhalation. This respiratory regulation is commonly referred to as "blowing off CO_2 ".

The bicarbonate–carbon dioxide equilibrium is the primary pH buffer system in the human body, where

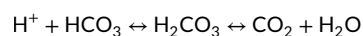


TABLE 4 Laboratory results in case #2

Antemortem clinical chemistries				
Specimen	Analyte	Result	Units	Reference range
POC testing ~16 min into resuscitation				
Venous blood	pH	6.64		(7.31-7.41)
Venous blood	pO ₂	12	mmHg	(35-45)
Venous blood	pCO ₂	157	mmHg	(41-51)
Venous blood	HCO ₃ ⁻	17	mmol/L	(23-29)
Postmortem toxicology				
Specimen	Analyte	Result	Units	
Peripheral autopsy blood	Amphetamine	95	ng/ml	
Peripheral autopsy blood	Methamphetamine	2,459	ng/ml	

[Correction added on 16 Aug 2022, after first online publication: Table 4 has been revised.]

TABLE 5 Physiologic parameters during rest and during strenuous exercise

	Resting	Strenuous Exercise	Increase
Oxygen consumption	300ml/min	3000ml/min	10x
Carbon dioxide production	250ml/min	3000ml/min	12x
Minute ventilation	5-6L/min	150L/min	25-30x
Respiratory rate	12-20breaths/min	40-50breaths/min	2-3x
Tidal volume	0.5L/breath	3L/breath	6x
Cardiac output	4-6L/min	25L/min	4-6x
Pulse	60-100bpm	150bpm	1.5-2.5x
stroke volume	100ml/min	160ml/min	1.5x

Note: These vary with an individuals health, fitness, and athletic training. (Reference [56], West & Luks, pp. 173-174; Reference [60], McArdle, p. 345; Reference [68], Levitsky, p. 251).

Expiration

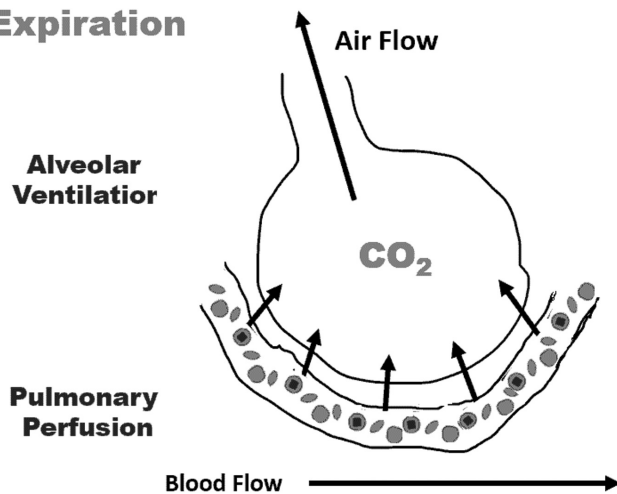


FIGURE 1 This figure depicts the elimination of carbon dioxide as involving perfusion and ventilation (see Reference [68], Levitsky; Reference [56], West & Luks)

The vast majority of carbon dioxide in the body is in the form of bicarbonate (HCO₃⁻). Bicarbonate functions as an alkalotic substance and carbon dioxide (CO₂) functions as an acidic substance.

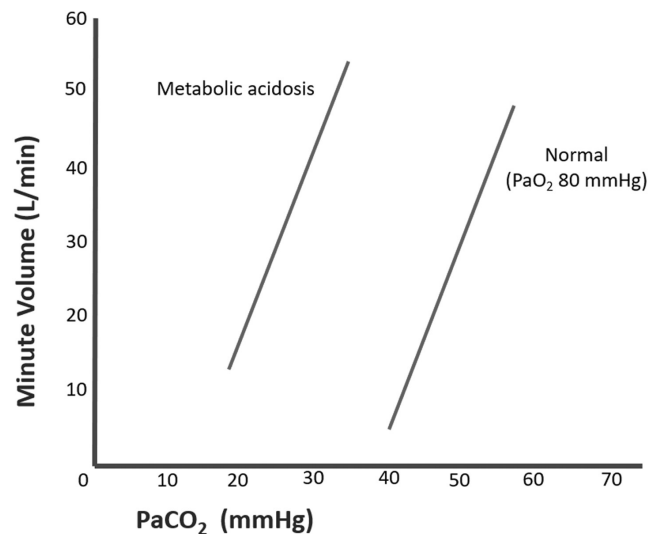


FIGURE 2 This graph demonstrates the direct ventilatory volume per minute response to the partial pressure of arterial carbon dioxide. The graph shows the dramatic increase in ventilation with increasing arterial pCO₂ and the greater need to ventilate at lower arterial pCO₂ levels during acidosis (see Reference [64], Yartsev; Reference [68], Levitsky, p. 219; Reference [56], West & Luks, p. 161)

The result is that blood pH is a direct function of CO₂ as defined in the Henderson-Hasselbach equation:

$$\text{pH} = 6.1 + \log_{10} \left(\frac{[\text{HCO}_3^-]}{0.03 \times \text{pCO}_2} \right)$$

The bicarbonate concentration is primarily regulated by the kidneys, but CO₂ is primarily regulated by the lungs. However, elimination of CO₂ is a function of both alveolar ventilation and blood perfusion (see [Figure 1](#)).

As seen in [Figure 2](#), small changes in pCO₂ result in relatively large ventilatory changes. In states of metabolic acidosis, both pCO₂ and pH will drive a several-fold increase in ventilation. During light to moderate exercise, ventilation increases linearly with work rate, but at the ventilatory threshold, near (if not at) the anaerobic threshold, pulmonary ventilation increases disproportionately to the oxygen demand [56, 60]. Short periods of maximal exercise may increase respiratory rates 2–3 times (from 16–20 breaths/min to 40–50 breaths/min) along with the increase in depth of breath, which together will increase ventilation 20–25 times (5–6 L/min to 150 L/min), while cardiac output increases only 4–6 times [56, 68].

5.3 | Prone restraint

The hyperventilation of metabolic acidosis involves both more rapid and deeper breathing (see [Tables 5](#)). The mechanics of inspirations are normally a combination of downward excursion of the diaphragm and raising of the ribs (outward “bucket-handle expansion”). During eupnea, the diaphragm is responsible for about two-thirds while supine and one third to one half of the air flow while seated or standing [68]. Expiration is normally a passive process. During hyperpnea, the excursion of the diaphragm will increase beyond the normal 1–2 cm of quiet breathing excursions; deep breaths may involve excursions up to 10 cm. Normal tidal volumes are about 500 ml, but a deep breath may have twice that volume, about 1000 ml [60]. Also, accessory muscles (scalene muscles, sternocleidomastoids) are recruited that elevate the first two ribs and sternum. The normally passive expiration will become active through contraction of the abdominal wall (rectus abdominis, transversus abdominis, the external and internal oblique muscles), increasing abdominal pressure and stiffening the intercostal spaces. Furthermore, faster flow rates require exponentially greater pressures to overcome turbulence. Thus, the work of breathing becomes considerably greater and will contribute to the metabolic demand.

5.3.1 | Restricted ventilation

The prone position can restrict inhalation in at least three ways: (1) chest compression, (2) resistance to upwards and outwards expansion of the chest, and (3) diaphragmatic excursion limitation (see [Table 6](#)). The chest wall has a certain amount of compliance from rib flexure and cartilaginous elasticity. In the prone position, the weight

TABLE 6 The effects of prone positioning are summarized here

Ventilation Restriction	Decreased Venous Return
1. Chest compression	1. Pressure on the inferior vena cava
2. Resistance to upwards and outwards chest expansion	2. Pressure on the cardiac atria
3. Pressure on the diaphragm	3. Pressure on the pulmonary veins

of the person will cause some decrement of the anterior–posterior dimension and resultant decreased chest volume due to elasticity of the ribs and cartilage. This initial compression compliance requires little weight, but further decrement would require greater force. The prone position will limit or resist the “bucket handle” upward and outward rib action that, by contraction of the external intercostal and accessory muscles, normally expands the chest. That the compression and/or limitation of the chest expansion will reduce pulmonary function was demonstrated by Barnett et al., when the effect of tucking the arms under the sides of subjects (“supported prone position”) resulted in improved ventilation [69]. Pressure to the abdomen will be transmitted as cephalad pressure to the underside of the diaphragm, limiting or resisting diaphragmatic excursion [70].

Steinberg has reviewed the peer-reviewed literature on in-custody atraumatic prone-related fatalities [42]. Studies have shown the prone position alone to cause a decrease in ventilation by 8%–16% [69, 71, 72]. Studies have also shown that hogtying subjects, who were placed prone, can decrease ventilation 13%–28% [17, 26, 27], with one study by Roeggla showing a decrease of 40% [73]. Additional studies with weights on the backs of individuals showed maximal decreases of 22% with 23 kg [26], 35% with 75 kg [72], and 30% with 102 kg [27]. The resultant changes were consistent with a restrictive pulmonary functional change rather than obstructive, with similar decreases in forced vital capacity (FVC) and forced expiratory volume (FEV1).

However, the San Diego group and their associates [17, 26, 27, 71] interpreted this decrement to be clinically insignificant, while other investigators found it to be significant [72–75]. For instance, Barnett et al. found that “restraint position has the ability to impede life-maintaining physiologic functions” [75]. Parkes and Carson noted that while others had found the reduction in pulmonary function to be clinically insignificant, they concluded “restraint position should be considered a risk factor for sudden death during restraint” [74]. Hick declared,

“The detriment to a struggling, profoundly acidotic patient whose life may depend on the ability to develop a respiratory alkalosis has not been studied, and thus we agree with previously expressed concerns that this reduction cannot be inferred to be ‘not clinically relevant.’” [76].

A healthy person will compensate for a mild to moderate ventilatory restriction by merely breathing faster to maintain oxygen levels and CO₂ levels. However, the same ventilatory restriction in the presence of a metabolic acidosis can become clinically significant. The

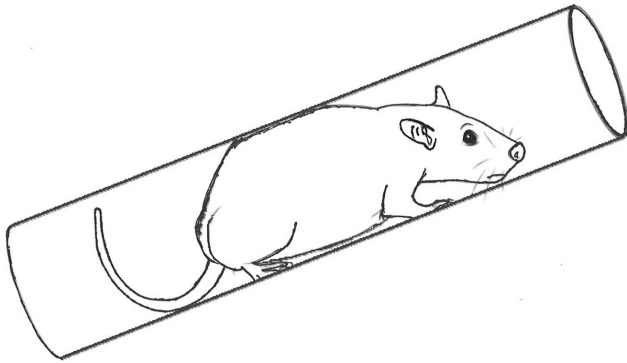


FIGURE 3 This cartoon of a rat in a tube depicts the animal model of restricted ventilation (see Reference [78], Pudiak & Bozarth)

large increase in ventilation needed will require both faster and deeper breaths and any restrictive impairment will become evident. If CO_2 is unable to be blown off, then the CO_2 in the blood will rapidly accumulate and the acidosis will be uncompensated and become more severe. With a sufficiently low pH, the heart will stop contracting.

In ventilation studies in healthy individuals in prone restraint at rest, there has been only a mild increase in pCO_2 [17], with Roeggla reporting a 14.7% increase in end tidal CO_2 [73]. There have been no reported significant changes in pCO_2 in studies of healthy subjects restrained in the prone position following exercise [17, 72, 77]. Above the anaerobic threshold, minute ventilation of exercising individuals is driven by their carbon dioxide level. Individuals in the study also did not achieve a significant metabolic acidosis, and their decrease in ventilation was compensated by the increase in respiratory rate maintaining a stable minute volume and pCO_2 . In the Cary study, individuals achieved a mild increase in pCO_2 of only 34.5 mmHg [71,72]. Individuals showed a 33% decrease in the maximum voluntary ventilation (MVV) when hogtied with weights on their backs. However, minute ventilation (MV) was maintained and compensated for by a 35% increase in respiratory rate. Thus, no increase in pCO_2 was noted or would be expected [72]. Chan also only achieved a pH of 7.28–7.33 and an average pCO_2 of 30.7 mmHg post exercise [17]. The reported decrease in ventilation in hogtied subjects of 21% was likely easily compensated also by the increase in respiratory rate which was not reported. Sloane studied obese hogtied subjects after exercise and achieved a pCO_2 of 32–33.3 mmHg [77]. He only reported MV, which was not different between seated subjects and subjects hogtied and in the prone position. He did not report FEV1, FVC or MVV nor respiratory rates. Chan notes that “It is unlikely that this period of exercise would simulate all the physiologic alterations that may occur with struggle and agitation. In addition, we did not reproduce the effects of trauma and psychological stress that often occur with apprehended individuals.” [17].

5.3.2 | Animal model of restricted ventilation

A relevant animal model of ventilatory restriction without pressure on the thorax is provided by Pudiak and Bozarth [78] (see Figure 3).

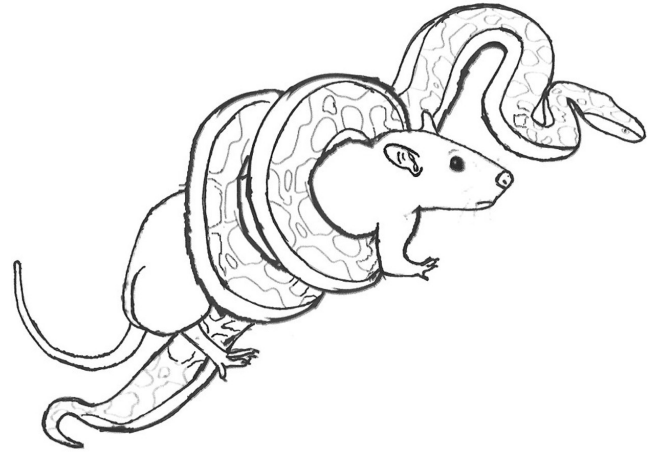


FIGURE 4 This cartoon of a rat in the coils of a boa constrictor snake depicts the animal model of decreased pulmonary perfusion (see Reference [85], Boback, et al.)

They restrained rats in cylinders which allowed some movement, but generally not the ability to turn around. Thus, the rats could breathe, but the confinement restricted chest expansion preventing deep breaths. When fed cocaine (increasing metabolic demand), 50% of the confined rats died compared to only 8% which were not confined.

5.3.3 | Decreased pulmonary perfusion

The prone position will also impair gas exchange through decreased pulmonary perfusion consequent to a decrease in venous return to the heart from pressure on: (1) the inferior vena cava (IVC), (2) the right atrium, and (3) the lungs (see Figure 1 and Table 6). Pressure on the abdomen will be transmitted posteriorly to the IVC, which has an internal pressure of 3–8 mmHg. Ho found large reductions in the diameters of the IVC in prone subjects [62]. Additionally, any decrease in anterior–posterior (AP) dimension from chest compression will result in pressure on the mediastinal structures, including the right atrium, which has an internal pressure of a mere 2–5 mmHg [56,68]. Finally, pressure on the thoraces will be transmitted to the lungs, where the pulmonary venous pressure is only 8 mmHg [56, 68]. The deficit in venous return may not be apparent until there is a greater demand for cardiac output.

The anesthesiology literature has documented decreased cardiac output of 18%–27% from reduced venous return in the prone position [79–81]. Noninvasive studies have shown prone position without a weight on the back can cause a decrease in cardiac output or cardiac index of 11% [82], with 25 kg on the back a decrease of 16% [83] and with 50 kg a decrease of 16% [84]. Roeggla noted a 37% decrease in cardiac output from hogtying alone [73].

5.3.4 | Animal model of decreased perfusion

A relevant animal model of pressure on the thorax was provided by Boback et al. [85] (see Figure 4). They studied the mechanism by

which boa constrictors kill their prey and found that it was not from hypoxic asphyxiation as traditionally thought. Rather, it was from cardiac arrest consequent to decreased venous return to the right heart from the thoracic pressure. Interestingly, in the constricted rats a significant metabolic and respiratory acidosis occurred, with pH dropping from 7.4 to 7.0 and $p\text{CO}_2$ rising from 40 to over 80mmHg. This was thought to be a result of tissue ischemia due to poor perfusion as well as from a decrease in ventilation. However, there was only a decrease in $p\text{O}_2$ from 130mmHg to a $p\text{O}_2$ of 89 mmHg. Thus, systemic hypoxemia was not a significant factor.

5.3.5 | Prone ventilation in disease states

The fact that recent studies have shown some benefit of prone positioning in certain hospitalized patients with acute respiratory distress syndrome and COVID-19 pulmonary infections should not cause confusion [86, 87]. Inflammation causes non-cardiogenic pulmonary edema which results in hypoxia. Prone positioning promotes alveolar recruitment in the dorsal portions of the lungs causing a more homogeneous distribution of ventilation. This along with a redistribution of perfusion and other mechanisms improves the ventilation-perfusion mismatch present in these patients. This mismatch does not exist in healthy people, and thus no benefit should be expected from prone positioning in them.

5.3.6 | Restraint

Restraint in the prone position means that subjects cannot extricate themselves from that prone position or increase their ventilatory capacity to compensate for the increased metabolic demands of the situation. Prolongation of this situation will result in continued carbon dioxide accumulation. Campbell has demonstrated that prone positioning results in increased work of breathing [88]. Moreover, hyperventilation requires substantial work, with the work of breathing increasing geometrically with the need. It will contribute to the hypercarbia and acidosis and at some point, the production of CO_2 exceeds the ability to eliminate CO_2 [89]. The resistive work of breathing is greatly increased with high airflow velocities due to turbulence [56, 68]. This work involves more rapid and stronger contractions of the muscles than normally involved in eupneic ventilation. Furthermore, the hyperventilation will require recruitment of accessory muscles. The accessory and abdominal muscles will fatigue with prolonged use.

5.3.7 | Air hunger

As noted above, hypercapnic metabolic acidosis generates a strong drive to hyperventilate. When an individual cannot satisfy this drive and adequately compensate for the acidosis, "air hunger" will result and explains the "I can't breathe" statements uttered in our Case 2

and other cases of arrest-related deaths [90–92]. Observers seeing someone who appears to be breathing hard and declaring they cannot breathe are likely to mistake the condition as hypoxia. Repeated vocalizations may, in fact, be evidence of the ability to ventilate, but the respiratory distress is from inadequate elimination of CO_2 , not inadequate intake of O_2 .

5.3.8 | Individual variation

The previously described studies also revealed a significant person-to-person variation, suggesting that some people may be more vulnerable to the effects of prone positioning than others. For example, Parkes reported one individual was found to manifest a 57% decrease in ventilation when prone [74]. Meredith tested eight individuals with COPD in prone position on a couch. Three could not tolerate the position due to deterioration in their clinical symptoms, while another five were able to stay prone with no significant change in ventilation capacity [93]. Even the San Diego group reported "certain outlier individuals" [17].

There is particular concern regarding obese individuals because of their greater body weight and larger abdominal fat pads that may cause pressure against the diaphragm and IVC [83], along with their poor physical fitness. In this regard, Han et al. [94] prospectively measured the intraabdominal pressures (IAPs) in three groups of patients undergoing spinal surgery: normal weight, overweight and obese. The IAPs were measured while they were being anesthetized in the supine position and then after they were rolled over prone onto Wilson Frames (the supporting frame at shoulder width). IAPs were essentially the same in the three groups while supine. But when they were rolled over onto the prone position, there was a difference in the IAPs, significantly higher in the overweight group as compared to the normal weight group and significantly higher in the obese group as compared to the overweight group. This occurred despite being placed prone on the Wilson frames. Thus, increased pressure on the diaphragm and IVC can be expected in obese individuals who are restrained in the prone position.

5.3.9 | First responder recognition of the danger of prone restraint

It is significant that since 2002 the National Association of EMS Physicians (NAEMSP) has had a position statement declaring restraint of agitated or combative patients in prone positions to be a prohibited technique. In 2021 this statement was joined by the National Association of State EMS Officials (NASEMSO), the National Association of Emergency Medical Technicians (NAEMT), and the American Paramedic Association (APA) [95]. Similarly, the International Association of Chiefs of Police (IACP) has directed departments to limit the use of prone restraint and to roll subjects onto their sides or to a sitting position as soon as possible to facilitate breathing [96].

5.4 | Cardiac arrest

Uncompensated metabolic acidosis may result in cardiac arrest. Therefore, the application of prone restraint to a person already in a physiologic state of increased metabolic demand and compensated metabolic acidosis may be catastrophic. This is not a matter of hypoxia because decreased ventilation has relatively little impact on arterial oxygen level. Rather it is due to an inability to hyperventilate and eliminate carbon dioxide (blow off CO₂) and thus an inability to correct the metabolic acidosis. This process can occur rather suddenly.

Hick et al. postulated that prone restraint may prevent compensatory respiratory alkalosis, with as little as 20% reduction in ventilatory capacity [43]. A 20% decrease in ventilation can result in a 25% increase in PaCO₂ and a decrease in pH by 0.1 [42].

Deaths have been reported during physical restraint of agitated individuals held in the prone (facedown) position, as detailed in case reports, case series, and inquests. In a recent review of the scientific literature, Strömmer et al. found physical restraint to be strongly correlated with death in subjects who were agitated and in excited states [39]. A Dutch study of 38 sudden deaths during restraint by police disclosed that 94.7% of the subjects were noted to have been in the prone body position, and 76.3% of subjects had been subjected to thoracic pressure [52].

5.5 | Cardiac rhythm

The evidence for prone restraint cardiac arrest is found in the initial electrocardiogram (ECG) readings recorded by the emergency medical services personnel. In these cases, the ECG does not show malignant ventricular cardiac arrhythmias, i.e., ventricular tachycardia or fibrillation, but rather a non-shockable rhythm, i.e., bradycardia, PEA, or asystole.

PEA and asystole generally occur from underlying non-cardiac etiologies, whereas a ventricular arrhythmia occurs from a primary cardiac etiology, such as myocardial ischemia, cardiomyopathy, or channelopathy. There is always an underlying cause for the PEA or asystole that constitutes a mechanism for the cardiac arrest. Specifically, when a clinician sees PEA or asystole, the clinician will consider the reversible conditions known as the "H's and Ts" (see Table 7) [97, 98]. Prominent among this group is metabolic acidosis, represented as an "H" for hydrogen ion. PEA or asystole generally indicates a non-cardiac basis of arrest, even in the presence of underlying cardiac pathology—although such conditions might contribute.

TABLE 7 The "H's and Ts" that cause PEA are listed here (References [97, 98])

5 H's	5 T's
1. Hypovolemia	1. Tension Pneumothorax
2. Hypoxia	2. Tamponade, cardiac
3. Hydrogen Ion (acidosis)	3. Toxins
4. Hypo/Hyperkalemia	4. Thrombosis (pulmonary)
5. Hypothermia	5. Thrombosis (coronary)

The "T" for thrombosis in the case of a coronary thrombosis (rather than for pulmonary thrombosis) is for the rare occlusive thrombus resulting in a sudden global myocardial infarction with secondary pump failure [99–101]. Rupture of the left ventricular free wall causing a cardiac tamponade or a posterior papillary muscle rupture with massive mitral valve incompetence are other causes of PEA secondary to coronary thrombosis and myocardial infarction [102]. In such cases of PEA, a 12-lead ECG might show signs of ischemia (e.g., ST-segment changes, T-wave changes, Q waves, or widening of the QRS complex), but regardless the underlying gross cardiac pathology will be obvious.

Dr. Jonathan Rich, a cardiologist testifying in the Derek Chauvin trial [103], declared that the George Floyd death was not of cardiac origin. Dr. W. Robert Graham, a cardiologist commenting after the trial [104], pointed out to the initial ECG recording showing PEA as a basis to dismiss the defense theories of cardiac causes of the death of George Floyd. Carotid body stimulation was a complicating consideration in that case due to the knee to the neck.

The survival of those with PEA and asystole is low (<5%) [105], and lower pH values are associated with lower rates of survival [106]. In PEA, the cardiac conduction system continues to function, but the heart fails to beat. The heart will cease contracting when the acidosis is sufficiently severe. It has been theorized that the strong decrease in cardiac contractility with acidosis may be related to potassium movement in and out of cardiac myocytes [107].

Steinberg in his review of 38 cases of prone restraint deaths from the peer-reviewed literature pointed out that most police in-custody deaths involving prone restraints have manifested either asystole (22/38) or PEA (13/38), with less than ten percent (3/38) manifesting an initial ventricular arrhythmia [42]. The predominance of asystole and PEA as the initial cardiac rhythm does not support cardiac mechanisms as the primary cause of death but is consistent with the concept of prone restraint cardiac arrest deaths. It should be recognized that the physiologic changes associated with prone restraint will lower the threshold for arrhythmias [108] and thus it is not surprising that some of these cases involve ventricular arrhythmias and may explain the other 10%.

In these situations, we are specifically discussing the initial ECG rhythm, as cardiac rhythms may convert into other forms. It is the initial rhythm that is diagnostic. Ventricular tachycardia and ventricular fibrillation will eventually deteriorate to asystole or PEA, but the median time noted for this transition is estimated at 31 min [109, 110]. At 12 min, 90% of these cases had not undergone degradation to asystole [111]. Resuscitation attempts may prolong the time to rhythm degeneration [112]. Thus, asystole or PEA as the presenting recording from the scene is, in fact, the likely initial rhythm.

5.6 | Other information

In addition to ECG recordings, other information may be gathered by emergency medical personnel when responding to such events to guide therapy. Pulse oximetry (photoplethysmography)

may be performed to determine oxygen saturation levels (SpO_2) and heart rate, as well as potentially other information [113–115]. Capnography, the graphical display of the end tidal partial pressure of carbon dioxide in mmHg in the airway (EtCO_2), may be performed to ensure proper airway placement, prevent resuscitative hyperventilation, and determine return of spontaneous circulation (ROSC) [116–119]. EtCO_2 correlates closely with cardiac output [119]. Quantitative capnometry uses infrared sensors to directly measure the carbon dioxide in the exhaled air as a direct correlate with blood CO_2 levels [119, 120]. Air flow sensors may directly assess ventilation [121]. Ultrasonography may directly assess heart function and may be important in recognition of pseudo-PEA, where the pulse is not detected [122].

Blood may also be drawn in the field or in the emergency department for either point-of-care (POC) testing or later laboratory analysis [123, 124]. Samples should be collected in air-tight containers or tested as soon as possible due to gaseous diffusion. In particular, pH, pO_2 , pCO_2 , lactic acid and pyruvate values would be of greatest value. As mentioned above, postmortem pH, particularly below 7.2, supports the diagnosis of acidosis [43]. When access is difficult, intraosseous samples have been shown to be comparable with venous samples for pH, bicarbonate, and base excess, and potentially for lactic acid [124, 125]. Many authors have shown in animal studies and in humans that arterial blood gases during cardiac arrest were shown to be misleading and that central venous or intraosseous samples were better [124, 126, 127].

5.7 | Our two cases exemplify PRCA

We presented two apparently healthy, but obese, individuals who were restrained in prone position and died suddenly. Both cases were videorecorded on police videocams. Both subjects were using stimulant drugs and were delirious. They were also both agitated, but neither extraordinarily so nor sufficiently so to be considered cases of excited delirium. In the first case, the subject physically struggled against the restraints and in the second case, the subject was involved in a minor struggle with the police, and so these cases involved physical exertion but not exhaustive physical exertion.

In both cases, their hands were cuffed behind their backs and their ankles were hobbled. In the first case, the subject was restrained in the prone position for approximately 10 min, with restrictive straps for approximately 7 of those 10 min. In the second case, the subject was restrained in the prone position for approximately 15 min, including $3\frac{1}{2}$ min sandwiched between a rigid board and the ground and with a significant weight applied on his back for a little over a minute. In neither case was pressure applied to the neck, and thus neither carotid body stimulation nor jugular venous obstruction was a consideration. In the second case, but not the first, the subject exclaimed that he could not breathe.

The initial cardiac rhythm on the ECG in the first case was sinus bradycardia, but pulses were found to be absent afterwards. The PEA deteriorated to asystole, before electrical activity was restored. Return of spontaneous circulation was achieved in the emergency room. The EMS report in that case noted that there was no indication of STEMI. The second case also manifested PEA; the ECG was not recorded and thus unavailable for review.

Although both subjects had cardiac pathology, it was not thought to be the cause of death in either case. We are unaware of any cardiac symptoms prior to the prone restraint in either case. The first subject was found to have a large and dilated heart, but the initial rhythm was bradycardia/PEA that indicated an underlying non-cardiac condition rather than a malignant ventricular arrhythmia expected of a cardiomyopathy, a myocardial infarction, or the stress of the event. An echocardiogram subsequent to the event documented normal heart function and ruled out clinically significant underlying cardiac pathology, such as cardiomyopathy. The second subject had cardiomegaly with left ventricular hypertrophy and focal severe coronary atherosclerosis, but again the initial PEA rhythm suggested a non-cardiac process. Neither showed the obvious acute cardiac pathology that could have resulted in PEA.

In Case #1, end tidal CO_2 monitoring of the subject in the ambulance revealed increased exhalation of CO_2 . Arterial blood gases were taken $1\frac{1}{2}$ hours after the cardiac arrest, when he was no longer restrained or in a prone position, and after the administration of Ringer's lactate and other medications. The testing revealed a profound acidosis (pH of 7.015) and hypercarbia (pCO_2 of 70.6 mmHg). The bicarbonate level was low consistent with acidosis. The pO_2 was elevated due to the administration of oxygen. He also had acute renal failure, hyperglycemia, and an elevated troponin as would be expected post cardiac arrest.

In Case #2, point-of-care blood gas testing on venous blood from the subject was collected approximately 33 min after the cardiac arrest, when he was no longer restrained or in a prone position, and after fluid resuscitation and epinephrine were administered. The venous blood revealed a profound acidosis (pH of 6.64) and hypercarbia (pCO_2 of 157 mmHg). The bicarbonate level was low, consistent with acidosis.

Autopsies were performed in both cases that failed to reveal significant trauma or obvious cause of death. Underlying cardiac pathology was found which may have contributed, but we do not believe were responsible for causing the deaths; see discussion above. In Case #1, evidence of recent ischemic injury from the cardiac arrest was found at autopsy—acute global brain ischemic injury and reperfusion injury of the heart. Neither case was thought to be a drug overdose; however, the stimulant drugs would have contributed to increased metabolic demand (but not to respiratory depression).

Both subjects would have had a high metabolic demand. Due to their prone restraint, they would have been unable to maximally ventilate and would have had a decreased venous return diminishing pulmonary perfusion. Thus, the subjects were unable to adequately compensate for their metabolic acidosis by exhaling sufficient

amounts of CO₂. These cases demonstrate the risks and dangerousness of prone restraint and serve as examples of PRCA.

6 | CONCLUSION

Deaths occur from atraumatic prone restraint as a likely complication of uncompensated metabolic acidosis rather than from hypoxia or underlying cardiac pathology. Prone restraint cardiac arrest provides a scientific explanation for these atraumatic police in-custody and arrest-related deaths. Since the vast majority of atraumatic arrest-related deaths involve prone restraint and present with pulseless electrical activity or asystole, prone restraint cardiac arrest may explain many, if not most, arrest-related deaths.

When approaching an arrest-related death, we recommend to forensic pathologists that venous blood gases (pH, pCO₂) be performed on an immediate STAT basis or more optimally as point-of-care testing. Forensic pathologists should study the EMS run sheets for the initial ECG rhythm, end tidal CO₂ values, and other EMS data, which may not be in the hospital charts. Police and EMS reports should be used to create a timeline of events (as in Figures 1 and 3), with an emphasis on the restraint used and position of the body. Any pressure on the side of neck should be noted as this may bring up other considerations. During the autopsy, the heart should be retained for cardiovascular pathology consultation.

CONFLICT OF INTEREST

The authors have no conflicts to report, except that A.S. is involved as a consultant in civil litigation involving ARD cases. The authors have received no financial support to produce this work.

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