Channa Pererra and Pollanen are not the first – nor, unfortunately, the last – “medical professionals” or “justice authorities” to erroneously blame Sickle Cell Trait for a restraint-related death, so as to absolve restrainers from suffering the responsibility for having caused an individual's death.[1,2,3]

In their “Sudden Death due to Sickle Cell Crisis During Law Enforcement Restraint” case study report[1], Channa Pererra and Pollanen (CP&P) failed to provide any kind of informational discussion of Sickle Cell Anemia (the “disease”), Sickle Cell TRAIT, the differences between the disease and the trait, or what kind of relationship sickle cell trait may (or may not) have to restraint-related deaths. Additionally, CP&P grossly neglected to provide critically important details regarding the manners and methods of restraint suffered by their case study subject – both before and during the event of his death.

**With this review, I intend to**

- Identify and Correct the FIRST of CP&P’s report deficiencies;
- Identify and Discuss the SECOND of CP&P’s report deficiencies;
- Clearly demonstrate how Channa Pererra and Pollanen FAILED to present anything even remotely-resembling a case study of “Sudden Death due to Sickle Cell Crisis [occurring] During Law Enforcement Restraint.”

**Sickle Cell Anemia** is a disease that is only transmitted via inheritance. To understand sickle cell anemia – and sickle cell trait – you first must understand the following facts:
An allele [pronounced “ah – LEEL”] is an “alternative form of a gene (one member of a pair) that is located at a specific position on a specific chromosome.”[4,5] To keep things simple, just think of an allele as half of a gene. One allele from your mother combines with one allele from your father, and that allele combination creates a gene you inherit.

There are two alleles important to whether you inherit “sickle cell anemia” or “sickle cell trait”: the A allele, and the S allele.[5,6]

“Individuals with two normal A alleles (AA) have normal hemoglobin, and therefore normal RBCs [Red Blood Cells]. Those with two mutant S alleles (SS) develop sickle cell anemia. Those who are heterozygous for the sickle cell allele [individuals who have one normal allele and one sickle cell allele] (AS) produce both normal and abnormal hemoglobin. Heterozygous individuals are usually healthy, but they may suffer some symptoms of sickle cell anemia under conditions of low blood oxygen, such as high elevation. Heterozygous (AS) individuals are said to be ‘carriers’ of the sickle cell trait.”[5]

Individuals who have sickle cell anemia – the “disease” – have received the mutant S allele from both their parents, thus they frequently suffer incidents of “vaso-occlusive sickle cell crisis.”[5,6]

“Under normal circumstances, your red blood cells are flexible and round, and they move easily through your blood vessels to carry oxygen to all parts of your body. In people with sickle cell anemia, the red blood cells become rigid and sticky and are shaped like sickles or crescent moons.”

“Periodic episodes of pain, called [‘vaso-occlusive sickle cell crisis’], are a major symptom of sickle cell anemia. Pain develops when sickle-shaped red blood cells block blood flow through tiny blood vessels to your chest, abdomen and joints. Pain can also occur in your bones. The pain may vary in intensity and can last for a few hours to a few weeks. Some people [who have sickle cell anemia] experience only a few episodes of pain. Others experience a dozen or more crises a year. If a crisis is severe enough, you may need hospitalization so that painkillers can be injected into your veins (intravenously).”[6]

Because most of them begin suffering signs or symptoms of the disease soon after reaching four months of age, individuals with both the mutant S alleles (SS) become diagnosed as having sickle cell anemia quite early in life.[6] Happily, if an individual who has sickle cell anemia is provided with emergency medical care in a timely manner when she/he suffers a “vaso-occlusive sickle cell crisis,” the sickle cell crisis episode does not have to be fatal.[6,7,8] Most often, someone with sickle cell anemia who suffers a sickle cell crisis “can be managed efficiently and quickly in a hospital’s emergency department with fluids and pain medicines.”[7]

Sickle cell “trait” has never been considered a “fatal condition” because those who only have the trait do not have the “disease.”[2,5,6,9-12] Individuals with sickle cell trait most
commonly live an entirely “normal” lifespan without ever suffering any form of sickle cell anemia-related signs or symptoms.[2,6] That is why individuals who only have the sickle cell trait frequently go “undiagnosed.” Thus, anyone with an “undiagnosed sickle cell trait” (such as Channa Pererra and Pollanen’s study subject) is very clearly someone who does not have a “condition that is fatal.”

Any circumstance or condition that causes an individual to suffer from hypoxia (a decreased amount of oxygen available to body tissues) is a circumstance or condition that will surely cause an individual with sickle cell anemia to suffer a “vaso-occlusive sickle cell crisis.”[6,7] For instance, a person with sickle cell anemia who simply drives up a mountain to a very high altitude, or flies in an unpressurized aircraft, will most likely suffer a sickle cell crisis when doing so.[5,6,7]

There are a variety of OTHER circumstances and conditions that may – all by themselves – cause an individual with sickle cell anemia to suffer a “vaso-occlusive sickle cell crisis”:[6,7]

- Dehydration
- Infection
- Fever
- Extremely exertive exercise
- Bleeding
- Exposure to extreme HEAT or COLD
- Drug and/or Alcohol use
- Pregnancy
- Stress
- Scuba Diving[10]

However! In order for someone who only has the sickle cell TRAIT – someone who does not have sickle cell anemia (the “disease”) – to be “triggered” to suffer a “vaso-occlusive sickle cell crisis,” one or more of the above-listed OTHER circumstances or conditions must ALSO be accompanied by a circumstance or condition that causes the individual to suffer from HYPOXIA.[5-7,9-12]

Postmortem findings of sickled red blood cells in the body of an individual with sickle cell anemia or sickle cell trait, do not – in any way – identify a “sickle cell crisis” as having caused that individual’s death! When an individual who has sickle cell anemia or sickle cell trait dies, the “agonal hypoxemia” that accompanies death due to any reason will cause previously un-sickled red blood cells to become sickled. Thus, sickled red blood cells found during a postmortem examination do not provide a concrete indication that red blood cell sickling occurred before death.
“... the presence of widespread intravascular sickling in post-mortem or in surgical specimens has no relationship to the amount (if any) of intravascular sickling that may have been present while the tissue(s) were living, for intravascular sickling may arise as an agonal event or after death as a result of tissue hypoxia.”[2,13]

“These were studied retrospectively by many groups with no definitive causal associations found, especially since agonal hypoxemia invariably causes intravascular sickling as an artifact.”[10]

“However, since sickling is known to occur postmortem, it remains controversial as to whether the pathogenesis of these exercise related deaths involves microvascular obstruction by sickled erythrocytes.”[12]

“The presence of generalized sickling at postmortem exam in the five patients reported does not constitute proof that intravascular sickling was pathogenic in the patient's death, for it is obvious that red cells from a patient with sickle trait will assume the sickled shape in the acidotic and hypoxic postmortem state.”[14]

In spite of that fact, in their case study discussion, Channa Pererra and Pollanen wrote:

Histological examination [postmortem] revealed widespread vaso-occlusive sickling of red blood cells in the brain, heart, liver, kidneys, adrenal glands, thyroid gland, intramyocardial coronary arteries, skeletal muscles, pancreas, testis and spleen (Fig. 1).

... Death was attributed to vaso-occlusive sickle cell crisis due to hypernatremic dehydration in a man with undiagnosed sickle cell trait.

Thereafter, CP&P failed to provide any kind of support for why they elected to consider their study subject's postmortem “widespread vaso-occlusive sickling of red blood cells” to concretely indicate that sickle cell crisis caused his death.

Again, it is not unusual for uneducated (or biased) individuals to erroneously attribute the death of an individual with sickle cell trait to sickle cell crisis, based solely upon the postmortem finding of sickled red blood cells.[2,3] However, when writing a case study report of this kind, responsible authors would sufficiently research the subject so as to become adequately educated. Upon discovering the cause-of-death attribution errors related to postmortem red blood cell sickling, responsible authors would discuss such a significant issue within their case study report.

By attributing their study subject’s death to sickle cell crisis, based solely upon postmortem sickled red blood cells – without offering any legitimate discussion of why they
felt “justified” for doing so – Channa Pererra and Pollanen have inarguably demonstrated that:

- they are terrifically uneducated and irresponsible reporters;
- **OR**
- they are incredibly BIASED reporters.

In their case study discussion, CP&P reasonably established that their subject was most likely suffering from dehydration prior to the physical exertion he experienced while struggling with – and being restrained by – police and EMS personnel. And, twice during their case study discussion, CP&P offered a description of the several “circumstances” they (apparently/allegedly) believed to have triggered their case study subject to suffer a “vaso-occlusive sickle cell crisis” that “caused” his death:

Nevertheless, under unusual circumstances such as dehydration, hypoxia, acidosis and physical exertion serious morbidity or mortality can result from complications related to polymerisation of deoxy-hemoglobin S.

Dehydration, hypoxia, acidosis and physical exertion are known aggravating factor[sic] for sudden death in sickle-cell trait due to vaso-occlusive sickle cell crisis.

I agree with CP&P; one or more episodes of hypoxia are absolutely required in order for someone with sickle cell trait to suffer a sickle cell crisis. Yet, nowhere in their report did Channa Pererra and Pollanen indicate WHAT happened to cause their case study subject to suffer one or more incidents of HYPOXIA.

Based upon my years of experience investigating restraint-related deaths, I believe that one or more incidents of HYPOXIA occurred during the manners and methods of RESTRAINT suffered by Channa Pererra and Pollanen’s case study subject. However, because CP&P grossly neglected to provide any of the critically important details related to the manners and methods of restraint suffered by their case study subject prior to (and at the time of) his death, my opinion cannot be substantiated – nor can it be unsubstantiated!

When investigating a death that is – in any way – related to an incident involving restraint, a responsible and unbiased medical examiner or forensic pathologist will obtain a detailed description of the manners and methods of restraint that were employed, prior to providing an opinion as to the “cause” of an individual’s death.[15-19]

“The questions asked need to have enough specificity to elicit answers to questions about how long the subject was restrained; in what positions he was
restrained; how much weight was applied to torso areas; how the subject reacted and verbalized; when the subject stopped moving, breathing, or talking; and whether he demonstrated clear signs of consciousness or life after the restraint process was completed or terminated.”[18]

“The description should include: (a) what type of restraint, (b) what period of time, (c) position of victim when the restraint was applied and during the restraint period, the final resting position, (d) any use of arms, shackles, handcuffs, flexcuffs, choke hold, the use of taser, pepper spray or any additional restraints, such as hogtying.”[19]

When investigating a restraint-related death – especially for the purpose of writing and publishing a case study of same – a responsible and unbiased reporter will similarly obtain and provide a detailed description of the manners and methods of restraint that were employed prior to providing an opinion as to the “cause of death.”

Below are the ONLY descriptions Channa Pererra and Pollanen offered in relationship to the manners and methods of restraint suffered by their case study subject prior to, and during, the moment of his death:

At the time of arrival of police and ambulance, he was conscious, alert and combative. He was then restrained by handcuffing and transferred to the ambulance in a supine position with a semi-agitated state.

He was given two separate intramuscular doses of 2 mg of midazolam while in the ambulance en route to the hospital. While he was supine on the stretcher he became unresponsive, and had no vital signs when brought into the emergency room.

That’s IT!!! That’s the ENTIRETY of the restraint information provided by CP&P!

Channa Pererra and Pollanen entirely failed to provide even a “sketchy” description of the manner or method of restraint employed

- by the police while “handcuffing” the subject.
- by the police while maintaining “control” of the subject prior to (and during) EMS personnel transferring him to a supine-restrained position on their stretcher.
- by EMS personnel to keep the subject restrained to their stretcher in a “supine” position.
IN OTHER WORDS, Channa Pererra and Pollanen entirely failed to RULE OUT an episode of hypoxia being suffered by their case study subject due to the manner or method of restraint that was employed:

- while he was being “handcuffed” by police.
- after handcuffing, while he was being “controlled” by police prior to, and during, the time that EMS personnel transferred him to a supine-restrained position on their stretcher.
- by EMS personnel to supinely restrain him to their stretcher – the manner and method of restraint he was being subjected to at the time of his DEATH!

[“He was given two separate intramuscular doses of 2 mg of midazolam while in the ambulance en route to the hospital. While he was [restrained] supine on the [EMS] stretcher he became unresponsive, and had no vital signs when brought into the emergency room.”]

[BTW: Common side effects of midazolam injection include “difficulty breathing, wheezing,” as well as other benzodiazepine-like respiratory suppression effects. But, because CP&P entirely failed to rule out an asphyxial form of supine restraint having been employed at the time of their study subject receiving the two midazolam injections – or at the time of his death – one cannot draw any concrete conclusions as to whether or not the midazolam injections contributed to one or more hypoxic events, or contributed to the ultimate cause of the case study subject’s death.]

Clearly, Channa Pererra and Pollanen grossly neglected to provide anything even remotely-resembling “details” related to the critically important manners and methods of restraint inflicted upon their case study subject prior to – and at the time of – his death in their “Sudden Death due to Sickle Cell Crisis During Law Enforcement Restraint” case study report. Thus, one cannot help but wonder how such a deficient case study report could have “passed” a “peer review” and become published!

At the end of their case study report’s “discussion” (just prior to their “In summary” paragraph), Channa Pererra and Pollanen offered the following “conclusion” based on the incredibly inadequate information they provided in their case study report:

**Therefore, we can conclude that dehydration likely triggered vaso-occlusive sickle cell crisis with the subsequent violent outburst and/or the physical exertion during restraint possibly contributing to the sickling process.**

Apparently, CP&P assumed that no one would check the reference citations they offered in “support” of their incredibly erroneous “conclusion.” Granted, few people *would check*
them! But, because I am a “responsible” and educated reviewer, I did!

Upon examining the FOUR references CP&P cited in support of their case study discussion’s incredibly erroneous “conclusion,” I was not at all surprised to discover that NOT ONE of them supports it![16]
The “WORST” aspect of Channa Perera & Pollanen’s case review report:

These authors had an opportunity to present this case study in a manner that would promote the requirement that all restrainers be trained in employment of SAFE restraint methods, prior to being allowed to restrain someone – a manner that would contribute to the prevention of children and adults suffering restraint-related deaths.

Instead, Channa Perera & Pollanen have quite clearly demonstrated their membership in the ranks of dishonorable and despicable individuals seeking ONLY to absolve restrainers who employ unsafe methods of restraint from being held responsible for killing children and adults.

SHAME on Channa Perera & Pollanen!

CHAS’ REVIEW REFERENCES


   The Sickle Cell Society Website Tables created from the information published in “Sickle cell anaemia and deaths in custody in the UK and the USA.” and posted at: http://www.sicklecellsociety.org/DeathUxpl.htm

(3) Wikipedia, the free encyclopedia. Martin Anderson http://en.wikipedia.org/wiki/Martin_Anderson"

(4) http://biology.about.com/od/geneticsglossary/g/alleles.htm


   http://www.emedicinehealth.com/articles/6504-1.asp#


