A Report of Four REFERENCES Cited Specifically To Support The “CONCLUSION” offered in Channa Perera and Pollanen’s case study report;
Sudden death due to sickle cell crisis during law enforcement restraint.

*J of Forensic Legal Med* V 14, Issue 5 (July 2007); pgs 297–300.

By Ms. Charly D. Miller (CHAS).

The discussion section of Channa Perera and Pollanen’s case study report ended with:

“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.11–14”

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**CP&P’s reference citation #11:**


**Exertional collapse and sudden death associated with sickle cell trait.**

**Kerle KK, Nishimura KD.**

Martin Army Community Hospital, Fort Benning, Georgia, USA.

Although rare, exertional collapse and sudden death are the most serious potential complications of sickle cell trait. Studies suggest that this condition may occur in susceptible persons when poor physical conditioning, dehydration, heat stress or hypoxic states precipitate sickling of the abnormal erythrocytes. Sickling leads to endothelial damage, which can cause vasoconstriction, disseminated intravascular coagulation and local tissue damage. Cardiac effects include acute ischemia and arrhythmias. Muscle damage results in acute compartment syndromes and release of myoglobin into the circulation. Acute renal failure is possible. Diagnosis is based on a high index of suspicion, and characteristic presentation and laboratory findings, including myoglobinuria, hyperkalemia, hypocalcemia, hyperphosphatemia and elevated creatine kinase levels. The differential diagnosis includes pulmonary embolism, acute cardiac events, anaphylaxis and heat stroke. Management is based on stabilization, rehydration, and the treatment and prevention of complications.

Publication Types:  **Review**

PMID: 8677839 [PubMed - indexed for MEDLINE]

**CHAS NOTE:** This publication was a “REVIEW” of studies and case reports published prior to 1996. Considering the fact that the studies and case reports published prior to 1996 only involved sickled cells discovered postmortem, it is highly unlikely that this review presented any evidence of an individual with sickle cell trait dying due to vaso-occlusive sickle cell crisis caused solely by dehydration and physical exertion.

**CP&P’s reference citation #11 does not support their case study’s “conclusion.”**

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**CP&P’s reference citation #12:**
CHAS NOTE: This reference also happens to be the one cited as reference (12) for my REVIEW of CP&P’s case study report. This reference’s full text is posted in the Restraint Asphyxia Library: http://www.charlydmiller.com/LIB09/2001MarchSickleCellTraitPhysicalExertion.pdf

Here is an important quote from that reference, directly related to CP&P’s case study report “conclusion”: “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.”

CP&P’s reference citation #12 does not support their case study’s “conclusion.”

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CP&P’s reference citation #13:

Exercise-induced death in sickle cell trait: role of aging, training, and deconditioning.

Clinical Sciences
Medicine & Science in Sports & Exercise. 28(5):541-544, May 1996.
GALLAIS, DANIEL LE; BILE, ALPHONSE; MERCIER, JACQUES; PASCHEL, MARC; TONELLOT, JEAN LOUIS; DAUVERCHAIN, JEAN

Abstract:
The pathophysiological process of exercise-induced death in subjects with sickle cell trait (SCT) remains unclear. Concerning the cause of death, authors have suggested stressful environmental conditions such as altitude, heat and humidity, or abnormal patient conditions such as deconditioning, fatigue, and disease. These conditions are thought to lead to hypoxemia, hyperlactatemia, acidosis, dehydration, hyperthermia, or exercise-induced rhabdomyolysis, all of which may initiate sickle cell crisis, disseminated intravascular coagulation, myoglobinuria, and renal failure.

We report the case of a 41-yr-old, healthy, and apparently well-conditioned subject with SCT who died during a cross-country race under normal environmental conditions in good weather (in terms of temperature and humidity). The medical and athletic history of the subject were unremarkable. We refer to an epidemiological study that reported a relation between age and exercise-induced sudden death in subjects with SCT. We then review the pathophysiological effects of aging in association with deconditioning and high-level training reported in the literature, particularly the decrease in aerobic metabolism in deconditioned subjects, and the exercise-induced hypoxemia in highly trained subjects. We discuss the consequences of deconditioning and high-level training in subjects with SCT during exercise, and conclude that these factors may be involved in the age-dependent risk of exercise-related sudden death in subjects with SCT.

CHAS NOTE: This 1996 case study report discussed a “subject with SCT who died during a cross-country race.” Thus, this case study involved sickled cells discovered postmortem. Therefore, this case study report did not provide any evidence of an individual with sickle cell trait dying due to vaso-occlusive sickle cell crisis caused solely by dehydration and physical exertion.

CP&P’s reference citation #13 does not support their case study’s “conclusion.”
CP&P's reference citation #14:

ORIGINAL ARTICLE

Sickle-cell trait as a risk factor for sudden death in physical training
JA Kark, DM Posey, HR Schumacher, and CJ Ruehle

Abstract

Case reports of sudden death during exertion have not established an association between the sickle-cell trait (hemoglobin AS) and exercise-related death. To test this association, all deaths occurring among 2 million enlisted recruits during basic training in the U.S. Armed Forces in 1977 to 1981 were classified from autopsy and clinical records as non-sudden deaths or as sudden deaths explained or unexplained by preexisting disease. On the basis of known numbers of entering recruits (according to race, age, and sex) and published prevalence rates for hemoglobin AS (8 percent for black and 0.08 percent for nonblack recruits), death rates (per 100,000) were 32.2 for sudden unexplained deaths, 2.7 for sudden explained deaths, and 0 for non-sudden deaths among black recruits with hemoglobin AS, as compared with 1.2, 1.2, and 0.7 among black recruits without hemoglobin S and 0.7, 0.5, and 1.1 among nonblack recruits without hemoglobin S. Among black recruits the relative risk of sudden unexplained death (hemoglobin AS vs. non-hemoglobin S) was 27.6 (95 percent confidence interval, 9 to 100; P less than 0.001), whereas among all recruits this risk was 39.8 (95 percent confidence interval, 17 to 90; P less than 0.001). The relative risk of sudden unexplained death among all recruits increased with age (P less than 0.04), from 13 (ages 17 to 18) to 95 (ages 26 to 30). We conclude that recruits in basic training with the sickle-cell trait have a substantially increased, age-dependent risk of exercise-related sudden death unexplained by any known preexisting cause.

CHAS NOTE: Again, studying the autopsy records of individuals with sickle cell trait who died during basic training does not contribute to establishing “an association between the sickle-cell trait (hemoglobin AS) and exercise-related death.” However, Kark et al probably were able to establish that “recruits in basic training with the sickle-cell trait have a substantially increased, age-dependent risk of exercise-related sudden death unexplained by any known preexisting cause.” Still, such a finding does not provide any evidence of an individual with sickle cell trait dying due to vaso-occlusive sickle cell crisis caused solely by dehydration and physical exertion.

CP&P’s reference citation #14 does not support their case study’s “conclusion.”

SUMMARY: NOT ONE of the FOUR reference citations offered in support of Channa Perera and Pollanen’s case study “conclusion” provide any support for same.