



Office of Chief Medical Examiner
Tarrant County Medical Examiner's District
Tarrant County, Texas
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AMENDED AUTOPSY REPORT

CASE NO: 1913146

Name: MILLER, Robert Geron

Age: 38 years

Sex: Male

Race: Black

Date and Time of Death: August 1, 2019 at 5:45 AM

Place of Death: 1575 S. Main Street, Fort Worth, Texas 76104 (John Peter Smith Hospital)

Autopsy Authorized By: Statute 49.25 of Texas Criminal Code

I, Richard C. Fries, D.O., hereby certify that I performed a complete autopsy on the body of ROBERT GERON MILLER at the Tarrant County Medical Examiner's District Morgue in Fort Worth, Texas on the 2nd day of August 2019, beginning at 8:00 AM, and upon investigation of the essential facts concerning the circumstances of the death and history of the case as known to me at this time, I am of the opinion that the findings, cause and manner of death are as follows:

FINDINGS:

- I) Investigative findings:
 - A. Decedent arrested at 10:13 a.m. on 7/31/19; noted to be agitated and was escorted to patrol car, noted to be combative during transport with kicking and spitting.
 - B. Arrived at police department holding facility at approximately 10:25 a.m.
 1. Video of the cell shows him to be initially agitated, moving around the room, gesturing, yelling, and repeatedly pounding on the windows and kicking the door. He is then seen to be sitting, standing, laying and using the restroom until transferred at 2:00 p.m.
 2. Escorted to court at 11:53 a.m., noted to be walking on return.
 3. A cup of water is provided at 12:04 p.m., which he immediately drinks.
 4. At 12:51 p.m. he attempts to drink again from the apparently empty cup.
 - C. Booked into jail at 2:15 p.m.
 1. He became combative in the change out room during booking process.
 2. Reported to have struggled with officers during which he lifted two officers on him off the ground.

TPA

3. Pepper spray deployed three times before he complied with procedure.
 4. Moved to decontamination at 2:20 p.m. for 10 minutes with cold running water decontamination .
 5. Nurse evaluated following decontamination and notes he reported right knee pain, with no visible injury. Also reports chest pain/shortness of breath; however was verbal without conversational dyspnea (difficulty breathing). Additionally reported headache. He then became agitated which prevented further evaluation.
 6. Decedent walked to housing unit at 2:42 p.m.
 7. Upon entering unit, decedent refused to walk; carried remaining distance.
 8. Placed, by himself, on floor of cell.
 9. Cell door was secured and he was noted to get up and drink water
 10. At 3:10 p.m. officer notes he is on the floor splashing water from the toilet on himself. Officer asked if he needed medical attention and he only responded by shaking his head no.
 11. Check log states:
 - a. Moved at 2:52 p.m.
 - b. At sink at 3:02 p.m.
 - c. On back at 3:12 p.m.
 - d. Medical attention called at 3:22 p.m.
 12. Medical personnel responded to cell with crash cart at 3:25 p.m., noted no breathing or pulse and begin resuscitation
 13. Emergency services called at 3:29 p.m.
 14. Emergency services takes over resuscitation at 3:32 p.m., notes low blood pressure and hypoglycemia (glucose 48); transports to hospital.
- D. Hospital arrival approximately 4:15 p.m.
1. Hospital diagnoses: cardiac arrest, acute respiratory failure, anoxic-ischemic encephalopathy, shock, acute kidney injury, refractory hyperkalemia, anemia, and shock liver
 2. Head CT:
 - a. no acute intracranial abnormality.
 - b. no acute fracture or traumatic malalignment of the cervical spine
 3. Chest CT:
 - a. Right second through fifth anterior rib fractures and left second, fourth, and sixth anterior rib fracture, likely secondary to CPR.



- b. Dependent areas of ground glass opacity and consolidation are concerning for aspiration. Pulmonary contusion is possible, though this appearance would be somewhat atypical.
 - c. Multiple findings and may be related to hypoperfusion and/or the administration of epinephrine during recent code. There is nonenhancement of the spleen, kidneys, and pancreas, despite apparent good opacification of the major venous structures. Arterial structures are diffusely small including the common carotid and subclavian arteries as well as the infrarenal abdominal aorta. This is of unknown etiology but could be secondary to arterial spasm.
 - d. Small bowel findings suggestive of ileus.
- 4. Urine toxicology screen negative
 - 5. Laboratory tests include Hgb 6.2 g/dL; PT 32.8; PTT 94.9; troponin I 0.270 and 1.640 ng/mL; potassium 5.7, 6.4, 7.2 mmol/L
 - 6. New swelling of neck noted at 5:25 a.m. on 8/1/2019
 - 7. Received red blood cell transfusions during hospitalization for anemia
 - 8. No source of blood loss identified
- E. Past medical history of asthma, bipolar disorder, and polysubstance abuse
- II) Autopsy examination findings:
- A. Sickled red blood cells stacked together with blood vessel occlusion, identified microscopically in multiple organs
 - B. Cerebral edema
 - C. Pulmonary congestion
 - D. No asthma changes of the lungs
 - E. Intact hyoid bone and laryngeal cartilages
 - F. No petechiae of the conjunctivae or oral mucosa
 - G. No external neck trauma
 - H. Soft tissue hemorrhage of chest and neck, consistent with resuscitation
 - I. Hemorrhage of the gastric mucosa
 - J. Abrasions of wrists, consistent with restraints
 - K. Superficial laceration of forehead, closed with adhesive
 - L. Focal subgaleal hemorrhage
 - M. Superficial abrasions of the left arm, right knee, right leg and left leg
 - N. Decayed right upper molar, tooth #2
 - O. Medical therapy
 - P. Early decomposition changes
- III) Blood cultures: growth of group B Streptococcus and Klebsiella aerogenes
- IV) Anthropology report: no trauma

- V) Novel psychoactive testing: none detected
- VI) Toxicology:
- A. Femoral blood:
 - 1. Negative for ethanol
 - B. Heart blood:
 - 1. Positive for ibuprofen and lidocaine
 - C. Hospital serum (collected 7/31/2019 @ 2045 hrs):
 - 1. Negative for ethanol
 - D. Hospital serum (collected 7/31/2019 @ 2104 hrs):
 - 1. Positive for lidocaine
- VII) Genetic testing: Sickle cell trait (Heterozygous Hb S mutation detected)

CAUSE OF DEATH: SICKLE CELL CRISIS

MANNER OF DEATH: UNDETERMINED


Signature

**Richard C. Fries, D.O.
Deputy Medical Examiner**

Comment: Robert Miller was arrested on 10:13 am on 7/31/2019. After being placed in the back of the police patrol vehicle Mr. Miller was noted to repeatedly kick the back of the vehicle such that after he was placed in jail custody the officer noted that "the door no longer lined up with the body of the patrol vehicle". Video of the holding facility shows him agitated, pounding on the windows and kicking the door repeatedly. Once transferred to the Jail facility Mr. Miller reportedly became combative in the change out room. No video is available of the encounter in the change out room. It is reported "As the officers were on top of him, Inmate Miller pushed his body as well as the two officers on top of him off of the ground. After his continual struggle and failure to comply with office's orders to stop resisting, Inmate Miller was eventually sprayed three times with Oleoresin Capsicum". Mr. Miller was then transferred to decontamination, where he was placed under cold running water for 10 minutes, per jail protocol. He was then examined by a nurse who noted he reported right knee pain, chest pain/shortness of breath, and headache. The nurse then reported that he became agitated precluding further evaluation. After this Mr. Miller is documented on video recording walking with an escort to the housing unit. Mr. Miller is observed by detention officers moving in his cell before being found unresponsive on the subsequent check at approximately 3:22 PM. Emergency Services is contacted at 3:32 PM and transports the patient to the hospital. Mr. Miller arrived at the hospital at approximately 4:15 pm on 7/31/2019. Initial assessment on the ICU admission H&P lists:

- a) Cardiac arrest
- b) Anoxic ischemic encephalopathy
- c) Hyperkalemia

- d) Normocytic anemia
- e) AKI (acute kidney injury)

Following evaluation and hospitalization Mr. Miller was subsequently pronounced on 8/1/2019 @ 5:45 am (approximately 13.5 hrs. after admission). The ICU doctor notes "In spite of bicarb, insulin drip, kayxellate he continued to have hyperkalemia and refractory acidosis", "no explanation as to what caused the initial cardiac arrest. Final diagnosis

Refractory shock
 Acute Renal Failure
 Refractory hyperkalemia
 Anoxic Ischemic Encephalopathy "

The following documented medical findings that are pertinent to the reviewing physicians include:

- Head CT: no acute intracranial abnormality and no acute fracture or traumatic malalignment of the cervical spine
- Chest CT: rib fractures secondary to CPR and multiple findings of hypoperfusion
- Urine Toxicology screen is negative
- No temperature recorded on admission, "euthermia protocol" listed (targeted temperature control)
- Anemia
 - o Hemoglobin: 8.2 g/dL @4:25pm(POC), 8.5 g/dL @4:445pm (POC), 7.1 g/dL @5:36 pm, 7.1 g/dL @8:14 pm, 6.8 g/dL @8:25pm (POC), 6.2 g/dL @9:04 pm
 - o Review of previous admission shows a baseline hemoglobin of 14 g/dL
 - o No source of bleeding identified
 - o Transfused red blood cell units; 1 unit@ 9:40 pm and 1 unit @ 10:14 pm
 - o RBC morphology: Moderate Poikilocytosis (poikilocytes are abnormally shaped blood cells, such as sickle shaped red blood cells, this finding is not further characterized in the medical record)
- Hyperkalemia
 - o Potassium: 5.7 mmol/L @4:25 pm, 7.2 mmol/L @5:36 pm, 6.4 mmol/L @8:25 pm, 6.4 mmol/L @ 8:45 pm
- Coagulopathy
 - o PTT: 68.8 @5:36 pm, 94.9 @ 9:04 pm
 - o PT: 17.4 @5:36 pm, 32.8 @9:04 pm
 - o INR: 1.54 @5:36 pm, 2.9 @9:04 pm
 - o Platelet count: 181 @ 5:36 pm, 121 @9:04 pm
 - o Fibrinogen and D-dimer were not ordered/reported
- Possible Rhabdomyolysis
 - o Creatinine: 1.3 mg/dL @4:25 pm, 1.7 mg/dL @ 5:36 pm, 2.0 @ 8:25 pm
 - o Creatinine can take up to 12 hours to begin increasing and peaks at 24-72 hrs (3)
 - o Myoglobin not ordered/reported
 - o Hyperkalemia present
 - o Metabolic acidosis with anion gap present
- Dehydration
 - o Not definitively demonstrated on review of laboratory results
 - o Fluid intake of 2975.86 ml and output of 10 ml reported @ 11:29 pm

Initial reporting of the circumstances leading up to the death of Mr. Miller was very concerning for an acute asthma attack resulting from the application of pepper spray to an individual with a medical history of asthma. Postmortem findings for asthma classically can include gross hyperinflation of the lungs and mucus plugging of the medium and small airways. Microscopic evaluation can show mucus plugging,

Charcot Leyden crystals, basement membrane thickening, goblet cell hyperplasia, smooth muscle hyperplasia, and prominent eosinophilic inflammatory infiltration. Grossly, hyperinflation was not identified on autopsy. Microscopy of Mr. Miller's airways showed focal mild mixed inflammation and abundant bacterial overgrowth. He does not have microscopic findings to support a diagnosis of acute asthma or reactive airway disease.

Sickled red blood cells are identified stacked together with occlusion of the blood vessel lumens of multiple organs on the postmortem microscopic examination despite fluid resuscitation and blood transfusion. The hospital findings of anemia without an identifiable source of blood loss and poikilocytosis (abnormally shaped blood cells) supports that the sickling of his red blood cells occurred prior to death. Subsequent genetic testing confirmed that he does have sickle cell trait (heterozygous for Hemoglobin S).

Superficial cutaneous injuries are identified on autopsy. No significant traumatic injury that would explain Mr. Miller's death was identified during his hospitalization or on autopsy.

Sickle crisis associated with sickle cell trait though uncommon is a well-documented phenomena increasingly reported in the medical literature and recognized by the Centers for Disease Control (CDC), the College of American Pathologists (CAP), the American College of Sports Medicine (ACSM), and the National Athletic Trainers' Association (NATA). The Death of Dale Lloyd II in 2006 from exertional rhabdomyolysis associated with sickle cell trait led to the NCAA recommendation for sickle cell trait testing in college athletics. In 2007 NFL professional football player Ryan Clark suffered a splenic infarct from sickle cell trait during a game that resulted in a surgical splenectomy, ending his season. Further studies have demonstrated a disturbing frequency of sickle crisis in sickle trait including a 10-year retrospective study showing it was the 3rd leading cause of nontraumatic death in high school and college athletes (9), even more than asthma. Sickle cell trait associated deaths was the leading cause of death during conditioning training for NCAA division I football for the 10 years from 2000 to 2010 (26). The number of deaths due to sickle cell trait in college athletics has also shown a significant decrease since the requirement for testing was instituted (40). Since the original study showing increased deaths in military recruits with sickle trait other cases have continued to be identified in the military (8,10,22,29,36). Multiple cases and case series are reported of sickle cell trait deaths associated with exertion during law enforcement encounters and arrests (13,19,25,27).

The pathogenesis of exertion related deaths associated with sickle cell trait is not well understood and is a subject of ongoing research. However, these deaths appear to share many features. The symptoms begin following intense exertion that can be as short as a few minutes. High levels of exertion increase oxygen demand resulting in a hypoxic state, dehydration, acidosis, higher tissue temperatures and dehydration of red blood cells, resulting in intravascular sickling of the red blood cells with vascular occlusion. Clinically, these patients frequently, though not always, exhibit rhabdomyolysis, hyperthermia, renal failure, disseminated intravascular coagulopathy (DIC), metabolic acidosis, and refractory hyperkalemia. One paper reported that evidence of both heat stroke and rhabdomyolysis is absent in about one third of cases, however profound hyperkalemia is frequently present (30). It is not clear why only some individuals with sickle cell trait are affected by exertion in this manner. Some of the risk factors identified include dehydration, hotter climate, fatigue, poor physical conditioning, and age.

In this case Mr. Miller exerted himself repeatedly on 7/31/2019. After being placed in the back of the police patrol vehicle Mr. Miller was noted to repeatedly kick the back of the vehicle such that after he was placed in jail custody the officer noted that "the door no longer lined up with the body of the patrol vehicle". Video surveillance footage demonstrates Mr. Miller pounding on the window of a holding room while in custody. Additionally, once he was transferred to the Jail facility Mr. Miller reportedly became combative in the change out room. It was reported "As the officers were on top of him, Inmate Miller pushed his body as well as the two officers on top of him off of the ground. After his continual struggle and failure to comply with officer's orders to stop resisting, Inmate Miller was eventually sprayed three times with Oleoresin Capsicum" (pepper spray). This encounter occurred during the summer in Texas

(hotter climate), the circumstance (homelessness and prolonged encounter with minimal hydration) and clinical findings (almost 3 liters of fluids provided in hospital without significant urine output or signs of fluid overload present on exam) are concerning for dehydration. He is overweight (BMI 28.1, consistent with poor physical conditioning), and he is 38 years old.

In summary, Mr. Miller's autopsy demonstrates no significant life-threatening trauma and no evidence of acute asthma. He does have sickled red blood cells, the hospital records demonstrate most of the clinical symptoms of sickle crisis in sickle trait, and he has several identified risk factors associated with sickle cell crisis in sickle cell trait. Therefore the cause of death was classified as sickle cell crisis.

Because officers were physically engaged with Mr. Miller in the last encounter in which he exerted himself and it is not feasible to determine which episode of exertion or if in fact the combination of the events resulted in sufficient exertion to initiate the process that resulted in his death, the manner of death in this case is best reclassified as "Undetermined".

Literature and citations:

1. CDC:
 - a. <https://www.cdc.gov/ncbddd/sicklecell/traits.html>
 - b. <https://www.cdc.gov/ncbddd/sicklecell/documents/sickle-cell-athletes.pdf>
 - c. <https://www.cdc.gov/ncbddd/sicklecell/documents/sickle-cell-coaches.pdf>
 - d. <https://www.cdc.gov/ncbddd/sicklecell/documents/sickle-cell-doctors.pdf>
2. CAP: <https://health.usf.edu/-/media/Files/Medicine/Orthopaedic/Sickle-Cell/SickleCellTraitCAP.ashx?la=en&hash=9400C4B40063DDDD94DEFB41C6BD454A13870EBB>
3. ACSM: <https://www.ncaa.org/sports/2013/12/18/acsm-and-ncaa-joint-statement-sickle-cell-trait-and-exercise.aspx>
4. NATA: <https://www.nata.org/sites/default/files/sicklecelltraitandtheathlete.pdf>
5. Jones SR, Binder RA, Donowho EM Jr. Sudden death in sickle-cell trait. N Engl J Med. 1970 Feb 5;282(6):323-5. doi: 10.1056/NEJM197002052820607. PMID: 5410817.
6. Kark JA, Posey DM, Schumacher HR, Ruehle CJ. Sickle-cell trait as a risk factor for sudden death in physical training. N Engl J Med. 1987 Sep 24;317(13):781-7. doi: 10.1056/NEJM198709243171301. PMID: 3627196.
7. Eichner ER. Sickle Cell Trait, Heroic Exercise, and Fatal Collapse. Phys Sportsmed. 1993 Jul;21(7):51-64. doi: 10.1080/00913847.1993.11710400. PMID: 27424860.
8. Gardner JW, Kark JA. Fatal rhabdomyolysis presenting as mild heat illness in military training. Mil Med. 1994 Feb;159(2):160-3. PMID: 8202248
9. Van Camp SP, Bloor CM, Mueller FO, Cantu RC, Olson HG. Nontraumatic sports death in high school and college athletes. Med Sci Sports Exerc. 1995 May;27(5):641-7. PMID: 7674867.

10. Murray MJ, Evans P. Sudden exertional death in a soldier with sickle cell trait. *Mil Med.* 1996 May;161(5):303-5. PMID: 8855065
11. Kerle KK, Nishimura KD. Exertional collapse and sudden death associated with sickle cell trait. *Am Fam Physician.* 1996 Jul;54(1):237-40. PMID: 8677839.
12. Le Gallais D, Bile A, Mercier J, Paschel M, Tonellot JL, Dauverchain J. Exercise-induced death in sickle cell trait: role of aging, training, and deconditioning. *Med Sci Sports Exerc.* 1996 May;28(5):541-4. doi: 10.1097/00005768-199605000-00001. PMID: 9148081.
13. Thogmartin JR. Sudden death in police pursuit. *J Forensic Sci.* 1998;43:1228-1231.
14. Wirthwein DP, Spotswood SD, Barnard JJ, Prahlow JA. Death due to microvascular occlusion in sickle-cell trait following physical exertion. *J Forensic Sci.* 2001 Mar;46(2):399-401. PMID: 11305451
15. Pretzlaff RK. Death of an adolescent athlete with sickle cell trait caused by exertional heat stroke. *Pediatr Crit Care Med.* 2002 Jul;3(3):308-310. doi: 10.1097/00130478-200207000-00023. PMID: 12780975
16. Bergeron MF, Cannon JG, Hall EL, Kutlar A. Erythrocyte sickling during exercise and thermal stress. *Clin J Sport Med.* 2004 Nov;14(6):354-6. doi: 10.1097/00042752-200411000-00005. PMID: 15523207.
17. Dincer HE, Raza T. Compartment syndrome and fatal rhabdomyolysis in sickle cell trait. *WMJ.* 2005 Aug;104(6):67-71. PMID: 16218320.
18. Eisenbach Ch, Pohl J, Dikow R, Stremmel W, Encke J. Severe rhabdomyolysis and renal failure triggered by a sauna visit in sickle cell trait: a case report. *Clin Nephrol.* 2005 Mar;63(3):229-31. doi: 10.5414/cnp63229. PMID: 15786826
19. Channa Perera SD, Pollanen MS. Sudden death due to sickle cell crisis during law enforcement restraint. *J Forensic Leg Med.* 2007 Jul;14(5):297-300. doi: 10.1016/j.jcfm.2006.05.004. Epub 2006 Aug 17. PMID: 16914356
20. Makaryus JN, Catanzaro JN, Katona KC. Exertional rhabdomyolysis and renal failure in patients with sickle cell trait: is it time to change our approach? *Hematology.* 2007 Aug;12(4):349-52. doi: 10.1080/10245330701255254. PMID: 17654064.
21. Mitchell BL. Sickle cell trait and sudden death--bringing it home. *J Natl Med Assoc.* 2007 Mar;99(3):300-5. PMID: 17393956; PMCID: PMC2569637.

22. Burke J, Seda G, Allen D, Knee TS. A case of severe exercise-induced rhabdomyolysis associated with a weight-loss dietary supplement. *Mil Med.* 2007 Jun;172(6):656-8. doi: 10.7205/milmed.172.6.656. PMID: 17615852.
23. Harrykissoon RI, Patel B, Warner MT, Estrada-Y-Martin R. Fatal rhabdomyolysis in a college athlete due to sickle cell trait. *Chest.* 2007 Oct 1;132(4):673A.
24. Connes P, Hardy-Dessources MD, Hue O. Counterpoint: Sickle cell trait should not be considered asymptomatic and as a benign condition during physical activity. *J Appl Physiol* (1985). 2007 Dec;103(6):2138-40; discussion 2140-1. doi: 10.1152/jappphysiol.00338.2007a. PMID: 18056520.
25. Scheinin L, Wetli CV. Sudden death and sickle cell trait: medicolegal considerations and implications. *Am J Forensic Med Pathol.* 2009 Jun;30(2):204-8. doi: 10.1097/PAF.0b013e318187dfcd. PMID: 19465821.
26. Eichner, E. Randy. Sickle Cell Trait in Sports. *Current Sports Medicine Reports: November 2010 - Volume 9 - Issue 6 - p 347-351* doi: 10.1249/JSR.0b013e3181fc73d7
27. Thogmartin JR, Wilson CI, Palma NA, Ignacio SS, Shuman MJ, Flannagan LM. Sickle cell trait-associated deaths: a case series with a review of the literature. *J Forensic Sci.* 2011 Sep;56(5):1352-60. doi: 10.1111/j.1556-4029.2011.01774.x. Epub 2011 Apr 11. PMID: 21480898.
28. Harmon KG, Drezner JA, Klossner D, Asif IM. Sickle cell trait associated with a RR of death of 37 times in National Collegiate Athletic Association football athletes: a database with 2 million athlete-years as the denominator. *Br J Sports Med.* 2012 Apr;46(5):325-30. doi: 10.1136/bjsports-2011-090896. PMID: 22442191.
29. Ferster K, Eichner ER. Exertional sickling deaths in Army recruits with sickle cell trait. *Mil Med.* 2012 Jan;177(1):56-9. doi: 10.7205/milmed-d-11-00106. PMID: 22338981.
30. Loosemore M, Walsh SB, Morris E, Stewart G, Porter JB, Montgomery H. Sudden exertional death in sickle cell trait. *Br J Sports Med.* 2012 Apr;46(5):312-4. doi: 10.1136/bjsports-2011-090521. Epub 2011 Sep 30. PMID: 21965838.
31. Harris KM, Haas TS, Eichner ER, Maron BJ. Sickle cell trait associated with sudden death in competitive athletes. *Am J Cardiol.* 2012 Oct 15;110(8):1185-8. doi: 10.1016/j.amjcard.2012.06.004. Epub 2012 Jul 16. PMID: 22809753.
32. Shelmadine BD, Baltensperger A, Wilson RL, Bowden RG. Rhabdomyolysis and acute renal failure in a sickle cell trait athlete: a case study. *Clin J Sport Med.* 2013 May;23(3):235-7. doi: 10.1097/JSM.0b013e3182625a37. PMID: 22894971

33. Quattrone, Richard D. DO, MPH¹; Eichner, E. Randy MD, FACSM²; Beutler, Anthony MD³; Adams, W. Bruce MD⁴; O'Connor, Francis G. MD, MPH, FACSM⁵. Exercise Collapse Associated with Sickle Cell Trait (ECAST): Case Report and Literature Review. *Current Sports Medicine Reports*: March/April 2015 - Volume 14 - Issue 2 - p 110-116 doi: 10.1249/JSR.000000000000137
34. Mitchell BL. Sickle Cell Trait and Sudden Death. *Sports Med Open*. 2018 May 23;4(1):19. doi: 10.1186/s40798-018-0131-6. PMID: 29796715; PMCID: PMC5966366.
35. Anzalone ML, Green VS, Buja M, Sanchez LA, Harrykissoon RI, Eichner ER. Sickle cell trait and fatal rhabdomyolysis in football training: a case study. *Med Sci Sports Exerc*. 2010 Jan;42(1):3-7. doi: 10.1249/MSS.0b013e3181ae0700. PMID: 20010136.
36. Hughes RL, Feig J. Sickle Cell Trait-Related Exertional Deaths: Observations at Autopsy and Review of the Literature. *Mil Med*. 2015 Aug;180(8):e929-32. doi: 10.7205/MILMED-D-14-00707. PMID: 26226538
37. Saxena P, Chavarria C, Thurlow J. Rhabdomyolysis in a Sickle Cell Trait Positive Active Duty Male Soldier. *US Army Med Dep J*. 2016 Jan-Mar:20-3. PMID: 26874092.
38. Longo T, Shaines M. Case Report: Exertional rhabdomyolysis in a spin class participant with sickle cell trait. *F1000Res*. 2018 Nov 2;7:1742. doi: 10.12688/f1000research.16326.2. PMID: 31372209; PMCID: PMC6659762.
39. Janga KC, Greenberg S, Oo P, Sharma K, Ahmed U. Nontraumatic Exertional Rhabdomyolysis Leading to Acute Kidney Injury in a Sickle Trait Positive Individual on Renal Biopsy. *Case Rep Nephrol*. 2018 Apr 15;2018:5841216. doi: 10.1155/2018/5841216. PMID: 29850311; PMCID: PMC5925017.
40. Buchanan BK, Siebert DM, Zigman Suchsland ML, Drezner JA, Asif IM, O'Connor FG, Harmon KG. Sudden Death Associated With Sickle Cell Trait Before and After Mandatory Screening. *Sports Health*. 2020 May/Jun;12(3):241-245. doi: 10.1177/1941738120915690. Epub 2020 Apr 9. PMID: 32271134; PMCID: PMC7222668.
41. Cools KS, Crowder MD, Kucera KL, Thomas LC, Hosokawa Y, Casa DJ, Gasim A, Lee S, Schade Willis TM. Sudden Death in High School Athletes: A Case Series Examining the Influence of Sickle Cell Trait. *Pediatr Emerg Care*. 2022 Feb 1;38(2):e497-e500. doi: 10.1097/PEC.0000000000002632. PMID: 35100753; PMCID: PMC8851953.

A complete autopsy is carried out at the Tarrant County Medical Examiner's Morgue.

GROSS ANATOMIC DESCRIPTION

I. CLOTHING AND PERSONAL EFFECTS: The body is presented to the Morgue in a white body bag, wrapped in a white sheet and unclothed.

II. THERAPEUTIC INTERVENTION: Evidence of medical intervention includes oral endotracheal tube, orogastric tube, a Foley catheter, intravenous lines in the left antecubital fossa, left arm, left femoral and right femoral, pulse oximeters of the left earlobe, right index and left middle fingers, pacer pads, bandaged venipunctures of the left antecubital fossa, left forearm and left wrist, a bandaged intraosseous puncture mark of the left anterior leg, right anterior rib fractures 2-4, left anterior rib fractures 2-6, and sternal fracture at the level of the fourth intercostal space, with patchy hemorrhage of the overlying soft tissue and muscle extending focally to the anterior muscles of the neck.

III. EXTERNAL BODY DESCRIPTION: The body is that of a normally-developed, black adult male weighing 174.1 pounds, measuring 66 inches in length, and appearing compatible with the stated age of 38 years. The body is cold following refrigeration. Rigor mortis is well developed in the small and large muscles. Livor mortis is indistinct. The body demonstrates early decomposition changes with bloating and faint discoloration of the abdomen. Body hair distribution is that of a normal adult male.

The head is normocephalic. The face is normal in appearance. There is a linear healing scab, 5/8 inch, of the forehead, covered with clear material. The head hair is black, braided and measures up to 3-1/2 inches. Facial hair consists of a mustache and goatee. The eyes when initially viewed are closed. The corneae are cloudy. The irides are brown, and there is no arcus senilis. The pupils measure 3 mm bilaterally. The conjunctivae are edematous. The sclerae are nonicteric. No petechial hemorrhages are identified. The nasal skeleton and septum are intact. The ears are unremarkable. The lips and tongue are atraumatic. The teeth are natural. There is an absent right upper molar, tooth #2, with marked decay. There is blood-tinged serous fluid in the external nares and oral cavity. There is no foreign material in the external auditory canals.

The neck shows no external evidence of injury. The trachea is midline. The chest is symmetric with normal male breasts. The abdomen is mildly protuberant and palpation non-revealing. The external genitalia are that of a normal adult male with an unremarkable penis and descended testes; the perineum and anus are unremarkable. The back and buttocks are symmetric and unremarkable.

The extremities are normally developed and symmetric with no deformities or fractures. The fingernails are intact. The legs show no edema or venous stasis changes. The toenails are unremarkable.

IV. IDENTIFYING MARKS:

A. Tattoos:

1. Upper abdomen: gun with "Thugz"
2. Upper abdomen: "Niggas For Life"
3. Right arm: design
4. Right posterior arm: five letters
5. Right anterior forearm: design
6. Right posterior forearm: "THUG"
7. Right wrist: indistinct
8. Right dorsal hand: crescent moon
9. Left posterior arm: three letters
10. Left anterior forearm and wrist: designs with indistinct writing
11. Left posterior forearm: "NATIO"
12. Upper back: "MILLER"
13. Back: cross

B. Scars:

1. Oval scar, left posterior thigh, 2 x 3/4 inches
2. Numerous irregular confluent scars of the anterior right and left legs and feet, 12 x 6 inches on the right and 13 x 5 inches on the left

V. EVIDENCE OF INJURY:

- A. Laceration of forehead, closed with adhesive, 5/8 inch
- B. Focal subgaleal hemorrhage, left scalp, 2.5 x 2.3 cm
- C. Patchy hemorrhage of the anterior soft tissue and muscle of the chest, consistent with resuscitation
- D. Hemorrhage of the left sternohyoid and right posterior cricoarytenoid muscle, consistent with resuscitation and intubation

- E. Intact hyoid bone, thyroid cartilage and cricoid cartilage (see anthropology report)
- F. Abrasion of the right dorsolateral wrist, 3/4 x 1/2 inch, consistent with restraint
- G. Abrasion of the left dorsolateral wrist, 3/8 x 3/8 inch, consistent with wrist restraints
- H. Superficial abrasion of the left medial arm, 3/8 x 1/4 inch
- I. Three superficial abrasions of the right knee, 5/8 x 1/2, 1/8 x 1/8 and 1/2 x 1/4 inch
- J. Three superficial abrasions of the right anterior leg, 3/8 x 1/4, 1/4 x 1/8 and 1/4 x 1/4 inch
- K. Superficial abrasion of the left lateral leg, 3/4 x 1/2 inch

VI. INTERNAL EXAMINATION:

BODY CAVITIES:

A Y-shaped thoracoabdominal incision is made; the organs are examined in situ and eviscerated in the usual fashion. The subcutaneous fat of the abdomen is moist and yellow. The musculature of the chest shows patchy hemorrhage.

The chest wall shows fractures of the anterolateral ribs and sternum (see Therapeutic Intervention). There are no clavicle fractures. The serous body cavity membranes are smooth and glistening with no adhesions or effusions. There is mild anterior and mediastinal hemorrhage. The pericardium shows a normal amount of serous fluid. The vertebral column shows no scoliosis or kyphosis. The left and right hemidiaphragms are in their normal location and appear grossly unremarkable. The pelvis is intact.

NECK:

A layered dissection of the anterior neck reveals focal hemorrhage of the left sternohyoid anterior strap muscle and the right posterior cricoarytenoid muscle. There is no hemorrhage of the deep anterior paravertebral muscles or muscles of the floor or the mouth. There is an intact hyoid bone as well as thyroid and cricoid cartilages (see anthropology report). There is agenesis of the left superior horn of the thyroid cartilage. The larynx is comprised of unremarkable vocal cords and folds, appearing widely patent without foreign material, and is lined by smooth, glistening mucosa. The epiglottis shows mild edema with no trauma or pathologic lesions. The vasculature of the anterior neck is unremarkable. There is no injury to the carotid arteries or jugular veins. The trachea and cervical spine are in the midline presenting no traumatic injuries or pathologic lesions.

CARDIOVASCULAR SYSTEM:

The heart weighs 313 gms. The left ventricle makes up the entirety of the apex. The endocardium is smooth. The foramen ovale is closed. The myocardium is red-brown without evidence of acute or remote infarction. The free wall of the left ventricle is 1.3 cm in thickness, the interventricular septum 1.2 cm, and the right ventricle 0.3 cm.

The coronary artery ostia are in the normal anatomical location. There is no significant atherosclerotic stenosis of the coronary arteries. There is a right dominant circulation. The cardiac valves are unremarkable with the tricuspid, pulmonary, mitral and aortic valves showing thin, delicate leaflets with no vegetations or lesions present. The aorta is unremarkable.

RESPIRATORY SYSTEM:

The tracheobronchial tree contains no edema fluid, aspirated gastric contents or other foreign material present. The airways are lined by smooth, glistening mucosa. The right lung weighs 878 gms and the left 836 gms. The pleural surfaces of the lungs demonstrate prominent anthracosis. On sectioning, the lungs show subpleural bullae at the apices, as well as congestion. There are no cysts, abnormal masses or other discrete lesions identified. The hilar lymph nodes demonstrate anthracosis. The pulmonary arterial system is unremarkable without thromboemboli or atherosclerosis.

GASTROINTESTINAL SYSTEM:

The esophagus is intact and lined by smooth, glistening mucosa without erosions or varices. The gastroesophageal junction is normal. The stomach shows normal rugal folds with diffuse mucosal hemorrhage and contains 100 mL of bloody fluid; no capsules or tablets are identified. The small and large bowel demonstrate decomposition changes. The bowel contains liquid stool throughout. There are no foreign objects or bowel obstruction present within the lumens. There is focal pigmentation of the cecum and proximal large bowel. There are early decomposition changes with green discoloration. The appendix is present and is unremarkable.

The pancreas has a yellow, lobulated cut surface without hemorrhage, calcification, fat necrosis, pseudocysts or masses.

HEPATOBIILIARY SYSTEM:

The liver weighs 2023 gms and has a brown, smooth, glistening surface. On sectioning, the hepatic parenchyma is red-brown and homogeneous, with softening and early decomposition. There are no discrete lesions. The gallbladder is unremarkable containing 5 mL of yellow-green bile and no calculi. The mucosa is decomposing. The extrahepatic biliary system is patent.

RETICULOENDOTHELIAL (HEMATOPOIETIC) SYSTEM:

The spleen weighs 104 gms with a gray, smooth capsule. On sectioning, the parenchyma is reddish-brown and fibrotic. Examination of the cervical, axillary, mediastinal, abdominal and inguinal lymph nodes reveals no lymphadenopathy. The examined bone marrow is red and firm without lesions. The thymus gland is involuted, appropriate for age.

GENITOURINARY SYSTEM:

The right and left kidneys weigh 134 gms and 158 gms, respectively. The capsules strip with ease, and the cortical surfaces are smooth. On sectioning, the cortex is of normal thickness, with congestion of the corticomedullary junction and unremarkable medullae. The pelves and calyces are of normal size and lined by gray, glistening mucosa. There are no calculi. The ureters are of normal caliber in the retroperitoneum. The renal arteries and veins are normal. The urinary bladder is unremarkable, containing no urine.

The prostate gland and seminal vesicles are unremarkable. The testes are not removed.

ENDOCRINE SYSTEM:

The thyroid gland is of normal size and shape with a red-brown cut surface and no lesions. Parathyroid glands are not identified. The adrenal glands have yellow cortices of normal thickness, and the medullae show no lesions or hemorrhage. The pituitary gland is of normal size with no gross pathologic lesions.

HEAD AND CENTRAL NERVOUS SYSTEM:

A scalp incision, craniotomy, and removal of the brain are performed in the usual fashion. There is a focal scalp lesion (see Evidence of Injury). The calvarium is intact without bony abnormalities or fractures. The dura is intact and unremarkable. On stripping of the dura, the base of the skull is intact with no fractures.

The brain weighs 1518 gms and has translucent leptomeninges. The cerebral hemispheres have a normal gyral pattern, however show marked edema and flattening of the gyri and sulci. The Circle of Willis is patent with no atherosclerosis or aneurysms. The cranial nerves are intact. Coronal sectioning of the cerebrum shows a compressed ventricular system. There are no space-occupying lesions present. Sagittal sections of the cerebellum and horizontal sections of the brainstem are unremarkable except for compression of the cerebral aqueduct. During examination, the brain developed decomposition changes with green discoloration. The spinal cord is not examined.

VII. IDENTIFICATION: The decedent's identity is confirmed by comparison of antemortem and postmortem fingerprints.

VIII. HISTOLOGY:

Multiple histology slides of multiple organs (liver, kidney, spleen, stomach) demonstrate sickle-shaped red blood cells stacked together with occlusion of the blood vessel lumens.

Brain (slides 1-5): edema

Heart (slides 6-9): no significant histopathologic abnormalities

Liver (slide 10): congestion

Kidney (slide 11): acute tubular necrosis

Spleen (slide 12): congestion

Pancreas (slide 13): autolysis

Thyroid (slide 13): no significant histopathologic abnormalities

Adrenals (slide 14): no significant histopathologic abnormalities

Prostate (slide 15): no significant histopathologic abnormalities

Lungs (slides 16-20): Focal mild mixed airway inflammation and abundant bacterial overgrowth. No smooth muscle hypertrophy, thickened basement membrane, goblet cell metaplasia or significant eosinophilic inflammation.

Stomach (slide 21): autolysis

Small bowel (slides 22 and 23): autolysis

Large bowel (slides 24 and 25): autolysis

Appendix (slide 26): autolysis

SPECIMENS AND EVIDENCE COLLECTED

1. Hospital fluid samples
2. 3 mL of femoral blood, 12 mL of heart blood, and 5 mL of vitreous for toxicology
3. Representative tissues in cassettes for histology
4. Blood card
5. Fingerprints for identification
6. Blood culture bottles
7. Larynx for examination
8. Representative photographs

Disclosure: Specimens retained for toxicology will be discarded in one year.

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RCF: sad