Expert Witness Summary of Dr. Richard Maunder
Chasse case
12/7/2009

Background

The case involves a 42 y/o man with chronic schizophrenia, who died approximately 70 minutes after an altercation with two Portland police officers. Aside from a brief encounter with paramedics within the first 10 minutes after he was seized, the patient was not in the hands of medical personnel until after death had occurred, at which point aggressive resuscitative efforts were unsuccessful in restoring his life. As such, there is a paucity of monitoring and assessment data during most of the seventy minutes that elapsed between Chasse's first physical contact with police and their observation (on 33rd Avenue) that he was no longer alive. Reports and observations from the police and jail personnel, paramedics, and Providence Portland Medical Center staff who had contact with Chasse are available, as well as bystander eyewitness accounts. A detailed autopsy performed by Dr. Karen Gunson revealed evidence of extensive trauma to the chest wall and left lung. Her opinion is that the death resulted from blunt force chest injuries.

Credentials

Because of my training and experience in Pulmonary and Critical Care Medicine, I have been asked to give an opinion of the most likely medical cause of this man's death. I received my M.D. degree from the George Washington University School of Medicine, completed residency training in Internal Medicine at Yale-New Haven Hospital, also serving as Chief Resident in Medicine there, and I received fellowship training in Pulmonary and Critical Care Medicine at the University of Washington. I am board-certified in Internal Medicine, Pulmonary Diseases, and Palliative Medicine. I maintained board certification in Critical Care Medicine from 1987 through 2008. In the mid-1980's I was elected to fellowship in the American College of Physicians, as well as the American College of Chest Physicians. During the 1980's and early 1990's I served as a faculty member at the University of Washington School of Medicine, where I was extensively involved in the study of lung injury due to trauma and other causes. I was based primarily at Harborview Medical Center, Seattle's premier Level I trauma center, where I worked as pulmonary and critical care consultant for a very busy trauma service. In the early 1990's I served as Director of Critical Care Services for Swedish Hospital Medical Center in Seattle, and between 1996 and 2005 I was Director of Critical Care Services at Providence Portland Medical Center in Portland. Since then I have worked as a Palliative Medicine physician, serving as Medical Director of the Providence Hospice and Palliative Care program in Portland Oregon from 2005 to 2009.

Records reviewed

In preparing this report, I reviewed the following materials, documents and records:

1. Detailed timeline of events of 9/17/2006
2. Prehospital Care Report (NW 13th/Everett) 9/17/2006
3. PFB Prehospital Care Report 9/17/2006
4. Photograph of Incident 9/17/2006

5. Prehospital Care Report (NE 33rd/Clackamas) 9/17/2006
6. Hospital Emergency Department Report 9/17/2006
7. DVD video from MCDC surveillance camera 9/17/2006
8. Nurse Gayman's Information Report (MCDC) 9/17/2006
9. Medical Examiner's Report (Autopsy) 9/18/2006
10. Photos from autopsy 9/18/2006
11. CD-rom of post-mortem CT scan 11/20/2006
12. Expert witness statement of William Long, M.D. 8/28/2009
13. Expert witness statement of Tom S. Neuman, M.D. 10/15/2009
14. Expert witness statement of Vincent J.M. Di Maio, M.D. 10/23/2009

Factual summary

Initial encounter at NW 13th and Everett. On 9/17/2006 Mr. James Chasse was pursued by police and taken into custody. He initially tried to run away but was apprehended and tackled by the police some time between 1718 and 1720 hrs. Chasse was forcibly taken to the ground. According to observers, his chest and head struck the sidewalk several times as he was lifted and forced face-down onto the pavement. A struggle ensued, estimated to have lasted approximately 3-4 minutes. He offered some resistance, but was heard to repeatedly cry out "no, no", "ow, ow, ow", and "mercy". He may have attempted to bite the officers involved. During the altercation Chasse was kicked and punched multiple times in the torso. He was eventually restrained in a prone position, with his arms pulled behind his back for application of handcuffs. Foot pressure was applied to his upper back, as well as knee pressure under the full weight of a police officer. According to one of the officers involved, Chasse then went limp and stopped breathing, at which point the officer thought Chasse had died. At 1723 a paramedic unit was summoned emergently to the scene. Eyewitness reports are variable as to timing, but it seems clear that Chasse was unconscious and not breathing for some period of time, beginning to come around gradually after the weight of the officers was removed. In fact, the emergency call was downgraded from Code 3 to Code 1 within a minute after the initial call was placed. AMR paramedics arrived on the scene at 1725, and Portland Fire Bureau paramedics were there at 1726. On evaluation by the paramedics, Chasse was found to be lying quietly on the sidewalk, initially not responding to questions. He cooperated with measurement of vital signs at 1728 but was resistant and uncooperative shortly thereafter when the paramedic attempted to obtain a blood sample to check blood glucose; this was eventually accomplished successfully at 1730. During the evaluation he was felt to be confused but alert. They found no overt evidence of medical instability on the basis of normal vital signs, normal blood glucose, and an abbreviated physical examination revealing only minor abrasions and lacerations and bleeding from the mouth. A secondary survey was not performed. Police informed the paramedics that Chasse was in possession of cocaine. The assessment of both the AMR and Portland Fire Bureau paramedics was that he was intoxicated due to the use of illicit drugs. He was deemed stable and cleared at 1741. Although medical transport was offered twice by the paramedics, it was refused by the police. At this point Chasse was carried in a hog-tied position and placed in a police car. He was observed by witnesses to be struggling and crying out in pain as he was carried. At the scene and while in the police car, the police report that he responded to their questions.

Multnomah County Detention Center. He was then transported to the Multnomah County Detention Center, arriving at approximately 1806. A gauze "spit sock" was placed over his head. He was carried from the car to a holding cell. The jail surveillance tape shows him

to be relatively quiet as he is being carried in to the cell. A minute later at 1807 he is heard to be crying out in the cell, although the cell is not visible to the camera, so it is not clear what he is happening or what position he is in. On the tape he yells intermittently over the next minute or so and then quiets down as he is told to relax and that the handcuffs are going to be removed. According to the arresting officer, after they removed the restraints Chasse again stopped breathing. At approximately 1810 staff summoned the jail nurse to evaluate Chasse, not only because he had stopped breathing but also due to a brief period of twitching activity. Within 30 seconds he resumed breathing, even before the nurse arrived. She performed her evaluation by observing Chasse through the window. On arrival at approximately 1811 she observed another brief (5-second) episode of twitching, which she interpreted as seizure-like activity. After that Chasse was seen through the window to be "breathing and moving his head and limbs". He continued to lie on the cell floor, either face down or on his right side. The nurse refused to accept him into the jail, due to his medical condition, and she recommended that he be transported to the hospital for medical evaluation. At approximately 1817 the handcuffs, ankle cuffs, and hobble restraint were re-applied, and at 1818 he was carried out of the holding cell to the police car, again face down, hog-tied with arms behind his back. The surveillance tape shows him to be writhing as he is being carried by the arms, and he can be heard crying out repeatedly in a groaning or grunting fashion. He was positioned upright in the back seat of the car with the mesh hood still in place. The plan was for Code 1 transport by the police to Portland Adventist Hospital. As the car paused outside the MCDC sally port, the police sergeant there noted that Chasse was quiet at that point but "breathing very rapidly..., hard..., like he was trying to catch [his] breath".

Subsequent events. At 1824 the officers requested advance notification of Portland Adventist Emergency Department that they had a "fighter" coming in. Some time after that, while en route to the hospital via I-84, the officers heard a thud in the back seat and looked back to find that Chasse had fallen against the door. One of the officers noted that Chasse was completely quiet and had stopped moving. Because of the spit sock, he was unable to see Chasse's face. No further assessment was made while on the road. They exited the freeway at 33rd Avenue and removed Chasse from the car, at which point he was apneic and pulseless. At 1829 the paramedics were called to respond. The mesh hood was removed, and the officer noted that blood was obstructing the airway. He cleared the upper airway using a finger sweep, and chest compressions were initiated at approximately 1832. A bystander brought an AED device to the scene. It is unclear when the AED was applied, but it reportedly read "No Shock Advised". The bystander observed that Chasse looked gray and lifeless and that his chest looked depressed on the left. The paramedics arrived at 1834. They encountered the police officers standing near their car. Chasse was lying on the ground, unattended. On their initial assessment, the paramedics found that Chasse was pulseless and not breathing. They resumed CPR, at this point with bag-mask ventilation, and established IV access. They observed that Chasse's chest wall had "no compliance" and that compressions felt "squishy". The cardiac rhythm was asystole. They administered epinephrine and atropine. Endotracheal intubation was accomplished by approximately 1841. He was transported to Providence Portland Medical Center at 1848. During transit, a wide-complex tachycardia was observed on the monitor, with transient recovery of a carotid pulse. However, on arrival at the hospital minutes later, there was no detectable pulse. Countershock was administered for ventricular fibrillation, with recovery of a slow wide-complex rhythm, but there was no pulse. Epinephrine, atropine, and bicarbonate were administered, with no response. Resuscitative efforts were terminated at 1904, and Chasse was declared dead. Apparently, the responding paramedics and hospital emergency staff were unaware of Chasse's chest wall trauma; they followed a cardiac arrest protocol and did not address

the trauma issues in their treatment. After the death, a staff nurse noted crepitus consistent with subcutaneous emphysema and notified the emergency physician Dr. Carlos Sanchez, who agreed that there was evidence of subcutaneous emphysema, as well as a left flail chest. He placed a 16-gauge angiocath in the left anterior chest, with no rush of air.

An autopsy was performed by the medical examiner, Dr. Karen Gunson, the following day on 9/18/2006. Toxicological examinations were performed on retained samples of blood, urine, and vitreous fluid, with negative findings. A post-mortem CT scan was performed by Dr. Gerald Warnock on 11/20/2006. The findings from these studies are reviewed in the case discussion. A second autopsy was performed, but I have not been asked to review the results.

Case discussion

Review of documented chest injuries. From the post-mortem CT scan and autopsy we know that this man sustained massive skeletal and soft-tissue trauma to the chest. At autopsy bilateral parasternal rib fractures (ribs #3-8) were noted, as well as fractures of the same ribs along the left anterior axillary line (6-8 inches lateral to the sternum). All of these fractures demonstrated serosal (parietal pleura) penetration and were associated with what is described as intense soft tissue hemorrhage. Extensive posterior rib fractures were seen as well, eventually found to involve all twelve ribs on the left and ribs #3-6 on the right. At least some of the left posterior fractures were displaced, leaving sharp bone fragments jutting inward toward the lung. In her post-mortem examination Dr. Gunson observed that several of the jagged rib ends had perforated the visceral pleural surface, penetrating the left upper lung adjacent to the 5th, 6th, and 7th vertebrae. CT imaging revealed a fracture in the body of the sternum as well.

The parasternal fractures and the fracture of the corpus sternum may have been caused by compressive forces or blows to the anterior chest during the altercation with police, but is also possible they may have occurred later due to aggressive chest compressions during cardiopulmonary resuscitation efforts. The lateral rib fractures probably occurred during the altercation with police at 13th and Everett. The displaced fracture of the distal clavicle, which was discovered by the post-mortem CT scan, also probably occurred at the time of the initial takedown, due to a forceful fall onto the point of the shoulder, or possibly a direct blow to the clavicle. The posterior rib fractures were caused by the blunt chest trauma inflicted during the initial takedown and restraint of the victim between 1718 and 1723 hrs.

Relevant anatomy and physiology of the lung and pleura. The human lung is made up of millions of tiny air sacs or alveolae, which as a whole might be compared to a partially inflated balloon, expanding and contracting in response to the pressure gradient between the inside and outside. The "visceral pleura" surrounding the lung surface is a skin-like layer of tissue, which functions to keep the air within the lung from leaking out. It is connected at the lung root to the "parietal pleura", which is a skin-like layer of tissue lining the chest wall inside the rib cage. Under normal circumstances the lung is fully inflated, with the inner (visceral) pleural layer nearly touching the outer (parietal) pleural layer and forming what anatomists call a "potential space" between the chest wall and lung. It is called a potential space because normally it is completely airtight and contains nothing more than a few teaspoons of lubricating fluid that allows the surface of the lung to glide along the inside of the chest wall as the lungs expand and contract during breathing. This

pleural space always has a slight negative pressure (vacuum) relative to the air-containing structures of the lung. This negative pressure is accentuated during inspiratory effort and transmitted to the lung surface, causing movement of fresh air through the upper airways and into the air sacs of the lung during chest expansion.

The consequences of rib fractures and pleural puncture. Rib fractures causing laceration of the visceral pleura, as seen in this case, break the normally airtight pleural seal. This type of injury, by definition, causes leakage of air directly from the lung into the surrounding pleural space. Due to the lung's elastic recoil, it then begins to separate from the chest wall and collapse. This shrinking of the lung typically creates a "ball valve" effect, causing air to be sucked outward by the negative pleural pressure but preventing movement of air back through the pleural tear during exhalation. The gradual build-up of air within the pleural space creates a profound disturbance of the normal respiratory cycle (sequential inspiration and expiration), which relies on the pleural surface being intact. The lung deflates progressively, like a tire that has been punctured. This is known as a "pneumothorax".

Running along the length of each rib is an intercostal artery and vein. When a rib is fractured, especially in a comminuted or displaced fashion, these vessels can be lacerated and bleed into the surrounding chest wall soft tissue. If the parietal pleura is penetrated, as it was by a large number of fractured ribs in this particular case, bleeding into the pleural space occurs; this is referred to as a "hemothorax". Chasse probably had a "hemopneumothorax", representing both air and blood within the pleural cavity.

Timing of cardiorespiratory deterioration after closed pneumothorax. There is usually relatively little in the way of immediate cardiac or respiratory compromise following pleural disruption, and oxygenation is minimally affected, as long as the alternate lung is functioning normally. If undetected and untreated, however, pressure will build up within the pleural cavity over time, causing progressive collapse of the underlying lung. The time frame for accumulation of air within the pleural cavity is variable following puncture, depending on the size of the hole(s) in the pleural surface and the degree of respiratory effort. It is slower in the spontaneously breathing victim than in one receiving artificial positive pressure ventilation from health care personnel. Increased respiratory effort, associated with periods of strenuous exertion or crying out, which occurred in this case, will cause more negative pleural pressure and accelerate the accumulation of air within the pleural space. Eventually, if treatment is delayed, the build-up of pressure within the chest can progress to the point where intrapleural pressure actually exceeds atmospheric pressure. This is referred to as a "tension pneumothorax". A simple pneumothorax can cause respiratory insufficiency, with low oxygen levels and breathlessness, but as a pneumothorax enlarges and begins to develop tension, the clinical picture is dominated more by cardiovascular instability. In the absence of appropriate therapy, this scenario inevitably leads to death from cardiovascular collapse, especially if circulating blood volume is low.

Pathophysiology of cardiac arrest following hypovolemia and pneumothorax. To function effectively as a pump (maintaining forward cardiac output and blood pressure), the heart, which is located within the chest, relies upon the return of blood from the veins. The pressure of blood in the veins is normally somewhat higher than the pressure within the chest, so venous blood moves along a pressure gradient back to the heart, where it can be oxygenated by the lungs and pumped into the arteries to support vital organ function. If intrathoracic pressure is increased and/or venous pressure is reduced, then the flow of blood back to the heart decreases, and cardiac output will drop. As described above, a

progressive increase in intrathoracic pressure commonly follows pleural laceration, due to the resulting pneumothorax. In the setting of blunt chest trauma, reduced venous pressure can result from two mechanisms: 1) direct loss of blood from torn intercostal vessels; and 2) indirect loss through translocation of plasma from the smaller vessels supplying traumatized body regions into the surrounding interstitial soft-tissue space. The degree of this interstitial (so-called "third-space") redistribution of fluid tends to correlate with the extent of the trauma. If pneumothorax, hemorrhage, and third-space losses occur in combination, as would have been the case following Chasse's injuries, it is only a matter of time before the combined effects of tissue hypoxia, progressive metabolic acidosis, and low blood pressure will lead to full-blown cardiac arrest. This is most often associated with asystole or pulseless electrical activity, exactly as observed in this case. CPR is unlikely to be successful unless the underlying problems are treated immediately, by resuscitation with massive amounts of intravenous fluids and evacuation of air from the pleural cavity.

Cause of death

The above considerations lead me to conclude that in all probability the medical cause of Mr. Chasse's death was sudden cardiovascular collapse due to hypovolemia and the mechanical effects of a pneumothorax. During the initial takedown and prone restraint by police, he was subjected to blunt injuries (direct blows and compressive force) to the chest wall, resulting in extensive skeletal and soft-tissue trauma, with associated hemorrhage and third-space fluid losses. Sixteen ribs were fractured posteriorly, and six ribs were fractured laterally. Most of these fractures were associated with penetration of the parietal pleura and intense hemorrhage into the adjacent soft tissue and musculature. The jagged ends of several displaced rib fractures adjacent to the left upper spine penetrated the surface of the lung, causing a closed pneumothorax. Damage to the chest wall and lung went undetected and untreated. The resultant pneumothorax was likely small at first, causing little in the way of cardiorespiratory instability. Chasse likely suffered agonizing pain from the fractured ribs and clavicle, undoubtedly aggravated by the way he was handled. Over the next 60-70 minutes, there was progressive accumulation of air within the left pleural space, which is the natural history of an untreated closed pneumothorax resulting from penetration of the lung surface by fractured ribs. In addition, over that time interval there was progressive blood loss due to intense soft-tissue and intrapleural hemorrhage, as well as third-space fluid losses from the vasculature. The resultant hypovolemia, together with a steady build-up of chest pressure from the expanding pneumothorax, led eventually to a profound reduction in venous return to the heart. While the development of cardiorespiratory instability was undoubtedly progressive over time, I believe it reached a critical point as Chasse was being prepared for transport from the jail to Portland Adventist Hospital. The rapid, "hard" breathing observed at that time is consistent with both the effect of acidosis caused by hemorrhagic/hypovolemic shock and impaired lung function from an expanding left pneumothorax. His condition steadily deteriorated, and he lost consciousness, due to low blood pressure from hypovolemia, aggravated by upright posture in the car, impaired myocardial contractility from acidosis and hypoxemia, and poor cardiac venous return due to the pneumothorax. Because he was alone in the back seat of a police car, it took several minutes before he could be assessed or treated. By the time medical intervention could be initiated several minutes later, his condition had deteriorated to the point where there was no cardiac electrical activity, and aggressive efforts to revive him were futile.

Evaluation of Expert Witness Statements

Dr. Tom S. Neuman (10/15/2009). Dr. Neuman is board-certified in Pulmonary Medicine and Emergency Medicine. He has published extensively in the field of Hyperbaric and Undersea Medicine. He reviewed an extensive amount of material in preparing his report.

1. Dr. Neuman finds it difficult to reconcile the extent and severity of the posterior rib fractures with the deposition testimony of the police. He believes it is unlikely the fractures were the result of a simple takedown or tackle. He concludes that "there is a major discrepancy between the fractures that were observed on Mr. Chasse and the mechanism by which they might have occurred."

   I agree in part. However, I was surprised by and do not agree with Dr. Neuman's later conclusion that "Mr. Chasse suffered the bulk of his rib fractures at the time of his CPR," especially if he means to include the posterior and lateral rib fractures.
2. Dr. Neuman suggests that some of the posterior rib fractures might represent "cough fractures".

   I disagree on this point. Based on my experience, the extent and location of Chasse's rib fractures are simply not consistent with cough as a mechanism.
3. He notes that Chasse was screaming and yelling as he was carried out of the jail, and his opinion is that this is inconsistent with someone with a flail chest going into respiratory failure. He notes that "an individual who is able to scream and yell in such a fashion is able to mount the minimal ventilatory movements necessary to live." He goes on to point out that if Chasse had died of respiratory failure, his deterioration would have been a slower, more prolonged process.

   Obviously, lung function was declining significantly by the time the police car approached the jail sally port at about 1823. Despite this, I agree with Dr. Neuman that in all likelihood Chasse's death was not simply the result of respiratory failure. For the reasons cited above, I believe the final event was cardiovascular collapse due to the combined effects of hemorrhagic shock, metabolic acidosis, hypoxemia, and the mechanical effects of pneumothorax – all resulting ultimately from extensive chest wall trauma.
4. Dr. Neuman does not believe a secondary exam would have revealed anything of concern or changed the decision to forgo hospital transport.

   I disagree. Had there been more thorough and straightforward transfer of information from the arresting officers to the paramedics, the potential for significant chest trauma would have been known, which would have led to a more complete examination by Paramedic Hergert. As it was, the paramedics were told by police to check vital signs only. If critical information had not been withheld, a secondary survey would likely have been performed, revealing injuries significant enough to necessitate hospital transport.

Dr. Vincent J.M. Di Maio (10/23/2009). Dr. Di Maio is a consultant in Forensic Pathology, based in San Antonio Texas. He has extensive experience in the field and has published a book on what has been called the "Excited Delirium Syndrome".

1. Dr. Di Maio devotes a good deal of attention to the issue of flail chest. He dismisses the physiological significance of a flail chest and hypothesizes that the flail segments seen at autopsy developed as a result of CPR. His opinion is that "the rib fractures did not cause or contribute to Chasse's death".

   For the reasons outlined in detail above, I disagree with Dr. Di Maio's opinion regarding the role of Chasse's rib fractures as a cause of death.

2. Dr. Di Maio points out that the ultimate mechanism of death was cardiac arrest, not chronic asphyxia.

   I agree that the ultimate mechanism of death was cardiovascular collapse. However, for the reasons cited above, I believe Chasse's cardiovascular collapse was the direct result of extensive chest trauma.

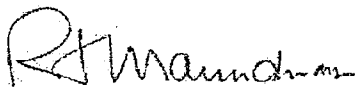
3. Dr. Di Maio provides a detailed description of excited delirium, and concludes that in all probability Chasse died as a result of the Excited Delirium Syndrome, which he explains "involves the sudden death of an individual, during or following an episode of excited delirium, in which an autopsy fails to reveal evidence of sufficient trauma or natural disease to explain the death."

   Dr. Di Maio is presented as an expert on excited delirium and the Excited Delirium Syndrome. However, using the diagnostic criteria and definitions Dr. Di Maio has laid out for these entities, it does not appear that Chasse actually had excited delirium, nor that his death could be reasonably attributed to the Excited Delirium Syndrome.

Summary

In summary, the death of James Chasse was, in my medical opinion, the direct result of chest wall trauma inflicted at the time he was arrested near the corner of 13th and Everett. Over the next hour, there was gradual but steady physiological deterioration due to massive hemorrhage, third-space fluid losses, and an evolving pneumothorax. The progression of these processes led inevitably to shock, metabolic acidosis, hypoxemia, and cardiac instability. At the time things eventually deteriorated to the point of complete cardiovascular collapse, Chasse was the back of a police car on the freeway, unattended and unmonitored. As a result, medical intervention was further delayed. At that point, resuscitation efforts following a cardiac arrest protocol were unsuccessful in restoring him to life. On the other hand, given the sophistication of hospital and pre-hospital emergency care in Portland, I would expect that appropriate medical evaluation and intervention at nearly any point in the preceding hour would very likely have averted Chasse's death.

I hold all of my opinions to a reasonable degree of medical probability. This report is not intended to be a complete or final statement of my opinions, and I reserve the right to expand, modify or otherwise amend my opinions as the discovery process proceeds. I have included the factual summaries of Drs. Neuman and Di Maio, where accurate, in my assessment of the factual basis of this case.



Richard J. Maunder, M.D.