



## **Arkansas State Athletic Commission**

### **COMBATIVE SPORTS REGULATION**

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## **FINAL REPORT**

### **Investigation Into Death of Anthony Jones**

**MEDIA/INQUIRY CONTACT:** Jason A. Stuart, Commissioner – 501.687.9000 – [Jason.Stuart@Arkansas.gov](mailto:Jason.Stuart@Arkansas.gov)

**PRELIMINARY REPORT DATE:**

MON-31-JAN-2011

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**FINAL REPORT AUTHOR:**

JASON A. STUART

**COMMISSIONER IN-CHARGE OF INVESTIGATION:**

JASON A. STUART

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**EVENT VENUE, DATE & SCHEDULED TIME:**  
FITNESS UNLIMITED, 1212 Hwy. 35 N, Benton, AR  
SAT-29-JAN-2011 @ 19:00 Hours

**NEAREST MEDICAL CENTER:**  
.9 Miles (Saline Memorial Hospital, 1 Medical Park Dr., Benton, AR – LEVEL 3 TRAUMA CENTER)

**NEAREST LEVEL 1 TRAUMA CENTER:**  
27.3 Miles (University of Arkansas for Medical Sciences Hospital, 4301 W. Markham St., Little Rock, AR)

**COMMISSIONER IN-CHARGE AT RINGSIDE:**

JASON A. STUART

**COMMISSION INSPECTOR:**

SONNY AXSOM

**BOUT REFEREE:**

MARTIN TUNSTALL

**POSSESSOR OF SEQUESTERED EQUIPMENT:**

JASON A. STUART

**RINGSIDE PHYSICIAN & NURSE ASSISTANT:**

DR. DEVN FRANDBEN, D.O.  
JULIA STONE, R.N.

**RINGSIDE AMBULANCE/EMT/PARAMEDICS:**

MEDTRAN; JERRY COHEN; HOPE CRESSLER

**PROMOTER:** LARRY HARRIS – THUNDERSTRUCK PROMOTIONS  
**PROMOTER'S LICENSE:** License #1427 Issued: 1-JUL-2010 Expires 30-JUN-2011  
**PROMOTER'S BOND ON FILE:** \$5,000 Bond Per Reg. §1.14.7.2  
**EVENT INSURANCE ON FILE:** YES. Per Reg. §1.27.3.1 – \$10,000 Per Contestant Medical Coverage;  
\$10,000 Per Contestant Accidental Death/Dismemberment; \$1,000 Deductible  
**CLAIM FORM ADMINISTERED:** YES  
**WEIGH-IN DATE & VENUE:** FRI-28-JAN-2011; FITNESS UNLIMITED, 1212 HWY. 35 N, BENTON, AR

**DECEASED CONTESTANT NAME & AGE:** ANTHONY JONES – Age 27  
**DATE & TIME OF DECEDENT'S WEIGH-IN:** Approx. 19:45 Hours FRI-28-JAN-2011  
**DATE & TIME OF DECEDENT'S PRE-BOUT PHYSICAL:** Approx. 17:45 Hours SAT-29-JAN-2011  
**DECEASED CONTESTANT AMATEUR EXPERIENCE:** Contestant Reported Record of 13-3-0 in Elimination  
Contest/Club Fighting  
**DECEASED CONTESTANT PRO EXPERIENCE:** NONE  
**DECEASED CONTESTANT VALID AR LICENSE:** YES  
**DECEASED CONTESTANT CHIEF SECOND:** DAVID CALDWELL  
**DECEASED CONTESTANT CHIEF SECOND VALID AR LICENSE:** YES  
**OPPONENT NAME:** QUINCY PALMER  
**OPPONENT AMATEUR EXPERIENCE:** Contestant Reported Record of 21-5-0  
**OPPONENT PRO EXPERIENCE:** FightFax Verified Record 1-0-0  
**OPPONENT VALID AR LICENSE:** YES  
**OPPONENT CHIEF SECOND:** NORMAN BAKER  
**OPPONENT CHIEF SECOND VALID AR LICENSE:** YES

## **SECTION I**

### **SCOPE OF INVESTIGATION**

This investigation's scope was comprehensive including, without limitation, a review and analysis of:

- 1.) Licensure Procedures & Standards;
- 2.) Bout Approval;
- 3.) All Event Procedures, Protocols and Actions;
- 4.) Event Emergency Action Plan;
- 5.) Medical Staffing, Pre-bout & Post-bout Protocols & Procedures;
- 6.) Jones' Medical Records, Autopsy and Specimen Analysis Reports;
- 7.) Jones' Personal History, Training History and other Background Information;
- 8.) Possible Causes of Injury and Contributing Factors;
- 9.) Previous Combative Sports Related Deaths in Arkansas; and
- 10.) Arkansas' Regulatory and Procedural Safeguards for Combative Sports Events.

In addition to utilizing its own internal resources and procedures, the Commission utilized the experience and knowledge of its medical team to ensure this Investigation and Report are both thorough and correct. The Commission contacted the relevant commissions and regulatory bodies within Mississippi, Nevada and Texas to obtain potentially relevant information learned by those sister commissions, as they have each experienced at least one (1) combative sports related death within the last twenty (20) years. During consultation with the sister commissions, the Commission inquired into the scope and findings of their respective investigations, to ensure potentially important areas of emphasis were not overlooked during this Investigation, and inquired into any procedural or regulatory changes resulting from those commissions' investigations.

## **SECTION II**

### **FACTUAL NARRATIVE SUMMARY OF EVENT ACTIVITIES**

This Event was sanctioned by the Arkansas State Athletic Commission (“Commission”) and held in accordance with the Commission’s Combative Sports Regulations. This incident occurred during and subsequent to the fifth (5<sup>th</sup>) bout of the Event. The fifth (5<sup>th</sup>) bout was between heavyweight Contestants, Quincy Palmer (1-0-0) (“Palmer”) and Anthony Jones (0-0-0) (“Jones”) (jointly referred to as “Contestants”).

The scales used at weigh-in were verified using a 35 lb. known standard measure and certified by Commissioner Stuart to weigh 9/10<sup>th</sup> lbs. less than standard weight and weights Pursuant to Reg. §§ 1.31, 1.32 and 1.33, Palmer weighed 251 lbs. and Jones weighed in at 233 ½ lbs. Both Palmer and Jones were within regulatory weights for the heavyweight division.

As part of the Pre-Bout activities the day of the Event, the Ring was inspected by Commissioner Stuart and found to be compliant with all subsections of Reg. §1.15.6. The Ring was additionally inspected and approved by Referee Tunstall.

Pursuant to Reg. §1.20, the Ringside Physician, assisted by the registered nurse, conducted Pre-Bout Physicals of all Contestants, including Jones and completed Combative Sports Medical Report (Pre-Bout) – ASAC Form CSMR057-2010 for all Contestants. Pre-Bout physicals were administered beginning at approximately 17:00 hours 29-JAN-2011. Following the Pre-Bout Physical, Jones was medically cleared by the Ringside Physician to participate in the Event.

After Jones completed his Pre-Bout Physical, Jones brought his Pre-Bout Physical form to Commissioner Stuart and in compliance with standard Commission protocols under Reg. §1.11.2, Commissioner Stuart administered a preliminary breath test (“PBT”) to Jones at approximately 18:00 hours 29-JAN-2011. Jones’ PBT was conducted using an Intoximeter IV portable breathalyzer, which is calibrated by the Arkansas State Police at least every six (6) months. Jones’ PBT result was .000 and he was cleared by the Commission to participate in the Event.

In accordance with standard Event protocol and immediately prior to the start of the Event, Commissioner Stuart visually verified the presence of an on-site ambulance with transport authority to the nearest medical facility. Commissioner Stuart visually inspected and verified the EMT’s pre-planned paths of emergency ingress and egress from the Ring to the ambulance. Commissioner Stuart visually inspected and verified the EMT’s equipment stationed at ringside and verified the on-site presence of advanced life support automatic defibrillator, oxygen, neck brace, spine board, collapsible gurney and other similar triage medical supplies. The on-site ambulance was stationed at the front/side exit and a direct route from the Ring to the exit was ensured prior to the start of the Event.

Approximately 15 minutes before the start of the Event’s 1<sup>st</sup> Bout, Commissioner Stuart held an emergency action plan (“EAP”) meeting with the Ringside Physician and EMTs to advise the Physician of the EMT’s ringside location (stationed in the neutral corner opposite the Ringside Physician so as to allow the Ringside Physician visual line of site and orientation upon entering the Ring) and vice versa. Commissioner Stuart also ensured the Ringside Physician and EMTs were clear on the protocols and procedures for implementation of the Commission’s EAP, should it become necessary during the Event. Neither the Physician nor EMTs had any questions regarding the EAP protocols. After clearance with the medical staff in attendance, the pre-planned emergency exit route was communicated to the Event Promoter with instructions to maintain continuous clearance of the emergency exit path during the Event.

Pursuant to Reg. §1.22, the Inspector inspected and approved both Contestants' Handwraps and Gloves. Handwraps used by the Contestants were in compliance with Reg. §1.35. Gloves used by the Contestants were 10 oz. gloves in accordance with Reg. §2.3 and in compliance with Reg. §1.34.

The Contestants entered the Ring at 21:40 hours and competed in a normal manner. Jones was clearly ahead on the scorecards at the end of the 1<sup>st</sup> Round. At the end of the 2<sup>nd</sup> Round, Palmer struck Jones with a legal punch or combination of punches, which Downed Jones prior to bell signaling the end of the 2<sup>nd</sup> Round. Jones did not return to his feet before the end of the Ten Count and the Referee stopped the fight. In accordance with standard protocols, the Ringside Physician entered the Ring to attend to Jones immediately after the Referee completed the Ten Count. The Ringside Physician was assisted by the registered nurse at ringside.

After initial examination, the Physician did not see any evidence of life threatening injuries. However, using an abundance of caution, the Physician implemented the EAP by signaling the EMTs and requested transportation to the nearest medical facility for further observation and testing of Jones. Jones was placed on the backboard, moved to the ringside collapsible gurney and transported via ambulance to Saline Memorial Hospital for evaluation. Commissioner Stuart provided Jones' Chief Second with the Promoter's Event Insurance Certificate and Claim Form with attached instructions on how to file a claim. Commissioner Stuart advised the Chief Second to accompany Jones to the medical facility, present the insurance information and follow the claim instructions.

Results of Jones' Ringside medical examination did not reveal life threatening injuries. After ensuring Jones had been loaded and transported, the Ringside Physician completed Jones' Combative Sports Medical Report (Post-Bout) – ASAC Form CSMR057-2010. During the subsequent Bouts, the ringside registered nurse provided follow-up reporting on Mr. Jones' condition and advised Jones was being transported to the University of Arkansas for Medical Sciences Hospital ("UAMS") for further precautionary evaluation. Jones' time of death was called early in the morning on SUN-30-JAN-2011.

At 08:40 on SUN-30-JAN-2011, Commissioner Stuart received notice of Jones' death and implemented standard protocols for notification of the necessary persons and agencies, as well as, beginning the Commission's investigation.

In accordance with standard protocols for unanticipated deaths, UAMS released Jones' body to the Pulaski County Coroner on 30-JAN-2011, who then transferred Jones' body to the Arkansas State Medical Examiner's ("ASME") office for forensic pathological examination and testing. The Commission received and evaluated the results of Jones' medical records, autopsy report, specimen analyses, as well as, generated its own investigatory notes. The purpose of this investigation was to determine all of the facts and circumstances surrounding Jones' death and review Arkansas' Combative Sports related health, safety and welfare regulations and procedures to ensure the continued safety of the Contestants, Officials and general public at Events sanctioned by the Commission.

## **SECTION III**

### **DETAILED TIMELINE OF RELEVANT INCIDENT INFORMATION**

The detailed, verified facts and information in this section are critical to the reader's understanding of the Arkansas State Medical Examiner's ("ASME") and Commission's respective opinions on Jones' primary cause of death and secondary/contributing factors. The information in this section was compiled directly from hundreds of hours of the Commission's work including interviews of Jones' teammates and friends, as well as, multiple reviews of Jones' medical records from Saline Memorial Hospital and University of Arkansas for Medical Sciences Hospital, the ASME's autopsy report, the video tapes of the Bout, and the Commission's own observations and activities.

The Commission's public disclosure of Jones' personal healthcare information in this report is pursuant to a HIPAA waiver and release Jones signed when applying to the Commission for a Combative Sports License. The public disclosures of Jones' personal healthcare information is being done only after due deliberation and consideration of the public's overriding interests in obtaining the public health, safety and welfare benefits associated with the public's receipt of full and factual information necessary to understand the facts and activities leading up to and contributing to Jones' death. As provided for in Ark. Code Ann. §25-19-105(b)(2), the Commission does not intend to, nor will it, disclose, provide or permit general access to Jones' actual medical records. This report fully discloses all relevant and potentially relevant information necessary to understand the ASME's and Commission's respective determinations on Jones' primary cause of death and secondary/contributing factors, as well as, the Commission's recommendations and position on adoption or enforcement of its Regulations.

While this section contains some of the Commission's interpretations of the evidence, such interpretations are limited to contemporaneous comments included in the charts for Jones' vital signs, blood and urine test results compiled by the Commission from Jones' medical records. The Commission's full conclusions are provided and fully explained in Section IV of this Report.

To save readers' time and help non-medically trained readers understand the complex medical terminology and test results, the Commission has provided the table of terms, definitions and explanations in Section III A, which table may assist with a proper interpretation of Jones' medical records including his vital signs, blood test and urine test results. The verified facts and relevant incident information timeline are provided in Section III B, C and D.

**A. DEFINITIONS & GUIDE TO READING & UNDERSTANDING BLOOD PRESSURE, BLOOD TEST & URINE TEST TERMINOLOGY & RESULTS**

**\*\*\* SPECIAL CAUTIONARY NOTE \*\*\***

The information in this chart is provided by the Commission **FOR INFORMATIONAL PURPOSES ONLY** and is not intended and should not be taken as medical advice or used to diagnose or treat any condition. **ALWAYS CONSULT YOUR LICENSED PHYSICIAN FOR INTERPRETATION OF YOUR RESULTS & TREATMENT.** For all reported values in this Report, the subject's actual test result is shown and is then followed by the generally accepted "normal range" in parenthesis. In the definitions/guide below, sentence fragments are purposely used to conserve space and save time. The definitions and indications or associations are **INCOMPLETE**, as they are only intended to include information the Commission deemed relevant or potentially relevant to this investigation rather than provide an exhaustive explanation of each term.

**BLOOD PRESSURE**

**Blood Pressure (BP)** – Measurement of the pressure exerted by circulating blood upon the walls of blood vessels. When used without further specification, BP usually refers to the arterial pressure of the systemic circulation. During each heartbeat, the BP varies between a maximum, known as the "systolic pressure," and a minimum, known as the "diastolic pressure." The measurement is usually expressed in terms of systolic pressure over diastolic pressure and has a normal range in adults between 110/65 and 140/90. Low BP can be caused by many things including sepsis (a generally used term for any type of systemic blood toxins/poisons), hemorrhaging/blood loss, and shock. At a certain point, low BP causes perfusion (blood supply) of the brain and other vital organs, such as the kidneys and liver, to be critically insufficient to the point of the cells within the organs dying and eventual organ failure.

**Systolic Pressure** – In relation to blood pressure, unless otherwise specified and because we typically measure blood pressure at a person's arm, the measured systolic pressure is that generated by contraction of the heart's left ventricle, which sends blood out of the heart and into the body.

**Diastolic Pressure** – In relation to blood pressure, diastolic pressure refers to the lowest pressure within the arterial blood stream during each Cardiac Cycle.

**BLOOD TESTS**

**Albumin Serum** – Protein produced in the liver. Responsible for maintaining oncotic pressure, flow of water into the circulatory system for filtration and excretion. Also responsible for transporting bilirubin and many drugs. Competitively binds calcium ions. Too much causes cells to dehydrate and too little causes cells to retain too much water. Low levels are caused by liver disease and excess kidney excretion. Excessive albumin is associated with high protein diets and chronic dehydration. Glomerulus (the kidneys' filtration devices) normally do not filter albumin because it is too large and negatively charged.

**ALK Phosphatase (ALP)** – Alkaline phosphatase is an enzyme responsible for removing phosphates including protein. High levels can indicate bile duct blockage. Should be viewed with GGT, since elevated GGT without elevated ALP indicates alcoholic liver disease and can indicate excess alcohol consumption in the preceding 3-4 weeks in some cases.

**Amylase** – Enzyme found in saliva and produced by the pancreas. Breaks down carbohydrates and fats. Normal level ranges from 0-137 U/L. Elevated level associated with pancreatic inflammation, kidney failure, alcohol abuse and dehydration. Helpful diagnostic marker since pancreas also produces insulin, thus elevated levels indicating pancreatic inflammations that may cause decreased insulin production and corresponding rise in blood glucose levels.

**ANION/GAP** – A calculation involving  $\text{Na}^+$ ,  $\text{Cl}^-$  and  $\text{HCO}_3^-$  (+/-  $\text{K}^+$ ) used to aid differential diagnosis of metabolic acidosis. Normal levels are between 3-11 mmol/L. Impaired renal function often changes the anion gap. High anion gap indicates lactic acidosis, ketoacidosis or renal failure caused by decreased acid excretion, decreased  $\text{HCO}_3^-$  reabsorption, and accumulation of sulfates ( $\text{SO}_4^-$ ), phosphates ( $\text{PO}_4^-$ ), urate and hippurate. Elevated anion gaps should be adjusted for abnormal albumin serum levels at the rate of approximately 2.3 times the change in albumin serum.

**BEecf** – Abbreviation for Base Excess Extracellular Fluid, a calculation using partial pressures of oxygen ( $\text{pO}_2$ ) and carbon dioxide ( $\text{pCO}_2$ ) and pH in the blood. The measurement evaluates the nonrespiratory component of the acid/base balance in clinically identifying cases of acidosis and alkalosis. Normal range is +/- 4 mmol/L. High levels indicate an excess of  $\text{HCO}_3^-$ , thus metabolic alkalosis. Low levels indicate a  $\text{HCO}_3^-$  deficiency, thus metabolic acidosis. Metabolic acidosis occurs when the body is producing too much acid or the kidneys are not removing enough acid through  $\text{HCO}_3^-$  production.

**Bilirubin** – Yellow byproduct of heme catabolism in the spleen. Heme is found in hemoglobin, the main component of red blood cells. Bilirubin is removed through liver functions and is excreted in bile and in rare instances, through urine.

**BUN** – Blood Urea Nitrogen. Should be read together with creatinine level. Basic measurement of renal function. Measures the amount of nitrogen present in the blood in the form of urea. Urea is a byproduct of protein metabolism in the liver and is removed by the kidneys. High levels are associated with impaired kidney function with extremely high levels indicating moderate to acute renal failure, as well as dehydration or shock, but can also result from very high protein diets. Elevated BUN in the presence of relatively normal creatinine levels may indicate physiological response to decreased blood flow to the kidneys resulting from heart dysrhythmia or dehydration without actual kidney damage. BUN will only be elevated outside of normal when more than 60% of kidney cells are no longer functioning. GFR and creatinine clearance are more accurate indicators of renal dysfunction.

**BUN/CREATININE Ratio** – Helpful in determining the reason for renal dysfunction because urea (BUN) and creatinine are both filtered by the glomerulus (kidneys' primary filtering structure), but urea reabsorption can be regulated while creatinine reabsorption remains relatively constant. Ratios of 10-20 are normal, but can also be postrenal disease. Low ratios are associated with liver dysfunction, excessive alcohol consumption or overhydration. Ratios less than 10 indicate intrarenal (within the kidney) or intrinsic kidney damage causing reduced reabsorption of BUN. Ratios greater than 20 indicate BUN reabsorption is increased and BUN is disproportionately elevated relative to creatinine in blood resulting in possible prerenal azotemia (decrease in blood flow to the kidneys caused by shock, decreased cardiac output or renal artery restriction without inherent kidney disease which can cause tachycardia). Ratios of 30 or higher strongly associate with upper gastrointestinal bleeding.

**CREATININE** – Is not Creatine, but is the byproduct of the breakdown of Creatine by muscle cells. Creatinine is removed by the kidneys with little

to no reabsorption, so elevated blood levels strongly indicate renal dysfunction. Elevated levels are observed only with notable nephronic damage. Best if tested and compared over time.

**Ca** – Calcium ion ( $\text{Ca}^{2+}$ ). Critically important signaling mechanism for many cellular processes via movement into or out of the cell's cytoplasm.  $\text{Ca}^{2+}$  together with  $\text{K}^{+}$  provides the ions required for cardiac cells' unique contraction mechanism. Low levels indicate renal dysfunction either through decreased reabsorption or diminished removal and excretion of  $\text{SO}_4$ , which in turn binds with the  $\text{Ca}^{2+}$  thereby decreasing the  $\text{Ca}^{2+}$  available for the heart and other cells' proper function.

**Cl** – Chloride ion ( $\text{Cl}^{-}$ ). Critical to proper cellular function. Reabsorbed through the kidneys. Low levels indicate renal dysfunction.

**CO<sub>2</sub>** – Typically carbon dioxide in the venous blood, which is most commonly used for laboratory testing, as opposed to arterial blood, which is used for pulmonary differential diagnosis by I-Stat or other arterial blood gas (ABG) immediate testing devices. Low levels in venous blood indicate impaired cardio-pulmonary function.

**CPK** – Creatine phosphokinase also called (CK). An enzyme found mainly in the heart, brain and skeletal muscle. Normal levels are 39-308 U/L. High levels indicate injury or stress to the heart, brain or muscle tissue. When muscles are damaged, CPK leaks into the bloodstream. Determining the specific type of CPK assists in diagnosing the damaged tissue. In skeletal muscle CK-MM is expressed at 98% with CK-MB expressed at only 1%. In myocardium (heart muscle) CK-MM is expressed at 70% with CK-MB expressed at 25-30%. Sarcomeric muscle, such as skeletal and cardiac muscle, expresses CK-MM, but CK-MB is almost exclusively expressed in cardiac muscle, thus making CK-MB a very good indicator of myocardial damage. Normal CK-MB levels are 0.2-5.0 ng/mL. High CK-MB levels indicate myocardial damage. Smooth muscle, like the vascular walls, certain layers of the aorta, small arteries, arterioles and veins, respiratory tract, and gastrointestinal tract, as well as, non-muscle tissue expresses CK-BB, thus making it a good contraindication of myocardial damage if CK-BB comprises the majority of the CPK value.

**GFR** – Glomerular Filtration Rate. Describes the flow rate of filtered fluid through the glomerular capillaries in the kidneys over time. Normal value is above 90 mL/min/1.73m<sup>2</sup>. Values of less than or equal to 59 mL/min/1.73m<sup>2</sup> may be associated with chronic kidney disease. Value of 60-89 mL/min/1.73m<sup>2</sup> evidences kidney damage. Value of 30-59 mL/min/1.73m<sup>2</sup> indicates moderate kidney damage. Value of 15-29 mL/min/1.73m<sup>2</sup> indicates severe kidney damage. Value of less than 15 mL/min/1.73m<sup>2</sup> indicates kidney failure.

**GGT/GGTP** – Gamma-glutamyltransferase or gamma-glutamyl transpeptidase. Found in many tissues, most notably the kidneys, liver, spleen, brain and heart. Provides a diagnostic marker for liver and kidney damage. Catalyzes the transfer of the gamma-glutamyl moiety of glutathione to an acceptor that may be an amino acid, a peptide or water (forming glutamate), thus leaving the cysteine product to preserve intracellular homeostasis of oxidative stress. Normal ranges from 40-78 U/L. Elevated levels are, like elevated ALK Phosphatase (ALP), associated with liver, biliary and pancreatic dysfunction/disease. GGT is also elevated by high alcohol intake. GGT should be viewed together with ALK Phosphatase (ALP) to confirm biliary disease, plus elevated GGT without elevated ALP indicates alcoholic liver disease and can indicate excess alcohol consumption in the preceding 3-4 weeks in some cases.

**Glucose** – Glucose. Normal level is 70-105 mg/dL. Used by the cells for aerobic or anaerobic energy production. Temporarily high levels could indicate stress, trauma, stroke, myocardial infarction, illness or recent alcohol ingestion if later accompanied by a drop in levels. Converted by the liver, adipose tissue and muscle cells and stored as glycogen for future re-conversion and use.

**HCO<sub>3</sub><sup>-</sup>** – Bicarbonate. Serves a critical role in pH buffering necessary to protect the body's tissues, especially those of the central nervous system, which are all highly sensitive to movement outside normal levels. Used during extreme cardiac emergencies to protect against the effects of acidosis in heart cells. Normal level is 22-26 mEq/L. Below 22 mEq/L is acidotic. Above 26 mEq/L is alkalotic. Should be read with pH level. If  $\text{HCO}_3^{-}$  is acidotic and pH is acidotic, then the acidosis is being caused by the metabolic or renal system. If  $\text{pCO}_2$  goes the opposite way from  $\text{HCO}_3^{-}$ , then the respiratory system is compensating. See BEecf above.

**HCT** – Hematocrit. Volume of red blood cells in 100 ml of blood expressed as a percentage. Evaluated in conjunction with Hgb. Most precise indicator of the severity of anemia or polycythemia. Low levels associated with decreased production, high loss or destruction of red blood cells, kidney and liver dysfunction. Increased levels associated with dehydration and testosterone supplementation.

**Hgb** – Hemoglobin. Levels must be evaluated in conjunction with HCT, RBC and MCV to determine the presence of anemia and its type. Increased levels associated with dehydration and testosterone supplementation. Decreased levels associated with tissue damage, kidney and liver dysfunction.

**INR** – International Normalized Ratio. Used to measure the patient's prothrombin time to a normal (control sample). Normal range is 0.9-1.3. High INR such as 5 indicates a high chance of bleeding. Low INR such as 0.5 indicates a high chance of clotting.

**K<sup>+</sup>** – Potassium ion. Used in opposition to  $\text{Na}^{+}$  to regulate the cells' action potential (electrostatic charge of a cell membrane).  $\text{K}^{+}$  is filtered and excreted by the kidneys in relation to the volume of  $\text{Na}^{+}$  present. The kidneys secrete  $\text{K}^{+}$  twice and reabsorb it three times before reaching the collecting tubules at which time it usually has the same  $\text{K}^{+}$  level as the blood, but if still too high then  $\text{K}^{+}$  is excreted. Minimum daily  $\text{K}^{+}$  excretion is 200 mg per day. High levels indicate tissue damage or destruction, renal dysfunction and extremely high levels indicating renal failure.

**Lactate** – An ion  $\text{CH}_3\text{CH}(\text{OH})\text{COO}^{-}$  formed by the loss of an ion from lactic acid. Constantly produced from pyruvate through the enzyme lactate dehydrogenase (LDH) during normal metabolic cell activity. Normal level is 1-2 mmol/L, but can rise to 20 mmol/L during intense exercise or exertion. Excess lactate is removed in several ways including oxidation to pyruvate from well oxygenated cells, then directly used to fuel the Krebs' cycle; or, conversion to glucose via gluconeogenesis in the liver and release back into the blood stream, as in the Cori cycle. High levels indicate metabolic acidosis.

**Lipase** – An enzyme found in saliva and also produced by the pancreas which breaks down fats. Normal level ranges from 12-70 U/L. Elevated level associated with pancreatic inflammation, kidney failure, alcohol abuse and dehydration. Since pancreas also produces insulin, pancreatic inflammations can cause decreased insulin production and corresponding rise in blood glucose levels.

**Lymphocyte** – A type group of white blood cells which include natural killer cells, T cells and B cells. Increased levels are associated with viral infections.

**MCV** – Mean Corpuscular Volume. Indicates the average size of the red blood cell and should be interpreted together with MCH. Increased levels associated with reticulocytosis (acute blood loss, reticulocytes are small, immature cells) and liver dysfunction. Decreased with vitamin B6 & C deficiency.

**MCH** – Mean Corpuscular Hemoglobin. Measures the amount of hemoglobin in a single red blood cell. Variant of the MCV measurement. Increased levels associated with reticulocytosis (acute blood loss, reticulocytes are small, immature cells) and liver dysfunction. Decreased with vitamin B6 & C deficiency.

**MCHC** – Mean Corpuscular Hemoglobin Concentration. A measure of the concentration of hemoglobin in a given volume of packed red blood cells.

**Mg<sup>2+</sup>** – Magnesium ion. Necessary for all life as the main source of cellular energy, ATP, must be bound to a Mg<sup>2+</sup> ion to be active. Over 300 enzymes require Mg<sup>2+</sup> for their catalytic action. Efficiently filtered and excreted or reabsorbed by kidneys. In cellular biology Mg<sup>2+</sup> is correlated with K<sup>+</sup> and holds onto water molecules more strongly than Ca<sup>2+</sup>, thus operate opposite from Ca<sup>2+</sup> and block Ca<sup>2+</sup> channels on the cell membranes. High levels are toxic to individual cells and are indicative of renal failure. Low levels cause muscle spasms, cardiovascular issues, migraines and cerebral infarctions.

**Monocytes, Absolute** – Type of white blood cell produced in the bone marrow and part of the immune system. Responsible for protecting cells from damaging foreign substances. High levels can indicate necrosis, severe infection and sarcoidosis. Low levels indicate impaired immuno-response.

**MPV** – Mean Platelet Volume. Measurement of the average size of platelets in blood sample. Average platelet size is larger when the body is increasing platelet production. Abnormally low MPV correlates primarily with thrombocytopenia.

**Myoglobin** – Iron and oxygen binding protein found in the muscle cells. Related to hemoglobin, which is the iron and oxygen binding protein found in blood. The only time myoglobin is found in the blood is when it is released following muscle cell destruction. Presence of myoglobin in the blood is an abnormal reading indicating muscle cell damage with high levels indicating severe damage. CK-MB is a better/more specific marker for myocardial/heart muscle cell damage. Myoglobin is filtered by the kidneys but is toxic to the epithelium of a portion of the nephron called the renal tubule, thus may cause acute renal failure. Naka T, Jones D, Baldwin I, *et al.* “Myoglobin clearance by super high-flux hemofiltration in a case of severe rhabdomyolysis: a case report.” *Critical Care* 2005;9:R90–R95. Renal tubules are the collecting tubes which transport urine to the ureters, reabsorb 99% of the water and secrete various ions such as Na<sup>+</sup>, glucose, and amino acids like glutamate with many cells having highly specialized functions, thus damage to these cells can cause severe dehydration. Presence of hazy, brown epithelial cells in urine is indicative of renal tubular damage from either exposure to a toxin such as myoglobin or ischemia, lack of blood flow/oxygen. However, ischemic renal tubular damage typically causes skip lesions through the tubules.

**Na<sup>+</sup>** – Sodium ion. Used in opposition to K<sup>+</sup> to regulate the cells’ action potential (electrostatic charge of a cell membrane). Na<sup>+</sup> is filtered and excreted by the kidneys. Decreases in blood pressure and decreases in Na<sup>+</sup> levels sensed by the kidneys stimulates production of renin, which generates aldosterone, which decreases the excretion of Na<sup>+</sup> in the urine. Retention of more Na<sup>+</sup> causes the body to retain more water. Na<sup>+</sup> is the chief cation in extracellular fluids and relatively little Na<sup>+</sup> actually resides inside the cells. Necessary to maintain the Na<sup>+</sup>/K<sup>+</sup>-ATPase pump.

**Neutrophils, Absolute** – Most abundant type of white blood cells. Generally known as a first responder for the body to fight infection and are highly anti-microbial in their function. Low counts are associated with aplastic anemia, leukemia and certain medications, such as chemotherapy. High levels indicate active microbial infection or cell injury.

**pCO<sub>2</sub>** – Partial Pressure of Carbon Dioxide. CO<sub>2</sub> is constantly produced as a byproduct of the body’s use of energy from glucose metabolism. Used in conjunction with pO<sub>2</sub> to determine the pulmonary efficiency, or rate of alveolar ventilation and perfusion. In simple terms, a measurement of the lung’s efficiency in exchanging oxygen for carbon dioxide. pCO<sub>2</sub>, along with pH, can be used to distinguish amount metabolic and respiratory acidosis or alkalosis. Respiratory acidosis describes decreased respiration causing increased carbon dioxide and decreased pH. Normal range is 35-45 mmHg. Below 35 mmHg is alkalotic. Above 45 mmHg is acidotic. If the pCO<sub>2</sub> is acidotic and the pH is acidotic, then the acidosis is being caused by the respiratory system. If the HCO<sub>3</sub><sup>-</sup> goes the opposite way from pCO<sub>2</sub>, then the metabolic or renal system is compensating.

**pH** – Measure of acidity or basicity of a substance. The body’s pH is closely regulated by acid-base homeostasis with blood normally having a pH of 7.365. Should be read together with HCO<sub>3</sub><sup>-</sup> and pCO<sub>2</sub> to determine source of imbalance. See HCO<sub>3</sub><sup>-</sup> above for interpretation of source. See BEecf above.

**PO<sub>4</sub><sup>3-</sup>** – Phosphate. Major intracellular anion., thus rhabdomyolysis and lactic acidosis cause extremely elevated levels sufficient to overwhelm the kidneys. Elevated levels associated with renal dysfunction or failure and lowered Ca<sup>2+</sup> levels due to Ca<sup>2+</sup>/PO<sub>4</sub><sup>3-</sup> precipitation in tissues. Hypoparathyroidism reduces levels of parathyroid hormone (PTH), which prevents renal absorption of PO<sub>4</sub><sup>3-</sup>, thus elevating PO<sub>4</sub><sup>3-</sup> levels due to increased reabsorption. Calcium Carbonate (CaCO<sub>3</sub>), calcium acetate (C<sub>4</sub>H<sub>6</sub>CaO<sub>4</sub>) and calcium acetate/magnesium carbonate can be used as a binder to reduce PO<sub>4</sub><sup>3-</sup> in the presence of renal failure.

**Platelet Count** – Fragments of cells which participate in clotting and initiate repair of blood vessel walls. Are considered an acute phase reactant to infection or inflammation. Increased levels associated with infection & tissue damage. Decreased levels associated with liver dysfunction & excessive hydration.

**Prothrombin** – Prothrombin time (PT). Measures blood’s extrinsic pathway of coagulation. Together with PR and INR determine blood’s clotting tendency, liver damage and vitamin K status. Prolonged PT can be caused by disseminated intravascular coagulation (DIC) (pathological activation of coagulation response caused by transmembrane glycoprotein called tissue factor (TF) coming into contact with general blood circulation. TF is normally not present in general circulation but results from massive tissue and cell destruction, such as in extreme rhabdomyolysis. DIC leads to formation of small blood clots inside the blood vessels, which disrupt the blood flow to the organs such as kidneys, causing dysfunction. DIC evidenced by prolonged prothrombin and partial prothrombin time with decreased platelet count.)

**PTT** – Activated partial thromboplastin time. Measures the efficacy of both the contact activation pathway (intrinsic pathway) and common coagulation pathways. Normal time is 23-36.9 seconds. Used in conjunction with PT. Lower times generally have little clinical relevance. Higher times require further testing to determine cause such as presence of “inhibitor” like heparin or a factor deficiency.

**RBC** – Red Blood Cell count. Checks for anemia and evaluates normal erythropoiesis (production of red blood cells). Mature red blood cells carry oxygen attached to iron in the myoglobin. The number of red blood cells is affected by many things including, liver and kidney function. Increased values are associated with anabolic metabolism (testosterone use) and decreased values are associated with liver and kidney dysfunction and catabolic metabolism.

**RBS** – Random Blood Sugar. Measures the glucose level in blood.

**RDW** – Random Distribution of Width. Measures the consistency of the size of red blood cells. To identify the anemic components of blood, you look for reticulocytes, as well as, B12 and folic acid deficient cells which are larger than iron deficient cells.. Increased RDW level is associated with B12 and folic acid deficiency and with alcohol abuse. Decreased RDW levels are associated with B6 and iron deficiency anemia.

**Serum Protein** – Measures total blood protein, both albumin and globulin. Albumin is produced in the liver and globulin is produced by the liver and immune system. Globulins combine with hemoglobin and help fight infection.

**SGOT (AST)** – Serum Glutamic Oxaloacetic Transaminase (SGOT) also called Aspartate Transaminase (AST). AST is found in the liver, heart, skeletal muscle, kidneys, brain and red blood cells. Elevated levels are associated with damage or disease in the heart, kidneys and musculoskeletal

system.

**SGPT (ALT)**– Serum Glutamic Pyruvic Transaminase (SGPT) also called Alanine Transaminase (ALT). Most commonly found in the liver. High levels indicate congestive heart failure, liver damage, bile duct obstructions and myopathy; however, ALT levels are significantly increased in response to extreme physical exercise. Best if used to narrow the source through analyzing ALT, alkaline phosphate and Creatine kinase levels together.

**Troponin** – Either TnC, TnI or TnT Complex of proteins required for contraction of skeletal and cardiac muscle, but not smooth muscle like walls of the blood vessels, certain layers of the aorta, small arteries, arterioles and veins, respiratory tract, and gastrointestinal tract. There are three types of troponin and each is specifically associated with the type of muscle in which it is found. Troponin is a component of thin filaments (along with actin and tropomyosin), and is the protein to which  $Ca^{2+}$  binds to regulate the action. Troponin has three subcomponents, TnC, TnI, and TnT. When  $Ca^{2+}$  is bound to specific sites on TnC, tropomyosin moves out of the way of the actin filament active sites to allow myosin (a molecular motor found in muscle thick filaments) to attach to the thin filament and produce force and/or movement. In the absence of  $Ca^{2+}$ , tropomyosin interferes with the myosin bonding process, thereby preventing contraction and forcing the muscle cell to remain relaxed. Troponin I is an inhibitor of cellular contraction and is a highly specific marker for cardiac muscle cell damage with higher levels indicating severe damage. Troponin's variants perform different specific functions as follows: Troponin C binds to  $Ca^{2+}$  to produce a conformational change in TnI; Troponin T binds to tropomyosin, interlocking them to form a troponin-tropomyosin complex; Troponin I binds to actin in thin myofilaments to hold the troponin-tropomyosin complex in place.

**WBC** – White Blood Cell. Measures the total of all white blood cells present in the blood sample. When done with a differential, it will include neutrophil granulocytes (may indicated infection), lymphocytes (higher with some types of viral infections and chronic lymphocytic leukemia or decreased by HIV), monocytes (increased in bacterial infections); eosinophil granulocytes (increased in parasitic infections, asthma and allergic reactions) and basophil granulocytes (increased in bone marrow conditions such as leukemia or lymphoma.)

### URINE TEST

**Appearance** – Normally clear. Many factors affect color as discussed elsewhere in these definitions. See Bilirubin.

**Bacteria** – Normally not present. Presence indicates high level infection.

**Bilirubin** – Yellow byproduct of heme catabolism in the spleen. Heme is found in hemoglobin, the main component of red blood cells. Bilirubin is removed through liver functions and is excreted in bile and in rare instances, through urine. Normally, bilirubin is not found in the urine, but if the liver is not functioning properly or the bile ducts are blocked, conjugated bilirubin leaks from the hepatocytes and appears in urine, giving the urine a dark amber color.

**Glucose** – See above for glucose levels and meanings.

**Ketones** – May be present if body is in ketosis.

**Leuk. Esterase** – Positive results from presence of white blood cells, either whole or destroyed. A negative result means that an infection is unlikely.

**Mucous** – Normally not present. Presence gives urine a cloudy color and is associated with infections or abnormalities such as kidney stones or STDs.

**Nitrites** – Positive nitrite tests means bacteria may be present in significant numbers.

**pH** – Initial filtrates from the glomerular tubes is usually acidified from pH of 7.4 to 6. One of the kidneys' functions is ridding the body of acid.

**Protein** – Typically not found in urine because the kidneys reabsorb it rather than filter it for excretion. Presence in urine indicates either impaired absorption or filtration functions. Causes can include damage to the kidneys' glomerulus (filtering mechanisms) and too much albumin in the blood. Levels of 300 mg/dL indicate severe kidney dysfunction.

**RBC** – Red Blood Cells. Normally, very few, if any, should be present in urine. High numbers of RBC indicate glomerular damage, kidney trauma, renal infarcts, acute tubular necrosis and physical stress on the body's systems as is associated with contact sports or long distance running. The presence of poorly shaped (dysmorphic) RBCs indicates glomerulonephritis, as the RBCs are distorted by passing through abnormal glomerular drainage structures.

**SG** – Specific Gravity. Normally between 1.003 and 1.035 g/cm<sup>3</sup>. Values over or under indicate urinary or renal disorders.

**Urobilinogen** – Colorless byproduct of bilirubin reduction. Some is reabsorbed by the kidneys for reuse in the body. High levels indicate haemolysis or liver disease. Low levels indicate biliary obstruction.

**WBC** – White Blood Cells. Normally not found in urine. See RBC above.

## **B. DETAILED EVENTS OF FRIDAY-28-JAN-2011**

**16:15 hours** – Jones left El Dorado gym riding with team mates to Benton for weigh-ins.

**16:30 hours until arrival in Benton** – Jones consumed one new container of Peach Timberwolf snuff and one new container of Red Seal – Natural snuff with his roommate on the drive between El Dorado and Benton.

**19:45 hours** -- Jones weighed in on the scales at 233.5 lbs (106 kg), but Commissioner Stuart certified the scales used for weigh-ins to weigh 9/10ths of a pound light, so Jones' actual weight was 232.5 lbs. (105 kg). Event scales were certified using a known, standard weight of 35 lbs (dumbbell).

**20:00 hours until unknown time** – Jones ate with team mates at Chili's restaurant in Benton then returned to his hotel. Jones consumed steak & ribs with broccoli at Chili's. Jones consumed an unknown quantity of tap water with his meal. Jones hung out with his roommate and friend in the hotel room the rest of the evening. Jones consumed no other food that night, but consumed an unknown quantity of water from his personal water cooler in the room. [NOTE: According to the United States Department of Agriculture, the minimum Potassium content of Jones' meal was 1,771 mg. calculated as follows: 6 oz. beef steak (572 mg), 6 oz. pork ribs (742 mg), one cup of broccoli (457 mg). The importance of this fact will become more evident later in this report.]

## **C. DETAILED EVENTS OF SATURDAY-29-JAN-2011**

**03:30 hours to 04:00 hours** – Jones went to sleep.

**09:00 hours** – Jones woke up.

**09:20 hours** – Jones ate breakfast at his hotel. Jones consumed an unknown quantity of biscuits & gravy, scrambled eggs and possibly some sausage. Jones drank unknown quantity, but at least two cups each, of apple juice, orange juice and coffee during breakfast. [NOTE: According to the United States Department of Agriculture, the minimum Potassium content of Jones' meal was 2,175 mg. calculated as follows: Two biscuits (42 mg), ¼ cup of beef gravy (47 mg), two cups of orange juice (992 mg), two cups of apple juice (590 mg), six large scrambled eggs (504 mg). If Jones also consumed two pork sausage patties, then we would also need to add 158 mg. to the total. The importance of this fact will become more evident later in this report.]

**11:00 hours** – Jones returned to his hotel room from breakfast and hung out.

**12:00 hours** – Jones consumed a 90 mg Potassium supplement obtained from roommate.

**12:00 hours until 16:15 hours** -- Jones was unobserved by roommate while roommate went shopping for clothes and Jones stayed behind to take a nap. It is unknown whether Jones actually took a nap. Upon the return of Jones' roommate at 16:15 hours, Jones was doing light exercises/warming up with his trainer. It is unknown whether Jones ate or drank anything during this time period.

**16:15 hours** – Jones performed a light workout/warm-up with his trainer. The team went to the Event Venue after completion of their workout/warm-up sessions.

**17:00 hours** – Jones arrived at Event Venue and participated in Contestants’ meeting and completed paperwork for his Pre-Bout Physical.

**17:45 to 18:00 hours** – Jones took and passed his physical exam given by the Ringside Physician using the Commission’s Pre-Bout Form. The Ringside Physician noted Jones’ **blood pressure as 142/86** and the presence of a small reducible umbilical hernia. Following his physical, Jones was approved by the Ringside Physician to participate in the Event.

**18:00 to 18:15 hours** – Jones passed a breathalyzer test given by Commissioner Stuart using an Intoximeter IV portable breath tester which is calibrated every six months by the Arkansas State Police (“ASP”). Due to insufficiency of Commission funds, the Intoximeter IV was provided to the Commission by the ASP via an agency to agency property transfer.

**18:00 to 21:00 hours** – Jones ate one medium size banana. [NOTE: According to the United States Department of Agriculture, one small size banana contains 422 mg of Potassium, thus it is assumed for purposes of this report that Jones’ medium size banana contained approximately 561 mg of Potassium. The importance of this fact will become more evident later in this report.]

**21:40:00 hours** – Contestants enter the ring.

**21:42:00 hours** – Bell rings to start 1<sup>st</sup> Round.

**21:45:00 hours** – Bell rings to end 1<sup>st</sup> Round. **1<sup>st</sup> Round Punch Statistics:** JONES received 8 punches to the head (2 jabs and 6 power punches) and no body shots; PALMER received 64 punches to the head (15 jabs and 49 power punches) and 6 body shots while being Downed once and receiving one Standing Eight Count. At the end of the 1<sup>st</sup> Round, Jones was ahead on all judges’ scorecards with Judge #1 scoring 10-8, Judge #2 scoring 10-8 and Judge #3 scoring 10-9.

**21:46:28 hours** – Rest Period ends with Seconds being cleared of the Ring. (NOTE: The 1 minute rest period was extended by approximately 25 seconds due to the Ringside Physician checking the physical condition of Palmer, who had taken significant head shots from Jones, had been Downed and was suffering a nose bleed at the end of the 1<sup>st</sup> Round.)

**21:46:33 hours** – Bell rings to start 2<sup>nd</sup> Round. **2<sup>nd</sup> Round Punch Statistics:** JONES received 26 punches to the head (1 jab and 25 power punches) and 3 body shots; PALMER received 18 punches to the head (2 jabs and 16 power shots) and 2 body shots.

**21:49:30 hours** – Following a right cross from Palmer, Jones hit the canvas approximately 3 seconds before the end of the 2<sup>nd</sup> Round. As Jones fell he hit his head on the padded canvas floor of the Ring. Referee Tunstall picked up the Count from the Timekeeper at 2 seconds and continued the Count. Between the Counts of 4 through 8, Jones attempted to get up; however, Jones returned to the canvas after the Count of 8 and Referee Tunstall finished the Ten Count without Jones rising from the canvas. In accordance with Commission Regulations, the Bell never rang to signal the end of the 2<sup>nd</sup> Round because Jones did not return to his feet before the end of the Ten Count. The Bout was officially scored as a Knockout win for Palmer at the end of the 2<sup>nd</sup> Round.

**21:49:41 hours** – Referee Tunstall concludes the Ten Count and calls an end to the Bout. The Ringside Physician and RN, who had both arisen from their seats and ascended the stairs to stand on the Ring apron during the Count, immediately enter the Ring to check Jones’ condition.

**21:49:44 hours** – Just three (3) seconds after the Bout was ended and fourteen (14) seconds after Jones hit the canvas, the Ringside Physician and RN are both at Jones' side evaluating his condition. During the initial assessment Jones was dazed and experiencing transient confusion, but verbally responsive and experienced no loss of consciousness. Ringside Physician did not request EMT presence inside the Ring; however, EMTs were in position with their equipment at Ringside.

**21:50:35 hours** – Ringside RN administers a pen light pupillary test and continues assessment while Jones remains on the canvas. Jones has reactive pupils and continues to be verbally responsive with transient confusion and memory deficit. Jones still has not and does not during this incident experience a loss of consciousness.

**21:51:52 hours** – Jones sits up and continues receiving treatment from Ringside Physician and RN.

**21:53:30 hours** – Jones stands up and is talking to persons around him including Ringside medical staff.

**21:54:30 hours** – Jones raises his hand and congratulates Palmer on the victory, then continues talking with Ringside medical staff. Jones is still a little dizzy and confused, but is otherwise not displaying any signs of life threatening injuries.

**21:56:00 hours** – Ringside Physician continues to assess and evaluate Jones' condition while inside the Ring.

**21:59:23 hours** – Jones was oriented times three, had reactive but sluggish and pinpoint pupils and failed the 3 names/3 objects memory test at 5 minutes. Ringside medical staff's initial assessment is that Jones has suffered an American Academy of Neurology guidelines Grade II Concussion, as generally evidenced by symptoms including transient confusion and memory deficit likely to last longer than 15 minutes with no loss of consciousness. No readily observable conditions indicate Jones has any life threatening injury; however, the Ringside Physician exercises an abundance of caution and exceeds standard medical protocols by requesting Ringside EMTs to transport Jones to Saline Memorial Hospital ("SMH") via ambulance for further precautionary observation and testing. Ringside EMTs and Physician begin preparing Jones for transport via ambulance. Jones' preparation includes backboard, cervical collar, etc.

**21:59:23 hours until 22:09 hours** -- Ringside EMTs and Physician prepared patient for transport via ambulance, loaded into ambulance, continued patient assessment and provision of further stabilization treatment detailed below.

**22:10 hours** -- EMTs provide O<sub>2</sub> at 15 Flow Rate and start Normal Saline IV using 18 gauge, Right Femoral, Saline Lock, Patient Response Unchanged with Confusion. Person, Other Person and Place Oriented, Able to Verbalize and Follow Commands.

**22:22:58 hours** -- Ambulance departs Venue en route to SMH. En route patient began showing signs of possible decorticate posturing, tensing both arms inward then relaxing them. Left eye would drift to the left while right eye maintained forward looking gaze. Both eyes were reactive, but sluggish and pinpoint. Respirations slowed and became shallow, but remained bi-laterally clear.

22:23 hours –

<b><u>VITAL SIGNS</u></b>
<b>Blood Pressure</b> – 101/53 A (110/65 – 140/90) <b>Heart Rate</b> – 80 R (60-80) <b>Respiratory Rate</b> – 16 R (16-18)
<b><u>COMMISSION INTERPRETATION</u></b>
At approx. 18:00 hours, Jones blood pressure was 142/86. At just 34 minutes after the end of the Bout, Jones' body was showing signs of distress of an unknown origin with heart and respiratory rates being within their normal ranges, but inconsistent with the low cardiac output evidenced by his below normal blood pressure. Slightly low systolic pressure is almost unremarkable, but in retrospect we can see contraction of Jones' heart's left ventricle, which sends blood out of the heart and into the body, was weakened by the electrolyte imbalance reflected in Jones' blood test results at 22:45 hours. Very low diastolic pressure further reflects the contraction/relaxation irregularities of Jones' cardiac cycle.

22:26:04 hours -- Ambulance arrives at SMH. **(Total Venue to Facility Transport Time 3 minutes 6 seconds covering 9/10ths of a mile.)**

22:27 hours -- SMH ER nurse begins triage assessment.

<b><u>VITAL SIGNS</u></b>
<b>Blood Pressure</b> – 142/78 A (110/65 – 140/90) <b>Heart Rate</b> – 118 R (60-80)
<b><u>COMMISSION INTERPRETATION</u></b>
At 38 minutes after the end of the Bout, Jones' body was still showing signs of distress of an unknown origin with his blood pressure having returned to normal from its "low" reading just 4 minutes earlier; however, Jones' heart rate has remarkably increased from its reading only 4 minutes earlier. Jones' vitals are consistent with his body's compensating response for low cardiac output/early stage cardiac dysrhythmia, primarily suspected tachycardia based upon a heart rate over 100 bpm. Jones' lower diastolic blood pressure is caused by an electrolyte imbalance causing heart to become too relaxed during diastole phases.

22:30 hours -- Cardiac monitor applied and provision of additional O<sub>2</sub>.

22:35 hours -- Blood draw for stat labs.

22:45 hours –

<u>BLOOD TEST RESULTS</u>	<u>COMMISSION INTERPRETATION/INDICATION</u>
<p><b>RBS/Glucose</b> – 224 H (70-105 mg/dl) <b>BUN</b> – 19 H (6-20 mg/dl) <b>CREATININE</b> – 2.7 H (0.5-1.1 mg/dl) <b>BUN/CREAT Ratio</b> – 7 (5-37 CALC) <b>Na<sup>+</sup></b> – 149 H (135-145 mmol/L) <b>K<sup>+</sup></b> – 5.4 H (3.5-5.0 mmol/L) <b>Cl<sup>-</sup></b> – 101 (100-109 mmol/L) <b>CO<sub>2</sub></b> – 11 L (22-31 mmol/L) <b>ANION/GAP</b> – 42 H (5-25 mmol/L) <b>Ca<sup>2+</sup></b> – 12 H (8.6-10.2 mg/dl) <b>CPK</b> – 1463 H (39-308 U/L) <b>Bilirubin</b> – .54 (0.20-1.2 mg/dl) <b>Serum Protein</b> – 9.4 H (6.4-8.2 g/dl) <b>Albumin Serum</b> – 5.1 H (3.4-5.0 g/dl) <b>SGOT(AST)</b> – 73 H (15-41 IU/L) <b>SGPT (ALT)</b> – 71 H (5-45 IU/L) <b>ALK Phosphatase</b> – 122 (50-136 U/L) <b>WBC</b> – 8.3 (3.0-12.0 K/uL) <b>RBC</b> – 5.46 (4.5-6.0 M/uL) <b>HEMOGLOBIN</b> – 17.3 (13.5-17.5 g/dL) <b>HEMATOCRIT</b> – 54.4 H (40-52%) <b>MCV</b> – 99.6 H (79-96 fL) <b>MCH</b> – 31.8 (27-36 PG) <b>MCHC</b> – 31.9 (30-36 g/dL) <b>RDW</b> – 14.8 H (11.0-14.0%) <b>Platelet Count</b> – 240 (150-500 K/uL) <b>MPV</b> – 9.9 (8.1-12.8 fL)</p>	<p>Just 55 minutes after the end of the Bout, Jones already showing signs of severe Exertional Rhabdomyolysis, renal dysfunction with likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration</p> <p><b>EVIDENCED BY:</b> Extreme fatigue/lethargy/heart dysrhythmia; High RBS/Glucose (indicates body still in energy generation phase, Mild Traumatic Brain Injury or kidney/liver dysfunction); BUN high end of range (possible damaged kidneys); Creatinine elevated to twice the upper end of normal; BUN/CREA Ratio of less than 10; High Na<sup>+</sup> (Na<sup>+</sup>/K<sup>+</sup>-ATP Pump impairment); High K<sup>+</sup> (muscle cell destruction) (Na<sup>+</sup>/K<sup>+</sup>-ATP Pump impairment); High Ca<sup>2+</sup> (associated w/rebounding effect after rhabdomyolysis &amp; renal failure); Low CO<sub>2</sub>; with Cl<sup>-</sup> still normal; Extremely High CPK 5 times the upper end of normal resulting from muscle &amp; kidney cell damage; Elevated Protein associated w/muscle cell damage; High SGOT (AST) levels more than 2 times upper end of normal (indicating muscle, heart, kidney damage); High SGPT (ALT) levels associated with physical exertion when viewed with CPK and ALK Phosphatase; Hgb &amp; HCT elevated (indicating dehydration); and High MCV with normal MCH &amp; MPV, but low MCHC.</p>

<u>URINE TEST RESULTS</u>	<u>COMMISSION INTERPRETATION/INDICATION</u>
<p><b>Appearance</b> – HAZY Abnormal (Clear) <b>Bilirubin</b> – NEG (Neg.)</p>	<p>Haziness, probably caused by renal tubular epithelial cells damaged from the myoglobin released by muscle cell destruction (rhabdomyolysis). Consistent w/blood indicators. Neg. bilirubin indicates proper liver function.</p>

22:48 hours -- SMH makes decision to transfer to UAMS for further treatment.

23:06 hours -- CT Brain Scan Routine w/out IV Contrast - No mass or mass artifact; No intracranial hemorrhage; Ventricles non-dilated; Lytic lesion involving right occipital bone, etiology unknown, recommend bone scan - 2.2 cm lytic lesion right occipital bone just posterior to the mastoid air cells should be correlated to bone scan.

23:09 hours -- CT Cervical Spine Routine w/out IV Contrast - Normal alignment; No fracture visible; No significant degenerative change.

23:15 hours -- Normal Saline IV at Flow Rate 1000

23:30 hours -- Foley Cath inserted (dark amber colored urine) (specimen sent stat lab)

23:45 hours – SMH provides UAMS with transfer report.

23:58 hours -- Ambulance departs SMH en route to UAMS.

## D. DETAILED EVENTS OF SUN-30-JAN-2011

00:08 hours –

<u>VITAL SIGNS</u>
<b>Blood Pressure</b> – 140/78 A (110/65 – 140/90) <b>Heart Rate</b> – 120 R (60-80)
<u>COMMISSION INTERPRETATION</u>
At 2 hours 19 minutes after the end of the Bout, Jones' body was showing all the signs of severe Exertional Rhabdomyolysis, renal dysfunction with likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration.
<b>EVIDENCED BY:</b> Jones' blood test results at 22:45 hours coupled with normal blood pressure reading and an elevated heart rate over 100 bpm. Jones' vitals are consistent with a compensating response for low cardiac output/early stage cardiac dysrhythmia, primarily suspected tachycardia based upon a heart rate over 100 bpm. Jones' lower diastolic blood pressure is caused by an electrolyte imbalance causing heart to become too relaxed during diastole phases.

00:20 hours – Arrival at UAMS ER. (Total Facility to Facility Transport Time 22 minutes covering 23.1 miles).

00:25 hours – Normal Saline IV – 18 gauge – Flowed 2000 mL; Glasgow Coma Scale score 15 – Mildest Concussion/MTBI – Eyes = spontaneous (4); Best Verbal Response = oriented (5); Best Motor Response = obeys command (6);

<u>VITAL SIGNS</u>
<b>Blood Pressure</b> – 105/85 A (110/65 – 140/90) <b>Heart Rate</b> – 116 R (60-80) <b>Respiratory Rate</b> – 38 (16-18) <b>Temperature</b> – 98 (97-98) <b>SpO2</b> – 95% (94-100) <b>Pupillary Dilation (L&amp;R)</b> – 3 cm (3-5 cm) <b>Abdomen</b> – Poor Rectal Tone (Normal) <b>Right Upper Extremity Movement</b> – Positive <b>Right Upper Extremity Sensation</b> – Positive <b>Left Upper Extremity Movement</b> – Positive <b>Left Upper Extremity Sensation</b> – Positive <b>Right Lower Extremity Movement</b> – Negative <b>Right Lower Extremity Sensation</b> – Negative <b>Left Lower Extremity Movement</b> – Negative <b>Left Lower Extremity Sensation</b> – Negative
<u>COMMISSION INTERPRETATION</u>
At 2 hours 36 minutes after the end of the Bout, Jones' body was continuing to evidence signs of severe Exertional Rhabdomyolysis, renal dysfunction with likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration. Now lower systolic blood pressure and slightly higher, but still low, diastolic pressure indicate Jones' heart is experiencing difficulty compensating for the extreme electrolyte imbalance shown by Jones' blood test results at 22:45 hours.
<b>EVIDENCED BY:</b> Jones' blood test results at 22:45 hours (High Na <sup>+</sup> (Na <sup>+</sup> /K <sup>+</sup> -ATP Pump impairment); High K <sup>+</sup> (muscle cell destruction) (Na <sup>+</sup> /K <sup>+</sup> -ATP Pump impairment); High Ca <sup>2+</sup> (associated with rebounding effect after rhabdomyolysis & renal failure)), coupled with slightly low blood pressure just after the end of the Bout, which then returned to normal and is now remarkably lower than the reading just 17 minutes earlier coupled with a still elevated heart rate over 100 bpm. Respiratory rate is now highly elevated above normal. Jones' vitals including his now very elevated respiratory rate are consistent with a compensating response for low cardiac output/early stage cardiac dysrhythmia, primarily suspected tachycardia based upon a heart rate over 100 bpm.

**00:30 hours** – **Tachycardia noted & Dr. advised**; Normal Saline IV – 18 gauge – Flowed 2000 mL; Glasgow Coma Scale score of 15 – Mildest Concussion/MTBI – Eyes = spontaneous (4); Best Verbal Response = oriented (5); Best Motor Response = obeys command (6); Complaining of generalized pain all over @ pain level of nine (9).

<u>VITAL SIGNS</u>
<b>Blood Pressure</b> – 92/80 A (110/65 – 140/90) <b>Heart Rate</b> – 114 R (60-80) <b>Respiratory Rate</b> – 37 (16-18) <b>Temperature</b> – 97.7 <b>SpO2</b> – 95% (94-100) <b>Pupillary Dilation (L&amp;R)</b> – 3 cm (3-5 cm)
<u>COMMISSION INTERPRETATION</u>
At 2 hours 41 minutes after the end of the Bout, Jones’ body was continuing to evidence signs of severe Exertional Rhabdomyolysis, renal dysfunction with likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration. Both systolic and diastolic blood pressures are continuing to fall in response to the electrolyte imbalance resulting from the Exertional Rhabdomyolysis.  <b>EVIDENCED BY:</b> Jones’ blood test results at 22:45 hours coupled with slightly low blood pressure just after the end of the Bout, which then returned to normal and is now remarkably lower than reading 5 minutes earlier. Elevated heart rate over 100 bpm and continued respiratory rate elevation. Jones’ vitals are consistent with compensating response for low cardiac output/early stage cardiac dysrhythmia, suspected tachycardia based on heart rate over 100 bpm.

**00:40 hours** –Glasgow Coma Scale score of 15 – Mildest Concussion/MTBI – Eyes = spontaneous (4); Best Verbal Response = oriented (5); Best Motor Response = obeys command (6)

<u>VITAL SIGNS</u>
<b>Blood Pressure</b> – 84/38 A (110/65 – 140/90) <b>Heart Rate</b> – 108 R (60-80) <b>Respiratory Rate</b> – 45 (16-18) <b>SpO2</b> – 100% (94-100) <b>Pupillary Dilation (L&amp;R)</b> – 3 cm (3-5 cm)
<u>COMMISSION INTERPRETATION</u>
At 2 hours 51 minutes after the end of the Bout, Jones’ body was showing increased effects of severe Exertional Rhabdomyolysis, renal dysfunction with likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration. Both systolic and diastolic blood pressures are continuing to fall and now at critically low levels in response to the electrolyte imbalance resulting from the Exertional Rhabdomyolysis.  <b>EVIDENCED BY:</b> Jones’ blood test results at 22:45 hours coupled with blood pressures, which fell just after the end of the Bout, returned to normal, and now are remarkably lower than each preceding reading within the last hour. Still elevated heart rate over 100 bpm now coupled with increasing and highly elevated respiratory rate. Jones’ vitals are consistent with a compensating response for low cardiac output/early stage cardiac dysrhythmia, primarily suspected tachycardia based upon a heart rate over 100 bpm.

**00:44 hours** – During interview and examination by emergency room physician complained of headache and generally hurting all over. Constantly asked for water.

00:49 hours –

<u>BLOOD TEST RESULTS:</u>	<u>COMMISSION INTERPRETATION/INDICATION</u>
<p><b>Na<sup>+</sup></b> – 131 L (135-145 mmol/L) <b>K<sup>+</sup></b> – &gt;8.9 CH (3.5-5.0 mmol/L) <b>Cl<sup>-</sup></b> – 110 H (100-109 mmol/L) <b>CO<sub>2</sub></b> – 12 L (21-32 mmol/L) <b>BUN</b> – 31 H (6-20 mg/dl) <b>CREATININE</b> – 3.4 H (0.6-1.3 mg/dL) <b>Glucose</b> – 184 H (70-105 mg/dL) <b>pH</b> – 7.2 L (7.35-7.45) <b>Hct/HEMATOCRIT</b> – 51 (40-52%) <b>Hgb/Hb/HEMOGLOBIN</b> – 17.3 (14-18 g/dL) <b>GFR</b> – 26 L (&gt;90 mL/min/1.73m<sup>2</sup>) <b>WBC</b> – 14.12 H (3.0-12.0 K/uL) <b>RBC</b> – 5.5 (4.5-6.0 M/uL) <b>MCV</b> – 90 (79-96 FL) <b>MCH</b> – 31.1 (27-36 pg) <b>MCHC</b> – 34.6 (30-36 g/dL) <b>RDW</b> – 13.1 (11.0-14.0%) <b>Platelet Count</b> – 276 (150-500 K/uL) <b>MPV</b> – 11.1 (8.1-12.8 fL) <b>Neutrophils Absolute</b> – 6.7 (2.5-8.2 K/uL) <b>Lymphocytes, Absolute</b> – 5.6 H (1.0-4.8 K/uL) <b>Monocytes, Absolute</b> – 0.5 (0.1-1.0 K/uL) <b>Prothrombin</b> – 16.5 H (12.0-14.0 sec) <b>INR</b> – 1.3 H (0.9-1.3) <b>PTT</b> – 39.5 H (23-36.9 sec) <b>Amylase</b> – 399 H (36-128 U/L)</p>	<p>Three (3) hours after end of the Bout, Jones had severe renal dysfunction, most likely due to effects of exertional rhabdo including necrosis of nephrons' renal tubules due to myoglobin toxicity and hyperkalemia. Likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration. Cascading failure of organ systems clearly in process.</p> <p><b>EVIDENCED BY:</b> Now low Na<sup>+</sup> - Was elevated (149) 2 hrs earlier (indicates renal tubules' failure to excrete Na<sup>+</sup> with ↑ K<sup>+</sup> forcing change in Na<sup>+</sup>/K<sup>+</sup>-ATP Pump); Now higher K<sup>+</sup> - Was 5.4 2 hrs earlier (indicates continued muscle cell damage w/out adequate compensation/filtering by kidneys; levels associated with critical imbalance) Cl<sup>-</sup> now elevated (possibly kidneys are reabsorbing); BUN now extremely high – Was high (19) 2 hrs earlier (indicates severe kidney damage to more than 60% of kidneys' nephron/cell structure); Creatinine still rising – Was high (2.7) 2 hrs earlier (indicates notably increasing nephronic damage); Glucose still high – Was higher (224) 2 hrs earlier (indicates body coming out of energy production phase but possible kidney/liver dysfunction); pH low (indicates acidosis); Hgb &amp; HCT still elevated upper end of range (dehydration indicated); GFR extremely low (indicates severe kidney damage and lack of blood flow/filtration); WBC &amp; Lymphocytes high (indicates body's infection response, likely due to sensing of cell destruction/attack); MCV now back within normal; High Prothrombin &amp; INR with PTT at high end of range (indicates possible disseminated intravascular coagulation); and Amylase extraordinarily high (indicates kidney failure &amp; dehydration w/high glucose also indicating possible pancreatic dysfunction).</p>

00:57 hours – Chest X-ray finds trachea midline. The aortic arch is not enlarged. Cardiomedial silhouette is within normal limits. The port is for effort identified. No focal consolidation or pleural fluid is noted. No fractures of the osseous structures are noted. Pelvic X-ray finds no acute fractures or dislocations. Hip joints appear well maintained. No diastasis of SI joints or pubic symphysis noted. Soft tissues grossly unremarkable.

01:00 hours – Glasgow Coma Scale score of 15 – Mildest Concussion/MTBI – Eyes = spontaneous (4); Best Verbal Response = oriented (5); Best Motor Response = obeys command (6)

<u>VITAL SIGNS</u>
<p><b>Blood Pressure</b> – 97/29 A (110/65 – 140/90) <b>Heart Rate</b> – 105 R (60-80) <b>Respiratory Rate</b> – 26 (16-18) <b>SpO<sub>2</sub></b> – 100% (94-100) <b>Pupillary Dilation (L&amp;R)</b> – 3 cm (3-5 cm)</p>
<u>COMMISSION INTERPRETATION</u>
<p>At 3 hours 11 minutes after the end of the Bout, Jones' body is beginning to lose its battle with the electrolyte imbalance resulting from severe Exertional Rhabdomyolysis, renal dysfunction with likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration.</p> <p><b>EVIDENCED BY:</b> Jones' blood test results at 22:45; 00:49 and 01:00 hours coupled with low systolic blood pressure and critically low diastolic blood pressure coupled with a heart rate still elevated above 100 bpm and respiratory rate above 18, but both now slowing over their previous readings.</p>

**01:00 hours –ECG Results:** Sinus Tachycardia w/ST change appearing junctional/peaked T's/suspect Hyperkalemia

<u>Vent. Rate</u>	<u>PR interval</u>	<u>QRS duration</u>	<u>QT/QTc</u>	<u>P-R-T axes</u>
104 BPM	138 ms	88 ms	322/423 ms	68 - 85 - 25

**01:00 hours –**

<u>BLOOD TEST RESULTS</u>	<u>COMMISSION INTERPRETATION/INDICATION</u>
<p> <b>Na<sup>+</sup></b> – 138 (135-145 mmol/L)  <b>K<sup>+</sup></b> – 5.5 H (3.5-5.0 mmol/L)  <b>Cl<sup>-</sup></b> – 112 H (100-109 mmol/L)  <b>CO<sub>2</sub></b> – 11 L (21-32 mmol/L)  <b>BUN</b> – 22 H (6-20 mg/dl)  <b>CREATININE</b> – 2.7 H (0.6-1.3 mg/dL)  <b>Glucose</b> – 180 H (70-105 mg/dL)  <b>pH</b> – 7.129 L (7.35-7.45)  <b>Hct/HEMATOCRIT</b> – 47 (40-52%)  <b>Hgb/Hb/HEMOGLOBIN</b> – 16.0 (14-18 g/dL)  <b>pCO<sub>2</sub></b> – 29.9 L (35-45 mmHg)  <b>HCO<sub>3</sub><sup>-</sup></b> – 9.9 L (22-26 mmol/L)  <b>BEecf</b> – -19 L (+/- 4 mmol/L)  <b>ANION/GAP</b> – 22 (5-25 mmol/L)                 </p>	<p>3 hr. 11 min. after end of the Bout, Jones body is trying to correct and compensate for severe renal dysfunction, most likely due to effects of exertional rhabdo including necrosis of nephrons' renal tubules due to myoglobin toxicity and hyperkalemia. Likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration. Cascading failure of organ systems clearly in process and escalating. Cardiac dysrhythmias beginning with sinus tachycardia resulting from hyperkalemia. Electrolyte &amp; other levels wildly fluctuating from previous results.</p> <p><b>EVIDENCED BY:</b>                      Na<sup>+</sup> and K<sup>+</sup> now both almost within normal – Both are wildly fluctuating, Na<sup>+</sup> and K<sup>+</sup> were both high (149) (5.4) 2 hr. 10 min. earlier, then Na<sup>+</sup> low (131) and K<sup>+</sup> critically high (&gt;8.9) just 10 min. earlier without any medical intervention/drug treatment (indicates body is trying to correct conditions, but damaged nephrons' &amp; renal tubules' failing to normally regulate Na<sup>+</sup> &amp; K<sup>+</sup>);                      (indicates continued muscle cell damage w/out adequate compensation/filtering by kidneys; levels associated with critical imbalance)                      Cl<sup>-</sup> increasingly high – Was (110) just 10 min. earlier (kidneys possibly overcorrecting via excessive reabsorption, will lead to cellular dysfunction/damage);                      BUN lower but still high – Wildly fluctuating was high (19) 2 hr. 10 min. earlier, higher (31) just 10 min. earlier, now lower but still high (consistent with severe kidney damage to more than 60% of kidneys' nephron/cell structure and body's counteracting corrective measures);                      Creatinine down but still high – Was high (2.7) 2 hr. 10 min. earlier &amp; (3.4) just 10 min. earlier (indicates continued nephronic damage);                      Glucose still high but lowering – Was higher (224) 2 hr. 10 min. earlier and (184) just 10 min. earlier (indicates body coming out of energy production phase but possible kidney/liver dysfunction);                      pH decreasing – Was 7.2 just 10 min. earlier (confirms acidosis);                      PCO<sub>2</sub> &amp; HCO<sub>3</sub><sup>-</sup> &amp; BEecf levels confirm extreme metabolic acidosis with the respiratory system attempting to compensate.                 </p>

**01:25 hours –** Taken to CT scan – tolerated well

**01:30 hours** – Glasgow Coma Scale score of 15 – Mildest Concussion/MTBI – Eyes = spontaneous (4); Best Verbal Response = oriented (5); Best Motor Response = obeys command (6)

**VITAL SIGNS**

**Blood Pressure** – 75/31 A (110/65 – 140/90)  
**Heart Rate** – 118 R (60-80)  
**Respiratory Rate** – 28 (16-18)  
**SpO<sub>2</sub>** – 100% (94-100)  
**Pupillary Dilation (L&R)** – 3 cm (3-5 cm)

**COMMISSION INTERPRETATION**

At 3 hours 41 minutes after the end of the Bout, Jones' body is continuing to lose its battle with the electrolyte imbalance resulting from severe Exertional Rhabdomyolysis, renal dysfunction with likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration.

**EVIDENCED BY:** Jones' blood test results at 22:45; 00:49 and 01:00 hours coupled with now lower/falling systolic blood pressure and continued critically low diastolic blood pressure. Above coupled with a heart rate still elevated above 100 bpm and respiratory rate above 18.

**01:35 hours** – CT Scan Thoracic Spine & Lumbar Spine Routine w/out IV Contrast – No evidence of fracture or subluxation. Vertebral heights and disc spaces are well preserved. Spinal alignment is maintained without evidence of spondylolisthesis. Natural kyphotic and lordotic curvatures are preserved. There is bibasilar atelectasis (collapsing of some of the alveoli in both lungs, usually caused by obstruction of the airways and bronchioles, pressure on the lung from fluid or air in the pleural space or other mechanism outside the lungs, here possibly from swelling of the chest muscles subsequent to the onset of rhabdomyolysis, this leads to further hypoxia, lack of oxygen to the body's various systems. Chest X-ray at 00:57 hours revealed neither an obstructed airway nor pleural fluid or air, thus hypothesize cause is one or more of the following: i) surface active proteins or acids causing monomolecular layer over pulmonary alveolar surfaces, lipoproteins, lecithins, and sphingomyelins that stabilize alveolar volume by reducing surface tension and changing ratio of surface tension to surface area; ii) Lack of pulmonary/alveoli cell structure due to rhabdomyolysis/hypoxia/anoxia; or iii) Lack of surfactant/moisture/alveolar cell volume due to dehydration) within the lungs. SI joints, lungs and abdomen are unremarkable.

**01:35 hours** – Normal Saline IV – 18 gauge – Flow Rate 100 mL/hr.

**01:35 hours** – Metoprolol 5 mg via IV push (selective beta blocker for tachycardia & to slow heart rate)

**01:35 hours** – Ca<sup>2+</sup> Gluconate 9.3 mEq (1 gram) via IV push (for heart muscle protection against hyperkalemia by reducing cardiomyocyte excitability but does not lower K<sup>+</sup>)

**01:38 hours** – Insulin 10 units via IV (to reduce glucose and lower extracellular K<sup>+</sup>)

**01:38 hours** – NaHCO<sub>3</sub> 50 mEq via IV push (to reduce K<sup>+</sup> and reduce metabolic acidosis)

**01:38 hours** – Dextrose (50%) (D50) 50 ml via IV push (to avoid anaerobic cellular respiration and reduce acidosis)

**01:38 hours** – Morphine 2-5 mg/4 hours via IV push (for generalized pain reduction)

**01:38 hours** – Famotidine 20 mg/4 hours via IV push (to reduce acidosis)

**01:40 hours** – Returns to room from CT scans and tells nurse he is still unable to move legs & experiencing generalized pain all over. Glasgow Coma Scale score of 15 – Mildest Concussion/MTBI – Eyes = spontaneous (4); Best Verbal Response = oriented (5); Best Motor Response = obeys command (6)

<b><u>VITAL SIGNS</u></b>
<b>Blood Pressure</b> – 112/33 A (110/65 – 140/90) <b>Heart Rate</b> – 107 R (60-80) <b>Respiratory Rate</b> – 20 (16-18) <b>SpO2</b> – 100% (94-100) <b>Pupillary Dilation (L&amp;R)</b> – 2 cm (3-5 cm)
<b><u>COMMISSION INTERPRETATION</u></b>
At 3 hours 51 minutes after the end of the Bout, Jones’ body is continuing to lose its battle with the electrolyte imbalance resulting from severe Exertional Rhabdomyolysis, renal dysfunction with likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration.  <b>EVIDENCED BY:</b> Jones’ blood test results at 22:45; 00:49 and 01:00 hours coupled with now rebounding systolic blood pressure and continued critically low diastolic blood pressure. Above coupled with a heart rate still elevated above 100 bpm and respiratory rate above 18.

**01:50 hours** – Glasgow Coma Scale score of 15 – Mildest Concussion/MTBI – Eyes = spontaneous (4); Best Verbal Response = oriented (5); Best Motor Response = obeys command (6)

<b><u>VITAL SIGNS</u></b>
<b>Blood Pressure</b> – 87/38 A (110/65 – 140/90) <b>Heart Rate</b> – 95 R (60-80) <b>Respiratory Rate</b> – 20 (16-18) <b>SpO2</b> – 94% (94-100) <b>Pupillary Dilation (L&amp;R)</b> – 2 cm (3-5 cm)
<b><u>COMMISSION INTERPRETATION</u></b>
At 4 hours 1 minutes after the end of the Bout, Jones’ body is critically losing its battle with the electrolyte imbalance resulting from severe Exertional Rhabdomyolysis, renal dysfunction with likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration.  <b>EVIDENCED BY:</b> Jones’ blood test results at 22:45; 00:49 and 01:00 hours coupled with once again low systolic blood pressure and continued critically low diastolic blood pressure. Above coupled with a heart rate still elevated above 100 bpm and respiratory rate above 18. Now experiencing contracted pupillary dilation and decreased SpO2 (blood oxygen) levels nearing the low range, as a result of electrolyte imbalance induced decrease in blood flow and inefficient cardio/pulmonary function.

**02:00 hours** – Glasgow Coma Scale score of 15 – Mildest Concussion/MTBI – Eyes = spontaneous (4); Best Verbal Response = oriented (5); Best Motor Response = obeys command (6)

<b><u>VITAL SIGNS</u></b>
<b>Blood Pressure</b> – 106/86 A (110/65 – 140/90) <b>Heart Rate</b> – 94 R (60-80) <b>Respiratory Rate</b> – 26 (16-18) <b>SpO2</b> – 100% <b>Pupillary Dilation (L&amp;R)</b> – 3 cm (3-5 cm)
<b><u>COMMISSION INTERPRETATION</u></b>
At 4 hours 11 minutes after the end of the Bout, Jones’ vital signs appear to improve in response to the medical intervention and medication given 15-20 minutes earlier; however, Jones’ body is still suffering from the electrolyte imbalance resulting from Exertional Rhabdomyolysis.  <b>EVIDENCED BY:</b> Jones’ blood test results at 22:45; 00:49 and 01:00 hours coupled with a heart rate still elevated above normal and respiratory rate above 18.

02:26 hours –

<u>URINE TEST RESULTS:</u>	<u>COMMISSION INTERPRETATION/INDICATION</u>
<p><b>Appearance</b> – HAZY/Light Brown Abnormal (Clear) <b>Glucose</b> – 300 H (0-15 mg/dL) <b>Bilirubin</b> – NEG (Neg.) <b>Ketones</b> – NEG (Neg. mg/dL) <b>SG</b> – 1.010 (1.003-1.030) <b>Blood</b> – LARGE Abnormal (Neg.) <b>pH</b> – 6.5 (5.0-8.0) <b>Protein</b> – 300 Abnormal (Neg. mg/dL) <b>Urobilinogen</b> – NORMAL (0.2-1.0 mg/dL) <b>Nitrites</b> – NEG (Neg.) <b>Leuk. Esterase</b> – NEG (Neg.) <b>RBC</b> – 6-10 H (0-2 #/HPF) <b>WBC</b> – 3-5 H (0-2 #/HPF) <b>Bacteria</b> – PRESENT (Absent) <b>Mucous</b> – PRESENT (0 #/HPF)</p>	<p>4 hr. 36 min. after the end of the Bout, Jones' renal function is severely impaired with damage appearing to be most likely associated with acute renal tubular necrosis from extremely high levels of myoglobin and K<sup>+</sup> from exertional Rhabdomyolysis. Likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration</p> <p><b>EVIDENCED BY:</b> Haziness (probably caused by renal tubular epithelial cells damaged from the myoglobin released by muscle cell destruction (rhabdomyolysis)); Glucose extremely high (indicates severe kidney dysfunction namely to glomerulus); Bilirubin Neg. (indicates proper liver function); Blood &amp; RBC &amp; WBC levels (presence of blood LARGE indicates, kidney damage (most likely acute tubular necrosis because of large shape evidencing no damage from passing through irregular glomerulus)); pH normal (indicates glomerular function may be normal more specifically identifying acute tubular necrosis); Protein extremely high (indicates either impaired glomerulus or tubular necrosis); Bacteria &amp; Mucous present (indicates response to infection/severe cell destruction)</p>

02:30 hours –

<u>VITAL SIGNS</u>
<p><b>Blood Pressure</b> – 87/29 A (110/65 – 140/90) <b>Heart Rate</b> – 91 R (60-80) <b>Respiratory Rate</b> – 22 (16-18) <b>SpO2</b> – 95% (94-100)</p>
<u>COMMISSION INTERPRETATION</u>
<p>At 4 hours 41 minutes after the end of the Bout, despite the temporary improvement resulting from the medical intervention, Jones' body is still unable to recover from the electrolyte imbalance resulting from severe Exertional Rhabdomyolysis, renal dysfunction with likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration.</p> <p><b>EVIDENCED BY:</b> Jones' blood test results at 22:45; 00:49 and 01:00 hours and urine test results from 02:26 hours coupled with once again low systolic blood pressure and now critically low diastolic blood pressure. Above coupled with a heart rate still elevated above normal and respiratory rate above 18. Now experiencing decreased SpO2 (blood oxygen) levels nearing the low range, as a result of electrolyte imbalance induced decrease in blood flow and inefficient cardio/pulmonary function.</p>

**02:38 hours** – CT Scan Brain Routine w/out IV Contrast – Gray-white matter differentiation is well maintained. Ventricles and sulci are w/in normal limits. Basal cisterns are well maintained. No intracranial hemorrhage or extraaxial fluid collection is identified. No evidence of acute territorial infarct, focal mass lesions, or midline shift. Visualized soft tissues and osseous structures are normal. No orbital abnormalities. Paranasal sinuses are well-aerated. Mastoid air cells are well aerated.

**02:38 hours** – CT Scan Pelvis & Abdomen Routine w/out IV Contrast – Dependent atelectatic changes are seen bilaterally. No pleural fluid identified. Visualized heart is normal in size w/out evidence of pericardial effusion. Distal esophagus is unremarkable. Liver, gallbladder, pancreas, spleen and adrenal glands are grossly unremarkable. No focal contour abnormality identified. Kidneys are grossly unremarkable and demonstrate normal contour. Abdominal aorta is normal in caliber w/out evidence of atherosclerotic changes. Stomach is

moderately distended w/out focal mass lesion. Large and small bowel demonstrate no evidence of obstruction. Appendix is well visualized and normal. No significant lymphadenopathy is seen w/in abdomen. Small umbilical hernia is present. W/in pelvis, bladder is collapsed and demonstrates a Foley catheter tip w/air present consistent w/catheterization. Prostate and seminal vesicles are unremarkable. No free fluid or significant lymphadenopathy is seen w/in the pelvis. Bone windows demonstrate no suspicious lytic or blastic lesions.

**02:58 hours – Ventricular Tachycardia.** Moved to ICU. Appeared sweaty; Verbally responsive w/appropriate responses to questions; Alert & Oriented Times 3; Unable to move R or L legs; Pupils – (L) & (R) reactive to light; Appears to go to sleep while talking to nurse

<b><u>VITAL SIGNS</u></b>
<b>Blood Pressure</b> – 76/52 A (110/65 – 140/90) <b>Heart Rate</b> – 175 R (60-80) <b>Respiratory Rate</b> – 35 (16-18)
<b><u>COMMISSION INTERPRETATION</u></b>
At 5 hours 19 minutes after the end of the Bout, Jones’ body was continuing to evidence signs of severe Exertional Rhabdomyolysis, renal dysfunction with likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration. Both systolic and diastolic blood pressures are continuing to fall in response to the electrolyte imbalance resulting from the Exertional Rhabdomyolysis.
<b>EVIDENCED BY:</b> Jones’ blood test results at 22:45 hours coupled with slightly low blood pressure just after the end of the Bout, which then returned to normal and is now remarkably lower than reading 5 minutes earlier. Elevated heart rate over 100 bpm and continued respiratory rate elevation. Jones’ vitals are consistent with compensating response for low cardiac output/early stage cardiac dysrhythmia, suspected tachycardia based on heart rate over 100 bpm.

**03:00 hours – CODE BLUE** called. CODE BLUE team immediately responded & hooked to crash cart. Transferred to SICU.

03:04 hours –

<b>BLOOD TEST RESULTS:</b>	<b>COMMISSION INTERPRETATION/INDICATION</b>
<p>Na<sup>+</sup> – 133 L (135-145 mmol/L) K<sup>+</sup> – 9.7 CH* (3.5-5.0 mmol/L) Cl<sup>-</sup> – 99 L (100-109 mmol/L) CO<sub>2</sub> – 15 L (21-32 mmol/L) BUN – 22 H (6-20 mg/dl) CREATININE – 3.8 H (0.6-1.3 mg/dL) Glucose – 238 H (70-105 mg/dL) Hct/HEMATOCRIT – 48.3 (40-52%) Hgb/Hb/HEMOGLOBIN – 17.1 (14-18 g/dL) GFR – 23 L (&gt;90 mL/min/1.73m<sup>2</sup>) WBC – 18.6 H (3.0-12.0 K/uL) RBC – 5.5 (4.5-6.0 M/uL) MCV – 88 (79-96 FL) MCH – 31.1 (27-36 pg) MCHC – 35.4 (30-36 g/dL) RDW – 13.0 (11.0-14.0%) Platelet Count – 246 (150-500 K/uL) MPV – 10.3 (8.1-12.8 fL) Neutrophils Absolute – 15 H (2.5-8.2 K/uL) Lymphocytes, Absolute – 2.3 (1.0-4.8 K/uL) Monocytes, Absolute – 0.9 (0.1-1.0 K/uL) Bilirubin – 0.6 (0.20-1.2 mg/dl) SGOT(AST) – 1330 H (15-41 U/L) SGPT (ALT) – 317 H (5-45 U/L) GGT – 51 H (7-50 IU/L) ALK Phosphatase – 131 H (32-91 U/L) Ca<sup>2+</sup> – 6.9 CL (8.6-10.2 mg/dL) PO<sub>4</sub><sup>3-</sup> – &gt;24.0 CH (2.5-4.5 mg/dL) Mg<sup>2+</sup> – 5.3 H (1.6-2.6 mg/dL) Amylase – 399 H (36-128 U/L) Lipase – 69 H (13-60 U/L) CK-MB – &gt;300.0 H (0.2-5.0 ng/mL) Myoglobin – 1143.7 H (3.0-70.0 ng/mL) Troponin I – 2.32 CH (0.04 ng/mL – Neg) (0.05-0.49 ng/mL – Suspect Myocardial Dmg.) (=&gt;0.5 ng/mL – Consistent w/Myocardial Injury)</p>	<p>5 hr. 14 min. after end of the Bout, Jones body is failing to keep up with the cascading systems failures caused by the severe renal dysfunction resulting from effects of exertional rhabdo including necrosis of nephrons and renal tubules due to myoglobin toxicity and hyperkalemia. Likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration. Cascading failure of organ systems clearly escalating from renal failure into heart failure. Cardiac dysrhythmias, rhabdomyolysis, lactic acidosis likely worsening from hyperkalemia, critically high PO<sub>4</sub><sup>3-</sup>, very low Ca<sup>2+</sup>. and CaPO<sub>4</sub> precipitation in tissues creating a potentially fatal feedback loop.</p> <p><b>EVIDENCED BY:</b> Na<sup>+</sup> still fluctuating with previous levels high (149) 4 hr. 14 min. earlier, low (131) 2 hr. 45 min. earlier, higher but normal (138) 2 hr. 4 min. earlier, now low (133); K<sup>+</sup> now critically high with extreme fluctuations always high (5.4) 4 hr. 14 min. earlier, critically high (&gt;8.9) 2 hr. 45 min. earlier, high (5.5) 2 hr. 4 min. earlier, now critically high (9.7) medical intervention/drug treatment ineffective (indicates body is trying to correct conditions, but damaged nephrons' &amp; renal tubules' failure to normally regulate Na<sup>+</sup> &amp; K<sup>+</sup> due to continued rhabdo and cellular destruction now resulting from multiple causes); Cl<sup>-</sup> low and fluctuating with normal (101) 4 hr. 14 min. earlier, high (110) 2 hr. 45 min. earlier, higher (112) 2 hr. 4 min. earlier, now low (99) (indicates renal failure &amp; dehydration); BUN lower but still high – Wildly fluctuating with previous levels high (19) 4 hr. 14 min. earlier, high (31) 2 hr. 45 min. earlier, lower but high (22) 2 hr. 4 min. earlier, now still high (22) (consistent with severe kidney damage to more than 60% of kidneys' nephron/cell structure and body's counteracting corrective measures); Creatinine high &amp; rising again was (2.7) 4 hr. 14 min. earlier, higher (3.4) 2 hr. 45 min. earlier, lower still high (2.7) 2 hr. 4 min. earlier, now very high (3.8) (indicates continued nephronic damage); Glucose rising; GFR extremely low &amp; decreased from 26 2 hr. 45 min. earlier (indicates worsening kidney damage and lack of blood flow/filtration); AST, ALT, GGT &amp; ALP levels indicate renal failure; Ca<sup>2+</sup> critically low and markedly decreased from its high level (12.4) 4 hr. 14 min. earlier (drastically lowered level probably caused by CaPO<sub>4</sub> precipitation in the tissues due to kidneys' failure to filter &amp; excrete the PO<sub>4</sub><sup>3-</sup>) (will cause severe heart dysrhythmias when coupled with channel blocking effects of critically high Mg<sup>2+</sup>); PO<sub>4</sub><sup>3-</sup> critically high (indicates release via extreme tissue destruction via rhabdomyolysis, lactic acidosis &amp; nearly complete renal failure); Mg<sup>2+</sup> very high (likely resulting from cellular destruction release of intracellular contents) (will cause heart dysrhythmias due to channel blocking of Ca<sup>2+</sup> and extracellular toxicity); Amylase still extraordinarily high (indicates kidney failure &amp; dehydration w/high glucose also indicating possible pancreatic dysfunction); Lipase high (indicates renal failure) CK-MB extremely high (confirms extensive cardiac/myocardial damage); Troponin I critically high (confirms severe cardiac muscle cell destruction); Myoglobin very high (confirmation of muscle cell destruction and toxic levels causing renal failure)</p>
<p>*Specimen slightly hemolyzed, so K may be falsely elevated</p>	

03:15 hours – **CODE GREEN** reported to ICU from SICU. Alert & Oriented times 3; No sensation from nipple line down; No external sign of injury, except bruise high on forehead; Difficult to palpate pulse in lower extremities; minimal dark reddish brown urine from catheter; Normal Saline IV @ 100%; **SINUS TACHYCARDIA w/Wide Complex** reported by MD.

<u>VITAL SIGNS</u>
<b>Blood Pressure</b> – 114/76 A (110/65 – 140/90) <b>Heart Rate</b> – 175 R (60-80) <b>Respiratory Rate</b> – 35 (16-18) <b>SpO2</b> – 92%
<u>COMMISSION INTERPRETATION</u>
At 5 hours 26 minutes after the end of the Bout, Jones’ vital signs appear to improve in response to the medical intervention minutes earlier; however, Jones’ body is still suffering from the electrolyte imbalance resulting from Exertional Rhabdomyolysis.
<b>EVIDENCED BY:</b> Jones’ blood test results at 22:45, 00:49, 01:00; 03:04 hours and urine test results from 02:26 hours coupled with now hyper-elevated heart rate and respiratory rate and decreased SpO2 (blood oxygen) levels.

**03:29 hours –**

<u>BLOOD TEST RESULTS:</u>	<u>COMMISSION INTERPRETATION/INDICATION</u>
<b>Prothrombin</b> – 16.5 H (12.0-14.0 sec) <b>INR</b> – 1.3 H (0.9-1.3) <b>PTT</b> – 33.9 (23-36.9 sec)	PT & INR high w/ normal PTT (indicates possible liver dysfunction – no contraindication for disseminated intravascular coagulation – possible compensation)

**03:30 hours – ECG Results: Diagnosis – Cardiac Dysrhythmia – Very wide QRS most compatible w/Hyperkalemia**

<u>Vent. Rate</u>	<u>PR interval</u>	<u>QRS duration</u>	<u>QT/QTc</u>	<u>P-R-T axes</u>
131 BPM	* ms	156 ms	* ms	*

**03:30 hours –** Given one amp  $\text{Ca}^{2+}\text{Cl}^-$  and one amp  $\text{HCO}_3^-$  (to treat critically low  $\text{Ca}^{2+}$  and control acidosis along with providing protection for cardiac muscle cells against elevated  $\text{K}^+$  and  $\text{Mg}^{2+}$ )

**03:37 hours –** D5W Solution IV – 18 gauge – Flow Rate 150 mL/hr.

**03:37 hours –**  $\text{NaHCO}_3$  100 mEq via D5W IV drip @ 150 mL/hr (to reduce  $\text{K}^+$  and reduce metabolic acidosis)

**03:41 hours – ECG Results: Diagnosis – Cardiac Dysrhythmia – Very wide QRS most compatible w/Hyperkalemia**

<u>Vent. Rate</u>	<u>PR interval</u>	<u>QRS duration</u>	<u>QT/QTc</u>	<u>P-R-T axes</u>
220 BPM	* ms	162 ms	202/306 ms	*--56--45

<u>BLOOD TEST RESULTS:</u>	<u>COMMISSION INTERPRETATION/INDICATION</u>
<p> <b>Na<sup>+</sup></b> – 132 L (135-145 mmol/L)  <b>K<sup>+</sup></b> – 6.6 CH (3.5-5.0 mmol/L)  <b>Cl<sup>-</sup></b> – 102 (100-109 mmol/L)  <b>CO<sub>2</sub></b> – 13 L (21-32 mmol/L)  <b>BUN</b> – 24 H (6-20 mg/dl)  <b>CREATININE</b> – 4.0 H (0.6-1.3 mg/dL)  <b>Glucose</b> – 207 H (70-105 mg/dL)  <b>Hct/HEMATOCRIT</b> – 46.7 (40-52%)  <b>Hgb/Hb/HEMOGLOBIN</b> – 16.4 (14-18 g/dL)  <b>GFR</b> – 22 L (&gt;90 mL/min/1.73m<sup>2</sup>)  <b>WBC</b> – 19.35 H (3.0-12.0 K/uL)  <b>RBC</b> – 5.33 (4.5-6.0 M/uL)  <b>MCV</b> – 88 (79-96 FL)  <b>MCH</b> – 30.8 (27-36 pg)  <b>MCHC</b> – 35.1 (30-36 g/dL)  <b>RDW</b> – 13.2 (11.0-14.0%)  <b>Platelet Count</b> – 231 (150-500 K/uL)  <b>MPV</b> – 9.9 (8.1-12.8 fl)  <b>Neutrophils Absolute</b> – 15.7 H (2.5-8.2 K/uL)  <b>Lymphocytes, Absolute</b> – 2.2 (1.0-4.8 K/uL)  <b>Monocytes, Absolute</b> – 1.2 H (0.1-1.0 K/uL)  <b>Bilirubin</b> – 0.6 (0.20-1.2 mg/dl)  <b>SGOT(AST)</b> – 2256 H (15-41 U/L)  <b>SGPT (ALT)</b> – 531 H (5-45 U/L)  <b>GGT</b> – 50 H (7-50 IU/L)  <b>ALK Phosphatase</b> – 104 H (32-91 U/L)  <b>Ca<sup>2+</sup></b> – 9.0 (8.6-10.2 mg/dL)  <b>PO<sub>4</sub><sup>3-</sup></b> – &gt;20.4 CH (2.5-4.5 mg/dL)  <b>Lactate</b> – 6.2 H (0.5-2.2 mmol/L)  <b>CK-MB</b> – &gt;300.0 H (0.2-5.0 ng/mL)  <b>Myoglobin</b> – 1079.0 H (3.0-70.0 ng/mL)  <b>Prothrombin</b> – 18.4 H (12.0-14.0 sec)  <b>INR</b> – 1.5 H (0.9-1.3)  <b>PTT</b> – 35.8 (23-36.9 sec)  <b>Troponin I</b> – 2.26 CH  (0.04 ng/mL – Neg)  (0.05-0.49 ng/mL – Suspect Myocardial Dmg.)  (= / &gt; 0.5 ng/mL – Consistent w/Myocardial Injury) </p>	<p>6 hr. 11 min. after end of the Bout, Jones' body is in the middle of its cascading systems failure. Jones is experiencing severe renal dysfunction and heart damage resulting from effects of exertional rhabdo including necrosis of nephrons and renal tubules due to myoglobin toxicity and hyperkalemia. Likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration. Cascading failure of organ systems clearly escalating from renal failure into heart failure. Cardiac dysrhythmias, rhabdomyolysis, lactic acidosis likely worsening from hyperkalemia, critically high PO<sub>4</sub><sup>3-</sup>.</p> <p><b>EVIDENCED BY:</b>  Na<sup>+</sup> stabilized fluctuation with previous levels high (149) 5 hr. 10 min. earlier, low (131) 3 hr. 41 min. earlier, higher but normal (138) 3 hr. earlier, low (133) 56 min. earlier, now low (132);  K<sup>+</sup> still critically high with extreme fluctuations always high (5.4) 5 hr. 10 min. earlier, critically high (&gt;8.9) 3 hr. 41 min. earlier, high (5.5) 3 hr. earlier, critically high (9.7) 56 min. earlier, medical intervention/drug treatment ineffective (indicates body is trying to correct conditions, but damaged nephrons' &amp; renal tubules' failure to normally regulate Na<sup>+</sup> &amp; K<sup>+</sup> due to continued rhabdo and cellular destruction now resulting from multiple causes now including significant heart damage);  Cl<sup>-</sup> fluctuating with normal (101) 5 hr. 10 min. earlier, high (110) 3 hr. 41 min. earlier, higher (112) 3 hr. earlier, low (99) 56 min. earlier, normal (102) (indicates renal failure &amp; dehydration);  BUN higher – Wildly fluctuating with previous levels high (19) 5 hr. 10 min. earlier, high (31) 3 hr. 41 min. earlier, lower but high (22) 3 hr. earlier, high (22) 56 min. earlier, now higher (24) (consistent with severe kidney damage to more than 60% of kidneys' nephron/cell structure and body's counteracting corrective measures);  Creatinine still rising was (2.7) 5 hr. 10 min. earlier, higher (3.4) 3 hr. 41 min. earlier, lower still high (2.7) 3 hr. earlier, very high (3.8) 56 min. earlier, now higher (4.0) (indicates continued nephronic damage);  Glucose lower but still high;  GFR extremely low &amp; decreased from 26 3 hr. 41 min. earlier and 23 56 min. earlier (indicates worsening kidney damage and lack of blood flow/filtration);  AST, ALT, GGT &amp; ALP levels indicate renal failure;  Ca<sup>2+</sup> back to normal from critically low and markedly decreased from its high level (12.4) 5 hr. 10 min. earlier drastically lowered level (6.9) 56 min. earlier (probably caused by CaPO<sub>4</sub> precipitation in the tissues due to kidneys' failure to filter &amp; excrete the PO<sub>4</sub><sup>3-</sup>) and now normal (9.0) due to the Ca<sup>2+</sup>Cl<sup>-</sup> treatment (fluctuations have likely already caused severe heart dysrhythmias when coupled with channel blocking effects of critically high Mg<sup>2+</sup>);  PO<sub>4</sub><sup>3-</sup> down but still critically high was &gt;24.0 56 min. earlier, now &gt;20.4 (indicates release via extreme tissue destruction via rhabdomyolysis, cardiac distress, lactic acidosis &amp; nearly complete renal failure);  Lactate high (6.2) (confirmation of metabolic acidosis);  CK-MB extremely high still &gt;300 as it was 56 min. earlier (confirms extensive cardiac/myocardial damage);  Troponin I still critically high down only slightly from 2.32 56 min. earlier (confirms severe cardiac muscle cell destruction);  Myoglobin still very high but down slightly from 1143.7 56 min. earlier (confirmation of muscle cell destruction and toxic levels causing renal failure);  Prothrombin high &amp; rising (16.5 3 hr. 10 min. earlier &amp; 31 min. earlier, now 18.4);  INR rising (1.3 3 hr. 10 min. earlier &amp; 31 min. earlier, now 1.5) ) with PTT high end of range (indicates probable disseminated intravascular coagulation) </p>

**04:05 hours** – Had received a total of six (6) Liters of Normal Saline IV fluids (1 L in ambulance en route to Saline Memorial; 1 L at Saline Memorial; 4 L in the UAMS Emergency Dept.). Scant urine production, what little was produced was dark brown/reddish. Additional two (2) Liters of fluids being administered (1 L normal saline @ 100 ml/hr. started at 01:35 hours; 1 L D5W @ 150 ml/hr. started at 03:37 hours). Total IV fluids administered since end of Bout is approximately 6.5 Liters.

**04:20 hours to 04:30 hours** – MD arrives in SICU to place right femoral artery line. Decision made to switch to Quinton catheter for emergency hemodialysis due to critically high potassium level.

**04:22 hours** – Insulin 10 units via IV push (to reduce glucose)

**04:22 hours** – Bumetanide (Bumex IV Drip), 24 mg/96 mL, 0.5 mg/hr titrate to effect, on demand, Max. drip rate 3 mg/hr (Loop diuretic used due to cardiac and renal dysfunction to increase renal blood flow and reduce edema/increase in interstitial fluid resulting from cardiac dysrhythmia and rhabdomyolysis. Operates via inhibition of Na<sup>+</sup>, Cl<sup>-</sup>, Mg<sup>2+</sup> and Ca<sup>2+</sup> reabsorption, which in turn inhibits water reabsorption and increases urine production, and should decrease Na<sup>+</sup>, Cl<sup>-</sup>, Mg<sup>2+</sup> and Ca<sup>2+</sup> through urine production without reabsorption.)

**05:10 hours** – **CODE BLUE** called. No pulse or respiration. Jones became unresponsive to questions while awaiting arrival of Quinton catheter, no hemodialysis started. **CODE BLUE TEAM responded immediately.**

**CODE BLUE CRASH SHEET LOG & INTERIM BLOOD TEST RESULTS**

<u>Time</u>	<u>Respiration</u>	<u>Pulse</u>	<u>Heart Rhythm</u>	<u>Medication</u>	<u>Notes</u>
<b>05:10 hours Until 05:15 hours</b>	Bagged, then intubated	Compressions	Bradycardic then Asystolic		
<b>05:15 hours</b>	Assisted	Compressions	Asystole	Epinephrine 1 mg, NaHCO <sub>3</sub> 1 mEq/kg and ½ dose after	
<b>05:17 hours</b>	Assisted	Compressions	Asystole	Vasopressin 40 units IV push, CaCl 1 gram, D50, Insulin 10 units	
<b>05:19 hours</b>	Assisted	Compressions	Asystole	Atropine 1 mg	
<b>05:22 hours</b>	Assisted	Compressions	Asystole	NaHCO <sub>3</sub>	
<b>05:24 hours</b>	Assisted	Compressions	Asystole	Epinephrine 1 mg; CaCl 1 gram	
<b>05:25 hours</b>	Assisted	SPONTANEOUS	Faint Pulse HR 150 Wide QRS		
<b>05:26 hours</b>	Assisted	Compressions/CPR Resumed	V-Fib		Defibrillator Shock 200 Joules
<b>05:27 hours</b>	Assisted	Compressions	Wide QRS	Epinephrine 1 mg	
<b>05:28 hours</b>	Assisted	SPONTANEOUS	Wide QRS		

<u>BLOOD TEST RESULTS</u>	<u>COMMISSION INTERPRETATION/INDICATION</u>
<p> <b>Na<sup>+</sup></b> – 135.2 L (135-145 mmol/L)  <b>K<sup>+</sup></b> – 8.46 H (3.5-5.0 mmol/L)  <b>Cl<sup>-</sup></b> – 101 (100-109 mmol/L)  <b>Glucose</b> – 136 H (70-105 mg/dL)  <b>pH</b> – 7.158 L (7.35-7.45)  <b>pCO<sub>2</sub></b> – 49.7 H (35-45 mmHg)  <b>HCO<sub>3</sub><sup>-</sup></b> – 17.2 L (22-26 mmol/L)  <b>BEecf</b> – -11.5 L (+/- 4 mmol/L)  <b>pO<sub>2</sub></b> – 32.2 L (75.0-100.0 mmHg)  <b>Ca, Ionized</b> – 1.11 L (1.15-1.33 mmol/L)  <b>Lactate</b> – 5.06 H (0.5-2.2 mmol/L) </p>	<p>7 hr. 39 min. after the end of the Bout, Jones has been in cardiac arrest for 18 minutes as the end stage result of severe Exertional Rhabdomyolysis, renal dysfunction with likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration</p> <p><b>EVIDENCED BY:</b>  Na<sup>+</sup> low w/fluctuation previous levels high (149) 6 hr. 38 min. earlier, low (131) 5 hr. 9 min. earlier, higher but normal (138) 4 hr. 28 min. earlier, low (133) 2 hr. 26 min. earlier, low (132) 1 hr. 28 min. earlier;  K<sup>+</sup> still critically high with extreme fluctuations always high (5.4) 6 hr. 38 min. earlier, critically high (&gt;8.9) 5 hr. 9 min. earlier, high (5.5) 4 hr. 28 min. earlier, critically high (9.7) 2 hr. 26 min. earlier, critically high (6.6) 1 hr. 28 min. earlier  medical intervention/drug treatment ineffective (indicates body is trying to correct conditions, but damaged nephrons' &amp; renal tubules' failure to normally regulate Na<sup>+</sup> &amp; K<sup>+</sup> due to continued rhabdo and cellular destruction now resulting from multiple causes now including significant heart damage/failure;  PCO<sub>2</sub> &amp; HCO<sub>3</sub> &amp; BEecf levels confirm extreme metabolic acidosis with the respiratory system now failing to compensate and in fact adding to acidosis through respiratory acidosis.</p>

<u>Time</u>	<u>Respiration</u>	<u>Pulse</u>	<u>Heart Rhythm</u>	<u>Medication</u>	<u>Notes</u>
<b>05:35 hours</b>	Assisted	SPONTANEOUS	Wide QRS	Atropine 1 mg, Epinephrine 1 mg.	Quinton catheter placed
<b>05:38 hours</b>	Assisted	SPONTANEOUS	Not Noted	D50, Insulin	
<b>05:40 hours</b>	Assisted	SPONTANEOUS	Not Noted	NaHCO <sub>3</sub> ; Arterial line started w/NaHCO <sub>3</sub> 150 mEq @ 500 cc/Hr	
<b>05:42 hours</b>	Assisted	SPONTANEOUS	Not Noted	D50	
<b>05:43 hours</b>	Assisted	SPONTANEOUS	Not Noted	Epinephrine 1 mg.; Started Epi get, Vasopressin Drip (Pitressin Drip) 250 units in D50 250 ml, 0.01 units/min on demand, Max. drip rate 0.04 units/min	
<b>05:46 hours</b>	Assisted	SPONTANEOUS	Wide QRS		
<b>05:48 hours</b>	Assisted	SPONTANEOUS	Wide QRS	NaHCO <sub>3</sub>	

<u>BLOOD TEST RESULTS</u>	<u>COMMISSION INTERPRETATION/INDICATION</u>
<p> <b>pH</b> – 6.973 L (7.35-7.45)  <b>pCO<sub>2</sub></b> – 66.8 H (35-45 mmHg)  <b>HCO<sub>3</sub><sup>-</sup></b> – 15.1 L (22-26 mmol/L)  <b>BEecf</b> – -17.3 L (+/- 4 mmol/L)  <b>pO<sub>2</sub></b> – 98.7 (75.0-100.0 mmHg)  <b>tHb</b> – 14.2 (12.0-18.0 g/dL)  <b>O<sub>2</sub>Hb</b> – 90.5 L (94.0-97.0 %)  <b>CO<sub>2</sub>Hb</b> – 0.2 L (0.5-1.5%)  <b>MetHb</b> – 0.2 (0.0-1.5%) </p>	<p>7 hr. 59 min. after the end of the Bout, Jones has been in cardiac arrest for 38 minutes as the end stage result of severe Exertional Rhabdomyolysis, renal dysfunction with likely damage to more than 60% of the nephrons (basic structural and functional units of the kidneys), and dehydration</p> <p><b>EVIDENCED BY:</b>  PCO<sub>2</sub> &amp; HCO<sub>3</sub> &amp; BEecf levels show worsening extreme metabolic acidosis with the respiratory system failing to compensate and in fact adding to acidosis through respiratory acidosis.</p>

<u>Time</u>	<u>Respiration</u>	<u>Pulse</u>	<u>Heart Rhythm</u>	<u>Medication</u>	<u>Notes</u>
<b>05:49 hours</b>	Assisted	SPONTANEOUS	Wide QRS	Zversed	
<b>05:50 hours</b>	Assisted	SPONTANEOUS	V-Tach	Mg 2 grams	Defibrillator Shock 200 Joules
<b>05:52 hours</b>	Assisted	SPONTANEOUS	Wide QRS	Increased NaHCO <sub>3</sub> get to 2 <sup>nd</sup>	

<b>05:53 hours</b>	Assisted	SPONTANEOUS	Wide QRS	D50 x2	
<b>05:56 hours</b>	Assisted	SPONTANEOUS	Wide QRS	Insulin 20 units	
<b>06:05 hours</b>	Assisted	Compressions	V-Tach		Defibrillator Shock 300 Joules
<b>06:06 hours</b>	Assisted	Compressions	V-Tach		Defibrillator Shock 360 Joules
<b>06:08 hours</b>	Assisted	Compressions	Asystole	CaCl 8-16 mg/kg	
<b>06:16 hours</b>	Assisted	Compressions	V-Tach	Epinephrine 1 mg.	Defibrillator Shock 360 Joules

<u>BLOOD TEST RESULTS</u>	<u>COMMISSION INTERPRETATION/INDICATION</u>
<p> <b>Na<sup>+</sup></b> – 131.8 L (135-145 mmol/L)  <b>K<sup>+</sup></b> – 9.77 CH (3.5-5.0 mmol/L)  <b>Cl<sup>-</sup></b> – 101 (100-109 mmol/L)  <b>Glucose</b> – 348 H (70-105 mg/dL)  <b>pH</b> – 7.003 L (7.35-7.45)  <b>pCO<sub>2</sub></b> – 51.3 H (35-45 mmHg)  <b>HCO<sub>3</sub><sup>-</sup></b> – 12.5 L (22-26 mmol/L)  <b>BEecf</b> – -18.9 L (+/- 4 mmol/L)  <b>pO<sub>2</sub></b> – 159.3 H (75.0-100.0 mmHg)  <b>Ca, Ionized</b> – 1.06 L (1.15-1.33 mmol/L)  <b>Lactate</b> – 11.82 CH (0.5-2.2 mmol/L)  <b>PEEP</b> – 5.0 cmH2O </p>	<p>8 hr. 27 min. after the end of the Bout, Jones has been in cardiac arrest for 1 hour 6 minutes as the end stage result of severe Exertional Rhabdomyolysis, renal dysfunction, and dehydration</p> <p><b>EVIDENCED BY:</b>  All electrolyte levels worsened and not recoverable;  PCO<sub>2</sub> &amp; HCO<sub>3</sub> &amp; BEecf levels worsening and likely not recoverable.</p>

**06:17 hours –**

<u>BLOOD TEST RESULTS:</u>	<u>COMMISSION INTERPRETATION/INDICATION</u>
<p> <b>Na<sup>+</sup></b> – 135 L (135-145 mmol/L)  <b>K<sup>+</sup></b> – 10.3 CH* (3.5-5.0 mmol/L)  <b>Cl<sup>-</sup></b> – 99 L (100-109 mmol/L)  <b>CO<sub>2</sub></b> – 13 L (21-32 mmol/L)  <b>BUN</b> – 24 H (6-20 mg/dl)  <b>CREATININE</b> – 4.8 H (0.6-1.3 mg/dL)  <b>Glucose</b> – 407 H (70-105 mg/dL)  <b>GFR</b> – 18 L (&gt;90 mL/min/1.73m<sup>2</sup>)  <b>Ca<sup>2+</sup></b> – 9.2 (8.6-10.2 mg/dL)  <b>Ca, Ionized</b> – 1.10 L (1.15-1.33 mmol/L)  <b>PO<sub>4</sub></b> – &gt;24 CH (2.5-4.5 mg/dL)  <b>Mg<sup>2+</sup></b> – 7.1 H* (1.6-2.6 mg/dL)  <b>Prothrombin</b> – 18.4 H (12.0-14.0 sec)  <b>INR</b> – 1.5 L (0.9-1.3 sec)  <b>PTT</b> – 35.8 (23-36.9 sec)  *Specimen slightly hemolyzed, so K may be falsely elevated </p>	<p>8 hr. 28 min. after the end of the Bout, Jones has been in cardiac arrest for 1 hour 7 minutes as the end stage result of severe Exertional Rhabdomyolysis, renal dysfunction, and dehydration</p> <p><b>EVIDENCED BY:</b>  All electrolyte levels worsened and not recoverable;  PCO<sub>2</sub> &amp; HCO<sub>3</sub> &amp; BEecf levels worsening and likely not recoverable.</p>

<u>Time</u>	<u>Respiration</u>	<u>Pulse</u>	<u>Heart Rhythm</u>	<u>Medication</u>	<u>Notes</u>
<b>06:18 hours</b>	Assisted	Compressions	V-Tach	CaCl 1 gram	
<b>06:22 hours</b>	Assisted	SPONTANEOUS	V-Tach	NaHCO <sub>3</sub>	
<b>06:23 hours</b>	Assisted	SPONTANEOUS	V-Tach	NaHCO <sub>3</sub>	
<b>06:24 hours</b>	Assisted	SPONTANEOUS	V-Tach	D50	
<b>06:25 hours</b>	Assisted	SPONTANEOUS	V-Tach	Insulin 20 units	

<b>06:26 hours</b>	Assisted	SPONTANEOUS	Wide QRS	Lidocane 100 ng.	
<b>06:27 hours</b>	Assisted	SPONTANEOUS	V-Tach	Mg 2 grams, Increase Epi get	
<b>06:30 hours</b>	Assisted	SPONTANEOUS	V-Tach	D50, CaCl 1 gram	
<b>06:32 hours</b>	Assisted	SPONTANEOUS	V-Tach	NaHCO <sub>3</sub>	
<b>06:33 hours</b>	Assisted	SPONTANEOUS	Wide QRS	CaCl 1 gram, NaHCO <sub>3</sub>	
<b>06:35 hours</b>	Assisted	SPONTANEOUS	Wide QRS	CaCl 1 gram	
<b>06:36 hours</b>	Assisted	SPONTANEOUS	Wide QRS	CaCl 1 gram	
<b>06:37 hours</b>	Assisted	Compressions	V-Fib	D50	Defibrillator Shock 360 Joules
<b>06:40 hours</b>	Assisted	Compressions	V-Fib		Defibrillator Shock 360 Joules
<b>06:42 hours</b>	Assisted	Compressions	V-Fib		Defibrillator Shock 360 Joules

**06:42 hours** – Asystole. CODE ENDED by Surgical attending physician – **TIME OF DEATH CALLED**

**06:43 hours** –

<u>BLOOD TEST RESULTS:</u>	<u>COMMISSION INTERPRETATION/INDICATION</u>
<b>Na<sup>+</sup></b> – 131 L (135-145 mmol/L) <b>K</b> – >9.0 CH (3.5-5.0 mmol/L) <b>Cl</b> – 110 H (100-109 mmol/L) <b>BUN</b> – 36 H (6-20 mg/dl) <b>Glucose</b> – 482 H (70-105 mg/dL) <b>Hct/HEMATOCRIT</b> – 27 L (40-52%) <b>Hgb/Hb/HEMOGLOBIN</b> – 9.2 L (14-18 g/dL)	8 hr. 54 min. after the end of the Bout, Jones is pronounced dead as the result of a cascading systems failure ending with cardiac arrest onset by hyperkalemia resulting from severe exertional rhabdomyolysis and resulting renal failure.

**08:40 hours** – Commissioner Stuart received notice of Jones’ death and implemented standard protocols for notification of the necessary persons and beginning the Commission’s investigation.

**12:45 hours** – Commissioner Stuart arrived at Venue, collected and sequestered all Event Handwraps and Gloves. The Handwraps and Gloves were later examined and found to be in compliance with the Commission’s Regulations with no evidence of illegal substances or objects having been involved in this incident.

Due to their voluminous nature and relative lack of importance, the Commission’s remaining investigatory details are not provided in timeline format.

**SECTION IV**  
**CAUSE OF DEATH FINDINGS, DETERMINATIONS & ANALYSIS**

**A. SPECIAL THANK YOU TO THE MEDICAL TEAMS**

The Commission wishes to extend its deepest thanks and appreciation for the extraordinary efforts of the Arkansas State Medical Examiner’s Office (“ASME”), especially the forensic pathologist of record for this matter, Associate Medical Examiner – Dr. Daniel Dye, M.D., who expended extra effort and many extra hours of his time on this case to accommodate the Commission’s extraordinary requests for additional forensic testing specific to Combative Sports related injuries.

The Commission would also like to thank the medical staffs of both Saline Memorial Hospital and the University of Arkansas for Medical Sciences Hospital (“UAMS”), especially Dr. Ronald D. Robertson, M.D. and the other treating physicians, all of whom provided world class treatment to Jones in his time of crisis. It is comforting to know the State of Arkansas is served by such dedicated, highly trained men and women.

The Commission would also like to thank the clinical and research staff at the University of California at Los Angeles Olympic Analytical Research Laboratory, without which the Commission would not have been able to receive the proper specimen analysis for collection of prohibited substance data.

Finally, the Commission would like to thank those physicians who consulted with the Commission in the preparation of this report. As a small, self-funded state agency, the Commission was not able to sufficiently compensate the consulting physicians, thus the Commission truly appreciates the time and knowledge each physician willingly donated to the good of the citizens of the State of Arkansas and the members of the general public outside the State of Arkansas, each of whom may benefit from the information and analysis proved by this report.

Data collection and analysis was critically important for this report. Without the information, observations, testing and data collected by the professionals working at Saline Memorial Hospital, UAMS, UCLA Olympic Analytical Research Laboratory and the ASME’s Office, it would have been much more difficult, if not impossible to actually find the cause of Jones’ death and to provide the teaching tools derived from this unfortunate incident.

**B. THE SHORT & OVERLY SIMPLISTIC ANSWER USING AN ANALOGY**

The short and overly simplistic answer is that Mr. Jones died as the result of a cascading systems failure caused by extraordinarily complex biological and chemical processes of multiple origins.

The 2004 Warner Brothers Pictures movie, Million Dollar Baby, is a very good movie about boxing and it provides great insight into not only the dangers of boxing, but also, what happens inside the ring and why contestants step inside the ring to participate. Million Dollar Baby is about a female, Maggie Fitzgerald played by Hilary Swank, who had a dream to start boxing and become the best boxer she could. In the movie, Maggie’s trainer/manager is Frankie Dunn played by Clint Eastwood. The movie is narrated by a character named Eddie “Scrap Iron” Dupris played by Morgan Freeman. Through the voice of Morgan Freeman, Million Dollar Baby describes in a very simplistic manner how different boxing is from almost everything else in life and what happens when a Contestant gets knocked out:

Boxing is an unnatural act, 'cause everything in it is backwards. You wanna move to the left, you don't step left, you push off the right toe. To move right, you use your left toe. Instead of running from the pain – like a sane person would do – you step into it. . . . Everything in boxing is backwards.

The body knows what fighters don't – how to protect itself. A neck can only twist so far. Twist it just a hair more and the body says, "Hey, I'll take it from here because you obviously don't know what you're doing. Lie down now, rest and we'll talk about this when you regain your senses." It's called the knockout mechanism.

Morgan Freeman as Eddie Scrap-Iron Dupris  
Million Dollar Baby (2004)  
Warner Bros. Pictures

By way of another overly simplistic analogy, when the human body cannot figure out how to resolve multiple problems with multiple organs or internal systems or otherwise suffers a major trauma, the body naturally sends out signals to shut down and restart its various systems, very much like a computer shutting down and rebooting itself to fix problems with its operating system. During the body's natural shut down and restart process, problems are many times resolved without any complications; however, in this case, Mr. Jones' critical systems, such as the heart and kidneys, were unable to be reset due to complex, underlying biological and chemical processes. Such an overly simplistic answer fails to give us a real understanding of exactly what happened to Mr. Jones and whether or not his death could have been prevented or whether a similar incidents can be prevented in the future through regulatory or testing protocols.

### **C. ARKANSAS STATE MEDICAL EXAMINER'S CAUSE OF DEATH FINDINGS & DETERMINATIONS**

Although the ASME and the Arkansas State Crime Laboratory, under which the ASME operates, are not specifically designed or equipped to analyze Combative Sports related injuries and deaths, the ASME's office and more specifically, the forensic pathologist of record, Dr. Daniel Dye, M.D., did an outstanding job reaching a sound, well reasoned medical and scientific conclusion with respect to Jones' cause of death. After performing an advanced autopsy including several tests and analyses with a specialized focus on Combative Sports injuries and conditions, Dr. Dye's findings were as follows:

**PRIMARY CAUSE OF DEATH:** 1.) Commotio Cerebri

**Secondary/Contributing Factors:** 1.) Cardiomyopathy of undetermined etiology (500 grams)  
2.) Dehydration  
3.) Rhabdomyolysis  
4.) Hyperkalemia

**Evidence of Old Injury:** NONE

**Evidence of New Injury:** 1.) Hemorrhage in right temporalis muscle.  
2.) Inferior/medial aspect of the right temporal bone has a 2.4 cm hairline fracture.  
3.) Hemorrhage is present in the soft tissues of the skull base under the fracture.  
4.) Microscopic subarachnoid hemorrhage and cerebral contusions are detected in a section (Slide 6) of the parietal lobe of the right cerebral hemisphere.

## **D. COMMISSION'S CAUSE OF DEATH FINDINGS & DETERMINATIONS**

**PRIMARY CAUSE OF DEATH:** 1.) Cardiac Arrest due to Cardiac Dysrhythmia – Specifically Left Ventricular Fibrillation onset by Hyperkalemia

**Secondary/Contributing Factors:**

- 1.) Exertional Rhabdomyolysis;
- 2.) Acute Renal Failure;
- 3.) Liver Steatosis;
- 4.) Lactic Acidosis;
- 5.) Dehydration;
- 6.) Severe Electrolyte Imbalance;
- 7.) Commotio Cerebri;
- 8.) Anabolic Steroid Use;
- 9.) Overuse of Nutritional Supplements; and
- 10.) Cardiomyopathy of Undetermined Etiology (500 grams).

**Evidence of Old Injury:** None noted; however, additional, highly specialized forensic testing and analysis would likely have revealed previous damage to left ventricular heart structures, kidneys, and liver resulting from alcohol and steroid abuse and overuse of nutritional supplements.

**Evidence of New Injury:**

- 1.) Critically High Potassium, Critically Low Calcium, Wild Fluctuation & Imbalance of Other Electrolyte Levels, and Blood & Urine Test Markers for the Various Positive Condition Markers.
- 2.) 80% of liver's hepatocytes involved by macro and micro steatosis.
- 3.) Metabolic Acidosis (notably Lactic Acidosis).
- 4.) Multiple Prolonged Prothrombin Time Results.
- 5.) Hemorrhage in right temporalis muscle.
- 6.) Inferior/medial aspect of the right temporal bone has a 2.4 cm hairline fracture.
- 7.) Hemorrhage in the soft tissues of the right temporal skull base under fracture.
- 8.) Microscopic subarachnoid hemorrhage and cerebral contusions detected in section of the parietal lobe (Slide 6) or the right cerebral hemisphere.

## **E. EXPLANATION OF WHY THE ASME & COMMISSION FOUND DIFFERENT PRIMARY & CONTRIBUTORY/SECONDARY CAUSES OF DEATH**

What follows in the next subsection is the much more complex and informative answer as to what caused Jones' death. Unfortunately, it would take several hundred pages to fully explain the science behind every medical, scientific and chemical process or compound causing or contributing to Jones' death; thus, the Commission has condensed this report as much as possible, while still retaining the details and meaningful summaries necessary to make this report both educational and informative to the widest array of readers possible. This report is intended to educate and inform Contestants, Seconds/Cornermen, trainers, Officials, physicians, researchers, and even members of the general public who otherwise do not participate in Combative Sports because they view it as an outdated, dangerous and barbaric sport, which serves no purpose.

A multitude of Jones' activities, habits and practices prior to participation in the Bout set in motion the extraordinarily complex biological and chemical processes resulting in his death. Each of Jones' biological, chemical and physical conditions, if existing alone, very likely would not have caused Jones' death; however, when all were present under the conditions Jones experienced during the Bout, his body simply could not overcome the cumulative effects. The complex biological and chemical processes involved in this case made it remarkably difficult for the professionals to reach a consensus as to the specific cause of death. In fact, due to the multiple origins and particularly complex nature of the biological, chemical and physical processes involved, it is possible for reasonable, medically and professionally trained minds to disagree on Jones' specific, primary cause of death; however, all agree as to the secondary/contributing conditions or factors.

Perhaps Jones' death is best explained through another analogy. The myriad physical and chemical tests and observations revealed Jones had many, very bad physical and chemical conditions present in his body, each of which could be compared to a single Domino<sup>®</sup> standing on its end. So, imagine a well known game where people stack Dominos<sup>®</sup> on their ends side by side with the object being to avoid knocking over all of the other Dominos<sup>®</sup>. If each Domino<sup>®</sup> is placed far enough from the others that it will not touch another when it falls, then any Domino<sup>®</sup> in the line can be randomly pushed over and the rest of the Dominos<sup>®</sup> will remain standing. However, if each Domino<sup>®</sup> is placed too close to the others, then when one Domino<sup>®</sup> falls each of the other Dominos<sup>®</sup> will also fall in succession until they have all fallen.

What happened to Jones' here is just like a line of Dominos<sup>®</sup> all placed too closely together. Due to the very specific circumstances present, when Jones' first system came under attack (i.e. the first Domino<sup>®</sup> fell), the failure placed insurmountable stress on his other internal systems; thus, each failed in rapid succession, just like a line of Dominos<sup>®</sup>.

Identifying Jones' "Primary Cause Death" is very much like having to identify which specific Domino<sup>®</sup> fell first. Thus, while every condition noted by both the Arkansas State Medical Examiner's Office ("ASME") and the Commission can be medically proven to have either caused or contributed to Jones' death, the "Primary Cause of Death" listed under the Forensic Pathologist's Findings and the Commission's Findings are different because each agency rendered their professional opinion based on which condition or factor it believes to be the most scientifically supported in view of all of the information respectively available to them at the time. The differences of opinion between the ASME and the Commission do not mean that one agency is right and the other is wrong. Instead, both agency's determinations are very likely correct, based on the information available to them and their particular agency's purpose and viewpoint.

For instance, the ASME's office falls under the jurisdiction of the Arkansas State Crime Laboratory and the ASME's office is most often asked to determine a cause of death for purposes of determining criminal liability. If the same person is in rapid succession mortally wounded by a gunshot from person A, then person B mortally wounds the person with a knife to the stomach after he is shot, and finally the same person after being shot and wounded with a knife is run over by a car driven by person C, the ASME's office has the responsibility of trying to determine whether it was the gunshot, the knife wound or impact from the car that actually killed the person in order that the person committing the fatal act can be charged with the appropriate crime. While the other actions no doubt will have contributed to the person's death and each, by itself, could have killed the person, the ASME must pick, with the appropriate degree of medical certainty, the act that actually killed the person and testify in court for criminal conviction purposes.

While the ASME investigates and analyzes every piece of relevant information actually presented to its office, the ASME's investigation is limited to the evidence with which it is presented. The ASME does not go out into the field and investigate leads, conduct interviews and find other relevant evidence as is depicted on the popular CBS television series, Crime Scene Investigators ("CSI"). Instead, the investigation portion is left to the detectives and police officers from the referring agency.

On the other hand, the Commission's purpose is regulatory in nature and primarily driven by an overriding directive to protect the health, safety and welfare of combative Sports Contestants, Seconds/Cornermen, Officials and general public. To this end, when reviewing a Combative Sports related serious injury or death, the Commission looks to find the underlying cause or causes of the particular injury or death and follows the evidentiary trails to determine what the Commission believes to be the most likely cause, along with all contributory factors, in relation to the specialized medical and scientific fields associated with Combative Sports and competitive physical activities.

Unlike the ASME, the Commission does have the authority and is charged with going into the field to investigate leads, conduct interviews and gather and find other relevant evidence during the Commission's investigations. As explained elsewhere in this report, the Commission conducted hundreds of hours of expert consultation, investigation, interviews, document and other evidence review prior to making its determinations and findings herein.

## **F. EXPLANATION & DESCRIPTION OF THE ASME'S & COMMISSION'S PRIMARY & CONTRIBUTORY/SECONDARY CAUSES OF DEATH**

### **1. The ASME's Primary Cause of Death – Commotio Cerebri**

The ASME found Jones' primary cause of death to be "Commotio Cerebri," a Latin term for "agitation of the brain" and most often used as a very general medical term describing a concussion. On the other hand, the Commission's findings and conclusions indicate commotio cerebri is only secondary/contributing factor to Jones' cause of death rather than the primary cause. The reasons for the difference of opinion are explained in this section through a discussion of the autopsy findings, medical history, analysis of the facts of Jones' case in relation to the various functions of each part of the brain, and the most current medical science and studies involving exertional rhabdomyolysis and mild traumatic brain injuries.

The physiological and biochemical explanations of commotio cerebri or concussion are also explained in this section to educate Contestants, Cornermen/Seconds/Trainers, Ringside Doctors and Officials on how to detect possible concussions and reduce the likelihood of occurrence and severity once they occur.

#### **(a.) What is a Concussion/MTBI? Did Jones Have One?**

Concussions are the most common type of traumatic brain injury and are also referred to as mild brain injury (MBI), mild traumatic brain injury (MTBI), mild head injury (MHI), and minor head trauma (MHT).

MTBI has a mortality rate of almost zero and only about one percent (1%) of those persons presenting at hospitals with MTBI actually need any type of surgical or other medical intervention. (Borg J, Holm L, Cassidy JD, *et al.* (2004). "Diagnostic procedures in mild traumatic brain injury: Results of the WHO collaborating centre task force on mild traumatic brain injury." *Journal of Rehabilitation Medicine* 36(Suppl. 43): 61-75.) However, the low mortality rate alone is not the reason for the Commission's movement of commotio cerebri from the primary cause to secondary/contributing factor category.

No singular, universally accepted definition of concussion exists within the medical profession; however, in general, neurologists agree a concussion is:

- a complex pathophysiological process affecting the brain;
- induced by biomechanical forces typically involving temporary impairment of neurological function;
- healed by itself over time; and
- a condition for which neuroimaging normally shows no gross structural changes to the brain as a result.

Intracranial hemorrhages (e.g. intra-axial hematoma, epidural hematoma and subdural hematoma) are not necessarily precluded in MTBI or mild head injury, but they are in concussion. Accordingly, concussion is typically and more properly used to imply a state of temporary brain function impairment, while MTBI implies a pathophysiological state.

When the facts outlined in the detailed timeline above are analyzed against the following discussion on the signs, symptoms and methods of detecting a concussion, it becomes clear Jones had a very mild concussion or MTBI.

**(b.) What Causes a Concussion/MTBI? Did Jones Suffer a Concussive Impact?**

The human brain is surrounded and suspended inside the skull by a somewhat thick fluid called cerebrospinal fluid. Concussions are caused by the brain being agitated in a way that overwhelms the cerebrospinal fluid's ability to adequately slow the motion before the brain impacts the skull. The exertional forces of the agitation can cause linear, rotational or angular movement of the brain or any combination of these movements before the brain impacts the skull. Concussions, especially American Academy of Neurologists Grade I concussions like the one Jones had in this case, can be very difficult to detect because they do not always involve the loss of consciousness. Here, Jones never lost consciousness and was verbally responsive both inside the ring and at the hospital.

Concussions are not only experienced in Combative Sports such as boxing and mixed martial arts, but also by participants in all manner of contact and non-contact sports on a daily basis. For example in non-contact sports, cheerleaders fall from the top of pyramid formations and hit their head on the ground or their teammates, basketball players fall and hit their head on the court, and baseball players run into each other or get hit by the ball. In other contact sports, football players often collide in a way (not always with helmet to helmet contact) that agitates their brains inside their skull. In boxing and mixed martial arts, it is unquestionable concussive impacts are delivered from not only the hands or feet to the head, but also from accidental headbutts and Contestants hitting their head on the canvas during either a take down or Knock Down.

In Jones' Bout with Palmer, there were no accidental headbutts, but Jones and Palmer both gave and received multiple punches to the head, which had sufficient force to cause a concussion. It was also observed and confirmed upon review of the video replay that Jones hit his head very hard on the padded canvas when he fell at the end of the 2<sup>nd</sup> Round. A review of thousands of rounds of boxing and mixed martial arts have enabled physicians to determine that Contestants often sustain concussions not from the punches received during the Bout, but rather, from hitting their head on the floor of the Ring when they fall. In this case, it is not possible to know for sure if Jones' concussion was caused by the punches, the floor of the Ring or a combination of both. From Commissioner Stuart's personal observation and from a review of the Bout's video, it was observed that it is very likely Jones and Palmer both sustained concussions from punches received by each during the Bout.

In fact, despite the indication on Palmer's Post-Bout Physical that a longer than 7 day mandatory suspension under Reg. §1.19.15 was not medically necessary, Commissioner Stuart nevertheless suspended Palmer for 30 days pursuant to Reg. §1.19.16 based on his experience and opinion that such 30 day suspension was justified by the number and severity of punches Palmer received from Jones, including the facts that Palmer was knocked down once and received a Standing Eight Count during the 1<sup>st</sup> Round.

Commissioner Stuart's 30 day suspension of Palmer is not unusual and the winners of Bouts, whether by Knockout or Technical Knockout or not, are often suspended by the Commission for longer than the 7 day mandatory rest period as a simple safety precaution based either on the Ringside Physician's or Commission Representative's observations during the Bout. The Contestants who are suspended for longer than the 7 day period often do not like it and loudly protest; however, the suspensions are routinely issued by the Commission for the Contestant's safety. Contestants are always permitted to request a hearing to contest any suspension and if sufficient evidence exists to lift the suspension, the Commission may do so, but only after the Commission is satisfied there is no substantially increased risk of harm to the Contestant from lifting the suspension early.

**(c) What are the Signs & Symptoms of Concussions/MTBI? Did Jones Display Them?**

Symptoms of a concussion include, but are not limited to, unconsciousness, pupils non-reactive to light, altered mental status; convulsions; dizziness, memory loss, severe, persistent headache; extremity weakness; vomiting; ringing or deafness in either or both ears. Here, Jones was never unconscious, had reactive pupils and was responsive with only slightly altered mental status and dizziness without memory loss. While at UAMS, Jones complained of lower extremity weakness (i.e. couldn't move his legs); however, such was in the Commission's opinion likely related to the other medical conditions existing within Jones' body at the time rather than a concussion.

As demonstrated in the timeline from 21:49 hours on 29-JAN-2011 forward, Jones never lost consciousness and his pupils remained reactive to light, but were sluggish and pinpoint at times. Jones did experience dizziness, transient memory loss of less than 15 minutes and later extremity weakness. While not displaying all of the signs and symptoms, Jones did display enough of the signs to safely diagnose him with a mild concussion.

**(d) What is Second Impact Syndrome? Did Jones Have It?**

For a period of minutes to days after a concussion, the brain is especially vulnerable to changes in intracranial pressure, blood flow, and anoxia. According to studies performed on animals (which are not always applicable to humans), large numbers of neurons can die during this period in response to slight, normally innocuous changes in blood flow. Also, as a result of the numerous vulnerabilities, the brain is much more susceptible to additional concussions and other secondary injury events.

Secondary injury events include damage to the blood brain barrier, release of factors that cause inflammation, free radical overload, excessive release of the neurotransmitter glutamate (excitotoxicity), influx of calcium and sodium ions into neurons, and mitochondrial dysfunction. Injured axons in the brain's white matter may separate from their cell bodies as a result of secondary injury, potentially killing those neurons. Other factors in secondary injury are changes in the blood flow to the brain; ischemia (insufficient blood flow); cerebral hypoxia (insufficient oxygen in the brain); cerebral edema (swelling of the brain); and raised intracranial pressure (the pressure within the skull). Intracranial pressure may rise due to swelling or a mass effect from a lesion, such as a hemorrhage. As a result, cerebral perfusion pressure (the pressure of blood flow in the brain) is reduced; ischemia results. When the pressure within the skull rises too high, it can cause brain death or herniation, in which parts of the brain are squeezed by structures in the skull.

Concussions have a cumulative effect and suffering one concussion greatly increases the likelihood of suffering another concussion, especially if the event is close in time to the previous concussion. In certain cases called second impact syndrome, the brain swells to dangerous levels after only a minor impact in the days or weeks following a previous concussion. In the most severe cases of second impact syndrome, the brain can herniate, resulting in intracranial pressure on the brain stem and death occurring within five minutes. (Bowen, A.P. (2003). "Second Impact Syndrome: A rare, catastrophic, preventable complication of concussion in young athletes." *Journal of Emergency Nursing*, 29(3), 287–289.) Although there is no scientific evidence to support the observation, empirical data suggests second impact syndrome is more likely to occur in younger athletes, especially those younger than twenty years old. (*Id.*).

There is no consensus on a definitively safe period of time between suffering concussions or concussive impacts, although the Commission and multiple regulating bodies from state athletic commissions and national association of high school athletics to the NFL and NBA are all constantly looking for verifiable scientific data to assist in the establishment of so called "Return To Play Guidelines." The NFL is currently conducting the most active and well funded ongoing research in the area of concussive impacts in contact sports. The Commission regularly receives updates on Return to Play Guidelines and studies on concussions in sports to maintain its currency on the latest scientific research in this area. Most recently, the Commission sent Commissioner Stuart and three (3) Arkansas physicians who regularly work Events for the Commission from ringside or consult with the Commission, to the Sports Concussion Institute's National Symposium on Concussions in Sports in May of 2010 to be briefed on the latest developments and research.

It is notable in this case that Jones did have at least one previous concussion recalled by his friends; however, the previous concussion was years earlier, during his days of playing football. Although Jones' brain sections did not reveal any injuries, such as scarring, consistent with previous concussive impacts, experience tells us that it is nevertheless very likely Jones suffered one or more mild to moderate concussions while playing high school and college football; however, such previous concussions likely went unnoticed and undiagnosed. Instead, historically, high school and college football players who "just had their bell rung" have been told the shake it off and get back in on the next play. Now however, modern science tells us that such attitudes toward mild concussions are no longer appropriate and athletes should follow strict return to play standards and guidelines appropriate for their particular sport. For example, the Commission's Regulations already contain mandatory 7 day, 30 day, 90 day and indefinite suspensions (Reg. §1.19.15; §1.19.16; §1.19.17; and §1.19.18) to safeguard Contestants' neurological health. During the time of each Contestant's suspension, the Contestant is supposed to not only refrain from participating in another Bout, but also observe a "no contact" training schedule which includes no sparring during the suspension period.

(e.) **How do We Test for Concussion/MTBI & Grade or Determine the Severity of Concussions/MTBI? How Severe was Jones' Concussion & Did the Tests Indicate the Need for Medical Intervention or Treatment for a Concussion/MTBI?**

The severity of a concussion is not necessarily related to the severity of the impact; however, studies by the Sports Concussion Institute in conjunction with the National Football League, as well as others, have directly linked the severity and recovery time of concussions to dehydration, glucose levels and electrolyte imbalances. The likelihood and severity of a concussion initially rises in a somewhat linear relation to increased dehydration, glucose reduction and electrolyte imbalance and at a certain point, the dehydration, glucose and electrolyte imbalance reaches levels where concussions become exponentially more likely and severe. In short, the more dehydrated an athlete is and more of an electrolyte imbalance or reduced glucose level he or she has, the more likely the athlete is to suffer a concussion and increase the severity of any concussion.

Here, it is without question and both the ASME and Commission agree Jones was experiencing some level of dehydration, although the reasons for and severity of his dehydrated state at the time of the Bout will not become evident until later in this report.

The preferred radiologic test in the emergency setting is computed tomography (CT) scans because they are quick, accurate, and widely available. Follow-up CT scans may be performed later to determine whether the injury has progressed. Magnetic resonance imaging (MRI) can show more detail than CT, and can add information about expected outcome in the long term. MRI is more useful than CT for detecting injury characteristics such as diffuse axonal injury in the longer term. However, MRI is not used in the emergency setting for reasons including its relative inefficacy in detecting bleeds and fractures, its lengthy time image acquisition, the inaccessibility of the patient while in the machine, and its incompatibility for use with metal items often used in the emergency care setting.

Although the tests are still recommended for all but very mildly concussed individuals, most concussions can be detected with neither an MRI nor a CT scan, since concussions themselves have no particularly identifiable characteristics in such images. (Poirier, MP (2003). "Concussions: Assessment, management, and recommendations for return to activity (abstract)." *Clinical Pediatric Emergency Medicine*, 4(3): 179–185.) While CT scans cannot detect most concussions, changes have been noted in MRI and SPECT imaging in concussed individuals with normal CT scans with post-concussion syndrome being possibly associated with SPECT and PET scan abnormalities. (Iverson, GL (2005). "Outcome from mild traumatic brain injury." *Current Opinion in Psychiatry*, 18(3): 301-317.)

Further, there is "strong evidence that clinical factors can predict computerized tomography scan abnormalities and the need for intervention in adults, but no such evidence for mild traumatic brain injury in children. [Plus, there is] substantial evidence that skull fracture is a risk factor for intracranial lesions, but the diagnostic accuracy of radiologically diagnosed skull fracture as an indication of intracranial lesions is poor." (Borg J, Holm L, Cassidy JD, *et al.* (2004). "Diagnostic procedures in mild traumatic brain injury: Results of the WHO collaborating centre task force on mild traumatic brain injury." *Journal of Rehabilitation Medicine* 36(Suppl. 43): 61–75.) It is presently unknown to what extent, if any, non-surgical intracranial lesions on CT scans are important in determining the long term effects of concussions.

With respect to concussed athletes, one Phase II study yielded evidence that recent memory questions are more sensitive than orientation questions in the assessment of cognitive function in concussed athletes. (Maddocks, DL, Dicker, GD, Saling, MM (1995). "The assessment of orientation following concussion in athletes." *Clinical Journal of Sports Medicine* 5:32-35.) Another Phase II study provided evidence for the validity of a brief measure of cognitive functioning, the Standardized Assessment of Concussion, for detecting the immediate effects of MTBI on cognition. (Barr, WB, McCrea, M (2001). "Sensitivity and specificity of standardized neurocognitive testing immediately following sports concussion." *Journal of International Neuropsychologists Society* 7:693-702.) While the two studies mentioned above revealed support for standardized testing to determine cognitive impairment due to concussion, other studies have not found such support; thus, more studies in this area are required to reach a consensus on the validity of the tests. In the opinion of the Commission, verbal, standardized assessment tests should be performed on each Contestant following every Bout, since such tests are easy and inexpensive to perform and have shown positive correlation to possible MTBI.

The following three (3) concussion grading systems are followed most widely:

- (i.) Robert Cantu Guidelines;
- (ii.) Colorado Medical Society Guidelines; and
- (iii.) American Academy of Neurology Guidelines.

Each divides a concussion into three grades, as summarized in the following table:

	<b>GRADE I</b>	<b>GRADE II</b>	<b>GRADE III</b>
<b>CANTU GUIDELINES</b>	Post-traumatic amnesia less than 30 minutes; No loss of consciousness	Loss of consciousness less than 5 minutes or amnesia lasting 30 minutes to 24 hours	Loss of consciousness greater than 5 minutes or amnesia greater than 24 hours
<b>COLORADO MEDICAL SOCIETY GUIDELINES</b>	Confusion; No loss of consciousness	Confusion; Post-traumatic amnesia, No loss of consciousness	Any loss of consciousness
<b>AMERICAN ACADEMY OF NEUROLOGY GUIDELINES</b>	Confusion; Symptoms last less than 15 minutes; No loss of consciousness	Symptoms last greater than minutes; No loss of consciousness	Loss of consciousness; (IIIa, Coma lasts seconds, IIIb for minutes)

The Glasgow Coma Scale (“GCS”) comprises three tests: eye, verbal and motor responses. The three values separately as well as their sum are considered to determine the existence of a concussion or traumatic brain injury. The lowest possible GCS (the sum of the numbers associated with each applicable response/observation) is 3, which equates to a deep coma or death, while the highest is 15, which equates to a fully awake person. For Glasgow Coma Scale scoring, the individual elements as well as the sum of the score are important. Thus, the score is expressed as: GCS 9 = E2 V4 M3. On the Glasgow scale, brain injury is classified by scores as follows:

- (i.) **Severe** – GCS  $\leq$  8;
- (ii.) **Moderate** – GCS 9 – 12; and
- (iii.) **Minor** – GCS  $\geq$  13.

	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5</b>	<b>6</b>
<b>BEST EYE RESPONSE (E)</b>	No eye opening	Eye opening in response to pain (e.g. Patient responds to pressure on fingernail bed OR supraorbital or sternal rub	Eye opening to speech (Not to be confused with an awakening of a sleeping person; such patients receive a 4 not 3)	Eyes opening spontaneously		
<b>BEST VERBAL RESPONSE (V)</b>	No verbal response	Incomprehensible sounds (Moaning, but no words)	Inappropriate words (Random or exclamatory articulated speech, but no conversational exchange)	Confused (Patient responds to questions coherently but there is some disorientation and confusion)	Oriented (Patient responds coherently and appropriately to questions such as the patient’s name and age, where they are and why, the year, month, etc.)	
<b>BEST MOTOR RESPONSE (M)</b>	No motor response	Extension to pain (abduction of arm, internal rotation of shoulder, pronation of forearm, extension of wrist, decerebrate response)	Abnormal flexion to pain (adduction of arm, internal rotation of shoulder, pronation of forearm, flexion of wrist, decorticate response)	Flexion/Withdrawal to pain (flexion of elbow, supination of forearm, flexion of wrist when supra-orbital pressure applied, pulls part of body away when nail bed is pressed)	Localizes to pain (Purposeful movements towards painful stimuli, e.g. hand crosses mid-line and gets above clavicle when supra-orbital pressure applied)	Obeys Commands (Patient does what is asked in an appropriate manner)

Here, the Ringside Physician diagnosed Jones with only an American Academy of Neurology Grade I concussion and Jones' was tested multiple times at UAMS using the Glasgow Coma Scale, each time receiving the best possible score of 15. Thus, while the clinical observations and Jones' responses to the concussion tests indicated he had a slight concussion, the results of Jones' tests did not indicate the need for medical intervention or treatment for concussion.

Just 1 hr. 15 min. after the end of the Bout, Saline Memorial Hospital performed a CT Scan of Jones' head, which produced the following findings:

CT Brain Scan Routine w/out IV Contrast which revealed no mass or mass artifact; no intracranial hemorrhage; ventricles non-dilated; lytic lesion involving right occipital bone, etiology unknown, recommend bone scan - 2.2 cm lytic lesion right occipital bone just posterior to the mastoid air cells should be correlated to bone scan.

Also, Jones received a CT Cervical Spine Routine w/out IV Contrast which revealed normal alignment; no fracture visible; no significant degenerative change. The lytic lesion discovered by Jones' CT Brain Scan was later determined by the post-mortem autopsy to be a scar from an old ear infection and have nothing to do with Jones' death or participation in the Bout.

At 4 hr. 49 min. after the end of the Bout, Jones had multiple CT scans performed at UAMS with the following results:

CT Scan Brain Routine w/out IV Contrast – Gray-white matter differentiation is well maintained. Ventricles and sulci are w/in normal limits. Basal cisterns are well maintained. No intracranial hemorrhage or extraaxial fluid collection is identified. No evidence of acute territorial infarct, focal mass lesions, or midline shift. Visualized soft tissues and osseous structures are normal. No orbital abnormalities. Paranasal sinuses are well-aerated. Mastoid air cells are well aerated.

CT Scan Pelvis & Abdomen Routine w/out IV Contrast – Dependent atelectatic changes are seen bilaterally. No pleural fluid identified. Visualized heart is normal in size w/out evidence of pericardial effusion. Distal esophagus is unremarkable. Liver, gallbladder, pancreas, spleen and adrenal glands are grossly unremarkable. No focal contour abnormality identified. Kidneys are grossly unremarkable and demonstrate normal contour. Abdominal aorta is normal in caliber w/out evidence of atherosclerotic changes. Stomach is moderately distended w/out focal mass lesion. Large and small bowel demonstrate no evidence of obstruction. Appendix is well visualized and normal. No significant lymphadenopathy is seen w/in abdomen. Small umbilical hernia is present. W/in pelvis, bladder is collapsed and demonstrates a Foley catheter tip w/air present consistent w/catheterization. Prostate and seminal vesicles are unremarkable. No free fluid or significant lymphadenopathy is seen w/in the pelvis. Bone windows demonstrate no suspicious lytic or blastic lesions.

In short, the CT scans performed by both Saline Memorial Hospital and UAMS provided no evidence of Jones' concussed state and did not indicate the need for medical intervention or treatment due to a concussion. However, the importance of Jones' CT scans lies not in what they showed, but rather, what the CT Scans did not show.

The most important thing the CT scans did not show is a skull fracture of any type; however, the ASME's report lists the following as evidence of new injury and the primary basis for finding commotio cerebri as Jones' primary cause of death:

- (i.) Hemorrhage in right temporalis muscle;
- (ii.) Microscopic subarachnoid hemorrhage and cerebral contusions are detected in a section (Slide 6) of the parietal lobe of the right cerebral hemisphere;
- (iii.) 2.4 cm hairline fracture in the inferior/medial aspect of the right temporal bone; and
- (iv.) Hemorrhage in the soft tissues of the skull base under the fracture.

However, none of the CT scans showed any evidence of the injuries discovered by the ASME's autopsy. The Commission does not dispute the existence of the ASME's autopsy findings and in fact, the Commission fully supports and believes the ASME's findings to be true; however, in the Commission's opinion, the 2.4 cm hairline fracture of inferior/medial aspect of Jones' right temporal bone and the ASME's other findings, while of concern are merely secondary/contributory factors in Jones' death. Based upon the best medical science available to the Commission and as also confirmed by the ASME, the above four (4) conditions are not serious enough, either individually or together, to have actually caused Jones' death, especially in light of the other physiological and biochemical processes simultaneously present in Jones' body.

With respect to the Commission's downgrading of Jones' head injuries to the secondary/contributory category, the Commission relies heavily on the following in addition to the medical records, expert opinions and studies available to the Commission:

- (i) Blood Test Results just 55 minutes after the Bout ended show Jones' BUN is 19, Creatinine is 2.7; BUN/Creatinine Ratio is 7; ANION/GAP is 42; and Albumin Serum is 5.1;
- (ii) Jones' alert, oriented and responsive condition from the time of initial assessment inside the Ring all the way through his time at UAMS up until he coded, most notably even though he began to experience sinus tachycardia at 01:00 hours and progressed into ventricular tachycardia and ventricular fibrillation, all of which is not normally associated with concussion/MTBI;
- (iii) The multiple CT scans specifically showing (i) No intracranial hemorrhage or extraaxial fluid collection; (ii) No evidence of acute territorial infarct, focal mass lesions, or midline shift; and (iii) Visualized soft tissues and osseus structures are normal;
- (iv) Jones' other blood and urine test results and ECG results while at UAMS;
- (v) Observation of Jones' activity during the Bout; and
- (vi) Evidence of Jones' personal activities and training prior to entering the Ring for the Bout.

The Commission's interpretation of these findings is that while the head injuries discovered upon autopsy were indicative of and associated with a concussion, they were relegated to secondary/contributory factors by other relevant medical evidence. Jones' blood test results, behavior and CT scans do not evidence the severity required to list commotio cerebri as anything other than a secondary/contributory factor.

More specifically, while Jones' high levels of glucose,  $\text{Na}^+$ ,  $\text{K}^+$ ,  $\text{Ca}^{2+}$ , and even the non-specific CPK could (at least at one hour post-Bout) all arguably be associated with the biochemical storm unleashed by a concussion/MTBI, Jones' other test results do not support a finding that Jones' brain was the source of these biochemical markers. Instead, all of Jones' other extremely abnormal electrolyte readings, and even the non-specific CPK readings are more logically and likely related to rhabdomyolysis, dehydration, renal failure and cardiac dysrhythmia.

As further support for the Commission's position, the Commission notes BUN will only be elevated outside of normal when more than 60% of kidney cells are no longer functioning and BUN/Creatinine Ratios of less than 10 indicate intrarenal failure, neither of which would have been caused by concussion/MTBI within the short time period observed in this case. Further, the high Albumin Serum and ANION/GAP are not normally associated with concussion/MTBI. Finally, Jones' alertness combined with the negative CT scans provide no evidence Jones' concussion was severe enough to cause the electrolyte and chemical storm evidenced by Jones' blood and urine test results.

(f) **What are the Pathological & Biochemical Processes Associated with Concussion/MTBI? Did this Happen with Jones and Contribute to or Cause His Death?**

Concussion involves diffuse (as opposed to focal) brain injury, meaning the dysfunction occurs over a widespread area of the brain rather than in a particular spot. Concussion is thought to be a milder type of diffuse axonal injury because axons (the long slender part of a neuron or nerve cell that conducts and provides a pathway for electrical impulses) may be injured to a minor extent by stretching.

Animal studies in which primates were concussed have revealed damage to brain tissues such as small petechial hemorrhages and axonal injury. Of particular importance to Jones' case is that axonal damage has been found in the brains of concussed persons who died from other causes; however, inadequate blood flow to the brain as a result of other injuries has been found to have contributed to the damage observed in the brain tissue. For example, studies have been conducted on the brains of deceased NFL athletes who received multiple concussions during their careers and the results of those studies suggests there is lasting damage to the brain after experiencing one or more concussions. The NFL athlete studies also suggest such lasting damage can lead to a variety of other health issues.

Among the many biochemical and physiological processes unleashed by a concussive impact are impaired neurotransmission, loss of ion regulation, deregulation of energy use and cellular metabolism, and a reduction in cerebral blood flow. Excitatory neurotransmitters, chemicals such as glutamate that serve to stimulate nerve cells, are released in excessive amounts as the result of the injury. The resulting cellular excitation causes neurons to fire excessively. This creates an imbalance of electrolytes, specifically ions such as potassium ( $\text{K}^+$ ) and calcium ( $\text{Ca}^{2+}$ ), across the neuron's cell membrane (a process like excitotoxicity). As will be explained in greater detail later, the body requires a very specific level of the electrolytes, potassium ( $\text{K}^+$ ) and calcium ( $\text{Ca}^{2+}$ ), to be present both inside and outside its cells in order for them to properly function.

Since neuron firing involves a net influx of positively charged ions into the cell, the ionic imbalance causes cells to have a more positive membrane potential (i.e. it leads to neuronal depolarization). This depolarization in turn causes ion pumps that serve to restore resting potential within cells to work more than they normally do. The cells' increased need for energy leads the cells to require greater-than-usual amounts of glucose, which the body transforms into ATP, an important source of energy for cells. Following a concussive impact, the brain may stay in this state of hypermetabolism for days or weeks, depending on the severity of the concussion and the individual's particular levels of dehydration, glucose and electrolytes at the time of the concussion.

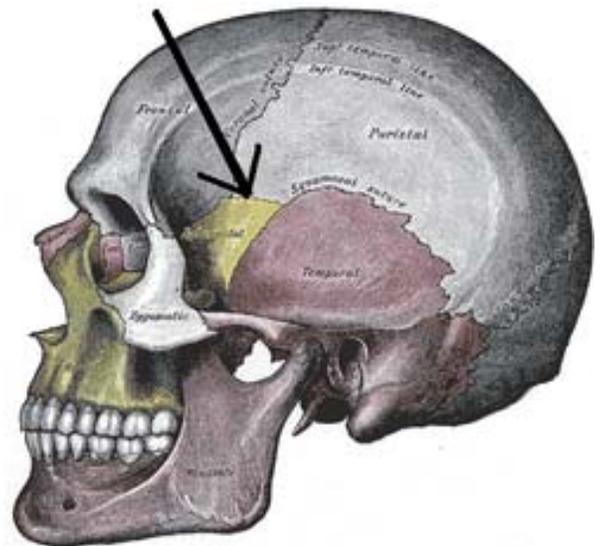
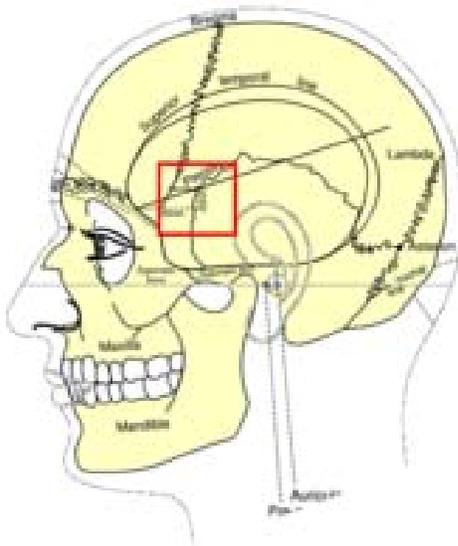
While myriad electrolyte imbalances are occurring, the cerebral blood flow is relatively reduced as a result of many other bodily functions including changed heart muscle cell firing patterns and the body's natural introduction of chemicals designed to restore the electrolyte imbalance, although the reduction in blood flow is not as severe as it is in ischemia. Due to the reduced blood circulation, the brain cells get less oxygen and glucose than normal, which in turn causes an "energy crisis," which is accompanied by excess glucose production throughout the body.

Concurrently with these processes, the activity of mitochondria may be reduced, which causes cells to rely on anaerobic metabolism, thereby increasing levels of lactate byproduct.

While Jones' blood test results evidenced all of the concussion related physiological responses described above, each of those physiological responses is also consistent with the body's physiological and biochemical response to exertional rhabdomyolysis, renal failure and cardiac dysrhythmia.

(g.) **What Areas of the Skull & Brain are Susceptible to Damage from Concussion/MTBI? Did Jones Have Damage in These Areas?**

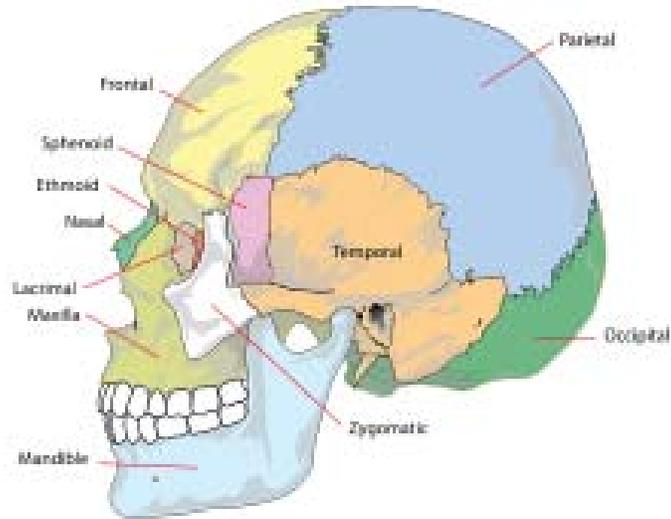
(i.) **Pterion Region of the Skull**



A particularly weak part of the skull that is vulnerable to damage causing extradural hematoma is the pterion (commonly referred to as the temple region). Deep within the pterion lays the middle meningeal artery which is easily damaged during pterion fractures. Since the pterion is so weak, pterion fractures and injury to the middle meningeal artery can easily occur and can be secondary due to trauma to other parts of the skull where the impact forces spread to the pterion. For example, in boxing, a blow to the pterion may rupture the middle meningeal artery thereby causing an epidural hematoma. Alternatively, the pterion may also be fractured indirectly by blows to the top or back of the head, which may not cause fracture at the site of impact, but may place sufficient force on the skull to fracture its weakest part, the pterion

Here, Jones' autopsy revealed his middle meningeal artery was intact with no extradural hematomas.

(ii.) **Temporal Bone & Mandible Bone Regions of the Skull**

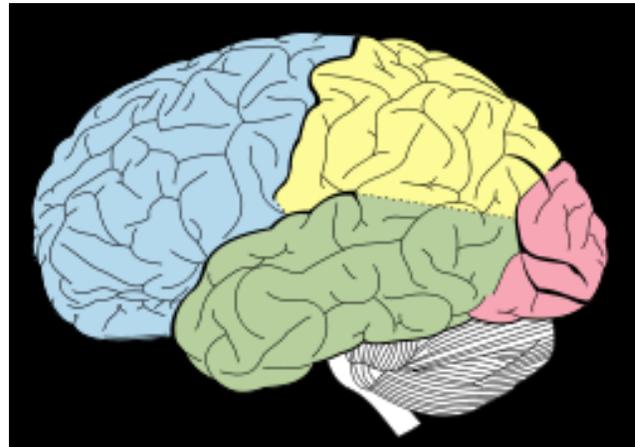
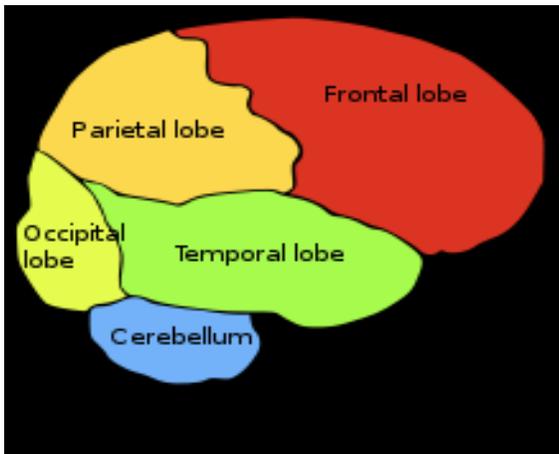


During the postmortem forensic analysis of Jones' central nervous system including his brain, Dr. Dye noted:

Hemorrhage courses throughout the right temporalis muscle. On the inferior/medial aspect of the right temporal bone is a 2.4 cm hairline fracture. Hemorrhage is present in the soft tissues of the skull base underneath the fracture.

The temporal and mandible bones can be seen in the above diagram. The 2.4 cm hairline fracture in the inferior/medial aspect of Jones' right temporal bone was determined to be caused by either an uppercut or straight right hand impacting Jones' chin. In the above diagram of the human skull, one can see where the mandible (jaw bone) joins and hinges into the rest of the skull at the temporal bone. When a punch (typically an uppercut) is delivered at a certain angle and with enough force, the mandible is driven backward and upward with the force of the impact being dispersed into the temporal bone and other surrounding bones. In this case, the inferior/medial aspect of Jones' temporal bone slightly fractured 2.4 cm while dissipating the force of one of Palmer's punches. The individual punch, which fractured Jones' temporal bone could not be definitively identified from observation of the fight or review of the video replays. The force of the punch that fractured Jones' temporal bone may or may not have been the cause of Jones' concussion.

### (iii.) Parietal Lobe Region of the Brain



Dr. Dye's autopsy of Jones also noted:

Section (Slide 6) from the right parietal lobe shows microscopic subarachnoid hemorrhage and microscopic cerebral contusions in the parenchyma. . . . Dystrophic calcifications are seen in one vessel wall. . . . No blood is in the epidural or subdural space. . . .

In the above diagram of the brain's various lobes, it can be seen that Jones' parietal lobe is in close proximity to his fractured temporal bone. The brain's parietal lobe is primarily responsible for integrating sensory information from all areas of the body and assists with our knowledge of numbers and their relation to one another and in our ability to manipulate and move objects from one place to another. The parietal lobe translates visual stimuli into spatial coordinates in relation to the body. In short, the parietal lobe allows the body to know where an object is located in relation to the rest of the body. For example, when you see an apple on the table to your left at about arm's length away from where you are standing, the parietal lobe is the primary part of your brain involved in making that determination.

Damage to the left hemisphere of the parietal lobe will result in problems in mathematics, long reading, writing and understanding symbols. Damage to the right hemisphere of the parietal lobe will result in loss of imagery, visualization of special relationships of objects and neglect of the left side space of the body (e.g. You might bump your left shoulder into the wall when you turn left around a hallway corner.). Most importantly here, the parietal lobe is the portion of the brain that would allow a person to follow the tip of a pen through a range of motion from left to right and vice versa.

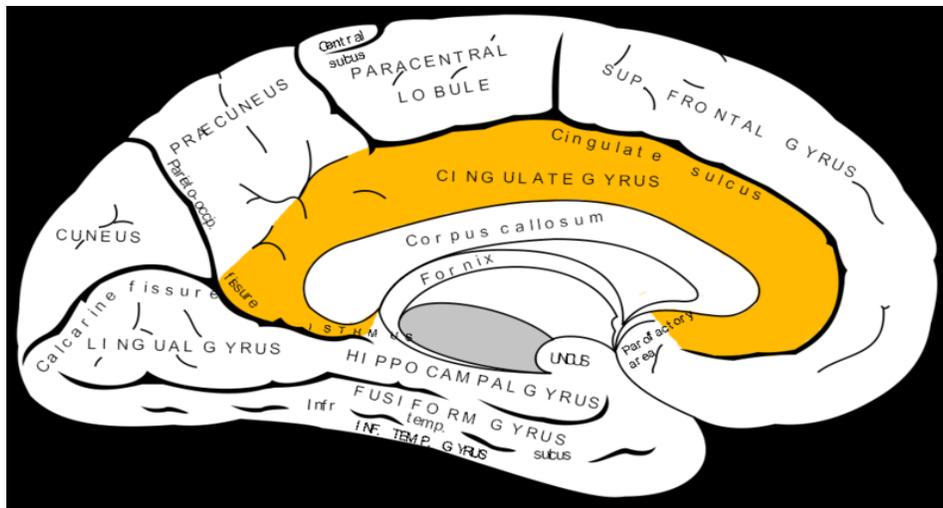
The term "parenchyma" is a latin term used to describe "the bulk of a substance." In the medical field, parenchyma is used to describe the functional parts of the body's organs as opposed to the stroma, referring to the structural tissues of the body's organs such as connective tissues. The subarachnoid hemorrhage and microscopic cerebral contusions Dr. Dye noted in Slide 6 from the parenchyma of Jones' right parietal lobe means there was some damage likely associated with this area of Jones' brain; however, this being the right hemisphere of his body, one would have expected Jones to have experienced or displayed a loss of imagery, visualization of special relationships of objects and neglect of the left side space of his body if there were any significant damage in this area. The medical records do not reflect Jones experienced any loss or impairment of functions associated with the right hemisphere of his parietal lobe. Thus, although it may sound serious to non-medically trained individuals, the observed actions and responses of Jones indicate the damage to his parietal lobe was *de minimus*.

Dystrophic calcification was observed in one of the blood vessel walls in Jones' right parietal lobe. Dystrophic calcification is the depositing of  $\text{Ca}^{2+}$  salts into a cell which occurs as a natural reaction to the cell's degeneration or death. However, a systemic imbalance causing the body's  $\text{Ca}^{2+}$  levels to rise to toxic levels can cause metastatic calcification or disseminated intravascular coagulation (precipitation of  $\text{CaPO}_4$  into normal tissues due to the kidneys' failure to filter & excrete the  $\text{PO}_4^{3-}$ ), which conditions would be indistinguishable in appearance from the cells observed during the autopsy.

Dystrophic calcification is usually localized in one injured area of the body; whereas, metastatic calcification may be found in many areas of the body. Metastatic calcification and disseminated intravascular coagulation can occur in almost any body tissue; however, they are most common in the interstitial tissues of the vasculature, kidneys, lungs, and gastric mucosa. In the kidneys, lungs and gastric mucosa, acid secretions and rapid changes in the body's pH level causes the formation of  $\text{Ca}^{2+}$  salts.  $\text{Ca}^{2+}$  salts, including  $\text{CaPO}_4$  are birefringent polarizable material.

Here, Dr. Dye's forensic analysis of the lung and kidney sections notes no birefringent polarizable material, thus indicating, but not requiring, a conclusion the calcification in the blood vessel of Jones' brain was localized dystrophic calcification rather than metastatic calcification. On the other hand, Jones' hyper-elevated  $\text{PO}_4^{3-}$  level ( $>24$ , more than 5 times the upper end of the normal range) reported in Jones blood test results at 03:04 hours and likely present, but not measured, prior to such time, when taken together with Jones' critically low  $\text{Ca}^{2+}$  level, which was markedly decreased from its high level (12.4) 4 hr. 14 min. earlier, provides significant evidence in favor of the likely occurrence of disseminated intravascular coagulation. Nevertheless, because voluminous serial sections of Jones' kidneys were not taken and reviewed in specific search for birefringent polarizable material, it is equally as likely the dystrophic calcification was localized to the damaged portion of the right parietal lobe as it is to have been the result of disseminated intravascular coagulation.

**(iv.) Tonsillar, Uncus & Cingulate Gyrus/Cortex Regions of the Brain & Brainstem**



Dr. Dye's autopsy of Jones also noted:

Brain is free of tonsillar, uncal and cingulate gyrus herniation. The vessels at the base of the brain are intact and free of dilatation.

In the diagram above, the cingulate gyrus and uncus portions of the brain can be seen. Notably, the absence of tonsillar, uncal and cingulate gyrus herniation indicates Jones' did not have any brain herniation, which is a deadly side effect of high intracranial pressure.

## **Tonsillar Herniation**

Tonsillar herniation compresses the lower brain stem and upper cervical spinal cord, which results in disruption of signals from the part of the brain controlling respiratory and cardiac functions. Although the medical records reveal Jones' experienced abnormal respiratory and cardiac function, the absence of tonsillar herniation and the results of Jones blood and urine tests, as well as ECG results, contraindicates brain injury as Jones' primary cause of death.

## **Uncal Herniation**

Uncal herniation compresses the uncus and puts pressure on the brain stem, mostly the midbrain, and the third cranial nerve, which causes the pupil of the eye on the side of the affected nerve to dilate and fail to constrict in response to light as a precursor to more substantial effects such as deviation of the eye to a "down and out" position due to a loss of innervation to all ocular muscles, except the lateral rectus and the superior oblique. Other signs of increased uncal herniation include extension of the leg, hemorrhages of the mesencephalon and pons area of the brain, and decorticate posturing, respiratory depression, slowed heart rate, lethargy and pupil dilation.

Again, Dr. Dye's autopsy revealed no hemorrhages of the mesencephalon or pons area of Jones' brain; however, at 22:22:58 hours, just 33 minutes after the end of the Bout and while en route from the Venue to Saline Memorial Hospital, one of the EMTs observed Jones showing signs of possible decorticate posturing, evidenced by tensing of both arms inward then relaxing them. At that time, the EMT also observed Jones' left eye drifting to the left while his right eye maintained a forward looking gaze. In addition, both of Jones' eyes were reactive, but sluggish and pinpoint, while his breathing slowed but remained bilaterally clear. Accordingly, it is possible Jones was experiencing some level of tonsillar, uncal or cingulate gyrus herniation; however, the fact that none of these symptoms of uncal herniation were observed at any other time, Jones' pupil dilation remained normal at all other times and the lack of hemorrhaging makes it much more likely Jones was simply experiencing transient disruptions to one or more of the tonsillar, uncal or cingulate gyrus regions as a result of the mild concussion rather than actual herniation pressure or damage.

Decorticate posturing, such as was noted by the EMT while Jones was en route to Saline Memorial Hospital, is simply abnormal posturing such as flexion or extension of the arms or legs and is usually, but not always, an indicator of some type of brain injury. Decorticate posturing occurs when one set of muscles are incapacitated while the opposing set is not and an external stimulus causes the operable set of muscles to contract.

In contact sports, decorticate posturing is most often seen as what is called the "fencing response." The fencing response is the response of the arms, following a concussion, where the person assumes the "en garde" position that initiates a fencing bout while laying on the ground (i.e. The person is laying on the ground with one arm stretched straight out at approximately 90 degrees from their body and the other arm is experiencing flexion (bending) while remaining at their side or just slightly raised to about the height of their hip.). Observation of the fencing response can be used by medical personnel as an indicator of the substantiality of the brain injury and localization of the injury to the midbrain most likely extreme stretching of the cerebellar peduncles.

Very good examples of decorticate posturing with the fencing response can be seen in the following videos:

- (i) Austin Collie during a National Football League game between the Eagles and Colts found at <http://www.youtube.com/watch?v=kufy7Q1FVSA> ;
- (ii.) Tie Domi's knockout of Ulf Samuelsson during a hockey match at approximately forty-five (45) seconds of <http://www.youtube.com/watch?v=VnKxV9ilsqo> ;

- (iii.) Arthur Abraham's 12<sup>th</sup> round knock out of Jermain Taylor at approximately one minute ten seconds (1:10) of <http://www.youtube.com/watch?v=j3XwWHwLwsI&feature=related> or two minutes fifty-five seconds (2:55) of <http://www.youtube.com/watch?v=ErNredfvwu0>

### **Cingulate Gyrus Herniation**

The cingulate gyrus region of the brain is an integral part of the body's limbic system and is involved in the formation and processing of emotions, as well as, learning, memory, respiratory control and the brain's executive functions (i.e. the collection of processes involved in planning, cognitive flexibility, abstract thought, rule acquisition, initiating situation appropriate actions, inhibiting situation inappropriate actions and selecting relevant sensory information).

Cingulate gyrus herniation is the most common type of brain herniation and does not put as much pressure on the brainstem, but does interfere with blood vessels in close proximity to the injured portion of the brain. Persons with cingulate gyrus herniation can present with abnormal posturing. Cingulate herniation is believed to be a precursor to other types of brain herniation.

The absence of tonsillar, uncal and cingulate gyrus herniation observed during Jones' autopsy is associated with the *de minimus* nature of the subarachnoid hemorrhage and cerebral contusions and a possibility that the same were the result of other biochemical abnormalities present in Jones' body.

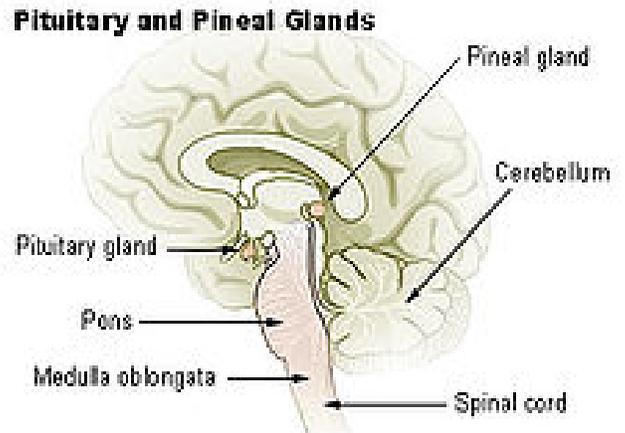
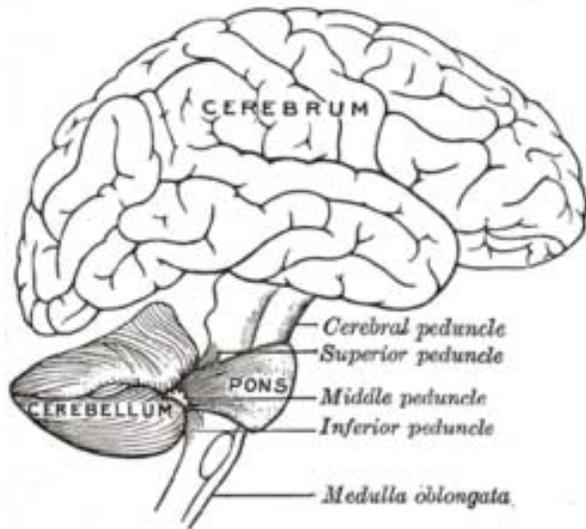
Dr. Dye's autopsy of Jones also noted:

The vessels at the base of the brain are intact and free of dilatation. The cerebral hemispheres, midbrain, pons, cerebellum and medulla [oblongata] are free of cystic scar, hemorrhage, and mass. . . . The ventricles are not enlarged and contain no blood. . . . The hippocampal nuclei are free of histopathic change. The vessels are not inflamed. Lytic lesion on skull, sections show cholesteatoma [evidence of previous, unrelated ear infection].

Although Dr. Dye's autopsy revealed Jones' cerebral hemispheres, midbrain, pons, cerebellum and medulla oblongata were free of cystic scar, hemorrhage and mass and multiple reviews of the Bout's video confirm Jones did not receive any blows to the top or back of the head, it is still very likely Jones' suffered some temporary dysfunction in one or more of the described areas as a result of either one or more of the punches he received or hitting his head on the floor of the Ring when he fell at the end of the 2<sup>nd</sup> Round. The Commission's determination of temporary dysfunction in the described regions of Jones' brain is supported by comparing Jones' actions and responses observed at Ringside, actions and responses contained in the medical reports and actions and responses contained in the timeline at the beginning of this report with the known functions of the various described regions of the brain.

Although appearing normal during Jones' autopsy, many of the following areas of the brain are affected in some way during a concussion or mild traumatic brain injury, thus the reason concussions are described as a "diffuse/widespread" MTBI rather than a "localized" injury.

(v.) **Midbrain/Mesencephalon Region of the Brain**



The midbrain or mesencephalon is the part of the central nervous system associated with vision, hearing, motor control, sleep/wake cycles, arousal/alertness, and regulation of the body's temperature. The midbrain is considered to be part of the brainstem and is responsible for, among other things, the production of dopamine, a neurotransmitter that increases heart rate and blood pressure and plays an important role in processing pain signals, cognition, voluntary movement, motivation, punishment/reward prediction and acknowledgment, sleep, mood, attention, working memory and learning.

Detection of possible injury to the midbrain or mesencephalon region of the brain is why vision and hearing tests are given post Bout and why healthcare providers advise frequently waking from sleep persons recently subjected to a concussive impact. In addition to being free of evidence of damage during the autopsy, Jones did not display any characteristics associated with damage the midbrain or mesencephalon regions of his brain.

(vi.) **Pons Region of the Brain**

The pons is also considered part of the brain stem and contains nuclei responsible for passing signals from the forebrain to the cerebellum and nuclei associated with sleep, respiration, swallowing, bladder control, hearing, equilibrium, taste eye movement, facial expressions, facial sensation and posture. The pons contains the nucleus responsible for the change from inhaling to exhaling; as well as, the portion of the brain responsible for sleep paralysis and dreams in REM sleep.

In addition to being free of evidence of damage during the autopsy, Jones did not display any characteristics associated with damage the pons region of his brain.

(vii.) **Medulla Oblongata Region of the Brain/Brainstem**

While almost all portions of the brain are critically important, the medulla oblongata is particularly important and is perhaps the most critical part of the brain. The medulla oblongata is the lower half of the brainstem which contains cardiac, respiratory and vasomotor control mechanisms affecting autonomic, involuntary functions such as breathing, heartbeat, blood pressure and to a large extent vomiting, coughing, sneezing and swallowing. The medulla oblongata also relays signals from the rest of the brain to the spinal cord.

Because of its location and the critical importance of the autonomic mechanisms it controls, great care is taken to protect a Contestant's medulla oblongata by absolutely prohibiting punches or strikes to any portion of a Contestant's head located behind the ear. Even light punches to the back of the head during a clinch, sometimes called "rabbit punches," can result in disqualification of a Contestant.

Although appearing to be undamaged during the autopsy, Jones' cardiac, respiratory and blood pressure abnormalities, when taken out of context without the additional information provided by Jones' medical records and the Bout's video, could lead one to believe Jones suffered from injuries to the medulla oblongata region of his brain. However, when all of the evidence is considered including, without limitation, Jones' blood test results, urine test results, multiple reviews of the Bout's video in which it is shown Jones' received no punches to the back of his head, and Jones' actions and inactions after the Bout, there is very little doubt Jones did not suffer any damage to his medulla oblongata; but instead, Jones' cardiac dysrhythmias, low blood pressure, and rapid heartbeat and respiration were all the result of hyperkalemia induced by exertional rhabdomyolysis.

### **(viii.) Cerebellum/Flocculonodular Lobe Region of the Brain**

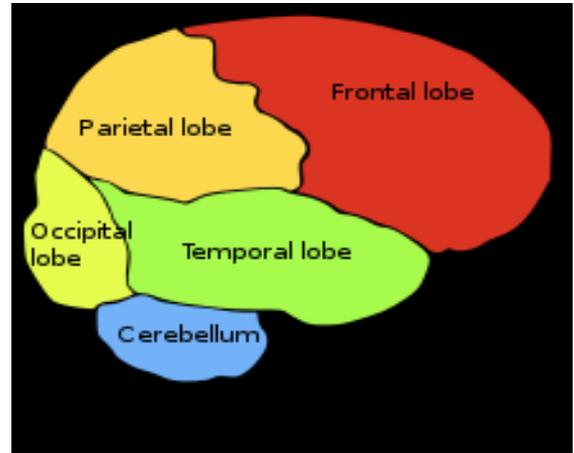
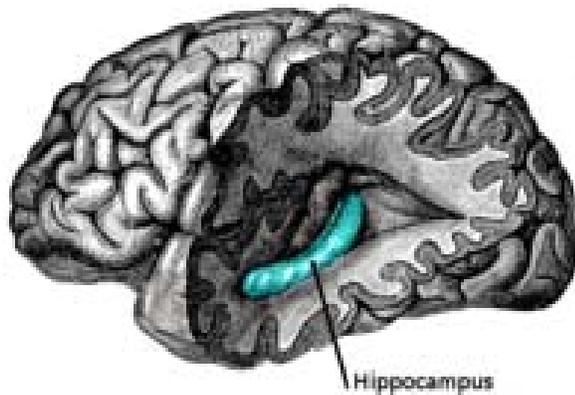
The cerebellum is a part of the bottom of the brain which plays a critical role in finely tuned motor control, cognitive functions, attention, language and some emotional responses such as fear and pleasure. The cerebellum does not initiate movement, but plays a role in coordination of movement including precision and timing. Damage to the cerebellum does not cause paralysis, but rather produces complications with fine movement, equilibrium, posture and motor learning. Among the standard tests for cerebellum injury is to reach with the tip of the finger for a target at arm's length from the body: A healthy person will move the fingertip in a rapid straight line toward the object; however, a person with damage to the cerebellum will reach for the object in a more slow and erratic manner with many mid-course corrections.

Because the cerebellum only processes information received from other parts of the central nervous system and does not otherwise generate any information of its own, the cerebellum functions very much like the central processing units in an extremely high speed supercomputer. The highly specialized and unitary function of the cerebellum greatly increases the human brain's processing speed and allows it to produce very quick, clear responses to hundreds of millions of simultaneous inputs being sent by other parts of the central nervous system.

Damage to the flocculonodular lobe of the cerebellum may present as loss of equilibrium most notably, an abnormal walking gait with a wide stance that indicates difficulty in balancing. Damage to the midline portion may disrupt movements of the entire body and damage specifically toward the lateral region of the cerebellum disrupts fine movements of the hands or limbs. Damage in the upper cerebellum also causes an irregular gait and other leg coordination issues; while, damage to the lower cerebellum causes uncoordinated and poorly aimed movement of the arms and hands with difficulty in regulating the speed of such movements.

Although appearing to be undamaged during the autopsy, Jones' temporary disorientation during the few minutes immediately following the Bout and Jones' inability to feel or move his legs at 00:25 hours and 01:40 hours provides inconclusive evidence of possible dysfunction in Jones' cerebellum or the lateral portion of the flocculonodular region of his brain. The inconclusiveness of these facts comes from the possibility that Jones' disorientation and inability to move his legs is also indicative of injuries to other portions of his central nervous system or decreased blood flow to the brain and outer limbs resulting from Jones' cardiac dysrhythmias and low blood pressure, which also presented at the same time.

(ix.) **Temporal Lobe Region of the Brain**



Temporal lobe of the brain is involved in auditory perception (hearing) with the primary auditory cortex being located within its structure. The superior, posterior and lateral parts of the temporal lobes are responsible for high-level processing of sound stimuli including the understanding speech, for which the left temporal lobe is particularly specialized. The left temporal lobe perceives not only low level sounds, but also extends to comprehension, naming, verbal memory and other language related functions.

The ventral temporal cortices of the temporal lobe are involved in visual processing of complex optical stimuli such as a person's face and scenery. The medial temporal cortices are responsible for declarative memory, for example memories which can be consciously recalled such as facts and events, as opposed to procedural memory, which is memory of skills like riding a bike. Declarative memory is further divided into two categories, episodic memory (i.e. the storing of personal experiences) and semantic memory (i.e. the storing of factual information). Damage to the medial temporal cortices results in anterograde amnesia, which is the loss of the ability to form new memories reflected by the person's inability to recall the recent past while still being able to recall memories from before the damaging event.

(x.) **Hippocampus Region of the Brain**

Inside the medial temporal lobe beneath the cortical surface is the hippocampus, which is the portion of the brain responsible for consolidating information from short term memory and placing it into long term memory, as well as, playing a major role in spatial navigation, which allows a person to know where they are in relation to their environment (such as knowing you are home, knowing you are at work, knowing when you are getting close to your destination and finding shortcuts and new routes between familiar places) and which direction the person is looking.

(h.) **What does the Commission do to Test Contestant's Brain, Neurological & Central Nervous System Functions Prior to and After Bouts?**

Due to their location and the twisting force on the head and neck when a Contestant receives a hard punch or kick or slams the back of their head on the floor of the Ring, the proper functioning of the midbrain or mesencephalon, pons and cerebellum (especially the flocculonodular lobe of the cerebellum) are often disrupted during and after a Combative Sports Event. Fortunately, these areas of the brain are highly resilient and often stabilize on their own shortly after slight disruptions in their function.

Question number 7 on the Commission's Combative Sports Medical Report (Pre-Bout) – Form CSMR057-2010 and question number 4 on the Commission's Combative Sports Medical Report (Post-Bout) – Form CSMR057-2010 are specifically designed to test and assess damage or dysfunction in all of the aforementioned areas of the brain and other areas of the central nervous systems, with a particular emphasis on the cerebellum and its flocculonodular lobe. Jones displayed only slight disruption of the tested areas while being observed and tested inside the Ring. All disruption of Jones' tested areas and the displayed dysfunction associated with such disruption had resolved itself by the time Jones arrived at UAMS, approximately 2 hours after the end of the Bout.

(i) **What Can Be Done to Prevent Concussions & Lessen the Effects of Concussion?**

There is currently no scientifically valid way to absolutely prevent concussions, save avoidance of all activities with any risk of concussive impacts to the head, neck and body. Since people are exposed to risks of receiving a concussion from almost anything, such as tripping over their own feet getting out of bed, slipping on the floor in their kitchen or being hit by an apple falling out of an apple tree, the best thing we can do is be prepared for the inevitable concussion and take precautions to lessen both the likelihood of occurrence and severity of its effects when it does occur.

(i) **B.E.H.G. – Balanced Electrolytes, Hydration & Glucose**

As previously discussed the likelihood of suffering a concussion and the severity of the concussion have been linked to the person's level of dehydration, glucose level and electrolyte imbalance at the time of impact. Accordingly, it is very important for athletes, as well as non-athletes, to remember the following:

- 1.) **Balanced Electrolytes** – Eat a well balanced diet designed to replenish the electrolytes lost during each day's activities. Ensure your diet contains a proper balance of the electrolytes potassium ( $K^+$ ), sodium ( $Na^+$ ), calcium ( $Ca^{2+}$ ), magnesium ( $Mg^{2+}$ ), chloride ( $Cl^-$ ), hydrogen phosphate ( $HPO_4^{2-}$ ), and hydrogen carbonate ( $HCO_3^-$ ) and that you do not consume too much of any one particular electrolyte, since many electrolytes are used as offsets or to counteract the electrical conductivity of others at the cellular level. At this time the Commission does not endorse or recommend athletes consume any type of electrolyte supplement without first having the requisite tests performed in consultation with their personal physician to establish whether their body or physical activities require any such supplementation.
- 2.) **Hydration** – Ensure that you stay well hydrated with plain water at all times including, before, during and after any physical activity.
- 3.) **Glucose** – Eat a balanced diet designed to provide the energy necessary for daily activities and modifying the diet to moderately increase glucose intake in the hours prior to upcoming events which have an increased risk of sustaining a concussive impact. In particular, unless it would not be recommended due to other medical conditions or individual circumstances, moderately (NOT EXCESSIVELY) increasing glucose intake (in the form of almost any type of fruit or candy bar) within an hour prior to the start of any activity in which there is an increased risk of sustaining a concussive impact. At this time the Commission does not endorse or recommend athletes make extensive modifications of their diet or consume any type of glucose or other nutritional supplement without first having the requisite tests performed in consultation with their personal physician to establish whether their body or physical activities require any dietary change or nutritional supplementation.

As can be seen by the description of Jones' pre-Bout activities provided later in this report, supplemental intake of electrolytes in addition to a well balanced diet can dangerously alter the balance of critically important electrolytes to such an extent that the body's systems cannot correct the imbalance, especially when simultaneously having to compensate and adjust its own physiological responses to one or more injuries sustained during daily activities or competition.

(ii.) **Omega-3 Docosahexaenoic Acid (DHA)**

Despite the foregoing warnings regarding the intake of dietary supplements, recent clinical and laboratory research by neurosurgeon Julian Bailes, M.D., and his colleagues from West Virginia University, has shown dietary supplementation with omega-3 docosahexaenoic acid (DHA) (*all-cis-docosa-4,7,10,13,16,19-hexa-enoic acid*; shorthand name is 22:6(n-3)) offers protection against the biochemical brain damage which occurs after a traumatic brain injury. (Mills JD, Bailes JE, Sedney CL, *et al.* (JAN. 2011). "Omega-3 fatty acid supplementation and reduction of traumatic axonal injury in a rodent head injury model." **Journal of Neurosurgery**. 114(1): 77–84.)

In Dr. Bailes' studies, rats who received the highest dose of DHA supplementation prior to traumatic brain injury experienced the least amount of tissue damage, by measurement of APP and caspase-3 markers as compared with rats given lower levels and no DHA. Dr. Bailes group wrote, "Omega-3 fatty acid supplementation significantly reduces the number of APP-positive axons [brain-cell connectors] at 30 days post-injury to levels similar to those in uninjured animals." (Bailes JE, Mills JD. (SEPT. 2010). "Docosahexaenoic acid reduces traumatic axonal injury in a rodent head injury model." **Journal of Neurotrauma**. 27(9): 1617–1624.)

"The potential for DHA to provide prophylactic benefit to the brain against traumatic injury appears promising and requires further investigation. The essential concept of daily dietary supplementation with DHA, so that those at significant risk may be preloaded to provide protection against the acute effects of TBI, has tremendous public health implications." (Mills JD, Hadley K, Bailes JE. (FEB. 2011). "Dietary supplementation with the omega-3 fatty acid docosahexaenoic acid in traumatic brain injury." **Neurosurgery**. 68:474-481.)

According to the study, which involved groups of adult male rats, which received daily DHA doses of 0, 3, 12, or 40 mg per kilo of body weight for 30 days prior to a traumatic brain injury, "Dietary supplementation with DHA resulted in increased serum DHA levels proportionate with the escalating dosage. Using a selective measuring technique, only the highest dosage group, 40 mg/kg, showed significantly ( $p < 0.05$ ) decreased numbers of APP positive axons, at 1.15 axons per high power field versus 6.78 in unsupplemented animals. CD-68, caspase-3, and water maze testing all were significantly ( $p < 0.05$ ) improved in the high dose group." (*Id.*)

The study's results showed that the animals receiving the highest dose of omega-3 DHA had significantly reduced brain damage, compared to the other animals. Specifically, levels of APP protein—a key marker for brain damage—were about six times lower in the high-DHA-dose group, compared with the animals that got no DHA. The high-DHA-dose animals also showed decreases in two major measures of brain cell death: a chemical called caspase 3 and macrophage-type immune cells. The rats receiving ample doses of DHA also suffered less behavior impairment, as measured by their performance in a water maze test.

DHA is the most abundant omega-3 fatty acid in the brain and retina and comprises nearly 40% of the polyunsaturated fatty acids in the brain and nearly 60% of those in the retina. DHA comprises nearly 50% of the weight of a neuron's plasma membrane. (Meharban, Singh (MAR. 2005). "Essential fatty acids, DHA and human brain." **Indian Journal of Pediatrics**. 72:239-242.)

Cold water oceanic fish oils are rich in DHA. However, not all Omega 3 fatty acids are the same and it is very important to note that the only form of Omega 3 fatty acids involved in the West Virginia University studies

was in DHA form. Many of fish oil supplements on the market today contain a different form of omega-3 fatty acid, namely eicosapentaenoic acid (“EPA”), and only limited quantities of DHA.

DHA is normally present in the human brain, while EPA is not; however, one study of pure DHA supplementation on children with attention deficit hyperactivity disorder showed no behavioral improvements, while another study found supplementation with both EPA and DHA improved behavior. (Vedin I, et al. (JUN. 2008). “Effects of docosahexaenoic acid–rich n–3 fatty acid supplementation on cytokine release.” *American Journal of Clinical Nutrition*. 87(6):1616–1622; *See also*, Voigt RG, Llorente AM, Jensen CL, et al. (AUG 2001). “A randomized, double-blind, placebo-controlled trial of docosahexaenoic acid supplementation in children with attention deficit/hyperactivity disorder.” *Journal of Pediatrics*. 139(2): 173, 174.)

Additional studies are necessary to continue investigating the potentially helpful and harmful effects of humans increasing their intake of DHA; accordingly, the Commission neither recommends nor discourages the use of DHA as a prophylactic measure to lessen the likelihood or severity of a concussion. The Commission encourages everyone to consult with their own personal physician and follow their physician’s advice before starting or continuing any dietary supplementation program. The Commission does recommend everyone, including athletes, consume plenty of water, avoid unhealthy weight loss programs and eat a well-balanced diet in conjunction with an exercise program suitable to their current physical condition and planned competitive activities.

## **2. The Commission’s Primary Cause of Death – Cardiac Arrest Due to Cardiac Dysrhythmia – Specifically Left Ventricular Fibrillation Onset by Hyperkalemia**

This section of the report is unavoidably complex as a result of the multifaceted biomechanical processes required for the proper function of the human heart. The following in-depth information on cardiac functions is provided by the Commission for the dual purpose of providing insight into Jones’ death and providing an educational resource for interested Contestants, Cornermen/Seconds/Trainers, Officials, physicians and researchers who may want to use Jones’ case to advance their knowledge and understanding of the importance of and relationship between a Contestant’s blood pressure, hydration and electrolyte balance (namely, potassium (K<sup>+</sup>), sodium (Na<sup>+</sup>), and calcium (Ca<sup>2+</sup>)) at all phases (before, during and after) of sports training and competition.

This section of the report also provides an explanation of why and how hyper-elevated potassium (K<sup>+</sup>) levels caused Jones’ heart and kidneys to enter into a lethal feedback loop whereby Jones’ heart was not sending enough blood to his kidneys because hyper-elevated potassium (K<sup>+</sup>) levels and other electrolyte imbalances were affecting his heart muscle cells’ ability to properly contract and relax. In turn, Jones’ kidneys were not sufficiently filtering and regulating the K<sup>+</sup> and Na<sup>+</sup> levels because of the decreased blood flow, associated nephronic damage and nephrons which had become clogged and damaged.

While Jones’ documented, dietary and supplemental intake of K<sup>+</sup> was definitely excessive, it was not sufficient to cause his cardiac arrest and kidney failure; instead, the most likely source of Jones’ lethal potassium (K<sup>+</sup>) level is revealed in this report’s later discussion of exertional rhabdomyolysis and Jones’ other pre-Bout activities.

### **(a.) What Is Hyperkalemia and What Are Its Effects/Symptoms?**

The electrolytes, sodium (Na<sup>+</sup>) and potassium (K<sup>+</sup>), are both essential mineral micronutrient in human nutrition. Sodium (Na<sup>+</sup>) makes up most of the cations (positive ions) of blood plasma (extracellular fluid) at a reference range of 145 milliequivalents per liter (3,345 mg/L). On the other hand, potassium (K<sup>+</sup>) is the major

cation (positive ion) inside cells (intracellular). Thus, both sodium ( $\text{Na}^+$ ) and potassium ( $\text{K}^+$ ) play very important roles in maintaining the body's fluid and electrolyte balance.

Hyperkalemia is the term used to describe an excessive level of potassium ( $\text{K}^+$ ) in a person's blood. Symptoms of hyperkalemia are fairly nonspecific and generally include malaise, palpitations and muscle weakness; mild hyperventilation may indicate a compensatory response to metabolic acidosis, which is one possible cause of hyperkalemia.

The symptoms of hyperkalemia are fairly nonspecific and generally include malaise, palpitations and muscle weakness; mild hyperventilation may indicate a compensatory response to metabolic acidosis, which is one of the possible causes of hyperkalemia. Often, however, hyperkalemia is detected while screening blood tests for other medical disorders, or only comes to medical provider's attention after the patient develops complications, such as cardiac arrhythmia or sudden death.

**(b.) What Are Normal Levels for the Electrolytes, Potassium ( $\text{K}^+$ ) and Sodium ( $\text{Na}^+$ )?**

According to the guidelines established by the United States National Academies of Sciences Institute of Medicine, the recommended daily intake of potassium ( $\text{K}^+$ ) is 100 mEq (3,909 mg). **2004 Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate**, pg. 187, The National Academies Press (2004). Surveys reveal the median potassium ( $\text{K}^+$ ) intake of adults in the United States is 72 to 84 mEq (2,815 to 3,284 mg) with median intake amongst the African American population typically averaging substantially lower than the overall median intake. While athletes may need more  $\text{K}^+$  than non-athletes, the amount of extra  $\text{K}^+$  necessary to offset the effects of strenuous training and exercise is not much. In fact, according to J. Anderson, a nutrition specialist and professor at Colorado State University, one cup of orange juice, a banana or a potato is sufficient to replace all of the  $\text{K}^+$  lost during one (1) to two (2) hours of hard exercise. <http://www.ext.colostate.edu/pubs/foodnut/09355.html>.

According to the University of Tennessee's Health Science Center Department of Nephrology, a normal adult's total body potassium ( $\text{K}^+$ ) is approximately 50 mEq per kilogram of body weight which is equal to 3,500 mEq (136,844 mg) in a 70 kg person. Ninety-eight percent (98%) of the total body potassium ( $\text{K}^+$ ) is stored in the body's intracellular compartments (inside the cells rather than in the blood, interstitial fluid & bone).

Fifty-seven percent (57%) of the total normal adult body weight is liquid, although obesity decreases the percentage to as low as forty-five percent (45%). Guyton, Arthur C. (1976). **Textbook of Medical Physiology (5<sup>th</sup> Ed.)**. Philadelphia: W.B. Saunders. pg. 424. Sixty-two and one-half percent (62.5%) of the body's total liquid is considered intracellular, while thirty-seven and one-half percent (37.5%) of the body's liquid is extracellular. Guyton, Arthur C. (1991). **Textbook of Medical Physiology (8<sup>th</sup> Ed.)**. Philadelphia: W.B. Saunders. pg. 275. The body's extracellular fluid can be further categorized as follows: (i.) Plasma contains seven and one-half percent (7.5%) of the total body liquid; (ii.) Interstitial fluid contains thirty percent (30%) of the total body liquid; and (iii.) Transcellular fluid contains a negligible amount of the total body fluid and is usually ignored in calculations. *Id.* See also, John T. Hansen, Bruce M. Koeppen, (2002). **Netter's Atlas of Human Physiology**. Teterboro, N.J.: Icon Learning Systems.

Jones, who weighed 232.5 lbs. (105 kg), was not obese, rather he had a very muscular body; therefore, under normal circumstances, it is assumed that prior to the Bout Jones' body had the potassium ( $\text{K}^+$ ) and liquid levels indicated in the following table:

**PHYSIOLOGY OF POTASSIUM (K<sup>+</sup>) BALANCE: BODY'S DISTRIBUTION OF LIQUIDS & POTASSIUM**

**(Jones' Normal Range Based on His Actual Weight Is Included in Bold Font)**

(Average Person Total – 3,500 mEq/136,844 mg)

**(Jones' Normal Total Potassium (K<sup>+</sup>) – 5,250 mEq/205,266 mg)**

**(Jones' Normal Total Body Fluid – 59.85 Liters)**

<u>Extra-Cellular Fluid</u>		<u>Intra-Cellular Fluid</u>	
(Average Person Potassium (K <sup>+</sup> ) – 70 mEq/2,737 mg – 2%) <b>(Jones' Normal Potassium (K<sup>+</sup>) – 105 mEq/ mg – 2%)</b> <b>(Jones Normal Fluid – 22.444 Liters)</b>		(Average Person Potassium (K <sup>+</sup> ) – 3,430 mEq/134,107 mg – 98%) <b>(Jones' Normal Potassium (K<sup>+</sup>) – 5,145 mEq/201,161 mg – 98%)</b> <b>(Jones Normal Fluid – 37.406 Liters)</b>	
<b>Plasma</b>	14 mEq/547 mg (0.4%) <b>Jones – 21 mEq/821 mg</b> <b>Jones – 4.489 Liters</b>	<b>Muscle</b>	2,660 mEq/104,001 mg (76%) <b>Jones – 3,990 mEq/156,002 mg</b>
<b>Interstitial Fluid</b> (Fluid Surrounding Tissue Cells)	56 mEq/2,190 mg (1.6%) <b>Jones – 84 mEq/3,284 mg</b> <b>Jones – 17.955 Liters</b>	<b>Liver</b>	245 mEq/9,579 mg (7%) <b>Jones – 367.5 mEq/14,369 mg</b>
<b>Transcellular Fluid</b>	Nominal/Disregarded	<b>Erythrocytes</b> (Red Blood Cells Which Deliver Oxygen to the Body's Tissues & Also Rich in Hemoglobin)	245 mEq/9,579 mg (7%) <b>Jones – 368 mEq/14,369 mg</b>
<b>Normal Serum K<sup>+</sup></b>	3.5 – 5.0 mEq/L 137 – 195 mg/L <b>Jones' Projected – 4.678 mEq/L</b>	<b>Bone</b>	280 mEq/10,948 mg (8%) <b>Jones – 420 mEq/16,421 mg</b>
		<b>Normal Intracellular K<sup>+</sup></b>	150 mEq/L – 5,865 mg/L

**(c) What Processes Does the Body Use to Regulate Potassium (K<sup>+</sup>) and Sodium (Na<sup>+</sup>) Levels?**

Although only 2% of total body potassium (K<sup>+</sup>) remains in the extracellular compartment, the extracellular potassium (K<sup>+</sup>) concentration plays a critical role in maintaining cell membrane resting potential (difference in voltage between the interior and exterior of a cell while at rest or not “firing”). Regulation of the cells’ resting potential is critically important for electrically excitable cells such as those involved in muscle and nervous system activity. The Nernst equation, which can be used to calculate resting membrane potential, shows that resting membrane potential is a function of the ratio of extracellular over intracellular potassium (K<sup>+</sup>) concentrations. Alteration of resting membrane potential alters the ability of electrically excitable cells like muscle cells and nerve cells to properly function. Since only small quantities of potassium (K<sup>+</sup>) are normally present in extracellular fluid, the addition or subtraction of small amount of potassium (K<sup>+</sup>) in this space significantly alters the extracellular potassium (K<sup>+</sup>) concentration thereby having a very substantial and adverse effect on muscle and nerve cell function. For this reason, the body must keep the extracellular potassium (K<sup>+</sup>) concentration within a very narrow range at all times.

The body must maintain its extracellular potassium ( $K^+$ ) concentration within a very narrow range of only 3.5 – 5.0 mEq (137 – 195 mg) per liter of fluid at all times despite daily fluctuations in dietary potassium ( $K^+$ ) intake. The process of regulating its internal environment to maintain stable, constant extracellular potassium ( $K^+$ ) concentrations is called homeostasis.

Potassium ( $K^+$ ) balance includes two major features: i.) Internal balance, which refers to getting potassium ( $K^+$ ) into the intracellular space, and ii) External balance, which involves matching daily intake of potassium ( $K^+$ ) to the amount excreted into the urine. External balance is a relatively slow process, such that the daily intake is excreted by the kidneys over an approximately twenty (24) hour time frame. In contrast, since humans typically ingest potassium ( $K^+$ ) through their meals in a bolus manner (meaning a large amount all at once), there is significant abrupt potassium ( $K^+$ ) entry into the body. If the potassium ( $K^+$ ) ingested through meals were allowed to remain in the extracellular space, hyperkalemia and its adverse effects on neuromuscular function would occur within an hour or so following a large meal. Thus, the body utilizes the much faster internal balance mechanism to move potassium ( $K^+$ ) into the intracellular space and prevent meal related hyperkalemia.

**(i) Regulation Via Intra-Cellular Storage/Release**

The body's primary mechanism for regulation of extracellular potassium ( $K^+$ ) concentrations is through intracellular storage of  $K^+$ . Intracellular storage of potassium ( $K^+$ ) is also the body's fastest method of reduction or release of potassium ( $K^+$ ) and is thus the method used for hour to hour regulation of extracellular potassium ( $K^+$ ) levels and regulation of potassium ( $K^+$ ) following food intake.

Intracellular potassium ( $K^+$ ) regulation is primarily achieved via an electrochemical process initiated by insulin as follows. Food ingestion, which includes potassium, leads to insulin release. When insulin binds to a cell's insulin receptor it activates the cell's  $Na^+/K^+$ -ATPase pumps which results in hyperpolarization of the cell membrane facilitation of potassium ( $K^+$ ) uptake by the cell (movement of potassium ( $K^+$ ) into the cell). Catecholamines stimulate cellular  $K^+$  uptake via the  $\beta_2$  adrenergic receptor ( $\beta_2R$ ). The generation of cyclic adenosine monophosphate (3'-5' cAMP) activates  $Na^+/K^+$ -ATPase pumps, causing an influx of potassium in exchange for sodium.

Other factors involved in internal balance include:

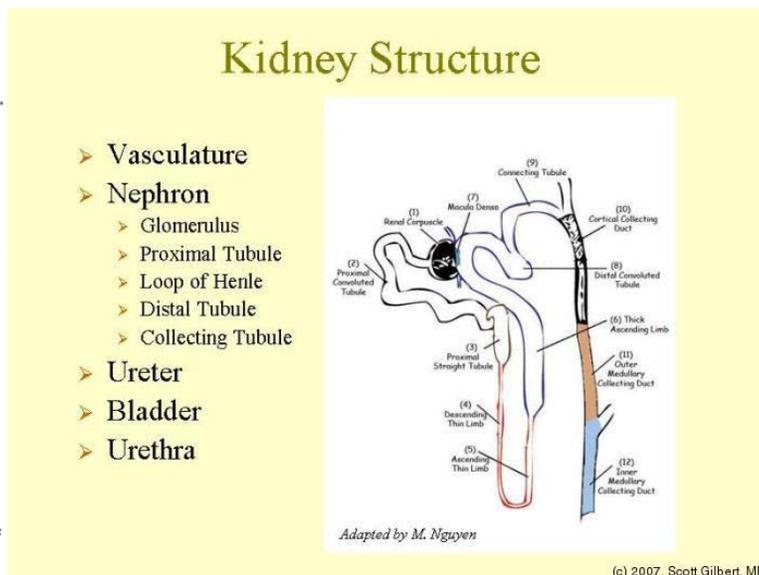
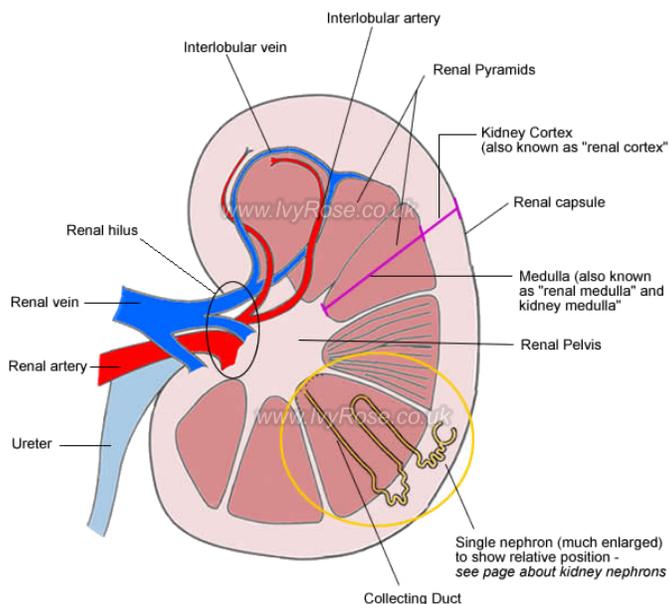
- Osmolality: increased osmolality in extracellular space leads to water movement from the intracellular compartment with potassium ( $K^+$ ) exiting the cell through a solvent drag effect.
- pH changes: metabolic acidosis with normal plasma anion gap results in hydrogen ions entering intracellular compartment for buffering by intracellular buffers, in exchange for potassium ( $K^+$ ) exiting from the intracellular compartment. This typically is a relatively small effect, which changes extracellular potassium concentration < 1 mEq (39 mg) per liter. Metabolic acidosis with increased plasma anion gap has minimal effects on internal potassium balance. In these disorders, hydrogen ions enter the intracellular compartment for buffering accompanied by the acid anion, such that no hydrogen ion potassium ion exchange occurs. Respiratory acid base disorders have trivial effects on internal potassium balance.
- Aldosterone has a modest effect to promote potassium entry into the cell.
- Beta 1 receptor activation promotes potassium entry to the intracellular space.

The body's  $K^+$  and  $Na^+$  levels are maintained by the enzyme Sodium-Potassium Adenosine Triphosphatase, also known as  $Na^+/K^+$ -ATPase or commonly referred to as the  $Na^+$ - $K^+$  Pump or sodium-potassium pump, found in the electronegative transmembrane ATPase. Increased extracellular  $K^+$  levels result in depolarization of the cell's membrane potential. This depolarization opens some voltage-gated  $Na^+$  channels, but not enough to generate an action potential (the firing or contraction of the cell), which means that after a short while, the open  $Na^+$  channels inactivate and become refractory, increasing the threshold needed to generate an action potential (i.e. making it harder for the body's cells to fire or contract). The increased cellular action potentials, caused by extracellular  $K^+$  levels, leads to impairment of the body's neuromuscular, cardiac, and gastrointestinal organ systems.

Of most concern and relevance in Jones' case is hyper-elevated extracellular  $K^+$  levels causing the impairment of cardiac conduction, which resulted in the ventricular fibrillation or asystole conditions experienced by Jones. To explain what happened to Jones in a more simply way, Jones' body, along with help from the UAMS medical teams' administration of insulin and other medicines, was trying its best to utilize natural processes to move the extracellular  $K^+$  back inside his cell walls; however, the combination of the overwhelmingly high extracellular  $K^+$  levels and the fact that so many of Jones' muscle cells had been destroyed by exertional rhabdomyolysis and the effects of the hyper-elevated extracellular  $K^+$  levels meant that there was too much extracellular  $K^+$  for Jones' body to handle and Jones' simply did not have enough healthy cells left in which to store all of the excess extracellular  $K^+$ .

## (ii.) Regulation Via Renal System Functions

Renal filtration and excretion of potassium ( $K^+$ ) is the body's primary method of regulating the external balance of potassium ( $K^+$ ). In addition, the kidneys secrete a variety of hormones, including erythropoietin and the renin enzyme. Erythropoietin is released in response to hypoxia (low levels of oxygen at tissue level) in the renal circulation. Erythropoietin stimulates erythropoiesis (production of red blood cells) in the bone marrow, which in turn expands the body's ability to correct the hypoxic condition by delivering oxygen to the cells. Renin is an enzyme component of the renin-angiotensin-aldosterone system, which is further described below, and is involved in the regulation of aldosterone levels.



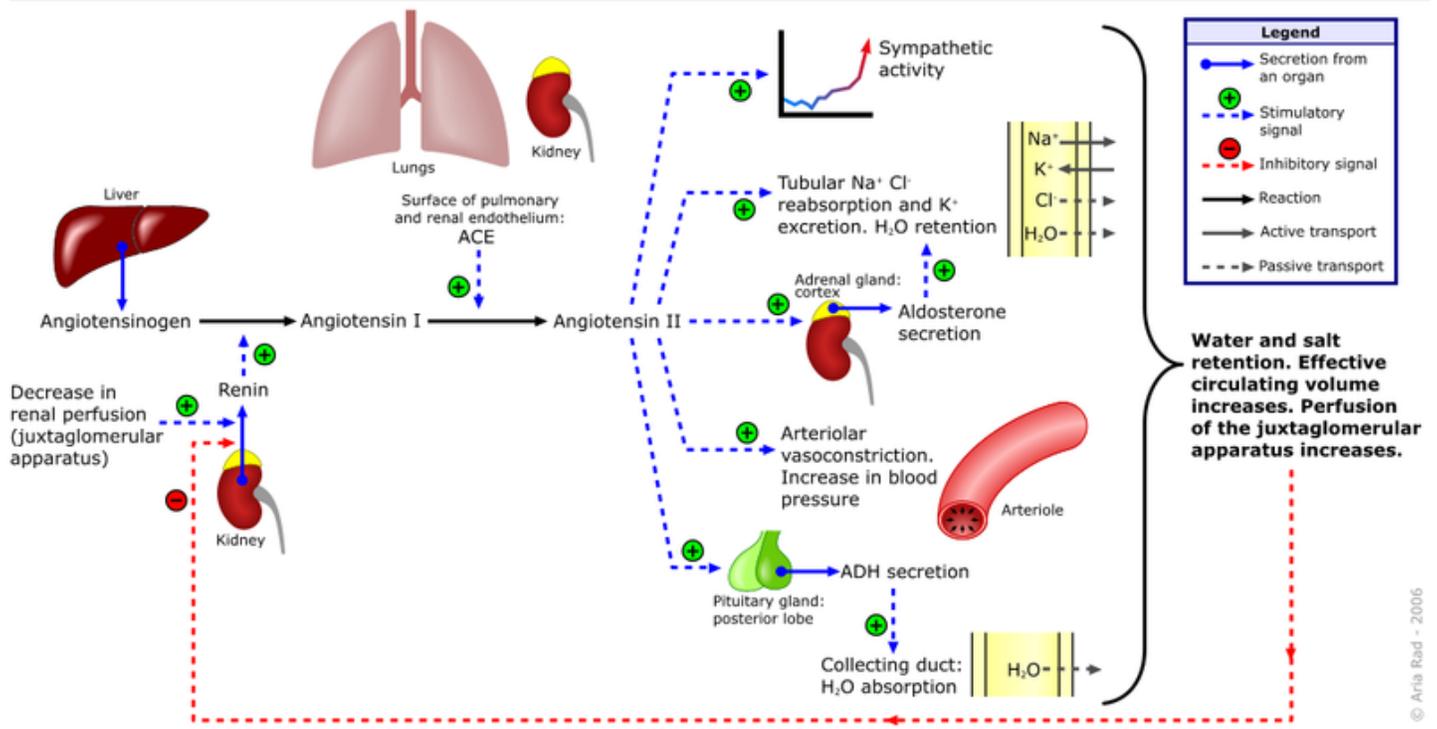
More than half of filtered potassium is passively reabsorbed by the end of the proximal convoluted tubule (PCT). Potassium is then added to tubular fluid in the descending limb of Henle's loop (see the diagram below). The major site of active potassium reabsorption is the thick ascending limb of the loop of Henle (TAL), so that, by

the end of the distal convoluted tubule (DCT), only 10% to 15% of filtered potassium remains in the tubule lumen. Potassium ultimately enters the urine through a process involving active secretion by the principal cells of the cortical collecting duct (CCD) and outer medullary collecting duct (OMCD). Potassium reabsorption occurs via the intercalated cells of the medullary collecting duct (MCD). Urinary potassium represents the difference between potassium secreted and potassium reabsorbed.

Major factors influencing potassium ( $K^+$ ) secretion at the cortical collecting duct include:

- Aldosterone
- Distal delivery of sodium and tubular fluid
- Presence of nonabsorbable anions in the tubular fluid perfusing the cortical collecting duct segment.

## Renin-angiotensin-aldosterone system



Aldosterone is a yellow steroid hormone (mineralocorticoid family) produced by the outer-section (zona glomerulosa) of the adrenal cortex in the adrenal gland sitting atop the kidneys. Aldosterone is responsible for the reabsorption of about 2% of filtered sodium in the kidneys, which is nearly equal to the entire sodium content in human blood under normal GFR (glomerular filtration rate). Aldosterone acts on the distal tubules and collecting ducts of the nephron (functional units of the kidneys) to cause the conservation of sodium, secretion of potassium, increased water retention, and increased blood pressure. Drugs that interfere with the secretion or action of aldosterone are used as anti-hypertensives and lower blood pressure by blocking aldosterone receptors.

Aldosterone is the primary of several endogenous members of the class of mineralocorticoids in humans. Deoxycorticosterone is another important member of this class. Aldosterone tends to promote  $Na^+$  and water retention, and lower plasma  $K^+$  concentration by the following mechanisms:

- Acting on the nuclear mineralocorticoid receptors (MR) within the principal cells of the distal tubule and the collecting duct of the kidney nephron, it upregulates and activates the basolateral  $Na^+/K^+$  pumps, stimulating ATP hydrolysis leading to phosphorylation of the pump and a

conformational change in the pump exposes the  $\text{Na}^+$  ions to the outside. The phosphorylated form of the pump has a low affinity for  $\text{Na}^+$  ions, hence reabsorbing  $\text{Na}^+$  ions and water into the blood, and secreting potassium ( $\text{K}^+$ ) ions into the urine.

- Upregulation of the epithelial sodium channel increasing apical membrane permeability for  $\text{Na}^+$ , thereby causing  $\text{Cl}^-$  to be reabsorbed in conjunction with the  $\text{Na}^+$  cations to maintain the system's electrochemical balance.
- Stimulation of uptake of  $\text{K}^+$  into cells.
- Stimulation of  $\text{Na}^+$  and water reabsorption from the gut salivary and sweat glands in exchange for  $\text{K}^+$ .
- Stimulation of  $\text{H}^+$  secretion by intercalated cells in the collecting duct, regulating plasma bicarbonate ( $\text{HCO}_3^-$ ) levels and its acid/base balance.
- Acting on the central nervous system via the posterior pituitary gland to release vasopressin (ADH), which conserves water by direct actions on renal tubular reabsorption.
- Acting through mineralocorticoid receptors, positively influencing neurogenesis in the dentate gyrus.

During states of total body potassium depletion, potassium reabsorption is enhanced. Reabsorbed potassium initially enters the medullary interstitium, but then it is secreted into the pars recta (PR) and descending limb of the loop of Henle (TDL). The physiologic role of medullary potassium recycling may be to minimize potassium “backleak” out of the collecting tubule lumen or to enhance renal potassium secretion during states of excess total body potassium.

As described above, when regulating both  $\text{K}^+$  and  $\text{Na}^+$ , the body moves  $\text{K}^+$  passively in counter flow to  $\text{Na}^+$  in response to an apparent (but not actual) Donnan equilibrium (the name used to describe behavior of charged particles to sometimes fail to distribute evenly across a semi-permeable membrane, thereby creating an uneven electrical charge). Thus, the  $\text{K}^+$  level in the body's urine can never sink below the concentration of  $\text{K}^+$  in the blood, except sometimes by actively excreting water at the end of processing.  $\text{K}^+$  is secreted twice and reabsorbed three times before the urine reaches the collecting tubules. At that point, urine usually has about the same  $\text{K}^+$  concentration as the plasma. Toward the end of serum processing by the kidneys,  $\text{K}^+$  is secreted one more time if serum levels are still too high.

#### **(d.) What Is the Body's Normal Rate of Potassium ( $\text{K}^+$ ) Filtration and Removal?**

If a healthy person, who normally eats a well balance diet, were to completely remove  $\text{K}^+$  from their diet, then their blood serum  $\text{K}^+$  level would decline to approximately 3.0-3.5 mEq (117-137 mg)/L in about one (1) week, yet there would still remain a minimum obligatory kidney excretion of  $\text{K}^+$  equal to about 200 mg per day. The body's excretion of  $\text{K}^+$  can never be completely shut off, thus death will result when the body's overall  $\text{K}^+$  level declines to somewhere around one-half full capacity.

All but the 1,000 to 10,000 mg of sodium ( $\text{Na}^+$ ) and the 1,000 to 4,000 mg of potassium ( $\text{K}^+$ ) likely to be in a person's normal diet must be reabsorbed by the kidneys to maintain proper electrolyte balance (homeostasis) and cellular function. Sodium ( $\text{Na}^+$ ) must be reabsorbed in such a way as to keep both the blood volume and osmotic pressure exactly correct and potassium ( $\text{K}^+$ ) must be reabsorbed in such a way as to keep serum concentrations as

close as possible to 4.8 mEq (188 mg) per liter. Even slight deviations from the prescribed levels cause the body to release bio-chemicals which activate both the internal and external balancing mechanisms to promptly restore the proper electrolyte levels.

Because the body has a relatively small amount of space in which to sodium ( $\text{Na}^+$ ), the kidney's sodium ( $\text{Na}^+$ ) pumps must always operate to conserve sodium ( $\text{Na}^+$ ). Sometimes potassium ( $\text{K}^+$ ) too must also be conserved, but since the amount of potassium ( $\text{K}^+$ ) in blood plasma is very small and the stores of potassium ( $\text{K}^+$ ) inside the body's cells is about forty times larger, the body's conservation of  $\text{K}^+$  is not quite as critical as it is for sodium ( $\text{Na}^+$ ).

If a normal, healthy, non-potassium deficient person were to consume the recommended daily amount of potassium ( $\text{K}^+$ ) (100 mEq (3,909 mg)), then in order to maintain homeostasis, 70 to 100 mEq of potassium ( $\text{K}^+$ ), must be removed from the body daily. The removal of potassium ( $\text{K}^+$ ) is primarily achieved through renal excretion. During periods of neutral potassium ( $\text{K}^+$ ) balance, the daily intake of potassium ( $\text{K}^+$ ) is equal to the daily excretion rate, primarily through urine. Only a small amount of potassium ( $\text{K}^+$ ) is excreted through the stool and sweat, as shown in the following chart.

<b><u>BODY'S NORMAL DAILY POTASSIUM (<math>\text{K}^+</math>) LOSS RATE &amp; SOURCE OF LOSS</u></b>	
<b><math>\text{K}^+</math> Loss in Urine</b>	90-95 mEq (3,519 – 3,714 mg) per day
<b><math>\text{K}^+</math> Loss in Stool</b>	10 mEq (391 mg) per day
<b><math>\text{K}^+</math> Loss in Sweat</b>	< 5 mEq (<195 mg) per day

**(i) Glomerular Filtration Rate (“GFR”)**

The glomerular filtration rate (“GFR”) is the volume of fluid filtered from the renal glomerular capillaries into the Bowman's capsule per unit of time. The GFR is used as a prognostic marker for kidney disease because the other markers may not detect the earlier stages of kidney disease or dysfunction. For example, blood urea nitrogen (BUN) will not be elevated above its normal range of 6 to 20 mg/dL until more than sixty percent (60%) of kidney function is lost due to damage or dysfunction.

GFR can be calculated by measuring the blood level of any chemical that has a steady level in the blood, and is freely filtered by the kidneys but is neither reabsorbed nor secreted during the process, then measuring the level of the same chemical in the urine.

The minimum GFR for a normal adult is 90 mL/min/1.73m<sup>2</sup>, meaning normal adult kidneys should filter and process a minimum of 5.4 liters of blood per hour or 129.6 liters of blood per day. Generally accepted norms place the typical adult male GFR at 120 mL/min, which equals 7.2 liters per hour and 173 liters per day. Therefore, using the upper end of the normal target values for  $\text{Na}^+$  at 145 mEq (3,334 mg)/L and  $\text{K}^+$  at 5.0 mEq (195.5 mg)/L, adult kidneys typically process and filter  $\text{Na}^+$  and  $\text{K}^+$  at the following rates:

<b><u>Time Period</u></b>	<b><u>Sodium (<math>\text{Na}^+</math>)</u></b>	<b><u>Potassium (<math>\text{K}^+</math>)</u></b>
<b>Hourly</b>	783 mEq to 1,044 mEq (18,004 mg to 24,005 mg)	27 mEq to 36 mEq (1,056 mg to 1,408 mg)
<b>Daily</b>	18,792 mEq to 25,085 mEq (432,022 mg to 576,782 mg)	648 mEq to 865 mEq (25,336 mg to 33,820 mg)

**(ii.) Estimating Glomerular Filtration Rate (“GFR”)**

Creatinine clearance or estimated creatinine clearance rates based on the serum creatinine levels are very commonly used to calculate the GFR. However, because creatinine is actively secreted by the peritubular capillaries in very small amounts, it is believed use of the creatinine clearance rate method actually overestimates GFR by ten to twenty percent (10% to 20%); but, this error rate is an acceptable tradeoff for the relatively inexpensive and easy manner of measuring blood serum creatinine levels. (Note: “Creatinine” should not be confused with “Creatine.” Creatinine is a by-product created by the breakdown of creatine phosphate, which is found in muscle cells.)

Aside from the over-estimation of GFR by using serum creatinine levels, another limitation of estimating GFR with serum creatinine levels is that all of the equations used to estimate GFR depend on a prediction of the twenty-four (24) hour excretion rate, which is a function of muscle mass. Therefore, estimated GFR values will be substantially affected by people with a greater muscle mass than is assumed by the formulas, thus it is often important for the formula used to contain a correction factor for both sex and race. In this regard, the Chronic Kidney Disease Epidemiology Collaboration Formula (“CKD-EPI Formula”) contains corrective factors for both sex and race, since it is known that on average men have more muscle mass than women and on average African Americans (both male and female) have more muscle mass than Caucasians. The CKD-EPI Formula for estimating GFR is as follows, including variables for sex, race and measured creatinine levels:

<b>CKD-EPI Formula for Estimating Glomerular Filtration Rate (GFR)</b>		
<b><u>Race &amp; Sex</u></b>	<b><u>Creatinine Level</u></b>	<b><u>Formula</u></b>
<b>African American Female</b>	Less Than or Equal to 0.7 mg/dL	Estimated GFR = $166 \times (\text{SCr}/0.7)^{-0.329} \times 0.993^{\text{Age}}$
<b>African American Female</b>	Greater Than 0.7 mg/dL	Estimated GFR = $166 \times (\text{SCr}/0.7)^{-1.209} \times 0.993^{\text{Age}}$
<b>African American Male</b>	Less Than or Equal to 0.9 mg/dL	Estimated GFR = $163 \times (\text{SCr}/0.9)^{-0.411} \times 0.993^{\text{Age}}$
<b>African American Male</b>	Greater Than 0.9 mg/dL	Estimated GFR = $163 \times (\text{SCr}/0.9)^{-1.209} \times 0.993^{\text{Age}}$
<b>Caucasian or Other Race Female</b>	Less Than or Equal to 0.7 mg/dL	Estimated GFR = $144 \times (\text{SCr}/0.7)^{-0.329} \times 0.993^{\text{Age}}$
<b>Caucasian or Other Race Female</b>	Greater Than 0.7 mg/dL	Estimated GFR = $144 \times (\text{SCr}/0.7)^{-1.209} \times 0.993^{\text{Age}}$
<b>Caucasian or Other Race Male</b>	Less Than or Equal to 0.9 mg/dL	Estimated GFR = $141 \times (\text{SCr}/0.9)^{-0.411} \times 0.993^{\text{Age}}$
<b>Caucasian or Other Race Male</b>	Greater Than 0.9 mg/dL	Estimated GFR = $141 \times (\text{SCr}/0.9)^{-1.209} \times 0.993^{\text{Age}}$

In Jones’ case, there is little doubt that Jones’ had a much greater muscle mass than any of the current formulas used to estimate GFR, even the CKD-EPI Formula; nevertheless, the following table shows the estimated GFR for Jones is reasonably close to the actually tested results. The following table provides both Jones’ estimated GFR (using the CKD-EPI Formula for an African American Male) and his actual, tested GFR compiled from hospital records from the time he arrived at Saline Memorial Hospital until the time of his death.

Jones blood urea nitrogen (BUN) levels are also included in the following table for use as a reference and comparison between the two test results. It is helpful to remember blood urea nitrogen (BUN) levels are only elevated above their normal range of 6-20 mg/dL when more than 60% of the kidney function has been lost due to damage or dysfunction.

**Anthony Jones' Glomerular Filtration Rate (GFR)**

<u>Time</u>	<u>Creatinine Level</u> (Normal 0.5 – 1.1 mg/dL)	<u>BUN</u> (Normal 6 – 20 mg/dL) Above 20 mg/dL = Evidence of 60% or Greater Kidney Damage or Dysfunction	<u>Glomerular Filtration Rate (GFR)</u> Normal – Above 90 mL/min. Mild Dysfunction – 60 to 89 mL/min. Moderate Dysfunction – 30 to 59 mL/min. Severe Dysfunction – 15 to 29 mL/min. Kidney Failure – Less Than 15 mL/min.
<b>22:45 hours (Saturday)</b>	2.7 mg/dL	19 mg/dL	Estimated – 36 Actual – Not Tested
<b>00:49 hours</b>	3.4 mg/dL	31 mg/dL	Estimated – 27 Actual – 26
<b>01:00 hours</b>	2.7 mg/dL	22 mg/dL	Estimated – 36 Actual – Not Tested
<b>03:04 hours</b>	3.8 mg/dL	22 mg/dL	Estimated – 24 Actual – 23
<b>04:00 hours</b>	4.0 mg/dL	24 mg/dL	Estimated – 22 Actual – 22
<b>06:17 hours</b>	4.8 mg/dL	24 mg/dL	Estimated – 18 Actual – 18
<b>06:43 hours</b> (1 Min. After Time of Death)	Not Tested	36 mg/dL	Estimated – Not Tested Actual – Not Tested

**(e) What Causes Abnormal Potassium (K<sup>+</sup>) and Sodium (Na<sup>+</sup>) Levels?**

Abnormal potassium (K<sup>+</sup>) levels can be caused by dysfunction or abnormality of the body's external balancing system, internal balancing system or both.

Ineffective potassium (K<sup>+</sup>) elimination can be hormonal (in aldosterone deficiency) or due to dysfunction of the renal parenchyma (improper kidney function, primarily with respect to the nephrons) which impairs excretion, as was the case here with Jones' renal tubular necrosis. Abnormalities in the external balance of potassium (K<sup>+</sup>) are primarily caused by dysfunction associated with renal tubular potassium (K<sup>+</sup>) secretion. Major factors which could lead to impaired potassium (K<sup>+</sup>) secretion include:

**(i) Decreased Glomerular Filtration Rate ("GFR")**

Decreasing glomerular filtration rates (GFR) typically trigger compensating responses within the body, such as increased blood pressure and flow through the kidneys, which allows the maintenance of normal external potassium balance. The body's typical compensation responses permit reduction of GFR down to severely reduced levels of 20 mL/min. or less for short periods of time. Thus, potassium (K<sup>+</sup>) is normally the last electrolyte for which homeostasis is lost in individuals with chronic kidney diseases.

As can be seen by Jones' table and medical records, Jones had severely reduced GFR evidenced as soon as an hour after the end of his Bout (see the blood test results at 22:45 hours). In fact, Jones had already suffered severe kidney damage and impairment at 22:45 hours, approximately one (1) hour after the end of the Bout, and had greater than sixty (60%) kidney damaged or dysfunction at 00:49 hours, just three (3) hours after the end of the Bout.

**(ii.) Decreased Renal Blood Flow or Decreased Renal Perfusion Pressure**

Decreased renal blood flow or decreased renal perfusion pressure (i.e. decreased blood pressure within the kidneys) causes the glomerulus to initiate an auto-regulation process to maintain a stable GFR. When the body maintains GFR at normal levels during conditions of decreased renal blood flow, a greater percentage of plasma reaching the glomerulus becomes glomerular filtrate (i.e. the volume of plasma passing from the lumen of the glomerular capillary to the space of the Bowman's capsule increases) in a process described as an increased filtration fraction. Filtration fraction equals GFR/renal plasma flow. Since normal glomerular filtration rate is 120 mL/min. and normal renal plasma flow is approximately 600 mL/min., the normal filtration fraction is .2 or 20%.

During auto-regulation, the plasma leaving the glomerulus at the efferent arteriole has decreased hydrostatic pressure (the pressure exerted by a fluid at equilibrium due to the force of gravity) and increased oncotic pressure (a form of osmotic pressure exerted by blood plasma proteins which tends to pull water into the blood stream). The efferent arteriole subsequently becomes the peritubular capillaries surrounding the proximal tubule. The Starling forces within these capillaries, decreased hydrostatic pressure and increased oncotic pressure, greatly increase proximal tubular reabsorption rates. The net result of enhanced proximal tubular reabsorption is decreased delivery of sodium ( $\text{Na}^+$ ) and tubular fluid to the distal nephron, including the cortical collecting duct. This significantly impairs the potassium ( $\text{K}^+$ ) secretory process.

Here, Jones' increased potassium ( $\text{K}^+$ ) levels cardiac dysrhythmias which decreased his heart's ability to effectively pump blood to the kidneys, thus reducing his renal blood flow and GFR. Despite his body's compensating responses and auto-regulation, damage to the epithelial cells of Jones' renal tubules from the release of myoglobin during muscle cell destruction prevented Jones' body from adequately compensating for the reduced renal blood flow.

**(iii.) Aldosterone Deficiency or Resistance to Aldosterone at Renal Tubular Level**

Adrenal insufficiency leads to both decreased cortisol and aldosterone production. Hyporeninemic hypoaldosteronism leads to decreased aldosterone because of decreased renin and, therefore, decreased production of angiotensin II, which is a primary stimulus for aldosterone secretion from the zona glomerulosa of the adrenal gland.

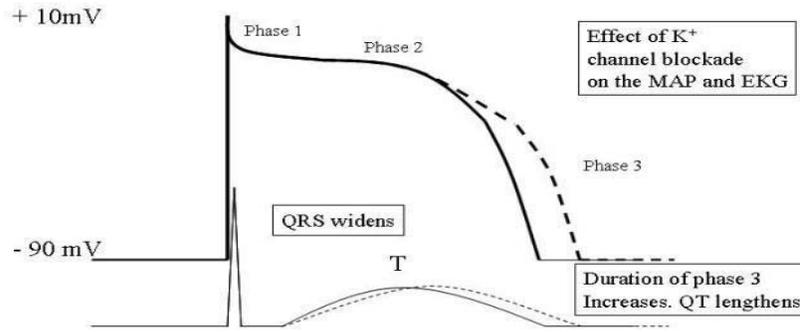
Aldosterone resistance is typically seen in conditions leading to direct tubular damage in the cortical collecting duct. Kidney diseases leading to chronic interstitial damage with renal tubular damage and corresponding aldosterone resistance include: analgesic nephropathy, allergic interstitial nephritis, polycystic kidney disease, etc..

Aldosterone production can be inhibited by the competitive antagonist, spironolactone, and other drugs which block the sodium channel in the luminal membrane of the cortical collecting duct (amiloride and trimterene). Angiotensive converting enzyme inhibitors and angiotensin II receptor antagonists may also lead to impaired aldosterone production. Trimethoprim and cyclosporine may also directly impair potassium secretion. Prolonged heparin usage can directly impair adrenal aldosterone production. Beta 1 receptor antagonists may have a mild effect to impair the rennin angiotensin aldosterone system. Severe digoxin toxicity may impair the sodium potassium ATPase.

Potassium secretion by the cortical collecting duct will be impaired by aldosterone deficiency, tubular resistance to the effects of aldosterone, or to drug related inhibition of the effects of aldosterone.

(f.) **What Medical Treatments Are Used to Lower Potassium (K<sup>+</sup>) Levels?**

**Monophasic Action Potential  
(Effect of Potassium Channel Blockers)**



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(i.) **Use of CaCl or Calcium Gluconate**

Most importantly for myocardial (cardiac) muscle cells in a hyperkalemic crisis is the prompt administration of Ca<sup>2+</sup> through either CaCl or calcium gluconate, as was done by the UAMS staff on numerous occasions while treating Jones. Administration of CaCl or calcium gluconate increases the myocardial muscle cells' threshold potential, thus restoring a normal gradient between threshold potential and resting membrane potential, both of which become abnormally elevated during hyperkalemic crisis.

One ampule of the CaCl used by the UAMS staff when treating Jones has approximately 3 times more calcium than calcium gluconate. Onset of CaCl action is less than 5 min, but only lasts about 30-60 minutes. Additional CaCl doses should be titrated with constant monitoring of ECG and blood test result changes during administration and doses should be repeated if ECG changes do not normalize within 3 to 5 minutes.

In addition, several other agents may be used to transiently lower K<sup>+</sup> levels. The agent employed depends on the degree and cause of the hyperkalemia, and other aspects of the patient's condition. Several medical treatments shift K<sup>+</sup> from the bloodstream into the cellular compartment, thereby reducing the risk of complications from hyperkalemia. The effect of these measures tends to be short-lived, but may temporarily ameliorate the critical condition until K<sup>+</sup> can be removed from the body through hemodialysis or other mechanisms.

(ii.) **Use of Insulin**

As copiously administered to Jones during treatment at UAMS, insulin (e.g. intravenous injection of 10-15 units of regular insulin along with D50 to prevent hypoglycemia) will lead to a shift of K<sup>+</sup> into cells, secondary to increased Na<sup>+</sup>/K<sup>+</sup> ATPase pumps activity, as also described above. The effects of insulin only last a few hours.

(iii.) **Use of Bicarbonate HCO<sub>3</sub>**

Also employed by UAMS staff in their valiant attempts to save Jones' life, was HCO<sub>3</sub> therapy (e.g. 1 ampule (45mEq) infused over 5 minutes), which is effective in cases of metabolic acidosis, which was experienced by Jones in the hours prior to his death. The introduction of HCO<sub>3</sub> into the body stimulates an exchange of cellular H<sup>+</sup> for Na<sup>+</sup>, thus leading to stimulation of the Na<sup>+</sup>/K<sup>+</sup> ATPase pump and movement of the excess intercellular fluid back into the cell structures.

**(iv.) Use of Salbutamol**

Salbutamol (albuterol, Ventolin) is a  $\beta_2$ -selective catecholamine that is administered by nebulizer (e.g. 10–20 mg). This drug also lowers blood levels of  $K^+$  by promoting  $K^+$  movement into cells.

**(v.) Use of Hemodialysis**

Severe cases of hyperkalemia require hemodialysis or hemofiltration, which are the most rapid methods of removing excess potassium from the body. Either hemodialysis or hemofiltration are typically used if the underlying cause of the hyperkalemia cannot be swiftly corrected while temporizing measures are instituted or there is no response to the medical team's other attempts to lower the potassium level.

In Jones' case, after all other means of reducing Jones' potassium level had failed, the UAMS medical team initiated the process to begin hemodialysis at 04:20 hours to 04:30 hours when the physician arrived in SICU to place the right femoral artery line. To increase the speed and efficacy of the treatment, the physician made the decision to switch to a Quinton catheter for emergency hemodialysis due to critically high potassium level.

**(vi.) Use of Polystyrene Sulfonate with Sorbitol (Kayexalate)**

Polystyrene sulfonate with sorbitol (Kayexalate) either orally or rectally is widely used with the goal to lower  $K^+$  over several hours. Complete removal of potassium ( $K^+$ ) is assumed to require defecation. However, well documented clinical trials to demonstrate the effectiveness of Kayexalate are lacking, and there are small risks of necrosis of the colon.

The medical team at UAMS utilized almost every weapon in its arsenal to counteract Jones' hyperkalemia including almost all of the above treatments. Despite their best efforts to get Jones'  $K^+$  back to normalized levels, neither Jones' body nor the UAMS medical staff could keep pace with Jones' ever increasing and consistently hyper-elevated potassium ( $K^+$ ) level. Virtually none of the medical intervention techniques provided adequate countermeasures for Jones' multiple, cascading medical conditions including the effects of exertional rhabdomyolysis, renal failure, heart damage and concussion.

**(g.) How and Why is Blood Pressure Important? How Is Blood Pressure Regulation Affected by Hyperkalemia?**

Blood pressure ("BP") measures the pressure exerted by circulating blood upon the walls of blood vessels. When used without further specification, BP usually refers to the arterial pressure of the systemic circulation. Blood pressure is the result of cardiac output increased by peripheral resistance; thus, abnormal change in blood pressure often indicates an underlying condition affecting the heart's output, vascular resistance, or both. In this instance, Jones' below normal and often critically low blood pressure readings were evidence of the decreased cardiac output resulting from the effects of Jones' hyperkalemia (elevated potassium  $K^+$ ).

During each heartbeat, the BP varies between a maximum, known as the "systolic pressure," and a minimum, known as the "diastolic pressure." Blood pressure is usually expressed in terms of systolic pressure over diastolic pressure with a normal, adult blood pressure ranging from 110/65 to 140/90. Jones' blood pressure during his pre-Bout physical four (4) hours before the Bout was 142/86, at the upper end of the normal range and completely normal for both experienced and inexperienced Combative Sports Contestants during pre-Bout physicals due to the inevitable nervousness and sometimes light warm-ups just prior to a Bout.

**(i.) Systolic Pressure**

Systolic pressure is created by the heart cells' coordinated contraction during the Atrial Systole, Isovolumic Ventricular Contraction and Ventricular Ejection phases of the "Cardiac Cycle." (A detailed discussion of the cardiac cycle and effects of electrolyte imbalances is provided in the following sections of this report.)

Unless otherwise specified and because blood pressure is typically measured at a person's arm, the measured systolic pressure is the pressure generated by contraction of the heart's left ventricle, which sends blood out of the heart and into the body. (The right ventricle sends blood from the heart to the lungs.)

**(ii.) Diastolic Pressure**

Diastolic pressure is associated with the decreased pressure within the heart's ventricular and atrial chambers during the Early Diastole and Isovolumic Ventricular Relaxation phases of the Cardiac Cycle. For instance, during Ventricular Diastole, the pressure inside the left ventricle is lower than that in the left atrium, which allows the mitral valve to open and blood to flow from the atrium to the ventricle. Diastolic pressure refers to the lowest pressure within the arterial blood stream during each Cardiac Cycle or heartbeat.

Low blood pressure can be caused by many things including sepsis (a very general term for any type of systemic blood toxins/poisons), hemorrhaging/blood loss, shock, and, as was the case here, cardiac dysrhythmia. If blood pressure drops too low, it will cause perfusion (blood supply) of the brain and other vital organs, such as the kidneys and liver, to be critically insufficient, eventually resulting in cell death and eventual organ failure.

Medical science has not yet been able to fully explain and understand all aspects of the endogenous regulation of (internal processes used to regulate) arterial blood pressure ("BP"); however, the following endogenous regulatory mechanisms have been well-characterized and explained as follows:

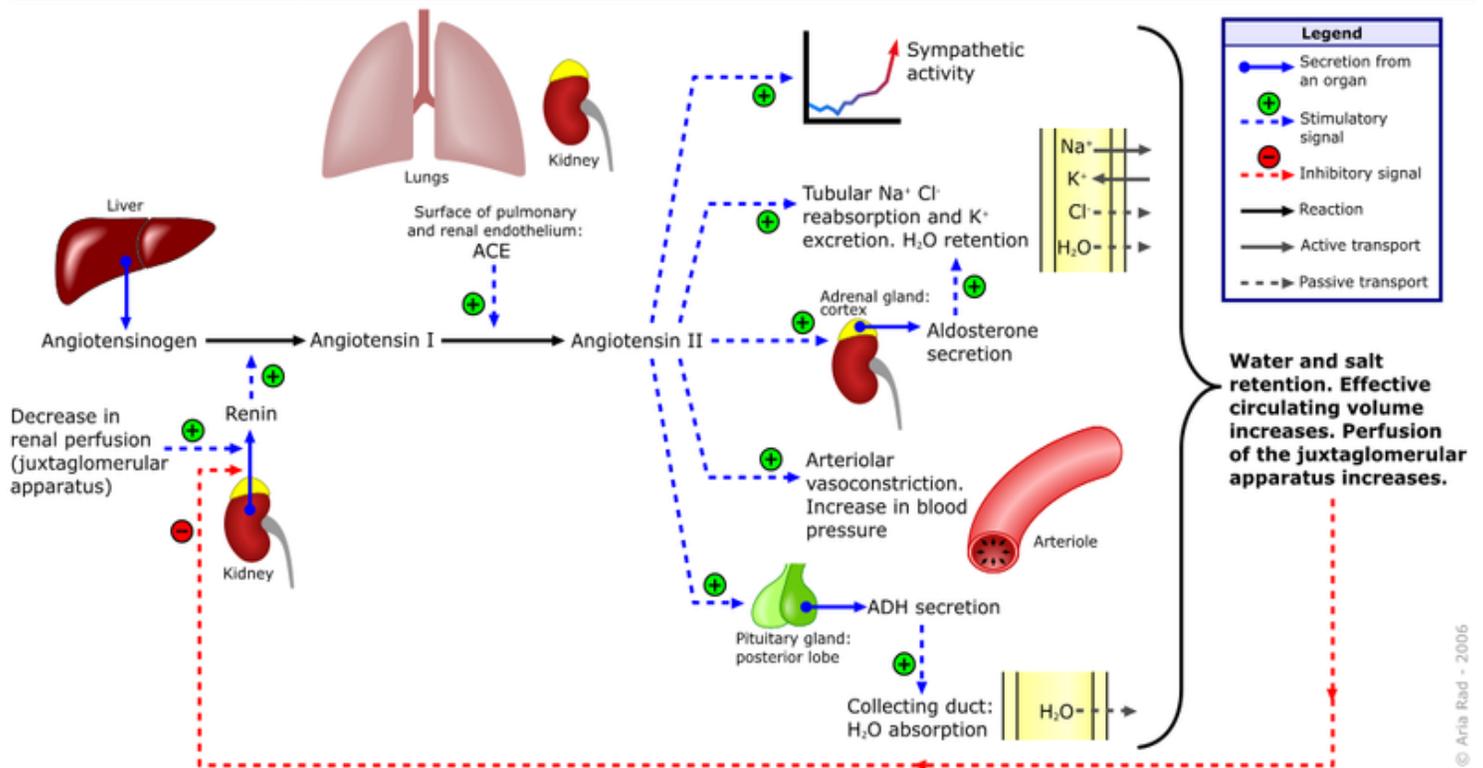
**(iii.) Baroreceptor Reflex**

Baroreceptors (a.k.a. the body's biologic pressure sensors) in the high pressure receptor zones (mainly in the aortic arch and carotid sinus) detect changes in arterial pressure. These baroreceptors send signals ultimately to the medulla of the brain stem. More precisely the signals are received by the Rostral Ventrolateral Medulla ("RVLM") region of the brain, which is a primary regulator of the sympathetic nervous system (i.e. the part of the nervous system responsible for mobilizing the body's systems in response to stress including, initiation of the "flight or fight response"). The medulla, by way of bio-chemical processes within the autonomic nervous system, adjusts the mean arterial blood pressure by altering both the force and speed of the heart's contractions, as well as the total peripheral resistance. It is believed the most important arterial baroreceptors are located in the left and right carotid sinuses and within the aortic arch.

In Jones' case, there is no medical evidence of any problem with his Baroreceptors; RVLM or medulla; instead, those regions of Jones' body were quite likely working just fine and sending the appropriate signals to increase his blood pressure from approximately 00:40 hours until his death. However, despite the signals being sent and body releasing the necessary chemicals to increase his blood pressure, the hyper-elevated levels of potassium  $K^+$  released into the blood stream by Jones' exertional rhabdomyolysis prevented Jones' heart from responding with the stronger and more coordinated and consistent contraction and relaxation cycles necessary to achieve an increase in blood pressure.

(iv.) Renin-Angiotensin System (“RAS”) & Renin-Angiotensin-Aldosterone System (“RAAS”)

## Renin-angiotensin-aldosterone system



Long-term regulation of blood pressure predominantly depends upon the kidney. This primarily occurs through maintenance of the extracellular fluid compartment, the size of which depends on the plasma sodium ( $\text{Na}^+$ ) concentration. Although the kidney cannot directly sense blood pressure, changes in the delivery of sodium ( $\text{Na}^+$ ) and chloride ( $\text{Cl}^-$ ) to the distal part of the nephron alter the kidney's secretion of the enzyme renin. When the extracellular fluid compartment is expanded and blood pressure is high, the delivery of these ions is increased and renin secretion is decreased. Similarly, when the extracellular fluid compartment is contracted and blood pressure is low, sodium ( $\text{Na}^+$ ) and chloride ( $\text{Cl}^-$ ) delivery is decreased and renin secretion is increased in response.

Renin is the first in a series of important chemical messengers that comprise the renin-angiotensin system. Changes in renin ultimately alter the output of this system, principally the hormones angiotensin II and aldosterone. Each hormone acts via multiple mechanisms, but both increase the kidney's absorption of sodium chloride, thereby expanding the extracellular fluid compartment and raising blood pressure. When renin levels are elevated, the concentrations of angiotensin II and aldosterone increase, leading to increased sodium chloride ( $\text{NaCl}$ ) reabsorption, expansion of the extracellular fluid compartment, and an increase in blood pressure. Conversely, when renin levels are low, angiotensin II and aldosterone levels decrease, contracting the extracellular fluid compartment, and decreasing blood pressure.

The Renin Angiotensin System (“RAS”) is generally associated with the body's long-term adjustment of arterial pressure. The RAS allows the kidney to compensate for loss in blood volume or drops in arterial pressure by activating an endogenous vasoconstrictor known as angiotensin II.

As seen in the diagram above, the Renin Angiotensin Aldosterone System (“RAAS”) involves complex interactions between the biochemical processes of the adrenal glands, kidneys, liver, lungs, and vascular systems.

As part of the RAAS processes, aldosterone is released. Aldosterone is a steroid hormone produced by the adrenal cortex region of the adrenal glands sitting atop the kidneys. The aldosterone release is triggered in response to angiotensin II or high serum potassium levels. Aldosterone stimulates sodium retention and potassium excretion by the kidneys. Sodium ( $\text{Na}^+$ ) ions are primarily responsible for utilizing principles of osmosis to control fluid levels inside the blood vessels; thus, the body's retention of Sodium ( $\text{Na}^+$ ) ions in response to aldosterone necessarily increases the body's fluid retention, and indirectly, arterial pressure by increasing the volume of fluid inside the blood vessels.

Baroreceptors in low pressure receptor zones (mainly in the venae cavae and the pulmonary veins, and in the atria region of the heart) provide feedback through release of chemical signals which regulate secretion of antidiuretic hormone (ADH/Vasopressin), Renin and Aldosterone. The resultant increase in blood volume increases cardiac output by the Frank–Starling law of the heart, in turn increasing arterial blood pressure. These different mechanisms are not necessarily independent of each other, as indicated by the link between the RAS and Aldosterone release.

#### (v.) Use of Food & Drugs to Regulate Blood Pressure

The most common method for lowering blood pressure is through modification of diet, primarily lowering sodium ( $\text{Na}^+$ ) intake. Meanwhile, current medical practice pharmacologically targets the RAS by the use of Angiotensin Converting Enzyme Inhibitors (“ACE Inhibitors”) and Angiotensin II receptor antagonists. ACE Inhibitors block the conversion of angiotensin I to angiotensin II thereby indirectly lowering blood pressure by lowering arteriolar resistance, lowering renovascular resistance, decreasing glomerular filtration rates of the kidneys, decreasing sodium ( $\text{Na}^+$ ) levels, and increasing potassium ( $\text{K}^+$ ) levels. Although ACE inhibitors lower blood pressure and result in the other counterintuitive effects described above, other effects of ACE inhibitors are very beneficial in certain situations which cause current medical practice to employ the maximum dose of ACE inhibitors during cardiac arrest and other situations involving prophylaxis of cardiac events.

The Aldosterone System is directly targeted by spironolactone, an Aldosterone antagonist. Fluid retention is targeted using either diuretics or anti-diuretics, which in turn affect blood volume and a resultant change in blood pressure. Generally, the Baroreceptor Reflex is not targeted in hypertension (high blood pressure) because if blocked, individuals may suffer from orthostatic hypotension (i.e. a very quick drop in blood pressure of more than 20/10 mm Hg, which results in a “head rush,” dizziness, light headedness and fainting).

#### (h.) The Cardiac Cycle – How Do Heart Muscle Cells Work?

The heart is comprised of a specialized group of striated muscle cells which pump blood throughout the body using a very highly coordinated and strictly regulated sequence and rate of individual cell contraction and relaxation. Each beat of the human heart involves a coordinated series of electrical impulses generated by the striated muscle cells and is identified by the following five (5) major stages known as the “Cardiac Cycle”:

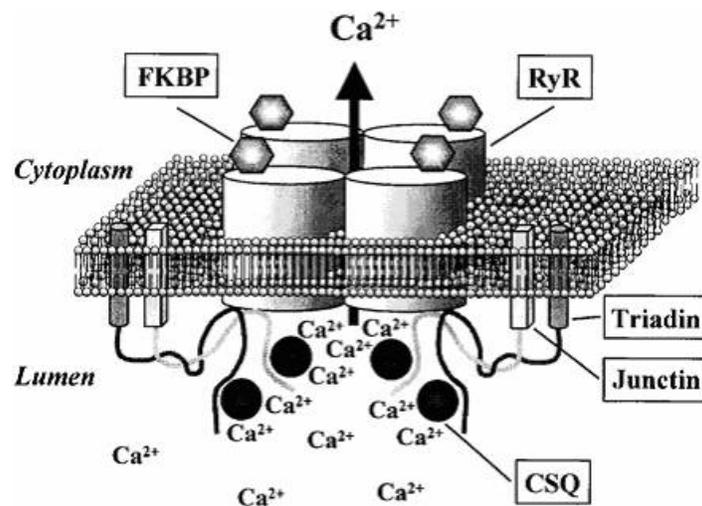
- (i.) Early Diastole – When the semilunar valves close, the atrioventricular (AV) valves open, and the whole heart is relaxed.
- (ii.) Atrial Systole – When the atrium contracts, the AV valves open, and blood flows from atrium to the ventricle.
- (iii.) Isovolumic Ventricular Contraction – When the ventricles begin to contract, the AV and semilunar valves close, and there is no change in volume.

- (iv.) **Ventricular Ejection** – When the ventricles are empty and contracting, and the semilunar valves are open.
- (v.) **Isovolumic Ventricular Relaxation** – Pressure decreases, no blood enters the ventricles, the ventricles stop contracting and begin to relax, and the semilunar valves close due to the pressure of blood in the aorta.

(i.) **Electrolytes – What Specific Role do Electrolytes Play in Proper Heart Cell Function?**

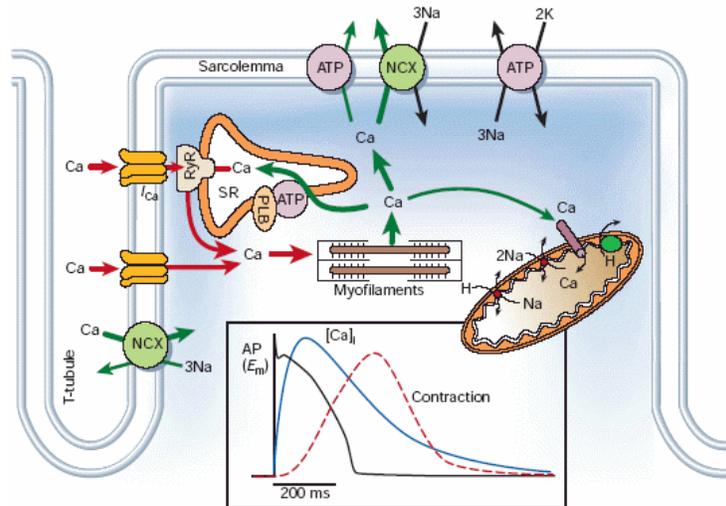
The concentration level of the three (3) electrolytes, potassium ( $K^+$ ), sodium ( $Na^+$ ), and calcium ( $Ca^{2+}$ ), both inside and outside the heart cell, as well as the timing of the concentration levels is critical to the proper function of a heart muscle cell. In order to contract, all muscle cells require the level of Calcium ( $Ca^{2+}$ ) inside the cell to increase anywhere from 100 to 10,000 times more than the levels observed during its normal, resting state. However, in order to relax, the level of Calcium ( $Ca^{2+}$ ) only has to return to its normal level. The level of Calcium ( $Ca^{2+}$ ) inside a muscle cell is controlled by a highly specialized process involving the  $Na^+/K^+$  ATPase Pump, the  $Na^+-Ca^{2+}$  translocator enzyme and sub-units of each cell called sarcoplasmic reticulum and troponin.

Like a neuron, a given myocardial cell has a negative membrane potential when at rest. Stimulation above a threshold value induces the opening of voltage gated ion channels with inducted flow of cations into the cell. The positively charged ions entering the cell cause the depolarization characteristic of an action potential. After depolarization, there's a brief repolarization that takes place with the efflux of  $K^+$  through fast acting potassium channels. Like skeletal muscle, depolarization causes the opening of voltage gated  $Ca^{2+}$  channels - meanwhile  $K^+$  channels have closed - and are followed by a titrated release of  $Ca^{2+}$  from the t-tubules. This influx of  $Ca^{2+}$  causes calcium-induced calcium release from the sarcoplasmic reticulum, and free  $Ca^{2+}$  causes muscle contraction. After a delay, slow acting  $K^+$  channels reopen and the resulting flow of  $K^+$  out of the cell causes repolarization to the resting state.

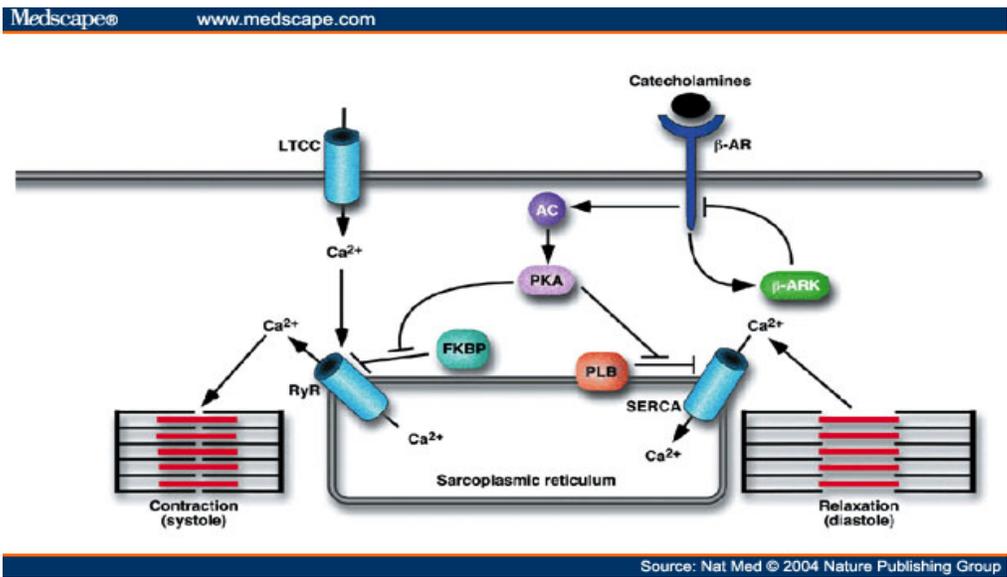


As depicted in the above diagram, unlike skeletal muscle, cardiac muscle requires extracellular  $Ca^{2+}$  for contraction to occur. The reason for the  $Ca^{2+}$  dependence is due to the mechanism of calcium-induced calcium release (CICR) from the sarcoplasmic reticulum that must occur under normal excitation-contraction (EC) coupling to cause contraction. Once the intracellular concentration of calcium increases, calcium ions bind to the protein troponin, which initiates contraction by allowing the contractile proteins, myosin and actin to associate through cross-bridge formation. Cardiac muscle is intermediate between smooth muscle, which has an unorganized sarcoplasmic reticulum and derives its  $Ca^{2+}$  from both the extracellular fluid and intracellular stores, and skeletal muscle, which is only activated by calcium stored in the sarcoplasmic reticulum.

In the absence of sufficient  $\text{Ca}^{2+}$ , tropomyosin interferes with the action of myosin; therefore, cardiac cells remain relaxed. If the level of  $\text{Ca}^{2+}$  in the blood (outside the cells) is too low, as it was for Jones at 03:03 hours, then there is insufficient movement of  $\text{Ca}^{2+}$  into the cell to trigger the sarcoplasmic reticulum (SR) to release more  $\text{Ca}^{2+}$  from inside the cell. The SR's failure to release additional  $\text{Ca}^{2+}$  results in the cell never contracting through the attachment of the  $\text{Ca}^{2+}$  ions to troponin. Alternatively, there may be insufficient extracellular  $\text{Ca}^{2+}$  to flow into the cell through L-type  $\text{Ca}^{2+}$  channels to sustain the depolarization and contraction of ventricular muscle cells for a long enough duration, thus shortening the cycle. The failure of Jones' heart cells to either not contract or sustain their contraction period caused Jones' irregular heartbeat (dysrhythmia) and further decreased blood flow to Jones' kidneys and brain.



Normally, the  $\text{Na}^+/\text{K}^+$  ATPase “pumps” two (2)  $\text{K}^+$  ions into the cell and removes three (3)  $\text{Na}^+$  out of the cell, which destabilizes the cell’s membrane potential and allows the  $\text{Na}^+-\text{Ca}^{2+}$  translocator enzyme to “pump”  $\text{Ca}^{2+}$  out of the cell and  $\text{Na}^+$  back into the cell during contraction and relaxation phases of the cell’s cycle. The initiation and upshoot of the action potential in ventricular muscle cells is derived from the entry of  $\text{Na}^+$  across the sarcolemma during the regenerative process. Then, the movement of  $\text{Ca}^{2+}$  into the cell triggers the sarcoplasmic reticulum to release more  $\text{Ca}^{2+}$  from inside the cell causing the cell to contract through the attachment of the  $\text{Ca}^{2+}$  ions to troponin. The inflow of extracellular  $\text{Ca}^{2+}$  through L-type  $\text{Ca}^{2+}$  channels sustains the depolarization and contraction of ventricular muscle cells for a longer duration.



**(j.) What Is the Source of the Calcium (Ca<sup>2+</sup>) Used By Cardiac Cells?**

The short answer is that Ca<sup>2+</sup> ions come from both inside and outside the cells and a very tightly controlled level in the blood must be maintained by the kidneys. The more complex answer involves the production of Ca<sup>2+</sup> by the smooth endoplasmic reticulum and sarcoplasmic reticulum. Calcium (Ca<sup>2+</sup>) plays a pivotal role in the physiology and biochemistry of organisms and the cell. It plays an important role in signal transduction pathways, where it acts as a second messenger, in neurotransmitter release from neurons, contraction of all muscle cell types, and fertilization. Many enzymes require calcium ions as a cofactor, those of the blood-clotting cascade being notable examples. Extracellular calcium is also important for maintaining the potential difference across excitable cell membranes, as well as proper bone formation. However, an improper balance of extracellular electrolytes including the hyper-elevation of extracellular potassium (K<sup>+</sup>), results in additional imbalances of the intracellular and extracellular levels of Na<sup>+</sup> ions and Ca<sup>2+</sup> ions which make it difficult, if not impossible for the cell to properly contract and relax.

Calcium levels in mammals are tightly regulated, with bone acting as the major mineral storage site. Calcium ions, Ca<sup>2+</sup>, are released from bone into the bloodstream under controlled conditions. Calcium is transported through the bloodstream as dissolved ions or bound to proteins such as serum albumin. Parathyroid hormone secreted by the parathyroid gland regulates the resorption of Ca<sup>2+</sup> from bone, reabsorption in the kidney back into circulation, and increases in the activation of vitamin D<sub>3</sub> to Calcitriol. Calcitriol, the active form of vitamin D<sub>3</sub>, promotes absorption of calcium from the intestines and the mobilization of calcium ions from bone matrix. Calcitonin secreted from the parafollicular cells of the thyroid gland also affects calcium levels by opposing parathyroid hormone; however, its physiological significance in humans is dubious.

The effects of calcium in humans are cell specific, meaning the different types of cells respond in different ways to Calcium (Ca<sup>2+</sup>). However, in certain circumstances, the action of Calcium (Ca<sup>2+</sup>) may be more general. Calcium (Ca<sup>2+</sup>) ions are one of the most widespread second messengers used in signal transduction. They make their entrance into the cytoplasm either from outside the cell through the cell membrane via calcium channels (such as Calcium-binding proteins or voltage-gated calcium channels), or from some internal calcium storages such as the endoplasmic reticulum and mitochondria. Levels of intracellular calcium are regulated by transport proteins that remove it from the cell. For example, the sodium-calcium exchanger uses energy from the electrochemical gradient of sodium by pumping calcium out of the cell in exchange for the entry of sodium. In addition, the plasma membrane Ca<sup>2+</sup> ATPase (PMCA) obtains energy to pump calcium out of the cell by hydrolyzing adenosine triphosphate (ATP). In neurons, voltage-dependent, calcium-selective ion channels are important for synaptic transmission through the release of neurotransmitters into the synaptic cleft by vesicle fusion of synaptic vesicles.

Calcium storage sites are intracellular organelles that constantly accumulate Ca<sup>2+</sup> ions and release them during certain cellular events. Intracellular Ca<sup>2+</sup> storage sites include mitochondria and the endoplasmic reticulum. In cell biology, an organelle is a specialized subunit within a cell that has a specific function, and is usually separately enclosed within its own lipid bi-layer. The name *organelle* comes from the idea that these structures are to cells what an organ is to the body. In Jones' case, the organelles were hemolyzed (destroyed) by the oxidizing side effects from rhabdomyolysis.

**(i.) Smooth Endoplasmic Reticulum & Sarcoplasmic Reticulum**

The smooth endoplasmic reticulum (SER) has functions in several metabolic processes, including synthesis of lipids and steroids, metabolism of carbohydrates, regulation of Ca<sup>2+</sup> concentration, drug detoxification, attachment of receptors on cell membrane proteins, and steroid metabolism. It is connected to the nuclear envelope. Smooth endoplasmic reticulum is found in a variety of cell types (both animal and plant) and it serves different functions in each. The Smooth ER also contains the enzyme glucose-6-phosphatase which converts

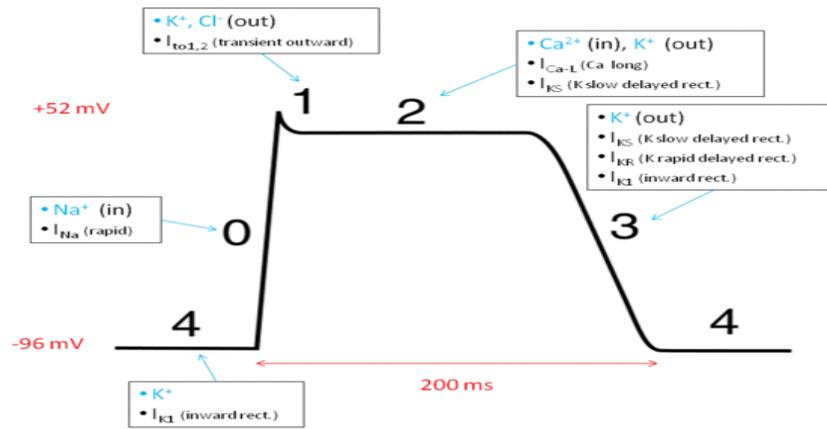
glucose-6-phosphate to glucose, a step in gluconeogenesis. The SER consists of tubules and vesicles that branch forming a network. In some cells there are dilated areas like the sacs of RER. The network of SER allows increased surface area for the action or storage of key enzymes and the products of these enzymes.

The sarcoplasmic reticulum (SR), is a special type of SER found in smooth and striated muscle. The only structural difference between this organelle and the SER is the medley of proteins they have, both bound to their membranes and drifting within the confines of their lumens. This fundamental difference is indicative of their functions: the SER synthesizes molecules while the SR stores and pumps  $Ca^{2+}$ . The SR contains large stores of  $Ca^{2+}$ , which it sequesters and then releases when the muscle cell is stimulated. The SR's release of  $Ca^{2+}$  upon electrical stimulation of the cell plays a major role in excitation-contraction coupling.

**(k.) What Happens During Each Phase of Heart Muscle Contraction Cycle?**

The five phase biochemical process involved in one heart muscle cell cycle is described in the following chart and depicted in the diagram which follows:

<b><u>Phase 4</u></b>	Phase 4 is the resting membrane potential. This is the period that the cell remains in until it is stimulated by an external electrical stimulus (typically an adjacent cell). This phase of the action potential is associated with diastole of the chamber of the heart. In addition to stimulus from adjacent cells, certain cells of the heart have the ability to undergo spontaneous depolarization, in which an action potential is generated without any influence from nearby cells. This is known as cardiac muscle automaticity.
<b><u>Phase 0</u></b>	Phase 0 is the rapid depolarization phase. The slope of phase 0 represents the maximum rate of depolarization of the cell and is known as $dV/dt_{max}$ . This phase is due to the opening of the fast $Na^+$ channels causing a rapid increase in the membrane conductance to $Na^+$ ( $G_{Na}$ ) and thus a rapid influx of $Na^+$ ions into the cell; a $Na^+$ current. The ability of the cell to open the fast $Na^+$ channels during phase 0 is related to the membrane potential at the moment of excitation. If the membrane potential is at its baseline (about -85 mV), all the fast $Na^+$ channels are closed, and excitation will open them all, causing a large influx of $Na^+$ ions. If, however, the membrane potential is less negative, some of the fast $Na^+$ channels will be in an inactivated state insensitive to opening, thus causing a lesser response to excitation of the cell membrane and a lower $V_{max}$ . For this reason, if the resting membrane potential becomes too positive, the cell may not be excitable, and conduction through the heart may be delayed, increasing the risk for arrhythmias. <p style="text-align: center;"><b><u>The Fast <math>Na^+</math> Channel</u></b></p> The fast sodium channel can be modeled as being controlled by a number of gates. Each gate (or gating variable) can attain a value between 1 (fully open) and 0 (fully closed). The product of all the gates denotes the percentage of channels available to conduct $Na^+$ . Following the model of Hodgkin and Huxley, the sodium channel contains three gates: $m$ , $h$ , and $j$ . In the resting state, the $m$ gate is closed (zero) and the $h$ and $j$ gates are open (one). Hence, the product denoting the percentage of conducting channels is also zero. Upon electrical stimulation of the cell, the $m$ gate opens quickly while simultaneously the $h$ and $j$ gates close more slowly. For a brief period of time, all gates are open ( <i>i.e.</i> non-zero) and $Na^+$ can enter the cell following its electrochemical gradient. If, as above, the resting membrane potential is too positive, the $h$ or $j$ gates may be considerably less than one, such that the product of $m$ , $h$ and $j$ becomes too small upon depolarization
<b><u>Phase 1</u></b>	Phase 1 of the action potential occurs with the inactivation of the fast $Na^+$ . The transient net outward current causing the small downward deflection of the action potential is due to the movement of $K^+$ and $Cl^-$ ions, carried by the $I_{to1}$ and $I_{to2}$ currents, respectively. Particularly the $I_{to1}$ contributes to the “notch” of some ventricular cardiomyocyte action potentials. It has been suggested that $Cl^-$ ions movement across the cell membrane during Phase I is as a result of the change in membrane potential, from $K^+$ efflux, and is not a contributory factor to the initial repolarization (“notch”).
<b><u>Phase 2</u></b>	This “plateau” phase of the cardiac action potential is sustained by a balance between inward movement of $Ca^{2+}$ ( $I_{Ca}$ ) through L-type calcium channels and outward movement of $K^+$ through the slow delayed rectifier potassium channels, $I_{Ks}$ . The sodium-calcium exchanger current, $I_{Na,Ca}$ and the sodium/potassium pump current, $I_{Na,K}$ also play minor roles during phase 2.
<b><u>Phase 3</u></b>	During phase 3 (the “rapid repolarization” phase) of the action potential, the L-type $Ca^{2+}$ channels close, while the slow delayed rectifier ( $I_{Ks}$ ) $K^+$ channels are still open. This ensures a net outward current, corresponding to negative change in membrane potential, thus allowing more types of $K^+$ channels to open. These are primarily the rapid delayed rectifier $K^+$ channels ( $I_{Kr}$ ) and the inwardly rectifying $K^+$ current, $I_{K1}$ . This net outward, positive current (equal to loss of positive charge from the cell) causes the cell to repolarize. The delayed rectifier $K^+$ channels close when the membrane potential is restored to about -80 to -85 mV, while $I_{K1}$ remains conducting throughout phase 4, contributing to set the resting membrane potential.



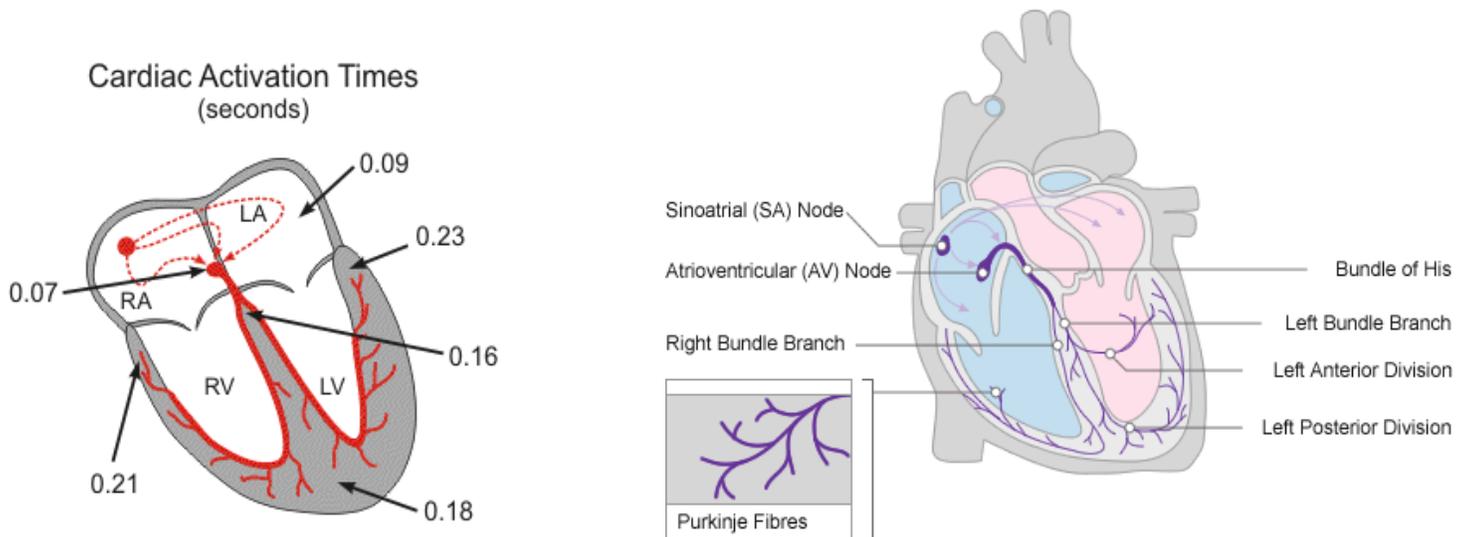
**(I.) What Makes Heart Muscle Cells Different Than Other Muscle Cells?**

Because the heart performs one of the most critical functions in the human body, pumping oxygen deficient blood to the lungs to exchange carbon dioxide for oxygen and pumping oxygen rich blood to the rest of the body, the approximately 500 million cardiac cells are all highly specialized, synchronized and coordinated to fulfill their respective role in creating a single heartbeat nearly 2.6 billion times during a normal person’s lifetime. The differences between cardiac cells and other muscle cells are important to a full understanding of Jones’ death.

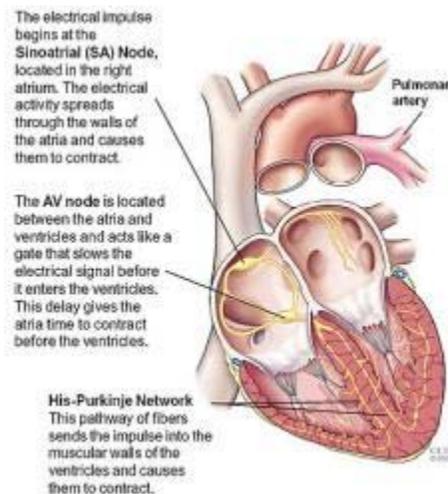
Cardiac cells are almost totally dependent on aerobic metabolism for energy production, have an automaticity characteristic, and have contractions that last nearly ten (10) times longer than other skeletal muscle cells; thus, cardiac cells are very sensitive to low blood oxygen levels and abnormal levels of the electrolytes, potassium ( $\text{K}^+$ ), sodium ( $\text{Na}^+$ ), and calcium ( $\text{Ca}^{2+}$ ). R. Matini, *Fundamentals of Anatomy & Physiology (5<sup>th</sup> Ed.)*. Prentice-Hall, Inc. (2000).

**(i.) Automaticity**

In the myocardium, automaticity is the ability of the cardiac muscles to depolarize spontaneously, i.e. without external electrical stimulation from the nervous system. This spontaneous depolarization is due to the plasma membranes within the heart that have reduced permeability to potassium ( $\text{K}^+$ ) but still allow passive transfer of calcium ( $\text{Ca}^{2+}$ ) ions, allowing a net charge to build. Automaticity is most often demonstrated in the sinoatrial node, which is the so called “Pacemaker of the Heart,” seen in the diagram below.



Cells with the greatest automaticity, thus capable of undergoing spontaneous depolarization the fastest, are the primary pacemaker cells of the heart, and set the heart rate. Usually, these “pacemaker cells” are located in the sinoatrial node (“SA node”) of the heart, as depicted in the diagram below. Consistent with their role as the “pacemaker cells,” electrical activity originating from the SA node is propagated throughout the rest of heart’s 500 million cells at an extraordinarily fast rate ranging from 0.5 meters per second in the SA node to 0.05 m/sec. in atrioventricular node (“AV node”) to 2.0 m/sec in the Bundle of the His and left and right bundle branches to 4.0 m/sec. in the Purkinje fibers of the left and right ventricles. In fact, the electrical conduction system of the heart contains the body’s fastest mechanisms for conduction of electrical activity.



The normal activity of the heart’s pacemaker cells is to spontaneously depolarize at a regular rhythm, generating the normal heart rate. Abnormal automaticity involves the abnormal spontaneous depolarization of cells of the heart. This typically causes arrhythmias (irregular rhythms) in the heart such as those experienced by Jones, sinus tachycardia, ventricular tachycardia and ventricular fibrillation. In cases of heart blockage, in which the activity of the primary pacemaker does not propagate to the rest of the heart, a latent pacemaker (also known as an escape pacemaker) will undergo spontaneous depolarization and create an action potential.

Of particular importance to our understanding of what happened to Jones, is an understanding of how the automaticity of Jones’ heart was affected by the increased level of potassium ( $K^+$ ) in his blood. The rate of depolarization and duration of the action potential in the pacemaker cells of the SA node is regulated by the parasympathetic and sympathetic fibers of the autonomic nervous system (see the description of the functions of the mid-brain and medulla oblongata earlier in this report) in addition to circulating catecholamines (commonly known as the “fight or flight” hormones produced by the adrenal glands).

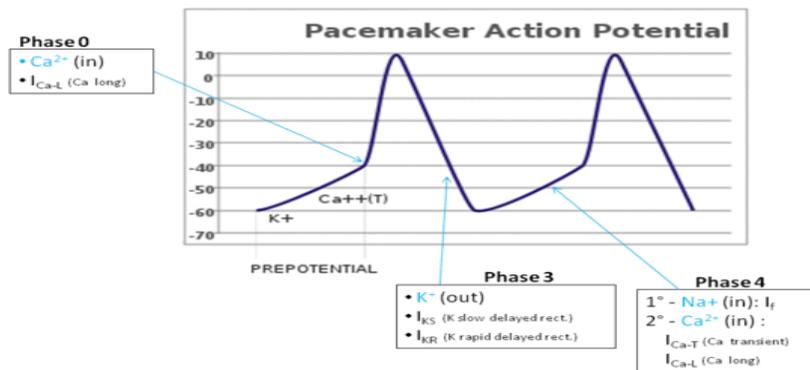
Specifically, acetylcholine (ACh) (a neurotransmitter released by several different parts of the nervous system) binds to the muscarinic acetylcholine receptors  $M_2$  and, via the  $\beta\gamma$  subunit of a G protein, opens a special set of potassium ( $K^+$ ) channels. The resulting increase in potassium ( $K^+$ ) efflux (outflow from the cell) slows the depolarizing effect of the “funny channels” circuits. In addition, activation of  $M_2$  receptors decreases cAMP in the cells and this slows the opening of the  $Ca^{2+}$  “L” channels. The normal result of the foregoing is a decrease in the cell’s firing rate. Conversely, sympathetic stimulation via  $\beta_1$  receptors results in an increase in cAMP levels which facilitates the opening of  $Ca^{2+}$  channels thereby increasing the rate of depolarization.

Abnormalities in the automaticity of Jones’ heart resulted, in part, from Jones’ hyper-elevated extracellular levels of potassium ( $K^+$ ) and were seen as rhythm changes in the tachycardia noted at 00:30 hours, sinus tachycardia noted in Jones’ ECG results at 01:00 hours, ventricular tachycardia noted at 02:58 hours, and eventual

asystole beginning at 05:10 hours. More specifically and probably not the only mechanism of arrhythmia, when Jones' body produced the ACh intended to slow down his heartbeat by binding to the  $M_2$  and opening the potassium ( $K^+$ ) channels of his heart muscle cells to permit a flow of potassium ( $K^+$ ) out of each cardiac cell, the potassium ( $K^+$ ) was prevented from flowing out of the cells as it normally would due to the already very high extracellular concentration of potassium ( $K^+$ ). Meanwhile, Jones' cardiac cells continued to their other functions as if the potassium ( $K^+$ ) had actually flowed outward which cast each cell into a chaotic state with some cells partially firing or contracting at a rapid rate which resulted in a very rapid and uncoordinated heartbeat.

It is interesting to note that most cardiac myocardial cells with an increased propensity to developing arrhythmia have an associated loss of membrane potential, the same type of lost membrane potential caused by Jones' very high extracellular concentration of potassium ( $K^+$ ) and described in the previous paragraph. In a lost membrane potential scenario, the maximum diastolic potential (relaxed potential) is less negative and therefore exists closer to the threshold potential (point at which the cell begins to contract). The ionic basis of automaticity is the net gain of an intracellular positive charge during diastole (cellular relaxation) in the presence of a voltage-dependent channel activated by potentials negative to  $-50$  to  $-60$  mV. Although Jones' experienced complications from increased extracellular potassium ( $K^+$ ), irregular cellular depolarization can be due to many different causes including a raised extracellular concentration of  $K^+$ , a decreased intracellular concentration of  $Na^+$ , increased permeability to  $Na^+$ , or a decreased permeability to  $K^+$ .

(ii.) Pacemaker Cell Action

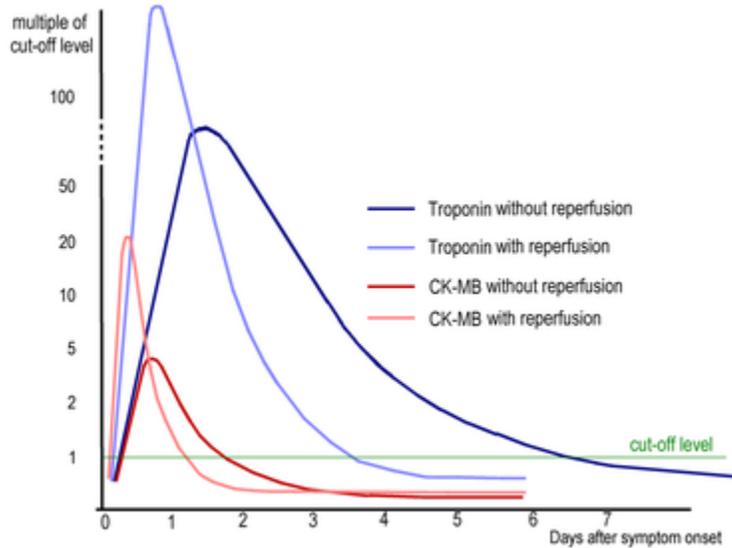


The mechanism of automaticity involves the so-called pacemaker channels of the HCN family, hyperpolarization-activated, Cyclic Nucleotide-gated channels. The pacemaker channels are poorly selective cation channels ( $K^+$  and  $Na^+$  are cations) which conduct more current as the membrane potential becomes more negative, or hyperpolarized by the flow of  $K^+$  and  $Na^+$  out of the cell. This, in turn, means the pacemaker channels are more susceptible to improper extracellular electrolyte levels, such as what Jones had here with the elevated extracellular potassium level associated with hyperkalemia onset by exertional rhabdomyolysis.

The initial “pre-potential” activity of these pacemaker channels in the SA node cells cause their membrane potential to slowly become more positive (depolarize as the ratio of  $K^+$  and  $Na^+$  inside cell changes relative to that outside the cell). The pacemaker cells’ “pre-potential” activity is conducted through what are known as “Funny” channels. The latter phase is due to the opening of “T” or “Transient”  $Ca^{2+}$  channels. The flow of  $Ca^{2+}$  ions further depolarizes the cell until, eventually, the “L” or “Long Lasting”  $Ca^{2+}$  channels are activated and an action potential is initiated, resulting in the cell contracting.

Jones' increased level of positively charged potassium ions in the extracellular fluid surrounding his "pacemaker" cells caused his poorly selective pacemaker channels, especially the "Funny" channels associated with their "pre-potential" activity, to depolarize too quickly and incompletely in an irregular manner, thus resulting in the rapid heartbeats seen in Jones' medical records from 22:27 hours until the time of his death, with peaks of 175 beats per minute occurring during his V-tach event at 02:58 hours and 220 beats per minute occurring during the continued V-tach event at 03:37 hours.

**(iii.) Troponin**

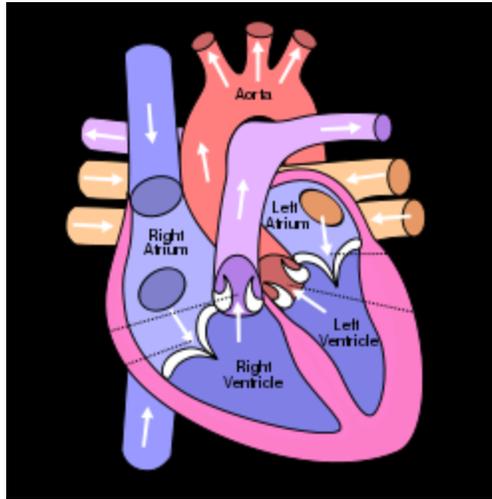


Troponin is a complex of proteins required for contraction of skeletal and cardiac muscle, but not smooth muscle like walls of the blood vessels, certain layers of the aorta, small arteries, arterioles and veins, respiratory tract, and gastrointestinal tract. Troponin is a component of thin filaments (along with actin and tropomyosin), and as previously stated, is the protein to which  $Ca^{2+}$  binds within a cell to regulate the level of  $Ca^{2+}$  and cause the cell to contract.

Troponin has three subcomponents, TnC, TnI, and TnT. When calcium is bound to specific sites on TnC, tropomyosin rolls out of the way of the actin filament active sites, so that myosin (a molecular motor organized in muscle thick filaments) can attach to the thin filament and produce force and/or movement. The different subcomponents of troponin serve different specific functions as follows: Troponin C binds to calcium ions to produce a conformational change in TnI – Troponin T binds to tropomyosin, interlocking them to form a troponin-tropomyosin complex – Troponin I binds to actin in thin myofilaments to hold the troponin-tropomyosin complex in place.

Each subcomponent of troponin is specifically associated with the type of muscle in which it is found, thus allowing specific diagnosis based on the level of the specific troponin subcomponent found in the blood. Troponin I is most specifically associated with heart muscle cells.

(m.) **What is Ventricular Tachycardia?**



The ventricular tachycardia (VT or V-tach) experienced by Jones at 00:30 hours and continuing through 02:58 hours is a potentially life-threatening cardiac arrhythmia that originates in the ventricles (typically the left ventricle), as shown in the above diagram. V-tach is usually a regular, wide complex tachycardia with a rate between 120 and 250 beats per minute. Here, Jones' ECG results from 03:37 hours reflected a very wide QRS and a rate of 220 beats per minute.

Ventricular tachycardia is a common, and often lethal, complication of a myocardial infarction (heart attack). Exercise-induced ventricular tachycardia is a phenomenon related to sudden deaths, especially in patients with severe heart disease (ischemia, acquired valvular heart and congenital heart disease) accompanied with left ventricular dysfunction. Ventricular tachycardia normally lasts for only a few seconds to minutes (*paroxysmal tachycardia*), but if V-tach persists it is extremely dangerous, often leading to the more serious condition known as ventricular fibrillation, as it did in Jones' case.

(n.) **What is Ventricular Fibrillation?**

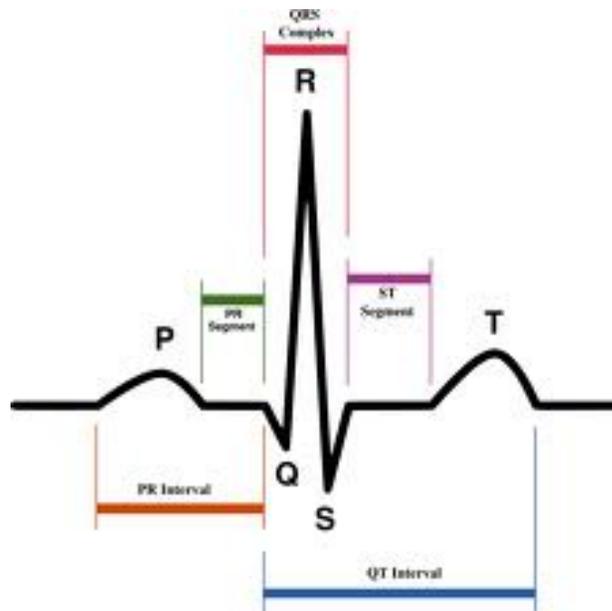
Ventricular fibrillation ("V-fib") describes a particular type of cardiac dysrhythmia (abnormal electrical activity in the heart) in which the muscle cells contract and relax in an uncoordinated manner, thus making them quiver or twitch rather than properly contract and relax. Without properly coordinated contraction and relaxation of the heart's ventricular cells, blood cannot flow either to the lungs through the right ventricle or to the rest of the body through the left ventricle.

Jones' medical records reflect that he virtually skipped the V-fib stage and moved from V-tach into an asystole condition, also known as flat-lining where there is no electrical conduction at all from the heart muscle cells. However, approximately fifteen (15) minutes into Jones' nearly two (2) hour, final code blue event, Jones' heart did restart, but experienced V-fib at 05:25 hours, then again experienced V-fib during the final five (5) minute period during which the UAMS medical staff tried to get Jones' heart restarted.

(o.) **What Are the Effects of Hyperkalemia?**

During extreme exercise,  $K^+$  is released from active muscle cells and the serum  $K^+$  raises to a point that would be dangerous if the body was at rest. Fortunately for athletes and active individuals, correspondingly high levels of other compounds, such as adrenaline and noradrenaline, also released during exercise seem to have a prophylactic effect in relation to cardiac electrophysiology.

(i.) **Effect on the Heart – Electrocardiograph Indications Associated with Excessive K<sup>+</sup>**



With mild to moderate hyperkalemia, there is reduction of the size of the P wave and development of peaked T waves, as seen in Jones' ECG at 01:00 hours. Severe hyperkalemia results in a widening of the QRS complex, and the ECG complex can evolve to a sinusoidal shape, as seen in Jones' ECG at 03:00 and 03:30 hours.

Hyperkalemia appears to have a direct effect on some of the K<sup>+</sup> channels which increases their activity and speeds membrane repolarization. Additionally, hyperkalemia causes an overall membrane depolarization that inactivates many Na<sup>+</sup> channels. The faster repolarization of the cardiac action potential causes the tenting of the T waves, and the inactivation of Na<sup>+</sup> channels causes a sluggish conduction of the electrical wave around the heart, which leads to smaller P waves and widening of the QRS complex. In other words, the K<sup>+</sup> and Na<sup>+</sup> is no longer moving in and out of the ventricular cardiac muscle cells at the proper rhythmic rate, so as to cooperate with the other muscle cells. Instead, the ventricular muscle cells begin to fire faster and faster (ventricular tachycardia) until they no longer fully fire, but just partially fire in a disorganized manner resulting in the fluttering effect associated with ventricular fibrillation. Both V-tach and V-fib are associated with remarkably lower blood pressure.

The serum K<sup>+</sup> concentration at which electrocardiographic changes develop is somewhat variable. Although the factors influencing the effect of serum K<sup>+</sup> levels on cardiac electrophysiology are not perfectly understood, the concentrations of other electrolytes, as well as levels of catecholamines, are known to play a major role. When arrhythmias occur or when K<sup>+</sup> levels exceed 6.5 mmol/L, as did Jones' from almost the end of the Bout until his death, emergency lowering of K<sup>+</sup> level is mandated.

From the time Jones' rhabdomyolysis was triggered during the match, his K<sup>+</sup> level began to rise. If extracellular K<sup>+</sup> levels are too high, then not enough Na<sup>+</sup> is pumped out of the cell. If not enough Na<sup>+</sup> is pumped out of the cell, then the raised intracellular Na<sup>+</sup> levels inhibit the Ca<sup>2+</sup>/Na<sup>+</sup> Pump and not enough Ca<sup>2+</sup> is pulled out. Ca<sup>2+</sup> then begins to build up inside the cell. Increased intracellular Ca<sup>2+</sup> concentrations cause increased Ca<sup>2+</sup> uptake into the sarcoplasmic reticulum (SR) via the SERCA2 transporter. Raised Ca<sup>2+</sup> stores in the SR allow for greater Ca<sup>2+</sup> release upon stimulation, so the heart muscle cell achieves faster and more powerful contraction by cross-bridge cycling. The refractory period of the AV node is also increased with higher intracellular Ca<sup>2+</sup> stores. If SR Ca<sup>2+</sup> stores become too high, some Ca<sup>2+</sup> ions are released spontaneously through SR ryanodine receptors,

which then leads to “after-depolarization” (depolarization of the cell at the wrong time), which leads initially to bigeminy (an irregular heartbeat occurring every other concurrent beat) then to tachycardia, as it did for Jones.

Jones’ progressively worsening cardiac dysrhythmia due to hyperkalemia onset by rhabdomyolysis can be seen by comparing Jones’ blood test results from 22:45 hours on 29-JAN-2011 to those at 00:49 hours on 30-JAN-2011 along with the notation of ECG readings showing sinus tachycardia at 01:00 hours. Then comparing the subsequent blood test results to the previous noting the two notations of ECG readings indicating ventricular tachycardia at 03:30 hours and 03:41 hours, followed by Jones’ experiencing ventricular fibrillation and cardiac arrest beginning at 05:10 hours and lasting until his death at 06:42 hours.

Additionally, Jones’ heart cells experienced a decreased internal  $K^+$  concentration and increased external  $K^+$  concentration (hyperkalemia), norepinephrine release and acidosis resulting from the rhabdomyolysis and renal failure. When Jones’ myocardial cells were exposed to the hyperkalemia, the maximum diastolic potential was depolarized as a result of the alteration of  $I_{K1}$   $K^+$  current, the intensity and direction of which is strictly dependant on intracellular and extracellular  $K^+$  concentrations. With  $I_{K1}$  suppressed, a hyperpolarizing effect is lost thereby activating a “funny current” even in myocardial cells. The “funny current” of only a few myocardial cells is normally suppressed by the hyperpolarizing effect of coexisting  $K^+$  currents; however, the imbalance of  $K^+$  and  $Ca^{2+}$  in Jones’ body was so great as to effect a greater number of cells in Jones’ left ventricle than could be regulated or overcome by the surrounding properly functioning cells.

While it is possible Jones’ concussion could have interfered with the regulation of his heart rate, the mildness of the concussion and the automaticity of the affected cardiac cells makes it highly unlikely Jones’ concussion caused an irregularity great enough to cause Jones’ cardiac arrest. Further, if Jones’ concussion was responsible for the cardiac dysrhythmias seen in Jones’ later tests, the dysrhythmias would have been visible much earlier including by the cardiac monitors to which Jones was hooked when transported from the Ring and while at Saline Memorial and en route to UAMS.

**(p.) What Were the Sources & Amounts of Jones’ Lethal Potassium ( $K^+$ ) Levels?**

Eliminating any doubt as to its lethal effect, it is noted here that  $K^+Cl^-$  is the lethal chemical administered by death penalty states using lethal injection. Typically, 100 milliequivalents (mEq)  $K^+Cl^-$  is used in lethal injections and the generally accepted, standard euthanasia calculation for  $K^+Cl^-$  is between 1 and 2 mmol/kg; thus, since 1 mmol  $K^+Cl^-$  = 1 mEq  $K^+Cl^-$  and Jones weighed 232.5 lbs. (105 kg), a fatal dose of  $K^+Cl^-$  for Jones would be between 105 and 210 mEq  $K^+Cl^-$ . Then, using the following formula for compounds, milliequivalent (mEq) = Total Atomic Weight/(Lowest Elemental Valence x 1000) for the compound  $K^+Cl^-$ , we find that one mEq of  $K^+Cl^-$  contains  $K^+$  = 39.0983 mg. and  $Cl^-$  = 35.4532 mg.

The following chart provides a reference for converting milliequivalents to milligrams:

CATIONS		ANIONS	
Milliequivalents	Milligrams	Milliequivalents	Milligrams
1 mEq Potassium ( $K^+$ )	39 mg	1 mEq Chloride ( $Cl^-$ )	35.5 mg
1 mEq Sodium( $Na^+$ )	23 mg	1 mEq Bicarbonate ( $HCO_3^-$ )	61 mg
1 mEq Calcium( $Ca^{2+}$ )	20 mg	1 mEq Phosphate ( $PO_4^{3-}$ )	31.67 mg
1 mEq Magnesium ( $Mg^{2+}$ )	12.2 mg		

Therefore, the fatal  $K^+$  dosage range for Jones was roughly between 4,105 and 8,210 mg  $K^+$ , although such is a very rough calculation and depends on multiple other factors including the rapidity of administration and proper functioning of Jones’ other systems. The faster the  $K^+$  level rise, the more lethal the dose becomes and the

less  $K^+$  it takes to become fatal because the body does not have sufficient time to regulate the level through both intracellular regulation (storage inside the cells) and extracellular regulation (filtration and excretion via the renal system).

In the twenty-four (24) hour period immediately preceding his Bout as detailed in the timeline in Sections III B & C of this report, Jones consumed a minimum of 4,507 mg of  $K^+$  through dietary intake (1,771 mg at dinner the night before, 2,175 mg at breakfast the morning of, and 561 mg via a medium size banana in the hours prior to the Bout). Notwithstanding the fact that Jones had already consumed enough  $K^+$  to replace all amounts he might lose during the Bout, Jones also supplemented his  $K^+$  intake with the 90 mg. supplement pill he obtained from his roommate at noon the day of his Bout, thus bringing Jones' total minimum  $K^+$  intake to 4,597 mg (or 118 mEq), very near the recommended daily intake for the twenty-four (24) hour period prior to his Bout.

Although the Commission was unable to obtain evidence of such and therefore did not take such into consideration for purposes of its findings and determinations, the Commission believes it is very likely Jones also consumed an additional 250 to 500 mg or more of  $K^+$  within the twenty-four (24) hour period prior to his Bout via consumption of additional food and/or multiple servings of the Creatine by GNC supplement (contains 80 mg  $K^+$  per serving) between his breakfast at 09:20 hours and his light workout at 16:45 hours, as well as by consuming at least one multi-vitamin (typically containing at least 50 mg  $K^+$ ) the day of the Bout.

$K^+$  salts are available and consumed as dietary supplements in tablets or capsules, which for therapeutic purposes are formulated to allow  $K^+$  to leach slowly out of a matrix, since very high concentrations of  $K^+$  (which might occur next to a solid tablet of  $K^+Cl^-$ ) can kill tissue and cause injury to the gastric or intestinal mucosa. For this reason, non-prescription  $K^+$  supplement pills are limited by law in the U.S. to only 99 mg of  $K^+$ . Excess intake of  $K^+$  is possible via salt-substitute,  $K^+$  containing dietary supplements, or  $K^+Cl^-$  infusion. However, for an otherwise healthy person with normal kidney function and no other conditions interfering with normal elimination, hyperkalemia via intake of  $K^+$  would be possible only with large intravenous infusions of  $K^+Cl^-$  or oral doses of several hundred milliequivalents of  $K^+Cl^-$ .

Although Jones' total minimum  $K^+$  intake to 4,597 mg (or 118 mEq) exceeds the lower level of the previously calculated lethal dose, it is important here to remember our earlier discussion of the body's safeguards and mechanisms for rapid control of extracellular  $K^+$  levels during food intake, as well as, our previous calculations for regulation by the renal system with a normal GFR. Under normal circumstances, Jones' body would have been able to filter and process approximately 1,056 mg to 1,408 mg of potassium ( $K^+$ ) per hour and Jones' total, known  $K^+$  intake of 4,597 mg (or 118 mEq) was over about a twenty-four (24) hour period. Accordingly, the amount of  $K^+$  Jones consumed through food and potassium supplements the day of his Bout should not have normally been a problem for his body to process and maintain a proper  $K^+$  balance. Unfortunately, Jones was not in a normal condition, as he experienced rhabdomyolysis and dehydration, which raising his body's  $K^+$  to toxic levels, then created the fatal feedback loop of electrolyte imbalance via renal failure and cardiac dysrhythmia described in the other subsections of this report.

Based upon the damage markers in Jones' blood test results beginning as early as 22:45 hours including, elevated CPK, SGOT (AST), SGPT (ALT), observation of Jones' actions during the Bout, and Jones' blood test results at 04:00 hours including, a myoglobin reading of 1,079 ng/mL, it is the Commission's opinion Jones' suffered from exertional rhabdomyolysis, which initially released toxic levels of both myoglobin and  $K^+$  into his blood within a period of less than thirty (30) minutes beginning with the start of the Bout and continued thereafter to release increasingly toxic levels of the same over the several hour period following the end of the Bout.

The initial shock of the rapid, hyper-elevation of  $K^+$  caused Jones' heart muscle cells to begin to experience tachycardic side effects; meanwhile, the rapid and hyper-elevated levels of myoglobin released into Jones' blood very quickly caused acute renal failure via renal tubule necrosis. The concomitant renal dysfunction and eventual

failure only exacerbated the hyper-elevated  $K^+$  levels and effects thereof, which eventually caused the heart to suffer ventricular fibrillation, ventricular systole and death. The volume of  $K^+$  initially released, when coupled with the renal dysfunction and failure soon thereafter, as well as the continued release of  $K^+$  was sufficient to provide a dose of potassium ( $K^+$ ) far in excess of the predicted lethal range calculated in the above paragraphs.

We know from our earlier calculations that Jones likely had approximately 3,990 mEq (156,002 mg) potassium ( $K^+$ ) stored in his muscle cells and that if Jones had a normal GFR, his body would have only been able to filter and process approximately 1,056 mg to 1,408 mg of potassium ( $K^+$ ) per hour. Thus, the damage or destruction of just two and a half percent (2.5%) of Jones' muscle cells under extreme exertion during the Bout would have resulted in the release of a lethal dose of potassium ( $K^+$ ). While it is unlikely, Jones received damage to two and half percent (2.5%) or more of his total muscle cells during the Bout, Jones' dehydrated state and reduced GFR associated with his renal dysfunction caused the eventual fatal concentration of potassium ( $K^+$ ) in Jones' blood.

### **3. Secondary/Contributing Cause of Death – Exertional Rhabdomyolysis**

Rhabdomyolysis is the breakdown or destruction of muscle cells which releases the cells' contents, myoglobin and  $K^+$ , into the bloodstream and causes the storage of  $Na^+$  and  $Ca^{2+}$ . While statistics vary from source to source and not all cases of rhabdomyolysis are actually reported, it is generally accepted that rhabdomyolysis accounts for eight percent (8%) to fifteen percent (15%) of the annual cases of acute renal failure. Springhouse. *Professional Guide to Diseases (8<sup>th</sup> Ed.)*. Lippincott, Williams & Wilkins (2005). Rhabdomyolysis is not all that rare and virtually all male athletes and a majority of all other males have experienced some level of rhabdomyolysis at least once in their life, if not on an almost weekly or monthly basis.

A 23-MAR-2011 article by the Associated Press reported that in January of 2011, thirteen (13) football players at the University of Iowa were diagnosed with rhabdomyolysis following an intense workout session, which included extended, explosive muscle group use exercises such as the athletes as quickly as possible performing 100 deep knee squats using half of the athletes' maximum weight.

A five-member university committee cleared the players, coaches and staff of any neglect or improper conduct. The university's committee reported the reason rhabdomyolysis occurred after the execution of the drills and not during or subsequent to any of the previous three (3) times the same workout had been used in 2007, 2004 and 2000 could not be determined by their investigation. The university committee further listed 10 recommendations, including the abandonment of the strenuous workout program. Another of the committee's recommendations was requiring athletes to be educated about rhabdomyolysis, which is a primary goal of this Commission's report here in the Jones case.

Go to <http://www.dailyiowan.com/2011/03/24/Photo/report.pdf> for a copy of the University of Iowa's Board of Regents' Report.

Additionally, the steadily increasing and prevalent prescription of statin drugs for use in controlling cholesterol levels has increased the importance for additional education and precautions for those persons being prescribed and taking statins while engaging in any type of athletic or physical activity. Due to the specific biochemical pathways and biomechanical methods utilized by statins (namely their effects on coenzyme  $Q_{10}$  and Cytochrome C), one of the rare, but potentially lethal side effects is rhabdomyolysis. In fact, a well known statin drug marketed under the brand name Baycol was very quickly pulled off of the market in 2001 after thirty-one people died from rhabdomyolysis after starting treatment with Baycol.

**(a.) What Are the Different Types of Rhabdomyolysis – Compressional vs. Exertional?**

Rhabdomyolysis comes in two forms, compressional and exertional. Compressional rhabdomyolysis occurs when muscle cells are exposed to extraordinary compression forces, which either destroy the cells by ischemia (restriction of blood flow to the cells) for a sufficient amount of time to induce cell death by anoxia (complete lack of oxygen) or by actually physically crushing or destroying the cells. One of the accidental causes of renal failure is crush syndrome, which is another name for a certain form of compressional rhabdomyolysis. Crush syndrome and certain forms of compressional rhabdomyolysis are reperfusion injuries that appear after the release of the crushing pressure or compressive impact. Large volumes of toxins are suddenly released into the blood when a long compressed limb is suddenly relieved from pressure which was causing ischemia by obstructing blood flow through the limb's tissues. The specific effect on the kidneys is not completely understood; however, renal failure does result in part from the nephrotoxic metabolites of myoglobin, primarily ferriheme (Mb<sup>+4</sup>). Upon release of the crushing pressure, the instant spike of potassium (K<sup>+</sup>), myoglobin and phosphorus leads to clogging and destruction of the kidneys' infrastructure, namely the epithelial cells of the renal tubules in the nephrons of the kidneys.

Exertional rhabdomyolysis, on the other hand, often occurs as a natural result of the processes associated with the conditions of hypoxia (very reduced oxygen levels) or anoxia (complete lack of oxygen) induced by extremely high muscle use and inadequate oxygenation of the blood during intense physical activity, such as Jones' heavyweight boxing Bout. Since Jones only received a total of three (3) body shots during the entire Bout, we can safely rule out compressional rhabdomyolysis. Instead, exertional rhabdomyolysis is the culprit here and will be the focus of this section of the report.

In order to fully understand the cause of Jones' exertional rhabdomyolysis, we must first understand several physiological and biochemical principals involved during periods of extreme physical exertion. First, we must understand the interplay between the level of physical activity and the two (2) ways cells produce the energy they need, including the side effects of such energy production. Second, we must understand the differences and interplay between the three (3) types of skeletal muscle cells and the two (2) types of contraction forces skeletal muscle cells are exposed to during periods of intense physical activity. Combining our understanding of the aforementioned processes, we can easily see how the perfect storm was created inside Jones' body and resulted in his death.

**(b.) What Causes Exertional Rhabdomyolysis – Aerobic vs. Anaerobic Energy Production?**

Muscle cell death resulting from exertional rhabdomyolysis is effectuated primarily through the body's natural processes associated with strenuous exertion or overuse of the muscle cells.

The energy required by cells is produced by the body's conversion of chemical energy stored in food (glucose) to high energy phosphate bonds in adenosine triphosphate ("ATP"). The high energy bonds of ATP are used in a number of biochemical reactions as an energy source for the cell. For example, ATP is converted to adenosine diphosphate ("ADP") by an energy releasing process which removes a phosphate group from the ATP molecule. The ADP is stored inside the cell for later use. Also, excess ATP is used during periods of low energy demand to convert creatine into phosphocreatine ("PCr"), which is stored for use in period of instant energy demand. The conversion and processing of ATP and ADP is constantly performed in the mitochondria of human cells via aerobic respiration.

If ADP levels inside the muscle cells become too high or the cells require a short, intense burst of energy, such as the intense energy required by Jones's muscle cells during his Bout with Palmer, then the body uses an enzyme induced fermentation process to break down phosphocreatine ("PCr"). The breakdown of PCr anaerobically produces the phosphate necessary to convert ADP back into ATP for the cell's use in satisfying

energy demand. The body's stores of PCr are extremely limited and can only support a muscle cell's ATP levels for two (2) to ten (10) seconds in the absence of any other sources of ATP. However, since ATP is also available from other sources, PCr ends up being a major energy source in the first minute or so of strenuous physical activity. Cells heavily rely upon PCr during intense bursts of muscle use because, unlike other resources used for ATP production, PCr is the most readily available resource due to its localized storage inside the cell. PCr is quickly accessed and used to restore and maintain ATP levels during intense exercises which require explosive bursts of muscle use such as sprinting, jumping, lifting, and throwing.

(i) **Aerobic Respiration (Energy Production)**

Human cells' default or preferred method of producing the energy they need is through the very efficient aerobic metabolic process known as aerobic respiration. Muscle cells are highly adaptive to fluctuations in blood oxygen levels and varying energy demands, thus utilize both aerobic and anaerobic respiration with aerobic respiration being the default method of energy production.

On the other hand, cardiac cells in particular, while classified as muscle cells, are so highly specialized with large numbers of mitochondria (required for aerobic respiration) and dense concentrations of myoglobin (oxygen storing pigments) that they are almost totally dependent on aerobic metabolism for energy production. R. Matini, *Fundamentals of Anatomy & Physiology (5<sup>th</sup> Ed.)*. Prentice-Hall, Inc. (2000). The intense reliance upon aerobic respiration makes heart muscle cells very susceptible to damage or death from decreased blood oxygen levels. At resting rates, heart muscle cells derive only about one percent (1%) of their energy from anaerobic metabolism and can increase their anaerobic energy production to around ten percent (10%) as blood oxygen levels decrease, but simply cannot produce enough energy from anaerobic metabolism (lactate production) to sustain ventricular contractions under severely hypoxic (oxygen deficient) or anoxic (absence of oxygen) conditions.

A very good explanation of the way muscle cells create and use various energy sources during different types of exercise was provided as part six (6) of the Physiology of Nutrition Lecture Series given by Dr. A. Scott Connelly and William H. Carpenter, M.S. at the UCLA Center for Human Nutrition and explains the process as follows (emphasis was added by the Commission):

With moderate exertion, a carbohydrate undergoes aerobic metabolism. Under these conditions, oxygen is used and the carbohydrate goes through both the Embden-Meyerhoff pathway of anaerobic metabolism in which glucose is converted to lactate, but, prior to the conversion of pyruvate to lactate, pyruvate enters the Krebs Cycle in mitochondria where oxidative phosphorylation results in a maximum extraction of energy from each molecule of glucose. If there is plenty of oxygen available and the exercise is of low to moderate intensity, then the pyruvate from glucose is converted to carbon dioxide and water in the mitochondria. Approximately 42 ATP equivalents can be produced from a single glucose molecule compared to only 4 ATP with anaerobic metabolism.

***Aerobic metabolism supplies energy more slowly than anaerobic metabolism, but can be sustained for long periods of time up to 5 hours.*** The major advantage of the less efficient anaerobic pathway is that it more rapidly provides ATP in muscle by utilizing local muscle glycogen. Other than PCr, it is the fastest way to resupply muscle ATP levels. ***Anaerobic glycolysis supplies most energy for short term intense exercise ranging from 30 seconds to 2 minutes.***

The disadvantages of anaerobic metabolism are that it cannot be sustained for long periods, since the accumulation of lactic acid in muscle decreases the pH and

inactivates key enzymes in the glycolysis pathway leading to fatigue. The lactic acid released from muscle can be taken up by the liver and converted to glucose again (Cori Cycle), or it can be used as a fuel by the cardiac muscle directly or by less active skeletal muscles away from the actively contracting muscle. ***Muscle glycogen is the preferred carbohydrate fuel for events lasting less than 2 hours for both aerobic and anaerobic metabolism.*** Depletion of muscle glycogen causes fatigue and is associated with a build-up of muscle lactate. Lactate production increases continuously but physiologists have defined a point at which breathing changes as a result of acid-base imbalance called the anaerobic threshold.

Both the nutrition and conditioning of the athlete will determine how much work can be performed in a specific exercise before fatigue sets in. It is possible to build up glycogen stores prior to exercise to improve performance. ***With exercises lasting for more than 20 to 30 minutes, blood glucose becomes important as a fuel to spare muscle glycogen breakdown.*** Both aerobic and endurance training lead to increases in glycogen stores, triglycerides, oxidative enzymes, and increased number and size of mitochondria. Both the oxidative enzymes involved in the Krebs cycle oxidation of glucose and the lipoprotein lipase needed to convert triglycerides to fatty acids are increased through training. ***This is not a general effect, but is specific to the muscle and muscle fiber type being used for the exercise.***

***Slow twitch muscle fibers provide for prolonged aerobic activity, while the fast-twitch muscle fibers are used for short intense activities. The fatigue that develops with intense exercise can be related to specific fiber types. In prolonged exercise at 60 to 75 percent of  $VO_2$ max Type I fibers (red, slow twitch) and Type IIa (red, fast twitch) are recruited during the early stages of exercise, but as the intensity increases Type IIb fibers (white, fast twitch) must be recruited to maintain the same intensity. It requires more mental effort to recruit Type IIb fibers and they produce lactic acid.*** As the glycogen levels drop in the red muscle fibers, they will rely more on fat. Since fat is less efficient than carbohydrate, intensity will decrease (pace will slow).

At the other end of the spectrum, during mild exercise such as a brisk walk muscles burn fat for fuel because the supply of ATP provided from fat is adequate to maintain intensity. . . . [F]atty acids are readily available from stored fat and the rate of lipolysis is three times the rate of fatty acid release at rest so that fatty acids can be supplied at an increased rate rapidly during the onset of low levels of exercise. ***So while fat is not very useful for short term, intense exercise, it is a great advantage for increasingly prolonged exercise especially when it is maintained at a low or moderate level of intensity.*** The advantage of fat as a fuel is that it provides extensive stores of calories in an easily portable form. Since fat is not hydrated it weighs much less per unit calorie than protein or carbohydrate (9 Cal/gm of fat vs. 4 Cal/gm of carbohydrate or protein). When you compare the number of ATP produced per carbon atom, fat is also more efficient.

A 6-carbon glucose molecule produces 36 to 38 ATP on average providing a ratio of 6 ATP/Carbon, while an 18 carbon fatty acid produces 147 ATP providing a ratio of 8.2 ATP/Carbon. However, carbohydrate is more efficient than fat when the amount of ATP produced per unit of oxygen consumed is considered. Six oxygen molecules are required to metabolize six-carbon glucose producing 36 ATP (ratio = 6 ATP/oxygen

molecule), while 26 oxygen molecules are required to produce 147 ATP from an 18 carbon fatty acid (5.7 ATP/oxygen molecule).

***Therefore, for a performance athlete it is important to maintain the efficiency edge provided by carbohydrate as long as glycogen is available in the muscles. Under usual exercise conditions, protein only provides about 6% of energy needs. With high intensity endurance exercise, the production of glucose from amino acids can be significant up to about 10 or 15% of total energy needs. The only food that provides energy for short-term fast-paced exercise is carbohydrate, while slow steady aerobic exercise uses all three primary fuels but primarily fat and carbohydrate.***

As explained, during aerobic metabolism, the cells use oxygen in the energy production process to create and convert the cell's primary energy source, adenosine triphosphate ("ATP"). The aerobic metabolic process can generate a net gain of up to 30 units of ATP for each molecule of glucose. However, to achieve such a highly efficient energy production rate, the aerobic respiration process requires oxygen from the blood, a primary component which is quickly in short supply in the bloodstream during intense or prolonged muscle cell use coupled with inadequate breathing techniques.

If insufficient oxygen is available for the cell to engage in aerobic respiration, then the cell can no longer satisfy the cell's energy needs through aerobic respiration. This creates a huge problem for a human (actually for any living, breathing animal), since the person would not survive very long if all of the cells in their body (or a substantial number of them) shut down and ceased to function every time blood oxygen levels dropped during periods of heightened exertion, such as running away from environmental threats or fighting off a threat. Imagine being faced with a threat or simply trying to complete a task involving average or above average physical exertion, then a few minutes after you start running away from the threat or begin the strenuous activity, you begin breathing a little harder, instantly feel very tired, fall down and die because all of your body's cells just immediately stopped working when your blood oxygen level decreased and the cells could consequently no longer keep pace with their energy needs through aerobic respiration. Without a backup plan for energy production without the use of oxygen, a human body would be very inefficient and not capable of actually performing much work or engaging in physical activities.

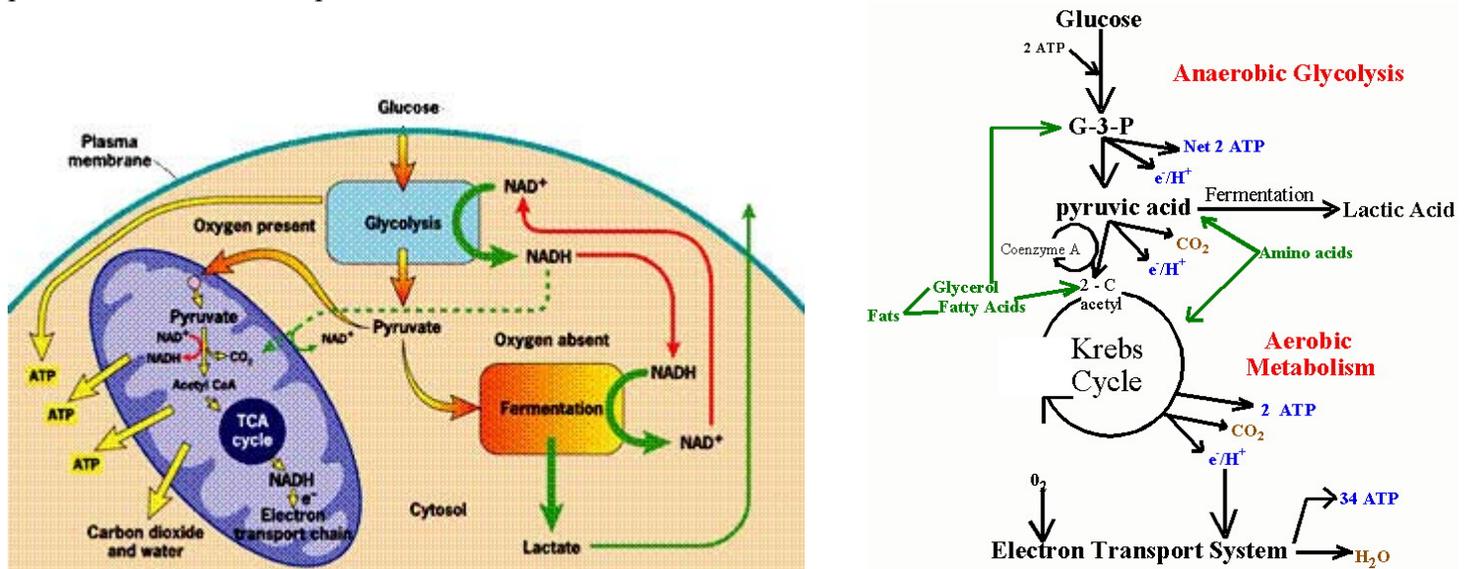
To prevent a full body shutdown from lack of cellular energy, the body's natural response to decreased blood oxygen levels and a continued need for energy is to initiate its "backup plan." The "backup plan" involves a complicated and potentially lethal process called anaerobic respiration which amazingly continues energy production by producing and converting ATP without using any oxygen. While potentially lethal, the body has substantial built-in buffers and safeguards to protect itself from the side effects of anaerobic respiration. Anaerobic respiration only becomes lethal when anaerobic respiration continues for such an extremely long period of time that it overwhelms the body's ability to compensate and protect itself or when other unusual conditions exist within the body, which impair the body's ability to buffer and safeguard against anaerobic respiration's toxic side effects.

Every athlete's body shifts into anaerobic energy production at some point during every workout in which their breathing and heart rate are elevated for more than a few minutes. Here, although the Commission has absolutely no way to confirm its hypothesis, based upon Jones' known pre-Bout warm-up exercises, extraordinarily high pace during the Bout, lack of controlled breathing, extraordinary muscle mass, and types of muscle cells used during the Bout, the Commission suspects the vast majority of Jones' muscle cells had converted to anaerobic respiration within the 1<sup>st</sup> minute of the 1<sup>st</sup> Round.

It is worth noting the Commission also believes it is very likely the muscle cells inside Jones' opponent, Palmer, had also undergone converted to anaerobic energy production around the same time as Jones, but Palmer's buffering systems were obviously sufficient to protect Palmer from the side effects of anaerobic respiration.

**(ii.) Anaerobic Respiration (Energy Production)**

While aerobic respiration is nineteen (19) times more efficient than anaerobic respiration, the body's production of some energy is better than none at all during periods of reduced blood oxygen levels. During anaerobic metabolism, the initial pathway of glycolysis (conversion of glucose into pyruvate (pyruvic acid) resulting in free energy being released in the form of ATP and NADH) is shared; however, in the absence of oxygen, the pyruvate is not metabolized in the mitochondria via the normal process of aerobic metabolism. Instead, the pyruvate undergoes conversion via fermentation. During fermentation, the pyruvate is not transported into the cell's mitochondrion, but remains in the cell's cytoplasm where it is converted into waste products, primarily lactate (lactic acid) which is later dispersed into the blood stream. The diagrams below demonstrate the process of anaerobic respiration.



During the first phase of the anaerobic metabolic process, glucose first enters the cell and is converted to pyruvate (a.k.a. pyruvic acid) in the cell's cytoplasm (main cellular compartment). The pyruvate is then converted through anaerobic metabolism using the lactate dehydrogenase enzyme to form lactate in the cell's cytoplasm, which is then used to breakdown the glucose for energy. While these two anaerobic metabolic processes release only a small amount of ATP and corresponding energy, neither requires oxygen, thus making them invaluable during periods of extreme or extended exertion or when oxygen in the blood is otherwise insufficient for aerobic respiration.

As mentioned in the quote from the Physiology of Nutrition Lecture Series, muscle cells can continue high rates of anaerobic energy production for only one (1) to three (3) minutes with the downside to such continuous anaerobic energy production being a buildup of very high levels of lactate within the cells which then forms lactic acid when it is released into the blood stream. Fortunately, because heart muscle cells are highly specialized to be resistant to fatigue, they are able to directly use lactate as an alternative fuel source, which becomes especially important during times of extreme exercise. J.C. Chatham, (2002), "Lactate - the forgotten fuel!" *J Physiol*. July 15; 542(Pt 2), p. 333. Excessive amounts of lactic acid begin to build in muscle tissue when the level of lactate exceeds both the oxidative capacity of the body's tissues and ability of the liver to convert the lactate back into glucose.

The process of generating energy from ATP happens in two steps: First, ATP is converted to ADP (adenosine diphosphate), then converted to AMP (adenosine monophosphate). ADP can be converted back to ATP with the help of the creatine kinase enzyme. When excess amounts of AMP accumulate in the muscle cell, the cell responds through the increased intake of glucose, which soon depletes the supply of glucose in the blood, unless the liver is able to increase glucose production to a level matching the cell's increased consumption.

In order for the liver to generate more glucose, it needs a substrate, which in the short term is provided by the self-cannibalization of muscle cells. During brief periods of starvation, human muscle cells adapt quickly by breaking down muscle proteins and converting them into a basic amino acid, alanine. Muscles then rely on a novel mechanism that involves an exchange system with the liver, the so-called glucose-alanine cycle. The alanine, derived from muscle protein, is released into the blood and shipped to the liver to be utilized for energy generation. The liver can then generate more glucose from the alanine through gluconeogenesis, while exporting the waste product, urea, to the kidneys for excretion. This also allows the liver to regenerate some ATP to help satisfy its own energy needs, which are very large during such stressful conditions. The glucose is shipped through the blood stream to the muscle cell, which eagerly takes it up to generate more ATP for itself.

The anaerobic processing of glucose yields pyruvate which can also be turned into alanine, but the process needs yet another enzyme to work. Thus, whenever insufficient oxygen levels prevent pyruvate from being processed in the mitochondria, pyruvate is instead be converted to alanine with the help of the enzyme, ALT (alanine aminotransferase), provided there is an adequate supply of glutamate.

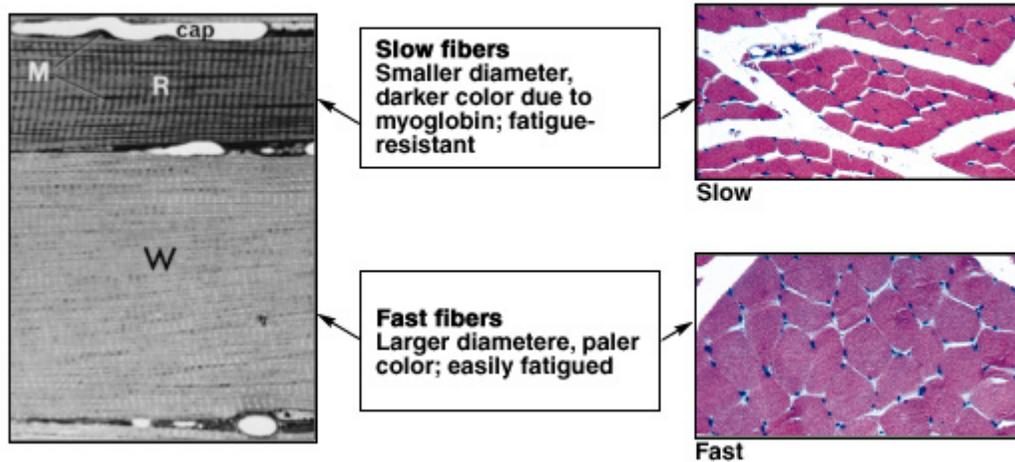
Scientists have identified several enzymes required for anaerobic cytoplasmic energy-generating processes to function. These anaerobic specific enzymes include creatine kinase, lactate dehydrogenase, and ALT; therefore, tests measuring the concentration of ALT, creatine kinase and lactate dehydrogenase concentrations in the blood will indicate whether muscle cells are preferentially processing glucose anaerobically in the cytoplasm rather than aerobically in the mitochondria. Here, at 22:45 hours only an hour after the end of the Bout, Jones' had an elevated SGPT (ALT) result of 71, elevated CPK result of 1463 and elevated SGOT (AST), all indications Jones' body was still preferentially processing glucose using much less efficient anaerobic metabolism.

Unfortunately for Jones, his muscle cells' extreme spike in energy demand during the 1<sup>st</sup> Round of the Bout took place during the perfect storm of unfavorable conditions. As is discussed in the following sections of this report, 80% of Jones' hepatocytes (primary functioning structures within the liver) revealed both macro and micro steatosis. Steatosis describes the abnormal retention of lipids/fats within a cell, also known as adipose degeneration. Steatosis interferes with the liver's ability to convert lactate or lactic acid back into glucose and generate glycogen for quick use in times of high energy demand. Due to the steatosis, Jones' liver was unable to either lower the level of lactic acid, thus permitting the lactic acidosis to become more severe. Additionally, the steatosis within Jones' liver impaired its ability to efficiently participate in the glucose-alanine cycle or otherwise sufficiently increase glucose production and release its most likely diminished glycogen stores at the rate required by Jones' muscle cells. Therefore, in addition to the numerous other factors contributing to Jones' rhabdomyolysis, Jones' muscle cells most likely died and released their contents when they not only ran out of oxygen, but also ran out of the glucose and glycogen necessary to produce the fuel (ATP) required for their continued functions.

As will also be discussed in greater detail later in this report, the steatosis in Jones' liver was very likely the result of multiple causes, not the least of which was his history of alcohol and steroid abuse.

(c) **What Are the Three (3) Types of Skeletal Muscle Fibers and How Do They Relate to Physical Activity and Aerobic and Anaerobic Energy Production?**

Muscle cells cluster together to form three distinct types of muscle fiber strands: Fast fibers, Slow fibers, and Intermediate fibers. The pictures below show the differences at the microscopic level.



The differences between and uses of the three types of muscle fibers is very well explained in chapter 10 of the Fundamentals of Anatomy & Physiology, 5<sup>th</sup> Ed. and is summarized in the following extensive quote. R. Matini, Fundamentals of Anatomy & Physiology (5<sup>th</sup> Ed.). Prentice-Hall, Inc. (2000).

Muscle performance can be considered in terms of **power**, the maximum amount of tension produced by a particular muscle or muscle group, and **endurance**, the amount of time for which the individual can perform a particular activity. Two major factors determine the performance capabilities of any skeletal muscle: (1) the types of muscle fibers in the muscle and (2) physical conditioning or training.

### Types of Skeletal Muscle Fibers

The human body has three major types of skeletal muscle fibers: *fast fibers*, *slow fibers*, and *intermediate fibers*.

(i) **Fast Fibers**

**Fast Fibers** Most of the skeletal muscle fibers in the body are called **fast fibers**, because they can contract in 0.01 sec or less after stimulation. Fast fibers are large in diameter; they contain densely packed myofibrils, large glycogen reserves, and relatively few mitochondria. The tension produced by a muscle fiber is directly proportional to the number of sarcomeres, so muscles dominated by fast fibers produce powerful contractions. However, fast fibers fatigue rapidly because their contractions use ATP in massive amounts, so prolonged activity is supported primarily by anaerobic metabolism. Several other names are used to refer to these muscle fibers, including *white muscle fibers*, *fast-twitch glycolytic fibers*, and *Type II-A fibers*.

(ii.) **Slow Fibers**

**Slow Fibers** **Slow fibers** are only about half the diameter of fast fibers and take three times as long to contract after stimulation. Slow fibers are specialized to enable them to continue contracting for extended periods, long after a fast muscle would have become fatigued. The most important specializations improve mitochondrial performance. Slow muscle tissue contains a more extensive network of capillaries than is typical of fast muscle tissue and so has a dramatically higher oxygen supply. In addition, slow fibers contain the red pigment myoglobin. This globular protein is structurally related to hemoglobin, the oxygen-carrying pigment in blood. Both myoglobin and hemoglobin are red pigments that reversibly bind oxygen molecules. Although other muscle fiber types contain small amounts of myoglobin, it is most abundant in slow fibers. As a result, resting slow fibers contain substantial oxygen reserves that can be mobilized during a contraction. Because slow fibers have both an extensive capillary supply and a high concentration of myoglobin, skeletal muscles dominated by slow fibers are dark red. They are also known as *red muscle fibers*, *slow-twitch oxidative fibers*, and *Type I fibers*.

With oxygen reserves and a more efficient blood supply, the mitochondria of slow fibers can contribute more ATP during contraction. Thus, slow fibers are less dependent on anaerobic metabolism than are fast fibers. Some of the mitochondrial energy production involves the breakdown of stored lipids rather than glycogen, so glycogen reserves of slow fibers are smaller than those of fast fibers. Slow fibers also contain more mitochondria than do fast fibers. . . .

(iii.) **Intermediate Fibers**

**Intermediate Fibers** The properties of **intermediate fibers** are intermediate between those of fast fibers and slow fibers. In appearance, intermediate fibers most closely resemble fast fibers, for they contain little myoglobin and are relatively pale. They have a more extensive capillary network around them, however, and are more resistant to fatigue than are fast fibers. Intermediate fibers are also known as *fast-twitch oxidative fibers* and *Type II-B fibers*. . . .

In muscles that contain a mixture of fast and intermediate fibers, the proportion can change with physical conditioning. For example, if a muscle is used repeatedly for endurance events, some of the fast fibers will develop the appearance and functional capabilities of intermediate fibers. The muscle as a whole will thus become more resistant to fatigue.

(iv.) **Muscle Performance & the Distribution of Muscle Fibers**

The percentages of fast, intermediate, and slow fibers in a skeletal muscle can be quite variable. Muscles dominated by fast fibers appear pale and are often called **white muscles**. Chicken breasts contain “white meat” because chickens use their wings only for brief intervals, as when fleeing from a predator, and the power for flight comes from fast fibers in their breast muscles. As we learned earlier, the extensive blood vessels and myoglobin in slow fibers give these fibers a reddish color; muscles dominated by slow fibers are therefore known as **red muscles**. Chickens walk around all day, and the movements are performed by slow fibers in “dark meat” of their legs.

Most human muscles contain a mixture of fiber types and so appear pink. However, there are no slow fibers in muscles of the eye or hand, where swift but brief contractions are required. Many back and calf muscles are dominated by slow fibers; these muscles contract almost continuously to maintain an upright posture. The percentage of fast versus slow fibers in each muscle is genetically determined. As we noted earlier, the proportion of intermediate fibers to fast fibers can increase as a result of athletic training.

(v.) **Muscle Hypertrophy**

As a result of repeated, exhaustive stimulation, muscle fibers develop more mitochondria, a higher concentration of glycolytic enzymes, and larger glycogen reserves. Such muscle fibers have more myofibrils than do muscles that are less used, and each myofibril contains more thick and thin filaments. The net effect is hypertrophy, or an enlargement of the stimulated muscle. The number of muscle fibers does not change significantly, but the muscle as a whole enlarges because each muscle fiber increases in diameter. Hypertrophy occurs in muscles that have been repeatedly stimulated to produce near-maximal tension. The intracellular changes that occur increase the amount of tension produced when these muscles contract. A champion weight lifter or bodybuilder is an example of hypertrophied muscular development.

(vi.) **Physical Conditioning & Muscle Cells' Relationship to Aerobic and Anaerobic Energy Production**

Physical conditioning and training schedules enable athletes to improve both power and endurance. In practice, the training schedule varies depending on whether the activity is supported primarily by aerobic or anaerobic energy production.

**Anaerobic endurance** is the length of time muscular contraction can continue to be supported by glycolysis and by the existing energy reserves of ATP and CP. Anaerobic endurance is limited by (1) the amount of ATP and CP on hand, (2) the amount of glycogen available for breakdown, and (3) the ability of the muscle to tolerate the lactic acid generated during the anaerobic period. Typically, the onset of muscle fatigue occurs within 2 minutes of the start of maximal activity.

Activities that require above-average levels of anaerobic endurance include a 50-meter dash or swim, a pole vault, and a weight-lifting competition. These activities involve the contractions of fast fibers. The energy for the first 10–20 seconds of activity comes from the ATP and CP reserves of the cytoplasm. As these reserves dwindle, glycogen breakdown and glycolysis provide additional energy. Athletes training to improve anaerobic endurance perform frequent, brief, intensive workouts that stimulate muscle hypertrophy.

**Aerobic endurance** is the length of time a muscle can continue to contract while supported by mitochondrial activities. Aerobic endurance is determined primarily by the availability of substrates for aerobic respiration, which the muscle fibers can obtain by breaking down carbohydrates, lipids, or amino acids. Initially, many of the nutrients catabolized by the muscle fiber are obtained from reserves in the sarcoplasm. Prolonged aerobic activity, however, must be supported by nutrients provided by the circulating blood.

During exercise, blood vessels in the skeletal muscles dilate, increasing blood flow and thus bringing oxygen and nutrients to the active muscle tissue. Warm-up periods are therefore important not only in that they take advantage of *trappe*, the increase in tension production . . . , but also because they stimulate circulation in the muscles before the serious workout begins. Because glucose is a preferred energy source, aerobic athletes such as marathon runners typically “load” or “bulk up” on carbohydrates for the last three days before an event. They may also consume glucose-rich “sports drinks” during a competition. **[COMMISSION NOTE: The risks and benefits of these practices is examined in a later chapter of the book and are not included in this report.**

Training to improve aerobic endurance generally involves sustained low levels of muscular activity. Examples include jogging, distance swimming, and other exercises that do not require peak tension production. Improvements in aerobic endurance result from altering the characteristics of muscle fibers and improving the performance of the cardiovascular system:

**Altering the characteristics of muscle fibers.** The composition of fast and slow fibers in each muscle is genetically determined, and individual differences are significant. These variations affect aerobic endurance, because a person with more slow fibers in a particular muscle will be better able to perform under aerobic conditions than will a person with fewer. However, skeletal muscle cells respond to changes in the pattern of neural stimulation. Fast fibers trained for aerobic competition develop the characteristics of intermediate fibers, and this change improves aerobic endurance.

**Improving cardiovascular performance.** Cardiovascular activity affects muscular performance by delivering oxygen and nutrients to active muscles. Physical training alters cardiovascular function by accelerating blood flow, thus improving oxygen and nutrient availability. . . .

Aerobic activities do not promote muscle hypertrophy. Many athletes train using a combination of aerobic and anaerobic exercises so that their muscles will enlarge and both anaerobic and aerobic endurance will improve. These athletes alternate an aerobic activity, such as swimming, with sprinting or weight lifting. The combination is known as *interval training* or *cross-training*. Interval training is particularly useful for persons engaged in racquet sports, such as tennis or squash, which are dominated by aerobic activities but are punctuated by brief periods of anaerobic effort.

The following table from chapter 10 of the Fundamentals of Anatomy & Physiology, 5<sup>th</sup> Ed. displays a quick reference summary of the differences between the three (3) types of muscle fibers.

TABLE 1 0-3 Properties of Skeletal Muscle Fiber Types

Property	Slow	Intermediate	Fast
Cross-sectional diameter	Small	Intermediate	Large
Tension	Low	Intermediate	High
Contraction speed	Slow	Fast	Fast
Fatigue resistance	High	Intermediate	Low
Color	Red	White	White
Myoglobin content	High	Low	Low
Capillary supply	Dense	Intermediate	Scarce
Mitochondria	Many	Intermediate	Few
Glycolytic enzyme concentration in sarcoplasm	Low	High	High
Substrates used for ATP generation during contraction	Lipids, carbohydrates, amino acids (aerobic)	Primarily carbohydrates (anaerobic)	Carbohydrates (anaerobic)
Alternative names	Type I, S (slow), red, SO (slow oxidizing), slow-twitch oxidative	Type II-B, FR (fast resistant), fast-twitch oxidative	Type II-A, FF (fast fatigue), white, fast-twitch glycolytic

R. Matini, *Fundamentals of Anatomy & Physiology (5<sup>th</sup> Ed.)*. Prentice-Hall, Inc. (2000).

(d.) **What Are the Different Types of Contraction Forces During Muscle Use – Concentric vs. Eccentric?**

Two different types of contraction forces can be exerted by muscle cells – concentric and eccentric.

(i.) **Concentric Muscle Cell Contraction**

A concentric contraction is a type of muscle contraction during which the muscles shorten while generating force. During a concentric contraction, a muscle is stimulated to contract according to the sliding filament mechanism (see troponin above). Concentric contractions occur throughout the length of the muscle, generating force at the musculo-tendinous junction, causing the muscle to shorten and changing the angle of the joint. For example, when assuming the proper stance with the hands held high and near the face, a concentric contraction of the biceps causes the arms to bend at the elbow and hands to move from near the waist to close to the face/shoulder. A concentric contraction of the triceps changes the angle of the elbow in the opposite direction, thus straightening the arm and moving the hand outward such as when throwing a jab.

In addition, it is typical to see less experienced Combative Sports Contestants compete in a more “tense” or “less smooth and relaxed” manner due to either nervousness or just their body’s natural response to a stressful, unfamiliar situation. The tensing of muscles involves constant or sustained concentric contraction of muscle cells, which in turn causes the cells to burn more energy at a faster pace. The increased energy use by the Contestant’s muscle cells speeds the Contestant’s entry into anaerobic energy production and rapidly introduces general fatigue and elevated risks of exertional rhabdomyolysis.

As Contestants gain experience they generally tend to learn to “loosen up” and compete in a more “smooth and fluid” manner. Observation of Contestants, during thousands of Rounds at both the amateur and professional levels, has established there is no proven method to ensure every Contestant is completely prepared for the emotional and physiological responses experienced each time the Contestant steps into the Ring or Cage. Every Contestant is biologically and emotionally different and may experience a unique set of personal and professional circumstances every time he or she steps inside the Ring or Cage.

Although “tense” or “tight” movement is without question more prevalent in younger, less experienced Contestants, even seasoned professionals with many high pressure Bouts on their record have experienced an “off night” or time when they properly trained for the Bout, yet when the lights came on and they stepped inside the Ring or Cage, they just “tensed up” and “couldn’t get loose.” Many trainers might refer to such phenomena as “lack of focus” or simply “being too tight.” Whatever the reason for the Contestant’s reaction, there is no way to prevent it altogether. Experience has shown training regimens which include mental focus and preparation in addition to the physical aspects of training will help a Contestant remain more relaxed physically while still retaining the speed and power to effectively compete. A Contestant who is relaxed and moving about in a “smooth, fluid manner” will burn less energy and reduce the risk of rhabdomyolysis.

Jones was observed coming into the Ring with a very tense posture and multiple reviews of the Bout’s video confirm Jones was indeed competing in a very tense manner during the 1<sup>st</sup> Round. In fact, when the opening bell rang, Jones exacerbated his tense demeanor by beginning and maintaining a very high, determined pace during the entirety of the 1<sup>st</sup> Round. The excitement of Downing Palmer in the 1<sup>st</sup> Round no doubt increased Jones’ adrenaline and excitement, thereby bringing with it additional energy demands from Jones’ substantial muscle mass.

### **(ii.) Eccentric Muscle Cell Contraction**

In contrast to concentric contraction, an eccentric contraction results when the muscle elongates while under tension due to an opposing force greater than the force generated by the muscle. Rather than working to pull a joint in the direction of the muscle contraction, the muscle acts to decelerate the joint at the end of a movement or otherwise control the repositioning of a load, such as when two boxers grab and hold each other with each resisting the movement of the other. Eccentric contractions can occur involuntarily (e.g. when attempting to move a weight too heavy for the muscle to lift) or voluntarily (e.g. when the muscle is “smoothing out” or decelerating the movement of a jab or a missed punch).

Muscles undergoing heavy eccentric loading suffer greater damage when overloaded (such as during muscle building or strength training exercise) as compared to concentric loading. When eccentric contractions are used in weight training, they are normally called “negatives.” Muscles under an eccentric load can actually support a greater weight (muscles are approximately 10% stronger during eccentric contractions than during concentric contractions), but also sustain greater muscle cell damage and delayed onset muscle soreness one to two days afterward.

To protect joints from damage, eccentric contractions normally occur as a braking force in opposition to a concentric contraction. Combative Sports necessarily require numerous concentric and eccentric contractions in alternating patterns and the use of which depends upon whether the Contestant is on offense or defense. During virtually any routine movement, eccentric contractions assist in keeping the motions smooth, but can also slow rapid movements such as a punch or throw, as was the case with Jones during this boxing match. Jones had an abundance of muscle mass, thus every punch he threw exerted the force of all of his muscle cells in that area using a concentric motion. Conversely, every punch blocked by Jones and the Contestants’ clinching required extraordinary and excessive eccentric contractions from Jones’ muscle cells. The extreme forces exerted by Jones muscle cells to rapidly alternate between concentric and eccentric contractions when throwing punches, taunting punches, blocking punches and clinching with Palmer caused Jones to suffer extensive muscle cell damage.

### **(e.) What Are the Symptoms of Rhabdomyolysis?**

The signs and symptoms of rhabdomyolysis are not all that specific and can vary depending upon the cause; however, the signs and symptoms include muscle pain, weakness, tenderness, and stiffness with paralysis

and severe weakness occurring due to extensive muscle cell necrosis and hyperkalemia. Knochel, J.P. (1998). "Pigment nephropathy." In A. Greenbert (Ed.), Primer on kidney diseases (2<sup>nd</sup> Ed.) (pp. 273-276). Fifty percent (50%) of rhabdomyolysis patients will present with symptoms involving thighs, calves and lower back muscles. Gabow, P.A., Kaehny, W.D., & Kellher, S.E. (1982). "The spectrum of rhabdomyolysis." Medicine, 61:141-152. Here, Jones' blood test results and complaint that he could not feel or move his legs after arrival at UAMS around 00:40 hours are all consistent with the signs and symptoms of severe exertional rhabdomyolysis coupled with resultant hyperkalemia.

Dead muscle tissue may cause a large amount of fluid to move from the blood into the muscle, reducing the fluid volume of the body and leading to shock and reduced blood flow to the kidneys. Risk factors include the following: Alcoholism; ischemia or necrosis of the muscles; low PO<sub>4</sub> levels; severe exertion & exercise; use of illicit drugs, especially cocaine, amphetamines, statins & heroin. Symptoms include: Abnormal urine color (brownish or reddish); general weakness; muscle stiffness or aching; muscle tenderness; elevated CPK levels; elevated serum myoglobin levels; elevated K<sup>+</sup> levels; presence of hemoglobin in urine without evidence of red blood cells; presence of myoglobin in urine; CPK isoenzymes; elevated urine creatinine; and elevated serum creatinine.

Rhabdomyolysis may be suspected in anyone suffering a trauma, crush injury or prolonged immobilization; however, rhabdomyolysis may also be identified at a later stage due to deteriorating kidney function (abnormally raised or increasing creatinine and urea levels, falling urine output) or typical brownish or pink-red discoloration of the urine. Here, Jones did not suffer any crush injuries, as he only received three (3) body shots during the Bout; however, Jones did fight at an extremely high pace during the 1<sup>st</sup> Round of the Bout and both Jones and Palmer engaged in an excessive amount of clinching during both the 1<sup>st</sup> and 2<sup>nd</sup> Rounds of the Bout, thus providing the mechanism for exertional rhabdomyolysis rather than compressional rhabdomyolysis. Jones' very high K<sup>+</sup> levels (hyperkalemia) in each of his blood tests also tend to be a classic feature of rhabdomyolysis. Low Ca<sup>2+</sup> levels may be present in the initial stage due to binding of free Ca<sup>2+</sup> to damaged muscle cells and precipitation of CaPO<sub>4</sub> in the presence of elevated PO<sub>4</sub> levels, each of which were exhibited by Jones' blood test results. In addition, the Commission's investigation revealed Jones had a history of steroid and illicit drug use and alcoholism.

**(i.) Elevated Transaminase (AST & ALT) Levels**

Transaminase (AST & ALT) levels are usually increased with rhabdomyolysis. Here, Jones' AST & ALT levels were elevated at 22:45 hours, only an hour after the end of the Bout, and his AST & ALT levels continued to elevate throughout the night. In the early stages of rhabdomyolysis or in less severe cases, elevated AST and ALT levels can lead to health care providers confusing rhabdomyolysis with acute liver injury. The incidence of true acute liver injury has been reported as high as 25% in one study of patients with non-traumatic rhabdomyolysis although the mechanism for this finding was uncertain.

**(ii.) Elevated Creatine Kinase (CK) & Creatine Phosphokinase (CPK) Levels**

One of the most reliable tests used to diagnose rhabdomyolysis is to test for the level of creatine kinase (CK) or creatine phosphokinase (CPK) in the blood. The creatine kinase and creatine phosphokinase enzymes are released by damaged muscle cells, and levels above five (5) times the upper limit of normal indicate rhabdomyolysis. Here, Jones had a CPK result of 1,463 U/L as early as 22:45 hours, which level was nearly five (5) times the upper end of the normal range of 39 – 308 U/L.

CPK levels depend upon the both the severity and duration of the rhabdomyolysis, and levels up to 100,000 units are not unusual in the most severe cases, usually involving compressional rhabdomyolysis. Initial and peak CPK levels have a linear relationship with the risk of acute renal failure in that the higher the CK, the more likely it is that kidney damage will occur. Typically, CPK levels rise 12 hours of the initial damage and remain elevated

for 1–3 days followed by a gradual decline; however, CPK levels may rise very soon after initial damage, as they did here in Jones’ case.

**(iii.) Presence of Myoglobin in Blood & Urine**

Myoglobin has a short half-life, and is therefore a useful diagnostic marker during the earlier stages of rhabdomyolysis and becomes less useful as a diagnostic test in the later stages of the disorder or beginnings of the recovery phase. Like Jones’ first urine test at Saline Memorial Hospital, a dipstick analysis of urine may reveal a positive result for “blood” in the absence of red blood cells on microscopy, as the reagent reacts with myoglobin, which will be present in the urine during rhabdomyolysis.

**(iv.) Abnormal Electrolyte Levels**

In the initial stages of rhabdomyolysis, electrolyte levels are often abnormal and require correction.  $\text{Ca}^{2+}$  levels initially tend to be low, but as the patient’s condition improves,  $\text{Ca}^{2+}$  is released from where it has precipitated within the remaining muscle cells and other tissues, along with phosphate. Vitamin D production resumes during compensatory or recovery phases, leading to hypercalcemia (abnormally high calcium levels). This “overshoot” occurs in 20–30% of those people who have developed kidney failure.

**(v.) Elevated Troponin Levels**

Cardiac troponin levels (normally used to diagnose heart damage) are increased in half of all cases of Rhabdomyolysis, but not associated with other evidence of heart damage in at least a third of those cases. However, in Jones’ case, Jones showed significantly elevated Troponin levels, notably the more specific Troponin I marker, but his elevated Troponin levels were correlated and confirmed as indicative of heart damage by CK-MB levels in excess of 300 at both 03:04 hours and 04:00 hours.

**(f.) What Are the Effects of Rhabdomyolysis?**

The effects of rhabdomyolysis are wide ranging and depending on the severity of the condition include, worsening of the condition via additional muscle cell death due to swelling, lactic acidosis and cellular oxidation, leading to additional increases in extracellular potassium ( $\text{K}^+$ ), extracellular myoglobin and its toxic ferriheme metabolite, renal tubule necrosis, acute renal failure, cardiac arrhythmias, and eventual cardiac arrest.

**(i.) Muscle Cell Death Via Swelling & Fluid Absorption**

As was the case for Jones, rhabdomyolysis causes renal failure, and subsequent cardiac dysrhythmia, by several mechanisms. Firstly, the release of  $\text{K}^+$  from the destroyed cells causes the remaining cells to store calcium ( $\text{Ca}^{2+}$ ), sodium ( $\text{Na}^+$ ) and water molecules, thus causing the remaining muscle and other tissue to swell. Swelling of the remaining muscle cells removes the calcium ( $\text{Ca}^{2+}$ ) necessary to offset the cardio-toxic effects of the hyper-elevated extracellular potassium ( $\text{K}^+$ ). Additionally, swelling of the remaining muscle and other cells removes water from the circulatory system which causes hypovolemia and a relative lack of blood flow to the kidney. If the muscle cells swell too much, they can burst thereby worsening the condition by releasing additional potassium ( $\text{K}^+$ ) and myoglobin into the blood stream and increasing the pace of multiple organ system deterioration.

The swelling and fluid reabsorption effects of this stage of rhabdomyolysis are evidenced by the fact that Jones weighed in for the Bout at 232.5 lbs. (105 kg), weighed 240 lbs (109 kg) upon check-in to UAMS and 252 lbs. (114 kg) at autopsy; further, Jones had been given at least 7.25 liters of IV fluids, yet produced scant amounts of urine. Each liter of IV fluid weighed approximately 2.2 lbs. (1.0 kg), thus accounting for nearly 16 lbs. (7.25 kg) of Jones’ increased weight (the remainder of Jones’ weight is likely accounted for by rehydration and food

intake between the time of the weigh-ins the evening before the Bout and the start of the Bout). Consistent with the natural biophysiological processes associated with rhabdomyolysis, Jones' body absorbed all of the IV fluids into the undamaged cells and muscle tissues.

**(ii.) Muscle Cell Death Via Lactic Acidosis**

Lactic acidosis is a distinct form of metabolic acidosis and most often occurs when the body's cells do not receive enough oxygen to continue aerobically producing energy (via ATP production), such as during vigorous exercise, and instead, begin producing ATP through anaerobic glucose metabolism. During anaerobic energy production, the body produces lactate to facilitate the breakdown of glucose for energy, then release the lactate into the blood stream as lactate levels inside the muscle cells become too high.

High levels of lactate in blood permit the formation of excessive amounts of lactic acid in the fluid surrounding the muscle cells. The metabolic pathways used for energy production cannot properly function in a highly acidic environment. As the acid levels increase, the cells produce less energy and the body is forced to slow down. The production and effects of lactate and other metabolites during extreme exertion is directly responsible for the burning sensation and fatigue often felt in active muscles. Thus, the entry into anaerobic energy production and increasing lactate and acidity in the muscle tissue is easily detected by athletes without any sophisticated tests. Every time an athlete says he or she "feels the burn," what they are actually saying is, "My muscle cells have been in anaerobic energy production for too long, my lactate production has increased beyond my body's ability to remove it and the areas surrounding my muscle cells are now too acidic."

In other words, the burning sensation is the body's natural defense mechanism intended to force (or at least warn) the person to slow down and allow the body to return to aerobic energy production before the lactic acid levels become too high and begin causing extensive damage. While the burning sensation does not mean athletes must immediately stop their activity (and in fact, some studies suggest that some lower levels of increased lactic acid in the muscle tissues help muscle cells perform better and more efficiently in anaerobic states), when athletes "push through the pain" they, and their trainers, must realize they have just entered into potentially lethal territory. The body is incredibly resilient to extreme exertion and abuse; however, the body has limits and each person's limit changes on a minute by minute basis due to an almost infinite number of factors constantly in play within the body.

The acidosis associated with increases in lactate concentration during heavy exercise actually arises from a separate reaction outside the muscle cell. During intense exercise, blood oxygen levels decrease and aerobic metabolism cannot produce ATP in sufficient quantities to meet the muscle cell's energy demand. Therefore, in such oxygen deficient, high energy demand situations, anaerobic metabolism becomes the dominant energy-producing pathway. When ATP is hydrolyzed (broken down), a hydrogen ion is released. ATP derived hydrogen ions are, together with the anaerobically produced lactate, primarily responsible for the decrease in pH. Due to the large amounts of ATP being produced and hydrolyzed in a short period of time, the buffering systems of the tissues are overcome, causing pH to fall and creating a state of lactic acidosis. Up to a certain point, the lactic acidosis is beneficial because it facilitates the easier dissociation of oxyhaemoglobin and allows easier transfer of oxygen from the blood; however, as the blood's pH continues to fall and the environment around the muscle cells becomes too highly acidic, the muscle cell walls begin to deteriorate and the cells die in massive quantities, resulting in the release of their potassium ( $K^+$ ) and myoglobin contents into the blood stream.

Lactic acidosis indicates cellular tissue damage and is characterized by lactate levels greater than 5 mmol/L and blood pH less than 7.35. Here, Jones had a steadily decreasing (becoming more acidic) blood pH of 7.2 at 00:40 hours, pH of 7.129 at 01:00 hours, pH of 7.158 at 05:28 hours, pH of 6.973 at 05:48 hours, and a pH of 7.003 at 06:16 hours. Blood pH should normally fall within a range between 7.350 and 7.450.

The massive volume of cell death, along with the consequential release of potassium ( $K^+$ ) and myoglobin, in such a short period overwhelms the body's ability to compensate for and regulate the increased acid levels through respiration, increased potassium ( $K^+$ ) levels through renal filtration, increased potassium ( $K^+$ ) the body's primary mechanism of intracellular storage, and excessive myoglobin (in ferriheme form) which poisons the renal tubules of the kidneys thereby preventing proper filtration of the blood.

Lactic acidosis can lead to not only the death of muscle cells, but also the death of neural cells within the brain. F. Staub, B. Mackert, O. Kempfski, J. Peters, A Baethmann, "Swelling and death of neuronal cells by lactic acid." *Journal of Neurological Science*, 1993: 119: pp. 79-84.

(iii.) **Muscle Cell Death Via Oxidation, Ferryl Myoglobin ( $Mg^{+4}$ ) & Other Toxins – Protective Benefits of the Anti-oxidants – Vitamin C, Vitamin E, Beta Carotene and Ubiquinone (Coenzyme  $Q_{10}$ )**

While the production and conversion of ATP without oxygen during anaerobic respiration can result in dangerous lactic acid build up in the muscle tissue, another more dangerous condition is created during aerobic respiration when there is an insufficient supply of ubiquinone, the coenzyme  $Q_{10}$  discussed earlier. Aerobic production of ATP in a coenzyme  $Q_{10}$  deficient environment results in the inefficient utilization of the electron transport chain. More specifically, hydrogen ions leak back into the mitochondrion through a passive process, which then requires a much larger expenditure of energy to push the hydrogen ions back outside the structure along with a corresponding reduction in the overall electrical charge. The increased energy expenditure used to eject the hydrogen ions results in a decreased ATP production during the aerobic cycle.

The net effect of reduced aerobic efficiency is very similar to the effect of insufficient oxygen; however, the situation is much more harmful for the cells because, instead of being absent, the oxygen is present but only partially converted to water. Various highly toxic charged ions containing oxygen, such as the highly reactive free radical hydroxyl ( $-OH$ ), superoxide ( $O_2^-$ ) and acidic hydrogen peroxide ( $H_2O_2$ ) will linger inside the cell with devastating effects on the muscle cell's internal and external structures. In particular, the hydroxyl ( $-OH$ ) free radical causes extensive damage to oxidative cells, especially myoglobin (iron and oxygen binding protein inside muscle cells), erythrocytes (red blood cells which are rich in hemoglobin for carrying oxygen in the blood), and the lipids and proteins comprising the muscle cell walls. More specifically, the hydroxyl ( $-OH$ ) promotes deterioration of the lipid structure of the cell walls, resulting in increased leakage of the cell's contents into the blood stream and eventually the complete destruction of the cell wall. Upon destruction of the cell wall, the potassium ( $K^+$ ) and myoglobin inside are released into the blood.

Additionally, with statin therapy, acidic hydrogen peroxide is generated in the mitochondria because the process of breaking down oxygen and converting it to water is incomplete due to the insufficient supply of coenzyme  $Q_{10}$ .

Myoglobin is a unique protein, which is specially adapted to satisfy muscle cells' tremendous need for energy with its corresponding need for the oxygen required by aerobic respiration. Myoglobin exists in at least three (3) distinct forms, which can be characterized as  $Mg^{+2}$  (Ferrous),  $Mg^{+3}$  (Ferric), and  $Mg^{+4}$  (Ferryl), depending upon the amount of charge that is present on the central iron atom (Fe). As  $Mg^{+2}$ , its healthy state, myoglobin will readily take up oxygen and store it and when converted to  $Mg^{+3}$  by the addition of a proton, myoglobin becomes harmlessly inert. However, the addition of yet another proton converts myoglobin to its ferryl form  $Mg^{+4}$  (ferriheme), a highly toxic reactive agent that will begin to break down the fatty acids contained in the outer cell wall of the muscle cell (so-called peroxidative damage), and go on to destroy the cholesterol in the cell wall as well. R.P. Patel, U. Diczfalusy, S. Dzeletovic, M.T. Wilson and V.M. Darley-Usmar, (1996) "Formation of oxysterols during oxidation of low density lipoprotein by peroxynitrite, myoglobin, and copper." *Journal of Lipid Research* Vol. 37, pp. 2361-2371.

Myoglobin becomes ferryl myoglobin in the presence of excess amounts of free radicals, i.e., under oxidative stress induced by highly reactive oxygen compounds like hydroxyl (-OH) and hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>). E.Y. Plotnikov, A.A. Chupyrkina, I.B. Pevzner, N.K. Isaev, and D.B. Zorov, (2009). "Myoglobin causes oxidative stress, increase of NO production and dysfunction of kidney's mitochondria." *Biochimica et Biophysica Acta (BBA) - Molecular Basis of Disease*. August, Vol. 1792, Issue 8:796-803; T. Pozefsky, R G Tancredi, R T Moxley, J Dupre, and J D Tobin (1976) "Effects of brief starvation on muscle amino acid metabolism in nonobese man." *J Clin Invest.* February, Vol. 57, No. 2, pp. 444-449.

Once the fatty acids in a muscle cell wall are broken down by exposure to toxic ferryl myoglobin (Mg<sup>+4</sup>), the cell rapidly disintegrates. Because the cell wall is no longer impermeable to ions, large amounts of calcium start rushing into the cell, and soon afterward the cell dies. J.L. Farber, (1994) "Mechanisms of cell injury by activated oxygen species." *Environ Health Perspect.* December, Vol. 102 (Suppl 10):17-24. The debris of the dead and dying muscle cells are then dispersed into the bloodstream and either consumed by the liver or filtered by the kidneys.

Ubiquinone (coenzyme Q<sub>10</sub>), vitamin C, vitamin E and Beta Carotene are all anti-oxidants which will block oxidation of muscle cells by the free radical hydroxyl (-OH) and acidic hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>). Therefore, increasing dietary intake of these anti-oxidants will provide protective benefits against the effects of rhabdomyolysis. The mechanisms by which muscle cells are protected from oxidative stress are as follows: "All aerobic cells generate, enzymatically or nonenzymatically, a constitutive flux of O<sub>2</sub><sup>-</sup>, H<sub>2</sub>O<sub>2</sub>, and possibly -OH. At the same time, the abundant antioxidant defenses of most cells, again both enzymatic and nonenzymatic, prevent these species from causing cell injury. Nevertheless, there are situations in which the rate of formation of partially reduced oxygen species is increased and/or the antioxidant defenses of the cells are weakened. In either case, oxidative cell injury may result." J.L. Farber, (1994) "Mechanisms of cell injury by activated oxygen species." *Environ Health Perspect.* December, Vol. 102 (Suppl 10):17-24.

The process of aerobic oxidation of food sources to generate energy is confined to the mitochondria in order to protect the constituents in the cytoplasm from oxidative stress as much as possible. But myoglobin is tasked with transporting oxygen from the cell wall through the cytoplasm to the mitochondria; therefore, myoglobin cannot avoid exposure to oxygen and when it delivers the oxygen, myoglobin is also forced to encounter the toxic intermediate products associated with the cell's conversion of oxygen to water. One of the most important roles of coenzyme Q<sub>10</sub> in the muscle cells is to neutralize the damage to myoglobin caused by these oxidative agents, including the prevention of myoglobin conversion into a highly toxic ferryl state (Mg<sup>+4</sup>).

#### (iv.) Renal Failure Via Elevated Myoglobin Levels

Finally, the most toxic and potentially deadly effect of rhabdomyolysis is the accumulation of the released myoglobin in the renal tubules. As explained, the myoglobin is an iron and oxygen binding protein found in muscle cells and should not be confused with the related protein, hemoglobin, which is found in the blood. The presence of myoglobin in the blood is abnormal and only occurs following the destruction of muscle cells. Once myoglobin makes its way into the blood, it is filtered by the kidneys' glomeruli; however, the myoglobin is toxic to the epithelium (lining tissue) of a portion of the nephron called the renal tubule. Renal tubules are the collecting tubes responsible for transporting urine to the ureters, reabsorbing 99% of the water, and secreting various ions such as sodium (Na<sup>+</sup>), glucose, and amino acids like glutamate. Many epithelial cells within the renal tubules have highly specialized functions, thus damage to these cells can cause severe dehydration. The renal tubules are also critically important to filtration and removal of potassium (K<sup>+</sup>), thus rapid, hyper-elevation of myoglobin in the blood very quickly causes acute renal failure via renal tubule necrosis. Naka T, Jones D, Baldwin I, *et al.* "Myoglobin clearance by super high-flux hemofiltration in a case of severe rhabdomyolysis: a case report." *Critical Care* 2005;9:R90-R95.

Myoglobinuria, the appearance of myoglobin in the urine, occurs when the levels in plasma exceed 1.5 mg/dL. At 22:45 hours, Jones had a blood plasma myoglobin level of 17.3 g/dL, well in excess of the levels normally associated with excessive myoglobin exposure. As the kidneys reabsorb more water from the filtrate passing through the nephrons, the myoglobin forms casts that obstruct the flow of fluid through the nephron; the condition is worsened further by high levels of uric acid and acidification of the filtrate. With the onset of renal tubular necrosis, the kidney is no longer able adequately perform its normal functions and a potentially lethal feedback loop is established via a fall in glomerular filtration rates and disruption of electrolyte regulation, which then leads to a further increase in potassium ( $K^+$ ) levels and hormone production (hence decreased vitamin D processing, further worsening the low calcium levels). In addition, iron (Fe) released from the myoglobin generates reactive oxygen species which leads to further oxidation and breakdown of muscle cells and other tissue within the kidneys. Together, the multiple degenerative processes lead to acute tubular necrosis, complete destruction of the renal tubule cells and total renal failure.

The presence of hazy, brown epithelial cells in urine is indicative of renal tubular damage from either exposure to a toxin such as myoglobin or ischemia, insufficient blood flow/oxygen resulting from decreased blood flow through the kidneys. Myoglobin toxicity is easily differentiated from ischemia because ischemic renal tubular damage typically causes skip lesions throughout the tubules. In this case, Jones' urine was hazy just 55 minutes after the end of the Bout and again at 02:26 hours with the presence of red blood cells, myoglobin and high glucose; however, upon autopsy, Jones' kidneys did not show any signs of skip lesions. As pointed out in more detail above, the results from every one of Jones' blood tests further confirm Jones' rhabdomyolysis, resultant intrarenal kidney damage, renal failure and ventricular fibrillation.

Each patient's prognosis significantly depends on the underlying cause and whether any complications occur. Rhabdomyolysis patients who experience acute renal failure may have a mortality rate as high as 20%. In 1995, USA hospital statistics reported 26,000 cases of rhabdomyolysis. Up to 85% of patients with major traumatic injuries will experience some degree of rhabdomyolysis. Of those patients with rhabdomyolysis, 10–50% will develop acute kidney injury. The risk is higher in patient populations with a history of illicit drug use, alcohol misuse and trauma when compared to muscle diseases, and has been found to be particularly high if multiple contributing factors occur together. Rhabdomyolysis accounts for approximately 7–10% of all cases of acute kidney injury in the United States.

**(g.) Acid – Base Homeostasis – How Do the Kidneys & Lungs Control Lactic Acidosis & Oxidation?**

Two organ systems, the kidneys and lungs, maintain the body's acid-base homeostasis, which is the maintenance of the blood's pH around a relatively stable value between 7.35 and 7.45. As the pH moves lower, the acidity of the blood rises. The kidneys contribute to acid-base homeostasis by regulating bicarbonate ( $HCO_3^-$ ) concentration. The kidneys have two important roles in the maintaining of the acid-base balance: (i.) Reabsorption of bicarbonate from the blood and (ii.) Excretion of hydrogen ions into urine.

**(i.) Arterial Blood Gas (ABG) Tests**

The Arterial Blood Gas test measures certain characteristics of arterial blood. The first measurement is the blood's pH level. The pH designates the acid-base balance of arterial blood. Ideally, blood pH should be 7.4. However, many variables affect the pH of the blood. If one of these variables forces the pH too far from 7.4, the cells of the body will be unable to properly function. Therefore, the body has two main buffering systems:

- 1.) The respiratory system; and
- 2.) The renal system.

The respiratory and renal systems generally balance each other to provide an optimum environment within the body. Balance must always be achieved by the opposite system, thus the systems can be thought of as being on opposite sides of a seesaw. When one side moves one direction the other must move in the opposite direction to maintain the body's acid-base balance. The balancing component of the respiratory system is the dissolved carbon dioxide (CO<sub>2</sub>) produced by cellular processes and removed by the lungs. The renal system's opposing or balancing component is the dissolved bicarbonate (HCO<sub>3</sub><sup>-</sup>) produced by the kidneys. In addition, the kidneys also help control pH by eliminating hydrogen (H<sup>+</sup>) ions.

The mechanism of action between the respiratory and renal systems is via formation of carbonic acid (H<sub>2</sub>CO<sub>3</sub>). Movement through the carbonic acid system is both fluid and constant, meaning that water (H<sub>2</sub>O) can combine with CO<sub>2</sub> to form carbonic acid, which offsets the alkalotic blood (i.e. low blood pH). On the other hand, if the blood's pH falls and the blood becomes too acidic, then carbonic acid (H<sub>2</sub>CO<sub>3</sub>) breaks up to form hydrogen ions (H<sup>+</sup>) and bicarbonate (HCO<sub>3</sub><sup>-</sup>) to provide the balance. Thus, by balancing back and forth in both directions, the body achieves and maintains its proper pH.

The respiratory system balances pH by manipulating the CO<sub>2</sub> level through increases or decreases in the respiratory rate. Faster and deeper breathing "expels" more CO<sub>2</sub>. Conversely, slower and shallower breathing "retains" more CO<sub>2</sub>. The renal system balances pH by producing HCO<sub>3</sub><sup>-</sup> or by eliminating hydrogen ions (H<sup>+</sup>).

The renal system reflects changes in the body's metabolic activity. For example, a patient, such as Jones, who becomes hypoxic (reduced blood oxygen levels) will undergo anaerobic metabolism, which produces lactic acid. The production of lactic acid will bind with HCO<sub>3</sub><sup>-</sup> thereby decreasing the body's HCO<sub>3</sub><sup>-</sup> level and causing acidotic conditions. Therefore, the HCO<sub>3</sub><sup>-</sup> level is an indicator used in determining the metabolic acid-base balance.

Since the body regulates pH by using the opposite system to balance pH, if the pH is out of balance due to a respiratory disorder, the renal system will make the necessary corrections to balance the pH. On the other hand, if the renal system is to blame for the pH imbalance, the respiratory system provides the balancing mechanism. This offsetting process is called compensation. Compensation may not always be complete. Complete compensation returns the pH balance to normal. However, there are times when the imbalance is too large for compensation to return the pH to normal. This is called incomplete compensation.

Two sets of information can be obtained from an arterial blood gas (ABG) machine. The first is the blood acid-base balance, and the second is blood oxygenation. The measures of blood oxygenation are the oxygen (pO<sub>2</sub>) and the oxygen saturation (O<sub>2</sub> sat). The dissolved oxygen in the blood is referred to as the pO<sub>2</sub> and is measured in mmHg. The second measurement is the oxygen saturation, which represents the amount of hemoglobin sites with attached oxygen. Oxygen saturation is expressed as a percentage of the total sites that have hemoglobin. The O<sub>2</sub> sat can be continually monitored non-invasively using pulse oximetry.

An ABG can detect four main states other than normal:

- 1.) Metabolic Acidosis;
- 2.) Metabolic Alkalosis;
- 3.) Respiratory Acidosis; and
- 4.) Respiratory Alkalosis.

Generally, the following six (6) steps are used in connection with an ABG analysis to determine which state exists in a patient.

- 1.) Look at the pH. Normal blood pH is 7.4, plus or minus 0.05, forming an acceptable normal range of 7.35 to 7.45. Blood pH below 7.35 it is acidic and blood pH above 7.45 is alkalotic. pH levels lower than 7.4 are normal/acidic, higher than 7.4 are normal/alkalotic.
- 2.) Examine the pCO<sub>2</sub>. Normal pCO<sub>2</sub> levels are 35-45mmHg. Below 35 is alkalotic, above 45 is acidic.
- 3.) Look at the HCO<sub>3</sub><sup>-</sup> level. Normal HCO<sub>3</sub><sup>-</sup> is 22-26 mEq/L. HCO<sub>3</sub><sup>-</sup> below 22 is acidotic and HCO<sub>3</sub><sup>-</sup> above 26 is alkalotic.
- 4.) Match either the pCO<sub>2</sub> or the HCO<sub>3</sub><sup>-</sup> with the pH to determine the acid-base disorder. For example, if the pH is acidotic, and the pCO<sub>2</sub> is acidotic, then the acid-base disturbance is being caused by the respiratory system (“Respiratory Acidosis”). If the pH is alkalotic and the HCO<sub>3</sub><sup>-</sup> is alkalotic, the acid-base disturbance is being caused by the metabolic (or renal) system (“Metabolic Alkalosis”).
- 5.) If either the pCO<sub>2</sub> or HCO<sub>3</sub><sup>-</sup> go in the opposite direction of the pH, then there is compensation by the system associated with the condition opposite from the pH. For example, if the pH is acidotic, the pCO<sub>2</sub> is acidotic, and the HCO<sub>3</sub><sup>-</sup> is alkalotic, then the primary acid-base disorder is respiratory acidosis because the pCO<sub>2</sub> matches the pH. The HCO<sub>3</sub><sup>-</sup> is opposite of the pH and evidences a compensation response from the metabolic system.
- 6.) Evaluate the PaO<sub>2</sub> and O<sub>2</sub> sat. If they are below limits there is evidence of hypoxemia.

For example, at 01:00 hours, Jones’ pH was 7.129 (acidotic), pCO<sub>2</sub> was 29.9 (mildly alkalotic respiratory system), and (HCO<sub>3</sub><sup>-</sup>) was 9.9 (very acidotic metabolic system). Thus, at 01:00 hours, because Jones’ blood was acidotic and his respiratory system was oppositely alkalotic but not as equally alkalotic as his metabolic system was acidotic, we know that Jones’ body was suffering from metabolic acidosis with his respiratory system incompletely compensating due to the very large imbalance between the two systems. As further proof, we can note Jones’ increased respiratory rate of 26 at the same time, which further evidences his body’s compensatory reaction by expelling more oxygen.

On the other hand, during Jones’ Code Blue episode at 05:28 hours, we can see that Jones’ pH was 7.158 (acidotic), pCO<sub>2</sub> was 49.7 (mildly acidotic respiratory system), and (HCO<sub>3</sub><sup>-</sup>) was 17.2 (acidotic metabolic system). Thus, at 05:28 hours, because Jones’ blood was acidotic and both his respiratory system and metabolic system were also acidotic, we know Jones’ body was suffering from metabolic acidosis with his respiratory system failing to compensate.

**(h.) How Is Rhabdomyolysis Treated?**

The main goal of treatment is to treat shock and preserve kidney function.

**(i.) Administration of Fluids – Volume Expansion**

Initial treatment consists of administration of generous amounts of intravenous fluids, usually normal saline solution (0.9% weight per volume Na<sup>+</sup>Cl<sup>-</sup> solution), in a process known as volume expansion. In victims of crush syndrome (e.g. in earthquakes), it is recommended to start the normal saline IV drip even before the victims are extracted from collapsed structures. Similarly, as was done in Jones' case, normal saline IV drips should be started by emergency medical transport crews immediately upon a ringside Physician's decision to transport a Combative Sports Contestant, even if rhabdomyolysis is not yet suspected. Immediate administration of IV fluids will ensure sufficient circulating volume to deal with the muscle cell swelling typically ensuing immediately after restoration of blood supply in crush victims and resulting from certain biochemical processes associated with exertional rhabdomyolysis. Additionally, the administration of copious volumes of IV fluids also helps prevent the deposition of myoglobin in the kidneys' renal tubules. Amounts of 6 to 12 liters over 24 hours are usually recommended.

Here, the ringside emergency personnel and medical teams at both hospitals performed exactly as they should with respect to their rapid initiation of volume expansion treatment via administration of IV fluids to Jones. Jones had received two (2) liters of normal saline solution prior to arrival at UAMS (one in the ambulance, while en route to Saline Memorial, and one while at Saline Memorial). While in the emergency department at UAMS, medical personnel administered another four (4) liters of normal saline solution via rapid administration using dual IVs, one in each of Jones' arms. Then, while Jones was in the ICU and SICU departments at UAMS, medical personnel began administering one (1) liter of normal saline solution at the rate of 100 ml/hr. starting at 01:35 hours and one (1) liter of D5W solution at the rate of 150 ml/hr. starting at approximately 03:37 hours. In total, Jones was administered approximately 7.25 liters of fluids during his emergency medical treatment. Unfortunately, despite the tremendous amount of fluid volume administered to Jones during treatment, Jones' body was already too far into the advanced stages of rhabdomyolysis and renal failure. In short, there were not enough healthy cells left to absorb and utilize the fluids for proper cellular functions.

**(ii.) Infusion of Blood & Crystals**

Clinical reports suggest that volume expansion therapy may be effective in interrupting the course of acute renal failure secondary to rhabdomyolysis; however, in very severe cases of rhabdomyolysis, it is critical to correct hypovolemia and shock by the infusion of blood and crystalloids. Bonventre, J., Shah, S., Walker, R., & Humphreys, M. (1995). "Rhabdomyolysis induced acute renal failure." *The Principles and Practice of Nephrology (2<sup>nd</sup> Ed.)*. (pp. 569-573); Knochel, J.P. (1998). "Pigment nephropathy." *Primer on Kidney Diseases (2<sup>nd</sup> Ed.)*. (pp. 273-276).

The ringside emergency personnel and medical teams at both hospitals performed exactly as they should have in treating Jones. Medical personnel immediately began aggressive crystalloid infusions via normal saline solution IVs, even before Jones left the Venue and such treatments aggressively continued during the entirety of Jones' treatment. Additionally, the medical team at UAMS blood typed Jones shortly after his admission to UAMS' emergency department and had sufficient quantities of blood (more properly in modern medical practice, had sufficient fresh frozen plasma) on standby for use in Jones' treatment if blood infusion was indicated by additional tests. However, the UAMS medical team was prevented from blood infusion treatment due to the rapidity with which Jones' medical condition deteriorated into more critical conditions, such as acute renal failure, ventricular tachycardia and cardiac arrest, which required different courses of treatment.

### (iii.) Mannitol, Furosemide & Other Loop Diuretics

While many sources recommend later stage use of mannitol, which acts by osmosis and renal vasodilation to ensure urine production, inhibit tubular reabsorption of glomerular filtrate and increasing tubular pressure which may prevent heme deposition and formation of casts within the kidney, there are no studies directly demonstrating its benefit. Similarly, it is suggested that the addition of  $\text{HCO}_3$  to the IV fluids tends to reduce or control metabolic acidosis, stop the dissociation of filtered myoglobin into the renal tubule destroying ferriheme compound ( $\text{Mg}^{+4}$ ), and reduces myoglobin cast formation in the kidneys, all of which accompany various stages of rhabdomyolysis.

Furosemide, or other similar loop diuretics, is also often used to ensure sufficient urine production, reduce sodium ( $\text{Na}^+$ ) transport and oxygen utilization by the kidneys, thus preserving energy stores in the renal tubular cells. Knochel, J.P. (1998). "Pigment nephropathy." *Primer on Kidney Diseases (2<sup>nd</sup> Ed.)*. (pp. 273-276). However, large doses of furosemide are ototoxic (damaging to the ear's cochlea and auditory nerve) and can cause pulmonary edema (accumulation of fluid in the lungs), thus should be used only in extreme cases accompanied by other indications for use. Star, R.A. (1998). "Treatment of acute renal failure." *Kidney International*. 54 (6):1817-1831.

In Jones' case, the medical teams at UAMS also performed according to medically accepted practices and standards for the treatment of rhabdomyolysis and other medical conditions by administering the following to Jones: 50 mEq of  $\text{NaHCO}_3$  at 01:38 hours; one ampule (44.6 mEq) of  $\text{HCO}_3$  at 03:30 hours; and 100 mEq  $\text{NaHCO}_3$  at 03:37 hours. Instead of Furosemide, the UAMS staff administered the loop diuretic Bumetide 24 mg/96 mL, 0.5 mg/hr. titrated to effect at maximum drip rate of 3 mg/hr..

### (iv.) Hemodialysis

In many cases kidney dysfunction or acute renal failure develops 1–2 days after the initial muscle trauma, and renal replacement therapy is required to correct the critically high levels of  $\text{K}^+$ . This may take the form of hemodialysis or hemofiltration. Certain types of peritoneal dialysis are also effective in removing the high levels of toxic solutes that can accumulate in rhabdomyolytic renal failure, and may be the only available option in some resource-limited settings. Renal replacement therapy through hemodialysis removes the excess  $\text{K}^+$ , lactic acid and phosphate ( $\text{PO}_4$ ) which accumulates in the body when the kidneys are not functioning normally and such therapy is required until the kidneys regain proper functionality.

In some severe cases of rhabdomyolysis, despite all of the above treatment methods, the potassium ( $\text{K}^+$ ) release is at such a rapid rate and muscle cell haemolysis (cellular release of its contents) is so fast that no other treatment option other than hemodialysis is available. During hemodialysis, the blood is directly filtered by a machine which acts as either a supplement for impaired kidneys or complete substitute for a failed renal system. Here, at 04:20 hours the SICU physician started to place a right femoral artery line; however, decided to switch to a Quinton catheter for emergency hemodialysis due to the failure of all other treatment options to reduce Jones' critically high potassium level. The UAMS medical team then ordered the equipment and began prepping Jones for emergency hemodialysis; however, Jones went into cardiac arrest very soon thereafter and before arrival of the equipment necessary to begin hemodialysis.

### (i.) How Is Rhabdomyolysis Prevented?

There is no scientifically proven and medically accepted way to absolutely prevent rhabdomyolysis; however, the following tips can help prevent its onset or lessen its effects:

(i.) **H.E.A.R.T – (Hydration, Exertional Avoidance, Recognition & Training**

- 1.) Hydration – Drink plenty of fluids before and after strenuous exercise to dilute the urine and flush myoglobin out of the kidneys;
- 2.) Exertional Avoidance – Do not try to engage in any type of explosive and continuous muscle group exertion without taking precautions such as building up to the level of activity through a training program or being carefully monitored by someone trained to recognize the signs and symptoms of rhabdomyolysis and administer preventative treatments.);
- 3.) Recognition – Learn to recognize the early signs of extreme muscle fatigue and avoid over-exertion of muscles; and
- 4.) Training – Train appropriately for the type of event or contest in which you will participate (e.g. if distance running, then create a program which builds up to the target distance rather than trying to do it all at once.)

(j.) **Anti-oxidants – Ubiquinone (Coenzyme Q<sub>10</sub>)**

Similar to this report's earlier discussion of the use of omega-3 docosahexaenoic acid (DHA) as a prophylactic measure against the effects of a concussion, although not yet a medically accepted or scientifically proven method for prevention of rhabdomyolysis, the administration or ingestion of increased amounts of the anti-oxidants, vitamin C, vitamin E, beta carotene and ubiquinone (coenzyme Q<sub>10</sub>, also known as ubidecarenone, coenzyme Q, and sometimes abbreviated CoQ<sub>10</sub>), may have a beneficial effect for muscle cells during periods of extreme exertion, thereby reducing or delaying entry into anaerobic energy production and subsequent cell death.

Coenzyme Q<sub>10</sub>, is an oil soluble, vitamin like substance present in most eukaryotic cells, primarily in the mitochondria, and is a component of the electron transport chain which participates in aerobic cellular respiration (energy production) by generating energy in the form of ATP. Roughly ninety-five percent (95%) of the body's energy production is through the aerobic energy production process of which coenzyme Q<sub>10</sub> is a critical component. Consequently, the cells within the body with the highest energy demands, such as the heart, liver and kidneys, have the highest coenzyme Q<sub>10</sub> concentrations. Okamoto, T; Matsuya, T; Fukunaga, Y; Kishi, T; Yamagami, T (1989). "Human serum ubiquinol-10 levels and relationship to serum lipids". *International Journal for Vitamin and Nutrition Research. Internationale Zeitschrift fur Vitamin- und Ernährungsforschung. Journal international de vitaminologie et de nutrition.* 59 (3): 288–92. In the most basic description of how coenzyme Q<sub>10</sub> in its various forms works to prevent or delay rhabdomyolysis is that it constantly engages in an oxidation reduction cycle, whereby it prevents oxidation of lipids and proteins as it gives up its own electrons during the oxidation reduction cycle, while the CoQ efficiently prevents the oxidation of bases in the mitochondrial DNA and the reduced form of CoQ effectively regenerates vitamin E from the a-tocopheroxyl radical.

Coenzyme Q<sub>10</sub> is a crystalline powder that is insoluble in water, thus absorption in the body follows the same process as that of lipids and the uptake mechanism is similar to that of vitamin E, which is another lipid soluble nutrient required by the body. Thus, the presence of lipids, through food intake, greatly enhances the body's absorption rate of coenzyme Q<sub>10</sub>. Three major factors lead to coenzyme Q<sub>10</sub> deficiency in humans: (i.) Insufficient dietary intake; (ii.) Reduced biosynthesis; and (iii.) Increased utilization within the body. Other factors reducing coenzyme Q<sub>10</sub> levels in the body include the use of statin drugs (reduces blood serum levels by up to 40%), aging (reduces internal organ levels in those over 20 years of age), and exposure to ultraviolet light (reduces levels in the skin).

Coenzyme Q<sub>10</sub> in higher doses is not usually toxic and one study showed that doses as high as 3,600 mg per day were well tolerated in both healthy and unhealthy persons alike. Hyson HC, Kiebertz K, Shoulson I, McDermott M, Ravina B, de Blicke EA, Cudkowicz ME, Ferrante RJ, Como P, Frank S et al (2010). "Safety and tolerability of high-dosage coenzyme Q<sub>10</sub> in Huntington's disease and healthy subjects". *Mov Disord*. 25 (12): 1924-1928. However, very high doses of coenzyme Q<sub>10</sub> have been reported as causing some adverse side effects, mostly gastrointestinal in nature. Clinical trials using the observed safe level risk assessment method indicate strong safety levels at intakes of 1,200 mg per day. Hathcock JN, Shao A (2006). "Risk assessment for coenzyme Q<sub>10</sub> (Ubiquinone)". *Regul Toxicol Pharmacol*. 45 (3): 282-288.

Peak levels of coenzyme Q<sub>10</sub> in the blood are normally reached two (2) to six (6) hours after oral ingestion, with some studies showing a second peak around twenty-four (24) hours after oral ingestion. Approximately half of the ingested coenzyme Q<sub>10</sub> is eliminated or used by the body within thirty-three (33) hours. Tomono, Y; Hasegawa, J; Seki, T; Motegi, K; Morishita, N (1986). "Pharmacokinetic study of deuterium-labeled coenzyme Q<sub>10</sub> in man". *International journal of clinical pharmacology, therapy, and toxicology*. 24 (10): 536-541.

Due to its fat soluble, rather than water soluble, nature, supplementation and absorption of coenzyme Q<sub>10</sub> by the human body is somewhat difficult; however, nanotechnology to reduce the size of the particles, emulsions in soybean oil using softgel capsules, and the addition of other chemicals to increase its solubility in water have all proven somewhat successful in increasing the body's absorption rate of coenzyme Q<sub>10</sub>.

Despite its relative difficulty in absorption, coenzyme Q<sub>10</sub> is the third most widely sold dietary ingredient in the United States, being surpassed only by Omega-3 and multi-vitamins. Natural means of increasing coenzyme Q<sub>10</sub> levels include adding or increasing the dietary intake of the coenzyme Q<sub>10</sub> rich parts of certain animals, such as beef, chicken and pork hearts and livers, as well as, certain types of fish, such as sardines and red flesh rich species. Soybeans, olive oil, parsley, perilla and grape seeds also provide very good sources of coenzyme Q<sub>10</sub>. However, frying foods reduces coenzyme Q<sub>10</sub> content by as much as 14 to 32%. Weber, C; Bysted, A; Hllmer, G (1997). "The coenzyme Q<sub>10</sub> content of the average Danish diet". *International journal for vitamin and nutrition research. Internationale Zeitschrift fur Vitamin- und Ernährungsforschung. Journal international de vitaminologie et de nutrition*. 67 (2): 123-129.

(k.) **Additional Resources for Information on Rhabdomyolysis**

Additional information on rhabdomyolysis can be obtained from the following articles:

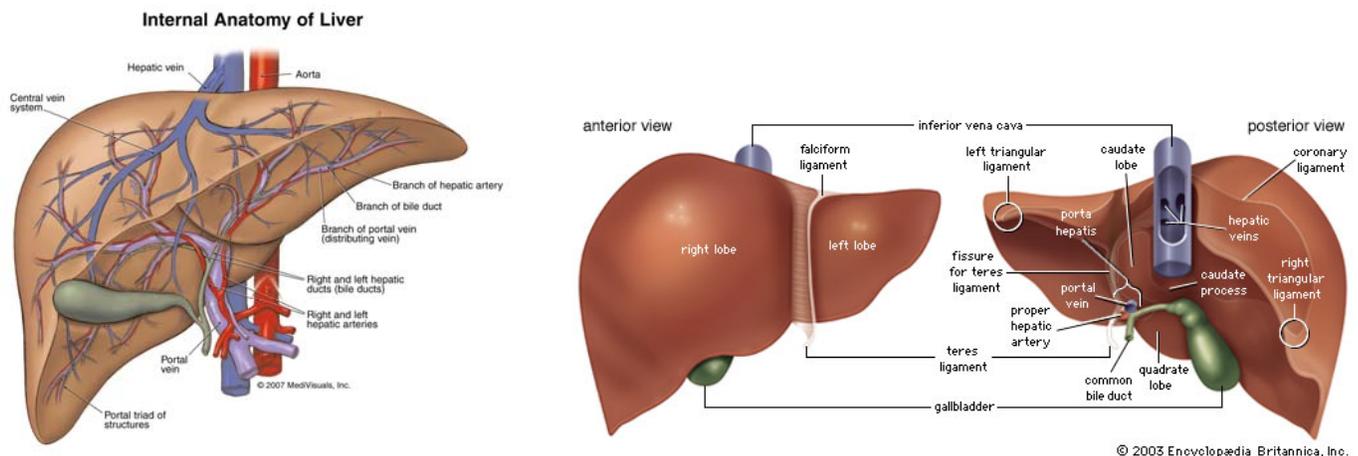
- Senert R, Kohl L, Rainone T, Scalea T. (1994). "Exercise-induced rhabdomyolysis." *Ann Emerg Med*. 23:1301-1306. (<http://www.charlydmiller.com/LIB04/1994/exerciserhabdo.html>)
- "Effects of Conditioning on Exertional Rhabdomyolysis and Serum Creatine Kinase after Severe Exercise," *Enzyme*. (1981) 26:177-181.
- "Catastrophic Medical Events with Exhaustive Exercise: White Collar Rhabdomyolysis," *Kidney International*. (1990) 38:709-719.
- "Myoglobinaemia and Endurance Exercise: A Study of Twenty-Five Participants in a Triathlon Competition," *American Journal of Sports Medicine*. (1984) 12:113.
- "Myoglobin, Rhabdomyolysis, and Marathon Running," *Ortho Journal of Medicine*. (1978) 188:463-472.
- (<http://www.sportsinjurybulletin.com/archive/rhabdomyolysis.html>)

#### 4. Secondary/Contributing Cause of Death – Liver Steatosis

Although the physiological and biological functions of the kidneys and the interplay between the human body's heart, kidneys, muscle cells and liver has been covered by previous sections of this report, certain aspects will be repeated or expounded upon in this section.

During the forensic analysis of Jones' renal/genitourinary system, liver and spleen, Dr. Dye noted:

The right kidney weighs 185 grams and the left kidney weighs 200 grams. Each kidney is composed of tan cortex surrounding maroon pyramids. The kidneys are free of cyst, mass, scar, hemorrhage, abscess, and stone. The ureters are not dilated. The bladder mucosa is smooth and cream colored. The bladder contains scant brown urine. The prostate is not enlarged. . . . The kidney section is free of birefringent polarizable material. The arteriolar walls are not thickened. . . . . [The liver section reveals] roughly 80% of the hepatocytes are involved by macro and micro steatosis. No significant inflammation or fibrosis is detected.



#### (a.) What Caused the Hepatocyte Steatosis Within Jones' Liver?

Notably, 80% of the hepatocytes (primary functioning structures within the liver) in Jones' liver revealed both macro and micro steatosis. Steatosis describes the abnormal retention of lipids/fats within a cell, also known as adipose degeneration. Steatosis impairs the cell's ability to synthesize and eliminate triglyceride fats. Macro vesicular steatosis describes involvement to such an extent as to distort the cell's nucleus; whereas, microvascular steatosis does not display gross involvement sufficient to distort the nucleus. Large accumulations can disrupt cell functions and cause the cell to rupture. Side effects of liver steatosis include increased release or failure to metabolize certain toxins, improper release of blood pressure signaling mechanisms and anoxia.

Here, the involvement of over three-fourths (3/4) of Jones' liver in both macro and micro steatosis provides significant additional insight into the reasons why Jones' muscle cells were generating extraordinary amounts of energy via anaerobic metabolism with premature muscle cell death resulting in rhabdomyolysis, rapid movement into lactic acidosis and failure to regulate his blood pressure.

The steatosis within Jones' liver likely reduced his liver's ability to produce

Liver steatosis is commonly caused by overabundance of lipids in the blood and sometimes results from alcoholism over a long period due to the large amount of energy resulting from the breakdown of ethanol. Energy produced from ethanol metabolism produces large amounts of energy in the form of NADH which signals the liver cells to inhibit the breakdown of fatty acids. Simultaneously, fatty acid synthesis is increased, thus causing an oversupply of lipids which are then deposited into the liver's cells.

Although, more commonly associated with long term alcoholism and Jones did have a history of alcohol abuse, it is more likely the liver steatosis revealed upon autopsy was the combined result of the massive lipid release associated with the extraordinary muscle cell destruction associated with rhabdomyolysis, the extraordinary amount of energy being produced in the form of NADH by Jones' aerobic respiration being supplemented by an equally extraordinary amount of energy from anaerobic metabolism and the very high levels of myoglobin present in Jones' blood, any or all of which could have degenerated or interfered with lipid regulation mechanisms in the liver cells and allowed the deposit of excess lipids in the liver cells.

Typically, the high lipid content of the cells will make the liver less bright on a CT Scan; however, here the CT Scan of Jones' pelvis and abdomen was performed without contrast, thus preventing a differential observation.

**(b.) How Did the Hepatocyte Steatosis Contribute to Jones' Death?**

If ADP levels inside the muscle cells become too high or the cells require a short, intense burst of energy, then the body uses an enzyme induced fermentation process to break down phosphocreatine ("PCr"), which is the most readily available energy source due to its localized storage inside the muscle cell. Cells heavily rely upon PCr during intense bursts of muscle use because, unlike other resources used for ATP production, PCr is quickly accessed and used to restore and maintain ATP levels during intense exercises which require explosive bursts of muscle use like that associated with Jones' very quick pace during the 1<sup>st</sup> Round of his Bout with Palmer.

The breakdown of PCr anaerobically produces the phosphate necessary to convert ADP back into ATP for the cell's use in satisfying energy demand. However, the body's stores of PCr are extremely limited and can only support a muscle cell's ATP levels for two (2) to ten (10) seconds in the absence of any other sources of ATP. The energy provided by Jones' stores of PCr provided just enough energy to let Jones throw his first five (5) or so punches. Normally, ATP is also available from other sources, which allows the PCr to be major energy source in the first minute or so of strenuous physical activity; however, the steatosis in the vast majority of Jones' liver prevented the liver from delivering the excess supply of ATP, glucose and glycogen to moderate the cells' consumption of their PCr stores. Thus, once the muscle cells very quickly consumed all of their PCr, the cells had to rely almost exclusively on less efficient and less expedient anaerobic respiration using glycogen from the cells' surrounding area. The major advantage of the less efficient anaerobic pathway is that it more rapidly provides ATP in muscle cells by utilizing locally available glycogen in the surrounding muscle tissue. Other than PCr, it is the fastest way to resupply muscle ATP levels.

The anaerobic glycolysis of locally sourced glycogen likely supplied most of the energy Jones' muscle cells needed for his intense activity during the next 30 seconds to 2 minutes; however, Jones' muscle cells now faced a critical problem. In addition to being 19 times less efficient at energy (ATP) production than aerobic respiration, another major disadvantage of anaerobic metabolism is that it cannot be sustained for long periods, since the accumulation of lactic acid in surrounding interstitial muscle decreases the blood's pH and inactivates key enzymes required by the glycolysis pathway leading to further inefficiencies and fatigue. The lactate released from muscle cell could ordinarily be taken up by the liver and converted to glucose again (Cori Cycle), or it can be used as a fuel by the cardiac muscle directly or by less active skeletal muscles away from the actively contracting muscle; however, Jones' activity during the Bout was demanding maximum output from nearly every type of muscle cell in his body, so there was no less active skeletal muscle, and worse yet, the steatosis in Jones' liver was preventing the

liver from both reducing the levels of lactic acid and from delivering extra glucose and glycogen to the energy starved muscle cells.

Depletion of Jones' muscle glycogen and other sources of producing muscle cell energy caused him to severely fatigue at the end of the 1<sup>st</sup> Round. Since Jones' breathing changes as a result of acid-base imbalance had long since placed him past his anaerobic threshold and Jones' liver was unable to process and convert the lactic acid back into usable glucose, Jones' respiratory system could not adequately compensate for the metabolic acidosis now ravaging his muscle cells and he was left with very few available resources for the muscle cell energy production necessary to make it through the 2<sup>nd</sup> Round.

In normally healthy persons, both the oxidative enzymes involved in the Krebs cycle oxidation of glucose and the lipoprotein lipase needed to convert triglycerides to fatty acids are typically increased through training regimens; however, the steatosis in over 80% of Jones' liver prevented Jones' body from properly responding to training by increasing its levels of oxidative enzymes. Therefore, when the bell rang to begin the 2<sup>nd</sup> Round, Jones' muscle cells were being lead into a battle in which he would once again demand maximum output, but this time ask the muscle cells to perform both without oxygen for aerobic energy production and without sufficient localized PCr, glucose or glycogen for anaerobic energy production. Being the brave and valiant warriors that his muscle cells were, they likely responded by also supplementing their energy production with the only mechanism for energy production left available to them – self-cannibalization (i.e. self destruction) to provide the substrate necessary for glucose production in the liver.

Remembering that the process of generating energy from ATP happens in two steps: First, ATP is converted to ADP (adenosine diphosphate), then converted to AMP (adenosine monophosphate). ADP can be converted back to ATP with the help of the creatine kinase enzyme. When excess amounts of AMP accumulate in the muscle cell, the cell responds through the increased intake of glucose, which along with the cell's other uses of glucose for energy production soon depletes the blood's supply of glucose, unless the liver is able to increase glucose production to a level matching the muscle cells' increased consumption.

In order for the liver to generate more glucose, it needs a substrate, which in the short term is provided by the self-cannibalization of muscle cells. During brief periods of energy depletion, human muscle cells quickly adapt by breaking down muscle proteins and converting them into a basic amino acid, alanine. Muscles then rely on a novel mechanism that involves an exchange system with the liver, the so-called glucose-alanine cycle. The alanine, derived from muscle protein, is released into the blood and shipped to the liver to be utilized for energy generation. The liver can then generate more glucose from the alanine through gluconeogenesis, while exporting the waste product, urea, to the kidneys for excretion. This normally also allows the liver to regenerate some ATP to help satisfy its own energy needs, which are very large during such stressful conditions. The glucose produced in the liver is then shipped via the bloodstream to the muscle cells, which eagerly take it up to generate more ATP for their own use.

While the foregoing self-cannibalization-glucose-alanine cycle works well for normally healthy people, it would have been a total disaster when added to the multitude of other processes already taking place inside Jones' critically stressed muscle, renal, respiratory and cardiac systems at the beginning of the 2<sup>nd</sup> Round. Accordingly, a partial list of the impaired liver related components of Jones' perfect storm, each of which were independently tolerable but combined into a lethal torrent resulting in a cascading systems failure, are as follows:

- 1.) At the start of the 2<sup>nd</sup> Round of the Bout, many of Jones' muscle cells had already been in anaerobic respiration so long that they had consumed all of their surrounding energy sources and were both so oxygen starved and energy starved that they had already died and released copious amounts of potassium ( $K^+$ ) and highly toxic myoglobin into the bloodstream.

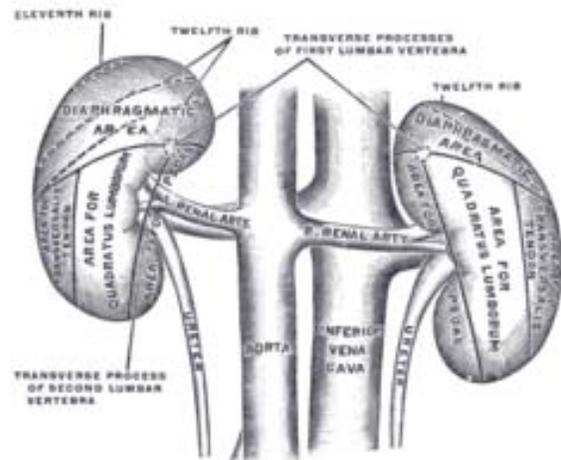
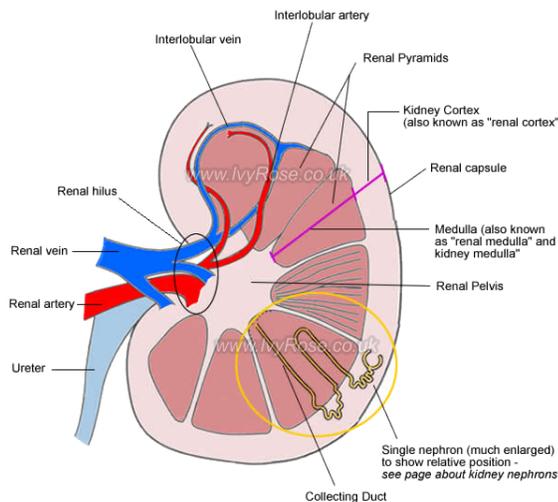
- 2.) The excessive anaerobic energy production required by Jones' high level of activity in the 1<sup>st</sup> Round resulted in the production of so much lactate and consequential lactic acid that Jones' steatosis impaired liver could not possibly begin to remove and convert enough lactic acid into ATP to both alleviate the dangerously high lactic acid level and resupply critically needed glucose, glycogen and ATP to the remaining muscle cells.
- 3.) The compensating response from Jones' kidneys was now being impaired by the death of epithelial cells in the renal tubules due to toxic exposure to myoglobin released from dead muscle cells, thus Jones' kidneys could not adequately compensate for the rising levels of lactic acid.
- 4.) Jones' breathing rate was rapid and uncontrolled, as is the case for many Contestants with little experience and who are competing more from instinct and emotion rather than training and calculated movement; thus, Jones' respiratory system was not only unable to fully compensate for the metabolic acidosis, but also failing to provide the oxygen necessary for his remaining muscle cells to cycle out of anaerobic energy production and back into aerobic energy to relieve both oxidative pressures on the cells and stop the rising lactic acid level by ceasing lactate production.
- 5.) The failure of multiple systems to compensate for the lactic acidosis caused additional muscle cells to die from the disruption of their energy generation process, thus exacerbating the stress on Jones' systems by releasing additional potassium ( $K^+$ ) and highly toxic myoglobin into the bloodstream.
- 6.) Jones' continued demand for energy from his muscle cells in the face of the foregoing circumstances left his body with no but to engage in the self-cannibalization-glucose-alanine cycle in hopes of gaining more glucose and ATP from the liver to sustain the remaining muscle cells' output; however, the death of the muscle cells through self-cannibalization only further increased the levels of potassium ( $K^+$ ) and myoglobin in Jones' bloodstream.
- 7.) The highly unstable, increasingly unregulated and completely abnormal environment outside the muscle cell walls provided the necessary reagents to permit formation of the ferryl form of myoglobin ( $Mg^{+4}$ ) (ferrihemate), which is a highly toxic reactive agent that begins breaking down the fatty acids in the outer cell wall of the muscle cell. Once the fatty acids in a muscle cell wall are broken down by exposure to toxic ferryl myoglobin ( $Mg^{+4}$ ), the cell rapidly disintegrates. Because the cell wall is no longer impermeable to ions, large amounts of calcium ( $Ca^{2+}$ ) start rushing into the cell, and soon afterward the muscle cell dies. The death of yet more muscle cells at the hands of the toxic ferryl myoglobin ( $Mg^{+4}$ ) releases still more potassium ( $K^+$ ) and myoglobin into Jones' bloodstream.
- 8.) And so the process continued in a dangerously escalating cycle of continuing muscle cell destruction, resulting in continued overloading of Jones' various compensating organ systems until the potassium ( $K^+$ ) levels rose to a point of interfering with the proper depolarization of Jones' cardiac cells which led to a decrease in blood flow to the very muscle cells so desperately in need of oxygen and nutrients necessary to stave off their own death and to the liver, kidneys and lungs charged with various compensating responses necessary to restore total body homeostasis.

- 9.) The foregoing fatal feedback loop continued, in whole or in part, until Jones' systems were all overwhelmed and Jones' heart went into cardiac arrest due to uncontrollable hyperkalemia onset by the exertional rhabdomyolysis.

## 5. Secondary/Contributing Cause of Death – Acute Renal Failure

The extensive details of the function of the kidneys, how the renal system's functions interplay with and affect the various functions of muscle cells and other organ systems, and why Jones' renal system failed is extensively covered above in Section IV (F)(2) of this report; therefore, such will not be covered in great detail by this section.

### (a.) How Does the Blood Flow Through the Kidneys for Filtration?



The kidneys receive blood from the renal arteries, left and right, which branch directly from the abdominal aorta. Despite their relatively small size, the kidneys receive approximately 20% of the cardiac output. Each renal artery branches into segmental arteries, dividing further into interlobar arteries which penetrate the renal capsule and extend through the renal columns between the renal pyramids. The interlobar arteries then supply blood to the arcuate arteries that run through the boundary of the cortex and the medulla. Each arcuate artery supplies several interlobular arteries that feed into the afferent arterioles to supply blood to the glomeruli. The interstitium (or interstitium) is the functional space in the kidney beneath the individual filters (glomeruli) which are rich in blood vessels. The interstitium absorbs fluid recovered from urine. Various conditions, such as excess levels of myoglobin in the blood, alcohol or drug abuse and highly acidic blood, can lead to scarring and congestion of this area, resulting in kidney dysfunction and failure. After filtration occurs the blood moves through a small network of venules that converge into interlobular veins. As with the arteriole distribution, the interlobular veins follow the same pattern to supply blood to the arcuate veins then back to the interlobar veins which come to form the renal vein exiting the kidney for transfusion of blood.

The kidney participates in whole-body homeostasis, regulating acid-base balance, electrolyte concentrations, extracellular fluid volume, and regulation of blood pressure. The kidney accomplishes these homeostatic functions both independently and in concert with other organs, particularly those of the endocrine system. Various endocrine hormones coordinate these endocrine functions; these include renin, angiotensin II, aldosterone, antidiuretic hormone, and atrial natriuretic peptide, among others. Many of the kidney's functions are accomplished by relatively simple mechanisms of filtration, reabsorption, and secretion, which take place in the

nephron. Filtration, which takes place at the nephrons of the renal corpuscle, is the process by which cells and large proteins are filtered from the blood to make an ultra-filtrate that will eventually be discharged as urine. The kidney generates 180 liters of filtrate a day, while reabsorbing a large percentage, allowing for only the generation of approximately 2 liters of urine. Reabsorption is the transport of molecules from this ultra-filtrate back into the blood. Secretion is the reverse of reabsorption whereby molecules are transported in the opposite direction, from the blood into the urine. The kidneys excrete a variety of waste products produced by metabolism. These include the nitrogenous wastes urea, from protein catabolism, and uric acid, from nucleic acid metabolism.

## 6. Secondary/Contributing Cause of Death – Dehydration

### (a.) What is Dehydration?

Dehydration is a condition caused by the body's excessive loss of fluids. There are basically three (3) types of dehydration:

- Hypotonic or hyponatremic (loss of electrolytes, sodium (Na<sup>+</sup>) in particular)
- Hypertonic or hypernatremic (primarily loss of water)
- Isotonic or isonatremic (equal loss of water and electrolytes) (most common in humans)

### (b.) How Does Dehydration Occur?

Dehydration can occur myriad ways but most often through failure to consume enough water to offset losses from physical exercise, high protein diets, use of drugs or supplements having a dehydrating effect, and over-exposure to heat. Once a person becomes dehydrated they can be rehydrated relatively quickly just by drinking water. Rehydration begins within 30 minutes of consuming water. Rehydration begins almost immediately upon administration of fluids via an IV.

### (c.) What are the Symptoms of Dehydration?

Dehydration symptoms generally become noticeable after a person loses more than two percent (2%) of their total body water volume and the symptoms include the following listed in conjunction with approximate stage of dehydration:

#### Two Percent (2%) or Greater Fluid Loss

(Athletes can experience a performance loss of up to thirty percent (30%))

- |  |  |
|--|--|
| <ul style="list-style-type: none"> <li>• Lack of Sweat During Warm-ups or Exercise</li> <li>• Low endurance</li> <li>• Rapid heart rate</li> <li>• Elevated body temperature</li> <li>• Rapid onset of fatigue</li> <li>• Thirst and discomfort</li> <li>• Loss of appetite</li> <li>• Dry skin</li> <li>• Constipation</li> <li>• Decreased urine volume</li> </ul> | <ul style="list-style-type: none"> <li>• Irritability</li> <li>• Headache</li> <li>• Dry mouth</li> <li>• Dizziness when standing</li> <li>• No urine output in a 24 hour period</li> <li>• Increased heart rate</li> <li>• Increased blood pressure</li> <li>• Increased respiration rate</li> <li>• Abnormally dark or amber colored urine</li> <li>• Unexplained tiredness</li> </ul> |
|--|--|

**Two Percent (2%) to Five Percent (5%) or Greater Fluid Loss**  
(Athletes can experience a performance loss of up to fifty percent (50%))

- Lack of tears when crying
- No urine output

- Confusion

**Five Percent (5%) or Greater Fluid Loss**

(Athletes can experience a performance loss of seventy-five percent (75%) or greater)

- Lethargy or extreme sleepiness
- Tingling sensation or lack of sensation in legs or arms

- Seizures or fainting

**Ten Percent (10%) to Fifteen Percent (15%) or Greater Fluid Loss**

(Athletes can experience a performance loss of one hundred percent (100%))

- Muscle spasms
- Skin shrinkage

- Dim or blurry vision
- Delirium

**Fifteen Percent (15%) or Greater Fluid Loss**

(Athletes can experience a performance loss of one hundred percent (100%))

- **Organ Failure & Death**

**(d.) What Are the Tests for Dehydration?**

Other than blood and urine tests, there is not a really good way to definitely test for a person's level of dehydration; however, the following are some simple tests for rough estimation of whether a person is dehydrated:

- i. **Skin Pinch Test** – Pinch the skin on the back of your hand in a way that raises it into a tent formation and hold it for a few seconds in that position, then release it. The skin should quickly flatten out and return to normal if you are not dehydrated. If the skin is slow to flatten and return to normal, then the person is experiencing some level of dehydration.
- ii. **Capillary Nail Refill Test** – Apply pressure for a few seconds to the nail bed by pinching a finger or thumb for several seconds. The nail bed should turn white from pressing the blood out of the capillaries in the surrounding tissue. Remove the pressure and a pink color should return within 2 seconds or less, if it does not, then the person is experiencing some level of dehydration.
- iii. **Urine Frequency Test** – One of the best tests for proper hydration is the frequency of urination. If the bladder becomes full to the point of making the person feel like they need to urinate every three (3) to five (5) hours, then it is likely the person is sufficiently hydrated for normal activities (as opposed to strenuous exercise or activities in very hot or humid environments).
- iv. **Urine Color Test** – Another one of the best checks for dehydration is to observe the color of your urine. Urine in properly hydrated individuals is normally clear or very lightly colored. Increasing darkness, especially yellow and amber or brownish shades, indicate some level of dehydration and possibly other medical problems.

The problem with each of the foregoing tests is that they will only indicate very dehydrated conditions, generally only those levels greater than two percent (2%) to five (5%), while failing to detect lesser dehydration levels still presenting a danger to Contestants.

The Commission's investigation here was unable to determine either the time or color of Jones' last urination prior to the Bout. Historically in all Combative Sports, there has been an increased likelihood Contestants in any weight classes with a designated maximum weight would dehydrate in order to "make weight" at the time of weigh-in; therefore, dehydration of Contestants in all designated maximum weight classes has been of particular concern and an area of emphasis for the Commission. In this regard, Reg. §1.33 specifically provides the Commission with authority to disqualify any Contestant who shows evidence of dehydration or use of other unsafe methods to "cut weight."

Contestants in the Heavyweight Divisions have not traditionally been a concern for the Commission and less emphasis on precautionary testing and observation has been applied to Heavyweight Contestants because such Contestants objectively had no incentive to engage in any unsafe weight loss or "weight cutting" practices known to cause dehydration with Contestants in lower weight classes. Jones' case is reminder to all persons involved in Combative Sports that all Contestants, even those in the Heavyweight Division, can experience dehydration prior to a Bout and observation and testing for dehydration should remain vigilant throughout all weight divisions.

**(e.) What Are the Treatments for Dehydration?**

**(i.) Oral Rehydration Therapy**

Mild dehydration can be treated with oral rehydration therapy, which generally consists of drinking several liters of water over the course of several hours, depending on the level of dehydration.

**(ii.) Administration of Intravenous Fluids – Fluid Expansion & The Fluid Challenge**

Severe dehydration requires prompt medical attention, such as was received by Jones in this case. Although the speed of fluid replacement depends on all of the specific variables associated with each patient's condition, the initial administration of fluids in the emergency medical environment involves initial fluid expansion called the "fluid challenge" period and is distinguished from the following period referred to as the "maintenance administration of fluids" period. During the "fluid challenge" phase, large amounts of fluid are administered in a short period of time with such procedure normally involving a nearly constant drip rate and a sixteen (16) or eighteen (18) gauge intravenous needle.

During the entirety of Jones' medical treatment, from the time he was loaded in the ambulance at the Venue until the time of his death almost nine (9) hours later, Jones was administered fluids via an eighteen (18) gauge intravenous needle. The maximum flow rate for a standard green eighteen (18) gauge needle is 105 ml/min. and for a standard procedural eighteen (18) gauge needle is 85 ml/min., thus one (1) liter (1,000 ml) of fluid can be fully administered in approximately ten (10) to twelve (12) minutes using an eighteen (18) gauge needle set at full drip. In critical cases of dehydration or other medical conditions necessitating such, an IV can be started in both arms to double the rate of fluid administration.

Jones had received two (2) liters of normal saline solution prior to arrival at UAMS (one in the ambulance and one while at Saline Memorial). While in the UAMS emergency department, Jones received another four (4) liters of normal saline solution via rapid administration using dual IVs. While Jones was in the UAMS ICU and SICU, Jones began receiving one (1) liter of normal saline solution at the rate of 100 ml/hr. starting at 01:35 hours and one (1) liter of D5W solution at the rate of 150 ml/hr. starting at approximately 03:37 hours. In total, Jones was administered approximately 7.25 liters of fluids during his emergency medical treatment.

**SECTION V**  
**JONES' RELEVANT PERSONAL BACKGROUND INFORMATION AND**  
**HOW IT PLAYED A ROLE IN HIS DEATH**

What follows is a compilation of Jones' relevant medical/forensic background information and activities compiled by the Commission from over two hundred (200) hours of research and investigation; medical and other records review; telephone and in person interviews with Jones' friends, co-workers, teammates, trainers and hotel roommate; and interviews with Commission Officials and numerous resources within the medical and scientific fields. Many of the interviewees, who claimed to know Jones personally, claimed to have known Jones since high school, were at the Event, and saw and talked to Jones prior to his Bout. Interviewees, who trained with Jones in one form or another, described Jones' general views toward supplements/non-prescription training aids as "More Is Better" and believed Jones was likely taking more than the recommended dosages on all of his supplements, although they stated they never actually checked the back of Jones' supplements to see if Jones was using excessive dosages.

The following products and drugs were either known to have been regularly used by Jones or were determined by post mortem chemical analysis to be present in his body and are considered by the Commission to have been primary contributory factors in Jones' death, in that they severely altered the levels of certain elements and compounds within Jones' body and created additional, unnatural stress or damage to Jones' heart, kidneys and liver:

**A. Anabolic Steroids**

The Commission prohibits, without limitation, the use of all substances listed on the World Anti-Doping Agency's ("WADA") list of prohibited compounds and substances. A list of the WADA prohibited list can be found at <http://www.wada-ama.org/en/World-Anti-Doping-Program/Sports-and-Anti-Doping-Organizations/International-Standards/Prohibited-List/>.

**1. Was Jones Using Anabolic Steroids?**

Yes. The Commission's investigation revealed that Jones had both an admitted history of using **Testosterone** and **Trenbolone**, as well as, a confirmed, post-mortem test for the presence of anabolic steroids.

Jones did not like discussing his steroid use; however, Jones did discuss with at least one person that he knew he should not mix the **Creatine** and **Testosterone** and knew he should drink more water while taking steroids.

**(a.) Trenbolone**

The half-life of **Trenbolone** and many other steroids is incredibly short and traces of such can be eliminated from the body through urine both quickly and easily. While very advanced urine testing protocols have been developed to detect various steroid metabolites and extend the detection period, hair specimen testing is preferable to urine testing for all illicit drugs since hair tests permit detection of prior use over a much longer period. However, after conducting extensive research on the issue and obtaining the opinion of several of the best biochemical research scientists and researchers in the field of steroid testing, the Commission understands the respective atomic charge of human hair and almost all steroids and their associated metabolites renders present technology scientifically unreliable for testing hair specimens for the presence of steroids and their metabolites. It is anticipated future technology will permit scientifically reliable hair tests using tests for steroid specific metabolites, by-products and isotopes.

## **2. Did Jones Test Positive for Anabolic Steroids After His Bout?**

Anabolic steroid testing is a highly specialized and very expensive endeavor. Nevertheless, in order to fully investigate every possible cause and contributing factor to Jones' death, the Commission followed the protocols necessary to conduct a prohibited substances test in accordance with the strict standards of the United States and International Olympic Committee and the World Anti-Doping Agency. Jones' post mortem urine specimen was collected by the Arkansas State Medical Examiner's Office, using the applicable chain of custody standards, then turned over to the Commissioner, Jason A. Stuart, who continued the chain of custody protocols for submission of Jones' urine specimen to the UCLA Olympic Analytical Research Laboratory ("UCLA's OARL").

The UCLA Olympic Analytical Research Laboratory is one (1) of only two (2) WADA certified laboratories in the United States. UCLA's OARL subjected Jones' specimen to a full steroid screen for the World Anti-Doping Agency ("WADA") prohibited list of compounds. The Commission also requested UCLA's OARL conduct a carbon isotope ratio test on Jones' urine specimen; however, the effects of Jones' rhabdomyolysis and overall dehydrated condition prevented the collection of a large urine specimen from Jones' body during the autopsy, thus the volume of Jones' urine specimen remaining after the full WADA screen was insufficient to also permit a carbon isotope ratio test for certain other performance enhancing drugs or methods of blood doping.

The anabolic steroid test report came back from the UCLA Olympic Analytical Research Laboratory with a positive test for several anabolic steroids.

## **3. What Anabolic Steroids Were Found in Jones' Post-Mortem Urine Sample?**

The UCLA Olympic Analytical Research Laboratory found Jones' urine sample to contain the following steroids, metabolites and other substances prohibited by the Commission's regulations:

(a) **Boldenone (1,4-androstadiene-3-one-17 $\beta$ -ol) & Boldenone Metabolites (5 $\beta$ -androst-1-en-17 $\beta$ -ol-3-one)**

Boldenone (1,4-androstadiene-3-one-17 $\beta$ -ol), also known under the trade names **Equipoise**, **Ganabol**, **Equigan** and **Ultragan**, is an anabolic steroid developed for veterinary use and has a low androgenic potency. In the United States Boldenone is not indicated for use in humans and is only available through veterinary clinics and the illegal, black market trade. Boldenone increases nitrogen retention, protein synthesis, appetite and stimulate the release of erythropoietin in the kidneys. Erythropoietin is the hormone that regulates red blood cell production and also has other known biological functions, such as playing an important role in the brain's response to neuronal injury and the body's wound healing process.

(b) **Nandrolone Metabolite 19-Norandrosterone (at a concentration higher greater than 25.0 ng/mL)**

**19-Norandrosterone** ("19-NA") is a Nandrolone and 19-norandrostenedione metabolite. 19-Norandrosterone is created as a byproduct of Nandrolone via the 5-alpha reductase enzyme and is on the list of substances prohibited by the World Anti-Doping Agency since it is a detectable metabolite of Nandrolone.

All anabolic/androgenic steroids promote muscle growth primarily via the cellular androgen receptor. Nandrolone has approximately two to three times greater affinity for the androgen receptor than does Testosterone. Testosterone converts to the more active steroid dihydrotestosterone with a three to four times greater affinity for the androgen receptor upon interaction with the 5-alpha reductase enzyme in targeted tissues such as the skin, scalp, prostate, central nervous system and liver. Meanwhile, Nandrolone converts to the much weaker steroid, dihydronandrolone, but with a very site specific targeting of the muscle tissues.

Nandrolone also differs from Testosterone in that Nandrolone is converted by the aromatase enzyme to estradiol, an active estrogen, at only 20% of the rate of Testosterone conversion; therefore, Nandrolone is much less estrogenic than Testosterone and as such, is much preferred by athletes seeking to reduce the estrogenic side effects of steroid use, such as water retention (which is likely a contributing cause of Jones' dehydration), increased fatty tissue deposits and gynecomastia (abnormal growth of large mammary glands in males).

In addition to metabolizing caffeine as discussed below, subsets of the cytochrome P450 enzymes also play important roles in the synthesis of steroid hormones (steroidogenesis) by the adrenals, gonads, and peripheral tissue: CYP11A1 (also known as P450<sub>scc</sub> or P450<sub>c11a1</sub>) in adrenal mitochondria effects "the activity formerly known as 20,22-desmolase" (steroid 20 $\alpha$ -hydroxylase, steroid 22-hydroxylase, cholesterol side-chain scission). CYP11B1 (encoding the protein P450<sub>c11 $\beta$</sub> ) found in the inner mitochondrial membrane of adrenal cortex has steroid 11 $\beta$ -hydroxylase, steroid 18-hydroxylase, and steroid 18-methyloxidase activities. CYP11B2 (encoding the protein P450<sub>c11AS</sub>), found only in the mitochondria of the adrenal zona glomerulosa, has steroid 11 $\beta$ -hydroxylase, steroid 18-hydroxylase, and steroid 18-methyloxidase activities. CYP17A1, in endoplasmic reticulum of adrenal cortex has steroid 17 $\alpha$ -hydroxylase and 17,20-lyase activities. CYP21A1 (P450<sub>c21</sub>) in adrenal cortex conducts 21-hydroxylase activity. CYP19A (P450<sub>arom</sub>, aromatase) in endoplasmic reticulum of gonads, brain, adipose tissue, and elsewhere catalyzes aromatization of androgens to estrogens.

### (c.) Nandrolone Metabolite 19-Noretiocholanolone

**19-Noretiocholanolone** ("19-NE") is a by-product of Nandrolone and together with 19-Norandrosterone, is one of the two main indicators used to prove the illegal use of Nandrolone by humans. Traces of 19-norandrosterone and 19-noretiocholanolone can be naturally produced by the human body, thus present in urine. However, in the present case, Jones had 19-norandrosterone levels greater than 25.0 ng/mL, which is more than twelve (12) times the WADA cutoff level and seventy-five (75) times the upper level naturally produced by the human body.

Several cases within the last few years have generated questions about the appropriateness of the official International Olympic Committee cutoff level, which is 2.0 ng/mL of 19-NA in male urine samples. In 1997, several athletes in France had concentrations of Nandrolone metabolites very close to the limit of 2.0 ng/mL. At that time, a debate took place about the capability of the human male body to naturally produce these metabolites without any intake of Nandrolone or related compounds. A 1998 study, "Urinary excretion of 19-norandrosterone of endogenous origin in man: quantitative analysis by gas chromatography – mass spectrometry" *Journal of Chromatography B: Biomedical Sciences and Applications*, Vol. 721, Issue 2, 22 January 1999, pp. 301-307, demonstrated the endogenous or naturally produced level of 19-norandrosterone in human males is within a range of .01 to .32 ng/ml, thus justifying the International Olympic Committee's threshold limit of 2.0 ng/mL, as such is the geometric mean of the study's samples plus four (4) standard deviations.

Another study, "Quantification and profiling of 19-norandrosterone and 19-noretiocholanolone in human urine after consumption of a nutritional supplement and norsteroids" *Journal of Analytical Toxicology*, Vol. 29, Issue 2, March 2005, pp. 124-134, demonstrated that many over-the-counter nutritional supplements can contain numerous prohibited anabolic steroid contaminants in addition to those specifically prohibited substances disclosed by the manufacturer. In fact, the nutritional supplement tested in the study revealed seven anabolic steroid contaminants in addition to the six listed by the manufacturer.

It is important to note that a Contestant's use of any anabolic steroids and other substances on the WADA prohibited list, including their metabolites, are prohibited and violates the Commission's Regulations. In the Commission's practice, it is never an excuse or justification for a Contestant's use that such anabolic steroid is sold over-the-counter or the steroid or another substance producing the steroid's associated metabolites is "unknowingly" contained in a readily available nutritional supplement.

Anabolic steroid use and abuse has been shown to severely damage and have an adverse effect on the endocrine system, blood lipids, liver and kidneys, even resulting in complete renal failure. A Columbia University Medical Center study, “Development of FSGS Following Anabolic Steroid Use in Bodybuilders” *Journal of the American Society of Nephrology*, November 16, 2009, associated anabolic steroid abuse with focal segmental glomerulosclerosis (“FSGS”) and proteinuria in ninety percent (90%) of the test subjects. The study’s full clinical research article can be found at <http://jasn.asnjournals.org/content/early/2009/11/17/ASN.2009040450.full.pdf+html>.

The Columbia University study’s subjects presented with renal insufficiency indicated by mean serum creatinine levels of 3.0 mg/dL and a range of 1.3 to 7.8 mg/dL. Here, Jones had the following blood creatinine levels:

<u>Jones’ Creatinine Levels</u>	
<u>Time</u>	<u>Blood Test Level</u>
22:45 hours	2.7 mg/dL
00:49 hours	3.4 mg/dL
01:00 hours	2.7 mg/dL
03:04 hours	3.8 mg/dL
04:00 hours	4.0 mg/dL
06:17 hours	4.8 mg/dL

The focal segmental glomerulosclerosis (“FSGS”) associated with anabolic steroid use is the formation of scar tissue in parts of the kidneys called glomeruli. As discussed earlier in this report, the glomeruli are the part of the nephron, which serve as the actual filtering mechanism for the body to filter substances into filtrate which is then either reabsorbed for used by the body or excreted through the urine. In this case, Jones had an overabundance of many chemicals and elements, namely Potassium (K<sup>+</sup>), which would ordinarily be filtered and excreted by the kidneys. However, it is probable that Jones’ steroid use combined with his heavy use of nutritional supplements and diuretics such as caffeine had already damaged his kidneys’ filtering ability to some degree; thus, it took a much lower myoglobin and potassium (K<sup>+</sup>) release during rhabdomyolysis to overwhelm Jones’ already impaired kidney function and heart muscle cells.

Additionally, the Columbia University’s study linking increased blood lipids to steroid use provides another explanation for the steatosis found in Jones’ liver and his resultant complications from decreased liver function. Because Jones’ steroid use was not discovered and confirmed until after the Arkansas State Medical Examiner’s Office had completed the autopsy, a complete biopsy and study of Jones’ liver was not performed for the purpose of determining the prevalence of FSGS; however, significant other clinical test results are consistent with the existence of FSGS.

Anabolic steroid use and abuse has been shown to cause enlargement of the heart and changes in the structure of the heart’s left ventricle including left ventricular hypertrophy (thickening of the ventricle walls). De Piccoli B, Giada F, Benettin A, Sartori F, Piccolo E (1991). “Anabolic steroid use in body builders: an echocardiographic study of left ventricle morphology and function”. *International Journal of Sports Medicine* Vol. 12, Issue 4, pp. 408-412. However, athletes, as a general population, have slightly enlarged hearts, even without using steroids. Dickerman RD, Schaller F, McConathy WJ (1998). “Left ventricular wall thickening does occur in elite power athletes with or without anabolic steroid Use” *Cardiology* Vol. 90, Issue 2, pp. 145–148.

During forensic pathological analysis, Dr. Dye noted Jones’ heart and lungs to be as follows:

The coronary arteries are free of dilatation and atheroma. Serial sections of the 500 gram heart reveal maroon myocardium that is free of softening, discoloration, and scar. The left ventricular free wall and

interventricular septum are of similar thickness. The chambers are moderately dilated. The septa are free of defect. The endocardium is free of mural thrombi. The valves are thin, pliant, and normal in form. The aorta contains rare fatty streaks. . . . The myocytes are normal in form. A section of the coronary artery is free of atherosclerosis. . . . The right lung weighs 920 grams and the left lung weighs 785 grams. The lungs are composed of edematous maroon tissue that is free of mass, consolidation, thromboemboli, and emphysema. The trachea and bronchi are patent. The tracheal mucosa is smooth and pink. . . . The alveoli are normal in form. The section is free of significant inflammation and birefringent polarizable material.

Jones aorta displayed very few fatty streaks with his endocardium being free of mural thrombi. In other words, Jones' cardiac arrest was not caused by fatty tissue in his heart or a blood clot obstructing the flow of blood. However, Dr. Dye further noted Jones had an enlarged heart weighing 500 grams (normal males have a 350 to 400 gram heart). Dr. Dye's autopsy also revealed Jones' left ventricular free wall and inter-ventricular septum were of similar thickness without noting the actual measurement. In most cases, left ventricular hypertrophy causes equal thickening of both the left ventricle wall and inter-ventricular septum. The exact etiology of Jones' enlarged heart is unknown; however, it is strongly suspected Jones' use of anabolic steroids and general athleticism were both significant contributing factors.

THE EXCESS MUSCLE MASS JONES GAINED BY THE USE OF STEROIDS ACTUALLY CONTRIBUTED TO MANY OF THE PROCESSES RESULTING IN HIS DEATH. IN ADDITION TO THE KIDNEY, LIVER AND HEART DAMAGE, JONES' EXTRA MUSCLE MASS MEANT THERE WERE MORE MUSCLE CELLS CONSUMING MORE OXYGEN, PCr, GLUCOSE & GLYCOGEN DURING THE BOUT AND MORE MUSCLE CELLS AVAILABLE TO DIE AND RELEASE MYOGLOBIN, K<sup>+</sup> AND Mg<sup>2+</sup> DURING RHABDOMYOLYSIS.

**B. Marijuana** – Tetrahydrocannabinol acid (“THC”) was detected in Jones' urine specimen tested by UCLA Olympic Analytical Lab; however, the quantity of such could not be confirmed due to an insufficient remaining specimen volume. THC was not detected at levels above the test screen cutoff in Jones' blood by the Arkansas State Medical Examiner's Office. Accordingly, the presence of THC in Jones' urine, but not his blood indicates Jones was likely not under the influence of marijuana at the time of the Bout, but had been using the illicit drug sometime within the one to two week period prior to the Bout. The serious harmful effects of marijuana use including, increased stress on the heart and cardiovascular system and decreased brain function, have been voluminously documented, thus will not be delved into further in this report as such harmful effects have been sufficiently reported upon by the general media and other governmental publications to the point where the Commission believes the harmful effects of marijuana use are sufficiently well understood by the general public so as to not require further discussion in this report. Marijuana use is prohibited by the Commission's regulations. For more information on the harmful effects of marijuana and drug use, please visit <http://drugabuse.gov/infofacts/marijuana.html>.

**C. JetAlert Caffeine Pills** – Manufacturer's Declared Active Ingredients in one pill: **Caffeine (200 mg)**. Interviewee stated he saw Jones take at least one 200 mg JetAlert at about 8:00 p.m. the night of the Event. The bottle of JetAlert (200 mg pills) was also found in Jones' gym bag at the Event. It is presently unknown how many pills are left in the bottle. Jones was observed regularly taking the caffeine pills everyday before workouts and often additional doses during workouts too, perhaps as many as 5 or 6 pills over a 2 hour or less workout period. A post mortem analysis of Jones' blood found his caffeine level to be 20 micrograms per milliliter. It is generally accepted that caffeine levels of 12 to 36 micrograms per milliliter are considered therapeutic with levels between 50 and 400 micrograms per milliliter being toxic. Depending on the human body's composition and other circumstances present, caffeine levels above 80 micrograms per milliliter can be lethal.

Caffeine is the most widely consumed psychoactive stimulant on Earth. Caffeine, a methylxanthine, is closely related to theophylline. Caffeine is rapidly and completely absorbed from the gastrointestinal tract and is

detectable in blood plasma 5 minutes after ingestion, with peak plasma levels occurring between 30 to 60 minutes after ingestion. Caffeine is primarily metabolized by the cytochrome P450 (“CYP”) oxidase system in the liver. The plasma half-life of caffeine varies considerably from person to person, with an average half-life of 5-8 hours in healthy, nonsmoking adults. Caffeine clearance is accelerated in smokers; clearance is slowed in persons with liver disease and in the presence of some CYP inhibitors (e.g. cimetidine, quinolones, erythromycin). In addition, the hepatic enzyme system responsible for caffeine metabolism can become saturated at high levels, resulting in a marked increase in serum concentration with small additional doses.

Caffeine directly stimulates respiratory and vasomotor centers of the brain and acts as an adenosine antagonist, resulting in peripheral vasodilatation and CNS stimulation. Caffeine is a potent releaser of catecholamines (norepinephrine and, to a lesser extent, epinephrine) that increases cardiac chronotropic and inotropic activity, bronchodilation, and peripheral vasodilatation. Caffeine is also a phosphodiesterase inhibitor.

In addition to its central nervous system (“CNS”) and cardiovascular effects, caffeine induces a number of metabolic changes, including hyperglycemia (by stimulating gluconeogenesis and glycogenolysis), increased renal filtration, ketosis, and hypokalemia. When ingested chronically in excessive amounts, caffeine produces a specific toxidrome (caffeinism), which consists of primarily CNS, cardiovascular and gastrointestinal (“GI”) hyperstimulation. CNS effects include anxiety, agitation, tremors, seizures and altered mental status. Additionally, pupils are dilated but reactive to light and cardiovascular effects are widened pulse pressure due to positive inotropic effect as well as vasodilation, sinus tachycardia, dysrhythmias (e.g. premature ventricular contractions, supraventricular tachycardias [SVTs], ventricular rhythms and hypotension along with respiratory tachypnea.

**D. Potassium Supplement** Jones consumed a 90 mg Potassium supplement obtained from his roommate around 12:00 hours on the day of the Event, telling his roommate that he wanted to take it because he “didn't want his legs to cramp up” and “it will make his legs be able to go longer.”

**E. Men’s Daily Complete Multi-Vitamin** Jones was not observed taking the multi-vitamins, but was known to take multi-vitamins almost daily and had a bottle of “Men’s Complete Multi-Vitamins” on the nightstand in his hotel room after the Bout.

**F. Apple Juice, Bananas, Orange Juice & Other Dietary Sources** – Jones’ dietary intake of at least 4,507 mg of potassium within the twenty-four (24) hour period immediately preceding his Bout is documented in the detailed timeline in Section III of this report.

**G. Smokeless Tobacco/Dip/Snuff** -- Partner stated Jones was using snuff (Grizzly Wintergreen) during the hours immediately prior to the Bout and while he was walking out and visiting people in the crowd prior to his Bout.

**H. Jack3D from USP Labs** – Manufacturer’s Declared Active Ingredients: **Arginine Alpha-Ketoglutarate; Creatine Monohydrate; Beta Alanine; Caffeine; 1,3-Dimethylamylamine (Geranium [Stem]); Schizandrol A.** Inactive Ingredients: **Citric Acid; Natural Lemon-Lime Flavor; Acesulfame-K; Sucralose; Vegetable Stearate; Silicon Dioxide; Chlorophyll** -- Manufacturer does not declare the actual dosage of each Active Ingredient compound, but does declare the total “Proprietary Blend” of these compounds is 4,145 mg. per 5.55 gram serving. Jones was a daily user of this supplement and was using it while training for this bout. Here is a link to the side effects page of the product’s website <http://www.jack-3d.com/side-effects>.

**I. Creatine from GNC** – Manufacturer’s Declared Active Ingredients in 45 gram serving: **Creatine Monohydrate (5.25 g); Taurine (1 g); Phosphorus (200 mg); Magnesium (60 mg); Sodium (95 mg); Potassium (80 mg).** Inactive ingredients: **Dextrose; Natural Fruit Punch; Citric Acid; Beet Color; Di-**

**Potassium Phosphate; mono-Sodium Phosphate, Dipentene (Limonene).** It is unknown at this time, which of GNC's 4 possible Creatine supplements that Jones was using; however, the foregoing information is for their CreaDrive supplement, which is the most probable product. Another possible supplement is their Creatine Plus product. Manufacturer's Declared Active Ingredients in 6 gram serving: **Creatine Monohydrate (5 g); Taurine (250 mg); L-Glutamine (250 mg); Taurine (250 mg); Glycine (250 mg); L-Arginine (100 mg); L-Methionine (100 mg).**

**J. Water** -- Usually consumed 1 to 3 gallons of water per day. Jones's water came almost exclusively from a Kangen Water Machine at the gym and from a supplier in El Dorado. Kangen Water is an ionized, high alkaline water having a pH of between 8.5 and 9.5. Ionization is achieved by passing the water over 7 platinum coated titanium plates. Jones often carried a big blue container of this water with him on a daily basis. Jones brought 2 gallons of this water from the machine to the Event, but still had a gallon left in his possessions. Jones also had either a 128 oz. or 384 oz. "Big Bubba Keg" brand cooler of this water in his hotel room at the Event, which roommate stated looked like it had been consumed and refilled because it was getting low the night before and had more in it when they inspected his room after the Event. Roommate drank out of this cooler of water too, but stated he thinks he only drank a glass or two from it, so it is unknown how much the Jones consumed.

**K. Protein Supplements** -- Jones was a daily consumer of protein shakes and supplements from various Manufacturers & Products. According to Vanderbilt University, the average sedentary human typically only needs about 1/3 gram of protein for every pound of lean body weight or between 30 and 60 grams of protein per day to replace the amino acids used by the body's daily activities. Houston, Michael. (1992) "Protein and Amino Acid Needs of Athletes" Nutrition Today, Issue No. 27, pp. 36-38. Athletes need slightly more daily protein; however, studies have shown the benefits of additional protein intake, even for strength training athletes, plateaus and there is little benefit from protein intake in excess of 2 grams per kilogram (2.2 lbs) of body weight. For example, a 235 pound, heavily muscled male, such as Jones, would only need a maximum of 175 to 200 grams of protein per day. One whole egg contains approximately 7 grams of protein and a small 6 oz. beef steak contains between 45-50 grams. By contrast, a 17 oz. Myoplex brand protein drink contains 42 grams of protein, a 14 oz. Muscle Milk brand protein drink contains 25 grams of protein and each scoop of most protein powder brands contain between 20 and 25 grams per scoop with most athletes consuming two scoops per drink.

The highly concentrated nature of protein supplements combined with current advertising and marketing campaigns touting the benefits of extra protein for athletes often leads to excessive protein intake for athletes and produces harmful or toxic effects. Most athletes can obtain adequate protein and nutrients by adjusting the type and quantity of foods they eat without the use of protein or nutritional supplements. According to protein expert Dr. Gail Butterfield, PhD, RD, the director of Nutrition Studies at the Palo Alto Veteran's Center and nutrition lecturer at Stanford University, daily diets deriving 30% or more of their calories from protein causes a toxic buildup of ketones. Ketogenic effects created by ultra-high protein intake stress and overwork the kidneys in an effort to rid the body of the toxic levels of ketones. In the process of eliminating and flushing the ketones from the body, the kidneys utilize excessive quantities of water which contributes to dehydration and added kidney and heart stress.

According to Vanderbilt University, A common misconception about excess protein in the diet is that it can cause kidney damage; excess protein cannot cause kidney damage even though it does make the kidneys work harder. When protein is metabolized nitrogen is a byproduct; the kidneys work to remove the extra nitrogen from the body. As of yet, no studies have found a high rate of kidney problems in strength athletes as would be expected if too much protein caused kidney damage. Also, Zaragoza et al. (1987) studied animals with very high protein intakes for more than half their life span and found no serious adverse effects.

High levels of protein intake can lead to increased water loss because the body excretes water to dispose of urea, a substance formed in the breakdown of protein. Water loss coupled with the fact that most athletes loose a

great amount of water through sweat, can lead to dehydration if fluid intake is not properly monitored. An excess of purified protein can, however, take calcium away from bones, thus predisposing one for osteoporosis.

**L. Other Supplements** -- Jones was known to be a large consumer of various supplements and frequented GNC. A list of additional supplements present at Jones's apartment is being compiled by his girlfriend and will be turned over to the family's attorney upon completion.

**M. Alcohol** -- Workout partner claimed Jones did not drink because Jones was a recovering alcoholic with 2 years of sobriety. Jones also worked at South Arkansas Substance Abuse Center as a counselor in training. However, another friend of Jones says he observed Jones consuming a few drinks a couple of times a month, but not ever really being drunk. Neither the roommate nor any one of the persons around Jones on the night of the Bout reported having seen Jones consume any alcohol the night before, day of or night of the Event. No alcohol was sold at the Event and no one observed any alcohol present at the Event. No alcohol was observed in his gym bag by his trainers after the Event. However, Jones blood did contain .005 ml/dL (approximate amount associated with Jones consuming one alcoholic drink before his Bout) of unaccounted for alcohol upon autopsy.

**N. Prior Concussion & Headaches** -- Jones had a concussion in high school, but does not know exactly what year or what "grade of concussion." Jones had not complained to anyone of headaches at any time in the few days prior to the Bout and did not complain of a headache to the roommate or team mates at any time the day before or day of the Event.

**O. Weight** -- Jones actually weighed 232.6 lbs at weigh-ins the night prior to the Event and was not re-weighed the night of the Event. Jones normally weighed between 235 lbs and 240 lbs with such weight being his normal weight for an unknown period, but well in excess of 6 months.

**P. Workouts** -- Jones was an avid weight lifter and did not like running, but did do extensive cardio through sparring, miscellaneous training exercises and participating in/teaching the "Insanity" workout class at the gym before or after his boxing training.

**SECTION VI**  
**REVIEW & ANALYSIS OF OTHER COMBATIVE SPORTS DEATHS IN ARKANSAS**

**A. JACOB GREENWALT – DECEASED 5-JAN-1997**

Jacob Greenwalt (“Greenwalt”) collapsed inside the Ring after the 2<sup>nd</sup> Round of his amateur, Silver Gloves Boxing Bout on 4-JAN-1997 and was transported by ambulance to Baptist Memorial Hospital in North Little Rock, Arkansas. Greenwalt died at the hospital the following day. The Silver Gloves Bout was not sanctioned by the Commission, as amateur boxing did not fall under the Commission’s jurisdiction at the time. Instead, the Bout was sanctioned by the Silver Gloves Association, the president of which is Ray Rodgers. The Contestants took physicals prior to their participation at the Event; however, the physicals did not comply with the more stringent standards and protocols currently used by the Commission.

Neither the Silver Gloves Association nor USA Boxing conducted any investigation or made an official report regarding the incident; thus, the Commission does not have the benefit of those reports and findings. However, the Arkansas State Medical Examiner’s Office (“ASME”) did perform an autopsy and investigation along with the North Little Rock Police Department (“NLRPD”). For the purposes of the present investigation, the Commission obtained the ASME’s records, which included the NLRPD records. Due to the obviousness of Greenwalt’s cause of death in the ASME’s report, the Commission did not obtain Greenwalt’s hospital records.

The ASME’s findings were:

- 1.) Acute left subdural hematoma.
  - Cerebral edema, with transtentorial herniation.
  - Brain death (clinical)
  - Multifocal ischemic changes, cerebral cortex and thalamus
  - Focal acute secondary hemorrhage, brainstem and midbrain
- 2.) Resolving left subdural hematoma.
- 3.) No skull fractures present.
- 4.) Status post organ donation: Absence of heart, lungs, liver, spleen, pancreas, kidneys, adrenals.

**PRIMARY CAUSE OF DEATH:** 1.) Acute subdural hematoma due to blunt force trauma during a boxing match

**Secondary/Contributing Factors:** 1.) Healing subdural hematoma

**Evidence of Old Injury:** Yellow brown discoloration of the dura at the left base of the cranial vault. Sections displayed evidence of prior subdural hematoma formation. Focal areas displayed increased numbers of fibroblasts, along with scattered hemosiderin laden macrophages. Section of left thalamus displayed focal areas of acute secondary hemorrhage, along with early dehiscence of the parenchyma. A section of the pons displayed a wide area of secondary hemorrhage. The areas of old, healing subdural hematoma, appeared to have been present for at least more than a month.

- Evidence of New Injury:**
- 1.) Diffuse, bilateral flattening of the cerebral gyri.
  - 2.) Bilateral grooving of the hippocampal unci along with mild coning of the cerebral tonsils.
  - 3.) Serial coronal sections of the brain revealed multifocal dusky coloration of the cerebral cortex consistent with ischemic damage most pronounced at the distribution of the posterior cerebellar arteries. Similar areas of discoloration in the left thalamus and cerebellum.
  - 4.) Brainstem sections revealed wide area of secondary hemorrhage in the pons.
  - 5.) Midbrain sections revealed extension of the pontine hemorrhage and elongation of the third ventricle.

According to the ASME/NLRPD investigation, Greenwalt was without complaints and in his usual good state of health at around 14:00 hours on 4-JAN-1997. At approximately 14:08 hours on 4-JAN-1997, Greenwalt was engaged in the Bout and suddenly collapsed at the end of the 2<sup>nd</sup> Round or beginning of the 3<sup>rd</sup> Round; however, the Pulaski County Coroner's preliminary information sheet states Greenwalt was described by spectators to have collapsed while throwing a punch during the 2<sup>nd</sup> Round. (NOTE: This factual discrepancy was not deemed significant enough for the Commission to verify or clarify during the present Investigation.) No ambulance was stationed onsite for the Event, thus an ambulance had to be summoned via 911 emergency dispatch. Arrival and transport time records were not available to the Commission for this report; however, Greenwalt arrived at Baptist in North Little Rock via ambulance at 14:26 hours. During the course of hospitalization, Greenwalt was diagnosed with an acute subdural hematoma and brain swelling. Greenwalt developed clinical brain death and was pronounced dead at 09:10 hours on 5-JAN-1997. At the request of the family, organ donation was made prior to referral to the State Crime Lab for autopsy.

The ASME/NLRPD investigation found forensic medical evidence of a previously incurred, healing subdural hematoma to be supported by statements from Greenwalt's father that approximately three (3) months earlier Greenwalt fell and hit his head while engaged in horseplay with his friends. As a result of the impact during horseplay, Greenwalt lost consciousness for about ten (10) minutes. Greenwalt's father reported Greenwalt did not display any signs of nausea, dizziness, or dilated pupils; however, Greenwalt did not receive medical attention after the horseplay injury.

After the horseplay incident, Greenwalt was reported to have participated in training and three (3) other boxing matches. Greenwalt was reported by his family and friends to have been beaten in a boxing match only a few weeks prior to the Bout during which he collapsed and had not been fighting as well since the time he was knocked unconscious during horseplay. Greenwalt was reported to not have received any "substantial" punches during the Bout at which he collapsed.

Mr. Ray Rodgers, the current amateur Arkansas Golden Gloves director, was responsible for supervising the Event and ensuring that a physician examined each Contestant prior to the bout on 4-JAN-1997; however, when questioned by the Pulaski County Coroner's office just two (2) days later, Mr. Rodgers did not know which, if any, physician had examined Greenwalt before the Bout. Mr. Rodgers also stated each Contestant used his own safety equipment, rather than safety equipment supplied by Mr. Rodgers or the Silver Gloves organization.

At the time of his death, Greenwalt was 15 years old and the Pulaski County Coroner's Report lists Greenwalt as 120 lbs. in the Summary Narrative; however, another section of the Coroner's Report states Greenwalt was 108 lbs.. (NOTE: This factual discrepancy was not deemed significant enough for the Commission to verify or clarify during the present Investigation.)

Due to the conclusive nature of Greenwalt's cause of death and other forensic medical evidence supporting Greenwalt's official cause of death, the Commission has not included the specific details of Greenwalt's course of

medical treatment, blood lab results or other information, such as has been provided above for Anthony Jones. Greenwalt's death was officially ruled and accident by law enforcement authorities.

The Commission does not express an opinion on Greenwalt's particular cause of death; however, the Commission does note its concern regarding the following:

- 1.) Neither Mr. Ray Rodgers nor the Silver Gloves organization followed appropriate health, safety and welfare protocols for pre-Bout and post-Bout physicals by qualified medical personnel;
- 2.) Neither Mr. Ray Rodgers nor the Silver Gloves organization followed appropriate health, safety and welfare protocols for medical questionnaires designed to elicit information regarding prior head injuries and periods of unconsciousness, then take such information into account when making a decision on whether or not to allow the Contestant to compete in the Event (e.g. Commission's Combative Sports Medical Report (Pre-Bout) Standard Form CSMR057-2010 Contestant's Questions #3, #4, #5, and #6, as well as Physician's Question #1);
- 3.) Neither Mr. Ray Rodgers nor the Silver Gloves organization followed appropriate health, safety and welfare protocols for retention of medical records on Contestants subsequent to the Bout;
- 4.) Neither Mr. Ray Rodgers nor the Silver Gloves organization followed appropriate health, safety and welfare protocols requiring the presence of a qualified ringside physician during the Bout; and
- 5.) Neither Mr. Ray Rodgers nor the Silver Gloves organization followed appropriate health, safety and welfare protocols for the presence of an on-site ambulance with transport authority together with emergency medical technicians or paramedics with advanced life support systems at ringside during the Bout.

At the time of Greenwalt's death, the amateur Silver Gloves Event did not fall under the Commission's jurisdiction; therefore, Mr. Ray Rodgers was solely responsible for regulation and implementation of the requisite health, safety and welfare protocols, as he deemed necessary or appropriate. However, now, all amateur and professional Combative Sports Events fall under the jurisdiction of the Commission and the Commission intends to increase its enforcement efforts with respect to those amateur Events wishing to obtain an exemption or to be regulated by a statutorily authorized and Commission approved alternate sanctioning body.

**B. BRANDEN TWITCHELL (24 Years Old) - DECEASED 14-FEB-2008**

Branden Twitchell ("Mr. Twitchell") deceased several days after participating in a boxing elimination tournament in Texarkana held in Texarkana, Texas. Mr. Twitchell's medical records are within the possession and control of CHRISTUS St. Michael Health System in Texarkana, Texas. While Ark. Code Ann. § 17-22-204(d) grants the Commission the power to subpoena records in connection with its investigations, the Commission's subpoena powers only extend to Arkansas residents or persons presently located in Arkansas. Further, Mr. Twitchell was not required to be licensed by the Commission under applicable Arkansas statutes in existence at that time; therefore, Mr. Twitchell did not sign a HIPAA release for the Commission. Accordingly, the Commission has been unable to obtain and review Mr. Twitchell's complete medical records for purposes of this Investigation and lacks the financial and other resources necessary to pursue Mr. Twitchell's records through an inter-agency request and legal proceeding in the State of Texas.

Based on the best information available to the Commission through interviews with witnesses and a few nurses, who would only speak off of the record, Mr. Twitchell's death appears to have been caused by an unknown, pre-existing condition which was not disclosed to the Event promoter at any time prior to the Bout. Additional reports indicate Mr. Twitchell did not evidence any external signs of injury prior or subsequent to his participation in the Bout. Although the Event was outside the scope of the Commission's jurisdiction at the time, the Event Promoter did have a board certified physician specializing in the field of neurology at Ringside and the same physician conducted both pre-Bout and post-Bout physicals of the Contestants. Mr. Twitchell was cleared by the neurologist both prior and subsequent to his participation in the Bout.

**C. JEREMY WOOD – DECEASED 18-AUG-2010**

Jeremy Wood ("Mr. Wood") deceased several days after professional wrestling training at Southern Wrestling Association's gym in Newport, Arkansas. Mr. Wood spent several days in a medically induced coma and subsequently died from the cumulative effects of subdural hematoma and cerebral edema. Very few details about the incident were made available to the Commission by those persons directly involved in the incident; however, the incident was not required to be reported to the Commission, since neither amateur nor professional training for any Combative Sport including, wrestling, are regulated by or fall under the jurisdiction of the Commission.

Subsequent to Mr. Wood's death, the Commission did make general, internal inquiries into whether or not training only activities were within the Commission's statutory jurisdiction and if so, whether the Commission possessed sufficient resources to adequately monitor and regulate both amateur and professional Combative Sports training activities on a statewide level. Because the Commission is required to be a "self-supporting commission," the Arkansas legislature makes no appropriation of the state's general funds for use by the Commission to carry out its regulatory duties. Accordingly, the Commission determined it lacks sufficient monetary, manpower and infrastructural resources to regulate both Combative Sports competition Events and Combative Sports training exercises on a continuous, statewide basis; therefore, the Commission did not reach the issue of whether or not it has the authority to regulate training exercises for Combative Sports. As of the publication of this report, the Commission's position on the regulation of Combative Sports training exercises remains unchanged.

**D. ROB PHIPPS – DECEASED 9-JUN-2011**

Robert Dale Phipps ("Mr. Phipps"), age thirty-nine (39), who wrestled under the ring name "Adrian Steel" for the Mid States Wrestling Association, collapsed inside the wrestling ring prior to the start of a wrestling Bout on 9-JUN-2011 in Harrison, Arkansas. While Mr. Phipps' death technically occurred while in competition, after receiving a verbal report from the Event promoter, Jason Jones, during the Commission's 2<sup>nd</sup> Quarter meeting on 21-JUL-2011 and performing some initial investigatory work, the Commission elected not to initiate a full scale, official investigation into Mr. Phipps' death. The Commission's election to not initiate a full scale, official investigation is based on the clear evidence received during the initial investigation indicating:

- 1.) Mr. Phipps suffered a heart attack and collapsed inside the ring prior to his opponent's entry into the ring and later died en route to the hospital;
- 2.) Mr. Phipps' death was not the result of any blows struck or received during competition;
- 3.) Mr. Phipps death was not the result of any type of physical exertion during competition; and
- 4.) Mr. Phipps death is not the result of and did not occur under any conditions involving actual or potential violation of Commission Regulations, such that further investigation would be warranted under the circumstances.

## SECTION VII

### COMMISSION'S PROPOSED ACTION, RECOMMENDATIONS & CONCLUSIONS

This Section provides an analysis of the Commission's operations, procedures and operations, in light of the information learned during the course of this investigation, and recommended actions to improve the health, safety and welfare, as well as, assure a uniform and unbiased regulatory scheme is designed and enforced to improve competition, entertainment and participation for all individuals and interested parties.

#### A. WHAT IS THE ARKANSAS STATE ATHLETIC COMMISSION'S PURPOSE?

The Arkansas State Athletic Commission's primary and controlling duty is the protection of the health, safety and welfare of the Contestants, Officials, and Licensees, as well as, ensuring fair, equal and uniform treatment of Contestants and Licensees, while maintaining the integrity and competitiveness of all amateur and professional Combative Sports in the State of Arkansas. Secondary to the aforementioned duties is the Commission's intent and objective to provide educational resources and easy access to information regarding the risks and benefits of participation in amateur and professional Combative Sports in the State of Arkansas.

Fulfilling the foregoing objectives is neither simple nor easy and the serious dangers to human health and safety associated with the activities under the jurisdiction of this Commission are always foremost in importance during the Commission's decision making processes; thus, the Commission attempts to at all times avoid regulatory or procedural changes in haste subsequent to any tragic event.

The Commission is mindful of those citizens who have expressed their opinion, both prior and subsequent to Anthony Jones' death, that all Combative Sports should be prohibited in the State of Arkansas and that if Combative Sports are permitted to continue, then the Commission should require more proof of a Contestant's experience prior to licensure as a professional. On the opposite end of the spectrum the Commission is faced by an equally opinionated portion of the citizenry which honestly believes the Commission has begun to over-regulate Combative Sports and that such over-regulation runs a substantial risk of driving all Combative Sports, especially those at the amateur level, out of the State of Arkansas.

James Madison was not only our Nation's 4<sup>th</sup> President and an integral framer of the Constitution, but also, a chief architect of the United States' federalist system. President Madison wrote,

If men were angels, no government would be necessary. If angels were to govern men, neither external nor internal controls on government would be necessary. In framing a government which is to be administered by men over men, the great difficulty lies in this: you must first enable the government to control the governed; and in the next place oblige it to control itself.

*The Federalist* No. 51, at 348 (N.Y. Heritage Press ed., 1945). *The Federalist* consists of 85 articles or essays written by James Madison, Alexander Hamilton and John Jay, advocating for ratification of the Constitution.

In this regard it is important for the Commission, and any branch of government for that matter, to remain circumspect and mindful of the dangers of quickly reacting to emotionally charged public outcries for change, whether calling for implementation of more or less strenuous regulation in an industry, without first fully understanding the facts and circumstances and exploring both the actual need for and potential impact of adding to or abolishing laws and regulations, unless prompt implementation of change is absolutely mandated for the protection of human health, safety or welfare.

Here, this Commission has undertaken an extensive review of Arkansas' Combative Sports statutes and regulations, which it is charged to enforce, received and listened to the public's comments and views on Combative Sports and the regulation thereof in Arkansas, and attended national conferences and symposiums to satisfy itself that its regulatory schema is reasonably consistent with the rest of the jurisdictions throughout the United States, while remaining adequately tailored to the unique requirements of Arkansas' citizens and those who chose to visit our State to either participate in or view a Combative Sports Event. During the course of this nearly seven (7) month investigation and report drafting procedure, the Commission has avoided making or implementing any reflexive regulatory changes in response to this unfortunate incident. Instead, the Commission has opted to wait until it has uncovered and come to an understanding of all relevant facts, the latest medical and scientific evidence, and its own capabilities and limitations.

To be clear, while fulfilling its regulatory duties, the Commission receives numerous complaints and public comments regarding the perceived lack of safeguards and perceived burdens implemented by the Commission's Regulations. The Commission always listens to the public with an open mind and sincere desire to maintain a balance between over-regulation and implementation of safeguards for the protection of the health, safety and welfare of the affected persons.

*I know no safe depositary of the ultimate powers of the society but the people themselves; and if we think them not enlightened enough to exercise their control with a wholesome discretion, the remedy is not to take it from them, but to inform their discretion by education.*

*Thomas Jefferson to William C. Jarvis, 1820.*

The Commission believes this report fulfills the obligations imparted upon the Commission by and the Commission's future operations are best conducted in accordance with the foregoing judicious words of President Thomas Jefferson. The Commission hopes this report, while quite lengthy and at times highly technical, will serve not only as an answer for any questions Mr. Anthony Jones' fans, friends and family may have regarding his death, but also, as an educational tool so that Mr. Jones' life will not have been given in vein pursuing a sport he loved and pursued with both love and passion.

## **B. What Is the Short Form Summary & Analysis of This Incident & Incident Response?**

Except for the blood testing recommended hereinafter and based upon the unique circumstances existing within the State of Arkansas, the Pre-Bout and Post-Bout physicals mandated by the Commission's regulations meet or exceed both the practical and medically recommended minimum examination which should be required for Contestants prior to and after participation in a Combative Sports Event in the State of Arkansas.

Because the heart's systolic function tends to remain normal with diastolic functions being slightly altered, an enlarged heart, such as Jones', is not readily or easily detected through routine blood pressure checks, but rather is most often only diagnosed using advanced, Doppler echocardiography. In addition, it was not Jones' enlarged heart, which caused his death; but rather, Jones' death was related to a series of underlying and cumulative medical conditions onset by a set of circumstances unique to Jones, as an individual. Furthermore, both steroid and immediate, I-Stat blood testing, which may have provided some advance indication of Jones' underlying conditions, are prohibitively expensive for administration to every Contestant at every Event. The present elements and observations required by the Commission for Pre-Bout and Post-Bout physicals are specifically

designed to and will detect almost all physical or medical conditions posing an immediate threat to the Contestant's health, safety or welfare when coupled with participation in the Event.

Less than fifteen (15) seconds elapsed from the time Jones was Down until a Physician and RN were at his side administering professional medical care and ten (10) seconds of that time was the Ten Count, during which time Jones was attempting to get up and continue the Bout. Just less than ten (10) minutes after Jones was Down, the Ringside medical staff finished their initial assessment and although they had not observed any symptoms of life threatening injuries, the Ringside medical staff decided to transport Jones to the nearest hospital via ambulance for further observation and testing. All of the Commission's emergency action plans worked as designed and ensured the most expedient medical care possible for Mr. Jones under the circumstances. All emergency exit pathways remained clear and provided the most expedient egress from the Ring to the on-site ambulance. No change to the Commission's emergency medical protocols is suggested by the findings of this investigation; however, this incident makes clear that it remains critically important for the Commission's representative at each Event to emphatically explain the Commission's emergency action plan during the pre-Event conferences and ensure the existence of pre-planned emergency pathways with the same being communicated to on-site medical staff, Officials and the Promoter.

The EMTs provided oxygen and started normal saline IV within ten (10) minutes of the Ringside Physician's transport decision. A total of approximately twenty-three (23) minutes was spent preparing and stabilizing Mr. Jones for transport, which at first may appear to be an abnormally long preparation and stabilization period; however, under the circumstances, Jones was received medically appropriate first response care and Jones' physiological responses were not indicating the existence of any emergent circumstances requiring an expedited pre-transportation stabilization procedure. Many of the tasks performed by the EMTs during the patient stabilization process were the same as would have been provided at the hospital, such as provision of oxygen, starting a normal saline IV, and triage assessment including vital signs. The on-site ambulance and crew performed as expected and no change to the Commission's EMT and on-site ambulance protocols is suggested by this investigation.

The total transport time from the Venue to the nearest medical facility was an extremely short three (3) minutes six (6) seconds. The nearest medical facility was Saline Memorial Hospital ("SMH"), a Level III trauma center; however, the total transport time from the Venue to the nearest Level I Trauma Center was well within the "Golden Hour" guideline at less than thirty (30) minutes via ground transportation.

Jones' use of alcohol and anabolic steroids, together with his extraordinary use of caffeine, tobacco, and nutritional supplements on top of additional multi-vitamins and potassium supplements without drinking sufficient water, created a biological environment which altered his normal physiological responses and facilitated his demise. The conditions within Jones' body the night he climbed into the Ring were not detectable by any standard, medically reasonable Pre-Bout Physical Examination; however, those conditions created the perfect and unfortunate environment necessary to cause his death from an occurrence the human body is ordinarily well-equipped to handle – the extremely high intensity bursts of energy and activity associated with participation in a Combative Sports Event, coupled with the slight neurological disruption caused by the impact of a punch or series of punches to the head. In laymen's terms, when Jones' brain tried to reboot his body like a computer following the end of the 2<sup>nd</sup> Round, Jones' underlying physiological and biological conditions prevented a normal, compliant rebooting response. Upon receiving the "reboot instruction" from the brain, Jones' enlarged heart failed to respond due to the extremely elevated potassium levels and Jones' kidneys failed to respond, as they were already being stressed beyond their capacity due to lack of water, extreme level of potassium and myoglobin, and high level of lactic acid. The stress on Jones' kidneys and his body's ineffective potassium regulation was compounded by the lack of blood flow from Jones' heart, which was suffering from the effects of the rhabdomyolysis induced hyperkalemia. The end result was simply a cascading, multi-system failure resulting in death.

Overall, it appears there was nothing the Commission could have done, from either the regulatory or procedural standpoints, to prevent the tragic loss of Mr. Jones the night of 29-JAN-2011. However, it is always possible to look back on a tragedy and play the “what if game.” In fact, asking the “what if” type questions was a primary purpose of this investigation because doing so allowed the Commission to spend a moment reflecting upon ways to improve the safety of combative sports not only for the Contestants in Arkansas, but also for those located throughout the world.

Mr. Jones’ death should not be used as weapon by those fundamentally opposed to Combative Sports in their quest to put an end to Combative Sports in Arkansas, nor should this incident be used as a springboard for hasty implementation or removal of regulations. Instead, we must take a moment to discern fact from fiction and opinion, then utilize verifiable facts as our guide on the path to conducting future Combative Sports Events in as safe a manner as possible. It is with the foregoing in mind that the Commission provides the following recommendations and analysis.

C. **Why Did the Commission License Anthony Jones & Why Does the Commission License Contestants With Little or No Amateur or Professional Competition Experience?**

As to why the Commission issued a Contestant’s license to Jones, the short answer is that Commission Regulation § 1.13 is the primary regulation related to licensure by the Commission and the requisite documentation and fees were paid to the Commission for issuance of a license to Jones. Jones’ paperwork did not contain anything unusual or out of the ordinary which would have caused a reasonably prudent person under similar circumstances to have made any different decision.

As to why the Commission issues Contestant licenses to persons with little or no amateur or professional experience, perhaps this is best explained by the following two quotes from the movie, Million Dollar Baby.

**Trainer – Frankie Dunn** – You’re not breathin’ right, that’s why you’re pantin’. So, it’s your birthday, huh? How old does that make you?

**Maggie Fitzgerald** – I’m 32, Mr. Dunn, and I’m here celebrating the fact that I spent another year scraping dishes and waitressing which is what I’ve been doing since 13, and according to you, I’ll be 37 before I can even throw a decent punch, which after working this speed bag for a month gettin’ nowhere, I now realize that may be the God’s simple truth. Other truth is, my brother’s in prison, my sister cheats on welfare by pretending one of her babies is still alive, my daddy’s dead, and my momma weighs 312lbs. If I was thinking straight, I’d go back home, find a used trailer, buy a deep fryer and some oreos. Problem is, it’s the only thing I ever felt good doing. If I’m too old for this, then I got nothing. That enough truth to suit you?

Clint Eastwood as Trainer/Mgr. Frankie Dunn  
Hilary Swank as Maggie Fitzgerald  
Million Dollar Baby (2004)  
Warner Bros. Pictures

If there's magic in boxing, it's the magic of fighting battles beyond endurance, beyond cracked ribs, ruptured kidneys and detached retinas. It's the magic of risking everything for a dream that nobody sees but you.

Morgan Freeman as Eddie Scrap-Iron Dupris  
Million Dollar Baby (2004)  
Warner Bros. Pictures

What should be remembered and taken in context with the Commission's use of the foregoing quotes to answer the question is that the persons who are licensed by the Commission are all either consenting adults of sound mind, or are consenting minors with the consent of their parents or guardians. The individuals who apply to the Commission for a Contestants license have a sincere to desire to participate in a Combative Sports Event. In this regard, the Commission has adopted a view that men and women of sound mind should remain free to engage in endeavors of their own choosing without undue constraint by the government or other regulatory bodies.

While certain activities are indeed so incredibly dangerous to the participants, others and society as a whole that it may be best for the government to proscribe its people from participating in such activities, Combative Sports do not fall into such a category. Participation in well regulated, competitive Combative Sports provide many well documented physical, psychological and emotional benefits to the Contestants; therefore, while risk is obviously involved, the Commission holds a view that the potential physical, psychological and emotional benefits to the Contestants outweigh the risks when participation is conducted under a well regulated schema with health, safety and welfare as the foremost concern for the regulators. Further, the Commission holds a view that it is better for persons to participate in Combative Sports within a centralized and well controlled regulatory environment rather than participating outside the bounds of the law in a wholly unregulated manner lacking any of the safeguards and protections provided by governmental sanctioning and licensing of the activity.

The Commission believes it has implemented reasonably appropriate regulations, procedures and protocols to make the Contestant's participation in a Combative Sports Event as safe as can be expected for an activity with well known an well disclosed risks of severe personal injury or death. In addition to the Commission's Regulations, every Bout for every Event regulated by the Commission is reviewed prior to the start of the Event and the Contestants matched by the Promoter for each Bout must be approved by the Commission's Competition Committee or Representative in charge of the Event, regardless of whether the proposed Contestants have received their Arkansas license or not prior to the proposed Bout. Prior to approving each Bout, the Commission's Competition Committee or Representative in charge of the Event conducts any and all research they deem necessary to satisfy themselves that the proposed Bout is between Contestants of sufficiently similar experience, skill, and/or training that the Bout will be reasonably competitive, fair and safe. It is understood that it is virtually impossible to evenly match every single Bout on every single Event; accordingly, due care is exercised under the circumstances unique to each Bout for the approval or denial of each proposed Bout in accordance with the expressed standards and protocols for approval or denial of a Bout.

**D. What Is the Commission's Analysis of Its Protocols, Regulations & Systems? What Are the Commission's Future Recommendations to Improve Safety?**

**1. Location of Appropriate Medical Facilities in Proximity to Event Venues.**

In the event of a Combative Sports related medical emergency, it is critically important for Contestants to have access to either a Level I or Level II Trauma Center within the "Golden Hour," less than one (1) hour from the time of the injury until receiving state of the art medical trauma center care. Trauma centers are divided into four (4) categories and provide levels of care as follows:

**Level I Trauma Centers** – Provide the highest level of surgical care to trauma patients and comprehensive clinical care, education, research and outreach. A Level I Trauma Center is required to have a certain number of general and specialized surgeons and anesthesiologists on duty 24 hours a day at the hospital to adequately respond to and care for various forms of patient trauma.

**Level II Trauma Centers** – Provide comprehensive clinical and trauma care supplementing the clinical expertise of a Level I Trauma Center. It provides 24 hour a day availability of all essential specialties, personnel and equipment, but are not required to have an ongoing program of research or surgical residency programs.

**Level III Trauma Centers** – Provide treatment of mild and moderate single systems injuries. A Level III center does not have full availability of specialists, but does have resources for emergency resuscitation, surgery and intensive care of most trauma patients.

**Level IV Trauma Centers** – Provides stabilization and advanced trauma life support prior to transfer. This center is only capable of providing initial evaluation, stabilization, diagnostic capabilities and transfer services. Emergency room is likely to only have trauma trained nurse and physicians available upon arrival.

Arkansas has only two Level I Trauma Centers, University of Arkansas Medical Center and Arkansas Children's Hospital, which are fortunately both located in the central part of the State. The Regional Medical Center in Memphis, Tennessee is a Level I Trauma Center capable of providing coverage to Events in eastern and northeastern Arkansas, while St. John's Hospital in Springfield, Missouri is a Level I facility capable of servicing northwest Arkansas. Thus, over 80% of the geographic area of the State of Arkansas has access to a Level I Trauma Center via ground transportation within the "Golden Hour."

The State's Level II Trauma Centers and their coverage areas are Baptist Hospital and St. Vincent's Infirmary in Little Rock – providing coverage to central Arkansas; Jefferson Regional Medical Center in Pine Bluff – providing coverage to southeast Arkansas; Wadley Regional Medical Center in Texarkana, Texas and St. Joseph's Mercy Hospital in Hot Springs – providing coverage to southwest Arkansas; Sparks Regional Medical Center in Ft. Smith – providing coverage to northwest Arkansas; Washington Regional Medical Center in Fayetteville – providing coverage to northwest and north central Arkansas. Thus, ninety-five percent of the State of Arkansas' geographic area has access via ground transportation to a Level II Trauma Center within the "Golden Hour."

Based on the current location of Level I and Level II Trauma Centers throughout Arkansas, there does not appear to be a need to restrict Event Venues due to unavailability of appropriate medical care in the event of a medical emergency. The expansion of Arkansas' trauma network can only operate to increase the safety of Combative Sports in Arkansas, as very many Events are held more than one hour from the state's only two Level I Trauma Centers in Little Rock and some Events are held on the outer threshold of the one (1) hour transportation limit to a Level II Trauma Center.

2. **Critical Need for Unified Computer System Using Centralized Databases to Track All Amateur and Professional Combative Sports in Arkansas and all Other States, Territories and Jurisdictions in the United States.**

(a.) **Lack of Nationally Integrated, Uniform & Accessible Electronic Database**

One of the biggest challenges for this Commission and sanctioning bodies across the United States is the availability of timely and accurate information regarding a Contestant's licensure and past Combative Sports participation on both the professional and amateur level and in all of the various disciplines included in Combative Sports. In this case, Jones reported a 13-3-0 amateur record to the Commission; however, during this investigation it was denied by Jones' family and friends that Jones had ever competitively participated in a boxing Event. Prior to licensure, the Commission did check its database to see if Jones had been licensed as an amateur, professional or elimination Contestant and could not find where any such license had been issued to Jones. However, the Commission does not have the capability to search surrounding states' amateur and professional licensing databases or otherwise fact check a Contestant's claimed amateur record.

In amateur boxing there exists a standardized national system to track the records of Contestants who have participated in amateur bouts sanctioned by USA Boxing; however, USA Boxing's system of Contestant names and records is not available online, nor is such electronically searchable by any sanctioning body. Instead, USA Boxing still uses what is called a "passbook system," whereby Contestants' records are kept in a written log book retained by the Contestants and initialed by the USA Boxing official presiding over the Contestant's Bout. USA Boxing also loosely maintains records of each of its sanctioned Bouts across the United States. One problem with USA Boxing's passbook system is that not all amateur boxing is sanctioned by USA Boxing, thus thousands of boxing matches are held annually in the United States without any sanctioning body presiding over the Event. For example, here in Arkansas, if the amateur boxing Event is not sanctioned by USA Boxing or another duly recognized amateur body, then the Commission sanctions the Event and will keep records of the Contestants' participation; however, Arkansas' records of amateur boxing Events are not presently available to any other jurisdiction and vice-versa. If the amateur Bout occurs outside of Arkansas and is not sanctioned by USA Boxing, then there is no way for the Commission to verify the Contestant's claimed amateur record. Furthermore, USA Boxing's "passbook system" is completely devoid of any truly reliable method for verifying the legitimacy of a passbook's claimed record and it is widely known to be accepted practice for Contestants to move to another jurisdiction and "start over" with their passbook or for a Contestant to "fill up" one passbook, then "start over" with a new record when they receive a new passbook from their own jurisdiction. Sometimes a USA Event Organizer will forget to record or "sign off" on a Contestant's passbook, thus that Contestant's passbook then is no longer accurately a reflection of that Contestant's skill and experience.

In professional boxing, there exists a "Federal ID" program administered by the Association of Boxing Commissions ("ABC"). A Contestant's Federal ID provides a way to track that Contestant's record from state to state through a multi-state, multi-jurisdiction compact requiring each jurisdiction to report the Contestant's participation and the results of each sanctioned event held in that jurisdiction to FightFax. However, again, there remains a problem ensuring the validity of a Contestant's complete record due to some jurisdictions, such as certain Indian reservations and foreign countries, failing to report or participate in the FightFax national reporting program.

Although the ABC recently implemented the "National ID" program for Mixed Martial Arts ("MMA"), problems, similar to those in boxing, also exist with MMA National ID database and other combative sports regulated by the Commission. Several jurisdictions do not regulate MMA at all, some jurisdictions only loosely regulate MMA and there exists a general overall lack of consistency in record reporting. Some jurisdictions regulate only professional MMA, but not amateur, thus the MMA National ID database cannot be fully relied upon to accurately reflect a Contestant's true record and level of experience.

Further, all of the various martial arts disciplines fall under the jurisdiction of the Commission; thus, very many of the disciplines have neither their own database nor their own standardized record keeping system, while others vary from jurisdiction to jurisdiction with no accessibility and standardized reporting system.

In addition to tracking and verification of Contestants' records, the Commission has suffered from tremendous inefficiencies caused by its own licensure and reporting systems being manually recorded rather than electronically stored and processed through an automatic, electronic system.

(b.) **L.I.G.H.T.S.O.U.T. (Leveraged Integration of Governmentally Hosted Technical Systems for Online Unified Tracking)**

Last year, the Commission began development of an online software system called LIGHTSOUT, which is an acronym that stands for "Leveraged Integration of Governmentally Hosted Technical Systems for Online Unified Tracking" and maximizes the use of automated, electronic licensure, event and bout operations, Commission, Contestant and Licensee record keeping, payments, receipts and reporting, as well as cross checking information in real time against other available electronic state and federal databases and being designed for integration and use by all fifty (50) states and other jurisdictions within North America. The Commission's development of its online software has been expanded in scope to cover all of the various Combative Sports falling under the Commission's jurisdiction and to automate and computerize every aspect of Event operations, Licensure of all Licensees, and Commission operations with a designed in flexibility to adjust to the needs of other commissions and jurisdictions which may either elect to or be mandated to use the LIGHTSOUT System.

The Commission's computerization efforts and software development have been severely hampered by the fact that the Commission is a self-supporting Commission which receives no funding from the Arkansas legislature or taxpayers; instead, the Commission is funded solely through gross receipts taxes and licensing fees from its regulated activities. Further, although the Commission's revenues are deposited into the Arkansas treasury, the Commission is required to seek legislative budget approval every two years prior to spending any of its own money.

For example, for fiscal year 2011-2012, the Commission only received an appropriation of approximately \$21,000 from its own funds to partially fund development of its LIGHTSOUT System. By comparison, the Commission's current best estimate of the money necessary to fully develop and beta test its LIGHTSOUT System is \$500,000 to \$750,000 (which amount does not include the hardware and mobile data services necessary for the integrated system to properly function); accordingly, the Commission has an extraordinarily dire need for immediate funding to complete the LIGHTSOUT System and improve the health, safety and welfare of not just the Combative Sports Contestants in Arkansas, but all over the United States.

Completion of the LIGHTSOUT System will provide a substantial benefit and increase the efficiency and safety for all stakeholders in and related to Combative Sports and its licensees. The LIGHTSOUT System has "designed in" features which, consistent with its name, provide an integrated approach to and benefit from inter-agency communication, data verification and notifications amongst many different state and federal agencies, hospitals and health care providers including medical records storage, access and transfer in real-time if necessary for treatment and reporting.

The Commission needs immediate, full funding and expedited design and implementation of the LIGHTSOUT System which is automatically linked and cross-referenced between its own databases and the following, which is only a partial list of the databases for which the LIGHTSOUT System is being designed for inter-operability and communication: ABC's Boxing Federal ID database, FightFax database, BoxRec database, ABC's National MMA database, Arkansas State Medical Board database, Arkansas State Board of Nursing

database, Arkansas Department of Motor Vehicles, Arkansas Office of Child Support Enforcement, hospital emergency rooms and Arkansas licensed physicians and all other state and federal jurisdictions which make their licensee or database information electronically available and provide added information verification and authentication capability to the LIGHTSOUT System.

Once the LIGHTSOUT System is brought online and eventually in use by all jurisdictions in the United States, every Contestant's and every Official's record of Bouts or participation in any manner will be instantly available online and available twenty-four (24) hours a day via a secured connection and login procedure. It is envisioned that a wide array of health, safety and welfare benefits will be achieved by the LIGHTSOUT System, as well as the achievement of many other ease of use and reliability metrics including, without limitation: (i) Contestants' and Officials' medical records could be made available during licensure, pre-Bout and post-Bout physicals, and treatment at hospitals or physicians offices nationwide; (ii) Any Licensee's amateur and professional licensure in any Combative Sport discipline and all Bout results could be automatically verified, accessed and electronically searchable and many, many more beneficial uses. The LIGHTSOUT System would only grow more reliable and beneficial to everyone as more and more jurisdictions adopted its use and brought their record keeping, licensure and other information systems online through the LIGHTSOUT System, as such would then be available to users in other jurisdictions and the safety of Combative Sports would be greatly improved by allowing a secure, verifiable way to exchange information and more evenly match Contestants based upon their verified experience level.

### **3. Need to Further Develop Commission's Form System & Mandate Use of Commission's Forms.**

Need promoters to immediately and without exception begin using all of the Commission's forms including the Event Application and Bout Card Application in electronic format. All Bout Card Applications should be completed in full and submitted at least ten (10) days prior to each Event, as required by the Commission's Regulations. If a Bout Card Application lacks information, it should be rejected with the reason noted for the Promoter to correct and re-submit. Bout Card Applications should not change Bouts or Event details less than 72 hours prior to the Event in order to give Commission sufficient time to perform due diligence.

### **4. Need for Increased Inspectors at Each Event**

Need minimum of two (2) inspectors plus one (1) Commission representative in charge at ringside for every event. Current testing for Inspectors is continually making available more Inspectors for Events and such testing by the Commission should continue until sufficient number of licensed Inspectors are available throughout the state to avoid and reduce travel requirements.

### **5. Need to Move Breathalyzer Testing Closer to Bout Time**

The Commission presently has five (5) portable breathalyzer testers ("PBT"), which it received via an inter-agency transfer from the Arkansas State Police. The PBT devices are calibrated by the Arkansas State Police at least once every six (6) months. Prior to every Contestant's participation in a Bout, the Contestant must pass a PBT test with a result of .000 ml/dL, which test has traditionally been administered in conjunction with their physicals. Jones was administered his PBT immediately after his pre-Bout physical and produced a result of .000 ml/dL.

While not clinically significant and thus not appearing in the ASME's final report or findings, forensic testing revealed Jones had .005 ml/dL of unaccounted for ethanol in his blood. The unaccounted for ethanol is consistent with Jones having had up to one alcoholic drink sometime between the time Jones passed the Commission's portable breath test and the time he entered the Ring for his Bout. The level of unaccounted for

ethanol was not sufficient to impair Jones' reflexes, coordination or judgment; however, the mere presence of unaccounted for ethanol in a Contestant who provided a .000 ml/dl result during pre-Bout screening raises a very slight concern regarding the timing of the Commission's PBT testing protocols.

In light of the findings in this investigation, the Commission has already modified its Event procedures to implement new PBT testing protocols which move the PBT testing closer to the actual time of each Contestant's Bout. More specifically, the Commission makes every effort to PBT test the Contestants at the time they "glove up" for their Bout, which is usually ten (10) minutes or less before they enter the Ring and during which ten (10) minute period the Contestant normally remains within view of the Commission's Inspector, if sufficient Inspectors are available for the Event.

In addition, increasing the number of Inspectors at each Event should further reduce the possibility that a Contestant could consume alcohol prior to participating in an Event.

## **6. Need for Urinalysis, HIV, Hepatitis & Blood Chemistry Testing**

Tests for many prohibited and illicit drugs and their metabolites can be quickly and relatively inexpensively performed on-site at each Event using disposable urinalysis test kits; however, many of the more sophisticated drugs, as well as, anabolic steroids and their metabolites must be tested by collecting the specimen in a specific, controlled manner and sent to a specialized laboratory for advanced urinalysis. Disposable test kits for on-site screening for many illicit drugs cost between \$25 and \$125 per kit; however, the tests for more sophisticated drugs, as well as, anabolic steroids and their metabolites costs between \$200 for a basic anabolic steroid panel screen up to \$725 for a full, Anabolic Steroid Screen for the WADA prohibited list with Carbon Isotope Ratio Testing.

Following President George W. Bush's 2004 State of the Union address, wherein he urged the Nation "to get tough and to get rid of steroids [in athletics]," many states implemented statewide steroid testing in their high schools. After spending four (4) years and \$400,000 of taxpayer money, Florida stopped its statewide high school testing because it could no longer cost justify the benefits after having only received one positive test among the 600 tests performed on its high school students. In 2008, Texas, Illinois and New Jersey were the only states known to have statewide random steroid testing programs. New Jersey only found one positive case in 500 tests during 2008. Texas' annual program costs the state over \$3 million per year and reported seven positive tests out of over 19,000 tests performed through the end of 2008. Meanwhile, Illinois spent \$150,000 on a pilot steroid testing program and had no positive tests through the end of 2008.

The Commission finds Jones' anabolic steroid use was likely a significant contributing factor in Jones' death and that mandatory or even random steroid testing could have possibly prevented or detected such use. However, based on other states' ratio of confirmed positive steroid tests to total amount of money spent on testing, perhaps implementation of a full steroid testing program along with incursion of or passing along of its associated costs is not the most efficient use of money at this time.

Instead, the Commission should implement a brief, standardized and very strongly worded verbal precaution to be given by the Commissioner or Representative in Charge at the Event sometime between pre-Bout physicals and pre-Bout dressing room Contestant instructions and rules.

Additionally, the Commission should consider placing an additional warning on either its annual license application or on the pre-Bout physical form with a certification to be signed by the Contestant confirming the Contestant has either never used or has been free of steroid use and other prohibited substance use for \_\_\_\_\_ months.

Portable I-stat devices provide almost immediate blood chemistry results using single use, disposable cartridges. Blood test results using an I-stat device include those that are seen in Jones' blood test results at 05:28 hours and can be expanded at an additional cost to include some of the results seen in Jones' blood test results at 04:00 hours. However, based upon consultation with the forensic pathologist who performed Jones' autopsy, Dr. Daniel Dye, M. D., and several other members of the Commission's medical team assembled for this investigation and report, the information derived from I-stat testing every single Contestant at every Event, both immediately before and immediately after they step into the Ring or Cage, cannot be economically justified. The I-stat testing becomes even less important in the opinion of the Commission and the Commission's medical team when the ringside physician is familiar with Combative Sports and exhibiting the appropriate level of Contestant observation, since any physiological medical condition which could be detected by the I-stat device at a level sufficient to pose a risk to the Contestant's participation or post-Bout activities, would very likely also be evidenced by other signs or symptoms easily detectable as part of both the Commission's standard pre-Bout and post-Bout physicals.

#### **7. Need for Crisis & Grief Counseling**

The Commission wishes to extend a special thank you to the Mississippi Athletic Commission and the Nevada State Boxing Commission, both of whom put forth a special effort to reach out to the Commission by not only offering their condolences on the loss of Mr. Jones, but also to offer any assistance they could based on their own experiences with this fortunately rare occurrence.

The Commission would also like to thank the many volunteers associated with the Arkansas Crisis Response Team who generously donated their time to provide grief and crisis counseling during the days and weeks following the loss of Mr. Jones. The Arkansas Crisis Response Team can be found at <http://www.arcr.org/> or by confidentially calling 501.766.3360.

As soon as was possible under the circumstances, the Commission arranged and made available crisis and grief counseling, free of charge, to the family, Seconds and teammates of both Jones and Palmer, as well as, the Ringside Officials including the Judges, Physician, Nurse and Referee. The very nature of the grief counseling process is confidential, thus preventing the Commission from knowing who actually took advantage of the offered services. Nevertheless, while the Commission can obtain no measurable participation results, it remains the Commission's opinion and recommendation that the Commission's continued crisis management and emergency incident protocols continue to include provisions to make available crisis and grief counseling, free of charge to those persons most directly impacted by the event. Immediate crisis and grief counseling is critically important to those affected by a tragedy such as this Incident.

#### **8. Need for Increased Enforcement & Oversight of Amateur Events Including Those Granted Exemptions Under the Regulations**

The Commission should increase its enforcement and oversight efforts for all amateur Events including, without limitation, those Events granted an exemption under the Commissions Regulation § 1.16 and its subsections.

#### **9. Need for Additional Training Opportunities & Experience for Judges & Referees**

The Commission should explore the implementation of a training and observation program for its judges and referees whereby referees and judges who have less experience overall, less experience in a particular Combative Sport, or less experience on either the amateur or professional side of a particular Combative Sport can shadow and receive mentored training from more experienced judges and referees. Additionally, the Commission should remain vigilant in its efforts to be familiar with its judges and referees and ensure either that the particular

referee or judge assigned to an Event has the requisite training or experience to work that particular Event. If an Event is being used as a cross-training or mentoring Event, then the Commissioner or Representative in charge at Ringside should remember to constantly remind all Judges and Referees of the rules applicable to that particular Event. The Commissioner or Representative in charge at Ringside should carefully observe the Referees' and Judges' activities during the Event for potential training tips and pointers and provide the same between Rounds, between Bouts or both.

In Jones' case, the Referee had extensive past and current Amateur Boxing experience with limited to moderate Professional Boxing experience. The Referee in Jones' Bout was being mentored during the Event by both the Commissioner in charge at Ringside, Jason A. Stuart, and the Commission's Inspector, Sonny Axsom, both of whom have extensive professional Boxing experience. Commissioner Stuart is certified by the Association of Boxing Commissions as both a boxing referee and a boxing judge. Despite a review of the Unified Rules of Boxing, which have been adopted by the Commission in Regulation § 2.4, with the referee prior to the start of the first Bout of the Event, the referee did make a mistake in the heat of the moment by issuing a Standing Eight (8) Count to Quincy Palmer during the 1<sup>st</sup> Round of the Jones vs. Palmer Bout.

While Standing Eight (8) Counts are mandated in Amateur Boxing, the Standing Eight (8) Count is prohibited under the Unified Rules of Boxing in effect during this professional Boxing Bout. During the one (1) minute rest period between the 1<sup>st</sup> and 2<sup>nd</sup> Rounds, Referee Tunstall was signaled and advised of his mistake by Commissioner Stuart at which time Referee Tunstall acknowledged the mistake. Following the end of the Bout Referee Tunstall was advised again and the matter was further discussed between Commissioner Stuart and Referee Tunstall to ensure a clear understanding. Referee Tunstall again acknowledged the mistake and stated that it had almost become a "reflexive response," due to his extensive experience as a referee under the Amateur Boxing rules. Referee Tunstall stated that he knew during the Standing Eight (8) Count that it was a mistake; however, rather than discontinue the count early and cause confusion or controversy, Referee Tunstall elected to continue the Standing Eight (8) Count and allow the Bout to continue at the end of the Standing Eight (8) Count, since he was satisfied Palmer was okay to continue. Commissioner Stuart agreed with Referee Tunstall's decision to continue with the Standing Eight (8) Count once it had been initiated rather than to discontinue the Standing Eight (8) Count early.

Referee Tunstall continued refereeing the remainder of the Bouts on the card at the Event, had no other mistakes and otherwise displayed good technique, judgment and control of the activities in and around the Ring. To the Commission's knowledge Referee Tunstall has not repeated the Standing Eight (8) Count error again since the Jones vs. Palmer Bout. The Commission believes the error was a small error due to reflexive action and that sufficient corrective action has been taken. The Commission does not recommend any disciplinary action against Referee Tunstall or that Referee Tunstall undergo additional Professional Referee training. It is further the Commission's opinion that Referee Tunstall's administration of a Standing Eight (8) Count did nothing to hasten, contribute or cause Jones' death nor would Jones' death have been prevented if Referee Tunstall had stopped the Bout instead of administering the Standing Eight (8) Count. The first Dominos had already fallen and Jones' cascading systems failure had already begun.

#### **10. Need for Continuous Review of Scientific Developments in Sports Injury Diagnosis, Treatment & Prevention, As Well As, Return to Play Guidelines**

The Commission should continually review, at least annually if not more often, the medical and scientific developments in the areas of sports injury diagnosis, treatment, prevention and return to play guidelines, especially in the context of combative sports. In this regard, the Commission should continue its current continuing education program including Commissioners, physicians and Officials attendance at various relevant seminars, presentations and forums on the subjects.

(a.) **Standardized Concussion Test**

In carrying out its functions and promulgation and enforcement of its regulations, the Commission should keep in mind the current medical and scientific practices providing updates to the following Standardized Concussion Test, which are generally followed by the Commission and were generally adopted and established in the practice parameter published by the American Academy of Neurology and reviewed by the American Association of Neurological Surgeons, American College of Emergency Physicians, American Academy of Pediatrics, American Academy of Family Physicians, National Athletic Trainers Association, and American Academy of Neurology Member Reviewer Network. (Lippincott, Williams, Wilkins, (1997). "Practice Parameter: The management of concussion in sports (summary statement)" Neurosurgery. 48:581-585):

(i.) **Orientation**

- Time (Day of Week/Day or Night)
- Place (Current Location, City, Building, Etc.)
- Person (Own Name, Name of Others Around Them)
- Situation (Circumstances of Injury)

(ii.) **Concentration**

- Counting Backwards
- Naming Months of the Year in Reverse Order

(iii.) **Memory**

- Name of Previous Opponent
- Time of Weigh-Ins
- Recall of 3 Words AND 3 Objects at Both 0 Minutes and 5 Minutes
- Recall of Recent Newsworthy Events
- Details of the Contest (Moves, Strategies, Etc.)

(iv.) **External Provocative Tests**

- 40 Yard Sprint
- 5 Push Ups
- 5 Sit Ups
- 5 Deep Knee Bends
- Any Appearance of Symptoms Associated with Concussion is Abnormal During This Test (e.g. Headaches, Dizziness, Nausea, Unsteadiness, Photophobia (abnormal sensitivity to light), Blurred or Double Vision, Emotional or Mental Status Changes)

(v.) **Neurologic Tests**

- Pupils (Symmetry of Motion and Reaction)
- Coordination (Finger-Nose-Finger, Tandem Gait)
- Sensation (Finger-Nose With Eyes Closed and Romberg's Test (i.e. Feet together with hands straight down at side and eyes closed. Observe movement in relation to a perpendicular object in the background to observe for any sign of swaying.)

**(b.) Standardized Return to Competition or Practice Guidelines**

In carrying out its functions and promulgation and enforcement of its regulations, the Commission should keep in mind the current medical and scientific practices providing updates to the following Return to Competition or Practice Guidelines, which are generally followed by the Commission and were generally adopted and established in the practice parameter published by the American Academy of Neurology and reviewed by the American Association of Neurological Surgeons, American College of Emergency Physicians, American Academy of Pediatrics, American Academy of Family Physicians, National Athletic Trainers Association, and American Academy of Neurology Member Reviewer Network. (Lippincott, Williams, Wilkins, (1997). "Practice Parameter: The management of concussion in sports (summary statement)" Neurosurgery. 48:581-585):

**(i.) Return to Competition or Practice Following Grade I Concussion Evidenced by Transient Confusion, No Loss of Consciousness, and Concussion Symptoms or Mental Status Abnormalities Resolving in Less Than 15 Minutes**

- Remove Contestant from Contest or Practice
- Examine Immediately and at 5 Minute Intervals for Mental Status Abnormalities or Post-Concussive Symptoms Both at Rest and at Exertion
- Allow Same Day Return to Competition or Practice if Mental Status Abnormalities or Post-Concussive Symptoms Disappear within 15 Minutes or Less
- Second Grade I Concussion During the Same Contest, Practice or Day Eliminates Contestant from Competition, Practice or other Activities that Day and Disallows Return to Competition or Practice Until Contestant is Asymptomatic for a Minimum of One (1) Week Both at Rest and at Exertion

**(ii.) Return to Competition or Practice Following Grade II Concussion Evidenced by Transient Confusion, No Loss of Consciousness, and Concussion Symptoms or Mental Status Abnormalities Lasting More Than 15 Minutes**

- Remove Contestant from Contest or Practice and Disallow Return to Competition or Practice that Day
- Examine Contestant On-site Frequently for Signs of Evolving Intracranial Pathology
- Trained Person Should Re-examine Contestant the Following Day
- Physician Should perform a Neurologic Examination to Clear Contestant for Return to Competition or Practice After One (1) Full Asymptomatic Week at Rest and at Exertion
- CT Scan, MRI Scan or MRA Scan is Recommended in All Instances Where Headache or Other Associated Symptoms Worsen or Persist Longer than One (1) Week
- Following a Second Grade II Concussion, Return to Competition or Practice Should be Deferred Until the Contestant Has Had at Least Two (2) Weeks Symptom Free Both at Rest and at Exertion

- Terminating the Contestant's Participation in Competition and Practice for the Entire Season (for Combative Sports, the Commission suggests a period of not less than 6 months) is Mandatory for any Abnormality on CT Scan, MRI Scan or MRA Scan Consistent with Brain Swelling, Contusion, or Other Intracranial Pathology

(iii.) **Return to Competition or Practice Following Grade III Concussion Evidenced by Any Loss of Consciousness Whether Brief (seconds) or Prolonged (minutes)**

- Transport Contestant from the Ring to the Nearest Emergency Room by Ambulance if Still Unconscious or If Worrisome Symptoms are Detected (Include Cervical Spine Immobilization, if Indicated by Conditions)
- Perform Thorough Neurological Evaluation Emergently, Including Appropriate Neuroimaging Procedures When Indicated
- Hospital Admission is Indicated if Any Signs of Pathology are Detected, or if the Mental Status of the Contestant Remains Abnormal
- If Findings Are Normal at the Time of the Initial Medical Evaluation, the Contestant May Be Sent Home. Explicit Written Instructions Will Help the Family or Responsible Party Observe the Contestant Over a Period of Time
- Neurologic Status Should Be Assessed Daily Thereafter Until All Symptoms Have Stabilized or Resolved
- Prolonged Unconsciousness, Persistent Mental Status Alterations, Worsening Post-Concussion Symptoms, or Abnormalities On Neurologic Examination Require Urgent Neurosurgical Evaluation or Transfer to a Level I Trauma Center
- After Brief (seconds long) Grade III Concussion, Contestant Should be Removed from Competition & Practice Until One (1) Full Asymptomatic Week at Rest & Exertion
- After Prolonged (minutes long) Grade III Concussion, Contestant Should be Removed from Competition & Practice Until Two (2) Full Asymptomatic Wks at Rest & Exertion
- Following a Second Grade III Concussion, Return to Competition or Practice Should be Deferred Until the Contestant Has Had at Least One (1) Full Month Symptom Free Both at Rest and at Exertion Which Time May be Extended by the Treating Physician Based on Clinical Evaluation and Other Circumstances
- CT Scan, MRI Scan or MRA Scan is Recommended for Contestants Whose Headache or Other Associated Symptoms Worsen or Persist Longer Than One (1) Week
- Terminating the Contestant's Participation in Competition and Practice for the Entire Season (for Combative Sports the Commission suggests a period of not less than 1 year) is Mandatory for any Abnormality on CT Scan, MRI Scan or MRA Scan Consistent with Brain Swelling, Contusion, or Other Intracranial Pathology. In Such Circumstances, Return to Competition or Practice in the Future Should be Discouraged when Consulting with the Contestant.

The foregoing Return to Competition or Practice Guidelines are provided as an educational service of the American Academy of Neurology and the Arkansas State Athletic Commission. The foregoing Return to Competition or Practice Guidelines are not regulations adopted by the Commission and are intended to neither include all possible proper methods of care for choosing to use a specific procedure nor exclude any reasonable alternative methodologies. Specific patient care should always remain the prerogative of the treating physician and be adjusted according to the patient's particular circumstances, condition and facts. Current and future medical and scientific developments and advances in understanding concussions will necessitate adaptation and modification of any Return to Competition or Practice Guidelines.

**ACKNOWLEDGMENT & SIGNATURE VERIFICATION**

**By my signature below, I certify the information herein is, to the best of my knowledge and information, true and accurate. No professional medical or legal opinion is expressed by the Commission or the undersigned individual by the dissemination of this report or the undersigned's signature hereupon. Any provision of this report which purports or appears to be medical opinion should be interpreted as the lay opinion of the Commission. No person or entity is permitted to rely upon the information provided in this report or in any discussion of this report for purposes of legal or medical advice or opinions.**

**The participation of the physicians on the Commission's medical team was on a consultative and informational basis only at the direct request of the undersigned Commissioner and Commission Investigator; thus, the professional medical opinion of none of the physicians on the Commission's medical team is expressed either by the Commission's opinions in this report, the Commissioner's signature below or the inclusion of the physician's name in the acknowledgment section of this report.**

**Signed:** \_\_\_\_\_  
**Jason A. Stuart, Commissioner**  
**Arkansas State Athletic Commission**

**Date:** 9-SEPT-2011