Effects of Alcohol on Traumatic Brain Injuries and Second Impact Syndrome

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INTRODUCTION

When Forensic Pathologist and or Neuropathologist evaluate a brain that has been subjected to **traumatic brain injury (TBI)**, they tend to focus on the anatomic injuries, i.e, fractures of the calvarium or base of skull; presents of epidural, subdural or subarachnoid hemorrhage; acute contusions, whether they be coup, contracoup or gliding; the presence of intraparenchymal hemorrhage; the presence of edema; and evidence of herniation to name a few of the anatomic injuries. Following noting the anatomic changes, they then attempt to relate the injuries to the circumstances that led to the TBI. In this process, they will note whether the patient was under the influence of alcohol and or other drugs, however, they typically do not explore the role alcohol and or other drugs had on the pathophysiologic process that occurs at the molecular level in a TBI. This is a case in which I was asked to examine two TBIs a patient sustained, separated by approximately two months. I was also asked to look at whether the patient's acute and chronic alcoholism played a role in the pathophysiological process of these TBIs, and his subsequent precipitous death following the second TBI due to an assault.

HISTORICAL INFORMATION

First Traumatic Brain Injury

A 44-year-old male was found lying on the side of a road. He was initially unresponsive according to bystanders. Upon arrival of the EMTs, he was noted to be awake but had an altered level of consciousness. The patient admitted to being intoxicated, stating he had "a lot to drink tonight." He did not know what had happened to him or where he was. He had injuries to his face and the back of his head.

On admission to the hospital he was noted to have a GCS (Glasgow Coma Scale) of 14 (GCS 3-15, with 15 being the highest and best score). His alcohol level was 372 mg/dL. He was not certain how he ended up along the road. He was also not certain if he had been struck by a car. He did have obvious facial trauma.

The patient was evaluated medically and noted to have a traumatic subdural hematoma with associated subarachnoid hemorrhage with loss of consciousness of unspecified duration; traumatic encephalopathy; scalp laceration; facial laceration; closed fracture of the nasal bones; abrasions of multiple sites; hypomagnesemia; traumatic cerebral edema; and hyponatremia.

The first CT of the head showed multiple areas of subarachnoid hemorrhage along the floor of the anterior cranial fossa with a small focus of subdural hemorrhage at the high left parietal convexity. A CT of the facial bones revealed displaced and comminuted fractures of the bilateral nasal bones.

The following day, the patient had another CT of his head, which showed an ovoid focus of high-density blood within the parenchyma of the right frontal lobe consistent with an intraparenchymal hemorrhage. There was evidence of acute subarachnoid hemorrhage in the inferior subarachnoid spaces of both frontal lobes and in the subarachnoid spaces of the basal cisterns, which now extended into the right insula. There was evidence of what was felt to be a subarachnoid hematoma near the vertex of the medial right frontal lobe manifested as a focus of blood. There was also a crescentic focus of high-density blood measuring 3 mm in thickness underlying the left frontal and parietal bones consistent with a subdural hematoma. Additionally, there was a high density focus of blood in the anterior interhemispheric fissure, which was consistent with another subdural hematoma.

The patient underwent a third CT of his head the next day. This CT showed areas of hemorrhagic contusions in the inferior frontal lobes bilaterally, right greater than left, which had increased in size when compared to the previous study the day before. There was a small area of hemorrhagic contusion in the right anterior temporal lobe, which also showed a minimal increase in size as compared to the first CT of his head. There was a small amount of blood in the right Sylvian fissure (lateral sulcus), which was unchanged. There was also a thin layer of subdural blood along the anterior aspect of the falx, which was unchanged.

Following the patients physical examination and CT scans he was diagnosed with traumatic subarachnoid hemorrhage and subdural hematoma with acute traumatic encephalopathy; Acute traumatic cerebral edema; Displaced and comminuted fractures of the bilateral nasal bones; Scaphoid and posterior lunate fractures; and Acute Alcohol Intoxication.

During the patient's hospital stay he had several psychological evaluations. One of these evaluations was a Cognitive Status Examination in which the patient's **personal safety** and **judgment** and **short term memory** were found to be impaired. **It was the opinion of the evaluator, both were due to his TBI exacerbated by his acute alcoholic intoxication.**

The patient was seen by a Licensed Clinical Counselor because of his history of drug and alcohol abuse. The Clinical Counselor found him to be defensive and distant.

When she first attempted to consult with him, he would not engage when asked questions about his drug and alcohol use. On the second attempt to consult, he did agree to engage, but his responses were limited to one word. She had to prompt him several times to get any meaningful information. She found the patient to be fairly angry-passively-and resistant. **He was described as being a fairly typical male alcoholic struggling with depression.**

The patient was noted to be disoriented to time and place intermittently, **which was believed to be due to his TBI exacerbated by his acute alcoholic intoxication.**

One of the physicians who examined the patient found him to becoming more restless and irritable, **which he attributed to alcohol withdrawal superimposed on a TBI.**

A physician assistant raised a concern whether the patient could care for himself considering his cognitive state. **He also believed the underlying causation of his dysfunctional cognitive state was his TBI exacerbated by his acute alcoholic intoxication.**

The consequence of the patient's dysfunctional cognitive state was emphasized when he decided to sign himself out of the hospital against medical advise because the nurses would not allow him to smoke a cigarette, while he was in a hospital unit. **Although the patient did return to the hospital the same day his inappropriate behavior was another example of his dysfunctional cognitive state due to his TBI exacerbated by his alcoholism.**

Ultimately the patient was discharged from the hospital seven days after his admission.

Second Traumatic Brain Injury

A little over two months later the patient sustained a second **TBI** following an assault due to the patient's inappropriate conduct. On arrival of the EMTs he is found to be unconscious. The EMTs noted bruising of the right eye, with a laceration at the corner. There was also an injury above the eyebrow. Due to the swelling of the right upper and lower eyelids, the patient's right pupil could not be examined. His left pupil was 4 mm in diameter, suggesting to the EMTs he had a head injury and there was possible drug and or alcohol use. The patient did smell of alcohol. The patient is placed in the ambulance at which time the EMTs noted his breathing was irregular and he had snoring respirations. **Snoring respirations** are also referred to as the "death rattle."

Snoring respirations are due to terminal respiratory secretions, which lead to the accumulation of salvia and bronchial secretions accumulating in the upper chest and throat (larynx and bronchial tree) due to the inability to swallow. Swallowing is a complex process requiring coordination within the autonomic nervous system, most especially the medial temporal lobe, limbic system, medulla oblongota, and pons. The snoring respirations are a terminal event with respiratory failure following shortly. **In this patient's case, they indicated he had suffered a catastrophic neurologic event.**

The nature of the catastrophic event, however, far exceeded the level of the assault, which the patient was subjected too.

He is subsequently seen in the Emergency Department of the hospital in which he is described as a 45-year-old gentleman who is essentially obtunded with a GCS of 3. He is described as a patient who smells of alcohol and has had trauma. He is noted to have ecchymosis and swelling around the right eye, as well as a hematoma to the scalp of the left parietal area and some ecchymosis to the left flank. His right eye could not be opened and the left sclera is normal but the pupil is 6-7 mm and nonreactive.

The patient's **Problem List** was as follows: History of alcohol and drug abuse; respiratory failure following trauma; head trauma; closed blowout fracture of right orbit; acute alcohol intoxication (327 mg/dL); GCS total score 3-8; TBI, with loss of consciousness of 1 hour to 5 hours 59 minutes; and a post traumatic subdural hematoma.

CT of the Head revealed a right blowout fracture, as well as a nasal bone fracture. Intracranially there was a large left subdural hematoma with a maximal thickness of approximately 15 mm. There was also an associated left to right shift estimated at 11 mm. There was obvious mass effect on the left cerebrum with loss of the basal ganglia cisterns and contralateral dilatation to the temporal horn. These findings are likely related to uncal herniation. There was diminished density in the subcortical white matter of the anterior inferior right frontal lobe, which suggested an acute contusion.

CT of the Facial Bones revealed a right blowout fracture with 3 mm depression of the major fracture fragment. The AP (anterior-posterior) dimension of the orbital floor fractures was 25 mm. There was a small intraorbital hematoma adjacent to the medial orbital wall fracture with slight deviation of the medial rectus muscle. There was also an intact right globe with extensive overlying soft tissue swelling.

Due to the likelihood of an extremely poor neurological outcome, the family declined surgical intervention and elected for comfort care, which the patient was placed on late in the evening of the day of his admission. The patient was pronounced early in the afternoon the following day. From the time of the assault to the withdrawal of life was approximately 5 hours, which is consistent with a precipitous neurologic decline.

POSTMORTEM EXAMINATION

An autopsy was performed on the patient the next day by a Forensic Pathologist.

Forensic Examination of the Body:
Body Build and Decompositional Changes: This is the unembalmed body of a 45-year-old, thin white male, who measures 5' 10" and weighs 132 pounds. The overall appearance of the body is compatible with the stated age.

Evidence of External Injury and other abnormalities:

Head:

1. Abrasion, elliptical shaped, right above right eyebrow measuring 1'' x 1/2" to 5/8".
2. Contusion, red/blue with marked swelling, right eye.
3. Abrasion, irregular shaped, beneath right eye associated with clotted blood, measuring 1½" to 11⁄8".
4. Contusion, inner aspect of right nose.
5. Nasal bones intact on palpation, please see x-rays.
6. Bloody fluid exuding out of nose.
7. Blood flow around right ear.
8. Purple changes, left ear.
9. Abrasion, elliptical, fresh, upper head/left forehead region, measuring 1/4" x 1/8".
10. Abrasion, polygonal shaped, left upper lateral forehead, measuring 5/8" x 1¼"
11. Abrasion, beneath lip, measuring ¼" x ½".
12. Contusion, fresh, circular, left upper forehead, measuring ¼" x 5/16".
13. Bruise, irregular shaped, measuring ¼" x ¼", left forehead, fresh.
14. Bruise, circular, measuring ¼" x ¼".
15. Bruise, left forehead.
16. Marked swelling, right orbit and right side of face.
17. Contusion, circular, right inner lower lip.
18. Hemorrhage, inner aspect of right upper lip.

Upper Extremities:

1. Bruise, irregular shaped, between second and third knuckles, back of left hand, measuring 3/8" x ¼".
2. Bruise, irregular shaped, polygonal shaped, back of left hand between third and fourth knuckles, measuring 1" x 1", compatible with defensive wound.

Lower Extremities:

1. Contusion, circular, irregular shaped, left medial knee, purple/green, measuring 2" x 2".
2. Contusion, red and green, triangular shaped, beneath left knee, measuring 3½" x 3½".
3. Abrasion, circular and irregular shaped, anterior left lower leg, fresh, measuring 1" x ¼.
4. Contusion, circular, inferior and medial to right knee, measuring ¾" x ½".
5. Contusion, left lateral hip, green/purple, measuring approximately 2¼" x 1½".
6. Contusion, left lower back, circular, red, measuring ¾" x ¾"±.

Internal Examination of the Body, Organ Systems:

Organ Weights:

Brain: 1276 grams (male normal average 1400 grams)
Heart: 427 grams (male normal average 300 grams)
Right Lung: 963 grams (normal average 450 grams)
Left Lung: 911 grams (normal average 375 grams)
Spleen: 146 grams (normal average 155 grams)
Right and Left Kidneys together: 423 grams (normal average 313 grams)

Head: Hemorrhages are noted in both petrous ridges consistent with congestion.

Head and Central Nervous System:
Examination of the scalp revealed two circular bruises in the left temporal region. There is an elliptical bruise in the left frontal region of the scalp just above the eyebrow. On the outside skin, there are petechial hemorrhages, but no additional bruise is noted. The right frontal and right lateral frontal region of the scalp also shows a bruise that is arising from the right orbital blowout fraction.

Removal of the calvarium reveals a subdural hemorrhage involving the left convexity and left basilar region of the brain. There are slight areas of hemorrhagic congestion in the frontal regions and the right central basilar frontal region of the brain. The dura is removed from this area and on comparing our observations of this area with the CT scans, the neuropathologic features of this area indicate contusions. The cerebral convexity are edematous. Examination of the unci, reveal bilateral uncal grooves. There is also evidence of a left to right shift. The blood clots in total weigh 34 grams.

The brain shows no evidence of neuropathologic features following fixation and coronal sectioning per the Forensic Pathologist who did the autopsy.

Chest and Abdomen Cavities:
Inspection of the chest and abdomen reveals an episternal hemorrhage as well as subcapsular hemorrhage in the right and left sides of the liver.

Cardiovascular System:
Examination of the heart reveals evidence of left ventricular hypertrophy (left ventricular thickness, 1.5 cm) and cardiomegaly (heart weight, 427 grams).

Respiratory System:
The lung parenchyma shows pulmonary edema, hemorrhagic congestion and areas of consolidation consistent with pneumonia most prominent in the lower lobes (right lung 963 grams, left lung, 911 grams).

Opinion

It was the opinion of Forensic Pathologist the cause of death was **Traumatic Brain Injury**. The manner of death was **Homicide**.

ANALYSIS

First Traumatic Brain Injury

The patient sustained a significant TBI when he was found lying in the roadway approximately two months before his fatal assault. This was determined by his medical evaluation and CT scans of his head, which showed acute subarachnoid hemorrhages, a subarachnoid hematoma, subdural hematomas, intraparenchymal hemorrhage, acute contusions, traumatic brain edema with an encephalopathy, and an unknown period of unconsciousness. These features are those of TBI, which would be classified as a moderate. The loss of consciousness was caused by acceleration-deceleration movements of the brain, which resulted in the stretching and shearing of axons.

A loss of consciousness, which is brief, that is under 6 hours, in a TBI is consistent with a concussion. However, by definition concussion is not associated with gross morphological neuropathology, such as subarachnoid hemorrhage, subarachnoid hematoma, subdural hematomas, intraparenchymal hemorrhage, acute contusions or traumatic edema. Thus, the patient's TBI is best classified as moderate rather than mild or severe.

When you look at the patient's behavior, he was unable to advise either the EMTs who treated him at the scene or the Doctors who were involved in his care at the hospital how he ended up along the road. Following regaining consciousness, as occurred with this patient, these patients often appear dazed or confused, **not uncommonly showing retrograde amnesia (what occurred immediately before the event, pretraumatic amnesia) and anterograde amnesia (what occurred immediately after the event, posttraumatic amnesia) surrounding the event, which caused the unconsciousness.**

The determination of acute subarachnoid hemorrhage and a subarachnoid hematoma on CT scan following being found lying in the roadway suggested the patient sustained a significant traumatic injury in which large vessels traversing the subarachnoid space were torn, causing focal and or diffuse subarachnoid hemorrhage being detected on CT. Typically, subarachnoid hemorrhage is minimal, detected only by CSF examination and is of little clinical importance. This was not the case with this patient. **This finding is consistent with the classification of this patient's TBI as moderate rather than mild or as a concussion.**

The presence of subdural hematomas on CT along with the acute subarachnoid hemorrhage was due to movements of the brain within the skull, which led to stretching and tearing of "bridging" veins that drain from the surface of the brain to the dural sinuses. As occurred with this patient, subdural hematomas are located typically over the lateral cerebral convexities, however, they may also collect along the medial surface of the hemisphere, between the tentorium and occipital lobe, between the temporal lobe and the base of the skull, or in the posterior fossa, Usually, they present as a crescentic collection of blood.

Another important aspect to this patients neuropathological changes involving his brain following the first TBI was the fact that he was acutely intoxicated and was a chronic alcoholic. When he is examined at the hospital his blood alcohol level is 372 mg/dL, which is over four times the accepted level for alcohol in many states (<0.08).

Alcoholic patients not uncommonly have cerebral atrophy and thus are especially prone to subdural bleeding; in these patients, large hematomas may result from trivial impact or even from pure acceleration-deceleration injuries without a rotational component, as would occur in a whiplash injury. This patient's brain weighed 1276 grams at autopsy. Keeping in mind, both the cerebral hemispheres were swollen due to edema, especially the left cerebral hemisphere. Thus, in the normal state, the patient's brain weighed less than 1276 grams. The average brain weight for an adult male varies between 1336 and 1400 grams. Consequently, the patient had cerebral atrophy due to his alcoholism. **The resulting cerebral atrophy of his brain increased the chances of subdural hemorrhage due to the enlargement of the subdural space, which placed the "bridging veins" in a very precarious state in that they are not only stretched but have little tissue support, and thus, are more prone to being easily ruptured.**

Another feature of alcoholism is the effect alcohol has on blood clotting. Alcohol can cause abnormally low platelet numbers in the blood (i.e., thrombocytopenia), impaired platelet function and platelet aggregation (i.e. thrombocytopathy), diminished fibrinolysis, as well as problems involving erythrocytes, leukocytes and other hemostatic factors. These effects can have serious medical consequences, such as an increased risk of hemorrhaging. **On reviewing the laboratory findings in this patient's case, his blood clotting ability would have been compromised, even though his platelet count was normal, due to impaired platelet function, the consequence of which would be to enhance intracranial bleeding as subarachnoid and subdural hemorrhage, hemorrhage associated with contusions, and intraparenchymal hemorrhage. As noted above the patient manifested all of these features.**

It was noted on a CT scan of his head during the first hospital admission he had an intraparenchymal hemorrhage, which was consistent with a rotational component to the acceleration-deceleration movements of his brain within the skull following the blunt force impact to his head. **The intraparenchymal hemorrhage was due to the tearing of small to medium sized vessels within the brain parenchyma, which in turn led to an intracerebral hematoma (intraparenchymal hemorrhage).**

What is of interest, the patient's intraparenchymal hemorrhage of his right frontal lobe was not seen on the initial CT scan of his head, however, it became visible on the CT scans the following day. **Delayed traumatic intracerebral hemorrhage usually becomes apparent within 48 hours after the head injury**. The precise mechanism of this delayed hemorrhage is uncertain. One current theory is that post-traumatic coagulopathy results in continued or delayed microvascular hemorrhage, which in the case of this patient would have been enhanced due to his alcoholism; another, that the forces associated with the primary injury do not produce frank rupture of the micro-

vessels at the time of the injury but initiate molecular changes that result in subsequent structural failure, the consequence of which is hemorrhage.

The acute contusions noted in the patient's brain were due to focal parenchymal hemorrhages that resulted from scraping and bruising of the brain as it moved across the inner surface of the skull. As occurred in this patient, the inferior surfaces of the frontal and temporal lobes is where brain tissue meets with the irregular protuberances at the base of the skull, and thus are the most common sites of traumatic contusions, which are referred to as **gliding contusions**.

The contusions that occur as a result of impact with acceleration-deceleration, such as occurs in a fall, may occur underneath the point of impact (coup contusions) or distant from the point of impact (contrecoup contusions). In forward-fall coup contusions, scalp bruising is over the forehead, with the contusions involving the frontal and temporal lobes. This patient sustained facial contusions with dried blood around the forehead, periorbital and oral areas, and bilateral closed fractures of the nasal bones. **These injuries to the patient's face and nasal bones would suggest the contusions he suffered to his frontal and temporal lobes were consistent with a forward-fall induced coup contusions and not due to blows to the head, such as would occur with a fist.**

The patient's acute contusions showed an increase in size between the first day of admission and the next two days. This was due to the fact acute contusions are dynamic lesions that evolve with time. They may become larger as a result of further hemorrhage some hours after the initial head injury. What can enhance this enlargement is the inherent inhibitory effect alcohol has on coagulation. His alcohol level at the hospital was 372 mg/dL.

On pages 2 and 3 of this Forensic Science Newsletter there is a discussion of the patient's dysfunctional cognitive state. **Although cognitive impairment is a feature of alcoholism, it is also a component of TBI.** This is what the physician was alluding to in his progress note when he found the patient to becoming more restless and irritable, **which he attributed to alcohol withdrawal superimposed on a TBI.**

Cognitive impairment is by far the most common and disabling problem associated with TBI. During the acute phase, disorientation and agitation are particularly common, both of which this patient demonstrated. Besides cognitive and motor deficits, such patients may experience headache, dizziness, vomiting or vertigo. These symptoms usually disappear in a few weeks, but may persist for months. Common chronic cognitive problems include impaired short- and long-term memory (this patient had impaired short term memory), attention, and concentration; slowing of psychomotor speed and mental processing and changes in personality (this patient clearly showed slowing of mental processing and changes in his personality). **There may be loss of memory for the events that occurred in the immediate period after recovery of consciousness and a similar amnesia for the events immediately**

preceding the injury (the patient could not remember what occurred immediately before or after his TBI).

Another feature of cognitive impairment associated with alcoholism and recovery from TBI is lack of inhibition to inappropriate conduct, such as this patient demonstrated when he signed himself out of the hospital so he could go and smoke cigarettes, and his inappropriate conduct, which led to his assault and subsequent second TBI within a few months of the first.

There is another issue that needs to be addressed and that is the effect alcohol has on a TBI. It has been shown, people who use alcohol or other drugs after they have a brain injury don't recover as much. TBI patients often say and or do things, as this patient did, without thinking first, a problem that is made worse by using alcohol and other drugs (this patient used alcohol, marijuana, cocaine and amphetamines). TBI patients have problems with thinking, like concentration or memory, and using alcohol or other drugs makes these problems worse. Following a TBI, alcohol and other drugs have a more powerful effect. There are studies, which have shown the presence of alcohol or drugs in a patient who sustains a TBI potentiates the effects of the TBI. Patients who continue to use alcohol or drugs while recovering from a TBI prolong the effects of the TBI. It has been noted that especially during the early period of recovery from a TBI, the first several years when the brain is attempting to heal or accommodate the injury, alcohol inhibits this healing and accommodating process.

Second Traumatic Brain Injury

A little over two months later the patient sustained a second **TBI** following an assault due to the patient's inappropriate conduct. On arrival of the EMTs he is found to be unconscious. The EMTs noted bruising of the right eye, with a laceration at the corner. There was also an injury above the eyebrow. Due to the swelling of the right upper and lower eyelids, the patient's right pupil could not be examined. His left pupil was 4 mm in diameter, suggesting to the EMTs he had a head injury and there was possible drug and or alcohol use. The patient did smell of alcohol. The patient is placed in the ambulance at which time the EMTs noted his breathing was irregular and he had snoring respirations. **Snoring respirations** are also referred to as the "death rattle."

Snoring respirations are due to terminal respiratory secretions, which lead to the accumulation of salvia and bronchial secretions accumulating in the upper chest and throat (larynx and bronchial tree) due to the inability to swallow. Swallowing is a complex process requiring coordination within the autonomic nervous system, most especially the medial temporal lobe, limbic system, medulla oblongota, and pons. The snoring respirations are a terminal event with respiratory failure following shortly. **In this patient's case, they indicated he had suffered a catastrophic neurologic event. The nature of the catastrophic event, however, far exceeded the level of the assault, which the patient was subjected too.**

He is subsequently seen in the Emergency Department of the hospital in which he is described as a 45-year-old gentleman who is essentially obtunded with a GCS of 3. He is described as a patient who smells of alcohol and has had trauma. He is noted to have ecchymosis and swelling around the right eye, as well as a hematoma to the scalp of the left parietal area and some ecchymosis to the left flank. His right eye could not be opened and the left sclera is normal but the pupil is 6-7 mm and nonreactive.

The patient's **Problem List** was as follows: History of alcohol and drug abuse; respiratory failure following trauma; head trauma; closed blowout fracture of right orbit; acute alcohol intoxication (327 mg/dL); GCS total score 3-8; TBI, with loss of consciousness of 1 hour to 5 hours 59 minutes; and a post traumatic subdural hematoma.

CT of the Head revealed a right blowout fracture, as well as a nasal bone fracture. Intracranially there was a large left subdural hematoma with a maximal thickness of approximately 15 mm. There was also an associated left to right shift estimated at 11 mm. There was obvious mass effect on the left cerebrum with loss of the basal ganglia cisterns and contralateral dilatation to the temporal horn. These findings are likely related to uncal herniation. There was diminished density in the subcortical white matter of the anterior inferior right frontal lobe, which suggested an acute contusion.

CT of the Facial Bones revealed a right blowout fracture with 3 mm depression of the major fracture fragment. The AP (anterior-posterior) dimension of the orbital floor fractures was 25 mm. There was a small intraorbital hematoma adjacent to the medial orbital wall fracture with slight deviation of the medial rectus muscle. There was also an intact right globe with extensive overlying soft tissue swelling.

Due to the likelihood of an extremely poor neurological outcome, the family declined surgical intervention and elected for comfort care, which the patient was placed on late in the evening of the day of his admission. The patient was pronounced early in the afternoon the following day. From the time of the assault to the withdrawal of life support was approximately 5 hours, which is consistent with a precipitous neurologic decline.

The significant autopsy findings related to the head and brain were as follows: the scalp revealed two circular bruises in the left temporal region. There was an elliptical bruise in the left frontal region of the scalp just above the eyebrow. On the outside skin, there were petechial hemorrhages, but no additional bruise was noted. The right frontal an right lateral frontal region of the scalp also showed a bruise that was arising from the right orbital blowout fraction.

Removal of the calvarium revealed a subdural hemorrhage involving the left convexity and left basilar region of the brain. There were slight areas of hemorrhagic congestion in the frontal regions and the right central basilar frontal region of the brain. The dura was removed from this area and when the Forensic Pathologist compared his observations of this area with the CT scans, the neuropathologic features of that area

indicated acute contusions. The cerebral convexity was edematous. Examination of the unci, revealed bilateral uncal grooves. There was also evidence of a shift from left to right. The blood clots in total weighed 34 grams.

No fractures to the calvarium or base of the skull were noted, which suggest the blows to the head, although significant, were less than severe, and do not in of themselves account for the precipitous neurologic decline of the patient.

The neuropathologic features shown on CT scan and at the time of autopsy were not consistent with the blunt force trauma inflicted during the assault of the patient, for they were far more expansive, suggesting other factors had played a role in their genesis, i.e., previous TBI and alcoholism.

What is also of interest is the very rapid neurologic decline the patient experienced following the blunt force trauma he sustained, as noted by the EMTs at the scene of the assault and the Emergency Department Medical Personnel at the hospital. This precipitous neurologic decline manifested as a fixed dilated left pupil within a few minutes of the assault indicated the left oculomotor nerve was no longer functioning properly due to brainstem compression; snoring respirations due to malfunctioning of the autonomic nervous system and impending respiratory failure due to brain swelling, most especially the left cerebral hemisphere with herniation; and a GCS of 3, which is the lowest possible score (the highest GCS is 15).

A GCS of 3 refers to the eyes do not open following stimulation, there is no verbal response nor is there evidence of a motor response. Patients with a GCS of 7 or less are considered to reflect severe injury to the brain with a poor clinical outcome. **The subsequent withdrawal of life support within 5 hours of the patient's blunt force trauma due to his dire neurologic state is very rapid and unusual for the level of trauma he sustained from the assault, which involved approximately 4 blows to the head and right orbital region.**

The rapidity of his neurologic decline indicated there were several existing complicating factors, which were additive to the blunt force trauma inflicted during the assault. The complicating factors, which played a significant role in this patient's catastrophic neurologic decline and death were his acute alcoholic intoxication (372 mg/dL) at the time of his first TBI; his chronic alcoholism, which caused a delay in the underlying healing process of his brain from his first moderate TBI; and his acute alcoholic intoxication (327 mg/dL) at the time he sustained his second TBI, all of which led to the immediate development of second impact syndrome and death.

A patient who has suffered a TBI and is still in the phase of healing is especially susceptible to the effects of a second TBI. This is especially true in a patient who is an alcoholic and continues to consume alcohol during the healing phase of the first TBI and has consumed alcohol immediately before the second TBI. As was stated on pages 9 and 10 of this Newsletter, **"It has been noted that especially**

during the early period of recovery from a TBI, the first several years when the brain is attempting to heal or accommodate the injury, alcohol inhibits this healing and accommodating process." Thus, when the patient sustained his second TBI, most especially while acutely intoxicated (327 mg/dL), and while still in the healing phase from the first TBI some two months earlier, he was especially vulnerable to a catastrophic outcome due to a phenomenon known as the **second impact syndrome**.

Pathophysiology

The pathophysiology of the **second impact syndrome** is generally believed to be caused by a loss of autoregulation of the cerebral vasculature. This dysautoregulation leads to a loss of the brain to autoregulate cerebral perfusion pressure and intracranial pressure. Such loss of autoregulation causes intense vascular congestion, hyperemia, which in turn leads to a marked increase in intracranial pressure. The patient suffered a very rapid onset of cerebral brain swelling, especially the left cerebral hemisphere, within a matter of a few minutes following the blunt force trauma to his head. The development of the cerebral edema was virtually immediate and was primarily vasogenic, but also had a cytotoxic component. The intense vascular congestion and cerebral edema of his brain, especially the left cerebral hemisphere led to an increase in intracranial pressure, which in turn led to inferomedial herniation of the temporal lobes and herniation of the cerebellar tonsils, which in turn distorted and compressed the brainstem. Once brainstem distortion and compression occurred, ocular involvement and respiratory failure followed rapidly, which is what occurred in this patient.

The usual time from the second impact to brainstem failure is a matter of a few minutes, which is what occurred with this patient. He had a fixed dilated left pupil, indicating brainstem compression, within a few minutes of the blunt force trauma to his head, as well as snoring respirations indicating failure of his autonomic nervous system.

Some cases of second impact syndrome will also have an acute subdural hemorrhage, which was the case with this patient. Such patients typically will show virtual instantaneous unconsciousness with prolonged coma due to direct injury to the reticular activating system. This patient showed virtual instantaneous unconsciousness.

From a pathophysiological standpoint the patient's first TBI some two months before his assault resulted in activation of an immune inflammatory response (immunoexcitotoxicity), which involved an interaction between immune receptors within the central nervous system (CNS) and excitatory glutamate receptors; this interaction triggered a series of events, including extensive reactive oxygen species/reactive nitrogen species generation; accumulation of lipid peroxidation products; and prostaglandin activation, which then led to dendritic retraction, synaptic injury, damage to microtubules, mitochondrial suppression and the death of neurons.

An important component of this immune inflammatory response is the activation of N-methyl-D-aspartic acid receptors (NMDA), which in turn increases the levels of

extracellular excitatory amino acids, glutamate and aspartate. A major release of these excitatory neurotransmitters is common after a TBI and is believed to be the proximate cause of a series of neurochemical sequelae of cortical (brain) injury. The subsequent chain reaction results in an intracellular influx of calcium, which in turn activates calcium dependent enzymes that cause mitochondrial lysis and neuronal cell death. There is also evidence that the neuronal cell death occurs through sodium intracellular influx, which in turn causes massive cellular swelling and thus brain edema, which this patient's brain demonstrated.

To further complicate the pathophysiology of second impact syndrome, the patient had an alcohol level of 327 mg/dL at the time of his second TBI. Such levels of alcohol inhibit the NMDA receptors, which in turn exacerbates neuronal damage. Thus, the patient's alcohol level of 372 mg/dl at the time of his first TBI, exacerbated neuronal damage in his brain, which normally occurs after a TBI, as well as extending the brains healing process for months. His second TBI approximately two months later, was also exacerbated due to the on going healing from his first TBI, which was further exacerbated by his acute alcohol intoxication of 327 mg/dL at the time of this injury.

The second impact syndrome occurs because the patient sustains a second TBI before the patient's brain has healed from the first. In this patient's case this was further exacerbated due to his intoxication during his first TBI, which enhanced the neuronal damage to his brain from the TBI; his continued use of alcohol following his first TBI, which enhanced neuronal cell death and prolonged his brain's healing process; and his acute alcohol intoxication during the second TBI, which enhanced the development of immunoexcitotoxicity and the development of second impact syndrome, ultimately leading to catastrophic neuropathologic damage to his brain and his death.