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### Tension Pneumocephalus

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# Tension Pneumocephalus

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## Introduction

Pneumocephalus is defined as the presence of air within the cranial vault; tension pneumocephalus occurs when air within the cranial vault becomes trapped and causes a mass effect or increased pressure, damaging the brain (Patel, Idicula, Carrau, & Prevedello, 2013). Small amounts of air within the cranial vault can be reabsorbed or metabolized by the body over a few days without causing symptoms, however, in tension pneumocephalus the air cannot escape, causing extensive neurological symptoms (Webber-Jones, 2005). Thus, asymptomatic pneumocephalus can be a common finding following a cranial surgery, where as tension pneumocephalus is extremely rare (Webber-Jones, 2005).

Markham (1967) noted that *H. Chiari* was the first to describe intracranial pneumocephalus in an autopsy he performed of a patient who died from ethmoiditis in 1884. *W. H. Luckett* was the first to use x-ray diagnostics to establish a diagnosis of pneumocephalus in 1913, and *E. Wolff* was the first to coin the term "pneumozephalus" in 1914 (Markham, 1967, p. 2). Of the 295 pneumocephalus cases

reviewed, Markham (1967) reported that 218 were attributed to trauma, 38 to a neoplasm, 26 to infection, 11 were postoperative, and 2 were idiopathic (p.13).

Revealing even a small amount of intracranial air (0.5ml), a non-contrast CT scan is the diagnostic tool of choice for tension pneumocephalus, as it can also exclude other diagnoses (Simmons & Luks, 2013). Mount Fuji sign is a CT scan finding in which a subdural air collection causes compression and separation to the frontal lobes giving a peaked appearance similar to the peaks of Mount Fuji (see Figures 1 & 2) (Michel, 2004). The Mount Fuji sign is associated in patients with tension pneumocephalus (see Figure 1 & 2) (Michel, 2004). Tension pneumocephalus is a surgical emergency requiring immediate attention (Patel et al., 2013). Without immediate and aggressive treatment, and in some cases even with treatment, neurological deterioration, cerebral cellular infarct and brain herniation can result, ultimately leading to death (Wohlgemuth, 1985). Thus, tension pneumocephalus has a high morbidity and mortality, if not recognized early with prompt intervention.

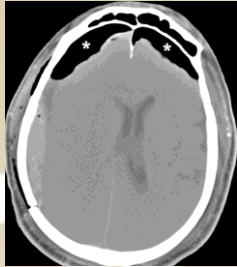


Figure 1. Unenhanced transverse CT image of the brain demonstrates bilateral subdural areas of hypoattenuation (\*) with compression of the frontal lobes. From "The Mount Fuji Sign" by S. J. Michel, 2004, *Radiology*, 232(2), p. 450. Copyright 2004 by The Radiological Society of North America. Reprinted with permission.

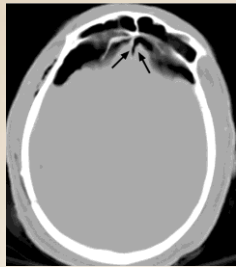


Figure 2. Unenhanced transverse CT image of the brain obtained caudal to image shown in Figure 1. Widening of the interhemispheric space between the tips of the frontal lobes is noted. The medial surface of each frontal lobe is marked (arrows). From "The Mount Fuji Sign" by S. J. Michel, 2004, *Radiology*, 232(2), p. 450. Copyright 2004 by The Radiological Society of North America. Reprinted with permission.

## Case Study

A 68-year-old man with a diagnosis of stage IV esophageal cancer presented to the emergency department, complaining of the worst headache of his life and was found to have an intracranial mass on computed tomographic (CT) imaging. (Patel et al., 2013). The patient was admitted to the hospital for IV corticosteroids and additional work up. He continued to have progressively worsening symptoms over then next 48 hours, including new onset seizures, vomiting, worsening headache, and eventually became obtunded. The patient was sent for an emergent repeat head CT scan, which revealed an astonishing finding, tension pneumocephalus.

The patient underwent emergent intubation and external ventricular drain (EVD) placement by the neurosurgical team. Upon further investigation the patient was found to have an esophageal-subarachnoid fistula caused by invasion of his esophageal tumor at the level of C7 and T1. The patient survived the event due to the prompt diagnosis and intervention by the medical and neurosurgical team, but ultimately went home with hospice care due to his progressive metastatic cancer. Due to rarity of the clinical findings in this case it was recently published in *Head and Neck, Volume 36, Issue 6*, under the title "Esophageal-subarachnoid fistula: A case of spontaneous tension pneumocephalus in the setting of esophageal cancer" (Patel et al., 2013). Patel et al. (2013) theorized that the air was most likely forced through the esophageal-subarachnoid fistula when the patient experienced his vomiting and reaching episodes. The initial CT imaging in the ED did not reveal any signs of air, where as the scan 48 hours later revealed the tension pneumocephalus after the progressively worsening neurological symptoms, including the seizure and vomiting (Patel et al., 2013).

## Pathophysiology

When trying to understand the pathophysiology behind the rare condition of tension pneumocephalus and the theories behind its manifestation, Gozur (1987) suggested that one must first understand the intracranial anatomy, physiology and the Monro-Kellie doctrine. There are three intracranial volumes all within the rigid confines of the human skull: 1) cerebral spinal fluid (CSF), 2) brain tissue and 3) blood (Porth, 2007, p. 826). Normally there is a reciprocal relationship that exists among these three intracranial volumes to maintain stable intracranial pressure (ICP) so that if and when a change in one volume occurs the others must balance the effect by an equal or opposite effect, known as the Monro-Kellie doctrine (Porth, 2007, p. 826). Thus, the brain has an auto regulatory system in order to maintain blood flow by changing the diameter of the blood vessels to get oxygen and glucose to the tissues to prevent brain damage (Patho Puzzler, 2007). If the body is unable to compensate for a change in volumes, increases in ICP can result.

Normal ICP is measured between 0 to 15 mm Hg in the lateral ventricles (Porth, 2007, p.825). Normal cerebral perfusion pressure (CPP) is 70 to 100mm Hg (Patho Puzzler, 2007). As ICP rises, cerebral pressure gets closer to the mean arterial pressure and it becomes more difficult for blood to enter the intracranial space (May, 2009). At this point, the brain is still able to auto regulate and reacts by decreasing the CPP (May, 2009). This causes the cerebral blood vessels to dilate, increasing blood volume and pressure, subsequently increasing the ICP even more (May, 2009). As this vicious cycle continues, ICP increases, CPP decreases and clinical symptoms begin to worsen. When ICP increases over 40mm Hg the CPP decreases to a point where consciousness is lost (May, 2009). Progressive increase in ICP leads to hypoxia, hypercapnea, and mass effect resulting in signs such as papillary dilation and Cushing's triad (May, 2009). Cushing's Triad is an early warning sign of brain herniation, consisting of hypertension with widening pulse pressure, bradycardia, and altered respiratory patterns. If excessive increase in ICP occurs, cerebral blood flow becomes obstructed, causing cerebral cellular infarct, brain tissue displacement and herniation, leading to death (Porth, 2007, p. 826).

In tension pneumocephalus it is the trapped air that enters the cranial cavity which causes the increased ICP and mass effect. sneezing would push the dura away from the bone and readily force the air through the relatively large opening of the dura into the cerebral defect; after this the heightened intracranial pressure would seal the dura against the depressed bone and effectively close the opening (p. 973).

These two theories, the *ball-valve mechanism* and the *inverted soda-pop bottle mechanism* for the entrapment of air in the intracranial compartment are still relevant today, and are consistently reported, accepted and used in the current literature.

As little as 25cc of trapped air can cause tension pneumocephalus (Webber-Jones, 2005). Thus, the signs and symptoms of tension pneumocephalus mimic the signs and symptoms of increased ICP.

### Early Signs of Increased ICP:

- Change in mental status (ex. confusion, restlessness, agitation)
- Increased respiratory effort
- Constant headache
- Purposeless movements
- Unilateral weakness
- Pupillary changes
- Headache,
- Nausea and vomiting
- Seizures (Patho Puzzler, 2007).

### Progressive and Late Signs of Increased ICP:

- Decreased level of consciousness
- Decreased/ altered respiratory patterns (ex. Cheyne-Stokes breathing),
- Bradycardia
- Increased blood pressure and widening pulse pressure
- Increased temperature
- Projectile vomiting
- Hemiplegia
- Decorticate or decerebrate posturing
- Loss of brainstem reflexes eventually leading to death (Patho Puzzler, 2007).

There are two major theories of mechanism to how air becomes trapped within cranial space. The first is the "inverted pop bottle syndrome" first theorized by Lunsford, Maroon, Sheptak & Albin (1979, p. 525). Lunsford et al. (1979) state "the mechanism of air entry into the intracranial compartment is analogous to the entry of air into an inverted soda-pop bottle. As the fluid pours out, air bubbles to the top of the container" (p. 525). The second is the "ball-valve mechanism" first described by Dr. Walter E. Dandy in 1926 (Dandy, 1926, p.973). Dandy (1926) describes this mechanism stating,

sneezing would push the dura away from the bone and readily force the air through the relatively large opening of the dura into the cerebral defect; after this the heightened intracranial pressure would seal the dura against the depressed bone and effectively close the opening (p. 973).

These two theories, the *ball-valve mechanism* and the *inverted soda-pop bottle mechanism* for the entrapment of air in the intracranial compartment are still relevant today, and are consistently reported, accepted and used in the current literature.

## Nursing Implications

Tension pneumocephalus, though a very rare occurrence, requires attentive nursing care to assess for signs of subtle neurological change and deterioration in order to initiate appropriate diagnosis and medical interventions. The Glasgow Coma Scale is a commonly used tool to objectively measure the level of consciousness (arousal and cognition) of a patient (May, 2009). The scale assesses best eye movements, best motor response, and best verbal response, with a score out of 15, where a falling score indicates neurological deterioration, and a score of less than 8 indicates that the patient needs immediate intubation (May, 2009). This tool can easily be used by nursing staff to assess subtle changes in consciousness, and initiate the appropriate interventions in a timely fashion to optimize the patient's recovery if neurological compromise is found.

The nurse must also be aware of preventative measures and predisposing factors to the development of tension pneumocephalus, especially in the post-operative setting. With cranial surgery more common today, pneumocephalus is often seen following surgery where the bone and dura are compromised (Webber-Jones, 2005). Schirmer, Heilman & Bhardwaj (2010) noted that predisposing factors to the development of tension pneumocephalus include such factors as head position during operation, nitrous oxide anesthesia, continuous CSF drainage via a lumbar drain, otitis media, intraoperative osmotherapy, hydrocephalus, hyperventilation, barotraumas, epidural/spinal anesthesia, and neoplasm. Tension pneumocephalus has also been associated with iatrogenic violation of the dura through the cribriform plate during endoscopic procedures (Simmons & Luks, 2013).

### Prevention Measures:

- Educate patients and families in neurological assessment (Webber-Jones, 2005).
- Assess for signs of a CSF leak, such as clear draining fluid from the nose or ears (Webber-Jones, 2005).
- Have the patient avoid activities that increase barometric pressures such as coughing, nose-blowing, valsalva maneuver, CPAP or BIPAP (Webber-Jones, 2005).
- Promote medications such as stool softeners and cough suppressants (Webber-Jones, 2005).
- Patients should be instructed not travel via airplane and to avoid exposure to changing air pressure (Webber-Jones, 2005).
- Plan for the patient to receive serial imaging to ensure the gradual reduction of intracranial air overtime (Schirmer et al., 2010).

Once tension pneumocephalus is confirmed, usually by non-contrast CT scan (Figure 1 & 2), all efforts are directed at preventing further mass effect and reducing ICP to restore blood flow, oxygenation and nutrients to the delicate brain tissues. (Webber-Jones, 2005). Oxygen is an intervention that has been found to be a vital treatment for tension pneumocephalus. Breathing high oxygen concentrations can create a pressure gradient, which increases the rate of nitrogen diffusion from the intracranial air collection to the blood stream, where it is then expelled by ventilation through the lungs (Simmons & Luks, 2013). For severe cases, surgical decompression may be needed (Simmons & Luks, 2013). Drug treatments following air evacuation may include hyperosmolar medications such as hypertonic saline or mannitol therapy to decrease brain swelling and prevent further mass effect (Webber-Jones, 2005). Additionally, sedatives such as propofol and barbiturates may be needed to suppress cerebral metabolism, and neural activity to decrease cellular damage (May, 2009).

### Additional Nursing Interventions:

- Placing the patient in a supine position to decrease additional air accumulation (Simmons & Luks, 2013).
- Reduce cellular metabolic demands by keeping the patient's head midline and controlling temperature with cooling blankets and medications (Patho Puzzler, 2007).
- Monitoring blood pressure and ICP (Patho Puzzler, 2007).
- Avoiding frequent arousal of the patient, and maintain a calm and noise free environment (Patho Puzzler, 2007).
- For non-intubated patients, ensure a patent airway and monitor respiratory patterns (Patho Puzzler, 2007).



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## Conclusion

Tension pneumocephalus is a rare, but deadly neurological complication if it is not diagnosed and treated promptly. It is important to remember that early detection is key. Nurses need to be attuned to subtle changes in their patient's condition as well as educate and teach the patient and family to be attuned to these changes as well. Any subtle signs of neurological compromise should not be taken lightly. Prompt evaluation and diagnosis needs to be made in order to preserve the delicate brain tissue needed for a complete recovery of homeostasis. If the diagnosis of tension pneumocephalus is made, continued monitoring of the patient's consciousness and condition is paramount. The patient's clinical condition and stability will help to direct the treatment course. Most likely neuro-surgical intervention will be needed emergently to evacuate the trapped air, though some nursing interventions can assist in preventing further damage and promote recovery.