

CASE STUDY: BRAIN INJURY

One in a series of case studies developed to stimulate enhancement of problem-solving techniques for physicians, nurses and paramedics. This case study is a composite developed from a number of patient transfers performed by REACH Air Ambulance.

This Case Study is provider approved by the California Board of Registered Nursing, provider number 9697, for 1.0 contact hour. This course has been approved for one hour of category one EMT-P continuing education by California EMT-P provider number 49-008.

LEARNING OBJECTIVES

After completion of this educational process the participant will be able to:

- Define cerebral perfusion pressure (CPP) and mean arterial pressure (MAP).
- Describe the roles of ventilation and Mannitol when treating patients with increased intracranial pressure (ICP).
- Define primary and secondary brain injury.
- Identify changes in ICP which occur as a result of central nervous system lesions which cause increased intracranial volume.

HISTORY

A 60-year-old female presents to the emergency department with a complaint of frontal headache for one week. On the day of admission to the ED she experienced an increase in intensity of the headache, it being more global in nature. She described the pain as "very intense." The patient also complained of being "weak all over," however, she denied any focal weakness. An additional complaint was loss of equilibrium. She denied change in hearing or vision.

PAST MEDICAL HISTORY

The patient had a history of high blood pressure. However, she had stopped taking her antihypertensive chemicals one year ago because they "made her feel bad." She denies other medical problems.

SOCIAL HISTORY

In the remote past she smoked. She denies alcohol abuse.

MEDICATIONS/ALLERGIES

She is not currently taking medications. She has no medical allergies.

PHYSICAL EXAMINATION

Temperature 98.1° F (36.7° C), heart rate 55, respirations 20, blood pressure 260/120. Pulse oximetry demonstrated oxygen saturations which were never lower than 98%. In general the patient is sleepy, but when aroused she is alert and oriented. Extra ocular motor activity is normal. GCS is 14-15. Pupils are equal and reactive. An attempt to visualize the fundus was unsuccessful because of lack of patient cooperation. The neck was supple and without pain during range of motion. Lungs were unremarkable. Heart was unremarkable: Extremities were unremarkable. Neurological exam demonstrated no evidence of lateralizing signs. Deep tendon reflexes 1+ symmetrical. Motor strength was slightly decreased but symmetrical.

What are your concerns? What tests would you have performed? Would you treat the patient's elevated blood pressure? Formulate your answer before proceeding.

COURSE IN THE EMERGENCY DEPARTMENT

Patient was treated with 10 mg Nifedipine (Procardia) by mouth with the intention of lowering her blood pressure. She was then sent for a head CT scan which revealed a large area of left frontal and parietal intracerebral hemorrhage. The intracerebral hematoma was of such an extent to produce a midline shift. In addition there was evidence of swelling of the brain tissue contiguous to the area of hemorrhage.

Upon return from the CT scanner the patient was noted to have experienced a decrease in her level of consciousness. Her GCS was now 8-9.

She was endotracheally intubated using a rapid sequence induction technique. Her blood pressure was controlled with intravenous boluses of Labetalol (Trandate). Subsequent blood pressures were in the range of 168-200 systolic and 70-110 diastolic. Chemicals were administered to sedate and

paralyze. The patient was administered one Gram of Phenytoin (Dilantin) for seizure prophylaxis.

In view of the fact the hospital did not have neurosurgery capability; arrangements were made to transfer the patient to a tertiary care center by helicopter air ambulance. REACH was contacted and dispatched to assess, stabilize and transport the patient.

AIR AMBULANCE TEAM

Assessment by the flight crew demonstrated an endotracheally intubated, chemically paralyzed patient. Blood pressure was 209/95, HR 72. The patient received additional Labetalol from the flight team.

En route the patient required additional intravenous sedation and chemical paralysis. No significant change in her status occurred while in the care of the REACH flight crew.

DISCUSSION

Primary brain injury is a consequence of the event which caused the initial brain insult. This event can be traumatic or non-traumatic in nature. An example of a traumatic lesions resulting in primary brain injury is a cerebral contusion as a consequence of an isolated blow to the head. Examples of non-traumatic primary problems are: hemorrhagic and thrombotic stroke, tumors or infectious lesions. These primary events cause immediate swelling, bleeding and, at times, "mass" effect, which means there is sufficient volume of blood and/or edema to displace normal brain tissue from its typical position.

Secondary brain injury occurs as a consequence of the primary insult. Its cause is multifactorial including any or all of the following: lack of oxygen supply, inadequate blood flow or diminished availability of energy substrate (glucose) to the area of injury. In addition, the cells injured at the time of the primary event subsequently release substances into the extracellular space which are toxic to marginally injured or non-injured brain tissue. The "ripple effect" is then "in play" with more and more brain tissue injured as the on going process unfolds.

To a significant extent controlling and decreasing the degree of secondary brain injury is within our control as medical care givers and is accomplished by skilled care emphasizing appropriate ventilation and oxygenation, proper blood flow (not too much, not too little) and delivery of adequate energy substrate to brain tissue so aerobic metabolism can be maintained.

Most caregivers when treating intracranial injury are very concerned, and appropriately so, about intracranial pressure. However, a key concept to understand is increased intracranial pressure occurs as a result of increased intracranial volume. The brain is enclosed in a rigid box, the skull, which has a limited volume capacity. When the capacity is exceeded by increased blood volume, swelling, hemorrhage, tumor or infectious mass, intracranial pressure increases rapidly.

(See below)

How may we as caregivers best prevent or minimize secondary brain injury? Pay scrupulous attention to:

VENTILATION/OXYGENATION

If the patient is able to maintain a stable airway and oxygen can be delivered to maintain oxygen saturations of 97% or greater, endotracheal intubation may not be necessary. If not, endotracheal intubation should be accomplished and the patient maintained in a paralyzed and sedated state. Carefully monitor the PaCO₂ or ETCO₂ (more on this under "Control of intracranial pressure").

CIRCULATION

An extraordinarily important concept for caregivers to understand is the significance of maintaining adequate cerebral perfusion pressure (Cerebral perfusion pressure (CPP) equals mean arterial pressure (MAP) minus intracranial pressure (ICP). $CPP = MAP - ICP$.

Most blood pressure measuring devices will provide an MAP. If yours does not, MAP can be roughly calculated using the following formula: diastolic pressure +1/3 of the difference between the diastolic and systolic pressures. An example: 200/140; diastolic 140, difference between 200 and 140 is 60. 1/3 of 60 is 20. $20 + 140 = 160$ (MAP).

To appropriately maintain CPP, it is clear MAP must be optimized and ICP controlled.

Our clinical goal should be to maintain CPP at or above 80 mmHg in the adult and 60 mmHg in the child. Normal MAP in an adult is 90-100 and in a child it is 70-80. Normal ICP in an adult and a child is less than 15. Knowing these

normals, evaluating the MAP, and assuming any patient with an intracranial lesion causing a change in the level of consciousness will probably have an elevated ICP, we can provide more precise care in our attempt to minimize secondary brain injury.

Methods to ensure adequate MAP include volume resuscitation with fluids and/or blood products and if necessary the use of a vasopressor such as dopamine. Hypotension should be avoided at all costs. One study demonstrated if there was even one systolic blood pressure measurement of 90 or below in a person with brain injury, mortality doubled.

It is of note a normal physiological response to an increase in ICP is an increase in BP and a decrease in pulse rate. This is Mother Nature's way of attempting to maintain CPP in the face of an increasing ICP. This is also termed the "Cushing response."

What about the patient who is Hypertensive?

Treatment of hypertension should be considered only after CPP is determined or estimated. In those cases where we do not know the ICP we have to make a guess as to what the ICP is. In most cases when the patient is unconscious you can assume an ICP in the range of 15-20 mmHg or higher. In general if we have no other means of knowing the ICP we should aim for an MAP in the range of 75-90mmHg. It is usually best to consult with the treating neurosurgeon but if this is not available these guidelines will suffice.

What measures can be used to control blood pressure?

Primary considerations are adequate sedation and analgesia, which could reduce BP to an acceptable range. However, it is essential to recall we are not attempting to achieve large decreases in blood pressure, nor do we want to administer chemicals which could cause a sudden precipitous drop in blood pressure. The result could be disastrous to brain cells. One chemical which could result in a precipitous drop is Nifedipine (Procardia). We do not believe it should be used in any circumstance as a chemical to control blood pressure regardless of the level of consciousness of the patient.

What chemicals should we use to control blood pressure?

An IV calcium channel blocker such as Nicardipine (Cardene) is an excellent choice. An infusion is preferred for continuing treatment because IV boluses and

oral medications can result in precipitous drops of blood pressure not easily or quickly reversible. The consequence could be an inadequate mean arterial pressure and secondary brain injury (brain cell death).

A guideline for desirable lower levels of systolic and diastolic blood pressure when treating with antihypertensive medications is a MAP of 75-90 mmHg or the range selected by the neurosurgeon.

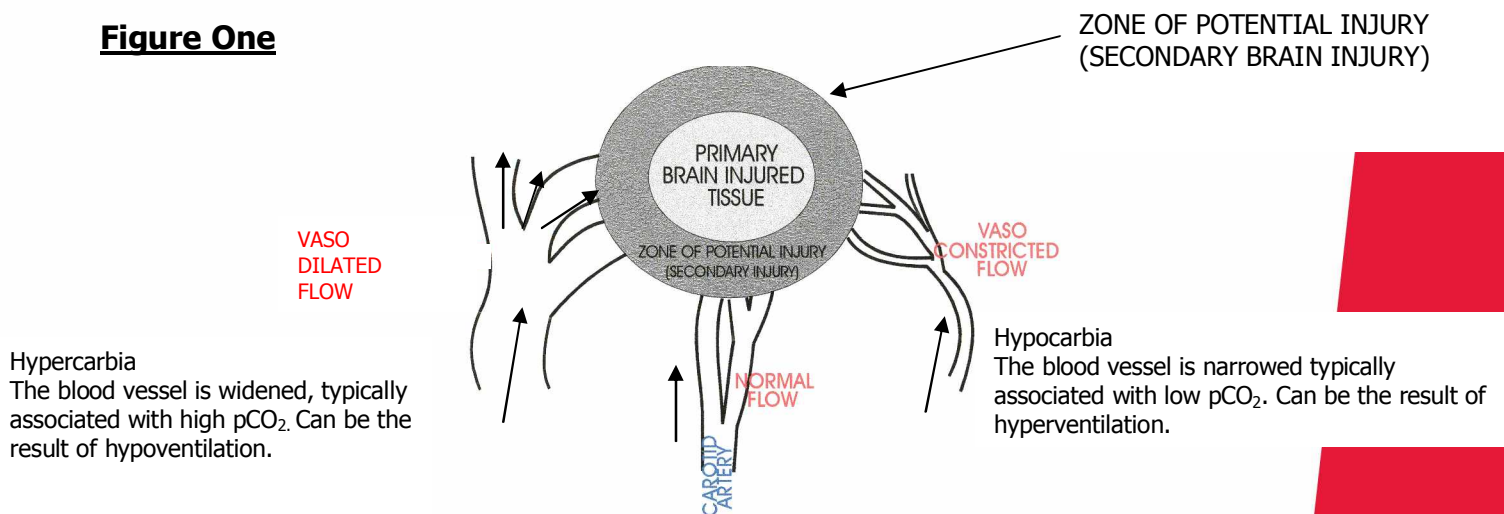
INTRACRANIAL PRESSURE/VOLUME CONTROL

In the past few years the recommendation to hyperventilate brain injured patients has been considerably modified. Ten years ago it was recommended PaCO₂ should be kept at approximately 25 mmHg. Current recommendations by the American College of Neurosurgeons are to maintain the PaCO₂ at near-normal levels. Our target at REACH is 35-45 mmHg. When using an end-tidal CO₂ monitor, a level of about 40 mmHg is optimal.

Why is hyperventilation no longer recommended?

Because hyperventilation and the resultant hypocarbia (low level of CO₂) results in vasoconstriction (narrowing) of cerebral vessels. The consequence could be inadequate blood flow to the injured tissue, increasing secondary brain injury. See Figure One, below.

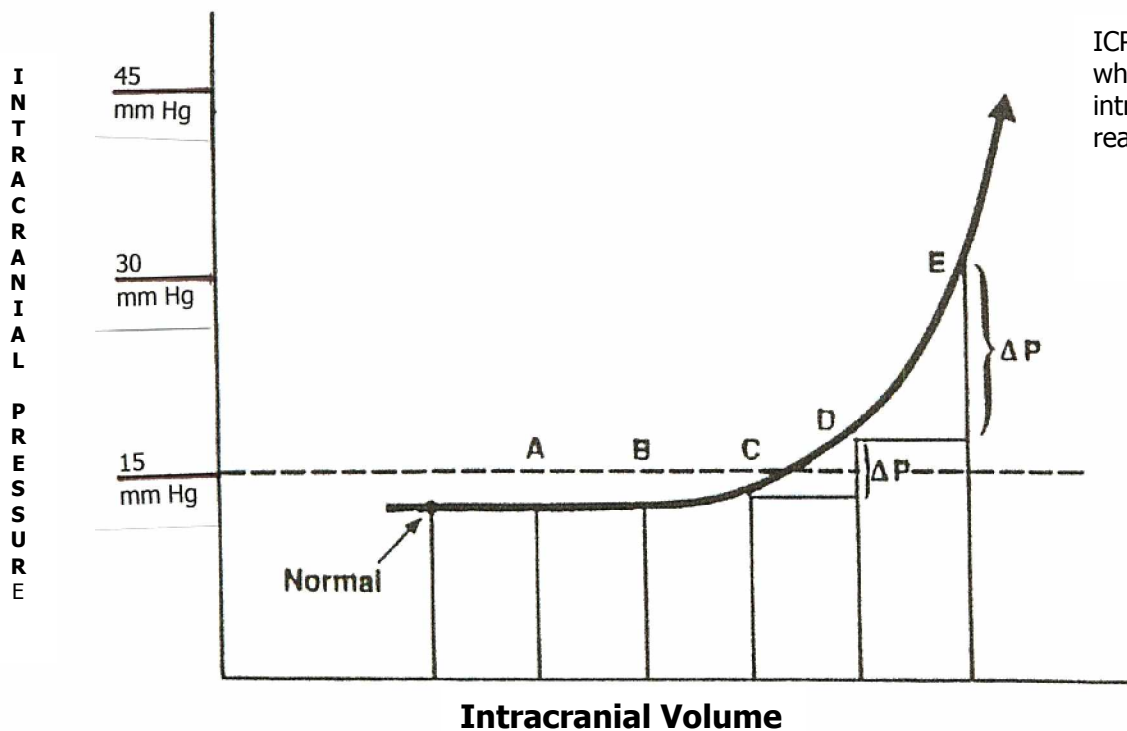
Figure One



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We recommend hyperventilation occur only if there are signs of increasing intracranial pressure such as pupillary inequality, decreasing level of consciousness, posturing, etc. We must remember, however, hypoventilation with resultant hypercarbia (high pCO₂) results in increased blood flow which in itself can be deleterious for a number of reasons. One significant consequence is the resultant increased brain volume which could lead to increased ICP. See figure two.

Figure Two



ICP increases rapidly when a critical intracranial volume is reached.

Idealized curve of relationship between intracranial volume and intracranial pressure.

Note: As intracranial volume increases a critical volume (in this graph at "C") is reached beyond which there is a rapid increase in intracranial pressure.

By what method should we hyperventilate a patient?

Increase the ventilator rate by approximately 10% for five minutes and observe for improvement such as a decrease of pupillary dilatation, improvement in the level of consciousness, decreased posturing, etc. As an example, if the patient is being ventilated at a rate of 16, increase the rate by about 10% to 18 for five minutes. If there is clinical improvement, decrease the rate again to 16 and observe. Also observe the ETCO₂ - it will probably decrease from 35-45 to the 25-30 range while you are hyperventilating.

Hypocarbica with its resultant decrease in cerebral blood flow (CBF) is not the only problem associated with ventilation. Hypercarbia or high carbon dioxide results in vasodilation or increased CBF. The consequence could be sufficient increase in brain blood volume to result in a dangerous increase in ICP. (See figure two, above)

CONTROLLING ICP BY ENHANCING VENOUS DRAINAGE

It stands to reason if we desire to control intracranial volume and thus ICP we should take measures to enhance drainage of venous blood out of the head. We can do so by elevating the head of the bed 20-30° to obtain an assist from gravity – unless the patient is hypotensive or has an inadequate MAP. If so, keep the head level with the torso to enhance blood flow into the head. Other methods of enhancing venous drainage are keeping the head in the midline, avoiding flexion or extension, and being careful not to allow constricting material such as endotracheal tube securing devices or C-collars to compress the jugular veins.

MANNITOL

Mannitol, a potent diuretic, draws fluid from brain cells into the blood stream then out through the kidneys. Its use is no longer recommended except in acutely deteriorating situations. For example: while hurrying the patient to an operating room for a craniotomy. The rationale for the use of Mannitol is to decrease brain volume and thus, indirectly, ICP.

Why is Mannitol risky?

Mannitol can result in hypovolemia leading to rapid drops in blood pressure and therefore MAP. CPP could then be inadequate to perfuse the injured brain tissue.

STERIODS

Steroids have not been proven useful in decreasing secondary brain injury associated with acute injury. Their use is not recommended, and in fact discouraged, because of potential serious side effects.

ENERGY SOURCE AND OXYGEN CARRYING CAPACITY

Another important consideration is adequate availability of glucose for the injured cells. Clearly, glucose should be measured at the time of initial treatment and frequently thereafter. This is particularly important when treating children whose low levels of glycogen reserves can be exhausted rapidly when the child is stressed. In addition, it is important to measure hemoglobin. Hemoglobin carries oxygen to the cells. If there is not sufficient hemoglobin to carry adequate oxygen to the cells, secondary brain injury could result.

SEIZURES

Seizure activity is very deleterious to cellular function, consuming oxygen and glucose in a rapid manner. Therefore, prophylactic anti-seizure medication should be given in most instances of brain injury. Phenobarbital is probably the best choice because it is longer acting than benzodiazepines such as diazepam (Valium), Midazolam (Versed) or Lorazepam (Ativan) and less likely to cause respiratory depression requiring endotracheal intubation. Dilantin is also a reasonable choice.

TEMPERATURE

At issue is at what level is the optimum temperature. Certainly, hyperpyrexia or high fever is deleterious. The febrile state increases oxygen consumption and can result in secondary brain injury. Use antipyretics in adequate dosages. There are those who believe hypothermia could be advantageous when treating brain injury. We favor slight hypothermia. There is no unanimity with regard to this recommendation and those who are treating primary brain injury and attempting to minimize secondary brain injury should seek the advice of an intensivist or a neurosurgeon when determining at what level to maintain the core temperature.

PROCEDURES

Procedures such as suctioning, laryngoscopy or endotracheal intubation can result in a patient response such as coughing or straining.

The result: surges in ICP by increasing intracranial venous pressure. Therefore it is critical adequate sedation and analgesia be administered. In addition intravenous or topical anesthesia, such as Lidocaine, could decrease these undesirable responses.

KEY POINTS

- Primary brain injury is avoidable only in terms of prevention.
- Secondary brain injury can be significantly influenced and moderated by proper attention to detail. See below.
 - Maintain oxygen saturations, by whatever means necessary, at 97% or greater.
 - Ensure adequate MAP (80 mmHg in the adult, 60 mmHg in the child).
 - Ensure adequate fluid volume to maintain MAP but not so much that intracranial volume becomes excessive.
 - Elevated blood pressure should be treated only after careful consideration of the CPP.
 - Scrupulously avoid hypotension
 - Maximize venous drainage from the head to prevent excess intracranial volume leading to increased ICP.
 - Monitor PaCO₂ or ETCO₂ - keep, in the absence of acute deterioration, at about 40 mmHg.
 - Ensure adequate hemoglobin levels to transport sufficient oxygen to the injured cells to maintain aerobic metabolism.
 - Ensure adequate glucose availability.
 - Provide adequate sedation and analgesia.
 - Treat/Prevent seizures.

We would welcome any questions or comments about this case study. We would also welcome any suggestions relevant to developing a case study from an interesting case involving your unit and REACH.

Let us hear from you. Should you desire to read previously published case studies and the opportunity to receive additional CEUs, visit our website at www.reachair.com. You can do so online.

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CASE STUDY

POST TEST

BRAIN INJURY

QUESTIONS: choose **all** correct answers.

1. Choose all correct answers. Lesions in the cranium (skull) which increase intracranial volume can cause:
 - A. Movement of the brain to the side opposite the lesion.
 - B. An increase in intra-cranial pressure.
 - C. A decrease in intra-cranial pressure.
 - D. Secondary brain injury.

2. If a patient has an MAP of 140, a known intra-cerebral hemorrhage, and is unconscious with an ICP of 30, what is the cerebral perfusion pressure?
 - A. CCP = 60
 - B. CCP = 80
 - C. CCP = 110
 - D. Unable to calculate.
 - E. Too high to measure.

3. In order to minimize secondary brain injury:
 - A. The patient's oxygen delivery should be kept as low as possible to prevent oxygen toxicity.
 - B. The patient's oxygen delivery should be kept at 3 LPM via nasal canula.
 - C. The patient's oxygen delivery should be monitored by pulse-oximetry and maintained at 97% or greater. The patient should be intubated in order to accomplish this, if necessary.
 - D. The patient should receive high flow oxygen by mask, which is usually enough if the patient is breathing.
 - E. Brain injury patients don't need oxygen.

4. Patients with brain injury should:
 - A. Never be given dextrose because it can make things worse.
 - B. Always be given 50 ml of D50.
 - C. Be placed on Dextrose 10% and water infusion.
 - D. Have their serum glucose checked and Dextrose administered as indicated.
 - E. Be administered IM Glucagon.

5. The Cushing Response (or reflex) is:
 - A. An increase in blood pressure and a decrease in pulse rate.
 - B. An increase in blood pressure and an increase in pulse rate.
 - C. A decrease in blood pressure and a decrease in pulse rate.
 - D. A decrease in blood pressure and an increase in pulse rate.
 - E. Vomiting in response to an increase in intra-cranial pressure.

6. A patient with a known or suspected brain insult who is confused, combative and choking on his/her own secretions should be:
 - A. Held down and suctioned aggressively.
 - B. Administered oxygen and told to calm down.
 - C. Provided anti-seizure therapy.
 - D. Chemically sedated, paralyzed, endotracheally intubated, suctioned and mechanically ventilated.
 - E. Both A and B.

7. Secondary brain injury is caused by:
 - A. Lack of adequate blood supply.
 - B. Lack of sufficient hemoglobin to transport adequate oxygen to the brain cells.
 - C. Hypoglycemia.
 - D. Toxins released from dead and dying brain cells.
 - E. All of the above.

8. All patients with complaints of severe headache (typically “the worst of my life”) and elevated blood pressure should:
 - A. Receive 10 mg of sublingual Nifedipine
 - B. Be evaluated for intracranial lesions.
 - C. Be transferred to a tertiary care hospital
 - D. Be referred to their primary care physician for evaluation of hypertension.

9. Patients found to have an intracranial lesion should:
 - A. Have their blood pressure monitored with the goal of maintaining appropriate ICP.
 - B. Receive emergent neurosurgical consult and/or transfer to a hospital with neurosurgical capabilities.
 - C. Be hyperventilated.
 - D. Have their blood glucose checked and treated if low (greater than or less than 70 mgm/dL in the adult, greater than or less than 60 mgm/dL in the child).
 - E. All of the above.

