QUIZ # 54 FLUID MANAGEMENT IN THE NEUROSURGICAL PATIENT

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I. 55 YEAR OLD PATIENT WITH A HISTORY OF HYPERTENSION PRESENTS FOR CRANIOTOMY AND RIGHT FRONTAL TUMOR RESECTION. AFTER RUNNING HER BIKE INTO A TREE, SHE HAS A HEADACHE AND NO DEFICITS. A HEAD CT WAS SIGNIFICANT FOR A 5CM WELL-CIRCUMSCRIBED TUMOR WITH SURROUNDING CEREBRAL EDEMA. NO OTHER INJURIES WERE NOTED. HER BP (130/70) IS WELL-CONTROLLED ON HCTZ. HER IV HAS BEEN HEP-LOCKED FOR THE LAST 48 HOURS AND SHE HAS NOT BEEN VERY HUNGRY. ALL OF THE FOLLOWING ARE TRUE REGARDING FLUID MANAGEMENT IN THIS PATIENT EXCEPT:

A. 0.9% Normal Saline is a better choice of crystalloid than 0.45% NS during this patient’s surgery
B. Intra-operative fluid restriction is in order as dehydration is good for this patient’s brain.
C. Goal directed fluid therapy may contribute to decreased postoperative complications
D. Hydrochlorothiazide (HCTZ) administered the morning of surgery should not prevent surgery from proceeding.
A. 0.9% NORMAL SALINE IS A BETTER CHOICE OF CRYSTALLOID THAN 0.45% NS DURING THIS PATIENT’S SURGERY

This is true. Administration of a hypo-osmolar solution like 0.45% NS (154 mOsmol/L) relative to plasma (295 mOsmol/L) will promote water movement into the brain exacerbating cerebral edema in this patient. 0.9% normal Saline is considered iso-osmolar with an osmolarity of 308 mOsmol/L (slightly hyperosmolar) and therefore administration of this fluid will be helpful in avoiding increases in brain water content and increased ICP.

B. INTRA-OPERATIVE FLUID RESTRICTION IS IN ORDER AS DEHYDRATION IS GOOD FOR THE BRAIN.

This is not true. The volume goal in neurosurgical patients is euvoemia. While excess fluid administration intraoperatively can exacerbate undesirable cerebral edema in this patient, fluid restriction in this patient whose only complaint is a headache; is on chronic diuretics; whose intake has been poor; and who has received no IVFs fluids for 48 hours can contribute to exaggerated intraoperative hypotension and decreased cerebral perfusion. Adequate IV access, intravenous fluid administration and prompt availability of vasopressors during induction is warranted to avoid hypotension.

C. GOAL DIRECTED FLUID THERAPY MAY CONTRIBUTE TO DECREASED POSTOPERATIVE COMPLICATIONS

This is true. Goal-directed fluid therapy relies on dynamic parameters like stroke volume variation (SVV) in order to guide intraoperative fluid administration. This strategy has been found useful in improving patient outcomes after abdominal, vascular and ortho procedures. In a study by Luo et al, Goal-directed fluid restriction (3ml/kg/hr) in neurosurgical patients (elective craniotomy for brain tumor resection, brain abscess, or intracranial aneurysm) combined with colloid boluses in the case of documented hypovolemia (SVV > 15% and CI < 2.5l/min/m² and MAP < 65 mmHg) lead to less colloid and crystalloid administration and decreased postoperative complications.

This is true. While HCTZ, a thiazide diuretic used for the treatment of hypertension will undoubtedly contribute to volume contraction in this patient, the risk of hypotension during anesthetic administration in this patient should not preclude surgery provided that the patients vital signs are adequate, and laboratory values, particularly K⁺ is within normal limits. Good IV access, IVF administration, readily available vasopressors and close attention to BP, during anesthetic induction and maintenance can help safeguard this patient perioperatively.

2. CHANGES IN INTRAVASCULAR VOLUME IN NEUROSURGICAL PATIENTS CAN BE CAUSED BY ALL OF THE FOLLOWING EXCEPT:

A. Hemorrhage
B. Osmotic Diuretics
C. Diabetes Insipidus
D. Volatile anesthetics
A. HEMORRHAGE

This is true. Hemorrhage is typically encountered in the neurosurgical head trauma patient who also has major multiple trauma causing blood loss leading to hemodynamic instability, decreased tissue perfusion, cellular hypoxia, and death. While healthy patients can often tolerate Hct 20-25%, in vitro studies demonstrate that oxygen delivery is maximal at Hct 30%. Fresh whole blood is preferable until bleeding is controlled. If not available then PRBC and coagulation factors and platelets. After blood for hemorrhage, crystalloid (preferably saline due to slightly increased osmolarity, see table) is probably the first choice for resuscitation of the head trauma patient. Concerns regarding colloid administration are shown in the table.

Rusa, R, Zornow M, Fluid management during craniotomy (Ch.9) in Cottrell and Young’s Neuroanesthesia, 2019

<table>
<thead>
<tr>
<th>Table</th>
<th>Solution</th>
<th>Osmolarity (mOsm/L)</th>
<th>Caution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crystalloids</td>
<td>0.9 % Normal Saline</td>
<td>308</td>
<td>Hyperchloremic acidosis</td>
</tr>
<tr>
<td></td>
<td>Lactate Ringers</td>
<td>&gt;273</td>
<td></td>
</tr>
<tr>
<td>Colloids</td>
<td>Hespan (6%)</td>
<td>310</td>
<td>Prolong PT/PTT</td>
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<tr>
<td></td>
<td>Dextran 40/70</td>
<td>300</td>
<td>Prolong PT/PTT</td>
</tr>
<tr>
<td></td>
<td>Albumin 5%/25%</td>
<td>290</td>
<td>Expensive ? Benefit and possible harm in the trauma patient with TBI</td>
</tr>
</tbody>
</table>
B. OSMOTIC DIURETICS

This is true. Mannitol (1098 mOs/L) is the most frequently used osmotic diuretic. This drug increases plasma osmolarity thereby drawing fluid from the intracellular to the extracellular space. In the case of the brain, drawing water out of the brain helps to decrease ICP and improve cerebral perfusion. The increase in plasma osmolality however may result in an acute expansion of the intravascular fluid volume, which could be very detrimental in the patient with poor myocardial function.

C. DIABETES INSIPIDUS

This is true. In neurosurgical patients, The syndrome of DI occurs typically in patients with lesions in the vicinity of the hypothalamus, after pituitary surgery or traumatic brain injury. Inadequate release of vasopressin from the supraoptic nucleus of the hypothalamus will lead to the loss of a large volume of dilute urine. High serum osmolality and high Na⁺ together with low urine specific gravity confirm the diagnosis. If untreated, this disorder may result in hypovolemia and hypotension. Treatment is with 0.45% normal saline and vasopressin.

Rusa, R, Zornow M, Fluid management during craniotomy (Ch.9) in Cottrell and Young’s Neuroanesthesia, 2019.
This is not true. Volatile anesthetics are potent vasodilators and while they contribute to decreased cardiac filling pressures they do not cause changes in intravascular volume.

Rusa, R, Zornow M, Fluid management during craniotomy (Ch.9) in Cottrell and Young’s Neuroanesthesia, 2019.
A. Osmolality of a solution is determined solely by the # of particles in solution
B. Equimolar concentrations of glucose, urea, and mannitol each exert the same effect on osmolality
C. An effective means of decreasing brain bulk is osmotherapy
D. Iso-osmolar crystalloid administration causes increased ICP
A. OSMOLALITY OF A SOLUTION IS DETERMINED SOLELY BY THE # OF PARTICLES IN SOLUTION

This is true. Osmolality is one of the physical determinants of water movement between the intravascular space and the central nervous system, because of the osmotic pressure that is generated within solutions of unequal osmolarity which are separated by a membrane permeable to water but not to solutes. When determining osmolality of a solution, the weight of the solute is not important solely the number of particles.

Rusa, R, Zornow M, Fluid management during craniotomy (Ch.9) in Cottrell and Young’s Neuroanesthesia, 2019.
This is true. Because it is the number of particles which determines the osmolality of a solution, equimolar concentrations of these molecules despite their different molecular weights: Glucose 180 g/mol; urea 60 g/mol; mannitol 182 g/mol they have the same effect on osmolality.

Rusa, R, Zornow M, Fluid management during craniotomy (Ch.9) in Cottrell and Young’s Neuroanesthesia, 2019.
This is true. Water has a tendency to move from the solution of lower osmolality across a member and into the solution of higher osmolality. This process continues until the solutions are of equal osmolality or the hydrostatic pressure is sufficient to preclude any further net flow of water.

With the goal of reducing brain tissue water in order to preserve perfusion, continued administration of hyperosmolar solutions like mannitol to the neurosurgical patients helps to produce more favorable osmolar gradients helpful for pulling water out of the brain. Mannitol begins to exert its effect in 10-15 minutes and lasts about 2 hours.

Rusa, R, Zornow M, Fluid management during craniotomy (Ch.9) in Cottrell and Young’s Neuroanesthesia, 2019.

This is not true. Fluid moves in and out of CNS according to the osmolar gradient. If no gradient is generated as with iso-osmolar crystalloid, there is no fluid movement, therefore no increase in brain water causing increased ICP.

Rusa, R, Zornow M, Fluid management during craniotomy (Ch.9) in Cottrell and Young’s Neuroanesthesia, 2019.
A. **Insist that the patient remain prone for the surgery as this position eliminates the risk of hemodynamic compromise to the patient**

B. **Tell the surgeon to wait until you adequately volume preload your patient**

C. **A couple of bolus doses of 100 mcg of IV phenylephrine should be adequate while you help the surgeon position the patient.**

D. **Increase volatile anesthetic concentration so that the patient does not move**
A. INSIST THAT THE PATIENT REMAIN PRONE FOR THE SURGERY AS THIS POSITION ELIMINATES THE RISK OF HEMODYNAMIC COMPROMISE TO THE PATIENT

Good communication with the surgical team should occur prior to induction of general anesthesia. Hypotension increasing the risk of myocardial and cerebral ischemia can occur in either the sitting or prone position. VAE, another problem in neurosurgery can occur in either the sitting or prone position and may potentially cause lethal hemodynamic changes. Increased abdominal pressure in the prone position decreases venous return causing a decreased CO. In Spine surgery, increased abdominal pressure causes venous congestion contributing to increased blood loss.

In the sitting position, increases in systemic vascular resistance leading to decreased venous return and decreased CO leads to more profound and consequential hypotension. Additionally, VAE is significantly more common in the sitting position. These issues are worthwhile discussing with the surgeon preoperatively.

From the surgeon’s standpoint, access to the posterior fossa may be more readily achieved with the patient in the sitting position. This position also facilitates drainage of blood and CSF thereby decreasing ICP.

This patient does not have any intracardiac defects and does not appear to be cachexic or severely hypovolemic (relative contraindications to the sitting position). Nevertheless, efforts to avoiding hypotension during positioning and surgery is imperative together with some method of monitoring for VAE.

Smith D, Anesthetic management for Posterior Fossa Surgery, (ch 12) in Cottrell and Young’s Neuroanesthesia, 2019.
B. TELL THE SURGEON TO WAIT UNTIL YOU ADEQUATELY VOLUME PRELOAD YOUR PATIENT

This is true. Volume administration will be important as this patient is placed in the sitting position. Pooling of blood flow in the lower extremities exacerbated by inhalational anesthetic administration which cause vasodilation and PPV which decreases preload all contribute to hypotension. In a prospective study of 20 pts having neurosurgical procedures in the sitting position, crystalloid preloading 20ml/kg before positioning prevented hypotension. There is also evidence for support of colloid administration to help maintain stable hemodynamics in the sitting position.

Ranjith, et al. Effect of crystalloid preloading (20ml/kg) on hemodynamics in relation to postural changes in patients undergoing neurosurgical procedures in sitting position. Journal of NS in rural practice, 2018;9(1)

This is not true. A few doses of phenylephrine may not be sufficient to prevent hypotension during positioning in this patient. Maintaining adequate CPP in this patient is essential to prevent additional untoward postoperative neurological sequelae due to cerebral ischemia. This patient with chronic hypertension on lisinopril and B blockers may be volume contracted and demonstrate exaggerated changes with positioning in the sitting position. Be prepared with IVF and vasopressors (infusions) in order to assure stable hemodynamics in this patient. Ideally it is beneficial to have the patient positioned gradually by the surgeon while the anesthesiologist monitors the hemodynamics while administering IV fluid boluses and adjusts vasoactive agents as indicated.

Smith D, Anesthetic management for Posterior Fossa Surgery, (ch 12) in Cottrell and Young’s Neuroanesthesia, 2019.
This is not true. All of the volatile anesthetics used today cause vasodilation and myocardial depression. Increasing volatile anesthetic therefore administration will exacerbate hypotension. Intravenous anesthetics like Propofol for maintenance anesthesia may have less and effect on cardiac function than volatile anesthetics. Muscle relaxation is for safety and decreased anesthetic depth.

Smith D, Anesthetic management for Posterior Fossa Surgery, (ch 12) in Cottrell and Young’s Neuroanesthesia, 2019.
5. 55 YEAR OLD FEMALE WITH A HISTORY OF HYPERTENSION PRESENTS FOR CRANIOTOMY AND ANEURYSM CLIPPING. SHE PRESENTED 2 DAYS AFTER THE WORST HA OF HER LIFE. A HEAD CT REVEALED SAH (FISHER GRADE II) AND AN ANGIOGRAM DEMONSTRATED A 6 MM RIGHT MCA ANEURYSM. TWENTY-FOUR HOURS AFTER ANEURYSM CLIPPING THE PATIENT IS AWAKE AND EATING AND SHE IS ON NIMODIPINE. HOWEVER BY POD 2, SHE HAS BECOME PROGRESSIVELY MORE OBTUNDED. BLOOD PRESSURE IS 120/70, TCD DEMONSTRATES MCA VELOCITY >120M/S. THE NEXT STEP SHOULD INCLUDE WHICH OF THE FOLLOWING:

A. Institute triple H therapy immediately
B. Avoid fluid boluses
C. Begin hypertensive Therapy
D. Observation
This is not true. This patient who is a Fisher Grade II (thin SAH; <1mm Thick without clots) and 4 days out from her bleed is at increased risk of developing vasospasm. Her neurological exam has changed and a TCD demonstrating an increased blood flow velocity is consistent with cerebral vasospasm. Triple H therapy (hypervolemia, hypertension, and hemodilution) a former treatment approach to vasospasm from SAH has as fallen our of favor due to the cardiopulmonary complications from hypervolemia with little benefit in improving outcomes after DCI. Focus now is on hypertension and normal volume status.

Datar, Rabinstein. Postinterventional critical care management of aneurysmal subarachnoid hemorrhage. Current Opini in Criti Care, 2017;23(2):87-93
This is not true. Avoiding fluid boluses in this patient may place her at risk for hypovolemia and hypotension potentially exacerbating cerebral ischemia in the setting of vasospasm. Increasing blood pressure is recommended for this patient effected through vasopressors together with keeping the patient euvolemic which may require fluid boluses preferably with normal saline. Invasive hemodynamic monitoring and goal-directed fluid therapy based on dynamic monitoring has proven to be beneficial in these patients.

C. BEGIN HYPERTENSIVE THERAPY

This is true. Increasing the blood pressure is the mainstay of medical management for cerebral vasospasm with vasopressor drugs and fluids as needed. Options include: Phenylephrine or Norepinephrine or additional inotropes. The goal is to increase MAP 20-25% above baseline and continue with neurological evaluation. Consideration of endovascular intervention if the patient does not improve is also indicated.

D. OBSERVATION

This is not true. This patient is demonstrating a significant change in neurological status. Prompt management of this patient is key to preventing vasospasm from evolving into cerebral infarction. If there is no clinical improvement after hemodynamic augmentation, endovascular intervention with a catheter angiogram is the next step which should be considered within few hours of failed hemodynamic augmentation.