The Big No-No That You May Not Know for Transcranial Doppler Imaging (TCDI)

Transcranial Doppler imaging can be a challenging exam for the sonographer to perform on the stoke inpatient. Small rooms, lots of equipment, lots of "lines" and often patient bandages. Many of these factors we have no control over, and we have to adjust our scanning accordingly. But the one no-no you may not know is not to move the bed or the patient. It could be catastrophic and life threatening for the patient. Why? Let me explain….

Some background first:

Approximately 30,000 hemorrhagic strokes occur annual, with over 50% of those patients not making it to the hospital and dying within just minutes. If the patient does make it to the hospital, obtains the needed CTA/MRA for diagnosis and is able to tolerate the coiling of the aneurysm, they still have a 50-70% chance of intracranial vessel vasospasm[1] which carries with it a 15-20% risk of another stroke or even death. Though the patient may seem to be doing better after the coiling, it is not until day 3-14 where the manifestations of vasospasm may occur. That is why a baseline (day after coiling) is so important to obtain, as the effects (vasospasms) caused by blood extravasation have not yet occurred. This baseline will aide in later detection of the severity of vasospasm and treatment. Why vasospasms and why that late?

The vessels in our body work efficiently when the blood is flowing within their lumen, but when extravasation of blood occurs and comes contact with the outer layer of the vessel, the vessel responds by contracting. This response is called myogenic vasoconstriction. "*This response is proportional to the duration of blood exposure and contributes significantly to the development of delayed vasospasm/ischemia after cerebral hemorrhage."*[2] So what's the big deal about moving the bed?

It all has to do with PCO_2 (partial pressure carbon dioxide). Carbon dioxide (CO_2) is the waste product of normal cellular metabolism. Approximately 10% is found dissolved in blood. PCO_2 is the pressure exerted by CO_2 dissolved in blood. What regulates the amount of CO_2 in our blood? Respiration. The medulla oblongata of the brain senses CO_2 & signals respiration. This is the "drive to breathe". Too much PCO_2 is known as hypercapnia, which is a vasodilator. In simpler terms, imagine your soda/pop bottle that is loaded with carbon dioxide. If you increase the carbon dioxide by shaking the bottle what occurs? The soda/pop bottle expands and will shoot out the soda/pop when you open it. It is the same with your blood vessels. Too much PCO_2 dilates the vessel and will increase cerebral blood flow (CBF) 100-120%, which creates hyperperfusion. Not good for a patient with a hemorrhagic stroke and cerebral edema.

The opposite is true when there is a decreased PCO_2 in your blood, known as hypocapnia. Again, imagine a soda/pop bottle that has lost its fizz (not much CO_2). What occurs to that bottle? It is easily indented and if you open the bottle, little or no soda/pop would shoot out as you open it. So too within your vessels. Hypocapnia is a vasoconstrictor, and reduces the cerebral blood flow (CBF) by 40-60% causing hypoperfusion to the brain. It is a delicate balance of pressure between too much and too little PCO_2 for the stroke patient. A normal PCO_2 is known as normocapnia and ranges from 35-45 mmHg.

Maintaining a consistent CBF hydrostatic pressure is critical. Change of head position, or elevation of the patient's bed can change intracranial pressure (ICP) and change PCO_2, which can be critical to an ischemic brain injury. The optimal neutral head position in patients with cerebral edema is essential for avoiding jugular compression and impedance of venous outflow from the cranium, and for decreasing CBF hydrostatic pressure. So, unless you have the permission of the physician, moving the patient or bed is a no-no. That means you may have to get creative in scanning: optimally and efficiently would be to

have 2 sonographers doing the exam. One to do one side, and the other to do the other side. Backing the machine in the room (so the front of the machine goes in first so you would be near the head of the bed looking toward the foot of the bed) is usually the best way to get around having to move the patient's bed. Unfortunately, it also means you are scanning in an awkward position, but that is much better than risking the life of that patient.

Always obtain the PCO_2 for correct interpretation of TCDI. It does make a difference.

Remember as well, it is the mean velocity (MV) that you should be documenting, not the peak systolic velocity (PSV). The MV is less affected by systemic factors such as heart rate, blood pressure fluctuations, etc, than PSV is. But never make the diagnosis of vasospasms on the MFV alone, use ratios in conjunction. Ratios help correct for dynamic states such as anemia, proximal disease and CHF.

In the not so past history, physicians would wait until a patient would have neurological symptoms before changing the treatment/medication of the stroke patient. This often led to disastrous consequences, as it was already too late for the patient. Today we rely heavily on the TCDI. Observing changes in velocities, signaling vasospasms, precedes neurologic symptoms and treatment can occur earlier, saving many more lives.

FOOD FOR THOUGHT: Have you ever wondered why people with sleep apnea often complain of headaches or migraines? A normal PCO_2 is maintained by respiration. The lungs remove the CO_2. People with sleep apnea are decreasing their respiration, which increases their PCO_2, which in turn causes vasodilation and cerebral edema.

1. Kirsch et al; RadioGraphics 2013; 33:E1-E14

***2. Extravascular Blood Augments Myogenic Constriction of Cerebral Arterioles: Implications for Hemorrhage-Induced Vasospasm*. Wensheng Deng, MD. et al.; J Am Heart Assoc. 2018;7:e008623. DOI: 10.1161/JAHA.118.008623.**