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MANAGEMENT OF
Traumatic Brain Injury

DRIP 5: CUSHING'S REFLEX

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Instructor: Dr. Christopher G. Byers, DVM, DACVECC, DACVIM, CVJ

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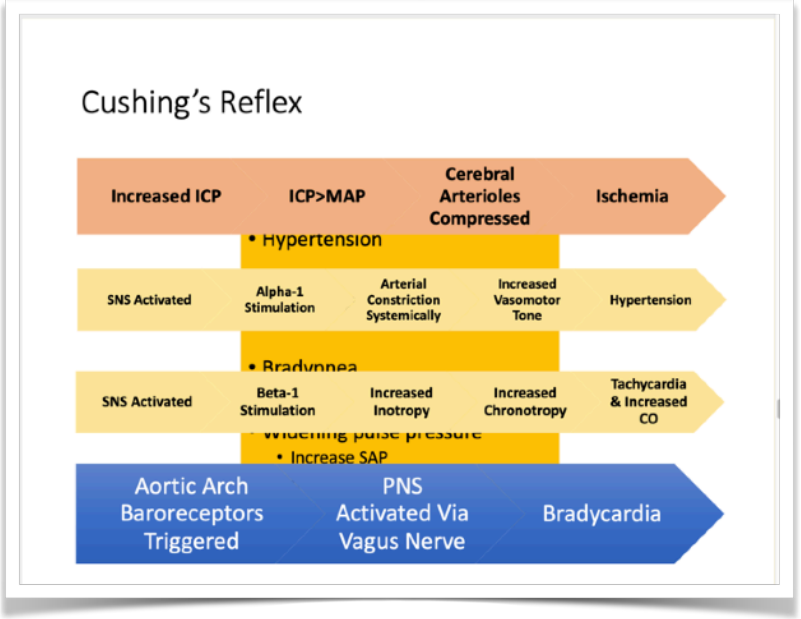
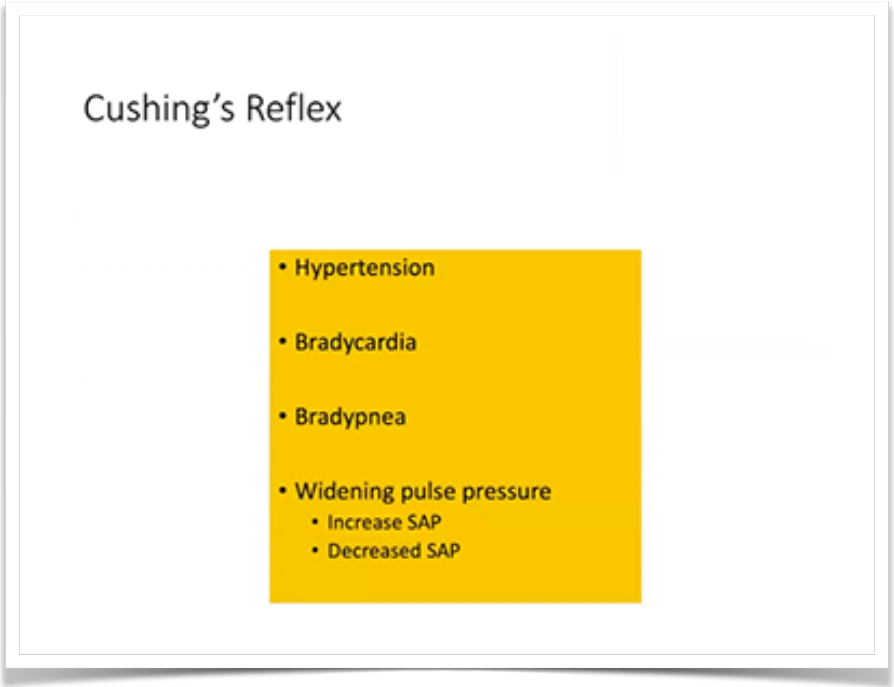


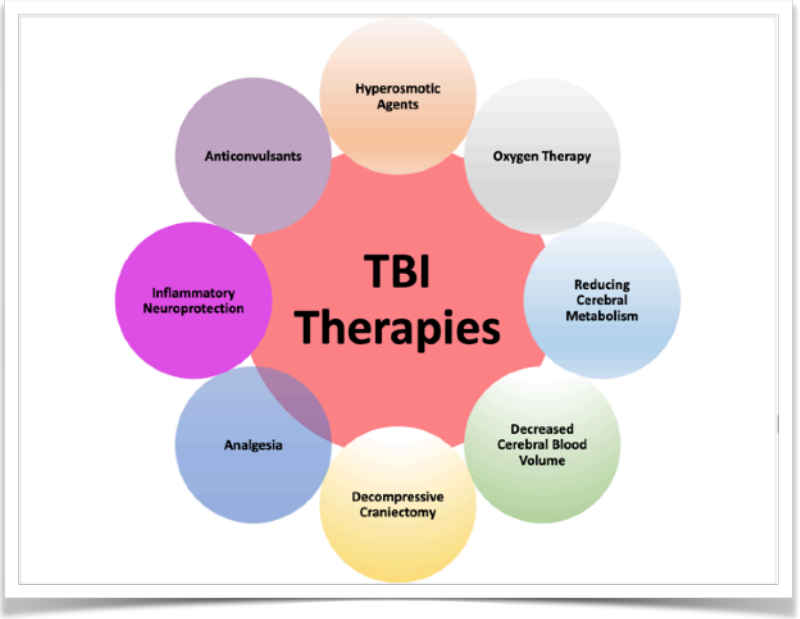
One thing that will get a emergency clinician a criticalist hopping is when a traumatic brain patient comes in and an assistant or nurse says blood pressure is 192 and the heart rate is 52 we're like oh no we don't like that that's hypertension and bradycardia that is the Cushing's reflex not Cushing's disease Dr. Cushing was a busy human being but this is the Cushing's reflex that is telling us that patient is trying to die through brain herniation so let's go through that important relevant pathophsyiology when intracranial pressure increases ICP you have a limit that you can go what you'd never want to have happen is for your intracranial pressure to get higher than your mean arterial pressure because when that happens the arterioles inside the brain get compressed and what happens when blood supply is cut off you get ischemia well this is a traumatic injury right so the fight or flight system has been triggered your central nervous system is

triggering happens you get stimulation of both alpha-1 and beta-1 adrenergic receptors when alpha-1's are triggered you get arterial constriction systemically and when the beta-1 receptors are triggered your heart does something it increases inotropy you get positive inotropic effects well when arterial vasoconstriction happens motor tone in those vessels goes up.

beta-1 stimulation also is a positive chronotrope you get increased inotropy increased chronotropy ultimately it means you get hypertension and initially some tachycardia but you know the body's a complicated organ and then tachycardia doesn't last very long because now the baroreceptors in the aortic arch get triggered everything's getting triggered and when that happens the parasympathetic nervous system is activated through the vagus nerve and that's where the bradycardia comes in so all as a result of this ischemia initially the CNS is like whoa what the heck then you get hypertension and tachycardia but the body doesn't like hypertension and tachycardia so it kicks in the parasympathetic nervous system which ultimately triggers bradycardia.

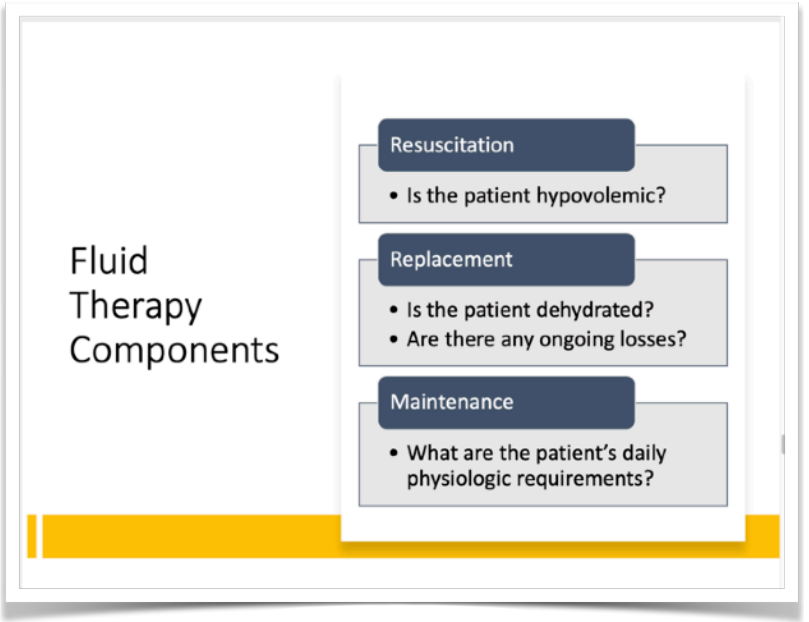


So if you see hypertension and bradycardia and people they'll talk about bradypnea and a widening pulse pressure because they do that type of monitoring for us a lot the patient's trying to die because their brain is trying to come up the back of their skull through the foramen magnum and you need to drop everything that you're doing and intervene.



So what are these interventions?

We have a lot of them and we're gonna spend a little bit of time going through these because they make a difference this is where you and I can positively impact whether or not our patient is going home or not.



First and foremost fluid therapy is essential we've talked about ischemia as a key component for triggering the Cushing's reflex we've talked about altered substrate delivery altered ATP delivery because _ cerebral blood flow has been compromised so we need fluid therapy and as we've talked about through the entire VetMed Live system when we talk about fluid therapy we ask ourselves three questions the first question is is my patient hypovolemic

If the answer is yes they need boluses you don't get to move on to question two or three until you are comfortable until you are satisfied that your patient is no longer hypovolemic when you reach that point then you ask yourself okay does my patient need any replacement therapy meaning is my patient dehydrated

Thirdly you ask what are my patient's daily requirements and not to sound like a broken record but those who know me know I love fluid therapy and we never say maintenance or twice maintenance I know what you mean when you're saying it but maintenance doesn't actually mean what you think it means maintenance is a type of fluid not a rate of fluid and we may actually want to use a maintenance type of fluid at some point in the traumatic brain injury patient but a maintenance rate which doesn't exist probably is not the safest approach so it's not just me standing on a little soap box and talking from an ivory tower there's a legitimate reason why we want to use proper terminology here and I know it's all ingrained in folk's heads and it's a habit but I'm going to challenge and I always will challenge you not to use the term maintenance.

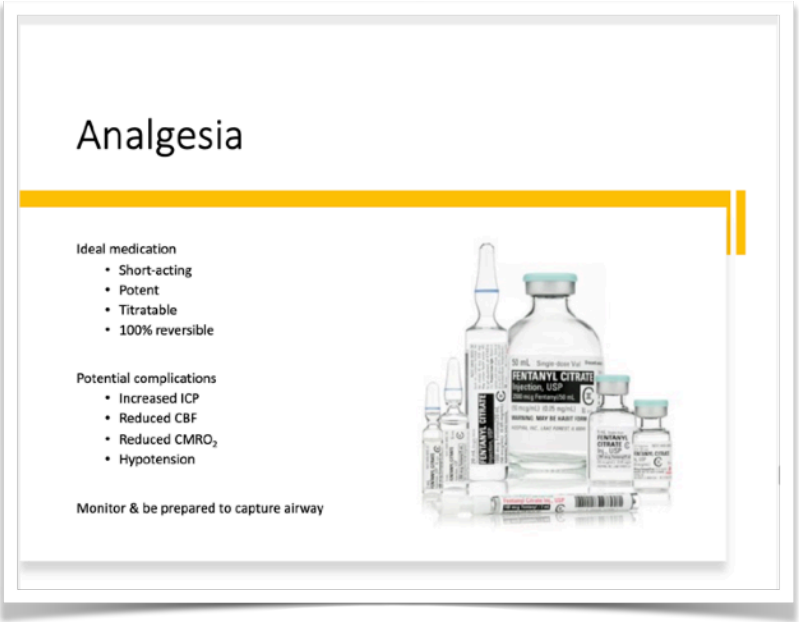
Image Credit: Central Texas Veterinary Specialty & Emergency Hospital

Image Credit: ABC News

Oxygen Therapy

- Cerebral hypoperfusion main precipitator of secondary TBI
- Target rapid restoration of adequate cardiopulmonary function
- Maintain oxygen carrying capacity
- Always provide supplemental O_2
  - Brain DO_2 may be inadequate with normal global perfusion parameters

Every patient with traumatic brain injury gets oxygen and I would say I would give supplemental oxygen for at least twelve hours and potentially longer even if they are not hypoxemic even certainly if they're not dyspneic and that's because while this area of the brain may be doing fine in terms of getting oxygen because of that altered cerebral blood flow maybe this area in this area and this area doesn't get enough cerebral blood flow and oxygen delivery perfusion to those brain segments is less than desired so even if they are not dyspneic even if they are not hypoxemic they get supplemental oxygen we don't know for how long I was always trained for at least half a day if not longer.



They’re trauma patients they're in pain they need pain medication but we all know that the most effective pain medication in an acute scenario is a narcotic there's no question about that but we also know that as effective as they are they have some potentially unfavorable side effects like causing hypotension like causing hypoventilation the last thing we want with hypoventilation the last thing we want is hypoventilation because that drives up your CO2 hypercapnea what happens to blood vessels with hypercapnea they dilate and that could increase the pressure inside the brain so we don't want to do that so we're usually reaching for a pure mu opioid initially and we like to use CRIs whenever possible but if you can't do a CRI because of the nature of your practice because you don't have the team to always be monitoring these guys I get it that's okay do your best but you deserve to know what is ideal and the ideal is a pure mu opioid given as a CRI

I always think it's important for folks to know the why I can sit up here and tell you what the best is but if you don't understand why I'm not doing my job as an educator.

The why is obviously these drugs are potent but when we use CRIs we tend to be able to use lower doses and we can titrate it so we start low we're not happy we can gradually ramp it up we don't like where we are because it's too much we can ramp it down those are potent they're titratable they're certainly efficacious we all know that but one of the major benefits is if we run into problems if our patient has an adverse drug reaction I can one hundred percent reverse a pure mu opioid with naloxone so a pure mu antagonist so that's why we prefer pure mu opioids either for intermittent injections if that's what you can do we have lots of options these days morphine fentanyl sufentanyl methadone I Love Me some methadone but I also love a fentanyl or a sufentanyl CRI and none of those are really expensive I'd say methadone is probably the most expensive one of those morphine is exceedingly financially friendly so you have some good options.