Central and Complex Sleep Apnea

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As mentioned in the section on *Obstructive Sleep Apnea (OSA)*, the word, "apnea" comes from the Greek word, "apnous" meaning breathless. We use the word apnea to mean "no breathing" or "no air flow". In OSA we can see the chest and the abdomen going up and down trying to breathe, but there is no airflow through the nose or mouth due to a blockage in the throat. In *Central Sleep Apnea (CSA)* the chest and abdomen are not moving because the "central nervous system" i.e.: the brain, is not telling you to breathe.

One cause of CSA is narcotic medication. Narcotics tend to suppress breathing and make breathing quite irregular. The irregular breathing in **Central Sleep Apnea Secondary to Narcotic Medication** is called "Biot's Breathing" or "ataxic breathing".

Primary Central Sleep Apnea is where the CSA does not seem to have an obvious cause. CSA can also co-exist in someone with OSA. Sometimes we see a patient in the lab who starts off with OSA. When we put them on treatment with *CPAP,* they develop CSA. We used to call this **Complex Sleep Apnea**. Now it is given a more descriptive but longer name called **Treatment Emergent Central Sleep Apnea**.

Normally, the trigger to take your next breath is a build up of carbon dioxide (CO2) in the blood. CO2 is the metabolic by-product of your body "burning" Oxygen and Glucose. A secondary trigger to breathe is a drop in blood oxygen. The body tolerates a slightly higher CO2 level when you sleep before it reaches the "breathing threshold" and triggers your next breath. Thus, as you transition from wake into sleep, there may be a slight pause in your breathing while your CO2 levels rise to that higher threshold. In conditions like Congestive Heart Failure (CHF) and Atrial Fibrillation (AFib) the output of the heart is reduced resulting in it taking a little longer for blood to circulate through the body and to the respiratory centers in the brain. This may throw off the delicate calibration of the feedback loop between your lungs and the brain. As a result, your CO2 levels go up too high and then down too low as the body overreacts. By the time the brain finds out you have not been breathing, it tells you to breathe too hard. Breathing more than normal lowers the CO2 levels below the breathing threshold. When the brain finds out your CO2 levels are too low, it tells you to stop breathing. This causes your CO2 levels to rise above normal. By the time the brain finds out you have not been breathing the brain tells the lungs to breathe hard again. This over-breathing and under-breathing is like loose steering on a car. The car is heading towards one side of the road and you make a small steering correction, but nothing happens because the steering is too loose. Then you make a big steering correction and now you are heading at the other side of the road. You make a small steering correction, and nothing happens, so you make a big steering correction and you are back heading to the other side of the road. This over and under correction is a bit like what is happening in the brain when the blood transit time is slowed a little due to CHF or AFib.

Another cause of CSA is from over breathing or “hyperventilation.” It is thought that in some people the CPAP used to treat OSA acts to “hyperventilate” the patient thus temporarily lowering their CO2 levels below the breathing threshold causing a central apnea. This is probably more likely to occur in people who are borderline “chronic hyperventilators.” Most people know what “acute hyperventilation” looks like. The person is taking big breaths and is complaining that they cannot get enough air. Often this is associated with an episode of *anxiety* or a *panic attack.* These people are getting more than normal amounts of air and oxygen but to them it feels like they are not. The majority of oxygen is transported in the blood attached to the hemoglobin protein in the red blood cell. The oxygen saturation levels in your blood hemoglobin are normally around 95%. When you hyperventilate the levels go up but cannot go higher than 100% which is not much of a change. However, CO2 in the blood acts like an acid. When you hyperventilate, this lowers the blood CO2 levels and the blood pH creating an alkalosis. Subtle changes in the blood pH can have significant effects on the physiology of the body. Acute hyperventilation can result in symptoms of light-headedness, numbness, tingling, muscle spasm (tetany), bloating, palpitations, shortness of breath, chest discomfort and dry mouth.

In “chronic hyperventilation” the person is not obviously over breathing although they may have some of the symptoms listed above. The most common symptom of chronic hyperventilation is frequent sighing. Sometimes you may feel like you have not taken a “satisfying breath”. The feeling makes you want to take a bigger breath, but you may have already taken the biggest breath possible. Chronic hyperventilators tend to have borderline low CO2 levels in their blood. When they go on CPAP, the additional hyperventilation lowers their CO2 levels below the breathing threshold resulting in a temporary apnea until the CO2 levels rise up again to trigger another breath.

People who tend to be more anxious may be more likely to experience chronic hyperventilation. However, most people who develop *treatment emergent CSA* are probably not anxious. Fatigue may also play a role. When you are tired from an underlying sleep disorder, you must push yourself more to get through your day. This results in living on more of the “fight or flight” hormones such as “adrenalin". This may increase your breathing a little bit causing some degree of chronic hyperventilation. This may predispose some people to treatment emergent CSA when they go on CPAP.

A particular kind of CSA is called “Cheyne-Stokes Breathing (CSB)”. This kind of breathing is commonly seen in people with stroke or CHF. In this condition, the breathing gradually gets shallower and shallower until it stops for a few seconds. Then the breaths gradually become bigger and bigger until they are much larger than normal. Then they gradually get smaller until they stop again. In some people with stroke or CHF you can see and hear their breathing waxing and waning even when they are awake. CSB is sometimes seen in the elderly even without a history of stroke or CHF perhaps due to undiagnosed cerebral deterioration affecting the respiratory centre.

Another type of CSA we see in the lab is **Periodic Breathing of Sleep Onset** or **Post Arousal CSA**. Breathing control tends to be a little unstable as you transition from wake to sleep. While you are waiting for your CO2 levels to rise up to the breathing threshold for sleep, a pause in breathing may occur that results in enough of a drop in oxygen to trigger an

arousal to breathe. Then the process to fall asleep starts all over again. This cycle may repeat a number of times until it resolves spontaneously when sleep stabilizes.

Diagnosis

CSA patients may have a history of pauses in breathing, but they are less likely to snore. They may have a history of CHF, AFib or stroke. They may be elderly or have a history of taking narcotic medication for pain or addiction management. The diagnosis requires a level 1, 2 or 3 sleep study to see the characteristic episodes of apnea associated without any significant chest or abdominal respiratory effort. Cheyne-Stokes breathing requires the presence of the characteristic waxing and waning of airflow. Biot's or ataxic breathing may be seen in patients taking narcotic medication. Except for CSB, the cycles in between central apneas tend to be less than 45 seconds. In CSB the cycles tend to be between 45 and 90 seconds. The EEG brain arousal is normally seen at the end of an obstructive or central apnea but tends to occur in the middle of the hyperventilation phase of CSB. Primary CSA has less than 5 obstructive apneas per hour. The diagnosis of both OSA and CSA can occur in the same patient. Treatment Emergent CSA starts as OSA on the diagnostic study but ends up with CSA during the CPAP trial.

Post arousal CSA occurs following an awakening. Most often this resolves spontaneously after a few events and sleep stabilizes fairly quickly. Sometimes it takes 10 to 20 minutes to resolve and can significantly disturb sleep.

As in OSA, the severity of the CSA is determined by the number of apneas and hypopneas (partial apneas) per hour of sleep. Five events per hour or less is considered within the normal range. Five to fifteen is considered mild sleep apnea. Fifteen to thirty is moderate, and thirty or more events per hour is considered severe sleep apnea.

Management

For **Central Sleep Apnea secondary to narcotic medication,** it is sometimes possible to have the patient wean off their narcotic medication and the CSA will usually resolve. Most of the time it is not possible for the patient to go off their narcotic medication. These patients need to come into the sleep lab to have a CPAP titration. If CSA persists after the OSA is controlled, then the patient is titrated on **BiPAP** (Bi-level Positive Airway Pressure). BiPAP has one pressure to breathe in and a lower pressure to breathe out. The higher inspiratory pressure assists the patient in taking a bigger breath. In CSA we add a backup rate to automatically ventilate the patient during the central apneas. This device is called **BiPAP S/T**. S/T stands for "Spontaneous/Timed" where spontaneous refers to patient initiated breaths and timed refers to the machine initiating a breath if you don't take a breath

spontaneously within a certain number of seconds. Modern machines with BiPAP S/T also have a setting called **AVAPS** (Average Volume Assured Pressure Support). This setting assures that the patient achieves a certain volume of air when the machine ventilates them. The machine will automatically increase the inspiratory pressure up to a predetermined level in order to achieve a prescribed tidal volume with each ventilated breath. This setting enables the patient to only get the higher pressures when they need them rather than with each breath. The AVAPS setting tends to make therapy a little more comfortable when first starting on BiPAP.

Periodic Breathing of Sleep Onset or **Post Arousal Central Sleep Apnea** is treated by deepening sleep to reduce the arousals that trigger this phenomenon. This is best achieved with *sleep hygiene* and other non-pharmacological measures to improve sleep. It can also be achieved with sedating medication. Zopiclone and Zolpidem are sleeping pills that have been shown not to make obstructive sleep apnea worse. However, if you are not on CPAP, other sedating medications may make OSA a little worse. For more persistent periodic breathing, BiPAP S/T may be considered.

Cheyne-Stokes Breathing, **Treatment Emergent Central Sleep Apnea** and **Primary Central Sleep Apnea** are usually best treated with **ASV** (Adaptive Servo Ventilation). BiPAP S/T may ventilate the patient during central events, but it also tends to hyperventilate them as well. This lowers the CO2 levels and perpetuates the cycling of the central apneas. ASV is designed not to hyperventilate the patient. It is a more sophisticated device that compares your apnea or hypopnea (partial apnea) to your normal breathing and gives you just enough pressure to take a normal sized or slightly smaller breath. If you do not breathe, it gives you 90-100% of your normal breath. If you take a 50% breath, it gives you a 40 – 50% supplement. If you take a 60% breath it gives you a 30 – 40% supplement. Thus, it does not hyperventilate you and perpetuate the CSA. What usually ends up happening is that the oscillations of over and under breathing gradually dampen down until you are breathing on your own. Then the machine just provides enough pressure to hold your airway open like a CPAP. Once you are breathing on your own, the arousals to breathe stop happening and you tend to sleep deeper than if you were being ventilated on just BiPAP S/T.

ASV can be used for CSA secondary to narcotic medication. However, the ASV works best when the pattern of CSA is fairly regular as seen in CSB, Treatment Emergent CSA or Primary CSA. It tends not to work as well in CSA secondary to narcotic medications because those apneas tend to be quite irregular.

Treatment Emergent CSA occurs in some patients treated with CPAP due to the hyperventilation effect that lowers their CO2 levels below the breathing threshold. In these patients, Provent may be an effective option because it causes a small amount of CO2 retention. This might prevent the emergence of CSA in these patients.