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Questions & Answers (Q & A) from:

Cerebral Palsy:
Management of Complex Hypertonicity

Presented by:

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Held 11 September 2025

Watch the recorded Clinical Webinar

Passcode: 1Vv+*V%m

Slides from the Clinical Webinar

Dr. Rose's written answers to the questions as written/asked during the live seminar:

Q. You say that CP is more motor. For us as general pediatricians, the greater concern is not motor but IQ and the mentality. What is your opinion concerning this?

A. While the diagnosis of CP is based on motor symptoms, comorbidities including impacts on cognitive function are very common and can be extremely functionally impactful for a patient. However, it is also important to remember that only approximately 50% of people with cerebral palsy have cognitive impairment, so this should certainly not be assumed about any patient with cerebral palsy.

Q. Regarding the other aspect - hypotonia. If the person has hypotonia, or hypertonia, will this give us indication about the etiology of CP?

A. The presence of hypertonia or hypotonia by itself does not give us a specific etiology as they can be seen in various genetic or acquired etiologies of CP.

Q. Is there any relation between or among the types of hypertonia and the brain damage severity? For example if you have a patient with only spaticity, or fragility, or dystonia, which one will be more worse than the others?

A. The severity of the brain injury doesn't necessarily dictate the types of movement or tone abnormalities that someone may have in the future. In fact, we are still trying to understand better how we can predict tone and movement abnormalities after brain injury. We do know

that certain types of brain injury may predispose you more to hypertonia. For instance, white matter injury is likely to lead to spasticity while injury to the deep grey matter predisposes you to the development of dystonia. However, we also know that you can get dystonia with isolated white matter injury or even cortical injury, so this is not the full picture.

Q. So usually when we are talking about spasticity, our thinking will go to the brain. Do you expect that a patient with a spinal cord lesion or tumor will present with spasticity also?

A. Yes, you can also have spasticity develop after a spinal cord injury as it is due to any injury to the upper motor neuron in either the brain or spinal cord.

Q. Regarding dystonia - its usually due to central nervous system disorder. May it also be due to myopathy?

A. The pathophysiology of dystonia is still being studied, but it seems to be due to a disorder of brain networks involving the nervous system. There is some involvement of afferent peripheral sensory nerves as well, as studies have shown modulation of dystonia with sensory input. However, it doesn't seem that dystonia is directly caused by muscle pathology.

Q. You said a person may have rigidity or spasticity. Will you expect that a patient that has rigidity will develop spasticity after that - and vice versa?

A. No, rigidity and spasticity are two separate things. Someone with rigidity has resistance throughout the entire range of motion, whereas spasticity has velocity dependent hypertonicity that is often unidirectional.

Q. So if you have a patient with dystonia during infancy, how can you differentiate between the physiological one and the pathological one? Because you say during infancy you may face some movement with slight dystonia?

A. As young infants are developing, their movements can appear dystonic. You will see this in young babies who are just learning how to reach out for objects and may appear to have some posturing and overflow movements to other body parts. However, this is part of their normal development and does not get in the way of their development. Something becomes pathologic dystonia when it impairs the development of the child.

Q. So what about dystonia vs. ataxia. For us, really, it can be quite confusing. If you have a patient with ataxia, or dystonia, or maybe both, what is the point that we should focus on?

A. Ataxia is a disorder of coordination whereas dystonia is a disorder impacting motor control. Someone with ataxia will have cerebellar signs on their examination, such as intention tremor, dysmetria, and a wide based ataxic gait. In contrast, someone with dystonia will have more posturing have an extremity when reaching, overflow movements to contralateral extremities and may experience a dystonic tremor, which is more jerky and may have a null point, which is not consistent with an intention tremor.

Q. Many (most?) of patients with CP will have epilepsy? Regarding anti-epileptic (medications) - can you correlate dystonia as a side-effect of the anti-epileptic (medication treatment)?

A. Not all patients with cerebral palsy will have epilepsy, although they are certainly at higher risk than the general population. There are not specific anti seizure medications that I would recommend in someone with dystonia, although it is important to be very careful to distinguish someone’s dystonia from their seizures. At times, dystonia can appear very much like a seizure and vice versa so it is important to get an EEG to distinguish between the two.

Typically, dystonia is not a side effect of an anti seizure medication, with the exception of vigabatrin, which at times can induce basal ganglia hyperintensities and rarely cause a movement disorder.

Q. You said most of the dystonia may be a part of the CP, or brain lesion, or it may be congenital. Is there any acquired dystonia that can appear after the age of 10 or 12, or are all of them due to other causes - congenital or signs/symptoms of another disorder?

A. Yes, you can have adult-onset dystonia as well. In adults, a lot of the acquired dystonia is focal or task specific dystonia, such as writer's cramp, laryngeal dystonia, or cervical dystonia.

Q. As you know many people today are asking about the role of nutrition in the treatment of (odd?) disease. Are there any foods that trigger dystonia or improve dystonia?

A. I'm not aware of specific correlations between particular foods and dystonia. You can see dystonia develop with nutritional deficiencies such as vitamin B12 deficiency or vitamin E deficiency, as well as with abnormal micronutrient levels such as iron or copper.

Q. You stated that one of the lines of treatment is the use of botulism toxin. In your practice, didi you experience any child develop botulism from use of this vaccine?

A. The bacteria that causes Botulism (Clostridium botulinum) is not present in botulinum toxin injections. Rather, the botulinum toxin injections contain the purified neurotoxin. If botulinum toxin is used incorrectly or spreads beyond the injection site, it could cause Botulism-like symptoms, but the person would not actually have Botulism.

Q. You state botulism toxin is for local (injection) use. How many joints can someone inject in one session?

A. This depends on how much toxin is being injected at each location. There is a maximum amount of toxin that can be administered in a given session depending on the weight of the child. For adults, there is a standard maximum cumulative dose of botulinum toxin that is recommended in a three month interval.

Q. So does a patient need a lot of physiotherapy for all of these hypertonic disorders?

A. Yes I would definitely recommend physical therapy as a key component of the therapeutic plan for any child with hypertonicity. Medications and bracing are also important components that are complementary to physical and occupational therapy.

Q. You stated one of the lines of treatment is giving cholonodine to the patient, and all of us know that this can eventually lead to hypotension. So how do we decide to give it - to which patients, which groups?

A. The doses of Clonidine that are used for treatment of dystonia are much smaller than those used to treat hypertension, so we typically don't encounter hypotension as a side effect of this. That being said, we do dose the medication carefully, taking into consideration the patient's weight, so that we do not overdose them and lead to hypotension.

Q. What is a minimum age at which we can use cholonodine?

A. There is no minimum age for clonidine usage. It is frequently used in the neonatal population for opioid withdrawal syndrome, so we have dosing available even for premature infants.

Q. You stated one of the causes is genetic dystonia? What are the criteria to make you think of possible genetic causes? And isn't the treatment totally different?

A. Yes we are discovering more and more causes of genetic dystonia. Traditionally, we always consider genetic dystonia when someone has evidence of dystonia clinically but has a normal brain MRI without evidence of brain injury. That being said, we are now finding some genetic causes that can contribute to dystonia even in patients with visible abnormalities on their brain MRI. Therefore, we are beginning to recommend genetic testing for all patients with dystonia. The treatment between genetic and acquired causes of dystonia does not necessarily have to differ; A lot of the treatments that we offer are the same. However, there are some genetic dystonias that we know respond especially well to a particular therapy, such as dopamine supplementation or deep brain stimulation. In those cases, the genetic results do guide our treatment recommendations.

Q. Best approach to treat status dystonicus?

A. In general, you want to treat the underlying trigger for the dystonia. While you are treating this trigger, you need to sedate the individual with IV sedation such as midazolam, propofol, or dexmedetomidine. While they are sedated, you can also adjust their enteral dystonia medications, so that when you withdraw the IV sedation, they don't immediately go back into status dystonicus.

Q. In cases of severe dystonia, is it possible for the abnormal involuntary contractions themselves or attempts by the patient to counteract these postures to cause bone fractures, joint dislocation, either through excessive muscle generated forces or accidental overcorrection? If so, how often is this encountered in clinical practice? and have you seen such cases?

A. Yes, this is unfortunately very possible, and I have seen this happen in my patients. For example, if the patient is experiencing an episode of severe tonic dystonia, their arm could get caught behind the arm of their wheelchair, causing a potential fracture. We try to control the dystonia as best as we can to decrease the risk of this happening, but it is always a possibility.

Q. Persistent Neonatal reflex has a role in diagnosis?

A. Generally, we do not use this in the diagnosis of hypertonicity. However, we do see a persistent upgoing toe, the Babinski reflex, in patients after upper motor neuron injury and this can be associated with spasticity.

Q. Some antispasticity medications, like oral baclofen or benzodiazepines, can cause sedation or respiratory depression. How do you balance effective spasticity management with minimizing respiratory risk in children with severe cerebral palsy?

A. This is a definite problem and consideration when we are coming up with a therapeutic plan. Many medications that we use for treating hypertonicity, both spasticity and dystonia, can cause sedation. We try to balance the need for pharmacologic treatment of the hypertonicity with sedation, often necessitating a careful titration of medications to find the best dose that will maximize therapeutic effect while avoiding excessive adverse effects.

Q. Any hope to reverse tone disorders in CP patients ? Any Gene therapy?

A. There is potential for gene therapy on the horizon for individuals with genetic causes of cerebral palsy, but at this point, there is not a known “cure” for acquired cerebral palsy secondary to brain injury. Even for those genetic causes, it is likely that a gene therapy would not fully reverse symptoms.

Q. In children who had hyperbilirubinemia in neonatal period and developed spastic

CP..which is best treatment?

A. Typically, hyperbilirubinemia causes dyskinetic cerebral palsy. I would recommend treating the dystonia and chorea with the treatment options that I highlighted- there isn't one treatment that is definitely better than the others.

Q. Proper management of constipation in CP as sometimes challengeable esp. if there is suspicious of surgical issues?

A. Yes, Constipation can be a real difficulty and can lead to significant exacerbations of hypertonia, especially dystonia. I try to manage Constipation as best as I can in my patients to stay ahead of it so it does not become a major problem.

Q. Role of gingo biloba as supportive medication for CP?

A. I do not use this as a supportive medication for cerebral palsy and I generally stay away from supplements as they are not as well studied or regulated as medications which are FDA approved.

Q. It's known that cerebral puasy is non-progressive disease; did you face any patient with progressive disease?

A. Yes, there are many patients with hypertonicity that do have a progressive disorder, which is not cerebral palsy. This is also part of the importance of doing genetic testing for patients, as we can identify those with genetic disorders that would lead to a progressive condition versus a static one.