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WHAT IS NEW IN PARKINSONISM, IT'S DIAGNOSIS, TREATMENT AND MANAGEMENT

Some interesting information from the MDS's meeting in Florida and Vancouver, the Vancouver meeting from June 4 to 8th, 2017

Naming the Parkinson clinical diseases is still not uniformly held. In part because each of the diseases have such varied clinical features and have much overlapping clinical signs and symptoms. Also the pathology has documented an overlap. Most are using Idiopathic Parkinson's Disease for what we know as IPD, yet realizing that on initial evaluation we may only be correct 50 to 75% of the time when compared to autopsy. Many, like myself, when uncertain as to the diagnosis, but thinking it is not exactly IPD, will use the term Atypical Parkinson's Disease or APD. Some others will use the term Akinetic-Rigid Syndrome for those patients that have just rigidity and akinesia. There are others who continue to use the older terminology called Parkinson Plus. I am sticking with ATP disease, but will use MSA, PSP, CBD (CBS), and FTD, when I get the more rapid progression with the corresponding signs and symptoms that may fit with each of these.
Please see my videos for the various features of these diseases.

Deep Brain Stimulation (DBS) has been shown in one large study, selecting younger patients, to be better somewhat than the best medical therapy at 5 years. Adverse side effects were intracerebral bleeding in about 0.5%, infection, and hardware difficulty. The key was appropriate patient selection.

Cell to cell transmission of the alpha synuclein in temporal and geographically somewhat consistent manner is thought to be the best explanation for the progression of the disease. It does appear that the alpha synuclein can be found in the olfactory nerves, the salivary glands, the skin, the GI tract and the vagus nerve. One study implanted alpha synuclein in the vagus nerve in animals and found it traveled into the brain.

There was not much on a topic that is getting a lot of press and that is the Microbiome, about the micro organisms in the gut. That is probably good news since we do not know what the normal microbiome is for most of us, not to mention when the human has some disease.

Ultrasound focused ablation in the thalamus, Gape, or STN is being used in limited centers and seems to be of benefit and is not invasive except for the destructive ultrasound in the target zone. Much more experience is needed as to: how long it takes to be clinically effective and how long is it effective, the adverse side effects over a long period of time, and who is the best candidate.

A new, longer lasting plasma half extended L-dopa (RYTARY 2??) has a longer plasma half-life (? 5 to 6 hours), seems to improve off time and will be going from clinical trial Level 2 to clinical trial level 3 in the future.
Many more genes are now being discovered for clinical enmities and we now find the genetic connections to diseases we could not group and did know the cause. However, the genetic marker does not always have the same exact clinical phenotype (same clinical features).

Myrbetriq (mirabegron) is a new medication for over active bladder and is used in patients with Parkinsonism and who are having symptoms of incontinence, frequency or urgency. Many anticholinergics have significant side effects such as dry mouth, confusion and even hallucinations. This drug in several studies has been shown to have fewer CNS side effects. A problem will be getting the insurance companies to pay for it. They probably will have to fail other drugs like oxybutynin first.

Several studies with following Parkinson patients and having autopsies have documented that Vascular Parkinson may be the second most common type of Parkinson pathology. It has been also found that 25 to 35% of autopsies on Parkinson patients have significant small vessel disease such as Leukoaraiosis and lacunar infarcts.

Use of marijuana has been of interest in treating some symptoms of PD especially tremor, fatigue, depression, rigidity and akinesia, however there has only been case reports and a few series of patients but no long term placebo controlled trials that I could find. In case reports there have been some improvements, however what is needed is long term, placebo controlled trials. Hence, there is insufficient evidence for its use and it is considered category U. What we do know is that marijuana with bring about apathy and loss of acquiring knowledge and that it is a gate way drug to addiction of more addicting drugs. In PD it is of concern because most experts believe that with PD there is a greater propensity to have and experience Impulsive Compulsive Behavior Disorders (ICD). There is a great deal of interest in the development of technology sensors to help document and understand off times and exercise. The Apple watch and others may be explored by the patient to find its benefit.

Protective factors for PD have been suggested and physical activity is consistently in a number of studies a protective factor as it is now shown to delay the progression of PD. Other protective factors are a lack of a family history for PD and caffeine intake. These protective factors may have a synergic effect.

Infusion trophic factor studies are being conducted in a few patients but the safety and value is uncertain.
The use of DATScan's are consistently showing about 90% sensitivity and specificity for deterring dopamine loss, however there is little benefit in diffracting IPD from APD however to benefit of the quantitative DATScan is uncertain.