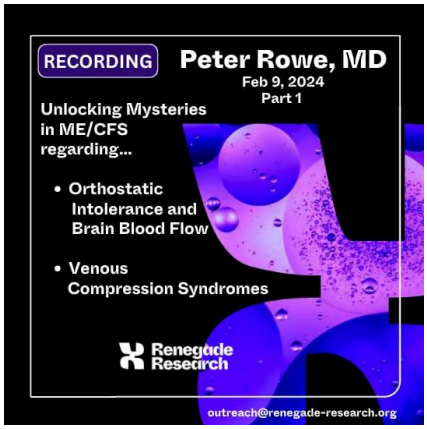
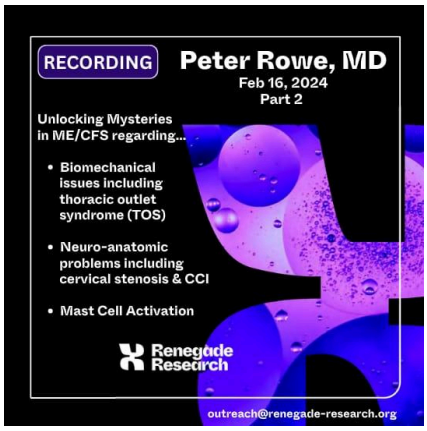
Extra Q&A with Peter Rowe, MD. May 2024

Not in either recording.
Inspired by events with Renegade Research / RemissionBiome:





Unlocking Mysteries in ME/CFS regarding...

Part 1

- **Orthostatic Intolerance and Brain Blood Flow**
- **Venous Compression Syndromes**
- **Recording** & **Transcript**

Part 2

- **Biomechanical issues including thoracic outlet syndrome**
- **Neuro-anatomic problems including cervical stenosis & CCI**
- **Mast Cell Activation**
- **Recording** & **Transcript**

If the information here helps you, and you can afford to, please consider donating to Renegade Research / RemissionBiome, or to Dr. Rowe's work via Rowe's Research Runners*, or both.*

Extra Q&A with Dr. Rowe

Q. Did the acrocyanosis disappear on treatment with ivabradine?

Dr. Rowe:
Acrocyanosis usually improves somewhat when treatments directed at orthostatic intolerance are successful, but it usually does not completely disappear.

Q. If the patient has high BP (blood pressure), how do you treat this if the patient has the symptoms you mentioned?

Dr. Rowe:
Patients with higher blood pressure and orthostatic intolerance are often treated with beta blockers or clonidine. Both beta blockers and clonidine are used as anti-hypertensive medications.

Another option is to use angiotensin-converting enzyme (ACE) inhibitor medications, which have been used extensively for the treatment of high blood pressure. A report by Zeng and colleagues (American Heart Journal 1998;136:852-8) showed that the ACE inhibitor enalapril was effective in preventing hypotension during tilt testing in those with recurrent neurally mediated syncope. In this study, the enalapril prevented the development of hypotension during tilt table testing, in part by reducing the release of catecholamines (adrenaline/epinephrine) which are known to provoke NMH.

Q. If OH or delayed OH (dOH) occur along side POTS symptoms, is it still called POTS?

Dr. Rowe:
If orthostatic hypotension occurs in the first three minutes of tilt or standing, that becomes the primary diagnosis. The Consensus definitions are less clear about whether the diagnosis of POTS should be made when the BP falls at a later point to the levels consistent with dOH. My tendency would be to list both if POTS occurs first and is followed by dOH.

Q. Hypoglycemia - Is there any relationship between me/cfs and hypoglycemia?

Dr. Rowe:
I am not aware of a clear association of hypoglycemia and ME/CFS. But, low blood sugar from reactive hypoglycemia can look a lot like orthostatic intolerance (OI), in part because they share a similar mechanism of causing symptoms, namely a rise in epinephrine levels.

For all of us, the physiological response to a prolonged period without food (which leads eventually to a drop in blood glucose levels), is to release epinephrine (adrenaline). Epinephrine, in turn, triggers a release of glucose from glycogen molecules in the liver, thereby raising blood sugar back to the normal range. But, this occurs at a price if the drop in sugar is rapid or there isn't a lot of stored glycogen: if epinephrine levels go high enough, they make anyone look pale and feel lightheaded and tremulous. As most people with the circulatory problems know well, those are also recognizable symptoms of orthostatic intolerance. In OI patients, epinephrine levels tend to be higher all the time, but especially so right before syncope. For those with OI, any physiologic stressor that causes a higher epinephrine level (stress, pain, exercise, standing, asthma medications) is capable of causing worse orthostatic intolerance, and a common, additional stressor includes a low blood sugar.

Thin people are more at risk for reactive hypoglycemia, especially (I think) if they also have OI. If they eat anything really sugary, the easily absorbed sugars lead to a big elevation in blood glucose. The body then responds with an equally big surge of insulin to bring the glucose into cells. In response to the big surge of insulin, blood sugar falls more precipitously, leading to a big counter-surge of adrenaline to bring blood sugar back into the normal range, but in the process making you feel sick. The key is to avoid the big surges in glucose, insulin and adrenaline, and to eat more carbohydrates that get absorbed more gradually (pastas and breads rather than confections, maple syrup, donuts).

Q. Thoughts on STAT device for home brain blood flow monitoring?

(many asked this)

Dr. Rowe:
We are following the research with interest, and the company has compared the STAT device to extra-cranial Doppler imaging of cerebral blood flow working with Drs. Van Campen and Visser in the Netherlands. I don't think the full answers are in yet though.

Q. Paralysis - Have you had patients experience paralysis?

Dr. Rowe: True paralysis, with loss of limb function, is not something we have seen in our clinic, with the exception of one patient following complex surgery.

Q. ADHD and POTS - Why does there appear a greater number with ADHD and POTS? (and also hypermobility)?

Dr. Rowe:
Research on this association is just emerging. This paper was one of the first to describe a link:

Joint Hypermobility Links Neurodivergence to Dysautonomia and Pain.
Csecs JLL, Iodice V, Rae CL, Brooke A, Simmons R, Quadt L, Savage GK, Dowell NG, Prowse F, Themelis K, Mathias CJ, Critchley HD and Eccles JA (2022)
Front. Psychiatry 12:786916.
doi: 10.3389/fpsyt.2021.786916

These authors write the following:

> As noted, in addition to these conditions, there is growing recognition that hypermobility is associated with the presence of one or more neurodevelopmental conditions, including autism and ADHD (17–19). Individuals with EDS are reported to be 7.4 times (95% CI: 5.2–10.7) more likely to be autistic than a comparison group (18). Autistic children were shown to have greater mobility of joints (maximum passive joint mobility in degrees of angle were measured for a finger, wrist, elbow, and ankle) compared to a matched comparison group (20). The association between autism and joint hypermobility syndrome/hEDS is further illustrated in a series of case studies (21), highlighting the need for more systematic research for robust characterization of these links (17). ADHD is also associated with GJH: One study reported generalized hypermobility in 32% of 54 patients with ADHD, compared to 14% of a comparison group (22). Another study reported the prevalence of GJH to be 74% in 86 children with ADHD, compared to 13% of a comparison group (23). Moreover, in a population-based matched cohort study in Sweden (n = 1,771), individuals with EDS were 5.6 times (95%CI: 4.2–7.4)more likely to have an ADHD diagnosis than those without EDS (18).

This study concludes:

> In neurodivergent adults, there is a strong link between the expression of joint hypermobility, dysautonomia, and pain, more so than in the comparison group. Moreover, joint hypermobility mediates the link between neurodivergence and symptoms of dysautonomia and pain. Increased awareness and understanding of this association may enhance the management of core symptoms and allied difficulties in neurodivergent people, including co-occurring physical symptoms, and guide service delivery in the future.

Q. Tinnitus. Is there a link between POTS and tinnitus?

Dr. Rowe: We have a number of patients with both diagnoses, and I have not done a search, but I am not aware of papers exploring whether the two disorders are diagnosed more commonly than tinnitus occurs in the general population.

Q. After OI is controlled, how do you treat residual symptoms such as fatigue?

Dr. Rowe:
We look into a variety of other co-morbid conditions as contributors to fatigue. An important one is the presence of restricted range of motion in the limbs and spine, often referred to as myofascial

restriction, or in some circles as adverse neural tension, or neural tension dysfunction. We are currently investigating how often thoracic outlet syndrome (which causes arm symptoms but also systemic fatigue with the arms overhead) is present in those with ME/CFS. It can be an independent contributor to fatigue.

Another is the presence of intolerance of specific foods, most commonly milk protein. Related to this, we look for evidence of mast cell activation syndrome.

More work is being done lately on different types of vascular compression problems as contributors to both orthostatic intolerance and other symptoms, including fatigue.

We also do a careful search and a careful neurological examination looking for signs of Chiari malformation, cervical spine stenosis, and cranio-cervical instability. Usually these patients have rather refractory orthostatic intolerance.

Certain supplements can be helpful in treating fatigue in specific patients, including intra-muscular or sub-cutaneous vitamin B12 injections, nicotinamide riboside (TruNiagen), curcumin, and others, like N-acetylcysteine.

Q. Lidocaine infusions

Q. In the Netherlands there is research being done into looking into Lidocaine infusions as a way of approaching the long covid theory of targeting and calming down the hyperreactivity of the immune system (for which I am being treated since 21 weeks and am noticing significant reductions in some symptoms such as fasciculations in the legs/brainfog). Is there any research that you (or Renagade Research) know of in the states that is also looking in this direction?

[Possibly related Intravenous lidocaine infusion in a case of severe COVID-19 infection
A novel role for lidocaine in COVID-19 patients?]

Dr. Rowe: I don't know much about using this medication for this purpose.

Q. Regarding rates of low blood pressure by different groups - do you know or have ideas if it is based on the patient pool specificity (for example, your clinic) may have a higher rate of severe ME/CFS population vs others?

Dr. Rowe:
The different rates reported in the past have had a lot to do with the methods used to ascertain for orthostatic intolerance. A lot depends on making sure the room is quiet (without for example an exercise test being performed in the next clinic space, with only a curtain between the tilt test and the exercise test, as one patient once told us), temperature controlled (overly cool environments would reduce the likelihood of an abnormal tilt), the duration of the test (shorter tests lasting 10

minute miss the vast majority of those with neurally mediated hypotension, as the time to an abnormal test in a median of 29 minutes among adults with ME/CFS), and whether the people supervising the test are talking to the patient (conversation should be minimized). The patient should be monitored to discourage movement. Some centers allow testing while the patient is on medications that can treat OI, which invalidates the test. If patients are tested after being encouraged to increase their salt and fluid intake (a partial treatment), this too would have the potential to lower the prevalence of OI.

Perhaps the most important issue in adults comes from the work of van Campen and Visser. Their 30-minute tilt test brought out heart rate and BP abnormalities in just 42% of adult patients. That number rose to 90% when they performed extracranial Doppler measurements of cerebral blood flow. So, without a measure of cerebral blood flow, we may be grossly under-estimating the proportion of patients who can be identified using HR and BP measures alone.

Other factors can include whether the patients actually have ME/CFS versus another fatiguing diagnosis. Also, the time of day matters. People are more likely to test positive for POTS if tested in the morning. We wrote about these methodological issues in the Institute of Medicine review in 2015 if you want to read more on methodological issues.

Q. Regarding Long covid vs MECFS - acute vs chronic -- Is the issue ongoing fibrosis building up? Senescent cells building up? Do we know?

Dr. Rowe: I do not think we know the answer to this.

Q. Any thoughts on how to scale treatment / access?

Dr. Rowe: I am not sure I know what you mean by this question. If you are asking how we get more people access to testing and treatment, this will involve a lot of continuing education of medical professionals about OI, and adoption of the passive standing test as an easy, cheap diagnostic maneuver that can be conducted by physicians without a lot of specialized equipment.

Links for speaker Peter Rowe, MD

Chronic Fatigue Syndrome (CFS) and Related Disorder Program at Johns Hopkins
Dr. Rowe's Research on Chronic Fatigue Syndrome and Related Disorders
Pre-order: Dr. Rowe's book" Living Well with Orthostatic Intolerance: A Guide to Diagnosis and Treatment"
General Information Brochure on Orthostatic Intolerance and its treatment | March 2014 (most recent)
Dr. Peter Rowe, Is The Physical Examination Normal in CFS? [No!]
- Part 1 (Orthostatic heart rate and blood pressure changes, Part 2 (Joint Hypermobility),
- Part 3 (Postural dysfunctions and movement restrictions)

Manual Therapy in CFS: Part 1 of 2
Manual Therapy in CFS: Part 2 of 2
An ME/CFS Expert Helps a Long-COVID Patient Recover | April 2023

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