As the wet, cold winter weather sets in, many people with CRPS notice a sharp increase in their pain and dysfunction.[1] Let's discuss why some of this happens and a few practical actions that may help mitigate it. This pain increase is primarily due to our vasomotor dysfunction, which as a result exacerbates nociceptive small fiber nerve signaling.

What's Going On

Cold makes blood vessels constrict. When the standard person is exposed to the cold, they experience a phenomenon known as the Hunting Reaction,[2,3,4] where they first experience temporary vasoconstriction for 5-10 minutes followed by a short period of vasodilation so fresh blood can flood the area before the vessels constrict again. This process repeats itself while the person is exposed to the cold to protect themselves from heat loss while ensuring their tissues remain oxygenated, balancing body temperature homeostasis, nutrient delivery, energy metabolism waste removal, and conservation of resources during inclement conditions.

In CRPS, our dysfunctional vessels get tighter with less provocation and stay that way longer than standard. In at least a large subset of patients, a contributing factor to this is a supersensitivity to the neurotransmitter noradrenaline, which—among other functions—tells blood vessels to constrict; this is because of either an upregulation of adrenoceptors (generally early / hot stage) or an autoimmune response damaging adrenergic receptors (generally chronic / cold stage), leading to an over-responsiveness to circulating noradrenaline.[5,6,7,8,9]

The resulting constriction decreases blood circulation in something called an ischemia-reperfusion injury (IRI), which is a core component of CRPS[10,11]—think of it like an oxygen starvation-corrosion cycle, which damages tissues both during and after the period of insufficient oxygenation. When you notice skin discoloration like blue, purple, gray, pale, blush pink, dark red, or mottling, particularly if associated with skin temperature change (as blood is blocked from or floods to an area), this is often an indication of an IRI cycle in CRPS-affected areas. IRIs are damaging to nerve, muscle, and bone tissue, creating a state of inflammation and activating pain neurons. As a result, the cold may increase sensations of deep aching, radiating, sharp, burning, throbbing, slicing, clamping or vice-gripping pains, as well as numbness, pins-and-needles, or other unpleasant perceptions.

As blood rushes back into the area after a period of impaired circulation, some blood plasma may fall out of gaps in the vein walls, filling the space in between tissue cells outside the circulatory system; this adds additional pressure to the small capillary beds from the outside, and the more pressure there is, the harder it is for them to reopen. However, capillary beds don't need much external pressure to be forced closed and prevent blood from making it to the tissues serviced by those beds; the more pressure there is, the more vessels and capillary beds are forced shut. This swelling may be quite noticeable or less noticeable, depending on the person, though generally it is more noticeable earlier in the condition and becomes less noticeable as the case becomes more chronic. The plasma leakage and

associated swelling is thought to start in deeper tissues and move towards the surface, and the accumulating fluid sets off pain neurons and causes allodynia and hyperalgesia.

The larger, fatty-sheathed nerves that are generally signaling properly also start having difficulty transmitting signals around 63F / 17C, getting worse as it gets colder.[12] The thinner, less- or non-fatty-sheathed nerves that are dysfunctional are less affected by the cold. Small C-fibers are our most dysfunctional nerves in CRPS and are responsible for the slow, deep, burning, radiating, diffuse pain,[13,14] as well as for controlling surface-level vasomotor dilation responses[15,16] in the case of neurogenic inflammation / the reperfusion part of the IRI cycle; these C-fibers are unmyelinated and will be some of the last nerves to cease sending signals due to cold-related transmission complications, and part of their sensory job is responding to thermal information, particularly extreme heat (around or above 105F / 40C)[17] or cold (around or under 60F / 15C).[18,19] In this case, as the larger fibers become less active, C-fibers become more active in response to the cold thermal information,[20] meaning there is considerably less "proper" signaling to contradict the dysfunctional sensory information our brains are getting from the small-fiber nerves.[21]

Because not enough fresh oxygen-rich blood can distribute to cells in tissues during ischemia in IRIs, we rely on non-oxygen-based energy production in those areas, which is about 15x less efficient,[22] burning through our glucose stores and creating a lot of lactate as a by-product,[23] which often gets trapped with sympathetic neurotransmitters and inflammatory chemicals in myofascial tissue, creating muscle knots,[24] which can increase muscular pain in addition to the neurogenic pain and vascular pain from the other aspects of the condition.

Additionally, as barometric pressures drop with winter storms, muscles and sinews, particularly around joints, can expand, nerves can become more easily irritated, and synovial fluid in joints can be thicker and less lubricating.[25]

Actionable Steps

The cold can make things extra difficult with CRPS, but if there are actions you can take to help prevent and break IRI cycles, then that will help you in both the short-term and the long-term. Here are some practical, inexpensive or free steps that may be useful in increasing quality of life, particularly during low temperatures.

-While cold constricts, heat dilates vessels. Moist heat penetrates more deeply into tissue than dry heat.[26,27]

-Dress warmly to prevent the loss of any body heat you do generate, even if your sensation of cold itself is dysfunctional; pay particular attention to insulating feet, hands, and face, as they have a lot of small vessels close to the surface where blood warmth can be lost,[28] as well as many nerves fed by those vessels that can be damaged if they do not receive proper circulation.[29]

-Consider a battery-pack-powered heated vest to help you keep circulating warm core blood while outside, like wearing a hot pad around.

-If you get right into a hot shower, especially while your limbs are discolored / cold, this can trigger a rapid vessel size switch and reperfusion via flushing blood and chains of electron stealing, which can be quite painful, like forcing a fully dead / numb limb to “wake up” very quickly. If that’s sounding like your situation, either try getting in with more moderate water, gradually turning it up to where you want it to let your vessels make the temperature shift in stages, or try to get out of the ischemia a bit more before getting into the water, so that the temperature shock isn’t as abrupt.

-If you’re swollen, the pelting shower water may also be aggravating allodynia, so trying to reduce fluid in the interstitial space or trying a bath instead could help with that.

-Release the trapped chemical messengers in muscle knots / trigger points through an array of options like manual myofascial release, counterstrain, dry needling, deep tissue massage, or “cold” low level laser light therapy to improve circulation, increase range of motion, and reduce pain;[30] these chemicals will then be in the lymph fluid in the space between tissue cells, which is the same place where the blood plasma leaked and is applying pressure to vessels from the outside.

-Lymphatic massage or lymphatic drainage helps increase lymph circulation, particularly in areas of stagnation or infectionless swelling; this can help reduce the external pressure on blood vessels, increasing their ability to circulate better, and on nerves, reducing their spontaneous firing and allodynia and hyperalgesia intensity.[31,32]

-Anti-oxidant-rich foods and other antioxidant treatments help counteract the free radicals / reactive oxygen species which damage cells during reperfusion in IRIs.[33,34,35]

-In addition to rapidly accelerating nerve signals, myelin sheathing acts as an oxygen-buffer for deprived nerves, protecting them from the effects of ischemia for a time; however, repeated oxygen-deprivation can damage both the nerves and the myelin sheath.[36,37] Foods high in omega-3 fatty acids, the amino acid choline, and vitamins B and D can help with nerve and myelin repair.[38]

-Gentle movements (whether that be something more robust like aerobic exercise, PT, walking, yoga or something more laid back like stretching in bed or wiggling feet and toes or deliberately flexing all the fingers in a hand, whatever you can manage) to prevent the body from locking up and keep blood circulating can help prevent IRIs that start due to disuse, as the body—in an attempt to conserve its resources—sends less blood to areas that are not being used and constricts blood vessels in response to sedentary behavior and lack of proprioceptive sensory input.

I hope this explanation shed some light on what’s happening internally during cold weather in CRPS, that some of these options assist you, and you are able to find additional solutions that

help mitigate the risks; winter is a tough season and can be particularly dangerous for those with mobility difficulties, particularly during periods of ice or snow. CRPS requires determination and often a fair amount of creativity to adapt; in my view, knowing why certain things are happening and at least one way to counteract it can be helpful.

Thanks for sticking with me. I hope you learned something, and I hope to see you next time.

contender

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