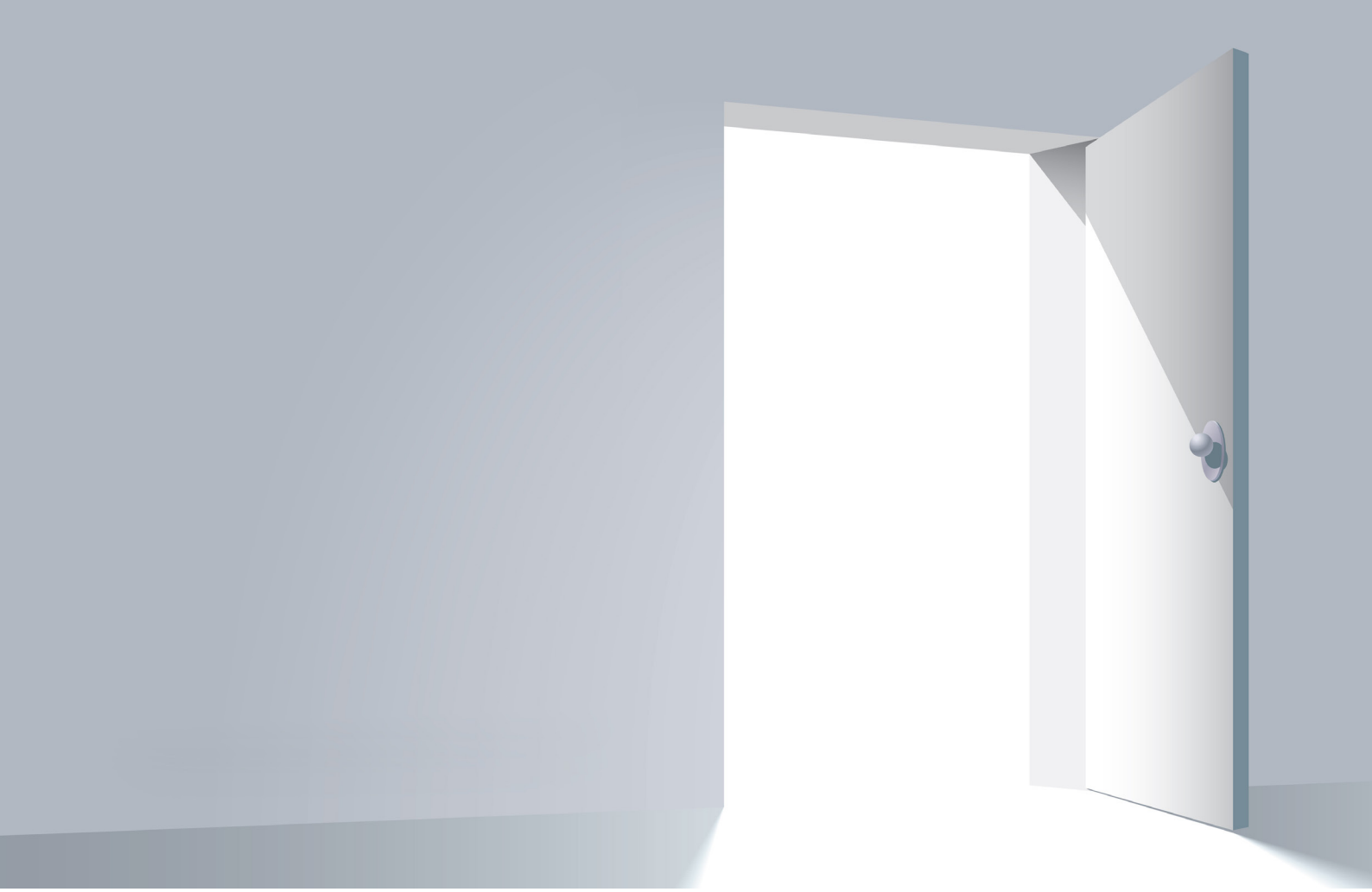

Peripheral Sensitivity

COMPLEX BIT

We could define peripheral sensitivity as an enhanced sensitivity to local stimulus, meaning that previously innocuous stimulus may now be sensed as noxious, this occurs through a decrease in the activation threshold of the nocicpetors.

It is difficult to separate what maybe peripheral or central sensitization and they may also co-exist. There are a number of changes that can happen to peripheral afferent neurons.

Previously silent or refractory noccieptors may now also become active increasing the number of functioning receptors able to generate an action potential.

We may also get an increase in sodium ion channels in the cell. These are manufactured within the cell body in the dorsal root ganglion (DRG) and antidromically transported to the nociceptor terminal endings to increase the amount of sodium able to get into the cell, causing depolarization and generating an action potential and an ascending afferent signal.

Increased nociceptive activation can also generate neurgenically mediated inflammation that further chemically sensitizes nociceptors through peptidergic chemicals such as substance p and calcitionine gene related peptide (CGRP)

Peripheral Sensitivity

SIMPLE SIDE

Simply put, a stimulus that was once not enough to cause pain or discomfort can become so when the peripheral sensors are more active. After injury or even hard exercise, such as delayed muscle soreness, we often become more sensitive afterwards with even light touch being felt more strongly than before.

This may become the 'default setting' however leading to an increased state of sensitivity driven by peripheral receptors firing far more easily and to much lower stimulus's. This could mean that even small movements cause mechanically activated receptors, which previously had a much higher threshold, to fire sending a signal.

Think people who 'flare up' at even the smallest amount of exercise or a specific movement or cannot even bear to be touched in certain areas of their bodies.

Simple story

The body is great at adapting unfortunately this is not always positive. The nerve endings in tissue can get better at sensing danger the more they are activated. They work simply through the transport of chemicals from outside the cell to inside the cell. Different things do this ranging from heat, movement and chemicals such as we get when we have inflammation.

One of the ways they do this is to put more open doors in the cell (ion channels) to enable it to get more of the stuff from outside to inside and send a signal.

In the end even a little bit of the stimulus can make it send a signal meaning you have more danger signals to deal with. It's not a reflection of the danger but the alarm becoming far to sensitive.

Imagine a car alarm that goes off when you get a little gust of wind. The wind is not dangerous instead the alarm is too sensitive. The danger system is not giving an accurate assessment of danger anymore.

Reading List

Hyperalgesia and allodynia: peripheral mechanisms.
Coutaux. Joint Bone Spine. 2005

Peripheral mechanisms of hyperalagesia.
Nagoya j med sci 1997