CAROL SAMUEL, PHD, FFHT, EXAMINES THE LINK BETWEEN STRESS AND PAIN AND LOOKS AT HOW REFLEXOLOGY CAN HELP

STRESS reflex

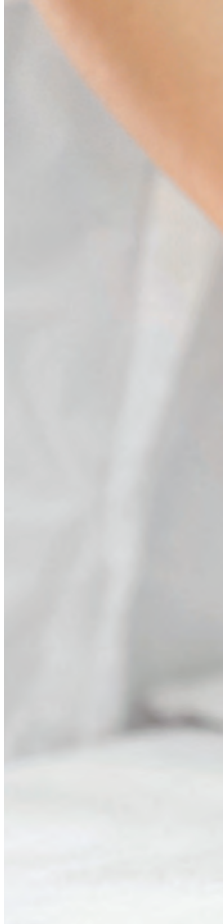
We live in a fast-paced, fast-track society where we are available 24 hours a day, 365 days a year. It's no wonder we find life increasingly stressful; but when stress becomes painful, pain can become stressful.

STRESS RESPONSE

There are two types of stress: eustress, which in the short term is positive, helps focus our energy on the job in hand and often comes with a feeling of excitement that helps improve our performance (Jackson, 2014); and distress – a tendency to cause anxiety and concern. Distress feels unpleasant and, when prolonged, decreases rather than enhances performance and leads to both mental and physical ill health.

Stress is not an illness as such, but a state that threatens the body's ability to maintain a normal internal environment (homeostasis) through the body's nervous, immune and endocrine functions (Charmandari, 2005). Chronic stress, however, leads to an aroused pathology that means the body is constantly moving away from these normal physiological parameters. The more accurate term for maintaining this internal environment is allostasis, which is about achieving stability through change, so ensuring the processes sustaining homeostasis stay within a normal range (Kiecolt-Glaser, 2010).

Stress operates through a negative feedback loop within the hypothalamic-pituitary-adrenal (HPA) axis through a series of hormonal signals that maintain autonomic nervous system activity. When the brain perceives something as dangerous, our fear centre, the amygdala, makes the initial response by sending a signal to the



hypothalamus, where corticotropin-releasing hormone (CRH) and arginine vasopressin are released into the anterior pituitary gland. This generates the release of adrenocorticotropic hormone (ACTH), which activates the release of glucocorticoids and catecholamines from the adrenal glands. At this moment, the sympathetic nervous system is activated and on high alert. As the stressful event subsides, the parasympathetic nervous system takes control and cortisol levels return to normal. In cases where the HPA axis is activated repeatedly, as in periods of chronic stress or pain, the negative loop fails and cortisol circulation is maintained, causing a lengthy neurochemical exchange that generates an imbalance that can initiate a long list of negative responses.

CORTISOL – FRIEND AND FOE

Cortisol is a steroid hormone that circulates through our blood and has many functions, but long-term exposure to high levels of it can have a detrimental effect on the body.

Cortisol assists in the metabolism of fats, proteins and carbohydrates, but when we are in a state of high stress we tend to crave more carbohydrates, which may result in increased fat deposits, weight gain and inflammation (Kiecolt-Glaser, 2010). When the negative feedback loop is unstable, there is a significant decrease in the release of peripheral thyroid hormones, which may also depress metabolic functions (Helmreich, 2005). In the immune system, cortisol works to prevent commotion of the inflammatory response by releasing anti-inflammatory cytokines. Increased cortisol circulation suppresses the activity of these important immune fighting cells, generating an increase in pro-inflammatory cytokines (Yeager, 2011). These increased levels of inflammation have a damaging effect on the cardiovascular system by increasing atherosclerosis (Black, 2002) and on collagen fibres by down-regulating collagen synthesis, and in decreased bone development (Gu, 2017).

WHY DISTRESS CAN CAUSE INFLAMMATION

As almost all our immune responses are controlled by glucocorticoids, in times of stress our immune and inflammatory responses are greatly impeded, which can cause an increase in pain (Charmandari, 2005).

Pro-inflammatory cytokines released into our cells can trigger many unpleasant and limiting symptoms. These inflammatory 'bad boys' can alter our neurochemistry and neuroendocrine communication systems and produce wide-ranging challenges to physiological functioning. Inflammation is experienced and seen as redness, heat, swelling and pain, but the unseen effects can actually cause more damage (Slavich, 2014). Chronic inflammation plays a major role in conditions such as asthma, arthritis, diabetes, atherosclerosis and some cancers, all of which have an element of pain attached to them. Whether from tissue or emotional injury, evidence shows that all pain stems from inflammation (Omoigui, 2007). When the sympathetic nervous system is under duress, the HPA axis prepares our body for the 'fight or flight' response and when this system is overloaded, it is unable to function effectively, and there is a decline in parasympathetic support and elevation in pro-inflammatory cytokines.

STRESS-INDUCED ANALGESIA/HYPERALGESIA

Stress, both good and bad, is also a major factor in the perpetuation of chronic pain conditions (Alexander, 2009). Our own in-built pain mechanism, the endogenous opioid system, can help to alleviate pain activated through fear, anxiety or stress, and delivers pain relief through a phenomenon known as stress-induced analgesia.

> "Reflexology can help to modulate the pain experience by increasing pain threshold and tolerance levels"

Stressful events can be complex, and may involve the pain-pleasure-reward circuit within the brain (Samuel, 2010), but increased fear, anxiety and a feeling of complete helplessness in managing persistent and disabling pain all amplify it. In fact, highly anxious people can create pain where there is neither pathological nor physical evidence for it (Ahles, 1987), and this can produce a phenomenon referred to as stress-induced hyperalgesia.

Fear and anxiety are both experienced as imminent threats to the body and motivate our defence mechanisms, making us hypervigilant. This increases our pain and our sensitivity to pain. Our emotions are influenced by, and influence, neural processes that shape our experience of pain (Vierck, 2009).

In short, stress can either suppress pain (stress-induced analgesia) or exacerbate it (stress-induced hyperalgesia) and this largely depends on the type of stress, the length of time experiencing that stress and its intensity, both emotional and physical (Alexander, 2009).

SUPPORTING CLIENTS IN PAIN

The biopsychosocial model of pain is based on the idea that ill health has both a social and a cognitive process that cannot be separated (Lumley, 2011). The model proposes that the body affects the mind and that the reverse is also true, so that an individual's response to pain is based on their emotional reaction to, and subsequent appraisal of, that pain experience. Cognitive behavioural therapy was designed to work alongside the biopsychosocial model by helping patients to break the pain cycle. They are taught how to become aware of negative thinking patterns that may link with their pain experience and then shown adaptive techniques to help overcome those thoughts (Samuel, 2010).

Another therapy for supporting clients in pain, and one I am much more familiar with, is reflexology. My own research has shown that reflexology can help to modulate the pain experience by increasing pain threshold and tolerance levels (Samuel, 2013). For the past 13 years I have been working alongside MNT-NR International® to bring nerve reflexology to the UK. This modified mode of reflexology works directly within the nervous system in a very precise and direct manner (Pauly, 2004; Veldhuizen, 2001). While there are a limited number of published research studies on the direct use of reflexology in the management of stress and anxiety

PICTURES: ALAMY; SHUTTERSTOCK

(Atkins, 2008; Hughes, 2008; McVicar, 2007), many of the pain studies and those on the physiological effects of reflexology have demonstrated a reduction in autonomic activity, which has a direct relationship with the stress response (Hughes, 2008; Choudhary, 2006; Tiran, 2005).

HOW REFLEXOLOGY CAN HELP – A CASE STUDY

A 34-year-old primary school teacher required treatment for her left-sided shoulder pain, which came with pins and needles in the hand.

Following a whiplash injury in 2009, an MRI revealed no damage to her spine. Steroid injections in both deltoid and supraspinatus muscles provided short-term relief.

Occipital headaches have been 'on and off' since the car accident but have been getting progressively worse recently.

Her job is stressful, and she was off sick with stress-related illness at the time of her visit. She is experiencing abdominal pain, bloating and slow gut motility.

CURRENT MEDICATION

She has been taking naproxen (a non-steroidal anti-inflammatory drug) and co-codamol for the past two years for the pain. These drugs have side-effects that can impact on the digestive tract, concentration, mood, sleep and cause hypersensitivity.

Omeprazole has been prescribed to protect her gut from the pain medication. In addition, she takes sertraline for depression. Common side-effects include drowsiness, dizziness, stomach upset and insomnia.

ASSESSMENT AND OBSERVATIONS

Assessment revealed restriction in abduction of her left arm, head rotation, and tilt. She also had sensitivity at lower cervical and upper thoracic transverse processes.

TREATMENT PLAN

The treatment sessions focused on lots of relaxation, providing a quiet space for the client to release both verbally and emotionally without judgement. I used a combination of nerve reflexology, classic foot reflexology and linking techniques.

When stress is involved in an illness it is important to work the limbic system to access the emotions and the autonomic nervous system to promote allostasis. As the only motor nerve of the diaphragm, I paid special attention to both the phrenic nerve and phrenic plexus, which also provide sensory feedback on the state of the upper abdominal organs. By helping to release tension in the thoracic cage, you help release tension in the organs, allowing them to move more freely. I also worked on the vagus nerve, which is the main parasympathetic outflow for the heart and gastrointestinal tract and plays an important role in peristalsis and glandular secretion. As gut bacteria produce the neurotransmitters serotonin and dopamine, they have a key role in mood, so work the digestive system with classic foot reflexology. The nerve reflex point for the nodose ganglia is also beneficial, as it contains all sensory nerve cells for the vagus nerve, as well as the HPA axis and hormonal system. Finish with the nerve reflex points for the brain, medulla and thalamus.

SUMMARY

The root cause of her current pain likely originated from an unresolved whiplash injury some years earlier in which there was sudden acceleration/deceleration. The body holds onto the memory of that incident, both physically and emotionally, long after the initial pain experience has subsided (Berry, 2017).

The client no longer obtained benefit from the pain medication and she was unhappy in her job, which increased stress and anxiety – and subsequently her sensitivity to pain. On a pain visual analogue score, she measured mild to moderate, which did not reflect the verbal account of her pain. Some of her symptoms were a result of her current medication, and her gut health was affected by this, impacting both her physical and mental wellbeing (Foster, 2017; Browning, 2017; Oriach, 2016). For this client, reflexology provided a safe space for relaxation and an opportunity to offload, but the cause of her stress was related to lifestyle choices, and that was something my client still needed to address.

Reflexology is just one of the many complementary therapies proven to be of benefit in supporting clients in pain. In the UK, chronic pain affects between a third and half of the population (Fayaz et al, 2016). However, it is also important to bear in mind that there may be times when you are not the right practitioner for a person in pain. You may need to signpost them to someone more able to address their emotional needs, so having a bank of professionals with whom you can refer, is always beneficial for your clients. IT

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REFERENCES

For full references, go to **fht.org.uk/IT-references**