A common consequence of many types of brain lesions is the development of muscle contractures: the stiffening of muscles that limits normal joint movement. Severe contractures cause deformities that are the most visible manifestation of brain damage.

Contractures arise when brain lesions cause paralysis or spasticity. Paralysis and spasticity change the mechanical environment of muscles – that is, they cause muscles to experience different patterns of activity, different changes in length and different forces than would normally be experienced. The muscles adapt in response to their altered mechanical environment by becoming stiffer, causing joints to become less mobile.

Contracture is common problem. In a recent study my colleagues and I monitored 200 consecutive people admitted to a Sydney hospital with the diagnosis of stroke (Kwah et al., 2012). Six months after admission half of all those people had developed at least one contracture. Contractures are also common in people with many other sorts of brain lesions. For example, contractures are prevalent in people who have had a traumatic brain injury, or who have multiple sclerosis or cerebral palsy.

Contractures prevent joint movement so they cause physical disability. For example, many people who have had a stroke or traumatic brain injury develop contractures of the calf muscles. Calf muscle contractures impede ankle motion, making it difficult to stand up from a chair or walk normally. In the same way, contractures of shoulder muscles can impair the ability to reach and contractures of wrist and finger muscles can impair grasp. Severe muscle contractures can cause the limb to adopt a fixed position. For many people, contractures become a much greater impediment to normal movement than the paralysis or spasticity that initially caused the contracture to develop.

There has been surprisingly little research into the mechanisms of contracture. As a result the mechanisms are poorly understood (Farmer & James, 2001). Studies on animals have shown that it is possible to make muscles become short or stiff with a number of experimental procedures. For example, leg muscles can be made short by immobilising the leg in a plaster cast and diaphragm muscles can be made short by inducing emphysema (a lung disease). These studies show that the stiffening of muscles can occur either because of changes in the muscle tissue (the muscle “fibres” or “fascicles”) or because of changes in the tendons that join muscle fascicles to bones. But studies on animal muscles can’t tell us about the mechanisms of contractures seen in human populations. Surprisingly, it is still not clear whether contractures in people who have had a stroke or traumatic brain injury are due to changes in the muscle fascicles or tendons.

There is just as much uncertainty about how to prevent and treat contracture. For the last half century physiotherapists and nurses have applied stretches to muscles, or passively moved limbs, or applied splints or casts to stretch the limb with the aim of preventing or treating contractures. But recent research suggests these interventions have little effect. For example in one study, 63 volunteers who had experienced a stroke were randomly allocated to receive either a wrist splint or
no splint. Two months later there was no discernable difference in the stiffness of the wrist of people who had or had not been splinted (Lannin et al., 2007). In another study, 20 volunteers who had quadriplegia after spinal cord injury were randomly allocated to receive passive movements to their ankles 10 times each week for 6 months or to not receive any passive movements (Harvey et al., 2008). At the end of the 6 months period the people who had received passive movements had, on average, just 2 degrees more ankle motion than those who did not receive passive movements. There have now been over 35 studies like these, and they quite consistently show little or no effect of stretch or movement-based interventions (Katalinic et al., 2010; Prabhu et al., 2013). For now at least, there are no treatments that have been clearly shown to prevent or reverse contracture.

Eventually scientific research will provide answers both about the mechanisms of contracture and about how to prevent and treat contracture. The development of new techniques for imaging and measuring the internal architecture of muscles using MRI provides one promising advance (Froeling et al., 2012). The first steps have been made in identifying the abnormalities of gene expression that are ultimately expressed as contracture (Smith et al., 2012). New ideas for treatments are being generated by basic research for subsequent testing in clinical trials. Hopefully the next decade will see major advances in both our understanding of the mechanisms of contracture and how to treat them.

References:


