The "outline of a talk" following was proposed to be given to the Royal Society, UK but had to be omitted due to a lack of time

# Epiphenomena of Radiculopathy

## Or: What happens when nerves start to go wrong

*(Outline of a talk given to the Royal Society, U.K.—February 1st, 1996 by Prof. C. Chan Gunn, M.D.)*

I wish to speak briefly about what happens to the body when peripheral nerves go wrong.

Although the brain and spinal cord are well protected within the skull and the spine, peripheral nerves, which conduct information to and from the central nervous system, are exposed to harm. Functional disturbances are therefore not uncommon in the peripheral nervous system, especially at the spinal nerve root, which because of its location, leaves the nerves particularly vulnerable to pressure, stretch and angulation. Wear and tear eventually leads to abnormal function in the nerve, a condition known as peripheral neuropathy.

Medical diagnosis typically regards pain as signals of actual or threatened tissue damage, and doctors usually have no difficulty in diagnosing and treating pain that is caused by injury (such as, a fracture), or by inflammation (such as, rheumatoid arthritis).

The profession, however, is often perplexed by painful conditions that have no apparent injury or inflammation to account for them (such as headache, backache, "tennis elbow" or repetitive strain injury). In these conditions, the perception of pain does not result from tissue injury (nociception); rather it occurs when innervated structures experience a disruption in the flow of arriving impulses, and compensate by becoming excessively sensitive (or supersensitive). Supersensitve receptors and pathways exaggerate ordinarily benign signals, which are misinterpreted as pain.

For example, in photophobia, it is not the light that is too bright and hurtful, but the eye that has become too sensitive. Pain, therefore, does not always signal tissue damage—more often than not it is a manifestation, or epiphenomenon, of "peripheral neuropathy"; just as fever is, of infection.

I would like to briefly describe several clinical tests that I had originally developed to determine neuropathy and explain chronic pain. These tests are simple, but have significance that goes far beyond pain: they are able to detect incipient or even presymptomatic clues to disease.

For example, in peripheral neuropathy, pressure receptors in muscle can become supersensitive: the muscle then becomes excessively tender to finger pressure. The anatomic distribution and the intensity of tender muscle points are good indicators of peripheral neuropathy. These can be used to differentiate mechanical back pain from one with neurological involvement. Tenderness can therefore be used as a simple and inexpensive screening test for workers in heavy labour. The Match Stick Test is a test for trophedema—the accumulation of fluid in subcutaneous tissue caused by increased capillary permeability and impaired lymphatic drainage. Trophedema, unlike oedema that follows congestive heart failure, cannot be indented by finger pressure, but when a blunt instrument, like the end of a match stick is used, the indentation is distinct and persists for many minutes. Not only is the Match stick Test easy to us, it can detect neuropathy earlier than electromyography.

Some of the tests are for finding shortening in muscles. In neuropathy, muscles go into "spasm" and shorten. This increases the tension in muscle and tendons, and ceaseless muscle pull can lead to a plethora of painful conditions, such as back pain, tension headache, tennis elbow, Achilles' tendon, or frozen shoulder. Many of these conditions are medical riddles; they appear dissimilar and unrelated, but in fact, they all have the same underlying pathology–muscle shortening following peripheral neuropathy.

Peripheral neuropathy also predisposes to arthritis. Muscle shortening upsets the alignment of a joint, limits its range of movement, and increases pressure on articular surfaces. These factors lead to the development of arthritis and eventually osteoarthritis. Tests that check for joint range can detect peripheral neuropathy long before there are any radiological changes.

Neuropathy also degrades the quality of collagen which provides strength to ligament, tendon, cartilage and bone, contributing to degeneration in weight-bearing and activity-stressed parts of the body.

The most critical consequence of peripheral neuropathy occurs when paraspinal muscles shorten. This draws adjacent vertebrae together, compresses the intervertebral disc and impinges on the nerve root. A vicious circle is created: pressure on the nerve leads to more shortening and to further compression of the nerve.

Compression compromises the nerve. Every structure in the body must receive a constant flow of motor impulses to regulate cellular function. Compression interrupts this "trophic" flow, causing the central nervous system to lose control over the periphery. This upsets the proper function of the structure and its associated spinal reflexes.

In a similar fashion, competent function of the autonomic nervous system (which is responsible for homeostasis or maintenance of a constant internal body environment) can also be deranged.

It is imperative, therefore, that any malfunction of the peripheral nervous system be urgently detected and rectified. While the causes of peripheral neuropathy are many, attrition of the spinal nerve root by wear and tear is, by far, the most common.

Although we can justifiably be proud of the many advances made in medical diagnostic procedures, there is no laboratory or imaging technique that can surpass a proficient clinical examination for the detection of the earliest signs of peripheral neuropathy. It is a false perception that "newer" (which usually means more expensive), equates to "better".