Some of the sensory problems may be generated from centres higher than the spinal cord. This is called central pain, and is due to hypersensitivity of the central nervous system.

It occurs when injury to the central nervous system is partial, and thus causes central sensitisation in the absence of total loss of function or sensation. It is commonly seen in patients with spinal cord injury (SCI).

Tasker ([1]) includes various conditions in his classification of central or deafferentation pain, and amongst these he specifies arachnoiditis as a cause.

The classical form of central pain is characterised by a constant, severe burning, often with a paradoxical component of cold, made worse by light touch. In some variations, pain from movement or from muscle tone is the major feature.

Recently, brain scans have revealed that the pain signal to the thalamus (the area of the brain that receives this input) may be severe enough to make it shut down to avoid cell death, which allows the uninhibited pain signal to reach the conscious brain.

Myers suggested in 1995 ([2]) that "ischaemic (poor blood supply) compression lesions may be the most common painful neuropathy" and noted that chronic constriction injury studies in rats cause hyperalgesia and allodynia in the distribution of the affected nerve.

This is a form of deafferentation pain.

Many patients with arachnoiditis seem to suffer from pain throughout the body, in areas that do not correspond to the site of the arachnoiditis.

Therefore, it seems that there may well be a component of central pain involved.

Devor, in 1995([3]), proposed that nerve damage causes pain by

"injecting abnormal discharges into the nervous system".

It is now widely thought that there is a form of "wind-up" of nerve cell firing, in which cells that normally do not deal with pain become pain receptors (nociceptors), which widens the area involved.

This can induce a "pain memory" which may involve various systems within the CNS (central nervous system). (Coniam and Diamond, 1992 [4]) The nociceptive input can generate within the spinal cord a state of "central sensitisation". Input into this is amplified. It also seems that the inhibitory system is affected the most by nervous tissue damage.

Central pain is recognised as being agonising. The brain is being bombarded with the same sort of information it would receive if severe tissue damage were occurring.

Mnemonic for central pain: **MD has CP:**

M uscle Pain (Gamma Pain)
D ysaesthesia
H yperpathia
A llodynia

S hooting Pain (Lancinating)
Circulatory
P eristaltic Pain (Visceral)
([5])

MUSCLE PAIN:

As well as the pain pathways discussed above, there is also the sensory arm of the gamma motor system, or pain sensation from the muscle spindle apparatus within the muscles.

Just as pain in the skin, so both allodynic and hyperpathic pain can be associated with muscle spindle pain. A very effective test for gamma pain can be effectively tested by asking the patient to go down into a squat position.

This causes a dysaesthetic burning "cramp" in the thigh or calf, along with a strong sensation of "pulling", resembling a muscle cramp after exercise. This rapidly becomes unbearable within seconds to minutes.

This pain may represent a hyperpathic response to pressure, and may resemble that felt by healthy people on sitting in one position for a very long period. Often patients report pain when trying to lie down in bed and they wake up feeling "battered".

Another type of muscle pain is that brought on by activity. In health people, only excessive and prolonged exercise such as that in a marathon run brings on what is termed ?lactic acid build-up' pain.

In patients with central pain, this type of pain is brought on within a very short period of activity. (Beric has termed this pain "kinesthetic dysesthesia" ([6]). It may be sufficiently debilitating that it effectively reduces a patient's functional ability.

"In the most common situation, if a Central Pain patient attempts to exercise, the tonic soreness in the muscles will be unbearable the next day. The onset of dynamic pain is immediate." ([7])

As we shall see below, muscle cramps are a common part of the arachnoiditis picture.

They may well be a feature of centralised pain. Localised cramps may persist almost continually. Exercise heightens gamma pain and is the principle reason why patients with arachnoiditis should not be considered non-compliant or even obstructive when they refuse to exercise.

"The physician should look upon dread of movement by the patient as a measure of the almost overwhelming severity of Central Pain, and not as an indicator of patient weakness." (508)

DYSAESTHESIA

This is bizarre pain that typically cannot be described but is generally burning in nature. It is not felt in normal people and is specifically a feature of incomplete nerve damage; it may be experienced in numb areas, but may extend further towards the centre of the body than the numb area.

Patients may find this pain experienced in virtually numb areas particularly difficult to describe and cope with. The loss of sensation seems to be partial rather than total, and the complication of the bizarre and highly unpleasant dysaesthesia is one of the predominant features of arachnoiditis.

It may sometimes be called deafferentation pain, or causalgia**.** Many patients suffer from burning feet in particular. Dysaesthesia is rather like an "afterburn", resembling the

pain of having just touched a hot stove, with a poorly defined ?flare' of pain.

Some patients describe a metallic quality to the pain. It may also seem simultaneously burning and freezing.

Tasker described a steady state level of "spontaneous" pain, which may be increased by light touch, clothes rubbing or temperature change. This increase is called "evoked pain" and is worse in areas where sensation is most reduced, nearly always in the extremities. It is an extreme pain, and may last for half an hour or so after the stimulus, although it does reduce by about a quarter once the stimulus is removed. It can be reduced more quickly by getting the skin temperature to the most comfortable level.

Evoked pain shows what is called "slow summation" i.e. it builds up gradually, with an initial time delay. Long exposure to stimulus (such as contact with bed sheets at night) is especially unpleasant.

HYPERPATHIA

This is increased pain from a stimulus that would be painful to normal people. Unlike dysaesthesia, it tends to be more proximal (nearer the centre of the body).

It also has a delay, but not one of time, rather what is termed "delay with overshoot", which means that there is a raised threshold for pain, but when that threshold is reached; there is an overshoot of pain, out of proportion with the stimulus.

Visceral hyperpathia can occur. This can affect both bladder and bowels, so that sensation of fullness is delayed and then becomes urgent and is often dysaesthetic i.e. burning (e.g. bladder

burning). This may lead to embarrassing accidents.

ALLODYNIA:

("other pain"): this is pain from normally non-painful stimuli or pain outside the area being stimulated (but is not the same as referred pain). It tends to be dysaesthetic.

It is very often triggered by changes in temperature, in which both hot and cold stimuli are perceived as hot, (termed "read only burning": ROB); light touch such as clothing may cause pain. Pain may be referred from other areas when they are stimulated. Continual or repeated stimulation can cause severe pain that persists after the stimulus is withdrawn.

CP (central pain)

sufferers have a narrow window of comfort as regards temperature changes. For this reason, cold draughts may be especially unpleasant, and hydrotherapy pools may be too hot.

LANCINATING (SHOOTING) PAINS

These are usually in areas where there is dysaesthetic burning, but they are very localised. The pain radiates, and may vary from being like an insect bite to an electric shock. The majority of patients have these transient shooting pains.

CIRCULATORY INSUFFICIENCY

This refers to nerve circulation (not blood), and manifests itself as "pins and needles".

These are usually in areas where there is dysaesthetic burning, but they are very localised. The pain radiates, and may vary from being like an insect bite to an electric shock. The majority of patients have these transient shooting pains.

These are usually in areas where there is dysaesthetic burning, but they are very localised. The pain radiates, and may vary from being like an insect bite to an electric shock. The majority of patients have these transient shooting pains.

Hyperpathia, as mentioned above, may affect the internal organs and may cause simple flatus to be experienced as severe cramping pains.

DISTORTIONS OF BODY SIZE PERCEPTION

In much the same way as your lip can feel odd after a dental anaesthetic, so too can parts of your body, usually those parts most affected by sensory loss and burning.

BIZARRE SENSATIONS

Typically, arachnoiditis patients report a variety of strange sensations affecting various parts of the body. These include feeling as if they are walking on broken glass, water running down the leg, sensation of insects crawling on the skin etc. They tend to be reluctant to report these experiences for fear of being diagnosed with a mental illness.

SENSORY ATAXIA

Sensory loss in the feet leads to trips and falls. Loss of proprioception (sense of where the feet are in relation to the ground) can mean that the patient feels their feet aren't really touching the ground.

AUTONOMIC EFFECTS (see below)

These include: excess sweating, changes of skin colour from white to purple, minor swelling, shininess of skin; also: fluctuating blood pressure (greater than usual response to physical stress such as change in temperature or position e.g. from lying to standing) These effects are exacerbated by cold or emotional upset.

In summary:

Central pain causes burning pain, often with a paradoxical component of cold, and is made worse by light touch or the rubbing of clothing (Bowsher's criteria [8]). It affects large areas of the body, or even its entirety, as it originates centrally rather than in the spinal cord. This may lead to fear that the disease has spread or may cause doctors to dismiss symptoms as psychological.

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[1] Tasker RR: Deafferentation, in Wall PD, Melzack R (Eds): Textbook of Pain. Edinburgh: Churchill Livingstone, 1984, pp 119-132

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[3] Devor,M, "Peripheral and central nervous system mechanisms of sympathetic related

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[4] Coniam and Diamond, "Chronic Pain Management" 1992

[5] http://www.painonline.org/mnem.htm

[6] Beric, A, Muscle Nerve. 1993 Oct; 16(10):1017-24.Central pain: "new" syndromes and their evaluation." 1993

[7] Pain Online resource http://www.painonline.org/muscle.htm

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