In arachnoiditis, the predominant pain tends to be neuropathic (neurogenic)i.e. arising from nerve damage.

However, one must also bear in mind that there may be musculoskeletal pain both from the original underlying spinal problem and also as a secondary feature due to muscle tension in response to unalleviated pain.

So there may be a variety of different types of pain experienced.

The commonest ones appear to be: Burning Shooting/stabbingDeep aching Muscle spasm Joint pain Headache Abdominal pain.

In Aldrete's survey (1) he describes a ?pathognomonic' (diagnostically definitive)feature of burning sensation in the feet and legs seen in 88% of the respondents.

In a further 11% the burning was confined to the lower back.

A common feature in arachnoiditis patients is that pain radiates upwards to the thoracic spine (29% in Aldrete's survey) and to the upper extremities.

This can lead to worry that the arachnoiditis has spread.

In the majority of cases of lumbar adhesive arachnoiditis, however, the cause may be either secondary musculoskeletal pain, and/or abnormalities of CSF (spinal fluid) pressure (impaired

CSF flow due to arachnoid scarring) and/or underlying spinal abnormalities at the relevant spinal level (e.g. degenerative disc disease or spondylosis in the neck can cause arm symptoms).

Centralised pain (CP): can occur when pain becomes chronic.

It arises from a sensitisation of the central nervous system, which becomes hypersensitive; it is almost as if it has been reset to be on constant ?red alert.

This means that pain perception is ?reset and pain signals are sent from nerve receptors that would normally only be involved in perception of pressure/touch etc.

In terms of symptoms, this can lead to a generalised effect throughout the body (not just in the lower half, as would be expected, say, in lumbar arachnoiditis).

As well as pain, the autonomic nervous system is also affected, (see below) which leads to hyperactive sympathetic nerves: increased sweating, difficulties with temperature regulation, fluctuating blood pressure etc.

CP may also be referred to as causalgia or deafferentation pain. It tends to involve a number of features:

1. Dysaesthesia: this term means pain which is very difficult to describe; it is unlike any other type of pain experienced; (people with undamaged nervous systems do not experience this type of pain). Most people use the word ?burning; in an attempt to describe the sensation. It is often an unremitting, relentless pain, and may well be worse at night.
2. Allodynia: this means pain is felt from what would normally be non-painful stimulus, such as light touch or change in temperature: these can become unbearable; the longer the stimulus persists, the more the pain increases, and it may well be felt over a greater area than is being stimulated (these features are known as temporal and spatial summation. So it is common for people with arachnoiditis to find it difficult to tolerate bedclothes or draughty rooms. Aldrete's survey (1) found that around 15% of arachnoiditis patients suffered from allodynia.
3. Hyperaesthesia: hypersensitivity to a variety of sensations; this might well include

photoaversion/photophobia (intolerance of light), and hyperacusis/phonophobia (intolerance of noise: see below

4. Hyperalgesia/hyperpathia: similar terms which refer to a heightened pain perception. It is not a question of a low pain threshold, rather, a delayed one, which, once reached, leads to an overblown pain response out of proportion to the painful stimulus (NB this is NOT a psychological response, but a physiological one). Examples of this which occur commonly in arachnoiditis patients are: painful urgency to empty the bladder; marked abdominal cramps with constipation.(these are both examples of visceral hyperpathia.
5. Hypoaesthesia reduced sensation: tingling or even pain may occur in numb areas.
6. Formiaesthesia a formidable term which literally means sensation of ants; bizarre sensations are common and can include a wide variety of sensations such as water running down the leg, insects crawling on the skin etc. etc. Patients are often reluctant to report these symptoms for fear of being diagnosed with a mental illness.

Neuropathic pain occurs in partially denervated nerves, that is to say, nerves that have some, but not total damage, so there may well be intact function as regards loss of sensation or impaired reflexes.

Indeed, damage of as little as 5% of the nerve function can lead to severe pain but all tests to date are unable to detect such a level of change.

If spinal nerve roots are affected, the resulting radiculopathy affects various target structures supplied by the affected nerve. Generally, there are motor, sensory, autonomic or mixed deficits.

Structures which are supplied by a nerve rely on this innervation to maintain function and integrity; loss of the nerve's regulatory (?trophic') effect leads to these structures becoming highly irritable; they develop abnormal sensitivity, becoming supersensitive.

Cannon and Rosenblueth's Law of Denervation:

"When a unit is destroyed in a series of efferent neurons, an increased irritability to chemical agents develops in the isolated structure or structures, the effect being maximal in the part directly denervated." ([1])

This phenomenon is known as disuse supersensitivity. It can affect;

- muscle (skeletal and smooth: the latter in viscera such as blood vessel walls).
- spinal neurons (which link the peripheral nervous system and the central nervous system and also deal with spinal reflexes).
- sympathetic ganglia (?junctions' in the sympathetic nervous system).
- adrenal gland (hence symptoms of ?arousal' to ?fight or flight' : rapid heart rate etc. which can give rise to feeling of anxiety/agitation).
- sweat glands (increased activity). brain cells.

These structures will inevitably behave abnormally, ?over-reacting' to many forms of input, not merely chemical (nociception), but also all types of physical input, including those that would not normally trigger such an exaggerated response:
stretch, pressure.

Furthermore, supersensitive muscle cells can generate spontaneous electrical impulses that trigger involuntary muscle activity (e.g. spasm) and pain.

Supersensitive nerves can become responsive to chemical neurotransmitters all along their length rather than at the end (terminal) and damaged nerves may sprout, forming connections with other types of nerve such as autonomic and sensory, thus creating a ?short circuit' between sensory and autonomic nerves (this may be a way of explaining RSD: CRPS Type I).

In areas of neuropathic pain, there may be the following signs:

- Affected areas are noticeably colder due to vasoconstriction.
- There may be ?goosebumps'; cold stimulus can increase this and also precipitate Pain.
- There may be increased sweating.
- Pressure on a tender muscle can produce goosebumps (piloerection) and/or sweating (sudomotor response).
- Skin may be pale.
- Localised oedema (swelling) due to increased tone in lymphatic vessel walls and increased blood vessel permeability:
- neurogenic oedema or trophedema:

- orange-peel skin; match-stick test: end of stick produces a clear-cut indentation in skin which may persist for several minutes
- Increased muscle tone.
- Tenderness over certain trigger points.
- Loss of joint range of movement due to muscle shortening (contracture).
- Fibrosis.
- Mild stimulation can cause extreme pain.
- Pronounced summation and after-reaction with repetitive stimuli.

Note: a neuropathic nerve can cause severe pain but be apparently normal, maintaining the ability to function: conducting nerve impulses, synthesising and releasing neurotransmitter substances and evoking action potentials and muscle contraction.

Tests of nerve function will therefore fail to reveal the source of the neuropathic pain.

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[1] Cannon WB, Rosenblueth A The Supersensitivity of Denervated Structures, a Law of Denervation. The MacMillan Company New York 1949