Slightly technical article intended for medical professionals, which means it has lots of long words. Each section has been extensively covered in other articles on the site.

This is the full article splt into chapters below

HANDBOOK ON ARACHNOIDITIS

INTRODUCTION:

Arachnoiditis is a chronic, insidious condition that causes debilitating, intractable pain and a range of other neurological problems. It has been regarded as rare by the medical community, but Burton reported as early as 1978([i]) that it is "common in patients with severe back and/or leg pain and functional impairment due to the failed back surgery syndrome."
Adhesive arachnoiditis is not a notifiable disease and is significantly under-diagnosed.
PATHOLOGY:
Arachnoiditis is chronic inflammation of the arachnoid layer of the meninges, which consists of trabeculae, a mesh of interwoven collagen fibrils resembling tissue paper. These secrete spinal fluid, which circulates through the cerebrospinal axis and is absorbed through the arachnoid villi in the brain.
The initial phase of the inflammatory process involves influx of white blood cells in response to an insult to the subarachnoid space, such as blood (trauma, surgery), foreign substance (dye, etc) or infectious agent (e.g. meningitis). This is initiated via the action of cytokines. There is infiltration by macrophages and mesenchymal cells; the latter transform into fibroblasts, which lay down collagen.
Usually the fibrinolytic process, which breaks down excess scar tissue, limits this, but in arachnoiditis the scar tissue continues to form. Authors such as Jayson ([ii]) have suggested that there may be a defect in the fibrinolytic pathway.
In the first stage involves radiculitis and the adjacent blood vessel hyperaemia. The subarachnoid space disappears. Deposition of collagen fibrils begins. In the second stage, (arachnoiditis) the scar tissue increases, and the nerves become adherent to each other and the dura.
The third stage, (adhesive arachnoiditis), involves complete encapsulation of the nerve roots. The subsequent compression causes them to atrophy.
The scarring prevents the arachnoid from producing spinal fluid in that area.

Complications include: Subarachnoid cysts: These are a recognised complication of arachnoiditis, in particular that caused by myelographic dyes or epidural anaesthesia. ([iii]
) They tend to be more common in the thoracic region than cervical or lumbar. In symptomatic cases, clinical presentation is generally non-specific, although there may be a sensory level, unlike in uncomplicated arachnoiditis. Surgical excision or drainage is often successful, provided that there is early intervention.

Syringomyelia is an uncommon complication of arachnoiditis, probably arising due to the pressure dissociation between the subarachnoid space and the central canal. Kamada et al ([iv]) recommend follow-up serial MRI imaging for patients with adhesive arachnoiditis in order to detect syringomyelia as early as possible.
It should be suspected if there is an increasing scoliosis (which is thought to be due to unequal nerve supply to the paraspinal muscles), with pain in a "cape-like" distribution in the upper body and areas of dissociated sensory loss, in particular, loss of temperature sensation in upper limbs which may lead to painless burns. There may be atrophy of small muscles in the hands and spastic paresis, gradually progressive, leading to difficulty in walking.
A further, rare, complication is communicating hydrocephalus. This is thought to be due to alterations in the cerebrospinal fluid dynamics, due to the effects of the scarring in the subarachnoid space.
AETIOLOGY:

1. Of mechanically-induced arachnoiditis (MIA)
Spinal surgery (especially multiple)
Multiple lumbar punctures
Trauma
Spinal stenosis (congenital/degenerative)
Chronic disc prolapse/ degenerative disc disease

2.Of Chemically-induced arachnoiditis (CIA)
Myelographic dyes (especially oil-based such as Myodil (Pantopaque))
Epidural steroid injections (e.g. Depo-Medrol)
Epidural anaesthesia
Other intraspinal drugs such as amphotericin B and methotrexate
Chemonucleolysis with chymopapain

3.Miscellaneous

Subarachnoid haemorrhage
Infection e.g. meningitis
SYMPTOMATOLOGY:

In MIA, these tend to be more directly related to the pathological lesion, although there may be an element of central pain.
In CIA, there is a more florid, systemic picture, with a higher number of widespread autonomic features and often autoimmune type symptoms.
The predominant and most distressing symptom of arachnoiditis is chronic, persistent p ain, which
is primarily neurogenic and thus difficult to treat.
Pain tends to increase with activity. There is may be a delay after onset of activity, with a slow summation, to a point where the pain suddenly becomes unbearable and then persists once the activity has ceased. This can make it difficult for patients and physicians or physiotherapists to assess what is the tolerable level of exercise.
Pain may be due to other factors besides nerve damage. These include musculoskeletal secondary to disuse, overuse or compensatory use of muscle groups, due to alteration of spine dynamics. There may also be muscle tension due to being in pain, or spasticity caused by nerve damage. Joint pain may be due to similar factors, or may be part of the autoimmune picture (see below).
Deafferentation pain (causalgia/dysesthesia): Pain is generally described as burning, but often people are unable to describe it. It is specifically a feature of incomplete nerve damage. Many patients suffer from burning feet, in particular.
Lancinating pain: The majority of patients experience transient shooting pains that may vary in intensity from an insect bite to an electric shock.
There is often an element of central pain, including feeling pain from normally painless stimuli especially from light touch such as clothing
(allodynia).
Changes in temperature commonly trigger this type of pain, so that sufferers have a very narrow window of comfort as regards temperature. (See also under autonomic effects).
Hyperpathia: an enhanced response to painful stimuli, suggestive of a low pain threshold. In fact, there is not a lowered threshold, rather a raised one, but once it is reached the response is magnified. This is called "delay with overshoot".
This is particularly noticeable in "visceral hyperpathia" in which normal bladder and bowel sensation is diminished, but once the signals of fullness are perceived, there is burning pain and urgency. This can lead to embarrassing accidents, especially if there is also nerve damage to bladder or bowel causing hyperactivity or sphincter dysfunction.

The areas commonly affected by pain are:
In most cases: lumbar, buttocks, legs (often both), feet, perineum, hip, abdomen.
In some cases: arms and hands, neck, head and face, chest.
However, it is important to remember that one of the aspects of central pain is that pain may be experienced over large areas of the body, rather than just in the lower part. This may lead the patient to fear that the disease has spread.

Parasthesiae and numbness are common features. Other sensory symptoms include loss of proprioception. This can result in tripping and falls. Temperature perception is sometimes diminished. There may also be bizarre sensations such as feeling as if walking on broken glass, water running down the legs, or insects crawling over the skin. These can be very distressing and many patients are reluctant to admit them to their doctor. A minority of patients may suffer from tinnitus and/or vertigo.

Motor nerve damage may cause loss of muscle strength, especially in the lower back and legs, in some patients. In most cases with weakness, it is mild, but it may progress sufficiently in some patients to necessitate use of walking aids or even a wheelchair. There seems to be a phenomenon that some patients reach a plateau in their symptoms, but can then rapidly deteriorate after a minor accident, such as a fall. The reason for this is unclear.
Also, many patients report that they fatigue quickly. There may be compensatory overuse of some muscle groups to allow the patient to walk, but this leads to the muscle fatiguing more rapidly than normal. This is similar to the picture seen in PostPolio Syndrome (PPS).
Increase in muscle tone is quite a common feature and makes the legs stiff, which may have an effect on mobility.
Muscle spasms and cramps may be violent and painful. Muscle fasciculations are a common feature.
A number of patients complain of symptoms suggestive of Restless Legs Syndrome, with nocturnal unpleasant sensations in the legs, accompanied by motor restlessness.
Less commonly there may be trouble swallowing, sometimes due to oesophageal muscle spasms. (See also under autonomic problems).
A common component of the arachnoiditis syndrome is the effect on the autonomic nervous system.
Principal symptoms of autonomic dysfunction include:

Bladder, bowel and sexual dysfunction. These are often very distressing to patients. Neurogenic bladder dysfunction may cause difficulty initiating urination and emptying the bladder, or hyperactive detrusor with sphincter disturbance causing incontinence. If the bladder is incompletely emptied (leaving a residual volume) there is a risk of recurrent urine infection.
Detrusor hyperactivity can give rise to high bladder pressures and possible reflux of urine to the kidneys, with a risk of hydronephrosis. Either problem may be exacerbated by decreased bladder sensation, which may lead to overflow incontinence, especially if there is an element of visceral hyperpathia. There may also be nocturia. Drugs such as antidepressants (e.g. amitriptyline) may worsen bladder dysfunction, causing difficulty in initiating micturition and emptying the bladder.
Bowel function may also be affected. Constipation due to drug treatment (especially opiates) and decreased mobility may complicate the picture.
Dyspepsia and intermittent vomiting are relatively common problems. They may be due to gastroparesis similar to that seen in diabetic autonomic neuropathy. Symptoms of gastroparesis include postprandial nausea, epigastric pain/burning, bloating, anorexia and vomiting. There may be vomiting of undigested food in the middle of the night or in the morning prior to eating breakfast. Prokinetic drugs such as Cisapride may relieve bowel motility disorders, including reflux oesophagitis.
Sexual dysfunction may affect potency and ejaculation in men, as well as causing problems with orgasm in both sexes.

Blood pressure disturbance (high, low or fluctuating); this may cause dizziness, syncope, or headaches. Orthostatic hypotension may occur.
Very rarely, there may be autonomic dysreflexia as seen in spinal cord injuries, with paroxysmal hypertension due to excess sympathetic activity reflexly activated by bladder or bowel distension, as described by various authors. (
[v]
)
Other cardiovascular symptoms include palpitations.
Cold extremities (Raynaud type phenomenon) are a common vasomotor problem. Sudo motor effects
of hyperhidrosis or anhydrosis may impact on temperature regulation, which is a very common problem.
Hyperhidrosis may be compensatory for loss of sweating in another area, or may be the initial phase before progression to anhydrosis. The majority of patients experience heat intolerance.
An uncommon problem may be facial pain, loss of sweating on one side of the face and change in size of one pupil (Horner's syndrome). There are also isolated reports of Adie's tonic pupil.
Oedema of the limbs is seen in some patients. A number of patients seem to be

diagnosed with Reflex Sympathetic Dystrophy (RSD), (Complex Regional Pain Syndrome Type I). This is characterised by severe burning pain in a limb, after trauma or surgery. There is usually an element of allodynia and hyperpathia. Autonomic effects include sudomotor and vasomotor abnormalities. There are changes in limb temperature, discolouration and oedema. Later stages may involve joint stiffness, loss of mobility and osteopaenia or osteoporosis, as well as skin texture and hair growth changes. The similarities between this condition and arachnoiditis suggest that the RSD type symptoms are in fact a part of the arachnoiditis syndrome, rather than a separate disease entity.

The following group of symptoms is reflective of the inflammatory nature of the condition and may point to an autoimmune component:
Most arachnoiditis patients experience a fluctuating course of the more systemic symptoms, with intermittent "flare-ups" and periods of relative remission. Some have intermittent low-grade fevers, malaise and raised ESR and/or white cell count.
They may also have lymphadenopathy.
A common feature is skin rash, often unexplained. Often this is urticarial or there may be angio-oedema , both suggestive of an allergic-type reaction. Some patients present with a vasculitic type rash. A few develop photosensitivity, but this may be related to medication.
Joint pains are also common, not just in weight bearing joints, but also small joints.
A number of patients complain of dry eyes and mouth (as seen in Sjogren's syndrome) but this is likely to be due to side effects of medication in most cases.
Other eye problems include iritis and uveitis, both inflammatory conditions seen also in association with autoimmune diseases.
Patients may have a dual diagnosis of arachnoiditis and fibromyalgia (or chronic fatigue). It is likely that the features of myofascial pain and malaise are part of the arachnoiditis syndrome itself rather than a separate condition.

A minority of patients also has a diagnosis of an autoimmune disease in conjunction with their diagnosis of arachnoiditis. These include Systemic Lupus Erythematosus, Sjogren's syndrome, Thyroiditis, Sweet's syndrome, Rheumatoid Arthritis, Primary Biliary Cirrhosis and Crohn's disease

Miscellaneous problems such as osteoporosis (c.f. in RSD, or due to decreased mobility) low potassium (possibly due to medication), chest pain mimicking angina, recurrent sinusitis, dyspnoea are seen in a few patients.
Eye problems (see autoimmune symptoms) seem to be quite common, with some patients who have undergone myelography complaining of photoaversion. Patients may describe stabbing pains or tingling and seeing "stars". There is an increased incidence of migrainous type headaches, often with auras. (but note the association between photoaversion and anticonvulsant treatment, particularly phenytoin and

carbamazepine.) ([vi])
Recurrent dental problems are quite common. Many patients undergo repeated root canal procedures but continue to suffer from facial pain and odontalgia without attributable dental pathology. A number of patients also suffer from bleeding gums (periodontal disease) and a few have "burning mouth syndrome". It is possible that some of these problems are related to medications that cause dry mouth, the lack of saliva contributing to reduced protection against infection and caries. The burning mouth symptoms could have a neuropathic component.

Dysphagia may affect some patients, especially those who have cervical pathology. In particular, this may occur if there is arachnoiditis accompanied by degenerative changes such as anterior osteophytes.
However, it may also be experienced by those with only lumbar pathology, possibly as a result of oesophageal reflux (due to autonomic effects on vagal tone), which may also cause non-cardiogenic chest pain.
Pharyngeal symptoms may include feeling as if a lump is stuck in the throat, and this may be dismissed by some clinicians as "globus hystericus".

Fatigue is a very common complaint, and can be due to a variety of factors.

Weight gain is a common problem. This is largely to do with decreased mobility and possibly to fluid retention secondary to medication (from drugs such as: Amitriptyline, Gabapentin, Ibuprofen, Morphine and other opiates, prednisolone/methylprednisolone). Alternatively, some patients may suffer weight loss, due to general debility and often, poor appetite.
The cognitive effects of arachnoiditis are anxiety and reduced ability to think clearly, with some short-term memory impairment. These are usually in direct proportion to the pain level being experienced. ([vii])
Sleep disturbance is common, and usually directly related to pain. It may contribute to depression, which is an understandable reaction to intractable pain, loss of function, loss of role and job, financial and relationship problems as seen in other chronic, debilitating conditions.
Fear for the future (prognosis cannot be predicted) and uncertainty about the diagnosis substantially increase this problem.
Many sufferers are reluctant to admit to depression.

Side effects of medication. These occur, to some extent, in most arachnoiditis patients, largely because of the potent drugs involved, which are often in combinations. Opiates alone can cause a wide variety of side effects, but when taken in combination with adjuncts such as antidepressants, anticonvulsants or muscle relaxants, there may be a cumulative effect.
The most common side effects are dry mouth, constipation, drowsiness, nausea, dizziness, urinary retention and blurred vision. Some drugs, such as opiates, NSAIDS and certain antidepressants may cause fluid retention, and thus weight gain.
There are a few patients who develop liver and kidney problems, probably due to adverse effects from medication.

CLINICAL ASSESSMENT

It is vital for the assessing physician to take into account that adhesive arachnoiditis does not present with a discrete clinical picture and that there may be symptoms that at first glance appear unrelated to any proven pathology.
Sadly, a significant proportion of patients may have had difficult previous experiences with the medical profession. Many have been labelled as having psychosomatic problems, although as the Mensana study in 1993([viii]) found, chronic pain patients do tend to have underlying organic pathology.
There is, moreover, a physician bias against patients involved in litigation and also women with chronic pain conditions (xli).
These factors may cause distrust from the patient. This can be compounded by feelings of anger about iatrogenic causes for the condition (if the patient is aware of this) and thus the patient may be either over-assertive or excessively anxious. It may therefore be unproductive to assess the patient's personality and coping abilities within the first interview, and this may be best postponed until a good rapport has been established.
Historical information may be convoluted and patients are often poorly able to communicate the sequence of events and the current, usually diverse symptoms.
Lack of information about the condition can lead to severe anxiety. Although the diagnosis of arachnoiditis is one of an incurable condition, most patients feel relief to have a name for their illness and feel that their suffering will now be recognised and legitimised.
Examination may or may not reveal significant neurological deficit. However, the possibility of pain of central origin should be borne in mind even if there is no obvious clinically observable abnormality.

PROGNOSIS

Arachnoiditis has been described as an insidious disease that is incurable. Guyer's paper on the prognosis of arachnoiditis ([ix]) suggests that there tends to be a

spectrum of the course of the disease, which varies from mild and non-progressive, to a fulminating progression that may cause paralysis and even death. Wilkinson (
[x]
) believes that progression after the first 24 months is unlikely to be due to the disease process alone. Most authors state that its onset may be years after the precipitating cause.
In general, arachnoiditis presents a highly variable clinical picture, with a fluctuating course.

DIFFERENTIAL DIAGNOSIS

Essentially, this involves excluding other causes of FBSS, such as recurrent disc herniation, disc fragments, stenosis, spondylosis or epidural fibrosis.
However, other causes of polyneuropathy should also be considered, especially those of an autoimmune origin.
It is interesting to note that a number of patients have a dual diagnosis of arachnoiditis and Multiple Sclerosis. This is presumably due to some similarities between the two conditions.
Fibromyalgic symptoms are likely to be part of the arachnoiditis syndrome, as opposed to being due to a separate disease entity.
Limb symptoms may be diagnosed as RSD.

DIAGNOSTIC TESTS

As arachnoiditis does not present with a discrete clinical picture of specific motor, sensory and reflex abnormalities, diagnosis tends to rest on tests such as MRI or CT scans. The current investigation of choice is a T2 weighted, fat suppressed, gadolinium enhanced, high resolution MRI scan. Ideally, a neuroradiologist experienced with the appearance of arachnoiditis should read this.
It is important that treatable causes of Failed Back Surgery Syndrome (FBSS) such as recurrent disc herniation, disc fragments or stenosis be excluded.
Further tests, which demonstrate nerve damage, include EMG and nerve conduction studies.
For bladder dysfunction, urodynamic studies may be required.

TREATMENT OPTIONS

Generally speaking, this complex neurogenic pain syndrome is best treated at a specialist pain clinic, with a multidisciplinary approach.

Of the well-established treatment regimes, opiates are frequently used.
However, these may be ineffective in combating any central component of the pain.

The issue of dependency concerns most practitioners and may lead to reluctance to prescribe. It is likely that there will be a risk of physical dependence, and thus of withdrawal symptoms if the opiate medication is discontinued. Also, there is an element of tolerance that may develop in long- term use, with the need for increasing doses for effective pain relief.
However, psychological dependence and abuse are less likely in chronic pain patients than in those who use opiate drugs recreationally.
It is best to start with short-acting morphine four hourly, until adequate analgesia is established. Breakthrough pain may require top-up doses.
Once control has been established, it is advisable to change to a slow release preparation such as MS Continus, which has a predictable duration of action for 8-12 hours, and can thus be given twice daily.
Fluctuations in dose requirement may occur, and in this case, the slow-release preparation should be replaced with a shorter acting one for the period of increased dose requirement.
Adjunctive treatment may also be necessary:
Antidepressants are useful for the background burning neuropathic pain, but are used in far lower doses than for depression (e.g. amitriptyline 25mg at night). It should be noted that the more selective antidepressants such as Prozac have been found to be poorly effective against neuropathic pain, first generation tricyclics being much more useful.

Anticonvulsants such as carbamazepine are particularly useful for the sharp, lancinating type of neuropathic pain. A relatively new drug, Neurontin (Gabapentin) is useful for pain relief and muscle spasms.

Antiarrythmic drugs such as mexiletine may also be used for neuropathic pain.

Muscle relaxants may be needed, including benzodiazepines such as diazepam.

Baclofen is a useful drug for spasticity. However, it should be noted that paradoxical increase in spasticity might occur. Also the Committee for Safety of Medicines (CSM) has advised that serious side-effects such as autonomic dysreflexia may be seen on withdrawal and a gradual dose reduction over at least 1-2 weeks should be undertaken to

avoid these. ([xi])
Non-steroidal anti-inflammatory drugs are not generally effective for pain relief and may cause significant gastrointestinal side effects and occasionally kidney problems after prolonged use.
Some patients have found that the troublesome nocturnal muscle cramps may be relieved by quinine.
Invasive treatments

These are not recommended by the Arachnoiditis Trust who believe that ANY invasive procedure carries a significant risk of exacerbating the inflammation of arachnoiditis, thereby worsening the patient's condition.

However, as always, it must be a question of weighing up possible benefits against possible risks, and individual needs must be assessed.
INA (Intraspinal narcotic analgesia): the "pump". This was originally developed for use in terminally ill cancer patients and thus was not being used long term. Of the studies of long term pump use, there are varying opinions as to its safety and efficacy. One recent paper states: "About one third of the patients get good long-term pain relief without
Complications or side effects, many require the addition of local anesthetics, and some never get effective relief. There are major questions to be answered before this form of therapy becomes widely disseminated."([xii])
Opiates are often supplemented with either local anaesthetics such as bupivicaine, or antispasmodics such as baclofen.
It is vital to ensure that preservative free solution is used.
Adverse effects of INA such as constipation, nausea, vomiting and itching tend to be short-term, whereas loss of libido and potency may persist for several months. The most persistent side-effects are sweating and oedema, the latter of which may necessitate INA being discontinued. The most serious adverse effect is respiratory depression
Spinal Cord Electrostimulation (SCS) involves electrical stimulation by implanted electrodes around the spinal cord, (in the epidural space), in the area that is most involved in causing pain. The very low energy current shuts down the input of pain fibres. Success rates seem to vary in different studies but are overall approximately 50% when all types
of chronic pain are considered, and the benefits may decrease with time. However, there is little literature on its efficacy in the specific case of arachnoiditis. Kumar ([xiii]) suggests that there is a favourable response to treatment of postsurgical arachnoiditis or perineural fibrosis if the pain is predominantly confined to one lower extremity.
Meilman et al (
[xiv]

) also state that SCS is of greater efficacy for unilateral lower limb pain than for more widespread nerve root involvement. It is best for controlling the dull, constant pain and poor for the sharp, lancinating pain. SCS may also be useful for neurogenic bladder problems. (
[xv]
)

Surgical treatment is generally regarded to have a low success rate. Resection of scar tissue is often followed by recurrence. Some specialists are now using laser techniques, but data on the outcomes is limited.
Epidural steroid and local anaesthetic injections: these are of questionable (and temporary) benefit and carry a risk of causing the very problem they are being used to treat. O'Connor et al ([xvi]) sum up the situation by stating that the "abnormalities of the epidural and subarachnoid spaces in such patients"(i.e. with chronic spinal arachnoiditis)... gives rise to "unpredictable and potentially dangerous results" following drug injection into these spaces.

Local nerve blocks
For those patients who have been diagnosed with RSD, sympathetic blockade may be offered. However, the literature is divided as to the efficacy of these techniques. Whilst they may be of use in the initial phases of the condition, when sympathetically maintained pain (SMP) is predominant, once central sensitization occurs (and thence what is termed "sympathetically independent pain: SIP") they are much less likely to be effective.
Other modes of administration
These include transdermal patches e.g. clonidine (but may cause hypotension) fentanyl (an opiate agonist). Fentanyl patches tend to produce fewer side effects than oral morphine.
Topical application of capsaicin is used to treat pain in peripheral neuropathies such as seen in diabetes mellitus. However, many patients find the initial (expected) increase in pain (which occurs prior to the anaesthetic effect) is intolerable, and few remain using it.

Non-pharmacological treatments
These include Transcutaneous Electrical Nerve Stimulation: TENS (of limited use)
Acupuncture (contact with patients who have tried this suggests that it is not as useful as could be hoped)
Physiotherapy: must be gentle as vigorous exercise may precipitate a flare-up. As in PPS, a non-fatiguing programme is likely to be the most beneficial.
Hydrotherapy: often very useful, but the water must not be too warm (heat intolerance is common in arachnoiditis patients)
Hypnosis

Biofeedback
Cognitive techniques
Relaxation/meditation: these are all helpful adjuncts to drug treatment, but few patients can manage on these pain management techniques solely.

3 relatively new techniques are becoming available:
1.APS (Action Potential Simulation) electrical stimulation (non-invasive) similar to TENS but of a different electrical waveform.
2.LLLT (Low-level Laser Therapy) again, non-invasive, resembling ultrasonic treatment in its application, it has been used with success in patients with various types of neuropathic pain, (e.g. post-herpetic) but mostly in more localized conditions.
3.PENS (Percutaneous Electrical Nerve Stimulation) is a technique that bridges acupuncture and electrical stimulation (TENS); low level electrical current (cf. TENS) is delivered via a series of ultra-fine, acupuncture-like needles. A recent study ([xvi i]) has demonstrated that PENS was more effective than TENS in providing short-term pain relief and improved physical function in patients with chronic low back pain.

MULTIPLE CHEMICAL SENSITIVITY

A few arachnoiditis patients may develop multiple chemical sensitivity. Some patients with arachnoiditis subsequent to injection of foreign substance into the subarachnoid space may have features suggestive of Multiple Chemical Sensitivity.

A number of patients have new allergies to antibiotics, especially penicillin related.

Some patients describe adverse reactions to dental anaesthetics, particularly those containing adrenaline.
These problems may lead to difficulties in prescribing for patients with arachnoiditis.

REFERENCES:

[i] Burton CV *Spine* 1978 Mar; 3(1): 24-30 Lumbosacral arachnoiditis

[ii] Jayson MI, Keegan A, Million R, Tomlinson I *Lancet* 1984 Nov 24; 2(8413): 1186-7 A fibrinolytic defect in chronic back pain syndromes.

[iii] Torres D, Bauso Toselli L, Vecchi E, Leiguarda R, Doctorovich D, Merello M, Guevara J, Nogues M *Medecina (B Aires)* 1993;53(50):391-396 [Spinal arachnoiditis as a complication of peridural anesthesia]

Sklar EM, Quencer RM, Green BA, Montalvo BM, Post MJ *Radiology* 1991 Nov;18(2):549-554 Complications of epidural anesthesia: MR appearances of abnormalities

Nogues MA, Merello M, Leiguarda R, Guevara J, Figari A *Eur Neurol* 1992;32(2):99-101 Subarachnoid and intramedullary cysts secondary to epidural anesthesia for gynecological surgery

Tseng SH, Lin SM *Clin Neurol Neurosurg* 1997 Dec; 99(4): 256-8 Surgical treatment of thoracic arachnoiditis with multiple subarachnoid cysts caused by epidural anesthesia.

[iv] Kamada K, Iwasaki Y, Hida K, Abe H, Isu T *No Shinkei Geka* 1993 Feb;21(2):135-140 [Syringomyelia secondary to adhesive arachnoiditis: clinical profiles and efficacy of shunt operations]

[v] Mathias CJ *Hypertension* 1991 Nov; 18(5 Suppl): III22-30 Role of sympathetic efferent nerves in blood pressure regulation and in hypertension.

Colachis SC 3d *J Am Paraplegia Soc* 1992 Jul; 15(3):171-86 Autonomic hyperreflexia with spinal cord injury.

[vi] Bayer A, Thiel HJ, Zrenner E, Paulus W, Ried S, Schmidt D *Nervenarzt* 1995 Feb; 66(2): 89-96 [Disorders of color perception and increase glare sensitivity in phenytoin and carbamazepine therapy. Ocular side effects of anticonvulsants.

[vii] Luoto S, Taimela S, Hurri H, Alaranta H *Spine* 1999 Feb 1; 24(3): 255-61 Mechanisms explaining the association between low back trouble and deficits in information processing. A controlled study with follow-up.
Kuhajda MC, Thorn BE, Klinger MR*Ann Behav Med* 1998 Winter; 20(1): 31-5 The effect of pain on memory for affective words.

[viii] Hendler NH, Bergson C, Morrison C *Psychosomatics* 1993 Dec; 34 (6): 49-501 Overlooked Physical Diagnoses in Chronic Pain Patients Involved in Litigation, Part 2.

[ix] Guyer DW, Wiltse LL, Eskay ML and Guyer BH. *Spine* 1989 Dec; 14(12): 1332-1341 The long range prognosis of arachnoiditis.

[x] Wilkinson HA *Alternative therapies for the Failed Back Syndrome* in The Adult Spine, JW Frymoyer ed.1991

[xi] British National Formulary Number 35 (March 1998)

[xii] James A. Stangl and John D. Loeser: *Current Review of Pain* 1997 1: 353-360. Intraspinal Opioid Infusion Therapy in the Treatment of Chronic Nonmalignant Pain [Review article]

[xiii] Kumar K, Nath R, Wyant GM *J Neurosurg* 1991 Sep; 75(3): 402-7 Treatment of chronic pain by epidural spinal cord stimulation: a 10-year experience.

[xiv] Meilman PW, Leibrock LG, Leong FT *Clin J Pain* 1989 Jun; 5(2): 189-93 Outcome of implanted spinal cord stimulation in the treatment of chronic pain: arachnoiditis versus single nerve root injury and mononeuropathy. Brief clinical note.

[xv] Ronzoni G, De Vecchis M, Rizzotto A, Raschi R, Cuneo L *Ann Urol (Paris)* 1988; 22(1): 31-4 [Long-term results of spinal cord electrostimulation in the treatment of micturition disorders associated with neurogenic bladder].

[xvi] O'Connor M, Brighouse D, Glynn CJ *Clin J Pain* 1990 Sep; 6(3): 240-2 Unusual complications of the treatment of chronic spinal arachnoiditis.

[xvii] El-sayed A et al *JAMA* 1999; 281:818-823 Percutaneous Electrical Nerve Stimulation for Low Back Pain. A Randomised Crossover Study.