PROGNOSIS OF ADHESIVE ARACHNOIDITIS

Introduction

As Day (2001) remarked in his review, literature concerning prognosis of arachnoiditis is scant. The seminal article by Guyer (1989) remains the most quoted authority on prognosis, based on his paper describing a group of 50 patients of whom 14 could not be followed up. The remainder were followed up for periods of up to 20 years.

Of course, there are some authors who dispute the very existence of the condition. Petty (2000) wrote scathingly about arachnoiditis, suggesting it was more fallacy than fact.

Other authors such as Wilkinson (1991), have contented themselves with stating that the condition is not progressive, although the basis for this conclusion is somewhat nebulous.

Shaw et al (1978) reported 25% progression and in the author's own experience, the majority of arachnoiditis patients who have had the condition for a number of years, consider it to be progressive, whilst those who have only recently received the diagnosis, hope that it is not.

Ways in which the syndrome appears to progress:

1.   Any underlying condition may progress
2.   Secondary musculoskeletal symptoms are likely to increase over time, particularly with deconditioning
3.   The onset of a chronic pain syndrome with autonomic effects etc.
4.   Centralisation of pain causing more widespread pain

These can all make it look as if the arachnoiditis itself is progressing whereas in fact this may not be the case. Individuals who seem to fare worst are those who mobilise in a limited way or those who require high doses of medication. This may reflect either a more severe initial level of the disease or misguided attempts to reduce symptoms by further intervention.

General patterns of symptoms:

1.   Constant

2. Fluctuating
3. Plateau
4. Deteriorating

Factors in symptom patterns based on the new database results:

1. weather especially barometric pressure; heat intolerance
2. intercurrent illnesses especially viral
3. falls/stumbles (note due to balance problems these patients have a higher risk)
4. trauma/surgery or other invasive treatment
5. stress
6. exercise

Principally, the problem lies with making a diagnosis. As the condition is regarded as rare, healthcare professionals tend not to consider it within differential diagnosis. Those few who are aware of the condition at all (the majority of doctors seem unaware of it) tend to reckon it is an anachronistic condition related to Myodil/Pantopaque and thus, since their use has been discontinued, no longer of concern.

As Day (2001) pointed out, prevention is going to be a vital aspect of future health strategies to manage this illness, but in order the achieve this, we first need a much clearer and fuller picture of the incidence and prevalence of the condition.

It is important to note that diagnosed as compared with undiagnosed patients show marked similarities of clinical presentation and more pertinently, of history, with matching causative events. Patients tend avidly to seek a diagnosis in the hope of treatment. As we know, there is no curative treatment.

Thus one might argue that a diagnosis is not the most important aspect of patient care. Nevertheless, we should pursue diagnosis diligently for three main reasons:

Firstly, on an individual level, patients may feel validated after years of being dismissed.

Secondly, although active curative treatment is not yet available, steps can be taken to manage the symptoms appropriately, including avoiding exacerbating factors such as further invasive interventions.

Thirdly, in a wider arena, we need to establish the true incidence of the condition and its prevalence within different patient communities, for example obstetric patients (who may be rendered disabled by a medical procedure having presented completely fit and well).

Recognition of cases other than those caused by oil-based myelogram dyes must be encouraged as to date, the medical profession tends to regard arachnoiditis as something of a medical dinosaur.

In many papers, single cases or a short case series are presented. Hence the evidence base is somewhat limited. However, this should not mean that doctors fail to be aware of the risk factors in a wide variety of medical specialties other than orthopaedics and neurosurgery, in patients other than 'back pain patients': such as paediatric, gynaecology, general surgery (breast cancer), obstetrics, oncology, haematology, infectious diseases, anaesthetics etc.

In other words, it behoves us to raise awareness throughout all medical fields.

Timing

Early diagnosis may be vital if we are to initiate treatment at the inflammatory stage (Aldrete 2000). Recognition and sustained follow-up of the transient conditions Transient Root Irritation (TRI), and Cauda Equina Syndrome (CES) may in fact lead us to uncover further cases of arachnoiditis.

There are papers which suggest early warning signs: Auld et al (1978) looked at 25 patients who developed spinal radiculopathy after surgery. In 7 of the patients, there was a post-operative syndrome lasting 3-20 days, involving leg spasms, radicular pain and chills. Although these resolved, all 7 went on to develop arachnoiditis.

Gutknecht (1987) wrote a letter to report a case of acute aseptic meningitis following epidural steroid injection. In this particular case, dural puncture was likely as CT scan of the brain showed air droplets in the subarachnoid space. In cases like this, one might expect longer-term sequelae.

The author has been involved for a number of years with a patient who has a similar history. She initially presented with low back pain without radiculopathy and was treated with epidural steroid injection under anaesthetic, with sacroiliac joint injection and manipulation. The first treatment was unsuccessful and it was repeated. It seems in hindsight that the second injection caused a dural puncture, because the patient went on to develop not only an epidural abscess but also meningitis, although regrettably a third injection was administered despite the patient being febrile (CT scan showed a similar picture to Gutknecht's patient). Nine years later, she

now has extensive arachnoiditis and is considerably disabled with widespread dysaesthetic pain, problems with balance, etc.

Generally speaking, the typical course of the condition tends to be one or more interchangeable phases:

1. Initial onset of symptoms: may be after a triggering event such as a fall
2. Plateau
3. Relapse/remission (known to most patients as a 'flare-up' pattern)
4. Rapid deterioration (often after a further trigger event)

Establishing a diagnosis in the early stages should not rely upon radiological findings as these might be absent or equivocal at this point. Essentially, the index of suspicion should be raised if the patient has undergone a potentially causative procedure.

Hence it is vital that we ascertain the true extent of risk of common procedures such as epidural anaesthesia and whether there is a particular group who are most at risk. There remains a great deal of research needed in this aspect of the condition and the iatrogenic origins should not deter us from undertaking this investigation.

Aetiology

As we have seen, there are a large number of somewhat diverse causes of arachnoiditis. Broadly speaking, the impact this has on the course of the condition depends on the type of causative factor:

1. Mechanical: trauma, surgery, lumbar puncture
2. Chemical: epidural injections, myelogram dyes, intraspinal chemotherapy
3. Miscellaneous: spinal haemorrhage, infection

In cases of mechanically-induced arachnoiditis, generally speaking the range of symptoms is relatively restricted to the spinal level involved. Hence we see a picture of more focal damage. Whilst there may be a chronic pain syndrome overlaid, the picture tends to be of circumscribed musculoskeletal and neuropathic pain, often in a dermatomal distribution depending on the nerve root affected.

However, in chemically-induced arachnoiditis, due to the more diffuse exposure of the arachnoid to the insult, the resultant picture is far more syndromic. This may indeed include

autoimmune conditions. It can be difficult and perhaps unnecessary (at an individual patient level) to distinguish the clinical presentation from that of a chronic pain syndrome with all the usual attendant widespread features such as autonomic, hormonal and immune dysfunction.

However, if we fail to identify cases, we will not accumulate an accurate database from which to estimate the prevalence of the condition or indeed to identify other at-risk patients.

Authors of the few papers looking at the clinical spectrum of arachnoiditis have tended to cover the most recognised causes of arachnoiditis: myelography and spinal surgery (Guyer 1989, Long 1992). Benner & Ehni (1978) looked at 68 patients, finding that most presented with leg pain, back pain and sphincter disturbance with some neurological deficits on examination, attributable to multi-level root involvement.

Benner reported an average of 3.6 procedures per patient prior to myelographic diagnosis and a further 2.2 following diagnosis. One must also note that the diagnostic myelogram itself constituted a procedure! 47% had new complaints or an exacerbation of previous symptoms.

Burton (1978) studied 100 patients with lumbosacral adhesive arachnoiditis: all had low back and leg pain exacerbated by exercise. The group averaged 3.6 spinal operations and 2.6 Pantopaque myelograms per operation.

Looking more closely at various causative factors, we can see from the literature that there are different presentations. The following examples are by no means an exhaustive review but should serve to demonstrate some recent findings:

 Post-myelogram: generally this arises after considerable delay. One might imagine that fresh cases post-myelogram with oil-based dyes (Myodil, Pantopaque, Ethiodan) would no longer appear. However, Gnanalingham et al. (2006) recently reported a case arising some 30 years post-procedure. This case involved arachnoiditis, and the complication of a thoracic syrinx and arachnoid cyst.

The presenting symptoms were chronic thoraco-lumbar pain, a spastic paraparesis and sphincter disturbance. Surgical intervention (laminectomies and shunts) led to improvement in sphincter disturbance and lower limb weakness but the authors do not comment on the pain level.

Navani et al (2006) also presented a case in which a 70 year old lady was receiving epidural steroid injections to treat degenerative disc disease. Imaging demonstrated widespread residual

iophendylate dye from a myelogram, although the patient herself was unable to recall undergoing the procedure.

Often litigation cases fail because there is a claim that arachnoiditis arose as a result of surgery, although there are cases that the author is aware of both anecdotally and in the literature in which the only risk factor for arachnoiditis was the myelography dye.

Shah et al. (2001) presented a case in which the patient had chronic back pain, radiculopathy, weakness and muscle wasting. Myelography had taken place 28 years prior to MRI demonstration of arachnoiditis with arachnoid cyst and cord tethering. The initial symptoms at the time of the myelography suggest a degree of acute inflammatory reaction as there was a febrile illness for a week and after about 1 month, onset of shooting pain in the abdomen and legs in addition to back pain. The frequency and severity of the symptoms increased over time and was noted as worse in the winter/wet weather. Most importantly, there was no history of spinal surgery, trauma, infection etc.

The author was consulted about the following case;

A 53 year old man underwent a lumbar Myodil myelogram in 1975 for cervical symptoms. At cervical laminectomy a month after the myelogram. a waxy substance was removed from the cervical subarachnoid space. The patient had no history of lumbar surgery. Post-operatively the initial presenting symptom of right shoulder pain failed to abate and in addition new symptoms of neck stiffness, numb fingers and reduced sensation over the left forehead were noted as was an oedematous left eyelid.

In 1995, he presented with a long history of low back pain with difficulty sitting/standing for prolonged periods and in getting up from sitting, with associated right leg pain. In 1996 MRI demonstrated a large spinal lipoma between T11 and L1. Arachnoiditis was reported in the 'thecal sac below'.

A CT myelogram performed in June 1996 using Iohexol via lumbar puncture, demonstrated a "considerable amount of mobile myodil" in the lumbar theca. No CSF could be withdrawn which suggests impeded CSF flow in that area.

The nerve root sheaths failed to fill with contrast and the thecal sac below L3/4 'had a featureless appearance'. Contrast medium did not flow freely into the thoracic region,

suggesting more cephaladad impairment of CSF flow, probably from diffuse arachnoiditis.

CT demonstrated "arachnoiditis…extended to the thoracolumbar junction". The conus was also noted to be adherent to the right side of the thecal sac and there was a possible cavity in the terminal part of the lumbar enlargement. [It is worth noting that retained iophendylate (Myodil) can mimic a lipoma as described by Suojanen et al 1988.]

At presentation to the author 10 years later, the patient's current symptoms included permanent stiffness in his neck, particularly making extending his neck difficult which makes drinking fluids a problem. His right shoulder was painful radiating down the arm to the hand and he was intensely sensitive to light touch.

He also had slight numbness at the tips of the fingers of his right hand (not the left) but denied any problems with grip or dropping objects. [He had apparently been diagnosed with alcohol-related peripheral neuropathy in his left hand 3 years ago by his GP].

Lower body symptoms comprised: Right buttock tenderness (including skin sensitivity), pain radiating down the lateral aspect of the right thigh to the foot. He also had a varicose ulcer on his right shin; the sole of his right foot and toes were numb. He described the pain as "soreness", "tingling" but did not describe lancinating pain. He also complained of 'cramp' in both feet, alleviated by standing up.

He was stiff in his joints but this improved as he moved about, increasing if he maintained a position for too long; hence he tended to awake 5 or 6 times a night to change position. He denied any sphincter disturbance or saddle anaesthesia/paraesthesia but suffered from constipation.

He only suffered from headaches on occasion and denied seizures or dizziness. He had decreased sensation over the right side of his scalp and around his right ear; hearing was not impaired. He described occasional problems with slight unsteadiness but did not bump into obstacles.

Although he had no change in sweating. cold weather aggravated symptoms, and he tended to improve in a warm climate. Examination revealed primarily sensory abnormality in C4-8, T1-11, L3-5 and S1 with significant neuropathic elements (allodynia, hyperaesthesia, hypoaesthesia).

Although this patient admitted excessive alcohol use in the past as an attempt to manage his pain, he had remained 'dry' for a number of years. He was using mild opiate medication as his doctor was reluctant to prescribe strong opiates bearing in mind his history of alcohol misuse. Hence this patient, as is so often the case, was being denied appropriate analgesia.

A 50 year old male invalided out of the UK services, was diagnosed (clinically) with arachnoiditis in 1991. He had undergone four myodil myelograms as well as spinal surgery in the early 1980s. He had been wheelchair bound since 1992, due to a lack of sensation in his left leg, and inability to completely weight bear through the right leg: balance difficulties had caused a number of falls when transferring from his wheelchair.

When he presented to the author in 2005, he complained of constant pain (Visual Analogue Score 8 out of 10) with 'no release' in his lower back from L4/5 radiating down the left leg to the foot and with associated pain in the left groin and hip. He described his foot feeling like a block of ice or as if he was standing on an ice skating blade. He had hyperaesthesia in his toes, allodynia, and patches of dysaesthesia.

He also complained of a band like pain in the thoracic region, to below should blades. (This is a relatively common symptom in arachnoiditis patients and resembles Multiple Sclerosis symptoms)Associated symptoms included pain down his left arm to the hand and paraesthesiae in his left hand with a weak grip. He experienced frontal headaches most days, associated with visual blurring.

He was completely unable to lie flat due to spasms in his lower back and was experiencing frequent myoclonic jerks in his left leg. He had difficulty urinating, but also urgency and high residual volumes.

He experienced profuse sweating over his back, neck and chest, as well as heat intolerance, dental problems, rashes behind ears, painful elbows, knees, hips. He also had reduced hearing in his left ear and whistling tinnitus.

There were also symptoms suggestive of hypothyroidism such as weight gain, feeling slowed up, coarse hair and skin and hoarse voice (?hypothyroid secondary to dye),

His sleep was very poor and he suffered severe depression, with marked social isolation, despite working in a voluntary capacity part-time. He reported that his quality of life had 'gone downhill'.

Examination showed he had marked increase in tone both legs but no muscle wasting. There was hair loss over the left leg in the L5 dermatome. Both feet were cool, with noticeable skin mottling, no pedal pulses were detected. He also had reduced light touch sensation overt the dorsum of the left foot. Power was 2/5 in the left leg, 3/5 in the right. There were absent knee and ankle jerks. A sensory level was detected over T9 on the right. There was also reduced light touch perception in the left arm C8 and T1 dermatomes. Sweating was noticeable and he was tender over the T7-9 and lumbar region.

Strangely, MRI scan was reported as showing no evidence of adhesive arachnoiditis. Treatment with a variety of medication was ineffectual in resolving his pain or the myoclonic jerks which became increasingly disabling.

A further case involved a 55 year old male who was diagnosed in 2004 by CT myelograms. He had undergone Myodil myelograms 25yrs previously, as well as L4/5/S1 fusion 14yrs previously. He presented with a two year history of left buttock pain with associated allodynia, paraesthesia radiating down the front of the thighs and shins to the dorsum of both feet and toes bilaterally. He had an associated tendency to lose control of his feet.

Interestingly, his past medical history included Ulcerative colitis. He was taking Oxycontin (which was causing itching), Gabapentin 1800mg a day, and Tramadol at maximum dose. He had stopped Amitriptyline due to side effects. He had poor sleep, low mood, and worries about the future, particularly relating to being able to continue work as he ran his own business and had some major financial obligations.

On Multidisciplinary review of his MRI scans, multilevel adhesive arachnoiditis was diagnosed. The pathology extended well above surgical level and radiologically showed the typical nerve root clumping and empty sac appearance.

Post-epidural steroid

A 64 year old female had been diagnosed with "most marked" lumbar arachnoiditis in 1992,

following 3 epidural steroid injections (1 of which was intrathecal) for osteoporotic pain in cervical region. She had no other precipitating factors.

Interestingly, her current diagnoses include Rheumatoid Arthritis, thyroid cysts, irritable bowel syndrome and oesophagitis. She has several autoimmune type symptoms including intermittent low-grade fevers, as well as systemic symptoms including sweating, shortness of breath, oedema. Of particular interest is that she has multiple drug allergies: to opiate drugs, anti-inflammatory drugs, penicillin etc.

Post-epidural anaesthesia: Ploteau et al (2004) reported on a case of medullar adhesive arachnoiditis occurring a mere 5 months after an epidural analgesia for repeat caesarean section. There was also a subarachnoid cyst and septated syringomyelic cavitation attributed to the preservative in the anaesthetic agent and to multiple lidocaine injections through the epidural catheter post-operatively. Unfortunately, the patient's status did not respond to surgical intervention and continues to worsen.

The author is aware of a similar, unpublished case in which a patient experienced an acute reaction to epidural anaesthesia during a caesarean section and shortly afterwards developed sphincter dysfunction and cognitive decline. She was about to be admitted to a Psychiatric ward when her husband insisted on further investigation.

She was found to have hydrocephalus and required urgent shunting. MRI has demonstrated extensive spinal arachnoiditis and the patient has ongoing chronic Cauda Equina type symptoms and a degree of lower limb paraparesis.

The only aetiological factors were the epidural immediately preceding onset of symptoms and a previous epidural during childbirth 8 years before. It may well be that the first epidural caused arachnoiditis and the second precipitated the complication of hydrocephalus.

A further case in which epidural anaesthesia is the only significant causative factor involved a 40 year old female who presented to the author in 2006 with constant low back pain worse on standing and walking, painful hips & knees, weakness in the left leg (needing to use a walker) tripping; cramps in her toes and feet; dysaesthetic pain and allodynia; bizarre sensations; pins and needles and pain in both hands. She also had urinary urgency over the previous two months, worse when cold.

She had undergone epidural anaesthesia for childbirth in 1993, but the administration was problematic due to mesh graft of burns from a childhood accident. Symptoms began to develop 4 years later, with stiffness in the joints and fatigue. She was diagnosed with cervical spondylosis in 2001.

Examination in 2006 revealed extensive burn scars over the back, neck and anterior thighs. Power was slightly reduced in the L4 myotome bilaterally. There was also reduced sensation to pinprick in the S1 dermatome bilaterally and over the dorsum of both hands. There was slight generalized increased tone in both legs, with brisk knee jerks. MRI scans demonstrated grade 2 adhesive arachnoiditis with nerve root clumping and adherence to dural sac in the Cauda Equina.

This patient admitted a history of alcohol dependency in the past as a result of trying to alleviate her pain. Fortunately this had now resolved. She was managing her pain fairly well on opiates, a non-steroidal anti-inflammatory and Pregabalin.

This case demonstrates not only the importance of recognizing the precipitating factor but also noting that the ongoing, unremitting pain can cause patients to 'self-medicate' with alcohol.

Post-meningitic: may well arise due to a combination of infection and trauma (lumbar puncture). Papavlasopoulos et al (2006) presented a case of a 30 year old man with a 3 year history progressive spastic paraparesis. He had a history of neonatal meningitis diagnosed by lumbar puncture and had begun to experience low back pain from the age of 23. Imaging revealed arachnoiditis ossificans with progressive syringomyelia and spinal arachnoid cyst. Despite surgical intervention, the patient failed to improve.

Neonatal meningitis remains a relatively common disease. De Goede et al recently (2007) reported 4 cases of myelopathy secondary to arachnoiditis as a late complication of neonatal meningitis.

One patient has E coli meningitis complicated by hydrocephalus but did well until age 8 when she had a fall which precipitated a progressive spastic quadriparesis.

Another case with hydrocephalus did well academically and in sporting activities until the age of 12 when he developed headaches, ataxia and spasticity. He was found to have thickened dura at the base of the brain and the MRI scan was abnormal throughout the spine.

A further case in a child with cerebral palsy prompted the authors to comment that in this situation, deterioration can be difficult to recognise and this late complication can easily be missed. Steinlin (1999) reported similar cases arising more than a decade after neonatal meningitis.   These cases particularly raise the issue of recognition of arachnoiditis in children.

Post-lumbar puncture: Etchepare et al. (2004) reported a case of a patient presenting with atypical inflammatory sciatica who had a history of a year of chronic low back pain. He was found to have severe vertebral stiffness and MRI scan revealed thickened L5 and S1 nerve roots. A Myelo-CT scan confirmed a diagnosis of arachnoiditis, the only precipitating factor being a traumatic lumbar puncture 17 years previously for suspected meningitis.

Post-surgical: as well as the recognised typical spinal surgery related cases, it is important to note that there are childhood cases : Wagner et al. (2002) described arachnoiditis as a consequence of closure of primary myelomeningocele.

The authors stated that the repair is 'inevitably followed by the development of arachnoiditis…' and that although the neurological symptoms may be reversible, later recurrence is a significant problem. Arachnoiditis also seems to be a complication of surgery for spinal congenital dermal sinuses (Ackerman & Menezes 2003).

In young adults who have a history of congenital spinal abnormalities, who present with low back pain, careful note of this predisposing factor must be made in order to avoid interventions that could exacerbate the condition.

Case history: A 32 year old female was referred to the pain service due to increased back and leg pain during pregnancy. It was noted only by thorough review of her medical notes that she had a diagnosis of adhesive arachnoiditis dating back 9 years.

She had been born with a complex dysraphic abnormality and had a shortened and hypoplastic left leg since birth; surgery had been performed on the foot to correct equinovarus deformity. There was also a history of recurrent pneumococcal meningitis in infancy due to CSF leak from a labyrinthine fistula; at age 2 she underwent exploration of the middle ear and closure of the CSF leak; she had myelography (Myodil in Germany; Metrizamide in UK) and then L3/4 laminectomy for tethering of spinal cord, with section of filum terminale .

Six years later, she required a calliper to her left foot to manage foot drop. She also developed

occasional problems with bladder control noted.

The diagnosis of arachnoiditis was made from MRI scan when she presented with left anterior thigh pain and numbness, with a year's history of urinary urgency. She was noted to have wasting of the left calf muscles, absent reflexes and reduced sensation in L3-5 and S1 dermatomes. MRI showed a split cord at T11-12, but no syrinx.

At the time of presentation at the pain clinic, she had an 11 year history of pain at the thoracolumbar junction at the upper end of laminectomy scar, but denied any leg pain.

She was scheduled for epidural analgesia for childbirth but bearing in mind her history, this was cancelled and she underwent uneventful general anaesthesia. Post-partum she did well and when seen again in pain clinic, was not taking regular analgesics, although she did have ongoing balance problems and tended to trip due to her left foot drop.

In this case, further deterioration from invasive spinal intervention was prevented by careful review of the history. Although the patient had relatively mild symptoms, the multiple historical risk factors of congenital abnormality, surgery, myelography and infection alerted the author to the diagnosis which was not clearly denoted but buried within the medical notes. The patient herself was unaware of the diagnosis.

This is commonly the case, patients thus being unable to alert clinicians to their diagnosis. The author saw a case in which arachnoiditis was noted on MRI but not disclosed to the patient, due to what the notes described as 'medicolegal connotations'.

The patient had undergone a Myodil myelogram in the 1970s and a sinogram to investigate facial pain in the 1980s (diagnosis then was trigeminal neuralgia). An X-ray report in 1985 noted residual dye in the sacrum as well as dye in sinuses. The patient presented in 1996 (a year after the MRI findings) with a 3 year history of increasing pain in the rectum, coccyx and right buttock, set against a background of a 12 year history of pain since a fall in 1984 onto the right buttock and lateral thigh.

An epidural steroid injection was administered without any benefit. Within the notes mention was made of various other diagnoses such as seronegative Rheumatoid Arthritis, Osteoarthritis, and Sjogren's. She was noted to have a number of different medical problems such as severe palpitations, gallstones, gastric erosions, inguinal herniorraphy, coccydynia, recurrent urinary

tract infections, urge incontinence, constipation, tenesmus.

She also suffered from Benign positional Vertigo for 5 years which was attributed to labyrinthitis. At her presentation in 2006, the author discussed her case with radiology colleagues who confirmed arachnoiditis on MRI, as well as spondylolisthesis, mild canal stenosis and lateral recess stenosis. The patient remained unaware of the diagnosis of arachnoiditis.

There are also isolated cases of miscellaneous causes, within the medical literature e.g. Cauda Equina syndrome complicating Ankylosing Spondylitis (Lan et al 2007), cervical osteomyelitis (Rajpal et al. 2007), brachial plexus avulsion (Sindou et al 2005).

Case history: a 44 year old male teacher diagnosed with acromegaly secondary to a large pituitary tumour in 2001 underwent surgical removal of the tumour in 2002. Unfortunately he developed a CSF leak for which a lumbar drain was inserted for one week. On its removal the patient experienced severe pains in the abdomen and legs, severe headache, nausea, diarrhoea, backache, pain and stiffness in the neck.

Four weeks later, he began complaining of pain in the legs radiating to the foot associated with a 'cold feeling' and paraesthesia which have persisted since then.

MRI scan in 2003 led to diagnosis of adhesive arachnoiditis in the cauda equina. In 2004 he was treated with epidural steroid injections and also underwent radiotherapy. He developed neck pain and headaches and when he presented to the author in 2004, had severe pain going up the right leg from foot to buttock and debilitating headaches probably due to low CSF pressure as they were positional, worse lying down, with increased pain in the temples, and better when 'hanging head over end of bed'.

Despite seeing a number of specialists, the exact cause of his headaches was not established and a variety of pharmacological strategies did not relieve his symptoms. He was finding it increasingly difficult to work due to being unable to drive or travel any significant distance.

Types of arachnoiditis: a varying picture

Burton has consistently reiterated that one should distinguish between arachnoiditis, which is a subclinical entity that has a relatively high incidence and is to be expected after surgery etc., and adhesive arachnoiditis which is the clinically significant form of the disease. One may assume that the majority of cases in the literature are in fact adhesive arachnoiditis as the

authors tend to describe the clinical symptoms and signs.

As already discussed, there is a spectrum of pathological damage and to some extent, one can argue that the prognosis will vary accordingly. This may however be an entirely erroneous assumption given the variability in the cases within the literature and also in the author's clinical experience. There seems, in fact, highly unpredictable correlation between pathological disease and symptomatology. This is, of course, not unique to arachnoiditis.

Nevertheless, within the author's experience, there do seem to be 2 discrete types as described previously: mechnically-induced and chemically-induced.

The author's Global Survey in 1999 of 317 patients found that there were a number of patients with particularly syndromic picture involving new autoimmune conditions. Virtually without exception, these patients had been exposed to a chemical insult to the arachnoid.

There seemed to be a correlation between the severity and frequency of the exposure and the extent of the comorbidities in that the greater the overall toxic load, the greater the clinical impact.

Case histories:

A 50 year old female diagnosed arachnoiditis more than 10 years previously had a history of four spinal surgeries, 6 Pantopaque myelograms, 2 Omnipaque myelograms.   She had been treated with an Intraspinal pump for 10 years. Current diagnoses include: Lupus, Sjogren's, Autoimmune hearing loss, Haemolytic anaemia, MS and possible Diabetes mellitus. She has general systemic symptoms and multiple allergies including rare ones such as albumin.

A second case involves 67 year old female who was diagnosed in 1990 with lumbar and cervical arachnoiditis. She had a history of at least 3 Pantopaque myelograms and 4 epidural steroid injections. She had also had Herpes Zoster in the past and an episode of acute renal failure secondary to calculi. Other medical problems have included recurrent gastric and duodenal ulcers necessitating gastrectomy, which resulted in Dumping Syndrome, then corrected by further surgery.

She has diagnoses of Sjogren's, Rheumatoid Arthritis, Raynaud's, Horner's syndrome, Iritis and Uveitis and Osteoporosis, as well as mild allergies. She also has continued gastric problems, mild congestive heart failure and osteoporosis. She continues to suffer from a very wide range of symptoms, throughout the body.

Arachnoiditis Ossificans

Arachnoiditis ossificans seems to be a more severe form of the disease. However, it appears mostly as isolated case reports in the literature.

Slavin et al (1999) described a case presenting with a 9 year history of progressive spastic paraparesis and a T8 sensory level. MRI demonstrated a large syrinx but no cause was elucidated. Unfortunately she gradually deteriorated over the ensuing few years of conservative treatment although radiologically the syrinx appeared unchanged. Eventually she underwent surgery but this was not beneficial. Immediately post-operatively the paresis worsened although this did resolve somewhat by 6 months post-op. The sensory impairment and pain were however persistent.

Mello et al (2001) reported 3 cases. The first initially presented with a 5 year history of back pain and more latterly, difficulty walking, with anterior and posterior thoracic pain. She underwent Pantopaque myelography which revealed an intradural block and subsequently underwent laminectomy at T3-5. Calcium plaques were seen at operation. The patient unfortunately deteriorated after the operation, suffering spastic paraplegia with sphincter incontinence, but pain had resolved.

Over the course of a few months, this picture reverse, with some resolution of the neurological deficit, and she was able to walk a year later. Within 4 years, the back and leg pain resumed and sphincter disturbance recurred. At a second operation, there were extensive arachnoid adhesions but no plaques. Despite resection, there was no recovery although pain was tolerable without medication. However, since a fall and fractured femur, the patient has been wheelchair bound.

The second case failed to show sustained improvement after surgery. Interestingly, the third case developed ossification of the arachnoid following 3 lumbar punctures and meningitis. Again, surgical intervention failed to improve her clinical picture.

In contrast, Faure's case (2002) was first symptomatic after a road traffic accident that led to brain injury and an L4 burst fracture with neurological damage. He required 2 surgical interventions and had scar sepsis. He improved considerably with almost complete resolution of the neurological damage, his only persistent symptoms being intermittent lumbar backache and moderate sciatica. 3 years later, the back pain was more severe and constant and sciatica had become bilateral due to a septic pseudarthrosis.

Again, surgery was very beneficial. Seven years on, he complained of backache and intermittent left limb weakness. His manual labour occupation was exacerbating the symptoms. After a further 9 years when he had recurrent low back pain and bilateral sciatica and CT scan revealed extensive lumbar intradural ossifications. However, the symptoms remained relatively mild and responded to conservative treatment such that he remained pain free 3 years later.

Matching clinical presentation with pathology.

We can see from the diverse clinical pictures in these various cases, that it can be very difficult to match the pathology with the clinical presentation, not least because there is no 'classical' presentation, as Jorgensen et al. (1975) and Long (1992) remarked.

Burton (1996) described typically chronic severe back and/or lower extremity/leg pain, of "a constant and burning nature" usually with "a poorly localised, paleospinothalamic pain pattern that is diffuse in nature". However, this is not exclusive to arachnoiditis.

As already mentioned, there is little discrepancy between diagnosed and undiagnosed cases where there is strong circumstantial evidence of matching aetiological factors (Day 2001).

Mooij (1980) describing 63 patients, suggested that new or typical symptoms of arachnoiditis did not occur and emphasised that most patients had the symptoms before arachnoiditis, only 20% developing new signs or symptoms from cauda equina arachnoiditis.

Long's (1992) series of 321 patients had almost universal back pain (94%) and 81% leg pain. Some degree of 'motion impairment' was present in 91% and what Long described as chronic muscle contractions secondary to surgery in 94%. 81% had sensory loss and almost three quarters (74%). Reflex changes were also almost universal (96%) but again this is not unique to arachnoiditis.

Long considered that only 1.8% of the patients had a progressive form of the condition. Functional status was also assessed: 84% walked alone, 15% walked with aids, 1.2% were wheelchair bound. Overall, the majority (92%) complained of claudication with limitation of the distance they could walk. Sexual function was severely impaired. Only 8% described had no problems; 45% were able to perform sexually with some impairment whilst 47% described severe impairment of sexual function.

As regards occupation, only 11% were working full-time and a further 37% were working part-time or with limitations and 167 were retired secondary to back complaints (but could not be directly attributed to arachnoiditis in most). In terms of driving, 82% drove without limitation; 12% were able to drive an automobile with aids; only 5.6% were unable to drive.

Quiles et al (1978) described 38 patients with lumbar arachnoiditis of whom 31 had had spinal surgery. However, in 7 the causative factors seem to have been myelography, spinal anaesthetic, serious trauma and epidural haematoma. In 2 there was no known cause. The authors remarked on the diversity of presentations and could not identify a uniform clinical picture.

Authors such as Brodsky (1978), studying cauda equina arachnoiditis, have the opinion that there is little correlation between radiological appearance and clinical presentation. However, Brodsky was reporting on myelogram findings not MRI imaging. Only 1 of the 93 patients he studied, had what he considered to be the 'classic severely disabling paraparesis, intractable pain, and loss of bowel and bladder functions.'

A contemporaneous paper by Shaw et al (1978) reviewing 80 cases, suggested that the lumbar region was most commonly affected and that this accounted for the persisting radicular symptoms with relatively low incidence of paraplegia. The authors noted that in 16 patients, symptoms developed within a month, in 23 within a year, in 21 between 1 and 5 years and in the other 14, after 5 years.

They also emphasised a shift in aetiology from infection and bleeding to myelography and surgery. Nowadays, infection remains a significant causative factor in the developing world, whereas surgery and intraspinal injections remain the most common factors in most developed countries.

Roca et al. (1993) found that patients with Type II and III myelographic appearances had bilateral leg pain and neurological deficit, and those with Type IV had dysaesthesia. More recent understanding of the nature of neuropathic pain suggests that the majority of arachnoiditis patients have dysaesthetic neuropathic pain, regardless of the degree of arachnoiditis demonstrated radiologically.

Warnke et al (2003, 2007) have recently found during thecaloscopy that there may be significant arachnoiditis not seen on MRI scan.

This is further complicated by other factors. Patients may present with both direct effects of arachnoiditis (which one can attribute to the site and degree of pathology) and indirect (mostly as a result of chronic neuropathic pain). There are similarities with other conditions, notably Multiple Sclerosis and Fibromyalgia, both of which are often mooted as possible or even probable diagnoses.

Chronic pain syndrome (or Intractable Pain as described by Dr Forrest Tennant in his Internet website) carries its own burden of symptoms which are now well recognised at least amongst algologists if not general medical practitioners. We know that the extent of the patient's suffering has little to do with the original pathology (Turk 1998) and that chronic pain is an illness of the whole person (American Medical Association).

Aside from the very few individuals such as Petty (2000) who seek to deny the condition exists at all, most clinicians who have seen arachnoiditis accept that it causes severe pain, mostly neuropathic in nature.

This type of pain is acknowledged by pain specialists to be particularly resistant to treatment. The phenomenon of central sensitisation, which is not particular to arachnoiditis, is a common feature, perhaps by virtue of the area of the body that is damaged: with central nervous system tissue being compromised, deleterious effects are to be expected. These occur at a much earlier stage than has been recognised in the past. The plasticity of the nervous system is now an established fact. In arachnoiditis, unfortunately, this can lead to early onset of symptoms, (cases of 'acute arachnoiditis').

Unfortunately, these tend not to be followed up by the teams that report the cases. We do not know if there is a later resurgence of symptoms or whether they resolve and remain in remission.

We do however, often see cases of delayed onset, maybe up to 30 years after the causative event (Oil-based dye cases). The likelihood is that in these patients, the body defended itself by encysting the chemical but at a later date the cysts are disrupted (e.g. by a fall), exposing the delicate arachnoid again to the toxic material.

Benner & Ehni (1978) looking at 36 patients, found that 19 had a short interval (3-10 months) from the time of spinal surgery to the time of the study (when they were diagnosed) , 8 had intermediate interval (18-36 months) and 9 long interval (5 years).

These delayed cases may well be missed because the link to a procedure some decades ago is not made either by the patient or the attending physician.

Precipitating factors for symptoms:

Wilkinson, whilst suggesting that cases of arachnoiditis are becoming less common overall,

remarked in a letter (2003) that there is a striking variability in clinical presentation and that patients often remain relatively asymptomatic for many years, then becoming acutely symptomatic following relatively innocuous trauma to the back.

De Goede et al. (2007) have also remarked that in their cases of arachnoiditis following neonatal meningitis, neurological deterioration arose after a fall in 2 cases and was exacerbated by spinal surgery in the other 2 cases. Steinlin et al (1999) have also noted that in one of their cases deterioration occurred after a fall and in another, after a hypotensive episode during surgery.

A COFWA (online USA-based support group) member, David Gaub, ran a small survey in late 1998, to assess the impact of arachnoiditis on walking. 49 COFWA members took part, the majority of whom had undergone multiple spinal insults.

On average, they had begun to experience sustained walking difficulties 12 years after their initial back problems. Mr. Gaub felt that the most significant finding of the survey was that most respondents developed walking difficulties after an incident such as a fall, auto accident, further surgery or meningitis. In some cases, the incidents were relatively minor and the resultant loss of function could not be directly attributed to damage from the incident itself.

However, 31 of them described a rapid deterioration since the incident. 38 of the respondents reported using an assistive device such as a cane or a walker, 10 used a motorised scooter or a wheelchair most of the time. They noted problems with fatigue, relationship issues and limits of socialisation.

Comorbidity

One of the most significant issues when attempting to define long-term prognosis is the contribution of other factors such as ageing and deconditioning (Guyer 1989).

Not all ongoing symptoms experienced by patients can or should be attributed directly to arachnoiditis. It remains unclear what link the condition has with autoimmune conditions and indeed other diseases.

The development of sphincter disturbance, urinary and bowel problems etc. was thought by Guyer (1989) not to be necessarily directly related to the pathology of the arachnoiditis. Whilst it

is true that chronic pain syndrome may incorporate a number of autonomic symptoms of this type, the symptoms experienced by those patients with arachnoiditis seem more profound and resemble a chronic cauda equina syndrome.

In some cases, they can be explained by the pathology, in others, they may represent one of the secondary features of the condition.

Guyer noted that 23% of his patients had urinary symptoms that could not be attributed to anything other than arachnoiditis and that these tended to develop late.

Case histories:

A 51 year old female had a longstanding diagnosis of arachnoiditis as well as Ankylosing Spondylitis and Tarlov cysts. She had undergone four spinal surgical interventions, 6 Pantopaque myelograms and 1 Metrizamide myelogram.

She had also had 2 spinal anaesthetics and 1 epidural steroid injection. She suffers from diffuse pain in most of the body, muscle cramps, weakness and stiffness. She has autonomic effects such as sweating increase and swelling. She also has a number of autoimmune features such as low-grade fever, raised ESR/WCC, enlarged lymph nodes, joint pains, bruising, fatigue, skin rashes (including cellulitis) and recently had pancreatitis (possibly autoimmune), with complete stenosis of the common bile duct (possibly primary sclerosing cholangitis) .

A 61 year old female was diagnosed with "Paraspinal multilevel denervation" S1-T10 in 1980, prior to surgery in 1981. She had undergone a Pantopaque myelogram in 1965 and had also had 2 other myelograms (one Metrizamide) 2 discograms and numerous epidural steroid injections. She had a longstanding diagnosis of arachnoiditis.

She also had cholecystitis (cholecystectomy 3 years ago) and pelvic infections plus endometriosis (requiring hysterectomy). Her symptoms were typical with pain throughout the body, the usual sensory and motor features plus autonomic (sweating, oedema) she also had various new allergies e.g. to pollen/dust etc. and one new drug allergy. She suffered from frequent eye inflammations. She also had recurrent oesophageal reflux and chest pain.

A 54 year old female was diagnosed with arachnoiditis in 1979 (intraoperatively) following Dimer-X and Amipaque myelograms. Her current diagnoses now include: Polyendocrine

Autoimmune Syndrome (Hypothyroidism, Pernicious Anaemia, premature menopause) also Vitiligo and osteonecrosis of femora and tibia, possibly Vasculitic/ neuropathic.

A number of cases of thyroid disorder have been seen in arachnoiditis patients, particularly those who have had myelogram, probably due to the iodine in the dye. These include Graves' Disease, Thyroiditis, Hypothyroidism, Thyroid tumour, Thyroid cysts, Lymphoma (Global survey 1999 as cited by Day 2001).

In some patients, there may be serious or even life-threatening comorbidity. A 63 year old man presented to the author in 2004 with a variety of symptoms of arachnoiditis. His history included a Myodil myelogram and two spinal surgeries in the 1960s. A thoracic spine X-ray in 1999 showed minor scoliosis but also a dense droplet at T10/11 suggestive of retained myodil.

In 1994, he developed upper lumbar pain radiating to the right hip and was treated with manipulation under anaesthetic and epidural steroid injections. X-ray at the time showed widespread osteoarthritis of the lumbar spine with osteophytes.

MRI scan in 1998 showed arachnoiditis below the level of L2/3 with nerve roots bunched and adherent to each other and at L5/S1 an empty sac appearance.

In 2002 he was investigated for loin pain and difficulty with micturition.

On presentation in 2004, he had a wide variety of arachnoiditis symptoms but retained a stoical and even cheerful outlook. He reported numbness in the legs associated with crawling sensations 'like worms' and intermittent change of temperature in the feet, with associated swelling, mottling and redness. He described the common symptom of
 'walking on broken glass' and complained of frequent cramps in his hands, shoulders, and feet (tonic spasms); he also had the typical burning feet.

However, the symptom of most concern to him was chest pain radiating to his left arm and jaw (ECG excluded a cardiac cause). At the time these symptoms had been attributed by his general practitioner to oesophagitis. It seemed unlikely that these upper gastrointestinal symptoms were related to arachnoiditis although there was a possibility of gastroparesis. However he was referred to a Gastroenterologist. Investigations proved normal.

In early 2005 he had perineal discomfort, urinary hesitancy; incomplete bladder emptying and was found to have unexplained microscopic haematuria.

Within a few months, he had an episode of hameoptysis and was fast-tracked through the chest clinic where investigations revealed large matted supraclavicular nodes, inguinal nodes and medial axillary nodes.

Diagnosis of Metastatic small cell carcinoma was made (note he had a history of smoking). There was a large volume of mediastinal disease which did not respond to chemotherapy. He was given Methadone for pain relief; he was very sensitive to pain medication. After palliative radiotherapy he developed numbness in the right face and his tongue deviated to the right. He developed liver involvement and died shortly afterwards.

One of the most important points raised by this case is that one must beware of missing other, life-threatening conditions which may present against a background of arachnoiditis.

Morbidity due to Complications of Arachnoiditis

Koyanagi et al. (2005) looked at the clinical features of syringomyelia associated with arachnoiditis in 15 patients over an 18 year period. The patients all presented with paraparesis or tetraparesis, of variable duration ranging from 1 to 33 years. 5 patients showed acute onset followed by gradual deterioration whereas the other 10 had developed paresis more gradually.

In 6, neurologic deterioration appeared 3 to 19 years after meningitis or spinal surgery.

All patients were treated surgically with shunts, 8 requiring repeat shunts. 9 patients showed neurologic improvement, one remained stable and 5 gradually deteriorated. Of interest, in 6 patients, tuberculous meningitis was the cause, 3 had unspecified meningitis, 2 had spinal surgery and 4 were unknown.

Ohata et al. (2001) described a case in Japan of a 47 year old man with a 6 year progressive history of numbness in the left hand gradually extending to all extremities and 6 months prior to presentation, onset of urinary dysfunction. A syrinx was found between C2 and T2 and an absent arachnoid space suggested arachnoiditis. The only causative factor was tuberculous

meningitis 28 years previously.

The author treated a 62 year old female with a history of low back pain since the 1970s had undergone two decompressive spinal surgeries and two myodil myelograms. Her leg pain had never improved. In 2001 there was a sudden onset of symptoms (although she had had progressive numbness in legs over the preceding year) of low back pain radiating to the right flank, with associated difficulty walking and numbness from the feet to the umbilicus. There was also urinary and faecal incontinence.

Examination revealed flaccid right lower limb, hyper-reflexic left lower limb and a sensory level just above the umbilicus. MRI demonstrated extensive syrinx and arachnoiditis. The patient underwent subarachnoid shunt which improved motor function but increased perineal numbness. Eight weeks later she had increased pain, numbness, and reduced right leg function. MRI showed reaccumulation of the syrinx due to a blocked shunt. She underwent a syringo-pleural shunt at T11.

By 2002 she had increased weakness in the left leg, with recurrence of neuropathic pain on the right. MRI revealed that the syrinx had collapsed below the level of the shunt tubing but was larger rostrally. A local neurosurgeon noted that even if the syrinx collapsed, pain and sensory disturbances tend to persist, commenting 'myodil-induced arachnoidal fibrosis…is unfortunately frequently extensive' making options such as Lumboperitoneal shunt or /CSF conduit) fraught with potential problems. He suggested that '50% of cases deteriorate inexorably'.

By 2004 she developed severe painful spasms lower half of body; initially responsive to Baclofen, although later these became refractory and a major source of distress to her as well as affecting her function.

In 2005 when she was first seen by the author, she had been admitted to a general ward with an acute chest infection, confused, delirious and completely immobile. Once she made a partial recover, she reported that she had migraines which initiated painful muscle spasms in her chest.

She also complained of cramps in both legs, burning from waist down; intermittent stabbing pain and a large patch allodynia over the left side of her back. She described a sensation of her perineum 'dropping' and unfortunately was not self-catheterising as had been advised. The faecal incontinence was also not being adequately managed. She was incidentally found to

have acute calcific tendonitis in both shoulders.

Unsurprisingly, as result of her increasing symptoms, she was very low in mood, having had to give up her teaching job which she had managed until a few months prior to the admission.

In 2006 her mood declined further, mostly due to her increasing disability. She was virtually wheelchair bound, although persisted in trying to ambulate around the house and therefore falling frequently. She also had blurred vision which compounded her problems. There was a normal ophthalmic examination so the blurred vision may well have been secondary to her polypharmacy.

She also had recurrent urinary tract infections due to residual urine of over 350 mls, which was probably contributing to her spasms (as in spinal cord injury patients). The spasms were unresponsive to Baclofen or Magnesium and she was on high doses of Oxycontin in a bid to control her pain, she was admitted in 2007 to the Hospice for respite care.

Morbidity related to Treatments

The treatment of any chronic non-malignant pain can itself cause significant morbidity. Severe neuropathic pain such as that caused by arachnoiditis tends to require a cocktail of pharmacological treatments and often patients seek help from all quarters, allowing themselves to be subjected to treatments for which there is scant evidence of effectiveness nor indeed safety.

In the 1999 Global survey, only 3% of respondents were not using medication regularly.

In the New Zealand survey (as cited by Day 2001), the figure was 20%. 12% found no treatment effective, 24% were on drug therapy alone, 44% were on drugs and other treatments, 20% were only on non-pharmacological treatments. Those on medication were on between 1 and 7 different drugs.

In Long's survey (1992), 14% were taking no drugs of any kind. From the author's experiences, this might result from patients finding either that medication was ineffective and/or that they could not tolerate the adverse effects. Generally, arachnoiditis patients are highly sensitive to medication and some develop multiple drug allergies.

19% of Long's patients were using non-narcotic analgesics (often in large quantities). One patient sustained severe kidney damage from Propoxyphene and two patients were found to have significant hepatic damage from the use of non-steroidal anti-inflammatory drugs. 52% used narcotics on a daily basis and a significant minority (14%) were reported as showing evidence of abuse and drug seeking behaviour.

This might in fact have included cases of pseudoaddiction where patients are receiving inadequate opiate doses. In addition, it is unlikely that these patients had been taught self-management techniques and been given a realistic explanation of the likely level of pain relief. A high percentage (81%) regularly used psychotropic drugs such as diazepam.

The Cochrane review on opioid treatment of neuropathic pain (Eisenberg et al 2006) found that although short-term studies provide only equivocal evidence about the efficacy of opioids in reducing the intensity of neuropathic pain, intermediate-term studies demonstrate significant efficacy as compared with placebo, which is likely to be clinically important.

Opioids are known to cause common adverse events, which can be burdensome to the patient, particularly chronic constipation which can act synergistically with the pathological effects of arachnoiditis. Fortunately these are not life threatening.

As the Cochrane authors pointed out, we need further randomized controlled trials to establish long-term efficacy, safety (including addiction potential), and, most importantly, effects on quality of life.

The adverse effects of analgesic medication, particularly anticonvulsants, including weight gain, demotivation, cognitive blurring, etc. can add considerably to the overall physical impairment and effects on balance can heighten the risk of falls in patients in whom balance is already impaired, and hence increase the risk of an event that can precipitate rapid deterioration. (Wilkinson 2003, De Goede 2007, Steinlin 1999).

Surgical treatment:

Johnston & Matheny (1978) suggested that progression is not the natural course of arachnoiditis, and that if it occurs it is usually due to subsequent surgical procedures. Guyer (1989) asserted,

"Surgery is not the answer, and carries a risk of increasing neurological deficit."

Wilkinson & Schuman (1979), however, advocated 'more frequent intradural exploration' to ascertain cases of spontaneous arachnoiditis mimicking lumbar disc disease. Their series of 17 patients drawn from a series of 681 being treated for lumbar disc disease, demonstrated multisegmental arachnoiditis in 5, with annular or subtotal adhesions in 12.

All the patients presented with severe pain and 14 had varying degrees of neurological dysfunction. Lysis of the adhesions did provide good pain relief and some improvement in neurological function within the first year although this declined at further follow-up.

Dolan (1993) studying 41 patients, asserted that once adhesive arachnoiditis is established 'intradural operation offers the best hope of at least partial relief'. However, he did acknowledge that this is a contentious viewpoint and suggested that other pain relieving strategies should first be tried.

Whilst he admits that undoubtedly adhesions 'can and do reform', in what he calls an 'appreciable number of cases' there is a 'marked' improvement. He does however concede that a definite percentage are not improved and become depressed, one of the study group having committed suicide whilst a further two or three had suicidal intentions. Of the 7 patients designated as having a 'poor' result, they were all depressed and some had gross neurological defects.

Even the 13 'good' results were unable to return to regular work and required regular medication. The length of follow-up was not specified.

The following cases illustrate how treatment for the pain of arachnoiditis can itself precipitate further morbidity:

A 60 year old female who had been diagnosed with arachnoiditis for over 10 years gave a history of 4 surgeries (including spinal fusion stimulator), 4 myelograms (Omnipaque), several diagnostic nerve blocks, discogram, and 3 epidural steroid injections. She was tried on an intrathecal morphine pump, but this had to be removed due to "blockage at L3-4-5" (due to

arachnoiditis) producing intolerable pain.

Notably now her current diagnoses include: Fibromyalgia, Sjogren's, Myofascial syndrome, also spinal stenosis. She has had an episode of "chemical hepatitis" due to the medication she was taking. She still suffers from widespread pain and various other symptoms.

In the second case, a man in his 60s who developed symptoms of arachnoiditis after treatment with epidural steroid injections for mechanical low back pain. He was given a clinical diagnosis initially and in 1998 a Dorsal Column Stimulator implanted, but relief from this was short-lived and a number of procedures were carried out to reposition the leads.

He also sustained a fall which dislodged the leads. Around this time, the patient was having thalamic infarcts and demyelination had been noticed within the brain.

In 2001, an intrathecal morphine pump was implanted. Unfortunately this lead to a number of problems, including bilateral leg oedema, and then a granuloma at the catheter tip. The catheter had to be resited on more than one occasion.

A number of different solutions containing a variety of medications were tried but failed to provide reasonable analgesia. The patient's quality of life was further reduced by ongoing thalamic infarcts the symptoms of which were difficult to distinguish from the effects of the pump mixtures.

At one point, the patient was told that he categorically did not have arachnoiditis, but had Multiple Sclerosis and that his thalamus was severely affected, but later the diagnosis of arachnoiditis was re-established. It is unclear as to the cause of his thalamic lesions, but it is obvious that he has consistently deteriorated over the course of the past few years and that his invasive treatments have been unhelpful and possibly harmful. That said, aside perhaps from Deep Brain Stimulation, there are virtually no other therapeutic options for thalamic pain.

Conclusion:

A thesis by Christine Hopkins, a New Zealand nurse, in 1998, details the experiences of 11

people with arachnoiditis. Only one had no pain, the others having a variety of pain sites and types. In addition to the severe, unremitting pain and physical impairment, the interviewees described the emotional impact of the condition.

Overall this is a tale of high levels of disability and pain causing widespread effects on quality of life. In particular, Hopkins noted how isolated the participants were. Whilst this is not peculiar to arachnoiditis patients, isolation in arachnoiditis is compounded by the patient encountering scant medical awareness of the condition and a generalised poor understanding of what is perceived as a 'back problem' whereas it is in fact a neuropathic pain syndrome.

Furthermore, the iatrogenic origins in most cases can heighten the patient's perception of being a victim.

Janet Kraal has written movingly about her story in "Released from the Web". She comments:

"It's hard to accept a condition which gives pain twenty-four hours a day. It's taxing on you mentally and one becomes exhausted with the pain, trying to do what may be the simplest of tasks. We are advised to try not to dwell on the pain, not to brood, to concentrate on other things, but when the pain is overwhelming one simply cannot be distracted or think of anything else...We feel trapped and helpless, we feel miserable, tense, angry and irritable."

Bourne (1990) commented:

"The relentless and progressive pain syndrome of arachnoiditis is taxing to the patient's morale. In many instances doctors, relatives and friends fail to realise that the pain can be as bad as terminal cancer, without the prospect of death to end the suffering. Well-meaning enquiries as to whether there is any improvement with the implication that there must inevitably be improvement...are distressing to the patient.

There are sympathetic doctors, relatives and friends who expect the patient to be brave, stoical and cheerful. In the end the patient yearns for less exhortation and more compassion. Compassion is an important consequence of comprehension of the existence and nature of

arachnoiditis."

Bourne, whose wife suffered from arachnoiditis, remarked that his paper was not aimed at specialists who are already aware of arachnoiditis (radiologists, neurologists and neurosurgeons) but at others such as rheumatologists and general practitioners, in order that in patients with 'seemingly bizarre symptoms and signs' the doctors might reconsider the diagnosis.

He further commented that it was to be hoped that this 'distressing iatrogenic disease' would be 'brought to public conscience'.

A variety of media articles (Daily Mail etc.) have investigated the links between epidural anaesthesia and neurological damage, to generalised outcry by anaesthetists. The problem with this format is that it is, by its nature, sensationalist and thus fails to adequately explore the complexities of the situation. The author doubts that there has been any significant benefit from these media exposés aside, perhaps, from a few individuals being made aware that they may suffer a condition that cannot be cured.

As such, although the author is far from deploring this avenue for raising awareness, she remains sceptical of its benefits.

Sadly, however, medical literature fares little better in terms of raising awareness. In these days of 'Evidence-based Medicine' (EBM) the typical arachnoiditis literature, which tends to comprise case reports and occasional case series, fails to attract medical attention. Furthermore, the average practitioner can scarcely keep pace with developments within his specialty, let alone explore other areas of medicine.

Policy makers are similarly unimpressed by the low level of evidence base for arachnoiditis. It simply does not have the figures to generate either research or indeed, a move to prevent future cases.

Nevertheless, as Dr. Charles Burton wrote in 1999,

"The subject of adhesive arachnoiditis is still something no one really seems to hear anything about. They will, I assure you, continue to hear about it because it is still a clear and present health affliction and we will do our best to continue to make the public aware of this condition and hopefully, to bring some recognition and respect to sufferers."

Dr Sarah Fox MB BS PGDip Pain Man- 2011

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