Suspecting and Diagnosing Arachnoiditis

A review of the symptoms noted in a group of patients with arachnoiditis presents an analysis of clinical observations of this disease.
By J. Antonio Aldrete, MD, MS

[1]Arachnoiditis (ARC) is a nonspecific inflammatory process usually caused by an invasion into the dural sac—whether by bacteria, blood, or injections of various irritant substances that produce a spectrum of pathological changes originating on the arachnoid membrane. The resulting inflammatory response eventually proliferates to other intrathecal neural elements leading to fibrosis and adhesions that involve the nerve roots, the arachnoid, the spinal cord, and the dura mater. This progression culminates in permanent disability characterized by severe intractable pain, neurological deficit, and other related symptoms.[1] Although this process may involve any site where the arachnoid membrane and neural structures are found, the most common presentation occurs in the lumbosacral region affecting not only the distal spinal cord, but also the cauda equina's nerve root and the sympathetic nerve fibers connected to them.,[2] In addition to pain sensory alteration and motor deficits, it may also be accompanied by other symptoms such as low-grade fever, profuse diaphoresis, heat intolerance, nausea, bladder and/or bowel dysfunction, and sexual impairment. Evidently, this syndrome is, by far, more complex than the involvement of the meningeal layers. Whereas the main clinical features represent a physical injury producing functional damage or alteration, the ensuing devastating disease is always associated with unrelenting chronic pain and major psychological and social impacts.

Due to the scarcity of objective data regarding the correct diagnosis of arachnoiditis, the author and a group of collaborators (see acknowledgements) associated with the author's clinical facilities have, over a period sixteen years, tabulated comprehensive data on arachnoiditis patients. The data was obtained from the clinical history from all the medical records available, patient physical and neurological exams, and a review all the available films in 489 patients in whom the diagnosis of arachnoiditis had been suspected or affirmed by other clinicians. All patients were seen personally by the author from 1989 to 2006. A good number of patients were refer to the author's pain management facilities for diagnosis and treatment, with most patients coming from distant states or from other countries. Many other patients came of their own accord after they developed symptoms resembling arachnoiditis following a medically-related event that included incidental invasion of the vertebral canal. In the last decade, by far, the largest number of patients have also received, at one time, the diagnosis of Failed Back

Syndrome (FBS). As a matter of clarification, it is important to note that not all the patients having arachnoiditis had FBS and not all the patients with FBS had ARC.

Historical Background

After Charcot and Joffroy[3] first described a syndrome resembling ARC in 1869, other series of cases caused by infectious diseases such as tuberculosis,[4] syphilis,[5] parasitic,[6] or fungal organisms[7] appeared in the medical literature nearly 150 years ago. In 1909, the british neurosurgeon Victor Horseley[8] wrote a classic description of the clinical symptoms, together with operative and postmortem findings in patients with arachnoradiculitis. This classical description was followed by Foix and Alajouanine[9] who, in 1926, correlated the symptoms with the location in the spinal cord and nerve root pathological findings. In 1930, Vincent et al.[10] described the first non-infective cases of ARC related to the introduction and increased popularity of myelography as a diagnostic test in the 1930's. However, it was not until two decades later[11] that the alarm was sounded about the possibility of pantopaque and other oil-soluble contrast media[12,13] producing permanent neurological deficit. This feasibility was initially doubted since many of the patients having myelograms either had an ongoing neurological disease[14] or had spinal operations[15] soon thereafter, so it was therefore difficult to discern the origin of subsequent neurological deterioration or to define the onset of new symptoms. This dilemma was aggravated by the fact that the only diagnostic procedure available to confirm the presence of ARC, at that time, was to perform another myelogram. Even when tomography became available, intradural sac pathology could only be defined when oil-soluble or some of the early water-soluble contrast media were injected intrathecally.[16] Although the use of oil-based dyes had been discontinued in Scandinavian countries in 1965, and despite multiple reports of severe arachnoiditis following the intrathecal injection of iophendylate and other oil-soluble dyes, the FDA did not stop its sale in the USA until 1986.

Possible Etiology of Arachnoiditis

Recognizing that arachnoiditis of the lumbosacral spine can also occasionally be caused by infections like meningitis, viruses like HIV, and parasites like echinococcus, readers are referred to a review on that subject.[2] This series include a group of patients that apparently acquired this disease while under going diagnostic or therapeutic procedures of the spine. Efforts to identify the possible causative factor(s) were made by reviewing all the pertinent medical records and correlating the radiological findings from the time of appearance of the low back pain. All subsequent procedures and interventions were reviewed. Since many of these patients were not evaluated until months or years after the apparent event that might have caused ARC, a retrospective assessment of the events related to spinal diseases, injuries, operations or any other invasive procedure performed in or near the spine were evaluated to identify any possibly related event in the available documentation. The precise probable cause was identified in 472 cases (96.5%); in the remaining 17 cases (3.5%), probable cause was deduced from sudden changes in the intensity and frequency in symptoms that coincided with a considerable change documented in the neurological exam, together with definite radiological findings reported at about that time. Previously, only a portion of the total number of patients included in this series was published.[1] This series encompasses all the patients seen from 1989 to 2006, making this the most current report.

As shown in Table 1, this updated series includes 489 patients (233 men and 256 women ranging from 24 to 94 years old). Most of the patients were middle-aged, with an average of 53.4 years (from 27 to 86 years of age) for men and 48.4 years for women (from 22 to 92 years old). Twenty six cases, in whom some of their medical records and some of their radiographs were reviewed but not examined directly by the author, were excluded from this series.

The type of pain, its frequency, and the location were noted; psychosocial factors such as litigation, Worker's Compensation claims, and work status were also recorded (see Table 2). Similarly, the frequency and type of bladder, rectal, and sexual dysfunction were noted. The occurrence of depression, anxiety and other emotional disturbances were evaluated in separate interviews by a

psychologist. The imaging studies were interpreted by an experienced radiologist and by the author.

Table 1. Probable Causative Events In Patients With Arachnoiditis

Myelogram with Pantopaque, (pre 1986)	12
Myelogram* followed by spinal surgery (post 1986)	16
One Laminectomy (first)	38
Laminectomy plus another procedure	18
Laminectomy, (2nd or multiple)	76
Spinal fusion with bone graft	36
Spinal fusion with hardware	71
Spinal anesthesia	45
Epidural anesthesia (Lumbar)	51
Epidural steroid injections (with incidental dural puncture)	53
Pseudomeningocele following dural tears at laminectomy	27
Other pain relief related procedure	29
Thoracic epidural anesthesia (syringomyelia)	5
Neuroplasty	5
Vertebroplasty	4
Spinal “taps"	3
Total	489

*In 1986, the production of oil-soluble contrast media for myelograms was discontinued in the USA.

Possibly Related Disorders

With certain frequency, some non-spinal related signs and symptoms appeared in patients diagnosed with ARC. Readers should be reminded that these statistics do not represent actual incidence of cases of ARC, but simply represent the frequency of each specific possible causative event in the group of patients included this series. Their incidence, as obtained from our group of patients, is shown in Tables 2 to 5. Patients related that these manifestations were not clinically manifested before the causative event which may have produced arachnoiditis, however this fact could not be specifically verified or denied from the review of their medical records.

Other illnesses, present in this group of patients, appeared to suggest the association of some ailments that may have more than a casual coincidence. For example, degenerative disc and/or facet joint disease of the cervical spine was noted in 32 patients (6.5%). Pulmonary disease was also diagnosed in 147 patients (30%)—all of which were, or have been cigarette smokers. Obesity (>20% over the patient’s expected body weight) was noted in 129 cases (26.3 %). Spondylolisthesis of the lumbar spine was present in 51 patients (10.4%) and lumbarization of S-1 or sacralization of L-5 vertebrae was reported in 129 (26.3%) and in 19 patients (3.8%), respectively. These abnormalities tend to render the spine unstable at a younger age. Similarly, the presence of short pedicles were noted in 112 patients (22.9%) from radiological studies and is important to consider since the narrower spinal canal appears to magnify symptoms caused by minor lesions. In such a case, a bulging disc, for example, would manifest as severe low back pain with radiculopathy. Another congenital defect is “spina bifida occulta" which again may not manifest with clinical symptoms until a mild injury to the spine results in exaggerated symptoms. See Figure 1 for the telltale presence of a “tuck-in" sacrum in the physical exam.

Table 2. Pain Related And Sensory Symptoms Observed In Patients With Arachnoiditis

Burning pain	
Gnawing pain in lumbosacra	

			Stabbing pain in lumbosacra
			Constricting pain in legs or
			Burning sensation in one fo
			Burning sensation in both fe
			Segmental numbness in the extremities
			Tingling sensation in one le
			Tingling sensation in both le
			Hypoesthesia
			Formication
			Dysesthesia
			Gait abnormalities
			Paraplegia
			Hyporeflexia in one leg
			Hyporeflexia in both legs
			Hyperreflexia in one leg
			Skin rash
			Muscle spasms
			Pruritus
			Allodynia
			Hyperalgesia
			Plantar Neuromas
Table 3. Systemic Symptoms Associated With Arachnoiditis	Low-grade fever of unknown origin	357	(73%)
	Frequent headaches (>4/week)	265	(54.1%)
	Chronic fatigue	231	(47.2%)
	Morning back stiffness	402	(82.2%)
	Heat intolerance	416	(85%)
	Profuse diaphoresis	409	(83.6%)
	Nocturnal diaphoresis	304	(62.1%)
	Frequ	42	(8.5%

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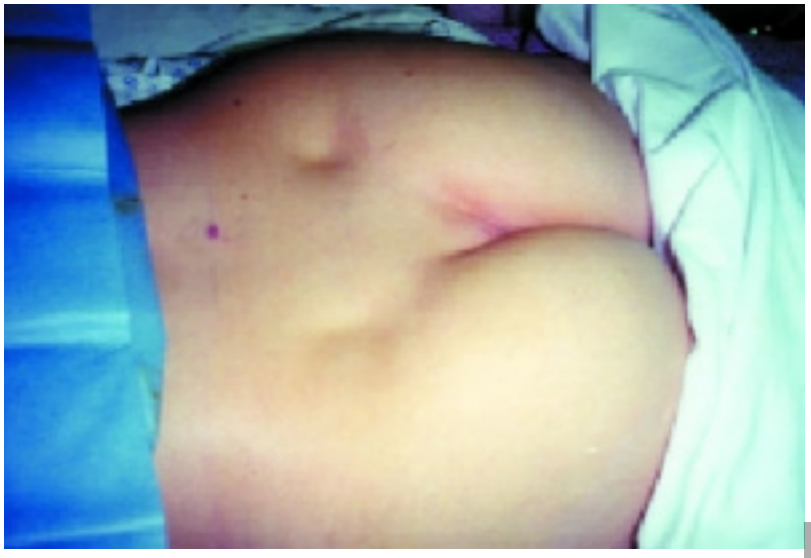


[2]**Figure 1.** *"Tucked sacrum" noted in a patient with "spina bifida occulta" .*

Dilatation or ectasia of the dural sac was common in those patients that had undergone spinal surgery through the posterior approach. Most of these, 126 (25.7%), occurred after laminectomy operations, and 84 others after spinal fusion (17.1%). All 210 of these patients (42.9%) also had clinical and radiological manifestations of ARC. Chronic obstructive lung disease was diagnosed in eight patients (1.6%) while five others (0.9%) had the ominous triad of arachnoiditis, pseudomeningocele, and pseudoarthrosis of the lumbar spine.

Clinical Symptoms in Patients with Arachnoiditis

Severe, unrelenting pain was the predominant symptom in patients with confirmed arachnoiditis. Although presentation had various characteristics, the common denominator was consistently burning pain that was present in 478 patients (97.7%). Low back and unilateral lower extremity pain occurred in 169 patients (34.5%) and 279 (57%) had pain in both lower extremities. The pain radiated upwards to the thoracic spine in 141 patients (28.8%) and 68 patients (13.9%) also had upper extremity pain, but these localizations of pain were possibly unrelated to ARC. The pain-related manifestations, the characteristics of the pain, the sensory and motor deficits, as well as the abnormalities found in the reflexes of the lower extremities are presented in Table 2.

Systemic Symptoms

These patients also presented with a variety of systemic symptoms not specifically related to spinal disease manifestations. Most were related to neurovegetative dysfunction that could, in part, be attributed to the intimate anatomical connection between the lumbosacral spinal cord and the corresponding sympathetic chain. Some of these manifestations have also been described in patients with traumatic spinal cord injuries, multiple sclerosis, and transverse myelitis. Among them are the symptoms listed in Table 3.

Genito-Urinary Dysfunction

Other frequently encountered symptoms were those related to bladder dysfunction, chiefly those

affecting the complex sphincter function and having greater prevalence in women (256) with arachnoiditis, therefore they were considered separately from the symptoms that occurred in men (233). Most of these can be encompassed in the so called “neurogenic bladder." To substantiate the origin of the symptoms, volumetric studies were performed by a urologist in order to differentiate them from cases in which pelvic floor weakness had resulted from obstetrical injury or any other cause.

Approximately two-thirds of the patients reported the onset of bladder dysfunction occurring after the injurious event (listed in Table 1). Most of these manifestations are typical of the syndrome referred to as “neurogenic bladder." Sixty one women were forced to do self-catheterization—a rather humiliating self-care procedure—in order to empty their bladder.

Gastrointestinal Symptoms

Bowel dysfunction consisted mostly of constipation in 312 (64%) of the patients and it was usually related to opioid oral intake and was generally treated with laxatives and occasional enemas. Intermittent bouts of diarrhea were noted in 132 patients (26.9%) some of which were also suspected to have irritable bowel syndrome. Severe constipation requiring either colostomy or frequent removal of fecal impaction was noted in 8 patients (1.6%) with either thoracic medullary lesions including syringomyelia (3 cases) or severe injuries to the cauda equina in 26 patients (5.3%). Rectal incontinence, noted in 51 women (19.9%) and 18 men (7.7%), was usually the most disturbing and embarrassing manifestation. Upper gastrointestinal disorders were related to the long-term ingestion of anti-inflammatory medications and occasional steroids. Two patients appeared to have developed fulminant liver failure after long term acetaminophen ingestion (3 to 4 gms daily); in both cases viral hepatitis was also present.

Table 4. Symptoms Related To Bladder Dysfunction In Patients With Arachnoiditis

Symptom
Dysuria
Hesitation
Incomplete emptying of bladder
Frequency
Urgency
Incontinence
Needed self- catetherization
*73 of them (28.5%) also had re

Sexual Dysfunction

Only six women (2.3 %) claimed no sexual dysfunction. One hundred and eighteen (46%) had pain at penetration. Two hundred and thirteen (83.2 %) noted loss of libido since the onset of their back pain. Lower back pain during intercourse was reported by 217 (84.6%) women, while 34 of them (13.2%) had pain in their lower extremities as well. 81 patients (31.6%) had exacerbation of their back pain after intercourse. When asked if any particular position for intercourse was better tolerated, 75 patients (29.2%) felt that the sitting on top position was better tolerated, whereas 56 patients (21.8%) preferred the supine position, 57 others (22.2%) were more comfortable on the lateral decubitus with the variant labeled as the “spoon" position, and 3 others (1.1%) felt it was less uncomfortable when sitting on a bench or sofa.

Symptoms and incidence of sexual dysfunction in the male patients are presented in Table 5. It is evident that impotence was the most common dysfunction; however no adjustments were made for the usual sexual dysfunction that may be present in a normally-aging population. The group of patients (233) is considered too small to conclude any significant result, especially since there was such a wide range of ages.

Table 5. Manifestations of Sexual Dysfunction In Male Patients With Arachnoiditis

Reduced libido
Partial and short lasting erection
Complete impotence
Perineal pain during erection

Perineal burning during erection
Low back pain during intercourse
Lower extremity pain during intercourse
Low back pain after intercourse
Received counseling
Unsuccessful counseling

There is no doubt that chronic back pain affects sexual activity. Although many of the patients in this series had low back pain before the apparent onset of arachnoiditis, no effort was made to differentiate it from the effect of chronic back pain, alone, on sexual performance. However, it was evident that sphincter and perineal muscle-related manifestations were more apparent after the symptoms of ARC became evident. While most of these symptoms were enough to have affected their sexual performance, it was impossible to discern what additional impact anxiety and fear of pain might have had on arousal and performance during intercourse. It was also not determined how sexual dysfunction was affected by pain during coitus and how it may have affected these patients' sex lives. While the sexual component has been generally ignored in the past, the impact on adult patients' quality of life and interrelationships requires that it be recognized and addressed. Physicians that care for this patient population need to regularly document the type of symptoms and psychological responses as they relate to sexual function.

Other Activities

Before the appearance of symptoms attributed to ARC, 87 of the male patients (37.3%) played some sport or performed regular physical exercise. Similarly, 112 (43.7%) women played some sport regularly. In all cases, regular physical exercise was interrupted after the injurious event. All patients reported that they had to abandon most outdoor leisure activities because exerting themselves usually exacerbated the intensity of the pain.

Habits and Circumstances

Certain habits and social conditions were present with sufficient regularity to establish a pattern which merits notice. For example, 469 patients (95.9%) complained of insomnia while 361 patients (73.8%) required hypnotics for treatment. Dreams were reported in only 132 patients (26.9%). In addition, 336 patients (68.7%), of which 182 were males and 125 females, smoked cigarettes, but only 18 patients (3.6%) admitted to drinking alcoholic beverages in excess. Total disability had been granted to 394 (80.5%) while 19 patients (3.8) were partially disabled. Only 29 of them (5.9%) worked full time and an undetermined number of them had some part time work. When first seen, 112 patients (22.9%) were involved in litigation and 22 others (4.4%) had settled previous claims.

Radiological Diagnosis

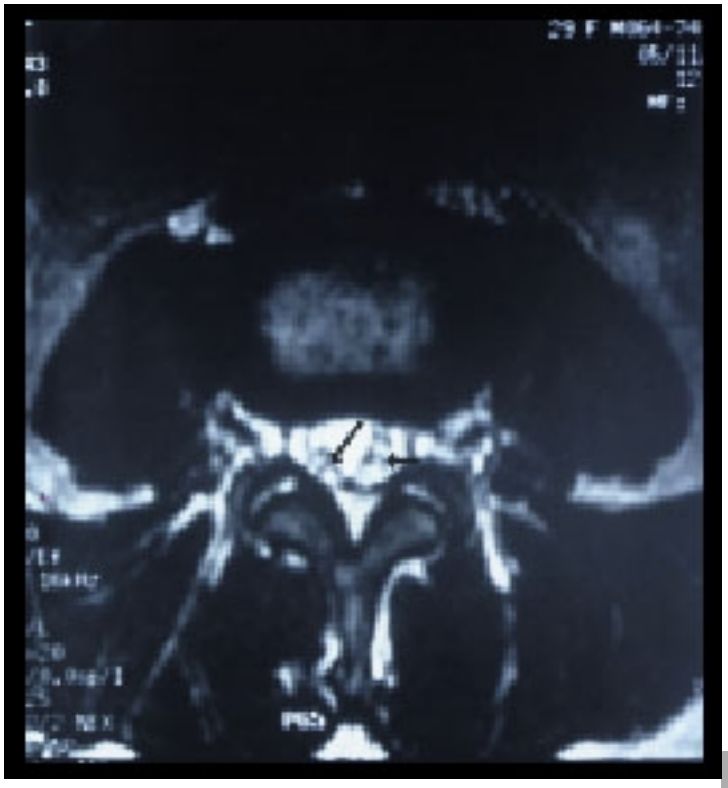
In every case, the clinical diagnosis was confirmed by diagnostic imaging studies including either magnetic resonance (MRI) or a myelogram followed by a computerized axial tomography (CAT) scan, when the former was contraindicated. It is suggested that if one or more of the following lesions is present in a patient, then a diagnosis of arachnoiditis is confirmed.

- intrathecal calcification
- enhanced nerve roots (see Figure 2)
- clumping of nerve roots (see Figure 3)
- adherence of nerve roots to the wall of the dural sac (see Figure 4)
- abnormal distribution of nerve roots within the dural sac
- extradural scarring in continuity with a deformed dural sac

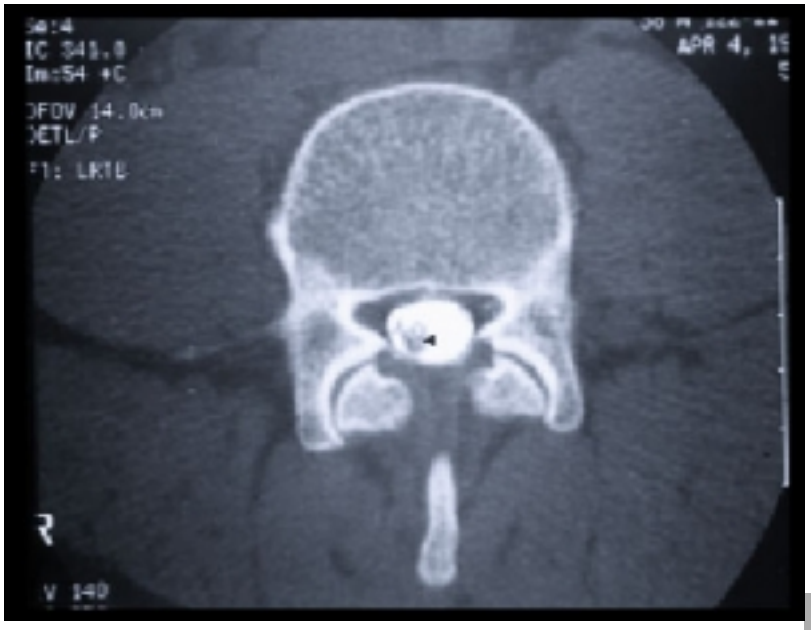
- Intrathecal pseudocysts
- Abnormal pattern of dye distribution within the dural sac pseudomeningocele (see Figure 5)
- intradural scarring
- dural sac deformity or narrowing
- residual oil-soluble contrast media in the dural sac
- multiple deposits of contrast media with irregular distribution (see Figure 6)

Extrapolation of Radiological Findings

Using plain films, only the recognition of an intradural calcification or a residual droplet of oil-soluble dyes (pantopaque) can suggest the presence of ARC. The wide acceptance of surface-coil magnetic resonance imaging (MRI) as a non-invasive diagnostic test—without danger of further re-injury—has made it possible to arrive at a more precise diagnosis, especially when thin slices are used. The axial and sagittal views allow the identification of nerve roots and dural sac pathology displaying, in detail, the type of abnormal findings as described by Ross et al,[17] Delamarter et al,[18] and others.[19,20] Limitation of the usefulness of MRI has been encountered in patients with metal devices in the proximity of the spine or near delicate organs such as the eyes or the brain. In these instances, a myelogram followed by a lumbar spinal CAT scan is indicated.[21]



[3]**Figure 2.** *Axial view of the MRI of the lumbar spine demonstrating “Enhanced (edematous) nerve roots (black arrows).*



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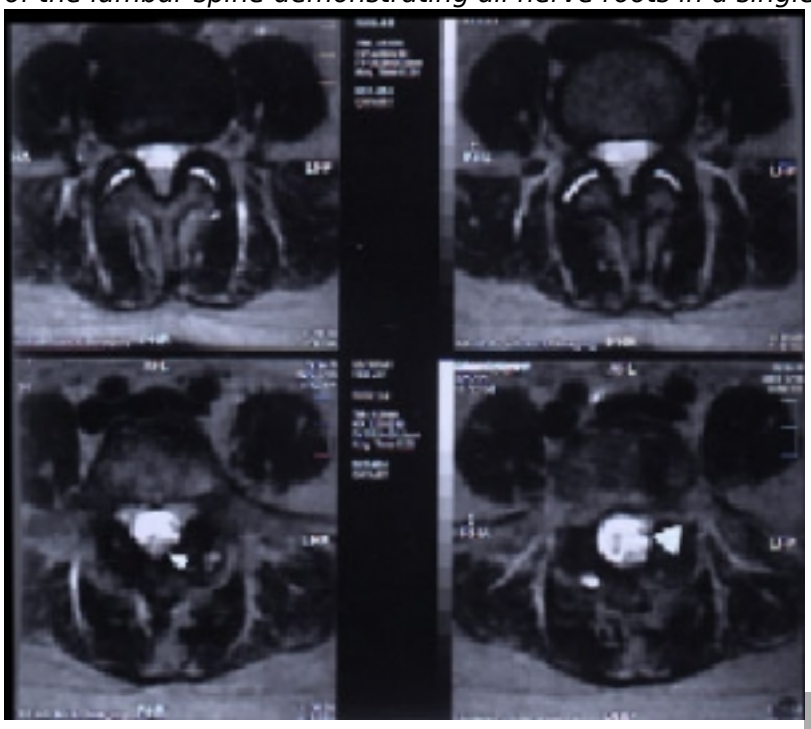
[4]**Figure 3.** *CAT scan of the lumbar spine demonstrating all nerve roots in a single clump (arrow head).*



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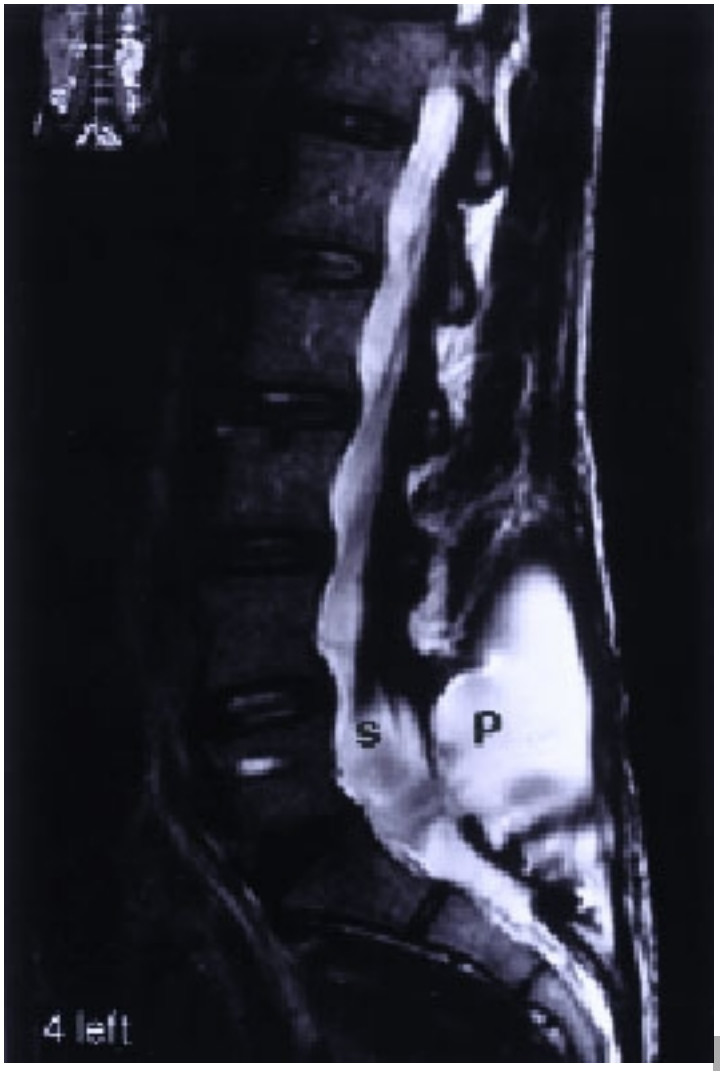
[5]**Figure 4.** *Four axial views of an MRI of the lumbar spine showing in the two upper frames, the normal location of the nerve roots in the posterior half of the dural sac, situated at the L3 vertebra. In the two lower frames, clumped and asymmetrically located nerve roots are seen at L4. (small arrow head). Nerve roots are also adhered to the dural sac on the right, lower frame (large arrow head).*



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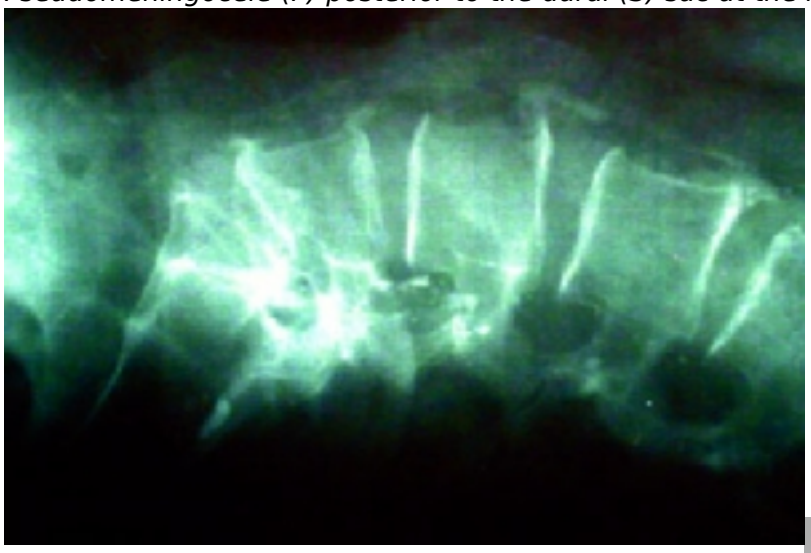
[6]**Figure 5.**
Pseudomeningocele (P) posterior to the dural (S) sac at the level of L5, after an L5-S1 discectomy.



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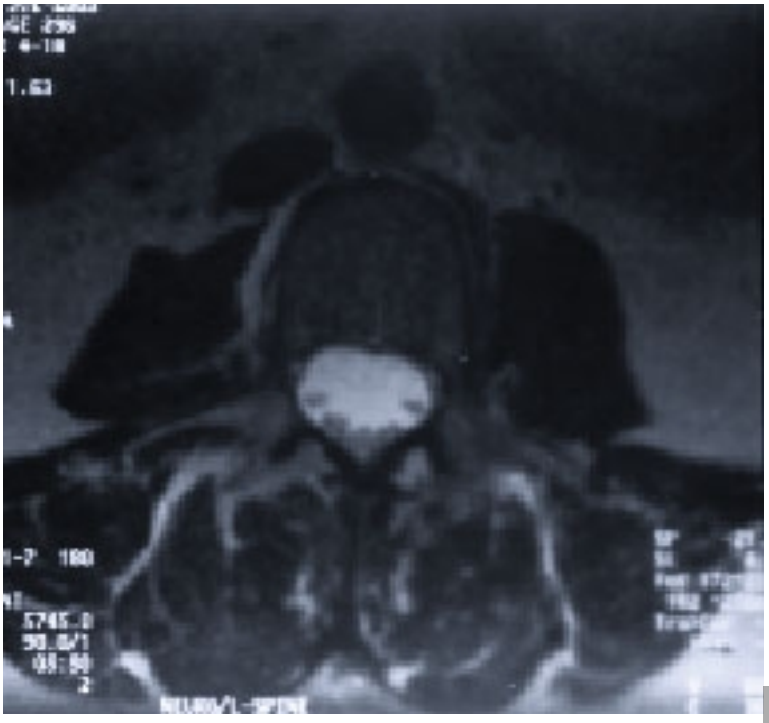
[7]**Figure 6.**
Distribution of multiple small droplets of pantopaque at different level, from L3 to the end of the dural



sac at S2.

[8]**Figure 7.** *Axial view of an MRI of a normal lumbar spine showing most of the nerve roots symetrically located in the posterior half of the dural sac. Two nerve roots are ascending toward the anterior corners of the sac in their way toward departing through the lateral foramen.*

Other radiological findings, such as when the conus medullaris and nerve roots are either deviated or tethered by adhesions or trapped or compressed by intrathecal scar tissue (identified in an MRI or a CAT scan following myelography[22,23]), suggest that an adhesive arachnoiditic proliferative process is already going on three months after the injurious event. In either of these imaging studies, the absolute confirmation is obtained when the nerve roots are clumped and asymmetrically located in comparing one side to the other. This abnormal distribution is in contrast to their normal appearance of being symmetrically located on both sides with the roots individually identifiable (see Figure 7). Nevertheless, in the first three months after the injurious event, “enhancement," meaning edema of the nerve roots, may be visualized by intravenously administering the contrast media gadopentate dimeglumine to patients.[24] In myelography, images of the dural sac appearing as having being “painted with a brush with little paint," blunting of axillary root pouches, dural cuff cysts, pachymeningitis, and abrupt obliteration of the dural sac are all suggestive of ARC.[21]

Not uncommonly, the type, location and distribution of symptoms in patients suffering from ARC are atypical because they superimpose the signs and symptoms produced by other underlying disease such as recurrent herniated discs, spinal stenosis, spondylolisthesis, facet arthropathy, meningitis etc.[25] This confusion makes the precise clinical diagnosis of arachnoiditis and each of the accompanying spinal conditions difficult and requires a meticulous and careful radiological interpretation. [17,18,21] From the author’s experience, a certain pattern of clinical manifestations were found and documented in every patient seen (see Tables 2 to 5). While such symptoms pointed toward the clinical suspicion of ARC, a case was not considered confirmed unless there was also an unquestionable and objective radiological diagnoses.

Myeloscopy

Other authors have claimed that arachnoiditis can be diagnosed by direct visualization with a myeloscope.[26,27] This assertion is questionable at best since the field of vision with this device is very

limited. At worse, the invasion of the dural sac does not warrant the information obtained since the arachnoid will undoubtedly be perforated and thus initiate a “flair-up" process if ARC is already present. In addition to having poor documentation from the photographed sites, the extent and location (multiple levels) cannot be determined convincingly. The likelihood that the endoscopist would initiate treatment (lysis of adhesions) in lesions that could not be confirmed by others, have cast a doubt of suspicion over this procedure. Moreover, since it has been shown that those adhesions will form again—and the procedure, itself, may stimulate further noxious stimuli that will exacerbate the clinical syndrome—this diagnostic and therapeutic modality is not recommended.

Electrodiagnostic Studies

Attempts to apply electrodiagnostic tests, including EMG, nerve conductions studies, and evoked potentials—commonly used to diagnose peripheral neuromuscular diseases[28,29]—have not rendered a consistent pattern that could be identified as that of arachnoiditis. Nevertheless, they have been useful in confirming the presence of other concomitant illnesses such as radiculopathy, diabetic neuropathy, etc. or to rule out neurological disease such as multiple sclerosis, amyotrophic lateral sclerosis, and others. Aside from these benefits, a definite electrodiagnostic pattern has not been found that would make any of these tests pathognomonic of arachnoiditis.

Current Epidemiology

As Table 1 shows, most cases of lumbar arachnoiditis represented in this series were most likely caused by therapeutic interventions such as laminectomies,[30,31] fusions,[32] epidural anesthesia,[33] spinal anesthesia,[34] epidural injections of steroids,[35] neuroplasty or adhesiolysis,[36] neurolytic agents,[29] some local anesthetics in high concentration,[38] epidural blood patches[39,40] and, less commonly, other invasive diagnostic procedures such as traumatic myelograms with water-soluble dyes,[41] discographies, lumbar punctures, and any other invasion of the vertebral canal. These cases may, in fact, be due not to the procedure itself but most likely to the accidental injury of the dura, with injection of an irritant chemical substance (methylene blue, acrylate, etc), blood into the intrathecal space, or even single traumatic spinal taps.

In order to reach the subarachnoid compartment, insertion of a needle that penetrates the dura undoubtedly has also to perforate the arachnoid membrane. It follows that intradural lesions can occur even after the slightest of trauma to the spinal cord or nerve roots (paraesthesiae) thereby initiating an inflammatory reaction in this trabecular membrane, followed by the appearance of some features common to ARC.[42] These include some cases of syringomyelia subsequent to a traumatic puncture of the spinal cord[43,44] or when pseudomeningoceles[45] appear after spinal operations. Cases of multiple invasive interventions with injections of chemically irritant substances such as depo-medrol[46] and methrotraxate,[47] by repeatedly producing inflammatory responses of the arachnoid and the adjacent dural wall, may eventually result in arachnoiditis ossificans,[48] arachnoid cysts,[49] or pachymeningitis,[50] all of which are extreme features of ARC .

In addition, cases of cauda equina syndrome and transient nerve root irritation that are usually thought to be only functional, temporary complications, are shown—through non-invasive diagnostic tests (coronal views in MRI images, nerve conduction studies and EMG)—to be quite the opposite by identifying definite lesions within the cauda equina. In the author’s opinion, for most of these cases, the inflammatory phase[51,52] behaves, clinically and functionally, as ARC and eventually progresses to permanent neurological deficits.[53]

Interpretation of Symptoms

Although the presence of signs and symptoms that do not conform with the representation of typical radiculopathy may cast some skepticism about the patient’s veracity and credibility, physicians must

realize that, in arachnoiditis, there are desynchronized reflex responses to random production of hyperesthesia that result in axonal injury.[54] Burning sensations which appeared in over 90% of our patients with arachnoiditis (see Table 2) are usually evoked by sustained pressure during nociceptor sensitization (noticed after long standing or walking), but always outlast the period of stimulation. These pain signals are most likely transmitted by fast-conducting primary afferents, as when severe burning pain is seen after repetitive stimulation or ectopic firing.[55] On the other hand, sensory deficit manifestations such as tingling, “pins and needles," formication, and even pruritus seen in some of our patients (see Table 2) may represent ectopically-generated nerve impulses to certain maneuvers such as stretching or flexing the lower extremities, turning from the supine to the lateral position, both of which induce excitation of deep tissue afferents.[56,57]

Hyperesthesia (see Table 2) appearing weeks after surgery may herald Wallerian degeneration accompanied by an increase of endoneurial fluid pressure[58,59] extending to the distal root ganglia which, in turn, generates a mechanical stimulus to evoke abnormal activity in sensory neurons.[60] Another mechanism, occurring 4 to 6 months after laminectomy, may have a role in the production of areas of hyperesthesia alternating with areas of sensory diminution. This mechanism involves considerable constrictive scarring and fibrosis surrounding a nerve root (extradurally) and thus compromising the intraneural blood flow sufficiently enough to result in localized ischemia.

Further, traumatic lesions of the nerve roots or the spinal cord by either traumatic punctures, spinal fractures, forceful surgical retraction,[61] or dissection, etc., may eventually affect some portions of the Lissauer’s tract and the substancia gelatinosa which are thought to participate in some of the mechanisms of pain suppression.[62,63] This finding suggests that at least some of the deafferentation type of pain may be due to an indirect dis-inhibition of the dorsal horn’s transmission cells. In these instances the pain-related symptoms will be evident in the immediate postoperative period.

Another mechanism for the production of ARC is the accumulation that may occur outside of the dural sac after surgical operations. Though is recognized that the blood, itself, does not usually cross the dural barrier, substances such as leukotrienes and cytokins resulting from the degradation of blood cells may do so.[64]

A recently identified source of discomfort, previously ignored, is the presence in post-lumbar spine laminectomy patients in whom fibrosis and scar tissue proliferates at the site of the operation constricting the dural sac and dilating it distally.[65] This complication appears three to six months following surgery and results in a gradual enlargement of the diameter of the sac, accompanied by markedly altered circulation of the cerebrospinal fluid from brain-to-spine and vice versa. Moreover, the function of the arachnoid villi, that allow unidirectional exit of the CSF toward the venous circulation is markedly reduced, if not obliterated, by this proliferative process. This condition manifests as a continuous gnawing and uncomfortable sensation of pressure in the lumbosacral spine and is usually not relieved by anti-inflammatory or narcotic analgesics.

Mechanical irritation (walking, pedaling, and gardening) may be associated with an erithematous discoloration (rubor) and a warming sensation of the feet that appears to initiate, by stimulation, a sympathetic mediated pain (see Table 2). The occurrence of antidromic vasodilatation, as well as mechanical and thermal hyperalgesia may be interpreted as a recruitment of the sympathetic nervous system into reflex action contributing, by default, to an unorganized vasomotor symptomatology. This would explain the complex mechanism of the neuropathic pain phenomenon experienced by patients with ARC that includes sympathetic-mediated symptoms and signs.[66]

The feasibility of having different mechanisms for different types of pain appears plausible, as allodynia, hyperalgesia, and burning sensations are subtypes of neuropathic pain.[60] This sympathetic-parasympathetic imbalance may be responsible for the frequent diaphoresis, heat intolerance, and nocturnal hyperactivity of the sweat glands seen in ARC. This also suggests participation of an autoimmune reaction, as some patients appear to be more susceptible to develop more scarring and adhesions in the healing process than others (see Table 3). This variant is perhaps an exaggerated

response generated by an antigen-antibody reaction in a hypersensitive patient.[66] These neurotoxic responses may repeatedly generate endogenous pirogens resulting in an intermittent low-grade fever, similar to that seen in patients with lupus erythematosus, polyarteritis nodosa, Reiter's syndrome, etc. Though, there is no specific pattern, low-grade fever implies an active autoimmune process[67] with its usual periodicity and is a concept supported by the findings of abnormal activity of gamma-glutamyl transferase in CSF,[68] defective fibronolysis,[69] and in some cases of arachnoiditis and failed back surgery syndrome.

As shown in Table 2, pain is a common denominator in every case, but to better understand this pain (usually derived from either a direct or an indirect injury to the intraspinal nervous system), it may be recognized that it is not uncommon that this neuropathic pain is also accompanied by some sensory loss phenomenon[1,25] as occurs with needle-produced paraesthesia. In brief, this painful dysesthesia,[42] caused by either isolated or repeated nerve root trauma, such as when the clumped nerve roots rub against each other or when pain is elicited as the patients extend their legs or flex their spine, and in so doing, essentially stretch the nerve roots. Portions of the these nerve roots—which are normally free floating in the CSF—are fixed as they adhere to each other or to the wall of the dural sac thus eliciting what amounts to ectopic nerve impulse activity with single or multiple burst charges.[55] This mechanism has some similarity to the paraesthesia provoked by stretching or tapping on a nerve trunk and somewhat similar to the perceptions that cause allodynia and/or hyperalgesia, indicating an increased mechanosensitivity.[56,57]

Contrary to the pain seen in a classic radiculopathy—that usually projects along a nerve trunk or follows a nerve dermatome and of which the most typical is sciatica—pain in patients with ARC does not extend along a continuous path, but is present in regions or patches either at the medial upper section of the thigh, or at the posterolateral aspect of the distal thigh or may appear irregularly on the lateral distant portion of the leg. When it does extend to the toes, it is accompanied by fasciculations in the early stages and by frank muscle spasms later on.[58,59] This unusual mapping may represent irritation of nociceptor afferent fibers transmitted to muscle fibers in the form of desynchronized reflex responses to random ectopic firing.[55] In some cases, certain plasticity has been shown when pharmacological manipulations have been performed on recovering fibers, axons or neurons after a variety of injuries.[70,71] In some instances, segmental numbness may be due to the predominant effect of descending impulses upon the dorsal horn, supposedly followed by the projection of neurons of the spinothalamic tract and possibly connecting with inhibitory interneurons located next to the dorsal root ganglion entry's zone. This may control the extent of the sensory deficit manifested on the skin distribution.[72] If followed in detail, some of these representations can be traced back to the location, the side, and the level where the clumping of the nerve roots is present.

The extent and severity of sexual dysfunction are alarming as reported by many patients that experienced pain during and after intercourse, which resulted in overt fear and hesitation for sexual activity, mainly because most physicians do not inquire about them and do not document this important aspect of the patient's disease. The prevalent symptoms were different among the genders; in the males, partial (60%) or total (40%) impotence was the most common and feared; loss of libido and difficulty in arousing where also common. In some cases, if erection was obtained, pain and burning sensation would occur at ejaculation. Sexual dysfunction in the female group consisted mostly of loss of libido and severe pain (pelvic, lumbar or in the lower extremities) during or after intercourse, resulting in worries and concern for their lack of active participation in marital sex. Frequently, in both groups, low back and/or lower extremity pain during or after intercourse was stoically tolerated by patients in an effort to please their partners (see Table 3).

Moreover, the partners' desire not uncommonly was tempered by the concern for causing pain and injury to the patient. Needless to say, this scenario lead to disappointment and friction that were prelude to intermarriage conflict. Though advice on trying different positions was at times helpful, counseling with the patient and or his/her partner did little to improve their actual sexual performance although it did help them to understand it and accept it as part of the disease. Since sexual dysfunction was a major contributor to the loss of self-esteem, depression, and relative isolation seen in many of

these cases, physicians are encouraged to ask patients about it, document it in their records, follow it, and refer patients to sex therapists when deemed necessary.

Smoking cigarettes is considered a prevailing factor in postoperative morbidity[73] since it contributes to respiratory complications,[74] delays healing, and probably increases the incidence of pseudoarthrosis and pseudomeningoceles[45] from frequent coughing and resultant increase in intradural pressure. This appears to be an ominous habit; in order to have the optimal chances of success, practitioners most insist that smoking cessation is achieved, especially if spinal surgical intervention is contemplated.[75,76] Of interest was the finding that excessive alcoholic beverage ingestion was reported in patients in whom the prescription of analgesics and other adjuvant medications had been considered inadequate and inappropriate.

Urinary hesitation and dysuria were found more frequently in males, however, this symptom is not unusual in middle-aged men with early benign prostatic hypertrophy. It emphasizes the need for a complete work-up in order to arrive at a precise diagnosis. In women, urinary incontinence was the most frequent sign of bladder dysfunction. Urinary retention was commonly seen as one of the alarming signs of cauda equina involvement in the immediate postoperative period. Bladder dysfunction included a variety of symptoms that may or may not be included within the label of “neurogenic bladder." These may include “dripping or leaking” as a result of a dysfunctional sphincter. Incomplete emptying produces persistent residual urine which may become easily infected. A combination of storage and emptying dysfunction is secondary to sympathetic receptors malfunction and afferent nerve dysfunction having a myriad of symptoms such as frequency, urgency, incontinence, and hesitancy. The symptoms were noted to be more frequent and more severe in females than in males for patients having ARC after spinal surgical interventions.

Conclusions

This review of the symptoms noted in a group of patients with ARC—with or without failed back syndrome—represents an analysis of the author’s clinical observations of these diseases. The author’s interpretations are aimed to inform those who care for these patients that the intensity, frequency, and characteristics of these manifestations are real. In brief, this article has attempted to identify the possible path of transmission and perception, while recognizing that much is still in the unknown category.

Since most cases of arachnoiditis and the so called failed back surgery syndrome are caused during a diagnostic or therapeutic intervention of the spine, there is a certain reticence to admit that, in most instances, it is a disease related to interventionism gone astray.[77] It raises the question as to whether diagnostic (myelograms, spinal taps, discograms, etc.) or therapeutic invasions of the spine (laminectomies, fusions, epidural or spinal anesthesia, neuroplasties, epidural injections of steroids, etc.) need to be limited to specific indications.

The majority of patients in this series were not working, required assisted care, made frequent doctor visits, underwent repeated procedures that only produced temporary pain relief and, even though most of them were taking a myriad of medications, including opiates, they continued to experience severe pain. Considering that the severity and chronicity of such morbidity includes life-term suffering, psychological dysfunction, and physical disability—resulting in an enormous cost to the health care system—prevention in this case, by far outweighs the effectiveness of any therapeutic modality attempted.

In caring for these patients, one cannot help but to emphasize that it is primordial to make thoughtful and objective decisions regarding interventional diagnostic or therapeutic modalities of the spine, given the potential for an adverse long-term outcome. Since, at present, there is no definitive cure for this condition, emphasis needs to be placed on prevention. Invasive interventions in the spine should only be performed when absolutely necessary and only when such procedures have been shown to offer a

definite benefit to the patient.

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References:

Resources

- 1. Aldrete JA. Clinical Diagnosis. In: Arachnoiditis: the silent epidemic, JA Aldrete (ed.) Futuremed. Denver. 2000. pp 201-220.
- 2. Aldrete JA. Infections. In Arachnoiditis: the silent epidemic; J A Aldrete (ed). Furturemed. Denver. 2000. pp 41-48.
- 3. Charcot JM and Joffroy A. Deux cas d'atrophie musculaire progressive avec lesions de la substance grise et des faiseeaux anterolateraux de la mo¢lle espiniere. Arch Physiologie. 1869. 2:354.
- 4. Krause F. Zur kenntnis der ruckenmarkslashmungen. Verh Kongr Inn Med Pt. 1907. 1:108-113
- 5. Schwarz E. Syphilitisehe myelomeningitis Mit Holenbildung in Ruchenmarke and Besodneren Dcgenerativen Veranderum Gen Der Neurologia. Wien Klin Wochenschr. 1897. 10:177. .
- 6. Madrazo I, Sandoval M, and Gonzalez D. Evaluation of the thalamic hypophyseal axis in patients with hypertensive hydrocephalus due to neurocysticereosis. Investigation Med. 1991. 2:113-119.
- 7. Woodal WC, Bertorini TE, Baklitian BJ, Gelfand MS: Spinal arachnoiditis with cryptococcus in a non-immunocompromized child. Pediat. Neurol. 1990. 3:206-208.
- 8. Horsley V. Chronic spinal meningitis: Its differential diagnosis and surgical treatment. Br Mewd J. 1909. 1:513-517.
- 9. Foix C and Alajouanine T. La myelite necrotique subaigue. Myelite ccntrale angiohypcrtrophe a evolument ascendante, d'abord spasmodique puis flasque, s'aceompagnant de dissociation albumino cytologique. Rev. Neurol. 1926. 2:1-42.
- 10. Vincent C, Puech P, and David M. Sur le diagnostic, le traitment chirurgical, des pronostic, des aracnoiditis spinales. Rev. Neurol 1930. 1:577-579.
- 11. Peacher WG and Peterson RC. Pantopaque myelography: Results, comparison of contrast media and spinal fluid reactions. J Neurosurg. 1945. 2:220-231.
- 12. Bering EA. Notes on the retention of pantopaque in the subarachnoid space. Am. J. Surg. 1950. 40:455-458.
- 13. Hurteau EF, Baird WC, and Siclair E. Aracnoiditis following the use of iodized oil. J. Bone Joint Surg. (AM) 1954. 36:393-400.
- 14. Erickson TC and Baaren HJ. Late meningeal reaction to pantopaque used in myelography (report of a case which terminated family). Trans. AM. Neurol. Assoc. 1953. 153:134-135.
- 15. Jorgensen J, Hansen PH, Steenskov V, and Oveson N. A clinical and radiologieal study of chronic lower spinal araehnoiditis. Neuroradiology. 1975. 9:139-144.
- 16. Phadke RV, Kohli A, John VK, et al: Tuberculous radiculomyelitis (arachnoiditis): Myelographic and CT myelographic appearances. Australasian Radiol. 1994. 38:10-16.
- 17. Ross JS, Masaryk TJ, Nodie MT, et al. MRI of lumbar arachnoiditis. AJR 1987. 149:1025-1032.
- 18. Delamarter RB, Ross JB, Masaryk TJ, et al. Diagnosis of lumbar arachnoiditis by magnetic resonance imaging. Spine. 1990:15.
- 19. Ross JS. MRI assessment of the postoperative spine. Degenerative disc disease. Rad Clin NA. 1991. 29:793-808.
- 20. Tratting VS, Stiglbauer R, Ungersbock K, et al. MRI in failed back surgery syndrome: comparison with CAT scan. Fortschr Roentgenstr. 1990. 152:369-373.

- 21. Boden SD, Davis DO, Dina TS, et al. Contrast enhanced MR imaging performed after successful lumbar disc surgery. Prospective study. Radiology 1992. 182:59-64.
- 22. Mangar D, Gonzalez W, and Linden C. Gadolinium-enhanced MRI and autopsy findings in a patient with cauda equine syndrome. Anesthesiology. 1993. 78:785-787.
- 23. Benveniste H, Huttemeier PC, Qiu H, et al. MRI of continuous spinal anesthesia with hyperbaric lidocaine: root or white matter lesion? Reg Anesth .1995. 20: (Suppl) 15.
- 24. Vroomen PC, Wilmink JT, and de Krom MC. Prognostic value of MRI findings in sciatica. Neuroradiology. 2002. 44:59-63.
- 25.Aldrete JA. Clinical diagnosis. In Arachnoiditis : the silent epidemic. J A Aldrete (ed). Denver, Futuremed. 2000. 201-220.
- 26. Blomberg R. A method for epiduroscopy and spinaloscopy: Presentation of preliminary results. Acta Anaesth Scand. 1985. 29:113-116.
- 27.Shimoji K, Fujioka H, Onodera M, et al. Observation of spinal canal and cisternae with the newly developed small-diameter, flexible fiberscopes. Anesthesiology. 1991. 75:341-344.
- 28. Katims JJ. Electrodiagnostic functional sensory evaluation with pain: A review of the neuroselective current perception threshold. Pain Digest. 1998. 219-230.
- 29. Magda P, Latov NBrannagan TH III, et al. Comparison of electrodiagnostic abnormalities and criterioa in a cohort of patients with chronic inflammatory demyelinating polyneuropathy. Arch Neurol. 2003. 60:1755-1759.
- 30. Matsui H, Tsuji H, Kanamori M, et al: Laminectomy induced arachnoiradiculitis: a postoperative serial MRI study. Neuroradiology. 1995. 37:660-666
- 31.Carrage EJ. The surgical treatment of disc degeneration: is the race not to the swift? Spine J. 2005. 5:587-588.
- 32. Deyo RA, Nachefson A, and Mirza SK. Spinal fusion surgery- the case for restraint. N Eng J Med. 2004. 350:722-726.
- 33. Reynolds F. Damage to the conus medullaris following spinal anesthesia. Anaesthesia. 2001. 56:235-247.
- 34. Poulakka R, Haasio J, Pitkanen M, et al. Technical aspects and postoperative sequelae of spinal and epidural; anesthesia; A prospective study of 3230 orthopedic patients. Reg Anesth Pain Med. 2000. 25:488-497.
- 35. Wilkinsonb HA. Dangers from methylprednisolone acetate therapy by intraspinal injection. Arch Neurol. 1989. 46: 721-725.
- 36. Aldrete JA, Zapata JC, and Ghaly R. Araclmoiditis following epidural adhesiolysis with hypertonic saline: Report of two cases. Pain Digest. 1996. 6: 368-370
- 37. Katz JA, Schchorst S, and Blisdard KS. Histopathologic changes in primate spinal cord after single and repeated epidural phenol administration. Reg. Anesth. 1995. 20: 283-290.
- 38. Kitagawa N, Oda M, and Totoki T. Possible mechanisms of irreversible nerve injury caused by local anesthetics. Anesthesiology. 2004. 100:962-967.
- 39. Aldrete JA and Brown T. Intrathecal hematoma and arachnoiditis after prophylactic blood patch through a catheter. Anesth & Analg. 1997. 84:233-234.
- 40. Diaz JH. Permanent paraparesis and cauda equine syndrome after epidural blood patch for post-dural puncture headache. Anesthesiology. 2002. 96:1515-1517.
- 41. Howland W J and Curry JL.: Experimental studies of Pantopaque arachnoiditis. Radiology. 1966. 87: 253-257.
- 42. Aldrete JA. Neurologic deficit and arachnoiditis from neuraxial anesthesia. Acta Anaesthesiol Scand. 2003. 47:3-12.
- 43. Bromage PR and Bernumoff JL. Paraplegia following intracord injection during attempted epidural anesthesia under general anesthesia. Reg Anesth Pain Med. 1998. 23:104-107.
- 44. Aldrete JA and Ferrari H. Myelopathy with syringomyelia following thoracic anaesthesia. Anaesth Intensiv Care. 2004. 32:100-103.
- 45 Aldrete JA and Ghaly RF. Postlamineetomy pseudomeningocele: an unsuspected cause of low back pain. Reg. Anesth. 1995. 20: 75-79.
- 46. Nelson DA. Intraspinal therapy using methylprednisolone aetrate: 23 years of clinical controversy. Spine. 1993. 18:278-282.
- 47. Weitz EM. Paraplegia following chymopapaine injection: A case report. J Bone Joint Surg Am.

1984. 66:1131-1135.
- 48- Ng P, Lorentzl, and Soo YS. Arachnoiditis ossificant of the cauda equine demonstrated on computed tomography scanogram. A case report. Spine. 1996. 21:2504-2507.
- 49. Roeder MB, Hazan C, and Jinkins JR. Ruptures spinal dermoid cyst with chemical arachnoiditis and disseminated intracranial liquid droplets. Neuroradiology. 1994. 37:146-147.
- 50. Barker LF and Ford FR. Chronic arachnoiditis obliterating the spinal subarachnoid space. JAMA. 1937. 109:785-786.
- 51. Avidan A, Gomori M, and Davidson E: Nerve root inflammation demonstrated by magnetic resonance imaging in a patient with transient neurologic symptoms after intrathecal injection of lidocaine. Anesthesiology 2002. 97:257-258.
- 52. Aldrete JA. Nerve root irritation or inflammation diagnosed by magnetic resonance imaging. Anesthesiology. 2003. 98:1294.
- 53. Lauretti GR. Pain transmission and modulation. In: Arachnoiditis: the silent epidemic..IA Aldrete (ed.), Denver Futuremed. 2000. pp 19-37.
- 54. Thelander V, Fagerland M, Friberg S, et al. Straight tests versus touch and pain afferents in single fascicles of the human ulnar nerve. Brain. 1990. 113: 1491-1500.
- 55. Nordin M, Nystrom B, Wallin U, and Habarth KE. Ectopic sensory discharges and paraesthesia in patients with disorders of peripheral nerves dorsal roots and dorsal columns. Pain. 1984. 20:231-245.
- 56. Ochoa JL. The newly recognized painful ABC syndrome Thermographie aspects. Thermology. 1986a. 2: 65-73; 308-316
- 57. Ochoa JL, Comstock WJ, Marohettini P, and Nizamuddin G. Intrafascicular nerve stimulation elicits regional skin warming that matches the projected field of evoked pain. Shaible, Vahle-Hinz (ed). Fine Afferent Nerve Fibers and Pain. Verlagsegesellschaft, Weinheim, 1987b. pp 476-479.
- 58. Powell HC, Myers RR, Costello ML, et al. Endoneurial fluid pressure in Wallerian degeneration. Ann Neurol. 1979. 5:550-557.
- 59. Myers P,R, Rydevick BL, Heckman HM, et al. Proximo-distal gradient in endoneurial fluid pressure. Exp Neurol. 1988. 102:368-370.
- 60. Howe JF, Loesscr JD, and Calvin WH. Mechanosensitivity of dorsal ganglia and chronically injured axons: A basis for the radicular pain of nerve root compression. Pain. 1977. 3:35-41.
- 61. Myers RR, Yamamoto T, Yaksh TL, et al: The role of focal nerve ischemia and Wallerian degeneration in peripheral nerve injury Produciong hyperesthesia. Anesthesiology. 1993. 73:308-316.
- 62. Rocha-Lauretti G. Pain Transmission and modulation. In Arachnoiditis: the silent epidemic. J.A. Aldrete (ed). Futuremed. Denver. 2000. pp 19-37.
- 63. Wall PD. The role of substancia gelatinosa as a gate control. In Bonia JJ (ed.) Pain. Raven Press. New York. 1980. pp 205-231.
- 64. Cassin B, Naidoo S, Ramsaroop R, et al. Immunolocalization of bradykinin receptors in human tissue. Inmuinopharmacology. 1997. 36:121-125.
- 65. Aldrete JA: Unpublished data.
- 66. Couto da Silva JM, Couto da Silva JM Jr, Aldrete JA. Body temperature and diaphoresis disturbances in a patient with arachnnoiditis. Anesth Analg. 2001. 93:1578-1579.
- 68. Akimov GA, Komahdenko NI, and Zinchenko VA. Activity of gamma-glutamyl transferase in the eerebrospinal fluid of various forms of cerebral arachnoiditis. Zh Nevropatol Psikiatr Im S S Korsakova. 1995. 86:682-684.
- 69. Cooper RG, Mitchell WS, Illingworth KJ, et al. The role of epidural fibrosis and defective fibrinolysis in the persistence of postlaminectomy back pain. 1991. 16:1044-1048.
- 70. Wall PD and Devor M. Surgery afferent impulses originate from dorsal root ganglia as well as from the periphery in normal and nerve-injured rats. Pain. 1983. pp 321-329.
- 71. Machettini P, Cline MA, and Ochoa JL. Innervation territories of touch and pain afferents in single fascicles of the human ulnar nerve. Brain. 1989. 78:213-216.
- 72. Mitchell C, Garrahy P, and Peake P. Postoperative respiratory morbidity: Identification and risk factors. Aust. NZ J.Surg. 1982. p 79.
- 73. Pearce AC and Jones RM. Smoking and perioperative morbidity. Anesthesiology. 1984. 61: 576-584.

- 74. Aldrete JA, Woodward ST, and Turk LH. Influence of cigarette smoking on the changes produced by spinal anesthesia on expiratory forced volume and flow rates. Anesth & Analg. 1973. 52:809-816.
- 75. Aldrete JA. Cessation of cigarette smoking by suggestion in the perianesthetic period. Anesthesiol. Rev. 1987. 14: 22-24.
- 76. Warner DO: helping surgical patient quit smoking: why, when and how. Anesth Analg 2005. 101:481-487.
- 77. Aldrete JA. Prognosis. In: Arachnoiditis: the silent epidemic. JA Aldrete (ed). Futuremed. Denver. 2000. pp. 273-280.

View Sources [9]

Resources

- 1. Aldrete JA. Clinical Diagnosis. In: Arachnoiditis: the silent epidemic, JA Aldrete (ed.) Futuremed. Denver. 2000. pp 201-220.
- 2. Aldrete JA. Infections. In Arachnoiditis: the silent epidemic; J A Aldrete (ed). Furturemed. Denver. 2000. pp 41-48.
- 3. Charcot JM and Joffroy A. Deux cas d'atrophie musculaire progressive avec lesions de la substance grise et des faiseeaux anterolateraux de la mo¢lle espiniere. Arch Physiologie. 1869. 2:354.
- 4. Krause F. Zur kenntnis der ruckenmarkslashmungen. Verh Kongr Inn Med Pt. 1907. 1:108-113
- 5. Schwarz E. Syphilitisehe myelomeningitis Mit Holenbildung in Ruchenmarke and Besodneren Dcgenerativen Veranderum Gen Der Neurologia. Wien Klin Wochenschr. 1897. 10:177. .
- 6. Madrazo I, Sandoval M, and Gonzalez D. Evaluation of the thalamic hypophyseal axis in patients with hypertensive hydrocephalus due to neurocysticereosis. Investigation Med. 1991. 2:113-119.
- 7. Woodal WC, Bertorini TE, Baklitian BJ, Gelfand MS: Spinal arachnoiditis with cryptococcus in a non-immunocompromized child. Pediat. Neurol. 1990. 3:206-208.
- 8. Horsley V. Chronic spinal meningitis: Its differential diagnosis and surgical treatment. Br Mewd J. 1909. 1:513-517.
- 9. Foix C and Alajouanine T. La myelite necrotique subaigue. Myelite ccntrale angiohypcrtrophe a evolument ascendante, d'abord spasmodique puis flasque, s'aceompagnant de dissociation albumino cytologique. Rev. Neurol. 1926. 2:1-42.
- 10. Vincent C, Puech P, and David M. Sur le diagnostic, le traitment chirurgical, des pronostic, des aracnoiditis spinales. Rev. Neurol 1930. 1:577-579.
- 11. Peacher WG and Peterson RC. Pantopaque myelography: Results, comparison of contrast media and spinal fluid reactions. J Neurosurg. 1945. 2:220-231.
- 12. Bering EA. Notes on the retention of pantopaque in the subarachnoid space. Am. J. Surg. 1950. 40:455-458.
- 13. Hurteau EF, Baird WC, and Siclair E. Aracnoiditis following the use of iodized oil. J. Bone Joint Surg. (AM) 1954. 36:393-400.
- 14. Erickson TC and Baaren HJ. Late meningeal reaction to pantopaque used in myelography (report of a case which terminated family). Trans. AM. Neurol. Assoc. 1953. 153:134-135.
- 15. Jorgensen J, Hansen PH, Steenskov V, and Oveson N. A clinical and radiologieal study of chronic lower spinal araehnoiditis. Neuroradiology. 1975. 9:139-144.
- 16. Phadke RV, Kohli A, John VK, et al: Tuberculous radiculomyelitis (arachnoiditis): Myelographic and CT myelographic appearances. Australasian Radiol. 1994. 38:10-16.
- 17. Ross JS, Masaryk TJ, Nodie MT, et al. MRI of lumbar arachnoiditis. AJR 1987. 149:1025-1032.
- 18. Delamarter RB, Ross JB, Masaryk TJ, et al. Diagnosis of lumbar arachnoiditis by magnetic resonance imaging. Spine. 1990:15.
- 19. Ross JS. MRI assessment of the postoperative spine. Degenerative disc disease. Rad Clin NA. 1991. 29:793-808.

- 20. Tratting VS, Stiglbauer R, Ungersbock K, et al. MRI in failed back surgery syndrome: comparison with CAT scan. Fortschr Roentgenstr. 1990. 152:369-373.
- 21. Boden SD, Davis DO, Dina TS, et al. Contrast enhanced MR imaging performed after successful lumbar disc surgery. Prospective study. Radiology 1992. 182:59-64.
- 22. Mangar D, Gonzalez W, and Linden C. Gadolinium-enhanced MRI and autopsy findings in a patient with cauda equine syndrome. Anesthesiology. 1993. 78:785-787.
- 23. Benveniste H, Huttemeier PC, Qiu H, et al. MRI of continuous spinal anesthesia with hyperbaric lidocaine: root or white matter lesion? Reg Anesth .1995. 20: (Suppl) 15.
- 24. Vroomen PC, Wilmink JT, and de Krom MC. Prognostic value of MRI findings in sciatica. Neuroradiology. 2002. 44:59-63.
- 25.Aldrete JA. Clinical diagnosis. In Arachnoiditis : the silent epidemic. J A Aldrete (ed). Denver, Futuremed. 2000. 201-220.
- 26. Blomberg R. A method for epiduroscopy and spinaloscopy: Presentation of preliminary results. Acta Anaesth Scand. 1985. 29:113-116.
- 27.Shimoji K, Fujioka H, Onodera M, et al. Observation of spinal canal and cisternae with the newly developed small-diameter, flexible fiberscopes. Anesthesiology. 1991. 75:341-344.
- 28. Katims JJ. Electrodiagnostic functional sensory evaluation with pain: A review of the neuroselective current perception threshold. Pain Digest. 1998. 219-230.
- 29. Magda P, Latov NBrannagan TH III, et al. Comparison of electrodiagnostic abnormalities and criterioa in a cohort of patients with chronic inflammatory demyelinating polyneuropathy. Arch Neurol. 2003. 60:1755-1759.
- 30. Matsui H, Tsuji H, Kanamori M, et al: Laminectomy induced arachnoiradiculitis: a postoperative serial MRI study. Neuroradiology. 1995. 37:660-666
- 31.Carrage EJ. The surgical treatment of disc degeneration: is the race not to the swift? Spine J. 2005. 5:587-588.
- 32. Deyo RA, Nachefson A, and Mirza SK. Spinal fusion surgery- the case for restraint. N Eng J Med. 2004. 350:722-726.
- 33. Reynolds F. Damage to the conus medullaris following spinal anesthesia. Anaesthesia. 2001. 56:235-247.
- 34. Poulakka R, Haasio J, Pitkanen M, et al. Technical aspects and postoperative sequelae of spinal and epidural; anesthesia; A prospective study of 3230 orthopedic patients. Reg Anesth Pain Med. 2000. 25:488-497.
- 35. Wilkinsonb HA. Dangers from methylprednisolone acetate therapy by intraspinal injection. Arch Neurol. 1989. 46: 721-725.
- 36. Aldrete JA, Zapata JC, and Ghaly R. Araclmoiditis following epidural adhesiolysis with hypertonic saline: Report of two cases. Pain Digest. 1996. 6: 368-370
- 37. Katz JA, Schchorst S, and Blisdard KS. Histopathologic changes in primate spinal cord after single and repeated epidural phenol administration. Reg. Anesth. 1995. 20: 283-290.
- 38. Kitagawa N, Oda M, and Totoki T. Possible mechanisms of irreversible nerve injury caused by local anesthetics. Anesthesiology. 2004. 100:962-967.
- 39. Aldrete JA and Brown T. Intrathecal hematoma and arachnoiditis after prophylactic blood patch through a catheter. Anesth & Analg. 1997. 84:233-234.
- 40. Diaz JH. Permanent paraparesis and cauda equine syndrome after epidural blood patch for post-dural puncture headache. Anesthesiology. 2002. 96:1515-1517.
- 41. Howland W J and Curry JL.: Experimental studies of Pantopaque arachnoiditis. Radiology. 1966. 87: 253-257.
- 42. Aldrete JA. Neurologic deficit and arachnoiditis from neuraxial anesthesia. Acta Anaesthesiol Scand. 2003. 47:3-12.
- 43. Bromage PR and Bernumoff JL. Paraplegia following intracord injection during attempted epidural anesthesia under general anesthesia. Reg Anesth Pain Med. 1998. 23:104-107.
- 44. Aldrete JA and Ferrari H. Myelopathy with syringomyelia following thoracic anaesthesia. Anaesth Intensiv Care. 2004. 32:100-103.
- 45 Aldrete JA and Ghaly RF. Postlamineetomy pseudomeningocele: an unsuspected cause of low back pain. Reg. Anesth. 1995. 20: 75-79.
- 46. Nelson DA. Intraspinal therapy using methylprednisolone aetrate: 23 years of clinical

controversy. Spine. 1993. 18:278-282.
- 47. Weitz EM. Paraplegia following chymopapaine injection: A case report. J Bone Joint Surg Am. 1984. 66:1131-1135.
- 48- Ng P, LorentzI, and Soo YS. Arachnoiditis ossificant of the cauda equine demonstrated on computed tomography scanogram. A case report. Spine. 1996. 21:2504-2507.
- 49. Roeder MB, Hazan C, and Jinkins JR. Ruptures spinal dermoid cyst with chemical arachnoiditis and disseminated intracranial liquid droplets. Neuroradiology. 1994. 37:146-147.
- 50. Barker LF and Ford FR. Chronic arachnoiditis obliterating the spinal subarachnoid space. JAMA. 1937. 109:785-786.
- 51. Avidan A, Gomori M, and Davidson E: Nerve root inflammation demonstrated by magnetic resonance imaging in a patient with transient neurologic symptoms after intrathecal injection of lidocaine. Anesthesiology 2002. 97:257-258.
- 52. Aldrete JA. Nerve root irritation or inflammation diagnosed by magnetic resonance imaging. Anesthesiology. 2003. 98:1294.
- 53. Lauretti GR. Pain transmission and modulation. In: Arachnoiditis: the silent epidemic..IA Aldrete (ed.), Denver Futuremed. 2000. pp 19-37.
- 54. Thelander V, Fagerland M, Friberg S, et al. Straight tests versus touch and pain afferents in single fascicles of the human ulnar nerve. Brain. 1990. 113: 1491-1500.
- 55. Nordin M, Nystrom B, Wallin U, and Habarth KE. Ectopic sensory discharges and paraesthesia in patients with disorders of peripheral nerves dorsal roots and dorsal columns. Pain. 1984. 20:231-245.
- 56. Ochoa JL. The newly recognized painful ABC syndrome Thermographie aspects. Thermology. 1986a. 2: 65-73; 308-316
- 57. Ochoa JL, Comstock WJ, Marohettini P, and Nizamuddin G. Intrafascicular nerve stimulation elicits regional skin warming that matches the projected field of evoked pain. Shaible, Vahle-Hinz (ed). Fine Afferent Nerve Fibers and Pain. Verlagsegesellschaft, Weinheim, 1987b. pp 476-479.
- 58. Powell HC, Myers RR, Costello ML, et al. Endoneurial fluid pressure in Wallerian degeneration. Ann Neurol. 1979. 5:550-557.
- 59. Myers P,R, Rydevick BL, Heckman HM, et al. Proximo-distal gradient in endoneurial fluid pressure. Exp Neurol. 1988. 102:368-370.
- 60. Howe JF, Loesscr JD, and Calvin WH. Mechanosensitivity of dorsal ganglia and chronically injured axons: A basis for the radicular pain of nerve root compression. Pain. 1977. 3:35-41.
- 61. Myers RR, Yamamoto T, Yaksh TL, et al: The role of focal nerve ischemia and Wallerian degeneration in peripheral nerve injury Produciong hyperesthesia. Anesthesiology. 1993. 73:308-316.
- 62. Rocha-Lauretti G. Pain Transmission and modulation. In Arachnoiditis: the silent epidemic. J.A. Aldrete (ed). Futuremed. Denver. 2000. pp 19-37.
- 63. Wall PD. The role of substancia gelatinosa as a gate control. In Bonia JJ (ed.) Pain. Raven Press. New York. 1980. pp 205-231.
- 64. Cassin B, Naidoo S, Ramsaroop R, et al. Immunolocalization of bradykinin receptors in human tissue. Inmuinopharmacology. 1997. 36:121-125.
- 65. Aldrete JA: Unpublished data.
- 66. Couto da Silva JM, Couto da Silva JM Jr, Aldrete JA. Body temperature and diaphoresis disturbances in a patient with arachnnoiditis. Anesth Analg. 2001. 93:1578-1579.
- 68. Akimov GA, Komahdenko NI, and Zinchenko VA. Activity of gamma-glutamyl transferase in the eerebrospinal fluid of various forms of cerebral arachnoiditis. Zh Nevropatol Psikiatr Im S S Korsakova. 1995. 86:682-684.
- 69. Cooper RG, Mitchell WS, Illingworth KJ, et al. The role of epidural fibrosis and defective fibrinolysis in the persistence of postlaminectomy back pain. 1991. 16:1044-1048.
- 70. Wall PD and Devor M. Surgery afferent impulses originate from dorsal root ganglia as well as from the periphery in normal and nerve-injured rats. Pain. 1983. pp 321-329.
- 71. Machettini P, Cline MA, and Ochoa JL. Innervation territories of touch and pain afferents in single fascicles of the human ulnar nerve. Brain. 1989. 78:213-216.
- 72. Mitchell C, Garrahy P, and Peake P. Postoperative respiratory morbidity: Identification and risk factors. Aust. NZ J.Surg. 1982. p 79.

- 73. Pearce AC and Jones RM. Smoking and perioperative morbidity. Anesthesiology. 1984. 61: 576-584.
- 74. Aldrete JA, Woodward ST, and Turk LH. Influence of cigarette smoking on the changes produced by spinal anesthesia on expiratory forced volume and flow rates. Anesth & Analg. 1973. 52:809-816.
- 75. Aldrete JA. Cessation of cigarette smoking by suggestion in the perianesthetic period. Anesthesiol. Rev. 1987. 14: 22-24.
- 76. Warner DO: helping surgical patient quit smoking: why, when and how. Anesth Analg 2005. 101:481-487.
- 77. Aldrete JA. Prognosis. In: Arachnoiditis: the silent epidemic. JA Aldrete (ed). Futuremed. Denver. 2000. pp. 273-280.

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