

PATIENT AND FAMILY HANDBOOK FOR ADHESIVE ARACHNOIDITIS

**By
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5th Edition

Information in this handbook can be shared if source credit is given.

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DEDICATION

This handbook and the research behind it would never have been written without the pioneering groundwork of Drs. Sarah Smith, Antonio Aldrete, and Charles Burton.

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CO-AUTHORS – “WE”

Who is Dr. Beak? A smart old buzzard who received his MD degree from the “Common Sense Medical School”.



Who is Nurse Rosey? A pesky, ever present nurse who wrote her nurse's thesis on “How to Shape Up Patients for Their Own Good”.

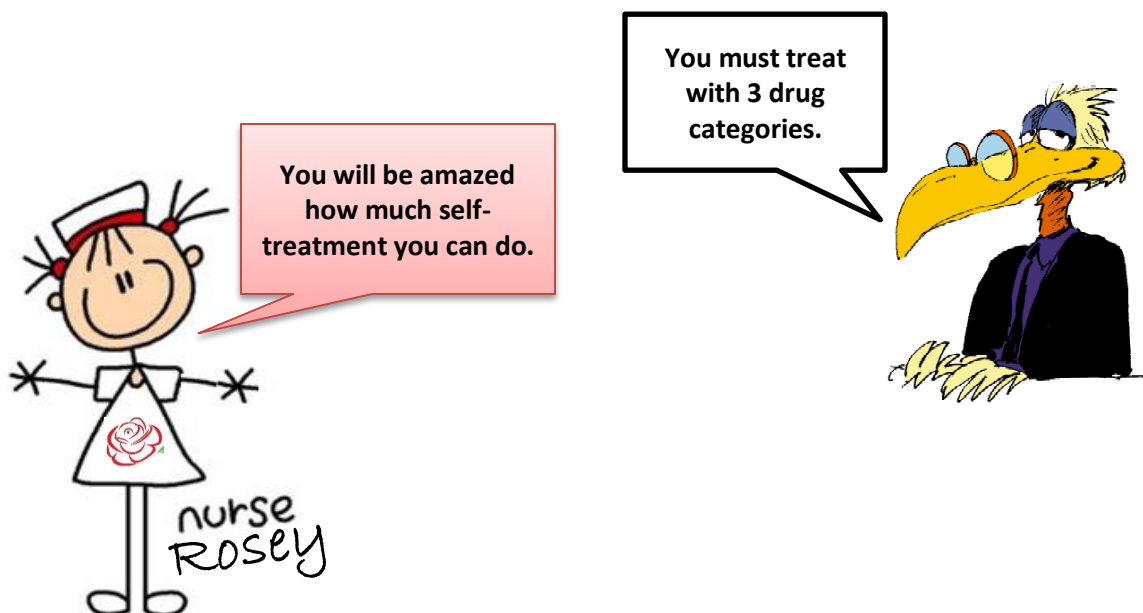
INTRODUCTION

This is our 5th handbook edition for patients and families with adhesive arachnoiditis (AA). Major goals of the “Arachnoiditis Research and Education Project” are to teach self-help and bring medical treatment, know-how to medical practitioners in every community. This goal is quite different from the one we initiated 5 years ago (1st Edition Handbook, 2013) which was to bring “awareness” of the disease to the World. Think back. Five years ago, the term “arachnoiditis” was considered a “spider bite” or a rare, hopeless, obscure disease that should be ignored and dismissed. We have pretty much achieved the “awareness” goal. If a medical professional practicing in 2019, including physicians, nurses, pharmacists, psychologists, and physical therapists, aren’t yet aware that AA is in every community and medical practice, I suggest they have been living under some rock. Let’s be clear. A high percentage of people who are diagnosed with “failed back syndrome”, who have had multiple surgeries or epidural injections, or who have a genetic, tissue degenerative disease like Ehlers-Danlos Syndrome likely have AA. Let’s also be clear that, after several years of research, AA can definitively and finally be recognized and treated.

The major message that I am trying to relate in this edition of the handbook is that AA is a neuroinflammatory disease that may develop dire complications and early death. To control it, three categories of medicinal agents must be simultaneously administered: (1) anti-neuroinflammation; (2) neuroregenerative/anabolic; and (3) pain control.

Our research over the past 10 years clearly reveals that patients and medical practitioners who try to avoid one of the 3 categories will not obtain much relief and recovery, and the patient will usually deteriorate.

Please share this handbook with any patient, medical practitioner, or other concerned party who wants to bring relief and recovery to AA patients.



CHAPTER ONE - HISTORY AND EXPLANATION OF ARACHNOIDITIS

1. Today's Arachnoiditis

Arachnoiditis means inflammation of the arachnoid which is the middle layer of the spinal canal covering, the outside layer being the dura and the inside, the pia mater. This covering is like a "pipe" that encompasses the spinal cord, nerve roots known as the "cauda equina", and fluid that flows up and down inside the canal.

The clinical term most used today is "adhesive arachnoiditis" (AA) which means that the spinal canal covering has attached itself to nerve roots by adhesions. Historically arachnoiditis has been caused by the infections, tuberculosis or syphilis, an injury to the spinal canal covering, or a diagnostic dye placed on the spinal canal. Today, however, the most common causes of AA are chronic spinal conditions such as slipped or protruding discs, spinal canal stenosis (narrowing), osteoporosis, kyphoscoliosis, and arthritis. These conditions may, over-time, compress nerve roots which results in friction, inflammation, and adhesion formation. Medical interventions to treat a chronic spine disorder can inadvertently accelerate the inflammatory and adhesion process. Combined factors such as aging, chronic spinal conditions, obesity, lack of exercise, and genetics can cause AA to develop. Unfortunately, AA may cause severe neurologic damage and severe pain as it may become a progressive, neuroinflammatory disease that entraps nerve roots and destroys nerve cells. Fortunately, new magnetic resonance imaging (MRI) done with contrast dye now permits physicians to suspect and diagnose AA in its early stage.

Besides pain, the common symptoms are leg weakness, bizarre sensations (e.g. bugs crawling) on the skin, and bladder and bowel dysfunction. Pain may be accelerated or relieved by changing positions. For example, persons with AA can't sit very long and may find relief by laying on the floor. In the past AA was considered a hopeless, progressive, and debilitating disease that could only be treated by symptomatic pain relief. Today, however, new therapeutic drugs and measures which suppress neuroinflammation and promote regeneration of nerves are bringing relief and recovery to AA patients.

2. Why A Need for This Handbook?

1. Until now arachnoiditis (ARC) has been considered a rare disease. No more. Its incidence is up several hundred-fold this past decade. Every community and medical practice now have cases, although they may not be recognized.
2. Adhesive arachnoiditis (AA) is a neuroinflammatory disease that not only has serious and even deadly ramifications, its treatment and control require very specific measures.
3. Treatment protocols have been recently developed thanks to abundant research on AA. At this time, however, only a few medical practitioners (MD's, NP's, PA's, DO's) are familiar with them.

Due to the 3 facts stated here, patients, families, and medical practitioners can aggressively diagnose and treat this disease.

3. Explanations and Definitions, You Must Know



You must know the "lingo".

Spinal Canal: The spinal canal consists of 4 components: (1) spinal cord; (2) nerve roots; (3) covering or lining; and (4) fluid. Think of the spinal canal as a closed pipe filled with structures bathed in fluid.

Central Nervous System (CNS): Brain, spinal cord, nerve roots of the cauda equina.

Nerve Roots: The actual spinal cord runs from the brain down to about the lumbar area. Below the spinal cord hangs about 2 dozen string-like structures called nerve roots. Collectively they are called the cauda equina. The nerve roots can become damaged, inflamed, clump together, and stick or adhere by adhesions to the arachnoid layer of the spinal canal cover. When sticking and adhesions occur, the term adhesive arachnoiditis is applied. If only enlargement, displacement, and clumping of nerve roots is seen on an MRI, the term "cauda equina syndrome" may be applied. Symptoms are similar to AA.

Neuroinflammation (NI): A special inflammation in the CNS.

Arachnoid (ARC): The cover or lining of the spinal cord is scientifically called the thecal sac or meninges. The inner layer is called the "Pia Mater". It is extremely thin and fragile. The outer layer is the dura which is thick and firm. The arachnoid is the middle layer. It contains blood vessels and inflammatory cells and can become inflamed if irritated or damaged.

Arachnoiditis: ARC is inflammation of the arachnoid layer of the spinal canal covering or lining which can be caused by trauma, infection, toxins, or friction between the covering and spinal cord or nerve roots. ARC most commonly develops in the lumbar spine area, but it also occurs in the cervical (neck) spine area. ARC has an official code listed in the international code of diseases (ICD-10, G03.9).

Adhesive Arachnoiditis (AA): This condition is present when there are adhesions between the arachnoid layer and the nerve roots in the cauda equina. Adhesions are seen on contrast magnetic resonance imaging (MRI). (ICD-10, G03.9)

Tarlov Cysts: A cyst or outpouching of a spinal nerve root. (ICD-10, G96.19) They are often called "perineural" cysts. Tarlov cysts are frequently associated with Ehlers-Danlos/Hypermobility Syndrome.

4. History of Arachnoiditis

- The word Arachnoid refers to a spider's web.
- The arachnoid is the second layer of the meninges, or neuraxis, the 3 protective layers covering the spinal cord. Web-like, it is located in the middle, between the dura and pia mater layers.
- The arachnoid layer is a fine, fragile, and cobweb-like tissue. When the arachnoid becomes inflamed this condition is known as the disease "arachnoiditis".

- The exact year the disease, “arachnoiditis”, was named is uncertain, however, the 1873 “Comprehensive Medical Dictionary”, published by J.B. Lippincott & Co., included this definition of arachnoiditis: “A faulty term, denoting inflammation of the arachnoid membrane”.
- In 1869 the famous neurologist, Dr. Charcot and his colleague first described a syndrome we now call the disease, arachnoiditis. The causes of the disease at this period in history were infections, primarily tuberculosis and syphilis.
- Dr. Addison, the physician who discovered adrenal failure, published his findings on 11 autopsied patients in 1855. Two cases had severe pain, atrophied adrenals and calcium deposits and fluid around the arachnoid layer, suggesting that long-standing, end-stage arachnoiditis was the likely cause of pain and adrenal failure.
- The first recorded attempt to treat arachnoiditis was probably in 1781 when Dr. John Fothergill, a renowned British physician, treated a patient with severe back and sciatic pain who had other symptoms compatible with arachnoiditis (ARC). Opioids had failed to relieve his patient’s pain, but he obtained positive results with a mercury concoction called calomel.
- Between 1930 and 1990 pantopaque and other oil-soluble dyes were infused into the spinal canal for diagnostic (myelogram) purposes. A small percentage of these patients developed ARC and other neurologic complications.
- Magnetic Resonance Imaging (MRI) replaced oil-based dyes in the late 1980’s, and ARC subsequently became a rare, unappreciated disease as toxic dyes were no longer used.
- Beginning around 2000 an extended life span, with increasing rates of chronic spinal conditions such as herniated discs and arthritis plus the increasing use of invasive medical interventions and surgeries to treat them, began fueling an increase in the incidence and prevalence of ARC that continues to this day.

5. Major Causes of Arachnoiditis (ARC)

- ✓ Today the most common causes are spine disorders related to aging, accidents, obesity, bucket seats, lack of exercise, poor posture, and the invasive medical interventions and procedures that are now used to treat common spine disorders.

Genetic Degenerative Disease

The arachnoid and pia mater layers of the spinal covering are thin, soft, and easily damaged. Tissue degenerative diseases, particularly Ehlers-Danlos, Marfan, and hypermobility syndromes may cause micro-tears in the arachnoid layer which lead to inflammation, cysts (Tarlov), and adhesions.

Autoimmune Disease

Many ARC patients have autoimmune diseases such as systemic lupus, psoriasis, or rheumatoid arthritis. ARC may be a direct result from autoimmunity.

Viruses

Some viruses are highly suspected to cause ARC. Epstein Barr is one possibility believed to do this.

Common Spine Disorders

Common spine disorders including chronic herniated discs, stenosis, osteoporosis, and vertebral arthritis may, over-time, cause nerve roots in the cauda equina to rub or be squeezed together causing friction (“sand-paper effect”), inflammation and adhesions.

Trauma: A puncture, tear, or traumatic injury to the arachnoid lining from an accident, needle puncture, or chemical irritant may initiate ARC. Inflammation and adhesions in the arachnoid lining may later capture the nerve roots that are close to the inflamed site and “glue” them to the lining with adhesions.

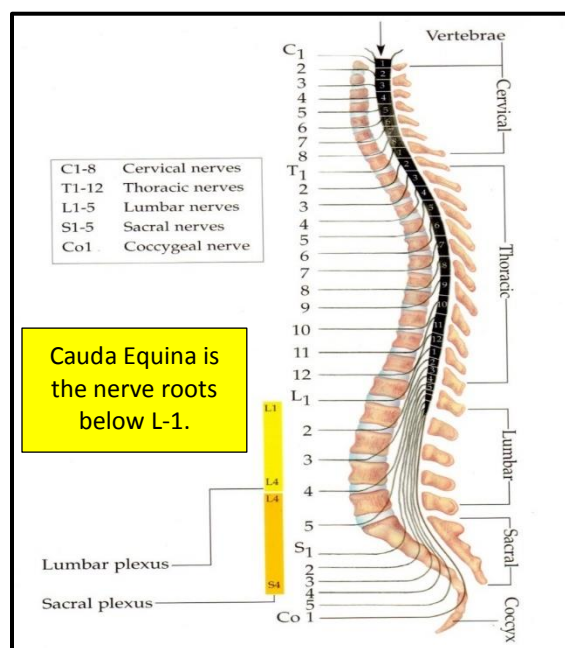
6. Spinal Conditions That May Cause AA

Herniated discs
Scoliosis
Collapsed vertebrae
Degenerative osteoarthritis

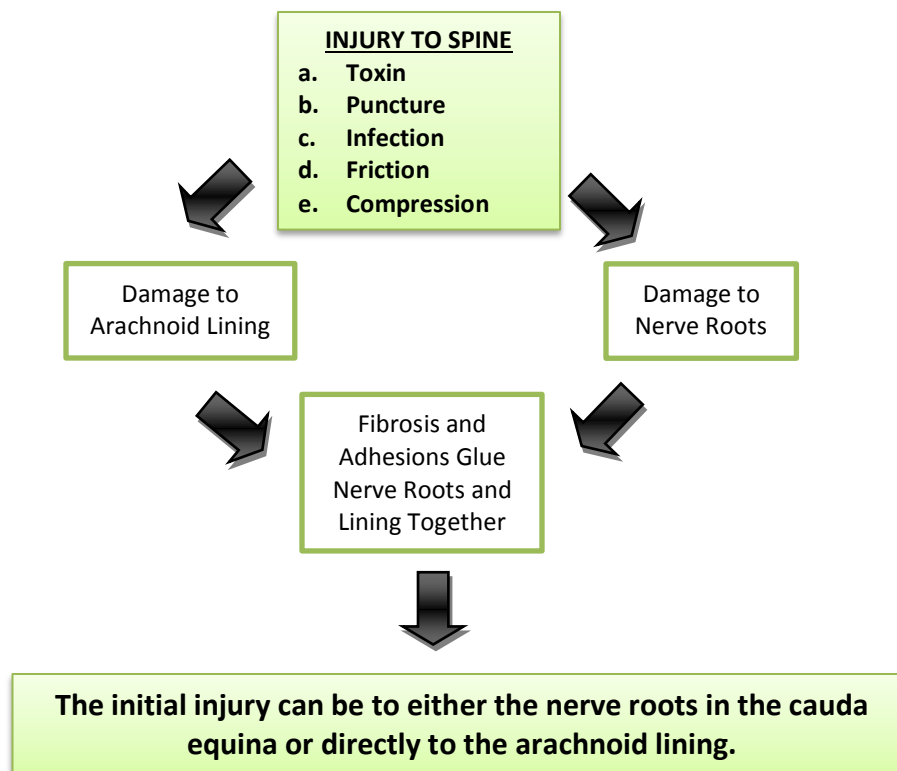
Spinal stenosis
Osteoporosis
Spondylolisthesis
Rheumatoid Spondylitis

7. The Cauda Equina Nerve Roots and AA

- Until now arachnoiditis (ARC) was considered a rare disease. No more. Its incidence is up several hundred-fold this past decade. Most every community and medical practice has cases.
- The cauda equina is comprised of about 2 dozen nerve roots that hang suspended in spinal fluid.
- AA occurs when some nerve roots in the cauda equina adhere, by adhesion, to the spinal canal covering rather than float freely in spinal fluid.
- Any condition such as multiple protruding intervertebral disc that push the nerve roots together may cause friction and irritation followed by neuroinflammation and adhesion formation.



8. Two Ways That AA May Develop



There is a myth and old belief that arachnoiditis only occurs when the spinal canal covering (arachnoid is the middle layer) is damaged by puncture or other insult.

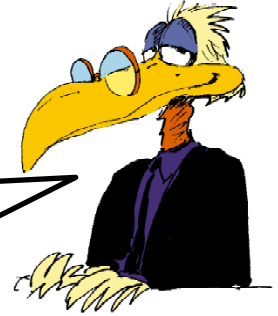
9. The Key Message: AA Is a Neuroinflammatory Disease

New research has shown that inflammation may develop in the CNS. It is caused by a group of cells called glia that are not found outside the CNS.

Glia cell inflammation is called “neuroinflammation” to call attention to the fact that it is not the same inflammation found in arthritis, allergies, or skin infections.

Since neuroinflammation is not regular inflammation, common anti-inflammatory drugs such as aspirin, Motrin®, or Advil® will not usually have much effect. AA is a neuroinflammatory disease that requires specific medications to suppress or control it. A failure to take some of these medications will usually allow AA to progress.

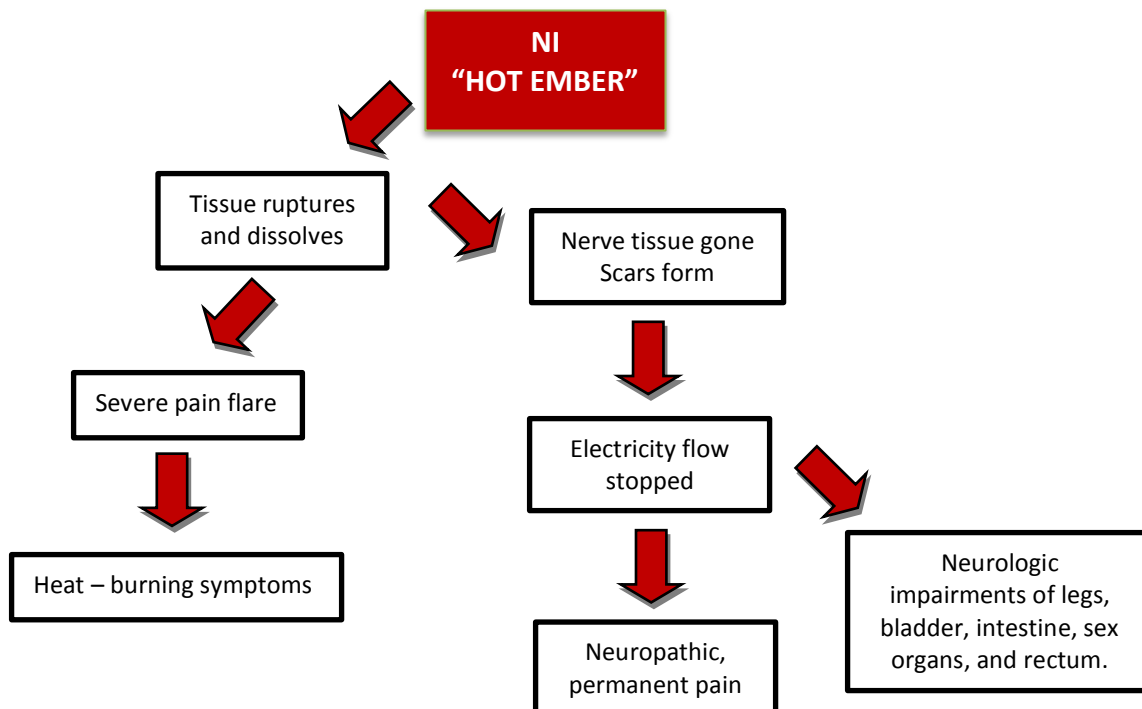
Neuroinflammation diagnosis and treatment is the key to successful control of AA.



10. How Neuroinflammation (NI) Causes You to Deteriorate

NI is fundamentally a hot ember or spot that causes nerve tissue to dissolve.

NI IS RESPONSIBLE TO THE 2 BASIC KINDS OF PAIN IN AA



11. Natural Course and Outcome of Adhesive Arachnoiditis (AA)

3 Possible Outcomes of Untreated AA

- Resolves
- Intermittent symptoms, impairments, and pain
- Progressive neurologic symptoms, impairments, and pain (e.g. wheelchair, bed-bound, early death)

LIFE SPAN: ONE STUDY REPORTS THAT AA SHORTENS A LIFE-SPAN AN AVERAGE OF 12 YEARS.

Ref: Guyer, et al. The long-range prognosis of arachnoiditis. Spine, 1989;14(12):1332-41.

The course and outcome of AA is directly related to the severity of neuroinflammation and the measures that MD’s, NP’s, and patients take to alter it.

Why Early Death Occurs in AA Patients

- Poor mobility
- Obesity, diabetes, hypercholesterolemia
- Endocrine (hormone) suppression
- Immune impairment with infections
- Hypertension
- Intestinal digestive defects with malabsorption

THE GOOD NEWS

We now know that the natural course and outcome of AA is greatly altered with suppression of neuroinflammation, promotion of neuroregeneration, and measures that normalize spinal fluid flow.

Final Cause of Death: (1) Cardiac arrest; (2) Adrenal failure; and (3) Overwhelming infection.

12. Self-Determination Test - Do You Have Neuroinflammation?

Answer each question based on your feelings and symptoms in the past week.

- | | | |
|---|------------------------------|-----------------------------|
| 1. Do you have periods of heat? | <input type="checkbox"/> Yes | <input type="checkbox"/> No |
| 2. Do you have periods of sweating? | <input type="checkbox"/> Yes | <input type="checkbox"/> No |
| 3. Do you feel like your body has too much electricity or “shock” at times? | <input type="checkbox"/> Yes | <input type="checkbox"/> No |
| 4. Do you have periods of burning in your feet, hands, pelvis or buttocks? | <input type="checkbox"/> Yes | <input type="checkbox"/> No |
| 5. Do you have periods or episodes of strong feeling on your skin like bugs crawling or pin stabbing? | <input type="checkbox"/> Yes | <input type="checkbox"/> No |
| 6. Are you sensitive or become nauseated and dizzy in heat such as a hot summer day? | <input type="checkbox"/> Yes | <input type="checkbox"/> No |
| 7. Do the areas over pain sites sometimes become red and hot? | <input type="checkbox"/> Yes | <input type="checkbox"/> No |
| 8. Does your temperature rise at times? | <input type="checkbox"/> Yes | <input type="checkbox"/> No |
| 9. Are your pain flares accompanied by sweating and heat? | <input type="checkbox"/> Yes | <input type="checkbox"/> No |
| 10. Do you have periods of stabbing, shooting, or jerking pains? | <input type="checkbox"/> Yes | <input type="checkbox"/> No |
| 11. Do you have recurrent pain flares you can’t control? | <input type="checkbox"/> Yes | <input type="checkbox"/> No |

INTERPRETATION: If you answered yes to over half of the above questions, you will most likely need specific treatment for neuroinflammation.

Take this test. If you have neuroinflammation, you must take quick action to stomp on it.



CHAPTER 2 – DIAGNOSIS AND COMPLICATIONS

1. Common Symptoms of AA

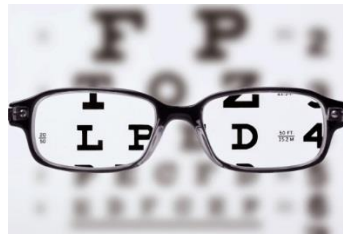
There are 4 fundamental physiologic causes of symptoms and impairments in AA patients: (1) neuroinflammation; (2) nerve clumping or trapping; (3) spinal fluid flow interference; and (4) nerve root destruction.

But which patients have arachnoiditis?

ARC patients have a peculiar and unusual set of symptoms that are not usually present in other back pain patients.

- | | |
|-----------------------------------|---|
| • Urinary Hesitancy, Poor Control | • Can't Stand or Sit Long in One Position |
| • Water Dripping on Legs | • Burning Feet |
| • Blurred Vision | • Constipation/Diarrhea |
| • Headaches | • Pain Walking Stairs |
| • Pins and Needles in Legs | • Weak Legs |

Some Strong Arachnoiditis Symptoms



2. Major Symptoms and Harms of AA

Nerve Entrapment and Destruction: Weird skin sensation, burning feet, can't sit or stand in one position very long, bowel/bladder difficulties, pain (comes in waves or is always present).

Spinal Fluid Flow Obstruction: Headache, blurred vision, ringing in ears.

Spinal Fluid Leakage: Back pain, decrease in ability to extend arm(s) & leg(s), difficulty walking.

Autoimmune Disorder: Arthritis, muscle aches, cardiac disorders, seizures.

Retained Electricity: Vibrations, tremors, overheating episodes, sweating.

Hormone Suppression: Weakness, fatigue, poor pain control, depression.

KEY MESSAGE: The sooner treatment with "curative" agents is started simultaneously with "symptomatic" agents for pain control, the prevention of symptoms and harms will start.

3. Self-Test - Do You Have Adhesive Arachnoiditis?

If you answer yes to 12 or more, you most likely have adhesive arachnoiditis.

		YES	NO
1.	Does it hurt to lie flat on your back?		
2.	When you stand with your leg straight and raise it, does this cause pain in your back?		
3.	Do you lose water (bladder) or stool (colon) without warning?		
4.	Does standing too long cause so much pain that you have to sit or lie down?		
5.	Do you have periods or episodes of intense sweating or heat (temperature)?		
6.	Do you sometimes have to stand to relieve your pain?		
7.	Do you sometimes have shooting pains, tremors, or jerks in your legs?		
8.	Do you have to sometimes sleep sitting up?		
9.	Do you sometimes have pain behind your eyes?		
10.	Do you have trouble starting your bladder to urinate or bowel to defecate?		
11.	Is your pain constant (always present)?		
12.	Is your vision ever blurred?		
13.	Have you ever collapsed while standing or walking?		
14.	Are your hands and/or feet cold a lot of the time?		
15.	Do you get twitching or crawling feelings over your back and spine area?		
16.	Do you get burning or electrical pains in your feet?		
17.	Do you have to sit on a pillow or cushion at times?		
18.	Do you have pain when you walk up steps?		

A final, clinical diagnosis of AA requires typical symptoms, physical findings, and laboratory tests. A contrast MRI is needed to confirm the diagnosis.

SPECIAL MESSAGE: The earlier the diagnosis in the course of the disease, the better the results of treatment.

4. Four Categories of Arachnoiditis - Which One Fits You?

Mild

- ✓ Occasional pain controlled by natural pain relievers and neuroinflammatory agents
- ✓ Full extremity range of motion
- ✓ Normal serum C-reactive protein (CRP) & erythrocyte sedimentation rate (ESR)
- ✓ No spinal fluid flow interference or leakage on MRI
- ✓ No bladder impairment
- ✓ Total recovery may occur

Moderate

- ✓ Pain requires intermittent, low dose, intermittent pain relievers, and neuroinflammatory and neuroregenerative agents
- ✓ Full extremity range of motion
- ✓ Normal serum CRP & ESR
- ✓ Adhesions and spinal fluid flow interference on MRI
- ✓ Some bladder hesitancy or urgency

Severe

- ✓ Constant pain requiring regular use of symptomatic pain relievers
- ✓ Some decreased range of motion in lower extremities plus weakness and some bed-bound hours
- ✓ Elevation of serum CRP or ESR
- ✓ MRI shows adhesions, spinal fluid flow obstruction, and leakage
- ✓ Bladder hesitancy/urgency
- ✓ Burning feet
- ✓ Some serum hormone deficiencies

Catastrophic

- ✓ Constant pain and flares requiring daily use of pain relievers
- ✓ Impairment of walking and standing, bed-bound hours
- ✓ Decreased range of motion in upper and lower extremities
- ✓ Elevation of serum CRP or ESR
- ✓ MRI shows adhesions, spinal fluid flow obstruction and leakage
- ✓ Bladder hesitancy, urgency, and some incontinence
- ✓ Headaches, blurred vision, mental confusion
- ✓ Serum hormone deficiencies

Cervical neck
arachnoiditis is
treated like lumbar-
spine arachnoiditis.



These categories naturally over-lap and treatment may require differences.

5. Diagnosis of Cervical (Neck) Arachnoiditis

The diagnosis of cervical (neck) AA is clinical and a physician's judgement. Since the cervical spine does not have nerve roots, the MRI is not as specific or as confirmatory as with lumbar AA. Adhesions are essentially not seen in modern day cervical AA. We have developed these criteria to make the diagnosis of cervical neck AA.

Criteria:

1. Severe pain which increases with forward flexing or backward extension of the neck.
2. History of trauma or disease involving the neck.
3. A contrast MRI shows little or no passage of spinal fluid on one side of the spinal cord. (Spinal fluid obstruction occurs because the arachnoid lining thickens. This MRI finding may sometimes be called stenosis.)
4. Blood tests may show elevated inflammatory markers.
5. One or more arms show weakness, decreased reflexes, or diminished range of motion.

Cervical neck arachnoiditis is grossly underdiagnosed. Patients are routinely told that they have neck arthritis, degeneration, or cervical radiculopathy.

BIG PROBLEM: A delay in diagnosis and treatment must be avoided because arachnoiditis is an inflammation of the spinal canal lining, and it will usually progress and worsen without treatment.

6. Centralized Pain – You Must Know If You Have It

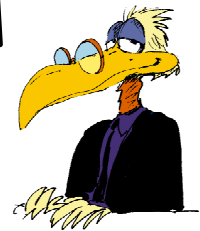
Each AA patient must ask themselves this question. Why? If your pain has centralized, you will likely have to take some special measures to stay comfortable and function.

Just What is Centralized Pain? Pain is a form of electromagnetic energy that can get trapped in the biologic matrix formed by glial cell activation and neuroinflammation. When this occurs the terms “intractable”, “central sensitization”, or “persistent” may be used to describe pain.

Hallmarks of Centralized Pain: You have it if your pain is constant. In other words, if your pain is intermittent or fluctuates, it has probably not centralized. In addition to constancy you will probably have insomnia to the point you will likely require a sleep aid. We have included a questionnaire you should take as it gives you some other information about other symptoms of centralized pain including heat episodes and sweating.

What to Do If You Have AA and Centralized Pain: You must take agents from three medications groups: (1) neuro-anti-inflammatory; (2) neuroregeneration; and (3) pain control.

You must aggressively take medication from all 3 groups.



Secondary Measures That May Be Necessary: Patients with centralized pain have a condition called “descending pain”. Normally pain goes from a disease or injured site on the body and then travels up (ascending) the nervous system and spinal cord to the brain. Centralized pain acts as an “electric battery” that sends pain down (descending) the spinal cord and nervous system.

To control descending pain, you will likely have to take a drug from one or both of these classes of drugs:

1. Stimulant Related to Caffeine
 - a. Methylphenidate (Ritalin®)
 - b. Amphetamine Salts (Adderall®)
 - c. Phentermine
2. Benzodiazepine or Some Muscle Relaxants
 - a. Clonazepam (Klonopin®)
 - b. Diazepam (Valium®)
 - c. Carisoprodol (Soma®)
 - d. Tizanidine (Zanaflex®)

There are a lot of good non-prescription drugs for self-help.



Note: Medical practitioners do not like to prescribe most of the drugs listed above because they can be abused and lead to overdose. Family members, not the patient, may have to assure the practitioner that the drugs will be taken only “as prescribed” and will be safely kept away from teenagers and visitors.

Non-Prescription Aids

These aids may substitute for the above-mentioned agents. Obtain these at a health food store:

1. Valerian Root: The name “valium” comes from this herb.
2. Garcinia Cambogia Extract: This is an herbal extract that is a mild stimulant that helps control weight.

7. Do You Have Centralized Pain?

If you have constant pain and answer “yes” to half or more of the questions, you can assume you have centralized pain.

1. Is your pain constant (“never leaves”)? Yes No
2. Do you have insomnia? Yes No
3. Do you have periods of great sweating? Yes No
4. Do you have periods when your temperature goes up (feel hot)? Yes No
5. Are your hands and/or feet usually cold? Yes No
6. Do you have periods that you have difficulty reading, analyzing, or remembering? Yes No
7. Do you have periods when you can’t smell, taste, or hear? Yes No
8. Do you sometimes have a lot of electricity?
(Shock others, burn out lights or watches) Yes No
9. Are you always “fatigued” even if you get some sleep? Yes No
10. Does some of your pain move from one location to another? Yes No
11. Do you have jerking or tremors? Yes No
12. Does the skin over your pain site really hurt if you touch or rub it? Yes No
13. Does water hitting or splashing on your skin irritate or cause you pain? Yes No

8. Do You Have Ehlers-Danlos/Hypermobility Syndrome?

AA occurs frequently in persons who have a genetic disorder called Ehlers-Danlos and/or Hypermobility Syndrome. These genetic disorders have a built-in gene that causes soft (non-bone) tissues to progressively deteriorate, degenerate, and form micro-tears in tissue. Tissues most affected are those that are known as “connective” in that they support such organs as the intestine, joints, skin, blood vessels, and spinal cord. Fundamentally, the disorder causes tissue to stretch, loosen, and collapse due to the dissolution of collagen in the tissue. Unfortunately, the spinal canal has many soft connective tissues like the pia mater and arachnoid. They may collapse and cause an array of spinal conditions commonly known as Chiari, Tarlov Cysts, tethered cord, and AA.

How Do I Determine If I Have EDS/Hypermobility?

You should particularly suspect you have this disorder if you developed AA without any significant trauma or surgery. The diagnosis of this disorder is made solely by history and your ability to hyperextend your skin and joints. The EDS Society has an established set of criteria for the diagnosis, and it can be obtained over the internet.

Self-Determination Screen for EDS/Hypermobility

If you answer “yes” to 3 or more of the questions, you most likely have it.

- Yes No Can you now (or could you ever) place your hands flat on the floor without bending your knees?
- Yes No Can you now (or could you ever) bend your thumb to touch your forearm?
- Yes No As a child did you amuse your friends by contorting your body into strange shapes or could you do the splits?
- Yes No As a child or teenager did your shoulder or kneecap dislocate on more than one occasion?
- Yes No Do you consider yourself double jointed?

Importance of Diagnosis of Eds/Hypermobility Syndrome

If you have an EDS/Hypermobility Syndrome you will need to be on extra neuroregenerative and anabolic (tissue growth) measures. EDS can be progressive, very painful, and debilitating, so it is critical to know if you have it.

9. Pretreatment Evaluation

We highly recommend a clinical evaluation prior to initiating treatment with the protocols outlined in this handbook.

Here Are Our Pretreatment Recommendations:

1. Physical exam: If back shows indentation, muscle asymmetry, or arms and legs can't fully extend, spinal fluid leakage is occurring or has occurred.
2. Blood test for inflammatory markers: (a) ESR; (b) CRP; and (c) cytokines. If any of these are elevated, it is evidence that neuroinflammation is out-of-control.
3. Blood test for 6 hormone levels: cortisol, pregnenolone, DHEA, progesterone, estradiol, testosterone. If any of these are low, they need to be replenished.
4. Neuroinflammatory clinical test – 2 options: (a) Ketorolac (Toradol®), take a 30 mg injection for 2 days in a row; or (b) Medrol® 6-day dose pack (methylprednisolone).

If either ketorolac or Medrol® reduce pain, insomnia, fatigue, or loss of appetite and simultaneously improve function and energy, a diagnosis of excess NI is essentially made and one or both of these NI agents should be continued.

NOTE: The above evaluation is also what we recommend if a long-term AA patient begins to deteriorate or their current treatment isn't working to control pain and improve function.



A pretreatment evaluation is ideal, but if you can't be evaluated start our "self-help" protocol. Don't wait!

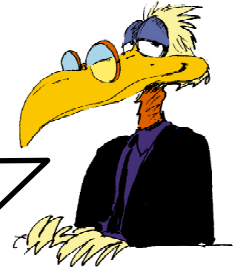
CHAPTER THREE – MEDICAL TREATMENT THAT WORKS FOR MOST PATIENTS

1. Major Goal of Treatment: Stop Progressive Deterioration

AA is a NI disease of nerve roots and the spinal canal covering. The neuroinflammation of the AA may take any of these paths:

1. Total recovery or resolution;
2. Cause progressive neurologic damage such as to involve the bladder, intestine, sex organs, or legs.

If you have developed nerve root clumping and adhesions as seen on your MRI, you should plan on being on an indefinite, treatment program. If you choose to ignore possible progression and crucial treatment, you can find yourself having sudden, without warning, neurologic impairments.



The medical treatment recommended in this handbook cannot be guaranteed as far as outcome. Unless an AA patient could not or would not access the 3-component program we use, they at least stopped deterioration and experiences better pain relief and function. Many AA patients have found what we term “near-cure”. We are hesitant to use the term “cure” as NI may remain almost asymptomatic and re-emerge later.

POSSIBLE OUTCOMES OF UNTREATED AA

2. Basics of AA Treatment – Consists of Two Types

Medicinal Agents

1. Anti-neuroinflammatory
2. Neuroregenerative/anabolic
3. Symptomatic pain relief

Physiologic Measures

1. Spinal fluid flow exercises
2. High protein anti-inflammatory diet
3. Contraction prevention exercises
4. Electromagnetic techniques

The following pages in this handbook provide multiple medicinal measures in each category. It is well-recognized that “one size does not fit all” and that medical practitioners, patients, and families have individual desires and biases.

Although this handbook gives multiple options of medicinal agents, the ones listed have shown effectiveness in AA. Be clearly advised. Only a small number of medicinal agents will cross the blood brain barrier and provide anti-neuroinflammatory and neuroregenerative effects.

There Are 4 Basic Outcomes With AA:

1. Total recovery or resolution
2. Burn out leaving nerve damage behind;
3. Go into remission and intermittently emerge with severe pain flares and more NI that causes further damage;
4. Worsening pain and neurologic/autoimmune impairments continue to a bed-bound state and early death.

How Does Recovery Occur?

When neuroinflammation and adhesions form between the nerve roots and arachnoid layer, the diseased area is soft and amenable to resolution and recovery if the NI is successfully treated and suppressed. Unfortunately, NI may form a scar and either trap and/or damage small nerve fibers which may cause pain and dysfunction of nerves that control such organs as the bladder and legs. Once a scar forms, we believe that small nerve fibers can grow around or through the scar and provide pain relief and improve nerve function.

3. The Earlier the Treatment—The Better the Outcome

Our conclusion in evaluating and treating hundreds of AA cases is that the earlier treatment is started, the better the result or outcome.

WHY? NI causes adhesions to form between the lumbar nerve roots and the arachnoid lining or covering of the spinal canal. In the early stage of AA, adhesions and NI can be at least partially resolved. After a time, however, NI and adhesions not only cause permanent nerve damage, the area of neuroinflammation and adhesions cause clumping, scarring, and even calcification which is not resolvable.

None of the medications recommended here need to be taken daily.

We recommend that medication be taken 2 to 5 days a week and daily use should be done only if intermittent administration is not providing enough relief and recovery.



4. The Anti-Inflammatory Agents

For an agent to be effective in controlling the NI of AA, it must be able to cross the blood brain barrier and suppress the over-activity of glial cells.

Most Potent

Prescription Agents

Low dose naltrexone*

Ketorolac (injection or troche)

Medrol® (methylprednisolone)

Dexamethasone

Non-Prescription Agents

Curcumin/turmeric

Serrapeptase

Bovine adrenal extract

Less Potent

Prescription Agents

Acetazolamide

Pentoxifylline

Minocycline

Metformin

*A potent pain reliever. Cannot be taken with opioids.

Special Notes:

1. Dosage of non-prescription agents are on their label. Starting specific dosages of prescription agents are listed in our protocols for medical practitioners which are available on request.
2. More than one agent can and should be taken in most cases.

5. The Neuroregenerative Agents

In order to be on an effective neuroregenerative (anabolic) agent, it must cross the blood brain barrier and stimulate the growth of damaged or degenerated nerve cells.

Prescription agents: (1) human chorionic gonadotropin (HCG); human growth hormone (HGH); and (3) nandrolone.

Non-prescription agents: (1) DHEA (dehydroepiandrosterone) 200 mg or more a day; (2) pregnenolone 100 mg or more a day; (3) bovine gonadal extract (Orchex® or other); (4) colostrum; and (5) deer antler velvet extract.

Special Notes:

1. Dosages of non-prescription agents are on their label. Starting prescription agent dosages are in our protocols for medical practitioners, and they can be obtained on request.
2. One or more agents can be simultaneously used, if necessary.

6. Pain Relievers – Multiple Types May Be Needed

AA patients will have 3 kinds of pain all of which can flare: (1) neuroinflammatory; (2) neuropathic (nerve damage); and (3) centralized and declining.

Prescription Non-Opioid

Low dose naltrexone (LDN)
Oxytocin
Ketamine
Clonidine

Neuropathic Non-Opioid

Gabapentin
Topiramate
Pregabalin (Lyrica®)
Duloxetine (Cymbalta®)
Tizanidine (Zanaflex® or other)
Carisoprodol (Soma®)

Prescription Centralized/Descending Pain

Amphetamine Salts (Adderall®)
Methylphenidate (Ritalin®)
Phentermine

Non-Prescription

Kratom
Palmitoylethanolamide (PEA-Comfort Max® or other)
Cannabinoid derivatives

Topical Skin

Lidocaine gel or patch

Opioids (Last resort)

Tramadol
Hydrocodone
Codeine
Morphine
Oxycodone
Buprenorphine (Suboxone® or other)
Hydromorphone (Dilaudid®) injectable is used if oral opioids are ineffective.

Pain relievers from the 3 different groups need to be used each week.



SPECIAL NOTES:

1. Only those agents that we recommend are listed here although others may be effective.
2. All pain relievers are symptomatic and not curative. They should only be taken if a patient is also on anti-neuroinflammatory and neuroregenerative agents. Otherwise, the dosages of symptomatic pain relievers will escalate and may reach a non-effective state.
3. Centralized pain is present if pain is constant. Descending pain means the CNS is sending out pain signals, and stimulants and other non-opioid agents are necessary for control.
4. Kratom is a non-prescription opioid.
5. PEA is a nutrient and pain reliever. It also has some anti-inflammatory actions.
6. LDN is the only agent that is a potent anti-inflammatory agent and pain reliever. Cannot be taken with opioids.

7. Symptomatic Versus Curative Care: The Big Confusion

Most AA patients do not understand the difference between “symptomatic” and “curative” drugs. The majority are taking multitudes of “symptomatic” but few “curative” drugs, AND THEY wonder why they are getting worse!

Critical to Know

- Symptomatic Drugs: provide temporary relief from pain, fatigue, depression, and hopelessness.
- Curative drugs directly attack and reduce the severity of a disease (e.g. neuroinflammation) to achieve a curative effect; either partial or total.

Reality

AA patients need both classes of drugs, but they are currently overloaded on symptomatic and light on curative agents. Symptomatic measures help one to function, but they don’t attack the disease.

<u>SOME CURATIVE AGENTS</u>	<u>SOME SYMPTOMATIC AGENTS</u>
Low dose naltrexone, ketorolac, methylprednisolone, dexamethasone, adrenal extract, acetazolamide, minocycline, pentoxifylline, metformin, curcumin/turmeric, Serrapeptase, palmitoylethanolamide (PEA), human chorionic gonadotropin, nandrolone, estradiol, DHEA, pregnenolone, progesterone, testosterone	Gabapentin, pregabalin (Lyrica®), duloxetine (Cymbalta®), clonidine, oxytocin, ketamine, oxycodone, fentanyl, hydrocodone, CBD oil, lidocaine, morphine, hydromorphone (Dilaudid®), clonazepam, carisoprodol (Soma®), alprazolam, topiramate, kratom, amphetamine salts (Adderall®)

What Disease Are We Treating?

AA is a NI disease of spinal nerve roots and spinal canal lining. Unless curative agents are taken, the disease will progressively entrap nerve roots and destroy nerve and spinal tissue.

Check out these lists. They are partial and will undoubtedly change over time, but every AA patient needs to be on some curative agents.

Note: If you are not taking 2 or more “curative” agents, you can expect your condition to worsen and deteriorate.

8. Neuroregeneration (NR): Essential Treatment For AA

Neuroregeneration (NR), sometimes called neurogenesis, is the new essential treatment measure for AA. NR simply means regeneration or regrowth of damaged or dead nerve cells.

Our research on ARC is clear. A person with ARC and/or AA must aggressively and daily take medical and other measures to regrow the damaged and dead nerve cells caused by NI.

Here is the problem. NI can cause a negative (“catabolic”) state in which nerve cells are overheated and degenerate. Although one’s natural regrowth and regenerative (“anabolic”) mechanisms will regrow some of the damaged or dead nerves, it may not keep up with the NI of ARC and AA. Therefore, one must, in our opinion, do the following each day:

1. Simultaneously take neuroregenerative (“anabolic”) and anti-neuroinflammatory medicinal agents;
2. Eat a high-protein (anti-inflammatory) diet;
3. Spinal fluid flow exercises;
4. Electromagnetic measures.

SPECIAL NOTE: Active NI usually has symptoms of heat, burning, sweating, and strange skin sensations but not necessarily. NI can silently and unexpectedly cause tissue degeneration, so ARC and AA patients must continually promote neuroregeneration.

9. Emergency Treatment for Early or Suspected Arachnoiditis (AA)

Indication: A patient who has these symptoms within about 60 days after an epidural or spinal tap.

Lumbar pain plus 2 of these symptoms:

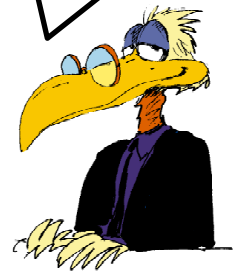
1. Burning/painful feet
2. Bladder hesitancy/difficult to empty, can't hold urine
3. Can't sit or stand in one place over 10 minutes
4. Headache
5. Blurred vision
6. Tinnitus (ringing in ears)

Treatment:

1. 6-day methylprednisolone (Medrol®) dose pack
2. Ketorolac (Toradol®) – 30 – 60 mg injection, daily for 3 days

Option: Medroxyprogesterone (PO) – 10 mg BID for 5 days, or minocycline 100 mg BID for 5 days

Any patient who has had a spinal tap or epidural injection and has severe pain and other neurologic symptoms, is a candidate for emergency treatment.



Interpretation: If the patient's pain and some other symptoms improve, a diagnosis of early arachnoiditis, cauda equina inflammation, or other CNS neuroinflammatory process has essentially been established. If, at the end of emergency treatment, clinical improvement is apparent, we recommend use of our regular AA protocol. If no improvement occurs, the logical conclusion is that pain and other symptoms are non-inflammatory.

10. Therapeutic Trials of Anti-Neuroinflammatory Drugs

The most common questions we get are: (1) Do I have AA? (2) If I have it, what do I do now? And (3) Why has my pain control stopped working?

If you or an acquaintance relates to one of the above 3 questions, you are a candidate for therapeutic trials with low dose naltrexone, ketorolac or Medrol® (methylprednisolone).

Low dose naltrexone, ketorolac, and methylprednisolone have been the most consistent, effective drugs to control the neuroinflammation.

Ketorolac: Take an injection or troche of 30 to 60 mg on 2 consecutive days.

Methylprednisolone: Take a 6-day dose pack (Medrol®).

Interpretation: If, after or during the therapeutic trial, you feel better, sleep more, have increased energy, less pain or other positive feelings, you know that you have uncontrolled neuroinflammation. In this case you need to add low dose naltrexone (LDN), ketorolac, and/or methylprednisolone to your treatment protocol.

Starting Usual Dosages

- a. Low dose Naltrexone .5 to 1.0 mg a day. **LDN cannot be taken with opioids.**
- b. Ketorolac 30 mg by troche or injection on 1 to 3 days a week;
- c. Methylprednisolone 4 mg, oral tablet on 3 to 5 days a week.

11. Flare Emergency Treatment

Every AA patient needs to be prepared for a severe flare. You need to break it fast not just for comfort but to prevent more damage and deterioration.

Physiologic Measures

- ✓ Ice
- ✓ Magnet and/or copper rubs
- ✓ Lidocaine gel or patch
- ✓ Rest
- ✓ Gentle stretching
- ✓ Short walks

Most common cause of a severe pain flare is NEUROINFLAMMATION!

Medical Options

1. Injection of Methylprednisolone (Medrol®) 20 to 50 mg with 15 to 30 mg ketorolac
2. Injection of Methylprednisolone (Medrol®) 20 to 50 mg with 2 to 4 mg of injectable hydromorphone (Dilaudid®)
3. Medrol® 6-day Dose Pack
4. Ketorolac 30-60 mg, on 2-3 days consecutive days (cannot take ketorolac for more than 5 consecutive days)

12. Biggest Mistake Made by AA Patients Who Deteriorate

- Our experience clearly shows that AA patients, families, and medical practitioners can be “self-defeating” in that they either don’t understand that 3 (not 1 or 2) categories of medicinal agents are necessary to stop progressive deterioration.
- Symptomatic pain relief is not curative. It is essential but AA patients equally focus on anti-neuroinflammation and neuroregeneration.
- Failure to walk and stretch each and every day. Walking and stretching are the fundamentals of physical therapy.
- Waiting to find a physician specialist in “AA”. They are just now developing.

- I can't take "steroids" or "anti-inflammatory" agents. Patients who claim they can't take steroids are almost always referring to cortisone or prednisone. In some AA patient's low dose, intermittent analogues of cortisone are all that will suppress NI. The term "steroid" is scientifically a chemical structure and our bodies make plenty of different "steroids" like estrogen, cortisol, and testosterone. The media has frightened patients off essential treatment in some cases.
- Doctors sometimes tell patients to stay away from anti-inflammatory drugs due to stomach bleeding if taken orally. Today, the critical anti-neuroinflammatory drugs can be taken as a sublingual (under-the-tongue), or troche (inside the mouth).
- Waiting for the "Big New Cure" like microsurgery, infusion, or stem cells. Basic treatment with the 3 medical categories should always be first and in place before trying the "latest miracle cure".
- All the medical agents recommended in this handbook do not have to be taken daily. To lower the risk of side-effects, take medications 2 to 5 times a week. Only take a medication daily if an intermittent schedule is not effective.



Therapeutic trials with LDN, Medrol®, and ketorolac should ideally be done with every new patient and any old one who isn't doing well.

If you have AA and are not taking LDN, ketorolac, or Medrol® I doubt that you will get better.



CHAPTER FOUR – SPINAL FLUID FACTS AND FLOW

1. Spinal Fluid Function and Necessity

The functions of spinal fluid are critical to the relief and recovery of an AA patient.

How is it made?

Spinal fluid is made by a small vegetative type tissue called “villi” in the brain. About 125 ml of fluid is produced every 4 to 6 hours for a daily total of about 500 ml.

How does it flow?

Once made, spinal fluid travels around the brain and down the spinal canal. Once it reaches the bottom (lumbar-sacral) area it flows back up the spinal canal to be filtered by lymph nodes in the neck. Filtered (impurities removed) spinal fluid is pumped into the blood stream to be excreted out of the body by urination. Breathing and moving helps the spinal fluid flow.

What does spinal fluid do?

1. Carries nutrients and anti-inflammatory chemicals to the spinal cord and cauda equina nerve roots;
2. Lubricates and protects the cauda equina;
3. Carries out toxins (e.g. viruses, metals and metabolic waste).

Good spinal fluid flow in AA is necessary to cleanse the nerve cells and act as an anti-neuroinflammatory agent.

2. Spinal Fluid Flow Obstruction

The clumping of nerve roots and the formation of adhesions causes disruption and obstruction of spinal fluid flow. This is like putting rocks or a dam in a stream – the water flow will slow down, back up, and reroute around the blockage.

When blockage occurs inside the spinal canal, certain symptoms may occur: headache, blurred vision, nausea, dizziness, mental confusion, loss of memory, ringing in ears, loss of balance, dysphoria.

One medication sometimes reduces the symptoms of spinal fluid flow obstruction, acetazolamide 50 to 500 mg a day. Start at 50 mg and work up the dosage as needed.

Listed below are special exercises and measures to keep spinal fluid moving to prevent symptoms and promote healing of the diseased and inflamed nerve roots.

SPECIAL NOTE: If an AA patient has several of the above-listed symptoms we recommend aggressive treatment with both anti-neuroinflammatory and neuroregenerative agents plus daily exercises to move the spinal fluid.

3. Spinal Fluid Leakage

We have learned from MRI reviews and physical examination of patients that the majority of AA patients chronically leak or “seep” spinal fluid into the tissues between the spine and skin over the lumbar spine area. The process is similar to a pipe that rusts and chronically leaks or “seeps” fluids out of the pipe. Unfortunately, the formation of inflammation between the nerve roots and arachnoid lining may cause microleaks through the spinal canal covering.



Spinal fluid is toxic to muscle, subcutaneous fat, and skin. This is why spinal fluid is in a closed canal away from non-CNS tissue.

4. Complications of Chronic Spinal Fluid Leakage: Over-time, these complications from spinal fluid may occur:

1. Contractures of paraspinal muscles to the point that you can't fully extend your arms or legs.
2. Pain over the spine – worse when pressure is applied.
3. Abnormal, off-balance leaning – asymmetry of back muscles.
4. Indentation (“caving in”) of tissues over the spine.

5. How to Improve Your Spinal Fluid Flow

All AA patients will have some disruption or blockage of spinal fluid flow. Good spinal fluid flow is necessary to wash away waste products and bring nutrition and positive autoimmune activity to the inflamed site.

Here are simple tips to enhance spinal fluid flow.

- Rock in a rocking chair
- Walk on a trampoline
- Use vibrator or massager over spine (Back scratchers and scrubbers are good)
- Soak or wade in water
- Walk and swing your arms (“Power Walking”)
- Rock back and forth on your feet
- Rub your spine with copper and/or a magnet
- Nod your head up and down
- Scrub your back with a brush
- Deep breathing (diaphragm) with stomach

KEY MESSAGE: Review the above list. Which things can and will you do daily to improve your spinal fluid flow?

Treatment of Spinal Fluid Leakage

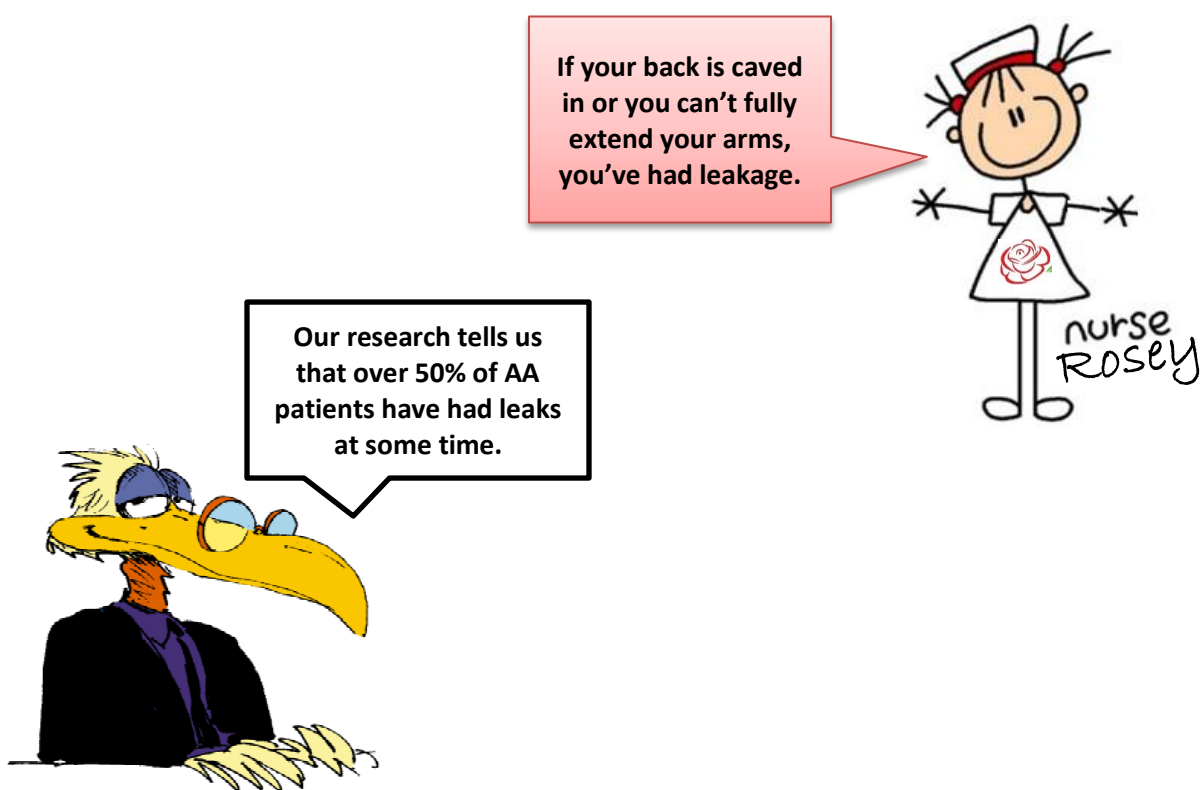
Treatment of spinal fluid leakage is an uncertain endeavor simply because active leakage may not be present. On an MRI leakage can usually be diagnosed, but it may have stopped by the time specific treatment is initiated.

Two of three of the following should be simultaneously administered as no one treatment measure may be effective.

- Increase dosages or add anti-inflammatory and neuroregenerative agents
- Massage into lumbar area topical creams which contain medroxyprogesterone (10 mg per ounce of cream) or estradiol (2 mg per ounce). Massage 2-3 times a day for 7 to 10 days.
- Local corticoid and/or homeopathic injection over lumbar area.

SPECIAL NOTES

1. We believe spinal fluid leaks stop rather rapidly with the anti-neuroinflammatory and neuroregeneration agents recommended in this handbook.
2. We do not recommend blood patches or epidural injections in a patient with known AA.



CHAPTER FIVE – PHYSIOLOGIC TREATMENT MEASURES

1. Walking and Stretching: The Most Basic Physiologic Measures

Walk Every Day with Correct Posture

An AA patient **must take walks every day** to move spinal fluid and prevent contractures (shrinking) of nerves and muscles. Walk with toes pointed straight ahead. Swing your arms during part of your walk. Lift your head so that your ears are directly over your shoulders. Breathe deeply. Quit at the first feeling of fatigue. Don't overdo or push too hard.

2. Proper Shoes

Persons with AA should wear supportive, tie shoes such as tennis shoes unless their feet are too painful. There are also some shoes that have copper or magnets in the soles. These are excellent to control pain.

Bare foot is often better for an AA patient than the modern-day practice of wearing thongs, sandals, flip flops, or slip-ons. Non-supportive footwear is a risk in 2 ways: (1) falls; (2) prevents correct walking posture.

One slip, slide, or fall can set an AA patient back to square one. A fall may tear adhesions which may cause severe pain which then re-heal with more permanent, nerve entrapment impairments!

3. Stretch Your Arms and Legs Each Day

Full-Body Stretch Laying Down: Lay down on the floor and do a full-body stretch. Count to 10.

Full-Body Stretch Standing: Spread hands and reach "to sky" until you feel pressure and tugging in your back. Count to 10.

Sit and Stretch Arms: Stretch your arms and spread your fingers. Count to 10. Can do while sitting in a car or plane.

Leg Raise While Laying Down: Raise leg until you feel tugging in your back. Count to 10.

Leg Raise While Standing: Stabilize yourself next to a table or wall. Raise your leg and flex your foot. Count to 10.

Knee Pull While Laying Down: Pull knee back until you feel tugging in your back. Count to 10.

Do one or more of these every day and stretch at least 3 times a day.

**Walk and stretch
each day. THEN
DO IT AGAIN!**



4. Contractures: An AA Patient's Worst Enemy

What is a contracture?

It is the scarring and shrinking or shortening of nerves, muscle, or tendons. The nerve roots of the cauda equina are the first part of long nerves (e.g. sciatic) that reach out to the legs, feet, bladder, intestine, stomach, rectum/anus, and sex organs.

NI leads to nerve root clumping, adhesion formation, and scarring. If NI isn't stopped, nerves can shorten or shrink causing great disability and impairments.

Muscles and tendons that attach to the vertebrae and joints can also contract particularly with chronic spinal fluid leakage.

Tragic Consequences of Contractures

If contractures of nerves and muscles may cause the following to occur: (1) leg paraparesis or paralysis – “wheelchair” or “walker”; (2) dysfunction and impairment of bladder, intestine, stomach, sex organs, and rectum/anus.

Stretching Principals

1. Stretch to a point you feel tugging or pulling but not pain.
2. Standing is best to stretch but sitting or lying down is OK.
3. You should do more than raise your arms. Stretch your arms and legs into positions that let you know you are tugging or pulling on a contracted area.

Best Stretch to Prevent Contractures.

1. Spread fingers.
2. Reach straight up with both arms until you feel tugging or pressure on your pain site. **DO NOT CAUSE PAIN!**
3. Hold for a count of 10.
4. Repeat at least 3 times a day.
5. Over time – try to extend your upward reach.



SPECIAL MESSAGE: Contracture prevention requires daily effort. No medication will prevent contracture – walk and stretch today!!

5. How to Eliminate Retained Electricity

What is retained electricity?

Nerves function and control our organs and structures by conducting electricity. With AA, nerve roots, the most critical part of a nerve, are clumped with adhesions and scarring, so electricity won't pass up or down the nerve root. Consequently, electricity is retained and accumulates.

Symptoms and Consequences of Retained Electricity

When electricity builds up it causes increased NI and will suddenly release itself in dysfunctional bursts. That is why AA patients get:

- ✓ Shooting and burning episodes of pain
- ✓ Leg jerks and tremors
- ✓ Burning feet
- ✓ Temperature rises with sweating
- ✓ Funny sensations on skin (“bugs crawling”, etc.)

When electricity accumulates you get more neuroinflammation and deterioration.



How to Help Eliminate Retained Electricity

Here are routine measures to eliminate electricity. Do some daily.

- ✓ Rub your spine with copper or a magnet 2 to 3 times a day
- ✓ Wear a copper anklet or bracelet
- ✓ Use magnets in your shoes or mattress
- ✓ Wear lots of jewelry
- ✓ Hold door knobs or other metal a second longer
- ✓ Soak in water (Epsom salts help)
- ✓ Pet your dog or cat (Any fur will do.)
- ✓ Walk barefoot on carpet or outside on your lawn

6. How and Why to Get More Oxygen

Why Oxygen?

Oxygen is necessary for healing, nerve function, and medication effectiveness. Without enough, you may progressively deteriorate.

Symptoms of Low Oxygen

- Fatigue and Lethargy
- Slow or Forgetful Thinking
- Depression and Feeling of Hopelessness
- Tired, But Can't Sleep
- Pain Medication Works Poorly

How Do I Get Oxygen?

Oxygen is breathed in through your lung and enters your red blood cells to be carried throughout your body. Regardless, if your pain site is spine, brain, joint, or muscle, you must have oxygen for pain relief and healing. The more oxygen, the better.

How Do I Get More Oxygen?

Your base oxygen intake and carrying capacity is what is in your blood when you are quietly sitting or lying down. Anytime you become active, your lungs breathe a little faster and deeper and your heart pumps a little faster, so you carry more oxygen in your blood. The healing and pain relief formula is simply to stay more active than what you are when you sit or lay down. Just increasing your breathing and heart rate will increase oxygen at your pain site.

First Steps to Acquire More Oxygen

1. Stay active! Walk every day.
2. Breathe as deeply as you can with your stomach (diaphragm) and hold it for 10 seconds. Do it while sitting or standing. Do it in a car, church, or home. Do this at least 10 times a day.

7. High Protein Anti-Inflammatory Diet for AA Patients

Protein

It provides the amino acid building blocks that are necessary for the production of neurotransmitters and tissue healing.

You Must Eat Some of The Following Each Day

Fish	Chicken	Turkey	Beef
Pork	Eggs	Cottage Cheese	

Spirulina and chlorella algae, black beans, and pumpkin seeds have a high protein content. If you can't or won't eat any of the above, you must obtain protein powder drinks and/or protein bars from the health food store.

Vegetables and Fruits: Some vegetables and fruits have anti-inflammatory activity. Eat some of these each day.

Carrot	Celery	Beets	Tomatoes
Broccoli	Brussel Sprouts	Spinach	Cucumbers
Radish	Onion	Lettuce	Watermelon
Blueberry	Blackberry	Raspberry	Strawberry
Apple			

Drinks: (Only use dietary sugars if weight is a problem): Coffee, Tea, Dietary Sodas, Water
Low dose, occasional alcoholic drinks are acceptable.

Banned to Control Weight: Milk, Regular Sodas, Fruit Juice, Bread, Rolls, Buns

Highly Restricted to Control Weight: (Eat these very sparingly) Potatoes including French Fries, Corn, Cakes/Pies, Pasta/Pizza

Gluten Restriction: bread, pasta, rolls, noodles, rice

8. Insomnia and Sleep

A regular sleep pattern enhances the hormone and immunologic systems that are necessary for neurogenesis. Here are some guidelines:

- ✓ Be in bed between 10:00 and 11:30 PM.
- ✓ Do your last stretches and medication dosage 30 to 60 minutes before bedtime.
- ✓ Keep your pain medications beside your bed. Take additional dosages during the night, if necessary.
- ✓ Take your first morning pain relief medications so you can be out of bed between 6:00 and 7:00 AM.
- ✓ Goal is 4 to 8 sleeping hours. Do not expect more than four hours of consecutive sleep.
- ✓ Most popular sleep aids are zolpidem (Ambien®) and temazepam (Restoril®).
- ✓ Take melatonin 5 to 20 mg with your sleep aid to assist sleep and help regulate your hormone and immune systems.



9. Insomnia with Centralized Pain

Intractable pain (IP) patients including those with AA who have centralized their pain will almost always have significant insomnia. Very few IP patients can get over 4 hours sleep at a stretch. Many only sleep for about 2 hours at a stretch. The cause of insomnia in IP patients is not just pain but the central nervous system is over-aroused and over-stimulated.

Follow these steps as IP patients **MUST** get some sleep each night.

STEP ONE—Use pain medication at bedtime. Take a dose of your pain medications just before bedtime. You may need to take another dose when you awaken in the night.

STEP TWO—If you can't get enough sleep with a bedtime dose of your usual medication, take one or more of these natural, non-prescription, over-the-counter preparations.

- L Tryptophan-1000 to 2000mg
- Valerian Root-1000 to 2000mg
- Benadryl® (diphenhydramine) – 25 to 50 mg
- Melatonin – 5 to 20 mg

10. Electromagnetic Measures

A new therapy that is gaining more and more supporters and advocates is electromagnetic energy (EME) therapy. We highly recommend it as we believe EME enhances the standard treatments of medicinal agents and physiologic measures.

What is electromagnetic therapy?

The human is alive and functioning because cells metabolize and communicate with each other by electromagnetic energy (EME). EME is biologically ½ magnetism and ½ electricity.

EME Devices

There are 3 medical devices that administer EME in various wave lengths and frequency to obtain a clinical effect: (1) radio wave, deep, long cord waves – slow frequency; (2) infrared, shallow – warm waves; and (3) laser, deep, hot – high frequency.

Difference with Other Devices

- ✓ TENS and electrical stimulators, provide pure electricity which gives a temporary, anesthetic effect
- ✓ Acupuncture – withdraws retained electricity

Benefits of EME

1. Reduction of inflammation, edema, and retained electricity;
2. Activation of cells which regenerate tissue including nerve roots, spinal canal lining, and soft tissues around the spine.

Spinal fluid leakage: We believe EME devices are very helpful to heal spinal fluid leakage.

Therapeutic Trials

At this point in time we recommend a one-month trial of EME to see if a positive clinical effect can be obtained. To date, about 2/3 of patients report a positive benefit in pain reduction and functional improvement.

11. Lifting and Bracing

The AA patient must be very cautious and careful while lifting and bending over. If you attempt to lift something that weighs more than about 10 pounds, you run the risk of tearing adhesions or scars in and around your lower spinal canal. When you bend over, raise up slowly because a jerk or rapid movement can cause a tear or rip. If this happens, severe pain follows, and the damaged area may end up being worse than ever.

The Importance of Spine Bracing

Shockingly, few AA patients have been told they need to periodically wear a brace to protect their damaged area.

Worst Situation: Riding in a car or plane that has bucket seats.

Danger Situation: Walking in unfamiliar areas such as a shopping center, grocery store, or social event.

**MOST IMPORTANT TIME
TO WEAR A BACK
BRACE:
PAIN FLARE**



12. Inversion Therapy

Inversion therapy (IT) is a form of super-stretching. It is usually done on a commercial stretcher. Rare persons with AA hang upside down.

The purpose of IT is to stretch out nerves, muscles, and tendons that are contracted. This is especially a problem with persons who have spinal fluid leakage.

It must be done gently and for only a short time (e.g. 3 to 5 minutes) to begin. Later, stretch time can be extended.

Only some AA patients seem to benefit from IT. Those persons who can't fully extend their arms or legs are the best candidates.

We only recommend IT for patients who are on a medication program with agents from the 3 key categories. IT is an ancillary measure and not a primary treatment.

CAUTION: Only stretch to the point of pressure. NEVER cause pain because pain means you may be tearing tissue that may scar and produce even more pain.



**Get on the 3-component
medical protocol and
start some physiologic
measures to get the best
relief and recovery.**

CHAPTER SIX – HOW YOU CAN HELP YOURSELF

1. Dealing with The Fright of AA

Every person who is informed they have AA is rightfully scared and frightened. To be otherwise would not be human. When one has pain, funny feelings on the skin, an irregular bladder, and blurred vision you will naturally be scared.

What's more, the stories of the past – paralysis, starvation, horrific pain, non-functioning mind and body, and early death were all true – at one time. But no more! Our medical protocols for AA aren't totally curative, but the plight of AA victims in the past doesn't have to occur today.

1. Don't let fright paralyze or cause you not to act. You must take immediate action once you find out you've got AA.
2. Tell someone about your fright – spouse, friend, child, doctor, clergyman – nothing calms fright like sharing it with another person.
3. Understand this new medical fact – our modern treatment will keep you from getting worse – just start the treatment! Bottom line – get started in treatment and you won't get worse.
4. Get started with treatment – take a walk – walking is a first-class medicine for AA. Second, go to the health food store and buy one thing known to help most AA patients – curcumin, Serrapeptase, pregnenolone.

2. No Doctor? Start Treatment Anyway!!

Adhesive arachnoiditis (AA) is a progressive, neuroinflammatory disease. Every day you delay puts you at greater risk for increased pain, suffering, paraparesis (partial paralysis), bladder dysfunction, and a bed-bound state. Too many persons with AA are waiting for a knowledgeable doctor to treat them. No need to wait. Our research tells us that persons with AA can truly help themselves get started with non-prescription treatment.

The best way to rid
yourself of fright is to
know your challenge
and that
**YOU HAVE THE POWER
TO CONTROL AA!**



<u>ANTI-NEUROINFLAMMATION</u>	<u>ANABOLIC/NEUROREGENERATION</u>	<u>PAIN RELIEVERS</u>
Curcumin/Turmeric Bovine Adrenal Extract Serrapeptase	Pregnenolone (100 mg or more a day) Dehydroepiandrosterone (DHEA) (200 mg or more each day) Colostrum Bovine Gonadal Extract (Orchex® of other)	CBD Oil Valerian Root Kratom GABA Taurine Icy-Hot Patches Palmitoylethanolamide (PEA)

Obtain and take at least one or more of the non-prescription agents listed in each group. These are available in health food stores or on the internet. Use the dosage on the label.

The most common complaint we hear is that an AA patient can't find a doctor who knows anything about AA. This is understandable. Only a couple of years ago the entire medical world thought arachnoiditis was a "spider bite". We've made good progress with more to come, but you can start your own treatment while waiting to find a knowledgeable medical practitioner.



CHAPTER SEVEN – HORMONES: THE WAY FORWARD

1. Anabolism and Catabolism – You Must Know the Difference

What Do These Terms Mean?

ANABOLISM and CATABOLISM are age-old medical terms that you don't hear much about anymore. Why? In regular medical practice they don't mean much – BUT they are CRITICAL with AA, EDS, Tarlov, and related disorders.

ANABOLISM is constructive or growth metabolism.

CATABOLISM is destructive or deterioration metabolism.

EDS, Marfan and some other genetic disorders patients have a “built-in” gene that causes catabolism. Simply put, metabolism in some tissues periodically or continually shift from anabolism into catabolism and causes tissues destruction. (e.g. microtears)

AA in advanced stages may create an autoimmune disorder that shifts some tissues from anabolism to catabolism. This is the reason some AA patients may develop symptoms of fibromyalgia, arthritis, or lupus.

Until recently, it was not well-appreciated that EDS is a catabolic disease. When catabolism gets in a given anatomic spot or organ such as the joints, uterus, or spine, tissue is destroyed. Microtears, ruptures, prolapses, and detachments may occur with production of severe pain, and disability.

2. Anabolic Measures

AA and EDS patients must constantly practice anabolic measures some examples are given here. Anabolic measures are basically of 3 types. A new anabolic therapy described in another chapter of this handbook is electromagnetic energy (EME) therapy.

<u>DIET</u>	<u>PHYSICAL MEASURES</u>	<u>HORMONES</u>
Amino Acids B-12	Weight Lifting	Testosterone DHEA

Neuroregeneration is the term that means anabolism for nerve tissue.

3. Hormone Replenishment: Essential Measure

AA and centralized pain will almost always require more, specific hormones than the body can produce. New research shows that the CNS produces specific hormones called “neurosteroids”. These include: (1) DHEA; (2) estradiol; (3) pregnenolone; (4) progesterone; and (5) testosterone.

We highly recommend that every AA patient have a blood profile test for the “neurosteroid” hormones at least 3 times a year. If you are low in one of the neurosteroids, you should take hormone supplements to replenish and bring your hormone blood level up to normal range.

Why are Neurosteroid Hormones Critical to AA Patients?

The basic functions of the neurosteroids are: (1) suppress neuroinflammation; (2) regenerate damaged cells; and (3) provide pain relief.

You have got
to have
hormones to
be HAPPY!



Ask your medical practitioners for a hormone blood profile test. Every community now has laboratories that do a hormone profile.

4. Anabolic Hormones: Best Hope for Cure or Near Cure

As explained in another section, anabolism means to generate or grow tissue and catabolism is the degeneration or deterioration of tissue.

Cold Hard Fact

Patients with AA who want to go for cure or near-cure will have to regrow or regenerate damaged nerve root and spinal canal lining tissue. We also believe that new nerve fibers may sprout and grow around scarred and damaged tissue which provides the same effect. Neurosteroids stimulate the growth of new nerves.

The Question: How does an AA patient promote anabolic growth of nerve roots and spinal canal covering?

Effective suppression of NI, a high protein diet, and some physiologic measures described in this handbook are critical. We also believe that one or more anabolic hormones must be taken to gain significant recovery.

Our Results to Date

Old ARC lore has it that AA is a hopeless disease that never gets better. In our experience, we see the great majority of AA patients get better. The ones who have usually done the best have taken one or more of the potent anabolic hormones listed below. Sometimes they have rotated the hormones.

While some would claim our results either didn't occur or the hormones didn't help, we will stand by our results and the statements made here. We have had many patients go from a bed or couch bound state to be functional. We have had AA patients with paraparesis (partial paralysis) or total paralysis start normal walking. Many AA patients have been able to stop daily opioids.

The Five Anabolic Hormones We Recommend

1. Dehydroepiandrosterone (DHEA) – The dosage must be 200 to 400 mg a day. DHEA is a neurosteroid that, at a 200 mg dosage, will metabolize to estradiol, progesterone, and testosterone.
2. Pregnenolone – The dosage must be 100 to 300 mg a day. At this dosage pregnenolone has direct spinal cord and nerve root anabolic effects. It also metabolizes to allopregnanolone which is a potent neurosteroid with anabolic effects.
3. Human Chorionic Gonadotropin (HCG) – This is one of the so-called “pregnancy” hormones. It goes up in the pregnant woman as it is THE HORMONE that grows the CNS in the embryo and fetus. When given to animals and humans it has a direct healing effect on spinal cord and nerve roots. In addition, it raises body levels of thyroid, progesterone, estradiol, and testosterone. Starting dosage is 250 to 500 units on 2 to 3 days a week. It can be taken as an injection, sublingual tablet, or buccal (cheek) troche.
4. Nandrolone – This is a synthetic testosterone derivative that is FDA labeled for wasting and deteriorating disease. AA patients certainly qualify. EDS patients with AA should most assuredly give this hormone a trial as it has generalized anabolic effects. It will grow or regrow tissue inside and outside of the CNS. Nandrolone can be taken as a weekly injection (25 to 50 mg) or it can be compounded as a troche to be taken (25 mg) on 2 to 3 days a week.
5. Medroxyprogesterone (MDP) – This is a derivative or analogue of progesterone and a member of a hormonal group called “progestins”. MDP has a more potent effect on CNS than plain progesterone. We use a trial of 10 mg a day for 5 days a week over 30 days. If pain and opioid dosage are reduced, we continue the therapy on 3 to 5 days a week.

Clinical Trial and Dosing

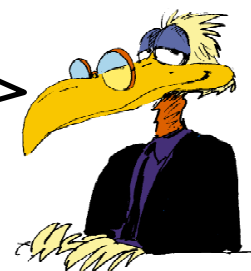
Patients can rotate the anabolic agents. Also, none have to be taken daily which obviously decreases any chance of side-effects. The minimal length of time necessary to experience positive effect is 2 to 4 weeks. Clinical trials should be at least 6 to 10 weeks.

DHEA and pregnenolone are both available without a prescription. Some patients now take both and report effects that are similar to HCG. Nandrolone is the back up hormone to be used in patients who have serious complications such as dysfunctional bladder, paralysis, intractable pain, Chiari incidents, and tethered cord. Patients with spinal fluid leaks should most definitely take anabolic hormones.

Prevention

“Knock on wood”. To date we have witnessed a lack of deterioration in AA patients who take one or more of the anabolic hormones along with anti-neuroinflammatory agents. In recent years we have not seen a new AA patient end up bed-bound, paralyzed, or develop contractures that limit arm and leg extension.

If you want a cure or near-cure you will have to take one or more anabolic hormones.



5. Biologic Hormone Extractions: Great New Development

One of the best things that has occurred for chronic pain patients, including those with AA, is the recent marketing of biologic hormone extraction products. These are not bioidentical, they are the “Real McCoy”. The makers of these products extract entire glands or fluids and don’t separate them into different, individual compounds.

History

Prior to the invention of prednisone and cortisone in about 1950, doctors would administer whole glands like the thyroid, pancreas, and adrenal. This practice was sometimes known as “glandular medicine”. The only survivor of this practice, until lately, is thyroid. Whole, dried (desiccated) thyroid is still marketed by the Armour Company. Patients who take whole “Armour” thyroid can tell its superior effectiveness over a singular, thyroid compound or chemical. Today, whole gland extractions of the adrenal gland and gonads (ovary/testicle) are marketed.

Why AA Patients Should Consider Biologic Hormone Extracts

Both patients and physicians are afraid of cortisone as steroids and the possible complications of cortisone, prednisone, estrogen, and testosterone. The biologic extracts are non-prescription and formulated so that complications either don’t exist or are minor and pass quickly. The US Food and Drug Administration regulates these non-prescription hormone extracts to insure safety. They can, therefore, be added to your current treatment program as a trial for a month.

Adrenal Extracts

Our studies show that essentially every AA patient, particularly if their pain is centralized, will be deficient in adrenal hormones including cortisol and pregnenolone. These hormones are essential for: (1) pain relief; (2) suppression of neuroinflammation; and (3) regeneration of damaged cells. Adrenal extracts are sold in health food stores or over the internet.

Gonadal Extracts

These extracts do about the same as the adrenal extracts, but they may have more neuroregenerative/anabolic effects than adrenal extracts. One trade name is Orchex®.

Colostrum

This is a milky hormonal substance secreted by primates (humans, animals with a spine and extremities) for a few hours after a female delivers an offspring. Colostrum supports and sustains newborns because it contains many hormones including human growth hormone.

Deer Antler Velvet Extract

Although new to the commercial market, it is hardly new. Deer Antler Velvet is a “velvet” type substance that grows in the lining of the antlers

**Some very good
anabolic
hormones don't
even require a
prescription.**



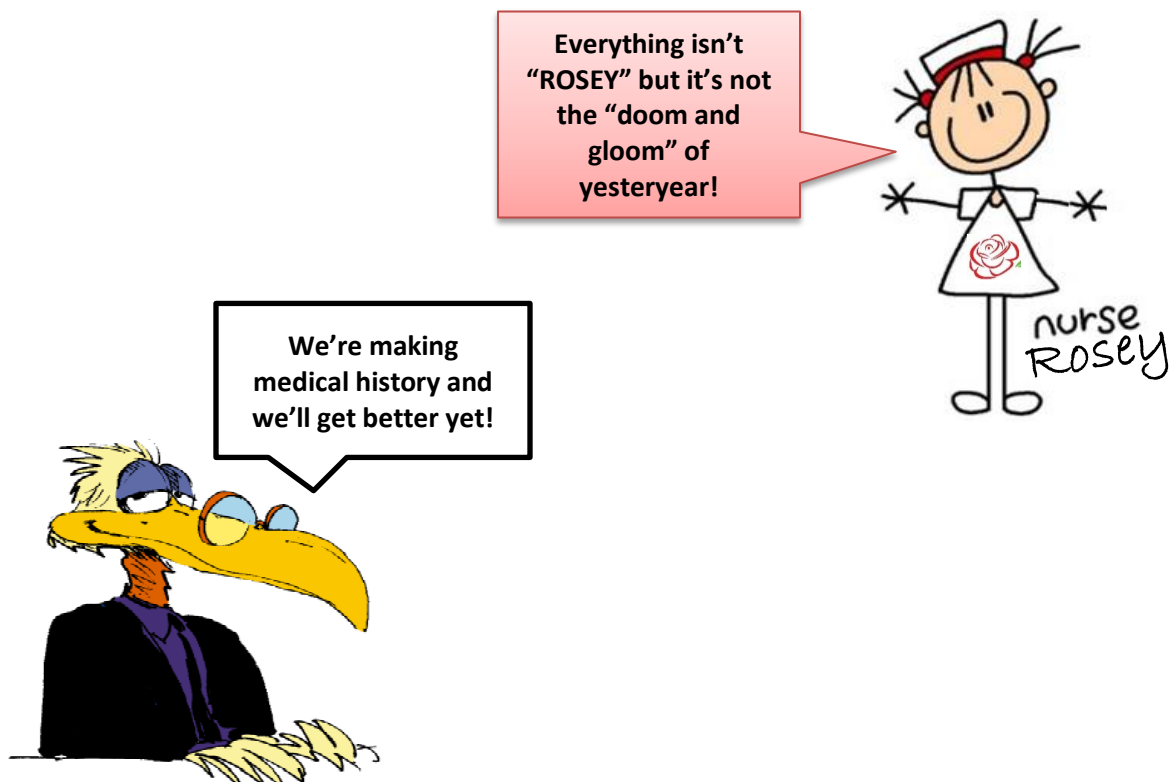
of a deer. It functions essentially as a pituitary gland to secrete hormones that nourish and grow antlers and other tissues. Historically, deer antler velvet is known as the medicine for "royalty". At this time, only a few AA patients have taken it, so we don't have reports to share. Deer antler velvet contains several hormones that are known to grow tissue. They include insulin growth factor and epidermal growth hormone. Theoretically deer antler velvet should help AA patients.

Dosage and Clinical Trials

Follow the directions on the label of biologic hormonal extracts. Try them for a month to see if you get a positive response before you take them on a long-term basis.

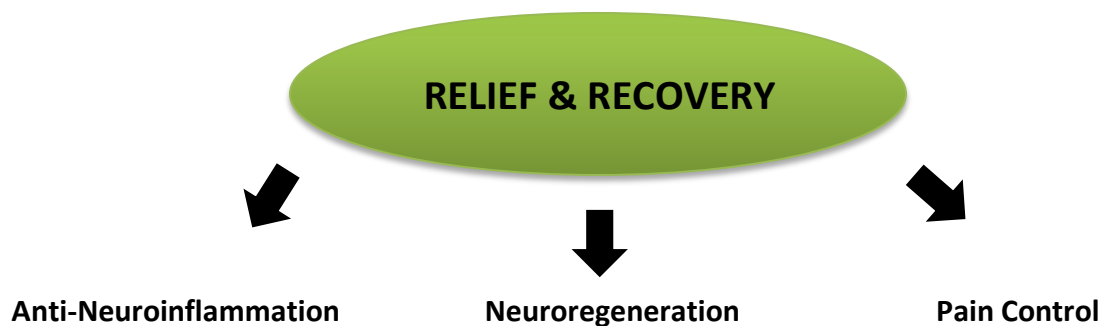
DISCLAIMER AND CAUTION

Physicians, including this author, who witness a new development that relieves suffering and brings a better life to patients are often accused of being over-zealous and/or biased. Ok! I'll admit to having a "missionary zeal" for anabolic hormones in the treatment of AA and EDS. Please excuse my enthusiasm, but I won't apologize. Based on my clinical experience, I firmly believe that hormone therapies are "THE WAY FORWARD".

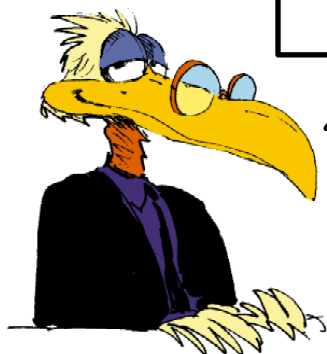


APPENDIX ONE

THE MEDICINAL THREE LEGS OF AA TREATMENT



Our research clearly indicates that AA patients usually don't improve much unless agents in all 3 categories are taken. Agents from all 3 categories do not have to be taken each day, but sometime during each week all must be taken.



Don't let anyone tell you that you don't need medicines from all 3 groups.



APPENDIX TWO

MEDICINAL AGENTS IN THE 3 ESSENTIAL CATEGORIES

I. ANTI-NEUROINFLAMMATION

Low dose naltrexone**

Ketorolac (Toradol®)

Methylprednisolone (Medrol®)

Metformin

Acetazolamide

Pentoxifylline

Minocycline

Non-Prescription

Curcumin/Turmeric

Serrapeptase

Bovine adrenal extract (Raw Adrenal® or other)

II. NEUROREGENERATION/ANABOLIC

DHEA (over 200 mg a day)

Pregnenolone (over 100 mg a day)

Human chorionic gonadotropin

Nandrolone

Non-Prescription

Colostrum

Bovine gonadal extract (Orchex® or other)

III. PAIN RELIEF

Non-Opioid

Oxytocin

Ketamine

Clonidine

Neuropathic*

Gabapentin

Topiramate

Pregabalin (Lyrica®)

Duloxetine (Cymbalta®)

Tizanidine (Zanaflex®)

Carisoprodol (Soma®)

Stimulant*

Amphetamine Salts (Adderall®)

Methylphenidate (Ritalin®)

Phentermine (Adipex® or other)

Non-Prescription

Kratom

Palmitoylethanolamide (PEA-Comfort Max® or other)

Cannabinoid derivatives

Topical Skin

Lidocaine gel or patch

Opioids (Last resort)

Tramadol

Hydrocodone

Codeine

Morphine

Oxycodone

Buprenorphine (Suboxone® or other)

Hydromorphone (Dilaudid®) injectable when oral opioids fail to relieve pain

*Agents from both these groups are usually necessary for centralized, intractable pain which is assumed to be present if a patient has constant pain, insomnia, and episodes of heat or sweating.

** Low dose naltrexone is also a pain reliever.

APPENDIX THREE

HORMONE BLOOD PANEL

Cortisol
DHEA
Estradiol
Pregnenolone
Progesterone
Testosterone

Any of the hormones listed above should be replenished if they show a low blood level.



All your hormones need to be in normal blood level range for relief and recovery. Test and replenish NOW!

All 6 of these hormones have neuroregenerative/anabolic properties. All AA patients need regular hormone blood tests.



APPENDIX FOUR**EMERGENCY TREATMENT**

Medrol® 6-Day Dose Pack

Plus

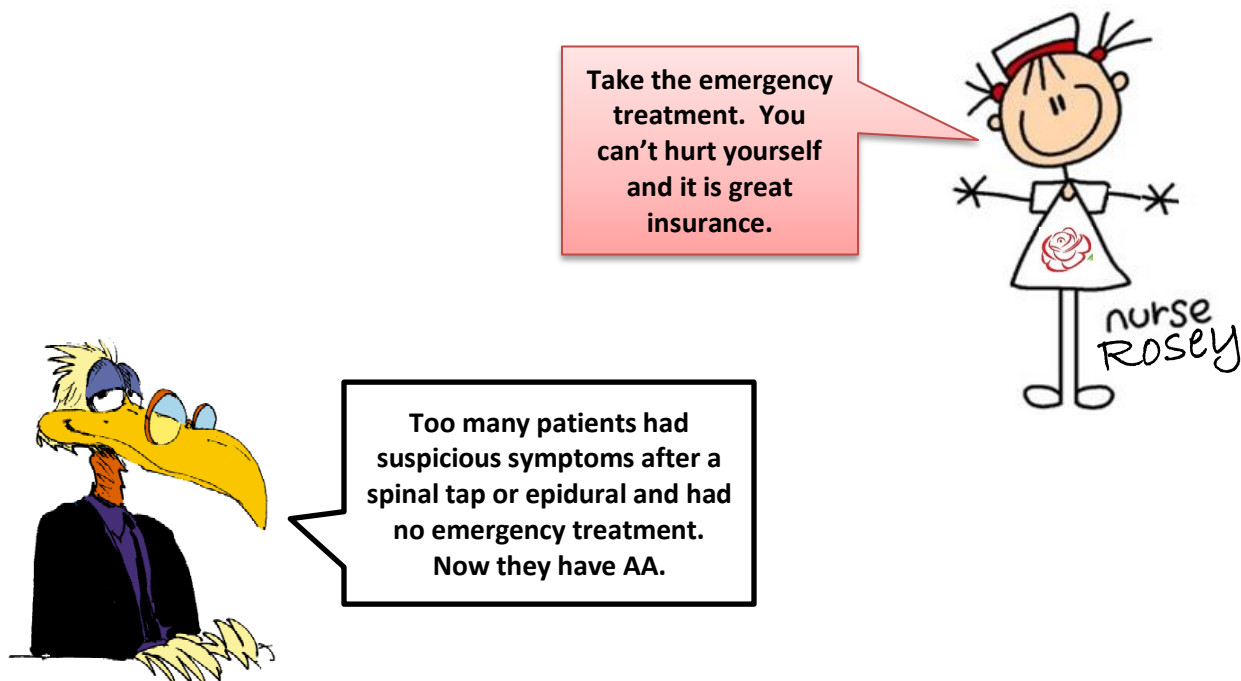
Ketorolac (Toradol®) 30 – 60 mg for 3 consecutive days

Options: Add medroxyprogesterone, 10 mg 2 times a day for 5 days.

Or

Minocycline, 100 mg 2 times a day for 5 days.

This treatment should be used within 60 days after a spinal tap or epidural injection if the procedure is suspected to have induced arachnoiditis.



APPENDIX FIVE

THERAPEUTIC TRIALS FOR DIAGNOSIS OF NEUROINFLAMMATION

1. Medrol® (methylprednisolone) 6-day Dose Pack

and/or
2. Ketorolac (Toradol®) 30 – 60 mg for 2 consecutive days

We recommend therapeutic trials under these 2 circumstances:

1. Undiagnosed patient who may or may not have adhesive arachnoiditis;
2. Patient known to have adhesive arachnoiditis but is having complications and/or inadequate pain relief. “Getting worse” or deteriorating.

Interpretation: If the person being tested experiences less pain, fatigue, insomnia, and immobility, it is because the therapeutic medication has reduced neuroinflammation. In this case the patient should remain on intermittent dosages of the medication that controls neuroinflammation.



You can't hurt anything with a therapeutic trial. Ask your doctor for one.

If you are getting worse, you need a therapeutic trial.



MATERIALS AVAILABLE ON REQUEST**BIBLIOGRAPHIES AVAILABLE ON REQUEST:**

Bibliography of Scientific Articles on Which are Adhesive Arachnoiditis Protocol is Based

Bibliography of Scientific Articles on Which the 2019 Update "Hormones and Pain Care: What Every Chronic Pain Patient Should Know - The Way Forward" is based

Bibliography of Scientific Articles on Neurosteroids and Pain

Bibliography of Scientific Articles on Which "Anabolic Measures for EDS/Hypermobility Syndromes" is Based

CLINICAL PROTOCOLS FOR MEDICAL PRACTITIONERS ON REQUEST:

Diagnostic Evaluation for Adhesive Arachnoiditis (AA)

Medical Treatment Protocol for Adhesive Arachnoiditis (AA)

REPORTS ON REQUEST:

Update 2019: "Hormones and Pain Care: What Every Chronic Pain Patient Should Know - The Way Forward"

"Anabolic Measures for EDS/Hypermobility Syndromes"

Arachnoiditis Research & Education Project**of the Tennant Foundation****334 S. Glendora Ave.****West Covina, CA 91790****Fax: 626-919-7497****E-mail: veractinc@msn.com**

Give us the following information:

Name

Address including City, State, Zip

E-mail

Fax Your Request