

RSD PUZZLE #63
METHADONE TREATMENT

Question:

My doctor started me on Methadone treatment. Is there any problem with Methadone treatment?

Answer:

Methadone treatment should not be applied to RSD patients. There are three different kinds of pain.

1. Acute pain such as a recent heart attack or car accident of a few weeks duration or a fracture of bone.

The treatment of choice for the acute pain is treatment with narcotics as well as correcting the damaged area by surgery or other methods which has originated the pain.

2. Cancer pain. In cancer pain the condition is called a "dynamic pain" which means there is a dynamic pathology ongoing damage in practically almost a continuous basis both acute and chronic due to the infiltration of cancer cells and or due to multiple operations, or radiotherapy. In treatment of cancer pain, anything goes. Methadone is no problem and should be used. Other strong narcotic such as Dilaudid, MS Contin or whatever treatment that relieves the patient's pain should be given. The patient has a short life expectancy and if sympathectomy relieves the pain, so be it. Even though sympathectomy is not indicated in RSD patients, it can be done in cancer patients who have diabetes or severe occlusive disease of the blood vessels in the extremity. In such diabetic or severe occlusive disease patients or cancer patients, the life expectancy is usually less than 5 years and sympathectomy can provide a few years of relief. On the other hand, truly chronic pain patients who are going to live several years or decades the sympathectomy is fraught with very high percentage of failure anywhere from a few weeks to 3 to 4 years after the sympathectomy is done.

3. The third type of pain is the chronic pain and complex chronic pain. In chronic pain, the original pathology has seized and has left scar and damage to the nerves. In some cases, the chronic pain has left the patient with no nerve damage but it is perpetuated because of the use of addicting (habituating and drug dependent) narcotics. On the other hand, in complex chronic pain either the

patient suffers from neuropathic pain (a pain that is due to neurovascular damage such as diabetic neuropathy) or sympathetically maintained pain (SMP) or further scar formation and involvement of the adjacent nerves due to scarring such as in the case of arachnoiditis which is the scar formation in the meninges of the spinal canal.

The treatment for chronic and complex pain is quite different from acute pain.

In the complex chronic pain, the patient should definitely be treated with strong analgesics which are not addicting. The best non-addicting analgesics are Trazodone and Prozac followed by some of the tricyclic antidepressants such as Desipramine. These medications have a Naloxone reversible analgesic effect meaning that if they are taken along with Naloxone then they cannot control pain, otherwise they can. In this regard they mimic the strongest narcotics. They are not addicting. They raise the threshold of pain and they provide good analgesia along with more normal sleep and along with by-product or side effect of being an antidepressant even though the patient usually in over 1/4 of the cases is not even depressed.

In addition, such patients can be treated with Morphine antagonists such as Stadol and Ultram which by nature of being Morphine antagonist they do not suppress the cerebral endorphins and other hormones in the brain. The addicting narcotics are not indicated in these patients because they cause perpetuation of pain due to the withdrawal (rebound) and tolerance (more demand by the brain for more of the medicine). What can change the simplest acute or subacute pain to a permanently chronic pain by the generous use of addicting narcotics?

Historically, Methadone has been used as an alternative for Heroin among the Heroin addicts. It doesn't mean it cures the addiction, it just replaces the Heroin. It is preferable because Methadone has got a several times longer half life lasting in the system from 3 to 6 or 7 days. So the patient does not develop a sharp withdrawal (rebound) as the patient experiences with Heroin.

Then the patient is provided with increasing dosages of Methadone at first once every day or every 2 or 3 days, then 2 or 3 times a day. At first it usually 10 mg three times a day and then gradually creeps up on the patient. The overlapping of the dosage of narcotics prevents withdrawal symptoms of pain, headaches, etc.

All this is achieved at the expense of Methadone causing inactivity and forcing the patient to regress into the use of a wheelchair or other assistive devices. In addition, as the patient regresses into the use of a wheelchair, the pain becomes worse because of the principle recently reported by Doctor Koltzenburg. Doctor Koltzenburg noted that an inactive extremity undergoes the development of the hyperexcitability of deep pain sensors in the muscles and bones. He calls this type of phenomenon a "sleeping nociceptor". The reason for the name is because there is such deep pain centers in the muscles and bones are usually silent and only becomes symptomatic as the extremity becomes inactive (such as the application of cast, braces or wheelchair).

The chain of events is as followed:

Methadone with increasing doses results in the patient not being motivated to be active and to get up and around. Secondarily the inactivity of the extremities wakes up the "sleeping nociceptors" and causes aggravation of pain. As the result, the patient needs to have more and more Methadone. Eventually the dosage gets to the level 10 to 20 mg 3 times a day up to even 50 mg 3 times a day. In such doses in the long term basis, the Methadone causes intoxication of the brain such as seen among Opiate addicts. This is in the form of prolonged bed rest, prolonged inactivity, drowsiness, and most importantly intoxication of the limbic system (the emotional centers of the brain).

This last complication of intoxication of limbic system results in the patients becoming chronically depressed, developing poor judgment, becoming argumentative and short fused.

Worst of all the problem of poor judgment to go to the toxic long term side effect of Methadone prompts the patient to beg his or her doctor to resort to any for of treatment. We are seeing an increased number of patients undergoing carpal tunnel surgery, thoracic outlet surgery, tarsal tunnel surgery, sympathectomy, spinal stimulator surgery, and other harmful operations among the Methadone users.

A similar problem also develops among other chronic habituating strong Morphine agonist pain medications these consist of patients who use MS Contin and similar slow release of strong narcotics. Such medications are duragesic and other skin patch treatments with strong pain medications a very similar effect as Methadone.

The use of Methadone, MS Contin and duragesic could be limited to cancer pain patient.

Unfortunately, in the past three years, there has been a major confusion mixing cancer pain treatment form of treatment and applying it to complex chronic pain patients.

Nobody can argue that cancer pain patients are not being treated with enough medication for pain. They are also not being treated with enough medication for nausea or for depression.

This obvious and pathetic fact does not justify crippling and practically maiming complex chronic pain patients who don't suffer from cancer.

The end results have been that the pain clinics and pain specialists are using MS Contin, duragesic and Methadone, generously on RSD patients with disastrous results. The same patients also undergo unnecessary operations which are quite dangerous such as lytic lesions, chemical sympathectomy, neurectomy, or cryosurgery. Cryosurgery refers to damaging the nerves by applying extreme cold in a focalized fashion. It's obvious that cold in the form of ice, cryosurgery or capsaicin destroys the small c fibers (small sensory sympathetic fibers), and cause more dysfunction of the sympathetic system. It is obvious that such procedures are harmful in RSD patients.

However, it's going to take a few years or decades to undo the damages that have been done with the use of such dangerous drugs to RSD patients. It's going to take a few decades for the doctors to rediscover, as has been discovered in the past three decades, that the use of strong addicting narcotic is going to cause more problems for chronic pain patients rather than helping them.

H. Hooshmand, M.D.

RSD PUZZLE # 64

CAN I STILL REMAIN ACTIVE WITH MY RSD?

Dear Mary Ann:

Due to the fact that you have RSD, it would be extremely harmful for you to stay inactive, to use braces, cane or crutches.

Realizing that you are in constant pain, your pain will be ten times worse if you are in active and if you give in to your pain. It would be suicidal for you to stay home, not to go out and not to be active.

If nothing else, even if it is a hot summer day, you should go to a mall and try to walk and sit down as frequently as possible.

Recent research by Dr. Koltzenberg has shown that being inactive stimulates the pain fibers in the deep structures of bones and muscles and as a result causes sever pain which in turn causes the patient to be inactive. This vicious circle is quite damaging to the patients. If you rest you'll have pain, if you walk you will have pain but if you rest all the time you will have a lot more pain.

H. Hooshmand, M.D.

RSD PUZZLE #65
THE SPREAD OF RSD
(ALSO SEE RSD PUZZLE #18)

You have inquired about the possibility of the spread of RSD.

I am enclosing more than a dozen references that show RSD spreads. It is quite common for RSD to involve other extremities. Because of the fact that the chain of sympathetic ganglia are connected with each other vertically and horizontally (on the same side vertically up and down and the opposite side horizontally in the anterior aspect of the vertebrae) the RSD has a strong tendency to spread from one extremity to the other extremity.

The research done by Dr. Basbaum in the University of California, San Francisco, also shows that the involvement of the spinal cord with RSD in the form of damage to and stimulation of a wide dynamic range type of nerve cells (WDR) causes spread of RSD from one side to the other side through the gray matter of the spinal cord.

I have also explained the same phenomenon in the book I have written on the subject of RSD published in 1993 by (CRC Press, Boca Raton, FL) in detail.

H. Hooshmand, M.D.

RSD PUZZLE #66

The Relationship Of RSD With Heart Disease

You have inquired regarding the relationship of RSD with heart disease. Apparently you have had a pre-existing heart disease that is becoming aggravated by RSD.

RSD causes three independent negative influences on cardiac function.

1. The sympathetic system is responsible for three main functions, i.e., temperature regulation, vital signs, and regulation of the immune system. The vital signs in the form of heart beat, blood pressure and respiration are up regulated and accelerated by stimulation of the sympathetic system. The RSD is not a simple hyperactivity only stimulation of the sympathetic system. It is the result of dysfunction of the sympathetic system. This dysfunction shows instability of the sympathetic system at times causing fluctuation of blood pressure and at other times causing attacks of fast heart beat.

2. The second reason RSD affects cardiac function is due to the anatomical innervation of the heart muscles. Of all the visceral organs, the heart has the richest innervation of the sympathetic system. This is in the form of cardiac plexus which is a rich plexus of nerves surrounding the heart. In any stressful condition, the natural response is rapid heart beat and rise of the blood pressure. The RSD being a distressful type of dysfunction of the sympathetic system, results in repetitive pathological and exaggerated response of the sympathetic system to stress, chest pain, palpation, and bouts of high blood pressure.

3. One of the main principles of development of RSD is inflammation. RSD is a condition with four major features. First, the allodynia and hyperpathia is typical with pains seen with sympathetic dysfunction.

Second, is motor response to such pain in the form of vasoconstriction, muscle spasm and muscle tremor.

Thirdly, inflammation in the form of skin rash, swelling of soft tissues in the extremities, increased circulation in the visceral structures resulting in osteoporosis, pelvic inflammation, and attacks of vascular headaches. The same inflammation and increased visceral circulation causes distress on the heart. Obviously if the patient has already had pre-existing cardiac disease, the distressful disease of RSD is going to cause further stress on the heart on the basis of the above mentioned three principles.

H. Hooshmand, M.D.

cc: Eric Phillips

RSD PUZZLE #67

WHAT IS WRONG WITH OPERATING FOR CARPAL TUNNEL SYNDROME, TARSAL TUNNEL SYNDROME OR THORACIC OUTLET SYNDROME IN RSD?

RSD is considered a regional disease. The new terminology for it is Complex Regional Pain Syndrome or CRPS. The reason it is called complex regional pain syndrome is because it is not limited to one nerve or one finger or one toe.

The difference between the somatic system and the sympathetic system is that the sympathetic nerves follow the path of the arteries and arterioles. On the other hand, the somatic nerves and nerve roots follow specific path of the trunk of the nerves such as for example the 7th cervical (C7) nerve root, the 5th lumbar nerve root (L5), or the 1st sacral (S1) nerve root. If the patient has a circumscribed lumbosacral disc herniation, then the S1 nerve root selectively is impinged and causes a specific nerve root distribution type of pain and sensory loss which would be mainly in the posterior aspect of the leg all the way down to the 4th and 5th toes of the foot. On the other hand, when the sympathetic nerve is injured then it does not follow a specific nerve root but it follows the distribution of the blood vessel. The sensory loss and the pain are in the distribution of for example brachial artery showing a more brachial plexus type of distribution of sensory loss and pain, or the femoral artery showing more of an arterial distribution of the femoral artery and its branches. This anatomical fact causes a major confusion in regards to the diagnosis and treatment of the sympathetic nerve dysfunction. The following is a true story of a patient who had minor injury that resulted in 5 operations before the proper diagnosis was made and further surgery was prevented.

Whereas in exceptional cases such as a torn ligament in the knee-an RSD patient may need surgery under the protection of simultaneous nerve block, in other nerve involvements at the wrist or ankle, surgery leads to devastating aggravation of RSD. The following is an example.

"T.B" was a 50 year old air condition repairman who was injured while carrying an air conditioning compressor. Accidentally, the compressor fell injuring the medial aspect of his right ankle causing burning pain and swelling over the medial aspect of the right ankle and foot. This was followed by sharp pain spreading up to the right groin. On the basis of the severe pain over the right groin region, an exploration was done in search of inguinal hernia. None was found and after the surgical procedure the patient's pain became more intense.

He started having pain and spasm involving the distribution of the femoral artery. A month later the left inguinal region over the left groin was explored in the search of hernia or impingement of any nerve. Again, no abnormality was found and the area was sutured. By this time the patient had the spread of the disease to both lower extremities in the distribution of both femoral arteries (see RSD Puzzle #18: spread of RSD).

Two months later the patient started having burning pain and swelling, inflammation, and tenderness over the palmar aspect of the right wrist. A nerve conduction time and EMG were done. The EMG was extremely painful because of the fact that the patient's hand was swollen and extremely tender with burning pain. The EMG was normal. The NCV showed no delay in nerve conduction times- regardless, the patient was diagnosed clinically as suffering from carpal tunnel syndrome. Carpal tunnel surgery was done over the right hand. The operation resulted in the development of tremor and weakness of the muscles of the right hand as well as difficulty with extending his fingers. He started developing flexion deformity of the fingers of the right hand.

He also developed radiation of the pain and inflammation all the way up to the right shoulder. He could not move his right shoulder. He had severe pain over the right shoulder. Five weeks later, because of the fact that the patient had a frozen shoulder and flexion of the right elbow and right wrist and because of the fact that he had swelling, tenderness and hypersensitivity to touch over the right shoulder, an MRI of the right shoulder was done. MRI was diagnosed as mild "rotator cuff tear". While awaiting surgery for this condition, the patient started having similar symptoms over the left hand and wrist with similar pain, tenderness and swelling of the left hand and wrist. Within one week the patient underwent carpal tunnel surgery for the left hand. After the surgery, the patient started having pain radiating to the left shoulder as well.

Because the patient had pain, tenderness and black and blue spots over the anterior aspect of the right elbow, he underwent a "supinator release surgery" over the anterior aspect of the right elbow. Needless to say, the right elbow surgery did not help the patient at all.

Since then the patient has had bouts of waking up in the morning having black and blue spots and swelling over the entire right upper extremity or entire right lower extremity for no obvious reasons. In addition, he has had tendency for neurodermatitis and swelling over the left hand of intermittent and spontaneous nature.

After the 5 operations, the patient was scheduled to have surgery for the right rotator cuff tear but because the patient was getting quite frustrated with side effects of operations he demanded another opinion. The last doctor who saw him proceeded with thermography which confirmed the diagnosis of the temperature changes involving the upper and lower extremities which were not in the distribution of any specific nerve roots but more in the distribution of blood vessels in the femoral and brachial arteries. The right upper extremity below the elbow where the patient had already had two operations, showed hyperthermia with 2 centigrade temperature higher than proximal portion of the right upper extremity. The left upper extremity and the lower extremities showed 1 to 3 centigrade temperature reduction in the distal portions of the extremities compared to the proximal portions.

The patient was diagnosed as suffering from RSD. He was given 3 epidural and 3 stellate ganglion nerve blocks on the right side which were quite painful and did not provide pain relief. By then he was advised that he needed to have rotator cuff surgical repair or sympathectomy.

Obviously the surgical procedures were canceled and the patient was treated conservatively for RSD.

The lessons to be learned from this case are the facts that the patient had injury to the right ankle yet he has had multiple operations in the areas away from the site of the injury.

The patient never had direct injury to his shoulder yet he showed rotator cuff tear. The reason for the rotator cuff tear is the fact that inflammation of RSD causes swelling and extravasation in the soft tissues of the extremity. Extravasation refers to the fact that RSD causes not only inflammation but also defective membrane function in the small blood vessels causing leakage of the blood and plasma outside the blood vessel. Eventually this swelling and inflammation becomes severe enough to cause separation of the rotator cuff tendon fibers and causes typical picture of rotator cuff tear. The same phenomenon causes the typical pictures of carpal tunnel syndrome, tarsal tunnel syndrome, tardy ulnar palsy or thoracic outlet syndrome.

Surgery in such areas of inflammation is only going to cause more traumas, more damage and more dysfunction of the sympathetic nervous system and it is going to spread the disease further into adjacent or remote regions of the extremities.

The diagram in RSD puzzle #18 shows the vertical and horizontal connections of the sympathetic ganglia around the vertebrae and explains the reason for vertical or across the midline spread of RSD. In the same RSD puzzle #18, there are 15 references that explain clearly the nature of the spread of RSD to other extremities.

The clue to RSD nerve entrapment rather than standard carpal tunnel or tarsal tunnel syndrome is the fact that the area of nerve entrapment has not undergone any kind of direct trauma. As is the case with Mr. T.B. whose history is outlined above, the original area of nerve damage is remote, and only as a regional spread such pictures of entrapment of nerves due to inflammation develops.

In RSD there are four main features. First of all the hyperpathic and allodynic pain which are typical characteristic pains of RSD manifested by elicitation of pain with simple touch or breeze (allodynia) and an out of proportion pain which spreads to the adjacent region (hyperpathia). The second feature of RSD is muscle spasm, muscle weakness, spasm in the wall of blood vessels (causing cold extremity) and tremor. The third manifestation of RSD is disturbance of immune system and secondary inflammation which becomes markedly aggravated by either inactivity or repeated operations. The fourth manifestation is disturbance

of limbic system causing emotional disturbance, poor memory, poor concentration, irritability, agitation and depression. The case of Mr. T.B. manifests all of these criteria.

The answer to such horrible spread of RSD due to unnecessary operations is to avoid surgery when there has been no direct trauma at the area that is being operated on; to treat the patient with medications that reduce the edema and swelling and inflammation (such as ACTH or IV Mannitol treatment). Treatment with physical therapy and exercise will help reduce the swelling and inflammation. The use of Epsom salt and warm water also helps correct this condition.

More importantly, to enable the patient to recover and heal his own body, it is necessary to provide proper and effective pain relief with the help of treatment with Naloxon, reversible antidepressants such as Trazodone, and treatment with non-addicting strong pain medications such as Tramadol (Ultram) and Stadol.

Heat, massage, ultrasound and exercise are essential. Avoidance of especially ice (which causes further damage to the sensory nerve fibers and spreads the disease), and avoidance of the use of brace and especially casts, is essential. Dr. Cardoso and Dr. Jankovic have shown that 10 out of 11 patients who developed Parkinsonian type of tremor after application of the cast, suffered from RSD [1]. If the sympathetic nerve blocks are not effective because of the long duration of the disease and because of the fact that treatments with multiple operations has already exhausted the sympathetic system (as noted above in the case of Mr. T.B., whose right upper extremity was warm rather than cold), then instead of doing sympathetic ganglion nerve blocks the patient should receive epidural and paravertebral nerve blocks. The most important preventive measures are encouragement of activity, prevention of pain, avoidance of the use of ice and avoidance of unnecessary surgical procedures and immobilization (e.g. with cast or brace).

H. Hooshmand, M.D.

Reference:

1. Cardoso F, Jankovic J: Peripherally induced tremor and parkinsonism: Arch Neurol 1995; 52:263-70.

RSD PUZZLE #68

Methadone is no different than other types of Morphine agonists in regard to tendency for physical dependence.

Dear Dr. Hooshmand,

I have been treated with Methadone for chronic pain. Because of the problems of fatigue, depression, and the fact that there are reports of deaths due to Methadone, I would like to get off this medicine. Can I just stop it cold turkey or should I replace it with other medications?

Thank you.

Ms. MT

Dear Ms. MT,

I appreciate your concern. It is easy to start on Methadone but it is not as easy to discontinue it. You should discuss with your pain specialist in detail regarding how you want to be detoxified and to come off medication. Methadone is no different than other types of Morphine agonists in regard to tendency for physical dependence. The only difference is that Methadone has a long half-life, and can last in the system for a few days. In that regard, the rebound phenomenon (withdrawal) is not noticeable when multiple doses of the medicine are prescribed such as in the dose of 2 or 3 times a day. By the time the previous day's is practically all out of the system, the second dose replaces it. In this regard, it is very similar to medications such as MS Contin or other long duration skin patches of the opiate agonist medications. Clinically, the fact that withdrawal is accelerated (rebound phenomenon) is camouflaged by overlapping dosages of the medications; the adverse affect on the brain is accelerated. This adverse effect consists of practically complete arrest of formation of cerebral endorphins and secondary side effects of reduction of Estrogen and other types of hormones related to the hypothalamus of the brain. As a result the patient becomes fatigued, has tendency to gain weight, has tendency to be inactive, and especially during the night while sleeping the extremities do not have the normal tossing and turning so the inactivity can aggravate the RSD and can aggravate the edema and inflammation of the RSD. Also such patients show a significant suppression of the brain endobenzodiazepines (endoBZs) and natural cerebral antidepressants.

There are a few safe ways to discontinue such long lasting opiates.

1. Recently the Harvard researchers have discovered a medication, Buprenorphine (Buprenex) [1]. Buprenex has been found by the Harvard researchers to be promising for the treatment of "polydrug" abuse. This analgesic medication has been tried on patients dependent on both opiates and Cocaine. Buprenex is mu opiate receptor agonist and antagonist. In addition, it has been found to have some advantages over Methadone in terms of relative safety in the treatment of Heroin addiction. Surprisingly, it is also effective in reducing the side effects of Cocaine withdrawal as well.

2. The second form of detoxification from Methadone and other opiate agonists' dependence is switching the patient to Stadol and Ultram in a cold turkey fashion as long as the patient also takes Klonopin to reduce any chances of potential for seizure disorder from Ultram.

H. Hooshmand, M.D.

Reference:

1. Ling E, Wesson DR, Charuvastra C, et al: A controlled trial comparing buprenorphine and methadone, maintenance in opioid dependence. Arch Gen Psychiatry 1996; 53:401-7.

RSD PUZZLE #69

MORE ON PHYSICAL THERAPY AND RSD

Depending on the specialist who is treating the patient, the treatment may be limited to only pain medications by one specialist, only antidepressant by another specialist, or only nerve blocks by a third specialist.

The treatment should be attacking the disease in a multidisciplinary fashion from all angles addressing all four pathogenic factors resulting in RSD.

1. For the allodynic pain, the patient should receive SSRI type of antidepressants or Trazodone. The tricyclics should be avoided because they have a tendency to aggravate obesity, exacerbate fatigue, and result in a drop in the blood pressure and, in rare cases cardiac irregularities. These complications may preclude the proper use of alpha blockers.

2. FOR THE PROBLEM OF COLD EXTREMITY, VASOCONSTRICTION, AND MOVEMENT DISORDER, ICE SHOULD NEVER BE APPLIED. THE PATIENT SHOULD BE TREATED WITH HOT WATER AND EPSOM SALT BATH. THE EPSOM SALT IS A HYPEROSMOLAR SALT, AND RELIEVES THE INFLAMMATION-AS WELL AS ACTING AS A CALCIUM CHANNEL BLOCKER

ICE SHOULD NEVER BE USED ON ANY RSD PATIENT BECAUSE THE USE OF ICE AGGRAVATES VASOCONSTRICTION IN THE CHRONIC RSD. IT CAUSES FURTHER HYPOTHERMIA OF THE SKIN, AND ACCELERATE THE COURSE OF THE ILLNESS. It expands the mechanoreceptors zone of recruitment and allodynia surrounding the lesion (Torebjork Principle). The majority of patients refuse the application of ice because of pain aggravation. The so-called "ice and heat challenge" with alternate application of ice and heat is of no use. Realizing that this procedure has been done on experiments and research work to study aggravation of sympathetic function, there is no therapeutic value for the alternate use of the two extremes of temperatures. The stress of the alternate treatment only aggravates the disease further.

The use of ice or Capsaicin has been shown to cause inflammation and death of the nerve fibers, especially the larger myelinated nerve fibers. Then the unmyelinated nerve fibers are left uninhibited and unopposed with acceleration of RSD (large myelinated fibers stop conducting at 20° C, but a myelinated fibers keep conducting down to zero degrees).

3. To counteract the hypothermia in the extremity due to the abnormal function of the sympathetic system, it is essential to encourage the patient to get rid of assistive devices (wheelchair, walker, cane, and crutches). The patient should be instructed to follow the golden rule of perpetual motion. In RSD the condition gets worse with prolonged inactivity or the stress of too much activity.

4. The patient should learn from the human heart which beats approximately once a second for 90 years without taking a vacation. The reason is the heart beats half a second and rests half a second. The same principle should apply to physical therapy in RSD. So of the 90 years heart span the heart works 45 years and rest for 45 years.

The same principle should apply for physical therapy. The patient should be instructed not to do any extensive resting or exercise for a long span of time, but to constantly keep changing position and alternating exercise with rest. If sitting up cause's pain, then walk. If walking causes pain, then lie down. If lying down cause's pain, then go back to the other forms of exercise, etc.

Inactivity gives the signal to the sympathetic system to preserve the circulation in the inactive extremity by vasoconstriction, which aggravates the RSD. Activity does the opposite by demanding more blood circulation to the surface of skin.

PAIN AND EXERCISE

Koltzenburg (1995) [1] has shown that inactivity and immobilization of the extremity, such as the use of cast, brace, wheelchair, etc., stimulates the so-called "sleeping nociceptors." Such small c-fibers nociceptors are usually dormant but with inflammation or increased muscle and deep tissue circulation secondary to CRPS, they become activated and aggravate the pain. These "sleeping nociceptors" are mainly chemoreceptors c-fibers and consist of 25% of all the chemoreceptors c-fibers in subcutaneous and deep structures of the extremity.

H. Hooshmand, M.D.

Reference:

1.Koltzenburg M: Stability and plasticity of nociceptor function, in IASP Newsletter. Jan/Feb 1995; 3-4.

RSD PUZZLE #70

Minor Injury, Common Cause Of RSD

Dr. H. Hooshmand M.D.
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August 20, 1996

Dear Dr. Hooshmand:

I can not thank you enough for making your written clinical studies entitles "RSD Puzzles 1995" available to RSD patients around the country. My college son did a lengthy internet search for me early this summer, and your valued information was sent to me several days later from The International Reflex Sympathetic Dystrophy Foundation, in Lakeville, MA.

Since I was diagnosed with RSD last fall, I have done extensive research into this disease and know that most of the cases begin with trauma to one's extremity, and that, if treated early, success rates increase. My RSD/Sympathetically mediated pain resulted in a very different area, through a quite unusual set of circumstances, so I wanted to present my case to you. Not much has been written about this particular situation.

In March 1992, I injured myself at home through an improper lifting accident, which separated my right sacroiliac joint. Not being one to run to doctors (I had always been very healthy, very active in sports-tennis, skiing, outdoor activities, had a great career in Corporate Training, etc.) I tried to ignore or manage the increasing pain for several months. By August of '92, the pain was so severe that I saw an Orthopedic surgeon (by that time, when I would try to walk, my pelvic bone would tip forward and wouldn't stay in proper position). I was told I would need surgery to fuse the S.I. joint, but I insisted on physical therapy first, which we tried until Dec. - to no avail.

In Jan.'93, I had surgery #1, anterior S.I. fusion with plate and 4 screws placed. Several months passed, pain continued to worsen, and I got 3 more opinions and was advised to wait 6 months before doing another surgery. In July'93, I underwent surgery #2, posterior S.I. joint fusion, bone scraping, and percutaneous placement of 5" screw through S.I. into lower lumbar/sacral area for fixation. Pain was so severe after this surgery, rendering me totally sedentary and bedridden until Sept.'93 when I underwent surgery #3, posterior formal bone block, bone graft, with follow-up body cast for 6 weeks to totally immobilize. I resigned my job and career in Nov.'93, and the pain never decreased. My pain was all across my lower back, and deep into rt. S.I. posterior area, presenting

constant throbbing pain. I couldn't sit, stand, or walk more than 10 minutes before I had to lay flat in bed. These were the symptoms I had since early on in the injury, even before the first surgery.

Having been on crutches most of 1993, and taking Vicodin and Percodan daily to control pain, I was at my wits end, because I could never get levels of pain below 6 or 7, even though I was taking the narcotics. Every several months, we would repeat CT scans, only to find the fusion wasn't happening. I was in physical therapy, pool therapy, hot tub therapy during most of this. By May 1994, I had surgery # 4 to do a percutaneous pinning. Results were still negative; I was still totally sedentary due to severe pain. By April of 1995, CT scan finally showed fusion, but pain levels were still, and I was symptomatic exactly as I was soon after the injury. A month later (May'95), they decided to remove the hardware, thinking maybe this was causing the pain.

Results were still negative as time went on, pain was still severe, and by now chronic and I was living on between 5 & 8 narcotics daily, with no real relief. Last fall, the surgeon had all but given up, and it was then that I was told he suspected I had RSD. He referred me to an anesthesiologist for a series of Lumbar Sympathetic Blocks. I had 8 blocks which injected bupivacaine and the longest relief I got was about 5 days.

THIS IS WHERE YOU BECOME SIGNIFICANT. In early June'96, I received your RSD Puzzles and began to study everything you recommended. (I had done research earlier from our local medical libraries, only to keep reading how hopeless RSD was if it hadn't been diagnosed early and how debilitating it would become). **I WASN'T GOING TO GIVE UP!**

Here are the key things I learned from you that I had never seen in print before or been told, and the success I have had so far:

1. Get off the narcotics because of the pain dependence (I did this 3 days before the next Lumbar block which I had scheduled for June 26, 1996) and I haven't had a need for narcotics since.
2. I got a prescription for Ultram in case I might need it after the block. I've only had about 15 Ultram since that block. I had my anesthesiologist read your suggestions from RSD Puzzle #31 where you recommend using Depo- Medrol in addition to local anesthetic, and he readily agrees.

3. I have combined physical therapy, moist heat, etc. along with the block.

Dr. Hooshmand, the block lasted from June 26-August 5, and I was free of pain the entire time (other than normal muscle soreness resulting from so much activity). The pain began to gradually return the first few days of August; we repeat blocked 3 days later, and I'm again free of pain. My anesthesiologist was so impressed and said he had not seen results like this very often in cases this severe. We are hopeful that the longer we can keep the cycle broken, the longer I can go between blocks, until maybe we can eliminate the altogether.

My husband and I feel so grateful to you for giving me my life back- if it hadn't been for your material, I don't know how my life would have proceeded. Chronic pain for long periods of time is so stressful, draining and debilitating. I have had a wonderful support system through this ordeal, and after several years had passed and the pain remained, we finally stopped praying that I would get well, but that I could put in the right direction to at least find answers. THANK YOU SO VERY MUCH for providing most of the answers.

Sometimes I worry that the pain will keep returning once the steroid wears out of my system, and that maybe I will become immune to it, but for now, we're trying to catch up on lost time and enjoy life.

I would welcome any other suggestions you might have based on my unusual case, and in the meantime, I want to thank you from the bottom of my heart for taking the time to make available such valuable information. I will always admire you and will pass on your information to others suffering from RSD whenever I can.

Sincerely,

Judy

RSD PUZZLE #71

VENIPUNCTURE RSD

An example of small nerve damage to the sympathetic system causing RSD, and one of the most severe type's causalgia (CRPS-II), is due to selective nerve damage to the wall of blood vessel. This disease (which fortunately is rare) is called "Venipuncture RSD". It is caused by the needle-used to draw blood from the vein-accidentally injuring the small nerve fibers (sympathetic nerve fibers) surrounding these blood vessels [1, 2]. It happens one in several million cases of venipuncture (IV needle injury) causing severe causalgic pain. This is one of the most severe and most painful forms of RSD, yet the only damage is in the form of damage done to the small nerves in the wall of the blood vessel. This may be the reason for the misconception that in some cases complex regional pain syndrome (RSD) there is no nerve damage. There has to be some sort of nerve damage to cause such severe pain and CRPS. The nerve damage is microscopic and can not be appreciated by standard tests. As is the case with practically all the other cases of RSD, EMG and nerve conduction times are normal. EMG and nerve conduction times become abnormal in RSD only due to disc complications (inflammation causing entrapment neuropathy) but the rest of RSD patients show no abnormality on EMG and nerve conduction times studies because the EMG and NCV study motor function rather than sympathetic function. Triphasic bone scan is nonspecific. As is the case with the other RSD patients, bone scan is abnormal in no more than half the cases [3].

In Venipuncture RSD, the sympathetic nerves in a very small area of the blood vessel have been injured causing causalgia without involvement of any other non-sympathetic nerves. This is in contrast to the more severe injuries (i.e., fracture of the extremity), where the somatic nerve stimulation overshadows the sympathetic nerve damage. Venipuncture RSD has a rapidly deteriorating course and a very poor prognosis. The prognosis is very poor in contrast to the case where the injury has caused fracture and damages to the non-sympathetic nerves as well. In the experience of Doctor Horowitz [1, 2], only one out of eleven patients showed a significant improvement. In our study of seven patients, only three patients have shown partial improvement. Such a low percentage of improvement is seen only in RSD patients who have ended up having amputation, or in patients who have gone undiagnosed and improperly treated for years. The Venipuncture RSD example is used as a model to contrast the CRPS type of pain as opposed to the usual, run of the mill acute somatic (non-sympathetic) pain seen in more major traumatic cases. Even in Venipuncture RSD, the therapeutic success rate improves if the proper diagnosis and proper treatment are applied in the course (the above example of 3 out of 7 versus 1 out of 11 partial improvements).

H. Hooshmand, M.D.

References:

- 1. Horowitz SH: Peripheral nerve injury and causalgia secondary to routine venipuncture. Neurology 1994; 44: 962-964.**
- 2. Horowitz SH: Iatrogenic causalgia classification, clinical findings, and legal ramifications. Arch Neurol 1984; 41:821-824.**
- 3. Lee GW, Weeks PM: The role of bone scintigraphy in diagnosing reflex sympathetic dystrophy. J Hand Surg [Am]. 1995; 20:458-463.**

For more information regarding Venipuncture RSD/CRPS please view the following article on our website.

Hooshmand, H., Hashmi, M., Phillips, E.M.: Venipuncture Complex Regional Pain Syndrome Type II. American Journal of Pain Management. Vol 11: no 4: 112-124, October 2001.

RSD PUZZLE #72

LUPUS AND RSD

You have brought up a few interesting questions regarding the relationship of RSD and Lupus.

Lupus and RSD have one common feature. Both of them are diseases of the immune system. Lupus is due to the pathological response of the immune system toward skin and blood vessels and the nerves in these areas. RSD is a disease of the sympathetic system. This is not the hyperactive or hypo-active sympathetic disease, but a dysfunctional sympathetic disease. In the early stages there is a tendency for hyperactivity of the sympathetic system and in later stages, especially after a few years and several treatments, the sympathetic system becomes dysfunctional.

There are some features that are common between the two diseases:

1. Pain. Both diseases result in neuropathic pain, which refers to the fact that there is some involvement of the sympathetic system in early stages of these two diseases. This is usually in the form of burning and or stabbing regional pain.
2. Muscle spasm, muscle weakness and poor circulation to the skin and muscles.
3. The disturbance of the immune system is invariably present in both diseases. In the early stages of RSD, there is an up regulation of the immune system, and in later stages, there is a down regulation of the immune system. Lupus also goes through different stages of pathological regulation of the immune system.

The next question you had was in regard to the position of RSD in existing Lupus. RSD can happen as a complication of many different immune system diseases, such as Multiple Sclerosis, Lupus, Rheumatoid Arthritis and Polymyositis. It is definitely logical to conclude that the RSD in your case has been a complication of Lupus.

You have asked "How can one tell if the RSD is progressing throughout the body?" In RSD Puzzle #18 I describe how RSD spreads to other parts of the body.

You have asked two questions regarding the use of Stadol and Zoloft. Zoloft is an excellent antidepressant, but has very little analgesic value. It is very well tolerated and the usual dose is anywhere between 50 to 150 mg. Sometimes it is most effective when it is increased up to 200mg.

In regards to Stadol, Stadol is an effective analgesic. It is quite potent, without evidence of physical addiction. Like any other drugs which are a strong analgesic, there is a tendency in a small number of patients for abuse, misuse and over use.

However, physical withdrawal is not a complication of Stadol. It is an opiate antagonist, meaning that it does not suppress the formation of endorphins in the brain. The main drawback of Stadol is its cost. Buprenex which is superior to Stadol is less likely to be abused by the patient, and its annual cost is one half the annual cost of Stadol. It is mainly used to detoxify patients from dangerous drugs such as Methadone, Morphine, Heroin and Cocaine.

The last question you brought up was in regards to physical therapy and to testing and treatment for an orthopedic problem. Physical therapy is beneficial for an orthopedic problem. Physical therapy is beneficial at any stage of RSD. In regard to undergoing any kind of invasive test for an orthopedic problem, not only should the patient have physical therapy for several days before the test, but also it is a good idea to protect the patient with sympathetic nerve block before, during and after the test.

H. Hooshmand, M.D.

RSD PUZZLES #73
SKIN LESION AND RSD

Many thanks for your letter of September 22, 1996.

You have mentioned the fact that you were involved in two accidents. The first one resulted in RSD and was treated effectively with nerve blocks. This was followed by another accident on October 24, 1995. This near fatal accident resulted in the development of RSD in the right leg. The nerve blocks helped with treatment of the RSD in the right leg.

Now you have developed an area of skin lesion over the scalp of the head, from the forehead all the way to the vertex. You have mentioned not only reddish discoloration, but pain and tenderness in the area. You have been told that this could not be related to RSD.

The most likely diagnosis in your case is that either due to the same car accident or some other previous injury, you had an area of damage to the scalp. This has been complicated by sympathetically maintained pain (SMP). It is not at all unusual for RSD being present in upper and lower extremities and then spreading to the head and neck regions.

First of all, you need to be seen by a neurosurgeon, as well as a good dermatologist. Secondly, this area should be treated with topical Lidocaine and topical Zonalon Cream, and you should definitely have a stellate ganglion nerve block to find out if the nerve block can improve your condition.

It makes no sense to come up with a separate diagnosis when you have already had RSD in two different parts of your body.

If the inflammation does not improve, I recommend treatment with I.V. Mannitol (80-100 grams, depending on your weight, in 1000 cc D5W). This should be given twice a week for three weeks and it will help the condition to clear up.

H. Hooshmand, M.D.

RSD PUZZLE #74

SPINAL CORD STIMULATOR (SCS)

Please, view RSD Puzzles 23 and 106 also regarding SCS

Dear Dr. S.,

Many thanks for your letter of 10/7/96. It was very interesting that you had quite a similar experience regarding the spinal cord stimulator treatment for CRPS (RSD).

I have seen 36 patients so far suffering from RSD who have received spinal stimulator for management of the pain. Of the 36 patients, the pain relief lasted an average of six weeks. Relief was as short lasting as a few hours and as long as over four months. Eventually the pain recurred with more severity.

More importantly, the foreign body of the stimulator in a patient who already had disturbance of plasticity due to long standing RSD, resulted in two phenomena:

1. Spread of RSD to the opposite extremity, as well as development of headache, neck pain and dizziness.
2. Reactive spinal cord ischemia resulting in transient paraparesis and incontinence in two cases, and the development of myoclonic type of jerks in the lower extremities in four other cases.

It is about time to differentiate the neuropathic pain of RSD from the somatic pain of failed neck or failed back syndrome.

In the same follow-up study of the patients, we have noted the effective response in control of the somatic type of pain in 32 failed neck and failed back patients with an average duration of relief being 18 months. No such complications of spread of the disease are noted in somatic (non-CRPS) patients.

In regard to your patient, I am glad she is being treated by you, and if I can be of any help, I will be at your service. Even though we have had excellent results (over 70% success) in 96 RSD patients treated with infusion pump, we always like to save the infusion pump treatment as a last resort because of problems of potential of infection (due to the disturbance of immune system secondary to RSD), and the patients intolerant of even the smallest doses of narcotics in the spinal fluid (noted in 11 patients).

H. Hooshmand, M.D.

RSD PUZZLE #75

FROZEN SHOULDER: NO SURGERY

In the case of frozen shoulder, we have adopted an aggressive approach for treatment with trigger point injections. The natural history of the frozen shoulder is the gradual deterioration of the condition complicated by inflammation due to RSD. The inflammatory effect mimics a clinical picture of rotator cuff tear, and even though almost all frozen shoulder cases are due to remote sources of RSD (e.g., hand and wrist injury, elbow injury, heart attack or stroke) without any history of direct trauma to the arm or to shoulder to cause rotator cuff tear, the orthopedists have a tendency to explore the shoulder to perform a "rotator cuff tear surgery". It is obvious that postoperatively, such a patient undergoes a severe deterioration of their RSD.

If the patient is not being treated by an orthopedist, and if a more conservative approach is applied, the patient unusually undergoes a few stellate ganglion blocks, followed by one or two trigger point injections of the shoulder, followed by manipulation of the shoulder under general anesthesia. This produces usually only provides a few days or weeks of relief and the condition recurs with more severity. By then, the trauma of manipulation of the shoulder may end up causing rotator cuff tear, to prevent such disastrous results, we have successfully resorted to aggressively injecting and eradicating the multiple trigger points around the should with Marcaine mixed with a minimal amount of Depo-Medrol®.

In the case of your patient, because of the severity of her frozen shoulder, and because the patient had not received any Depo-Medrol® in the past, the patient was treated with the maximum allowable trigger point injections. The patient had excellent results from it.

Once the patient has had these injections, the patient is instructed to immediately exercise the shoulder and keep using it in order to prevent further recurrence of the immobilization and frozen shoulder. We have not had any case that the patient had to come back for recurrence of the frozen and repeat of the treatment.

H. Hooshmand, M.D.

RSD PUZZLE #76

Deformed Hands And Extremities In RSD

The medical files of 495 RSD patients were divided into two categories:

Category A. Patients with no extremity deformity.

Category B. Patients with extremity deformity.

The following parameters were studied:

1. Duration of the illness before the deformity started.
2. Risk factors:
 - 2A. Application of ice for longer than 2 weeks versus shorter than 2 weeks.
 - 2B. Surgery for entrapment syndromes (carpal tunnel, tarsal tunnel, ulnar nerve transposition, and resection for thoracic outlet syndrome)
 - 2C. Use of assistive devices such as cast, brace, or wheel-chair
 - 2D. Little or no physical therapy
3. Risk factors consist of surgical treatments in the form of:
 - 3A. Spinal cord stimulator (SCS)
 - 3B. Amputation
 - 3C. Chemical sympathectomy
4. Surgery for neuroma exploration
5. Rotator cuff surgery
6. Single versus multiple operations in the injured area

CRPS I (RSD) VERSUS CRPS II (CAUSALGIA)

RESULTS

1. Time duration. The patients with deformity had an average lag of 22.3 months delay between the onset of the disease and the first diagnosis of RSD. This was in contrast with the no-deformity patients who had a lag of 14.5 months between the onset and diagnosis.

2. Treatment with ice or heat and cold challenge, 2 weeks or more. The patients with deformity were treated with ice or hot and cold challenge for an average of 4.6 months versus the patients with no deformity for an average of 3.1 months. In both groups, the hypothermia therapy was usually discontinued due to the persistent protestation of the patient against ice treatment because of aggravation of pain.

2B. Entrapment surgery. The surgery for carpal tunnel syndrome, tarsal tunnel syndrome, thoracic outlet syndrome, rib resection, and ulnar nerve transposition was performed in 38% of the patients with deformity versus 14.5% of the patients without deformity.

2C. Assistive devices. In this category, the use of assistive devices after the development of deformity was excluded from comparison. This was done because of the fact that the patients suffering from deformities were in more need of assistive devices than the ones with no deformity. After excluding such patients, the use of assistive devices showed no statistical significance between the two groups.

3A. The use of spinal stimulators. The non-traumatic spinal stimulator procedures showed no statistically significant higher rate of deformity. On the other hand, of the 44 patients who had received spinal stimulator treatment, 3 patients had traumatic complications of temporary or permanent paralysis of the lower extremities which required assistive devices.

Of the 42 patients who received spinal stimulator treatment, followed for more than 2 years, the majority of the patients lost any beneficial pain relief from the spinal stimulators after as early as 7 days and as long as 13 months duration.

3B. Infusion pump: Infusion pump done on 96 patients proved to be no risk for development of deformity. If anything, the patients who received Baclofen in the pump had a significant relief of spasticity and reduction of deformity.

3C. Sympathectomy: This group was divided to standard sympathectomy, and chemical sympathectomy. The post-sympathectomy deformity developed within a few months after the surgery. Of the 62 patients who had undergone standard or chemical sympathectomy, 14 developed post-surgical extremity deformity.

4. "Neuroma" exploration: This was usually done over the foot and ankle area by podiatrists. This group consisted of 42 "neuroma" explorations, and 18 ended up with deformity post-operatively

Interestingly, of the 42 "neuroma" explorations, only 3 patients were in possession of the pathologic microscopic biopsy sample. None of the three biopsies showed neuroma.

5. Rotator cuff surgery: Fifty-eight patients had undergone "rotator cuff surgery" procedure. Only 1 patient had the pre-operative MRI diagnosis of rotator cuff tear. 14 others had MRI which was either negative or showed inflammatory changes over the shoulder region. In this group, the frozen shoulder and the flexion deformity of the arm and forearm became aggravated and more disabling post-operatively in 29 patients.

Of the rotator cuff tear surgical patients, only 3 had direct injury to the shoulder. The rest had no history of direct injury to the shoulder. These consisted of patients who suffered from hand, wrist, forearm, or elbow injuries and a small number (5) patients had the onset of the disease after heart attack or stroke. There was not even one patient in the no-deformity group who had already undergone "rotator cuff surgery" procedure.

6. CRPS I (RSD) versus CRPS II (Causalgia) categories: There was no statistical difference between the two categories in regard to the incidence of complication of limb deformity. However, the causalgic group developed the limb deformity earlier in the course of the illness. The average lag time between trauma and the development of the deformity in CRPS II (causalgia) group was 7 months.

CONCLUSION

The risk factors contributing to the development of limb deformity consist of surgical procedures, exploratory operative procedures (such as looking for neuroma or looking for entrapment neuropathy), immobilization with cast or wheelchair, and prolonged use of cryotherapy (application of ice).

The deformity evolved earlier in the CRPS II (Causalgic) group than in the CRPS I (RSD) group.

H. Hooshmand, M.D.

RSD PUZZLE #77
RSD and MRSA

Question:

Is there any relationship between MRSA and RSD?

Answer:

I have treated six MRSA patients among RSD and Electrical Injury patients. This disease is due to the combination of poor immune system function secondary to neuropathic system dysfunction, inflammation, and chronic illness contributing to the above. In regards to treatments the following are the do's and don'ts.

Don'ts:

Do not let anyone operate or resect it because it causes spread and rapid deterioration making it much worse.

Do not cover the area with bandage because it will heal slower and will become infected.

Do not be hospitalized: inactivity and nosocomial (hospital germ infections) aggravate it.

Do's:

Keep the area open and dry. Nerve blocks can help, but not at the area of damage.

Soaking with warm water and Epsom salts.

I.V. Immunoglobulin

I.V. Mannitol Treatment

Move around, become active. Inactivity aggravates the infection.

Avoid long term antibiotic treatment.

H. Hooshmand, M.D.

RSD PUZZLE #78

Dangerous RSD Treatments (The Fads That Cause Failure of RSD Treatment)

Question:

"What are the reasons for failure of treatment of RSD"? - To put it another way, what are the dangerous forms for treatment of RSD?

Answer: Here are some reasons for failure of treatment:

1. "RSD burns itself out and goes away after a few years." This is a big lie! It only goes away if the patient has had multidisciplinary treatments. The doctor is not intentionally lying. But after a few years of no effective treatment the patient goes to another doctor, does not see the original doctor and then the original doctor concludes that the patient must have been cured, because the patient did not return.
2. Instructing the patient to be confined to a wheelchair because he or she in pain.
3. Clonidine patch applied to the area of lesion in the extremity rather than the area of referred pain in cervical or lumbar spine region. (See #16 below)
4. Only one or two trigger point injections for frozen shoulder.
5. Bier Block needle insertion in the area of flared up RSD.
6. Treatment with ice.
7. Hot and cold challenge treatment.
8. Only 10mg or 25mg tricyclic anti-depressant per day for treatment of pain.
9. Repetitive Sympathetic nerve blocks after extremity becomes warm and has undergone virtual sympathectomy with nerve blocks.
10. Monotherapy with nerve block, or opioid medication.

11. Indiscriminate use of Neurontin. Neurontin is exclusively effective for burning type of pain, so if the patient has dull ache or electric shock type of pain then Neurontin won't work.

12. Small doses of anticonvulsant such as 100mg-300 mg of Neurontin or 1-2 Tegretol a day. Two principles should be followed: First of all, the dosage should be enough anticonvulsant to manage the burning pain (Neurontin), or the stabbing electric shock pain (Tegretol). Secondly, generic anticonvulsants are useless (according to the American Academy of Neurology)

13. Instructing the patient to bathe while wearing the clonidine patch.

14. Mistaking paravertebral nerve blocks for articular facet injections.

15. Mistaking diagnostic sympathetic nerve block with simple Marcaine injection as a therapeutic block.

16. Treatment of high blood pressure with newer anti-hypertensives rather than the alpha blockers. When the patient is treated with alpha blockers such as Dibenzylamine, Hytrin, or Clonidine, not only is the hypertension managed, but also the patient receives systemic sympathetic block. It is true that RSD is a dysfunction of the sympathetic system, but in different parts of the body the dysfunction is different. At the area of the nerve damage at the apex of the sympathetic nerve injury, there is focal hyperthermia pointing to an ephaptic nerve damage paralysis of the sympathetic function. In earlier stages of the disease, this area is surrounded by vasoconstriction and SMP (sympathetically mediated pain) pointing to the compensatory hyperactivity of the sympathetic system. The wide dynamic range (WDR) at the spinal cord level as well as the stimulation of the sympathetic system through the paravertebral sympathetic ganglia causes regional and remote sympathetic stimulation. Treatment with alpha blockers helps ameliorate this condition. The opposite is also true. The application of Clonidine patch over the area of sympathetic nerve damage (the vortex of the sympathetic paralysis and heat emission) only aggravate the condition. On the other hand, the application of the Clonidine patch over the referred pain area in the involved paravertebral region relieves the sympathetic dysfunction.

17. Trigger point injections and nerve blocks in the nerve damaged area while paravertebral nerve blocks and trigger point injections are helpful when applied to the paravertebral nerves in the regional referred pain area of the spine, the same trigger point injections or nerve blocks when applied to the area of nerve damage aggravate the RSD. One example is BIER blocks. BIER blocks are very effective unless the intravenous injection is done in the involved area of the extremity. If the patient has had an injury to the dorsal aspect of the foot resulting in RSD, insertion of an IV needle in this area flares up the condition and

adds insult to injury, in this situation, The BIER block does not relieve the patient's pain, but aggravates the disease.

18. Reading too much into the Phentolamine block result. In the late stages of RSD, the SMP changes to SMP and SIP or purely SIP (sympathetic independent pain). This may be due to the therapeutic trauma such as multiple nerve blocks or surgical procedures (surgery for carpal tunnel syndrome, tarsal tunnel syndrome, or thoracic outlet syndrome), or simply long-standing vasoconstriction of the region of CRPS (Chronic regional pain syndrome) causing long standing hypoxia involving somatic as well as sympathetic nerves. The end result is that frequently after several months or a few years, the CRPS pain is not sympathetically mediated anymore. Even in early stages of the disease, the hyperpathia is mainly transmitted through the thermal receptors (SMP) whereas allodynia is transmitted to the mechanoreceptors (a-delta fibers resulting in mechanoallodynia in contrast to small c-thermal receptor fibers). So, sooner or later the mechanoallodynia becomes the main feature of the illness and the SMP changes to SIP. Treatments such as application of ice, immobilization of the extremity, excessive use of narcotics such as MS Contin, Duragesic, or Methadone, which cause a reduction of the mobility of the extremity, result in secondary aggravation of the mechanoallodynia. This is also aggravated by stimulation of the so-called sleeping nociceptors secondary to immobilization. In such patients, SIP is the general rule of thumb rather than being an exception. In patients receiving large doses of narcotics, there is a tendency for edema of the lower extremities, attacks of inflammation secondary to stimulation of the sleeping nociceptors. This inflammation is manifested by spontaneous bruises, neurodermatitis, along with painful edema of the extremity.

19. Amputation

20. Chemical sympathectomy

21. Cingulotomy

22. Neuroectomy

23. Cryosurgery

24. Alcohol nerve block

25. Phenol nerve block

H. Hooshmand, M.D.

RSD PUZZLE #79

ATTACKS OF STABBING AND ELECTRIC SHOCK PAIN IN RSD PATIENTS

The type of pain, described above by a patient is the classic "Causalgic pain". The word Causalgia was first coined by Doctor S.W. Mitchell, the first physician who reported the existence of RSD. This type of pain is caused by practically and electric shock short between adjacent damaged nerve fibers.

The normal nerve fibers are insulated with a fatty sheath called "myelin". If an injury (usually minor, occasionally due to sharp objects, or even bullets) causes damage to the adjacent nerve fibers, the insulating sheath is damaged resulting in the electrical current in the nerve fiber (which works as an electrical wire conducting messages) to spread to the adjacent damaged nerve fibers and to irritate the adjacent nerve fibers. Eventually, the cumulative effect of the multiple areas of nerve damage and irritation results in a sudden electrical discharge that is strong enough to stimulate an electric shock type of pain sensation.

At times, it is so severe that the arrival of such an electric shock to the spinal cord causes an extremely transient shock to the nerves that are responsible for the posture and balance, and as a result, the patient has a tendency to either completely or partially fall to the ground.

This symptom, like any other symptom of RSD, becomes more severe and more prominent due to aggravation of the disease by inflammation and edema. The falling attacks are seen in late (3rd and 4th) stages of RSD.

This electric shock type of pain is usually seen in causalgia which is also called CRPS II.

The treatment of choice for this condition is an anticonvulsant called Tegretol. Unfortunately, because of the cost problems, the pharmacist dispenses the generic form called Carbamazepine. Unless the physician specifies "no generic" the patient ends up with treatment with the generic Carbamazepine. This is a rare situation where the generic does not at all work the same as the brand name Tegretol.

In patients suffering from epilepsy, it has been a well known fact for decades that Tegretol is an excellent anticonvulsant, but the generic form Carbamazepine is not even half as effective.

Due to the above mentioned facts, other anticonvulsants have been tried for treatment of causalgic, electric shock pain. These have consisted of Dilantin, which is only partially effective, and, recently, Neurontin (Gabapentin).

Unfortunately, there has been a tendency for overuse and disuse for Neurontin (Gabapentin). There is no sense and no proof that Neurontin (Gabapentin) can do any good with a patient with CRPS I (disuse RSD). It is mainly an anticonvulsant and unless the patient has the above described type of pain, the Neurontin doesn't do much for the continuous sharp, burning pain.

Even though Gabapentin (Neurontin) has a beneficial psychotropic effect (makes the patient feel better), Tegretol has even a stronger psychotropic beneficial effect.

If the patient cannot tolerate any of the above medications, then treatment with Klonopin or even Valproic Acid should be considered.

In addition to the above mentioned anticonvulsants, the patient should also be treated with an effective antidepressant with least side effect. In this regard, Amitriptyline (Elavil) should be avoided because it has a tendency to cause inactivity, fatigue, obesity, and disturbance of pulse and blood pressure. The SSRI antidepressants such as Prozac and Paxil are effective analgesic type of antidepressants, but one in five patients treated with these SSRI antidepressants develop sexual difficulties in the form of lack of desire and poor potency. Antidepressants such as Effexor which have a tendency for stimulating the patient due to dopamine re-uptake inhibition should also be avoided.

After review of the above list of antidepressants, one is left with two antidepressants with the least side effects and best results for RSD patients, i.e. Trazodone and Desipramine.

H. Hooshmand, M.D.

RSD PUZZLE #80

COLD AND HEAT TOLERANCE

As you are well aware, there are early, intermediate, and late stages of RSD (stages I - IV).

Stage I, is referred to as reflex sympathetic dysfunction. Stage II is dystrophy. Stage III is characterized by atrophy. Stage IV is characterized by sympathetic system's eventual failure and destruction. The stage IV is also characterized by the failure of sympathetic function manifested in some patients by low, rather than high blood pressure and pulse, as well as low, rather than high regulation of the immune system.

In stage I, the injured area shows hyperthermia (increased temperature) rather than hypothermia. This is because of a temporary shock to the sympathetic system not being able to preserve heat and to control any heat loss over the skin of the injured area. In a matter of a few weeks, the majority of such patients (over four-fifths of RSD patients) regains the function of the sympathetic system and develops cold skin over the skin surrounding the area of nerve damage. Such patients cannot tolerate ice. If anything, ice aggravates the disease and exaggerates the constriction of the blood vessels and hypothermia (coldness) of the extremity. Even in these patients, the small central area of nerve damage at the area of maximum brunt of the trauma, an area is left with total paralysis of the sympathetic function showing as a pin-point area of hyperthermia on thermography. In these patients which are the majority of RSD patients, ice application should be avoided by all means.

In one-fifth of the cases when hyperthermia persists for a few more weeks, the patient will continue to be intolerant of heat or ice.

In stages II and III, the extremity progressively develops more and more hypothermia due to persistence of the dysfunction of the sympathetic system.

Eventually, towards the end of stage III and beginning of stage IV, in some cases the hyperthermia has a tendency to recur. This phenomenon is in part due to the fact that the patient has had repeated sympathetic ganglion blocks. The repetitive, numerous sympathetic ganglion blocks cause what is called "virtual sympathectomy". This results in gradual hyperthermia of the extremity (warming of the extremity). Even though the extremity becomes warm due to the virtual permanent damage to the sympathetic system, the pain does not get any better. In these patients, again, heat should be avoided.

Regardless of which type of heat intolerance or cold intolerance the patient is dealing with, the so-called heat and cold challenge treatment does no good in RSD patients. It only confuses the diagnosis and treatment and it should be avoided.

In many patients, in stages II and III, in the same extremity, there are islands of sympathetic paralysis, (hyperthermia) and islands of marked hypothermia due to sympathetic nerve irritation. These are the cases that do not respond properly to sympathetic nerve blocks and are classified as SIP (sympathetically independent pain).

The above only reflects how sophisticated the sympathetic nervous system function is and how confusing the clinical picture is.

Even though we don't use thermography for diagnosis of RSD and we consider the diagnosis of RSD being a clinical diagnosis, thermography can shed a lot of light on the above confusing issues. Both the Mayo Clinic Group and Doctor Ochoa consider thermography as the diagnostic tool of choice for understanding the nature of the RSD pathology (see Muscle and Nerves, 1994, Cordoso and Ochoa, see Mayo Clinic Proceedings 1995).

H. Hooshmand, M.D.

RSD PUZZLE #81

Diagnosis Of RSD With Nerve Blocks

There have been emphatic statements saying that RSD can only be diagnosed by sympathetic nerve blocks. This may be true in approximately 90% of RSD patients in the first six months, 60% of RSD patients in the first one year or around 50% of RSD patients in the first two years. However, as the disease progresses, the sympathetic nerve block test shows Sympathetically Independent Pain (SIP) in a high percentage of the patients. As a matter of fact, after five years more than 4 out of 5 of the patients are SIP and the nerve block is negative. There have been many patients that have been told that they do not have RSD just because the sympathetic nerve block is negative, even though the patient had been diagnosed with RSD for five to seven years.

RSD is not a simple hyperactivity of the sympathetic system. It is a dysfunctional and rogue sympathetic system. The more chronic it becomes, the less likely it is to be sympathetically mediated pain. RSD is not a diagnosis by exclusion or by nerve blocks. It is a diagnosis made clinically and inclusively. The whole principles of:

1. Hyperpathic allodynic pain.
2. Muscle spasm with constriction and movement disorder.
3. Inflammation.
4. Disturbance of the limbic system causing insomnia, agitation and depression.

The above are the four minimum requirements and the most effective diagnostic criteria for the accurate diagnosis of RSD. Bone Scan or Thermography are not accurate enough to exclusively diagnose RSD.

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RSD PUZZLE #82

Chemical Sympathectomy

There have been questions regarding chemical sympathectomy, chemical neurectomy, injection of phenol, alcohol, etc... as well as radio frequency lesion of the sympathetic system. These are extremely dangerous. They are similar to neurectomies, rhizotomies, thalamotomies and other forms of surgical nerve damages which cause severe aggravation of RSD and the spread of the disease to other parts of the body.

Any kind of alcohol, phenol or hypertonic saline nerve blocks aimed at destroying the nerves (Chemical Sympathectomy) are apt to fail, to cause serious complications, and aggravation of the pain- by leaving a large scar behind.

In my 35 years of Neurosurgery and Neurology training as well as teaching I have yet to see any of the above procedures helping a single soul. Unfortunately the young surgeons who do these procedures do not have time to read the 1950, 1960 and 1970 literature which reflects how damaging and destructive such operations are.

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RSD PUZZLE #83

Antidepressants and Pain

Narcotics are essential in treatment of pain (not the morphine agonists causing withdrawal pain, but the agonists. (See below for more on this)

Elavil has been around for over a quarter of century. It has been most exhaustively studied. It is an excellent analgesic. If you cold turkey Elavil your pain will recur with a vengeance. However in the first year of taking Elavil an average patient gains 18 pounds of weight. It also causes fatigue, low blood pressure and is stressful to the heart muscle. *

Another tricyclic, Desipramine, is an excellent antidepressant and analgesic without the above mentioned side effects of Elavil.

Even though antidepressants prevent pain, they do not replace the proper use of narcotic analgesics. The two are complimentary.

Trazodone is as effective as Desipramine or Elavil in pain control. It should be started in a small dose and gradually increased and adjusted upward or downward to achieve 8 hours of uninterrupted sleep, without the side effects of drowsiness the next day.

The SSRI's: Prozac, Paxil and Zoloft are excellent antidepressants. However one out of five patients loses their sex desire with SSRI's. This loss is bad for anybody's health. Zoloft is the least analgesic. Prozac is the most likely to cause agitation.

* The use of Elavil should be avoided- It should be replaced with Desipramine, Trazodone, etc... according to the patient's tolerance.

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