RSD PUZZLE #21
Symptoms used to diagnose RSD

"I have suffered from RSD for the past eight years. In the first few years, sympathetic nerve blocks helped me. Last month the doctor did a diagnostic sympathetic nerve block which did not relieve my pain. The doctor says that I do not have RSD because the diagnostic test ruled it out. How can I not have RSD when I have had all the other findings of it in the past eight years?"

One of the most accurate ways of diagnosing RSD in a patient who has had other manifestations of it (constant burning pain, weakness or movement disorder of the extremity, emotional disturbance, and insomnia, as well as evidence of swelling and inflammation of the extremity) is to do a sympathetic ganglion or IV Phentolamine) tests. In the first two to three years, such a sympathetic nerve block test definitively relieves the patient from her pain. Such a positive response of pain relief proves that the patient suffers from "SYMPATHETICALLY MEDIATED PAIN" (SMP).

However, after two to three years of suffering from RSD, the longstanding poor circulation and constriction of the blood vessels as well as the inflammation and swelling secondary to RSD, affect the non-sympathetic (somatic) nerves as well. So the patient develops not only SMP but also the pain that is independent of sympathetic system function due to the lack of oxygen to the somatic nerve fibers (non-sympathetic nerve fibers). This type of pain, which does not respond to sympathetic nerve blocks, is called "SYMPATHETICALLY INDEPENDENT PAIN" (SIP).

The end result is that longstanding RSD causes the development of a pain that is independent of the sympathetic system due to the poor circulation and the swelling. The patient develops pain that is severe, has the sympathetic component of a constant burning pain, but sympathetic block loses its effect due to the long- standing damage to the nerves.

Because of lack of familiarity with such a complex phenomenon, a lot of patients are accused of having never had RSD and they are deprived of treatment due to the SIP component of the pain.

As long as in the early stages of the disease the patient has had SMP (sympathetically maintained pain) confirmed by complete relief of pain to sympathetic nerve blocks, there is no reason to doubt the illness later on when the condition becomes more complicated.

Another factor is that even though Phentolamine IV nerve block relieves the sympathetic pain, attempts at sympathetic nerve block by direct injection to the sympathetic nerve ganglia (such as stellate ganglion block) even in the best of hands and in the hands of the most experienced physicians faces a one-fourth (approximately 25%) risk of the block not being successful due to anatomical variation and due to the fact that the sympathetic ganglion is not exactly where it is supposed to be. So, because of this one-fourth failure rate of technically and successfully blocking the sympathetic ganglion, the RSD cannot be ruled out on the basis of "SIP" diagnosis.

There is no one test in the world that can definitively 100% rule in or rule out RSD. Even IV Phentolamine test is fraught with handicaps of not completely relieving the pain of RSD due to the simultaneous complication of SIP if the patient's original injury has also caused some somatic (non-sympathetic) nerve damage to the area involved with RSD.

As the world literature reflects, the fact that the triphasic bone scan test is successful in only 55 to 65% of the patients [1]. This is due to the fact that RSD not infrequently causes symmetrical bilateral involvement in the extremities also due to the fact that in chronic RSD that has been partially treated the bone scan may be insensitive and may not show the abnormal isotope uptake in the extremities. Thermography is not, by far, 100% positive in RSD patients, and at best has a sensitivity of around 80%. Both bone scan and thermography, like any other test (including MRI and CAT scan), are handicapped by false-positive and false-negative results and showing changes expected in RSD patients when the patient already has had an old injury and does not suffer the full picture of RSD anymore.

The diagnosis of RSD should always be a clinical diagnosis. The diagnosis of RSD cannot be made on the basis of "ruling out other causes". It is an insensitive and inaccurate way of diagnosing RSD.

There is no way one can "rule out" other causes. The patient with cancer, RSD, epilepsy, or any other serious illness can also suffer from the clinical manifestations (conversion reaction) and/or malingering. Just to prove conversion reaction or malingering does not rule out co-existence of cancer, RSD, multiple sclerosis, or other complex and serious illnesses.

The best guideline for the diagnosis of RSD is the presence of the following criteria:

1. A constant burning pain that is elicited even with a breeze or a touch (allodynia).

2. Any manifestation of the disturbance of motor function in the extremity such as constriction of the blood vessels (cold extremity and poor circulation), or movement disorder such as tremor, dystonia and flexion spasm, atrophy or weakness of the muscles of the extremity.

3. Evidence of inflammation (swelling) in the involved area. This may be in the form of simple swelling (edema), skin rash (neuro-dermatitis), spontaneous bleeding, blotchy skin, and other forms of discoloration of the skin.

4. Disturbance of limbic system function. The sensory sympathetic nerve fibers ascend through the spinal cord up to the brainstem and thalamus and terminate in limbic system (marginal system which is at the margin of old and new brain). This system, which is mainly over the temporal lobe and frontal lobe regions, is responsible for control of emotion, expression of proper judgment, and memory function, and control of diurnal cycle (through the brainstem influence on the sleep wakeful cycle).

A true RSD patient suffers from insomnia, agitation, depression, disturbance of judgment manifested by willing to have any type of operation and any other type of treatment that comes by, and complications of attempted suicide, as well as weight fluctuation.

Without some manifestation of the above four categories, one cannot make the diagnosis of RSD. RSD cannot be ruled in or out by trust of exclusion. For example, if the patient has carpal tunnel syndrome secondary to RSD, then the patient's diagnosis is not a simple carpal tunnel syndrome but RSD causing carpal tunnel syndrome, etc.

H. Hooshmand, M.D.

Reference:

1. Lee GW, Weeks PM: The role of bone scintigraphy in diagnosing reflex sympathetic dystrophy. J Hand Surg [Am]. 1995; 20:458-463.

RSD PUZZLE #22
Possible Timelines Of RSD Recovery

"Will RSD always be with you even if you recover from an original injury?

If RSD is treated early and properly, it will not hang around forever. It will definitely improve and in better than 80% of the cases when treated in the first 6 months it completely cleared up. After two years the percentage of success drops precipitously. The main exception is among children and teenagers. In this group there is such a strong recovery power that the prognosis is usually excellent and it is hard to mess them up with improper treatment with these patients the sympathetic dysfunction after successful treatment becomes asymptomatic. On can always pick the abnormalities up on thermography or in occasional cases on bone scan tests in a patient who is otherwise asymptomatic, but these tests show sympathetic dysfunction. The sympathetic dysfunction alone is not the same as RSD (Please see RSD Puzzle #1 "What is RSD").

H. Hooshmand, M.D.

RSD PUZZLE # 23
WHY NOT SPINAL CORD STIMULATOR (SCS)?
Please view RSD Puzzles #74 and #106 also regarding SCS

Spinal cord stimulator (SCS) has a limited role in treatment of CRPS. As you are well aware, there are two different types of pain, in two different stages of CRPS. In the early stages, in the first few months, the pain is sympathetically maintained pain (SMP) meaning that the pain responds to sympathetic ganglion blocks. However, on the average of almost a year, the nature of the pain changes from sympathetically maintained pain (SMP) to sympathetically independent pain (SIP).

The spinal cord stimulator is effective in the SMP phase of the CRPS, not the SIP. The recent research has shown that the later in the course of the disease spinal cord stimulator (SCS) is started, the less likelihood SCS will relieve the pain.

As a rule of Thumb, if the stellate ganglion nerve blocks have lost their effect, about the same time the SCS loses its effect. This is because both treatments aim at the sympathetic system. With passage of time, as the pain gradually changes to SIP, such treatment cannot be expected to help.

What it boils down to, is the fact that if the stellate ganglion nerve blocks have lost their therapeutic effect, then what is the sense of doing SCS? If the pain has become SIP, what is the sense of doing SCS treatment?

SCS is a digital stimulator utilized for treatment of an analog symptom (the analog pain modality is random and not time locked or digital). It is not a type of treatment that would be successful in every form of chronic pain.

The reason we do not apply SCS is because if the sympathetic ganglion nerve blocks do not work, then epidural nerve blocks which contain Depo-Medrol® applied to the epidural space in the spinal canal are far more effective, and their pain relief lasts longer. On the other hand, even in a patient who suffers from SMP type of pain, after about a year the successful treatment with SCS will fade away because the SMP has changed to SIP. Then, the patient is left with a foreign body in the spinal canal not providing any decent pain relief. This foreign body causes disturbance of immune system resulting in skin rash, and dermatitis [1] and skin lesions and allergic reaction to SCS [2].

In CRPS/RSD, the immune system is rogue. This is because the immune system is modulated by sympathetic system. The sympathetic system, under pain input, responds by releasing T-cell lymphocytes (in early stages CD4 or helper lymphocytes, and in late stages CD8 or killer T-cell lymphocytes) [3]. So, after the SCS has lost its effect, the sympathetic system considers the foreign body of the

spinal stimulator as a source of sympathetic dysfunction. This causes neuroinflammation manifested as skin rash, edema, and infection.

As the condition becomes chronic, the SCS can lead to spread of pain from the original site to other parts of the body [3].

In rare cases, there are other complications noted with SCS application. These complications consist of the following:

1. Epidural abscess or blood clots.

2. In occasional cases, the sensitization of the spinal cord by the spinal cord stimulator causes spinal cord sensitization in the form of myoclonic akinetic seizures [3]. The sensitization is due to prolonged electrical stimulation causing exhaustion of the inhibitory nerve cells. Treatment with Klonopin® and removal of the stimulator prevents the sensitization.

Such attacks of myoclonic seizures originating from the spinal cord due to the spinal cord sensitization are not limited to the SCS. They are also seen in other spinal procedures [4]. The diagnosis of spinal cord originated myoclonic seizures is quite difficult, and usually these patients are labeled as "functional" or "hysterical." Such patients respond very nicely to treatment with Klonopin®, brand name rather than generic.

The removal of SCS, as well as Multidisciplinary treatments, aiming at desensitizing the spinal cord, helps this condition.

Another problem with the SCS is the tendency for electrode movement due to improper anchoring, and the necessity for the surgeon try to correct the position of the stimulator. Every operation is going to be another new source of CRPS pain.

In the rare and severe cases of spinal cord sensitization, the patient may develop myoclonic jerks, and urgency, frequency, and even incontinence of urine, secondary to SCS irritating the urinary bladder and interstitial cystitis.

H. Hooshmand, M.D.

References

1. McKenna KE and McCleane G: Dermatitis induced by spinal cord stimulator implant. Contact Dermatitis.1999; 41: 229.

2. Ochani TD, Almirante J, Siddiqui A, et al: Allergic reaction to spinal cord stimulator. Clin J Pain. 2000; 16; 178-180.

3. Hooshmand H, Hashmi H: Complex regional pain syndrome (CRPS, RSDS) diagnosis and therapy. A review of 824 patients. Pain Digest. 1999; 9: 1-24.

4. Rosenblum, JA: Spinal abdominal myoclonus. The Neurologist. 1996; 2: 784-787.

RSD PUZZLE #24
Visual And Hearing Problems

RSD patients frequently develop blurring of vision, reading difficulty, problem with focusing, and dizziness in the form of vertiginous attacks (either the body or the objects moving around). As well as hearing problems such as buzzing in the ear (tinnitus).

It is immaterial which part of the body has had the damage causing RSD. As the enclosed figure shows, the sympathetic nervous system is intermingled and connected through sympathetic ganglia which are on each side of the vertebrae from lower cervical spine region all the way down to the tail bone. This chain of sympathetic connections causes the spread of RSD to symptoms and signs both across the midline to the opposite side (from hand to hand or from foot to foot) and vertically up and down the spine. As a result, the patient may have RSD due to a knee injury or injury to the foot or hand and yet may develop stimulation and abnormal function of the sympathetic system causing constriction of the blood vessels to the brain. When the blood vessels are constricted in the distribution of vertebral arteries in the cervical spine and in the distribution of the blood vessels providing circulation for the hearing center and brainstem, the patient develops attacks of dizziness, trouble with focusing with the eyes (due to brainstem dysfunction which has the responsibility of coordinating the eye movements), and buzzing in the ears (tinnitus).

Treatment with alpha blockers (such as Clonodine, Hytrin, etc.), as well as newer antidepressants such as Trazodone or Zoloft, provide excellent relief for the above symptoms (figure enclosed) and Muscle relaxants such as Baclofen and Trizanidine.

At times the original injury that has caused RSD may cause retinal detachment (damage to the retina of the eye) or bleeding of the eye. For this reason, the patient should have careful eye examination by an ophthalmologist as well.

H. Hooshmand, M.D.

RSD PUZZLE #25

Failure Of Repetitive Sympathetic Nerve Blocks In Chronic Stages Of RSD

A patient came to see me today because of persistence of pain in all four extremities. She is very upset because she has been told that she needs to have the following course of treatment:

1. Sympathetic nerve blocks. She had no objections to it and I welcome it.

2. Surgery for carpal tunnel syndrome.

3. Post operatively she is to be scheduled to see a psychologist to follow her case.

This is a preordained algorithm which already predicts failure of response to sympathetic nerve blocks necessitating surgery. It predicts failure to surgical treatment necessitating psychotherapy. What is most confusing is if the blocks and surgery are failure, why try them? If the blocks and surgery are failures, why expose the patients to psychotherapy as if to begin with there was nothing wrong with the patient and it was "all in her head"?

Being at the long term receiving end of the type of treatment that has been prescribed for the patient, the stereotyped pattern has convinced me that year-in year -out repetitive sympathetic nerve blocks in chronic RSD are a failure. Surgery is not just a failure, but a disaster in chronic stages of RSD.

The architectural design of the nerve block-surgery-psychotherapy is very similar to the work of an architect in a small town in Florida who designed a dead-end street to start with the police department, followed by the fire department, followed by hospital, followed by nursing home, followed by mortuary, followed by grave yard. The grave yard was quite large so it blocked the road and the sign declared "dead-end".

An old wise farmer once said "there is no lesson to be learned from the second kick of a mule".

The occurrence of carpal tunnel syndrome (CTS) is late stage of RSD is common place. The occurrence of tarsal tunnel syndrome (TTS) in late stages of RSD in the lower extremities is common place. In either case, it is the inflammatory nature of RSD which causes inflammation, swelling, and entrapment neuropathy. In either case surgery for the secondary carpal tunnel or tarsal tunnel syndrome results in disastrous acceleration and deterioration of RSD.

H. Hooshmand, M.D.

RSD PUZZLE #26
"CAN RSD BE INHERITED?"

The answer is a qualified no. RSD is a syndrome brought on by multiple factors (e.g., minor trauma, inactivity, pre-existing injury, and trauma to specific water-shed zones). It cannot be simply inherited.

In our series of 386 consecutive patients diagnosed as suffering from RSD, only 4 families (with more than one member of the family suffering from RSD) were identified.

1. In the first family, two sisters suffering from Fabre' Disease (lipid metabolism disease of genetic nature). Fabre' Disease selectively involves small blood vessels and secondarily has a high tendency for development of RSD (or, more frequently, simple sympathetically maintained pain). As it affects the small arteries, it involves the sympathetic nerves surrounding them. It is not RSD, but the pre-existing genetic disease that made the members of the family susceptible to it.

2. In the second family, there was a strong tendency for auto-immune disease. A mother and her two daughters suffered from RSD. The mother also suffered from Sjogern's Disease, and one of the sisters suffered from lupus (LE). All three ladies had abnormal T-cell lymphocyte ratios. The treatment was aimed at both treating the RSD and treating the auto-immune disease. It is well known that 5% of patients with auto-immune disease (e.g., rheumatoid arthritis, MS, lupus, etc) show a hereditary familial tendency.

3. In the third family, a brother and a sister suffered from RSD. The brother developed RSD after a lightning strike and the sister developed RSD after a mild lumbar spine injury. Both patients had a rough course and eventually needed infusion pump treatment.

4. In the forth family, two sisters and one cousin suffered from RSD at different stages of life, yet every one of them was precipitated by spinal trauma.

Realizing that RSD comprises somewhere between 5-6% of the chronic pain patients, the familial tendency is explained by either coincidence, or other type of hereditary diseases rather than the RSD itself being a pure genetic disease.

Mailis and colleagues [1] have observed abnormal human lymphocytic antigens (HLA) elevation in some RSD patients. Eighty percent of such patients were resistant to treatment. Further study in this regard may help us understand the role of genetics in RSD.

H. Hooshmand, MD

Reference:

1. Mailis A, Meindole H, Papagapiou M, et al: Alteration of the three-phase bone scan after sympathectomy. Clin J Pain 1994; 10:146-5

RSD PUZZLE #27
"I HAVE HAD BLOCKS AND OTHER TREATMENTS THAT DID NOT WORK"

Unfortunately, the above statement is too self-repeating and does not prove that the condition is hopeless. The majority of RSD patients are treated in a piece-mill and partial fashion. Usually, the patient has a few invasive stellate ganglion or lumbar sympathetic blocks with the effect lasting for a short period of time and after 2-3 trials arbitrarily it is decided that the patient has had enough nerve blocks. During the time the patient undergoes nerve blocks; other forms of treatment are not applied simultaneously. If the therapy is continued, usually it is challenged and its beneficial effect is neutralized by the use of ice or alternated heat and ice. Obviously, what ever good the sympathetic nerve block is doing to increase the circulation to the extremity by blocking the sympathetic dysfunction, it is completely neutralized by the use of ice which reinforces constriction of the blood vessels over the skin and counteracts the sympathetic nerve block. In addition, the patient is kept on the same addicting medications such as addicting tranquilizers or strong narcotics which in and of themselves perpetuate the anxiety and pain.

During the sympathetic blocks, the patient is not treated on antidepressants. As a matter of fact, it is shocking to see how rarely the patients with RSD are treated with antidepressants on a long term basis. Antidepressants are not given to RSD patients because they are depressed. Somewhere around one- fourth of the RSD patients do not suffer from any form of depression. However, they need antidepressants, too.

Antidepressants are not given for depression, but because antidepressants are the treatment of choice for chronic pain. In this regard the principle of the use of antidepressants is similar to the principle to the use of aspirin for heart attack or stroke. Aspirin is supposed to be an arthritis medication, but additionally it is one of the most effective modes of treatment for heart attack or stroke. Improper medicine may be capable of exerting more than one therapeutic role in the body.

No single RSD treatment can be written off as a failure unless other modes of treatment are simultaneously and properly applied. The patient should be on antidepressant, proper exercise and physical therapy without application of ice, the patient could be on plenty of pain medications that are less likely to be addictive (such as Talacen, Nubain, Stadol, or Ultram), and the patient should definitely be taken off addictive benzodiazepams. The addictive narcotics and benzodiazepams cause a rebound phenomenon (withdrawal pain). The best example of this rebound phenomenon is that the patient may have a focalized regional CRPS (complex regional pain syndrome of RSD) involving the right foot and yet every four hours after withdrawal from the intake of Percocet or Lortab may develop the severe headache, neck pain, and pain in every part of the body due to withdrawal effect of the addicting pain medications. The patient develops pain all over the body because of withdrawal regardless of how many sympathetic

nerve blocks the patient is having. The withdrawal from addicting benzodiazepams (Valium, Halcion, Ativan, Xanax, Tranzene, Librium, etc) results in generalized muscle spasm and low threshold for pain as well as moderate depression.

The same patient after undergoing a few nerve blocks is then exposed to treatments such as spinal stimulator or infusion pump. We have already discussed the futility of the use of the spinal stimulator in RSD Puzzle #23.

However, even a treatment as powerful as infusion pump is apt to fail if the patient is already loaded with intake of strong narcotics.

In conclusion, regardless of how extensively and repeatedly the patient has had different independent modalities of RSD treatment, the patient should be started from scratch with multiple treatments of antidepressant as an analgesic for chronic pain, effective pain control with the addition of non-addicting strong narcotic medications, muscle relaxants especially in the form of Baclofen (Lioresal), and nerve blocks.

The nerve blocks should not be just simply limited to a few sympathetic ganglion blocks or Bier block, but they should also include epidural and paravertebral nerve blocks.

The epidural and paravertebral nerve blocks are quite effective as a maintenance form of nerve block. They block not only the somatic nerve, but also the sympathetic nerves as well.

H. Hooshmand, M.D.

RSD PUZZLE #28
"THERE ARE CONTRADICTIONS IN REGARD TO THE USE OF NARCOTICS"

This is an excellent question. First of all no RSD patient will have a day of rest or any improvement unless the pain is effectively suppressed with the help of strong pain medications, nerve blocks, and large enough doses of antidepressants.

On the other hand, addiction is a handicap in long term treatment of RSD because addiction in itself aggravates RSD, causes stress and alarm in the sympathetic system resulting in more severe sympathetic dysfunction, and results in perpetual presence of rebound (withdrawal) pain.

To be able to understand the principles of narcotic use as well as the reason for non-addicting nature of Morphine pump, one has to understand the principles of addiction. The following may be helpful.

THE FOUR PRINCIPLES OF ADDICTION:

1. COMPETITION: Competition refers to the fact that if the brain is naturally providing chemicals to relieve pain, or anxiety, or to provide good rest and sleep, the extraneous intake of similar chemicals in the form of medications will compete with the brain's own natural chemicals (such as endorphines and endo BZs). If the patient takes Morphine by mouth, through skin patch, or by injection at the usual doses of 60-100mg a day, then the brain will cease the formation of the natural endorphines. This is only to protect the human body so that the combination of natural endorphines and the strong narcotics does not cause deep coma, arrest of breathing, and death.

On the other hand, if the pain medication is taken that does not compete with endorphines and is not complimentary or similar to the endorphines, then the body will continue forming endorphines without disruption.

In the first case when the brain does not form its own endorphines, 4-6 hours after the intake of Morphine the body is left with no protective effect of pain medication. As a result, the withdrawal (rebound) pain develops.

In the second case, when the brain does not stop the formation of endorphines there is no withdrawal effect and no rebound pain.

The patient to begin with may have taken the Morphine for an acute condition such as a recent trauma, heart attack, or surgical procedure. If after the original trauma has cleared up, the dosage of Morphine is continued, then the patient will have a perpetual pain due to the withdrawal effect of the strong addicting narcotics. What makes the narcotic addicting is the fact that it competes and forces the arrest of secretion of the body's natural endorphines.

2. REBOUND PHENOMENON: The rebound phenomenon has been discussed in detail in principle #1.

The rebound (withdrawal) pain is quite different than the original pain of the disease that requires pain medication. The disease may be nothing but a fracture of the ankle. The pain in this situation is quite excruciating and limited to the ankle. On the other hand, the rebound (withdrawal) phenomenon due to the use of strong addicting narcotics causes pain all over the body in the form of headache, neck pain, low back pain, pain in every bone and every extremity. It is a non-specific continuous pain and has nothing to do with the pain due to the original focalized injury. Long after the disease has been fixed and cured, the patient will continue to have rebound (withdrawal) pain which will continue indefinitely.

3. TOLERANCE: This refers to the fact that as the rebound pain becomes more disabling and severe due to long standing use of addicting narcotics, the brain learns to tolerate larger doses of narcotics and to control the vicious circle of the rebound pain adding to the original pain. As a result, the patient demands more and more of the pain medication, and even after taking large doses of pain medications, still has very poor and irregular sleep during the night.

4. INCREASING INTAKE OF THE MEDICATIONS DUE TO THE TOLERANCE: It is true that any pain medication or tranquilizer even though non-addicting may be abused. This abuse is usually in a small number of patients who try to accelerate the frequency and dosage of the medication to get pleasure out of it.

However, in the case of abuse of non-addicting narcotics or BZs sudden discontinuation of the non-addicting medications will not cause the severe withdrawal and the stressful symptoms and signs of the rebound. These consist of excessive sweating, rapid heartbeat, rise in the blood pressure, tremor, and severe anxiety and practically psychotic reaction. Such a withdrawal phenomenon is limited to the withdrawal from addicting medications.

In the case of application of Morphine Pump, none of the above four principles of addiction are involved. The dosage of medication given to the patient is so small (a dosage of 1 day is given in prescription form in 1 month). That does not compete and stop the formation of endorphines. There is no withdrawal phenomenon (no rebound symptoms) because of the strict drip irrigation form of application of medication. There is no tolerance because of the fact that the patient has no control over the intake of medication, and the body is not being exposed to the rebound phenomenon.

The problem of addiction as outlined above directly affects the sympathetic nervous system by causing stress (with resultant severe pain, sweating, tremor, etc.) and aggravating RSD.

The Morphine Pump is not given to facilitate the intake of an addicting medication. It is given to prevent the above mentioned four principles of addiction and tends not to cause problems of withdrawal and tolerance. The dosage of medicine is too small to stop the formation of other biogenic ammens, endorphines, endo BZs, and other hormones in the brain.

On the other hand, the use of pain medication in long term pain narcotic patients should be limited to the non-addicting type of pain medications (such as Stadol or Ultram) or at least to the less addicting pain medications such as Nubane and Talacen. In the meantime, it should be understood that one cannot keep the patient on any narcotic for ever. Simultaneously, the antidepressant medications should be increased until the analgesic effect of the antidepressant makes it unnecessary for the patient to take long term doses of strong medications.

The stress of severe pain and the stress of the rebound pain are two major aggravators of RSD. With the above mentioned methods, one should eliminate such distressors.

Stadol and Ultram as well as to some extent Nubain are Morphine agonists antagonists meaning that they enhance the pain relief and drowsiness in the presence of endorphines or other non-narcotics. Yet, when they are given along with other narcotics which are addicting, the combination is so strong that it causes nausea, vomiting, excessive drowsiness, and even breathing problems.

It is foolish to ask the patient to simply "live with the pain" and not to take strong pain medications. However, the most effective strong pain medication is proper antidepressants.

H. Hooshmand, M.D.

RSD PUZZLE #29

"THEY TRIED THE MORPHINE PUMP ON ME AND IT MADE ME VERY SICK, I WAS ALLERGIC TO IT."

It is not at all uncommon for the patient to be tried on infusion pump while she is not properly tapered off other narcotics. As a result, the combination makes the patient quite sick.

In some patients the patient is intolerant of even a very small dosage of Morphine or Dilaulid in the spinal fluid. I such patients an addition of a very small amount of Clonidine in the pump (small doses of 75-125 microgram) help enhance the pain control. As a result, the dosage of the Morphine or Dilaulid can be reduced to quite a small amount and yet effective relief of pain can be achieved without nausea.

In addition, the infusion pump should not be tried unless the patient is already detoxified for at least 1 week from other forms of narcotics.

The infusion pump usually requires small enough dosage of Morphine or Dilaulid so that there would be no suppression of secretion of cerebral endorphines. This is usually between 2 to 8 (up to 9) milligrams per kilogram Morphine. On the other hand if one goes above the 9mg dosage, the pain recurs due to the suppression of the endorphine function. This is a frequent cause of failure of infusion pumps.

In our series of over 80 patients with infusion pumps, there has been 82% success in patients who have otherwise been totally 100% failure in control of their pain. The 18% failure has been due to either intolerance to the medication, or infection. Infection alone is not a contraindication because the pump can be removed and replaced later on. In two patients among the failure group, the body rejected the foreign body of the pump by either forming a lot of scar and granulation at the tip of the catheter or around the pump itself. This is a rare complication.

H. Hooshmand, M.D.

RSD PUZZLE #30

"YOU DID HAVE RSD BEFORE, YOU DON'T HAVE IT NOW.
THE SYMPATHETIC NERVE BLOCKS HELPED, NOW YOU DON'T HAVE IT"

The early stages of RSD sympathetic nerve blocks can be both diagnostic and therapeutic. The temporary elimination of pain following the sympathetic ganglion or regional block is the best diagnostic proof that the patient suffers from sympathetically maintained pain (SMP). However, as the disease becomes chronic and complicated with improper treatment, such as application of ice, the long term immobilization with braces and wheelchair, and unnecessary surgery such as exploration for carpal tunnel syndrome, then the pain is not a simple SMP. Instead, damage to somatic nerves secondary to the vasoconstriction and additional surgical trauma changes to sympathetically independent pain (SIP) which does not respond 100% to sympathetic nerve blocks.

The end result is that in the chronic stage of this disease the patient develops both sympathetic and somatic pain. By then, because the sympathetic block does not control the pain, the patient is told that she does not have RSD any more. Abruptly the treatments are discontinued and the patient is abandoned. This has become a serious problem.

By the time the above phenomenon develops, the patient has suffered from RSD for any where from a year and a half to four to five years. The treatments have complicated the disease, yet the patient is being punished for not responding to sympathetic nerve block.

Another problem is that in the best of hands the sympathetic nerve blocks are successful in only about 75-80% of the patients. This is because of the phenomenon mentioned above as well as the sympathetic anatomical structure being so variable from person to person that there is no guarantee that the nerve block can be done. The guaranteed forms of nerve block are Bier block and IV drip nerve block.

Finally, as the disease becomes chronic if the patient has been left totally untreated then one sees the classical four stages of RSD. On the other hand, any treatment for RSD changes the clinical picture of RSD and as the condition becomes atypical making the diagnosis that more difficult. There is a definite need in orienting and educating the care giver regarding the fact that treatment may change the clinical picture of RSD.

By the time the patient has suffered from RSD for quite a few years, the infusion pump may be the only form of treatment to control the patient's pain.

H. Hooshmand, M.D.

RSD PUZZLE #31
"WE HAVE GIVEN YOU ALL THE NERVE BLOCKS WE COULD"

Nerve blocks are done in the following forms:

1. Sympathetic ganglion blocks.

2. Regional blocks (Bier block, bretylium block, etc).

3. Paravertebral and epidural blocks.

4. Regional somatic nerve blocks.

5. Systemic sympathetic nerve blocks such as:

5a. Treatment with alpha I (Clonidine, Dibenzyline, Hytrin) or alpha II (Yohimbin).

5b. IV Phentholamine

Two principles should be kept in mind. Sympathetic nerve blocks- without other forms of treatment for RSD-are apt to fail. The sympathetic nerve blocks should be combined with physical therapy, moist heat, exercise, and the use of newer (Trazodone or SSRI) antidepressants as well as detoxification.

A lot of people mistake diagnostic blocks for therapeutic blocks. A simple diagnostic block with the use of local anesthetic doesn't last more than four hours. This is not necessarily a treatment but simply a confirmation of SMP. The therapeutic blocks not only contain the local anesthetic but also should contain some other chemicals to help the blocks last longer (chemicals such as Celestone or Depo-Medrol®).

To simply treat the patient with a painful diagnostic or therapeutic sympathetic block, one can’t expect the patient to improve without other forms of treatment is unrealistic and is apt to fail. Every chronic pain patient (such as RSD) should be first treated with antidepressants as the ideal analgesic.

The second principle to keep in mind is that as the condition becomes chronic and a combination of SMP and SIP (sympathetically independent pain) develops, other forms of blocks especially epidural nerve blocks, paravertebral nerve blocks, trigger point injections, and regional somatic nerve blocks become excellent additional tools for treatment of RSD. These blocks should not be given into the areas of severely painful scars that initiated RSD. They should be given proximal to the area of nerve and scar that is tender to touch.

Injection of the area of the scarred nerve (due to scar of original trauma or the scar of surgery) is extremely painful, and flares-up the RSD. This flare-up is not simply in the form of severe incapacitating pain but it is accompanied by inflammation, development of skin rash, swelling of the tissues in the area of traumatic injection. It simply aggravates and traumatizes the nerve causing the scar which started the RSD to begin with. The best way to achieve this nerve block is to block the proximal part of the nerve immediately above the area of the scar.

The treatment for RSD in early stages should start with physiotherapy, antidepressants, and sympathetic nerve block and should be reinforced and followed by topical or regional somatic block, especially epidural and nerve canal blocks such as spinal canal or myelin canal nerve blocks. The role of the treatment should be to achieve control of the pain with repeated alternating sympathetic and somatic blocks. This buys enough time to control the condition with the help of more long term and permanent control of pain such as the use of antidepressants, anticonvulsants (Tegretol or Neurontin), and physiotherapy, mobilization, and application of heat.

H. Hooshmand, M.D.

RSD PUZZLE #32
"My RSD Started From An Injury To The Hand And After Carpal Tunnel Surgery. Why Is It I Can't Remember Anything?"

RSD is not just the disease of peripheral nerves. The condition is not a simple burning or stabbing pain. Besides the pain the patient also has other manifestations such as movement disorder, constriction of blood vessels in the extremity, swelling of the soft tissues (mistaken for "fibromyalgia"), and disturbance of the limbic system (the temporal-frontal lobe regions). The limbic system is the primitive cortical system in the cerebral hemispheres. Disturbance of this system results in depression, poor judgment, poor memory, fatigue, irritability and agitation. It also results in insomnia.

The memory loss is not simply because of depression. As a matter of fact, not all the RSD patients are depressed. Somewhere around one-forth of the RSD patients have a normal depression test on psychological examination.

The number one factor in memory loss among RSD patients is the pain causing disruption of normal natural REM sleep. This results in the patient being fatigued, irritable, and edgy and having poor memory the next day. Sleep is probably the most important recuperative state of our life. We need to sleep to give the brain a chance to put its house in order after a stressful day. Especially during REM sleep the brain stem and the limbic system undergo recovery and recuperation.

The same patients who suffer from memory loss also have a tendency to suffer from chronic fatigue as well.

The effective and successful treatment for memory loss in RSD are as follows:

1. Antidepressants that provide natural REM sleep. At the top the list is Trazodone.

2. Pain relief. Like any other RSD complication, the patient is not going to get better unless proper relief is provided.

3. Nerve blocks. The anesthesiologists who have extensive experience with nerve blocks, and the patients who have undergone the blocks, describe the phenomenon that the first successful nerve block that the patient receives is immediately followed by a deep sleep which helps the patient feel much improved.

4. It is imperative to discontinue existing BZ'S (especially Ativan, Xanax and Ambien) to give the patient a chance to have natural, normal REM sleep. Realizing that Ambien does not cause significant rebound (withdrawal), still it reduces the REM sleep.

Another medication that seriously disturbs the memory function and the general state of RSD patients is barbiturates in the pain medications such as Fiorinal, etc. The barbiturates which for decades have been used as a sleeping pill are more anesthetic than sleeping pill. They provide an unnatural sleep and deprive the patient of REM sleep.

Alcohol is another drug that cannot be used in any amount in any RSD patient because of not only the caustic (damaging) effect of alcohol on the nerves but also because of depriving the patient of REM sleep.

In more severe cases, to counteract fatigue and memory disturbance, the patient may require Paxil, Zoloft, or Prozac in the morning and Trazodone and night. The same outline antidepressants are also effective in control of the chronic pain.

H. Hooshmand, M.D.

RSD PUZZLE #33

"I Can't Antidepressant. Elavil Made Me Sad And Exhausted Caused Dizziness And Fast Heart Beat".

As we have repeatedly discusses, the treatment of choice for chronic pain is antidepressant. This is independent of any depression. Antidepressant is the most effective medication for chronic pain.

However, there are actually different types of antidepressants.

1. The tricyclic antidepressants have been around for over 25 years, and there has been extensive medical literature regarding the efficacy of Elavil, Tofranil, Norpramine, etc., for pain control. However, these anticonvulsants have been recently labeled as "dirty" because they have multiple side effects. These consist of obesity, aggravation of fatigue, tendency for inactivity, low blood pressure, and excessive drowsiness. Obviously, the RSD patient cannot afford such side effects. However, until recently there was not much of an alternative.

2. Twelve years ago a non-tricyclic antidepressant became available, called Trazodone. This antidepressant has the side effects of pelvic genitalia congestion so it improved erection in men, Twelve years of use has showed no more than 3 or 4 serious erection complications.

On the other hand, Trazodone in judicious careful doses provides an excellent REM sleep. The best time to take the medication is at bedtime and with the help of adjustment of the dosage and blood level of Trazodone an accurate dosage can be figured for each patient. It is an excellent analgesic. However, it does have a tendency to mildly increase the appetite.

3. In the past 8 years, a new series of antidepressants called selective serotonin re-uptake inhibitors (SSRIs) have been available. The most commonly used of this group of medications that raise the concentration of serotonin in the central nervous system are Prozac, Paxil, and Zoloft. This group of antidepressants do not cause the serious complications of obesity, hypotension (low blood pressure), and fatigue. If anything, they are quite effective in counteracting fatigue.

Of the three, Prozac possesses the most analgesic effect. The Zoloft, on the other hand, has very little analgesic effect. Paxil, which is not as effective pain medication as Prozac, is better tolerated in the long run and causes fewer side effects. It is the treatment of choice for chronic fatigue.

Prozac has been the most widely used SSRI, and when used on a long term basis can cause significant complications. These consist of bouts of agitation, lack of sexual desire, and weight loss. The weight loss actually in many patients is a plus as such for some patients who have weight problems but in occasional patients the Prozac may be abused because of obsessive tendency for the patient to lose weight.

The main problem with Prozac is the problem with drug interaction. Paxil and Prozac should not be used along with Valium, Xanax, alcohol, Risperdal, or tricyclic antidepressants. Addition of Paxil or Prozac to tricyclic (such as Elavil or Tofranil, raises the tricyclic drug level by 400% and can cause serious complications).

Of the three SSRIs, Zoloft is the cleanest in regard to interaction with other medications but has the least analgesic value.

If the patient has any tendency for seizure disorder, then Prozac, Wellbutrin, or Clozaril should not be used:

1. Prozac is reported in PDR (Physicians' Desk Reference), on page 946, to cause convulsion.

2. Wellbutrin is reported in PDR, on page 826, to cause seizures, myoclonus seizures, and abnormal EEG.

3. Clozaril is reported in PDR, on page 2152, 2nd column under "Central Nervous System", to cause myoclonus, status epilepticus (continous seizures), and abnormal EEG.

At times a combination of two antidepressants may become necessary. One example is the patient who has such severe pain and insomnia that Trazodone, Prozac, or Paxil alone do not control her pain. In such patients, a combination of a tricyclic such as Norpramine given at bedtime in small doses, and Prozac given in the morning in small doses can provide good pain control and a good night's sleep. This is because of the fact that Prozac inhibits the enzyme (P450) that breaks down the tricyclic antidepressant. As a result, giving 20mg Prozac in the morning and a minimum dose of Norpramine (25mg) at bedtime enhances the function of the Norpramine with good antidepressant and pain control affect without serious side effects without side effects of the large dose of the drug.

As is the case with any medication that influences the function of the brain, the patient should abstain from any alcohol intake while he is on antidepressant medication.

Finally, antidepressants are not addicting. The antidepressants provide three beneficial therapeutic effects.

1. Pain control.

2. Counteracting depression.

3. Helping the patient to be detoxified from addicting drugs.

H.Hooshmand, M.D.

RSD PUZZLE #34
"CAN RSD INVOLVE INTERNAL ORGANS?"

RSD invariably involves the internal organs. Usually the skin surface is cold at the expense of increased circulation to the internal organs. This increased circulation can cause osteoporosis, fractures of bone, abdominal cramps and diarrhea, disturbance of absorption of foods with resultant weight loss, water retention with aggravation of premenstrual headaches and depression, persistent nausea and vomiting, as well as severe vascular headaches mistaken for "cluster headache".

In addition, the sympathetic dystrophy can cause the complication of intractable hypertension which responds best to alpha I blockers (Dibenzyline, Hytrin, or Clonodine). The RSD can cause attacks of irregular or fast heart beat, chest pain, coronary artery spasm (angina), as well as disturbance of function of other internal organs. A few examples are frequency and urgency of urination, respiratory disturbance such as dyspnea and apneic attacks, and attacks of severe abdominal pain.

Laparoscopy may reveal congestion and inflammation of the ovaries, uterus or small bowel.

Attacks of fluctuating blood pressure may also be accompanied by constriction of the blood vessels to the kidney resulting in periodic bleeding in the urine as well.

The internal organs complication may become aggravated by traumatic effect of sympathetic nerve blocks. One such complication is accidental trauma to the kidney with resultant hematuria (blood in urine) and aggravation of hypertension.

Nerve blocks and more importantly physical therapy help improve the skin circulation and reduce the deep circulation calming down the inflammatory affect of RSD over the internal organs. As mentioned above, alpha I blockers are quite affective in treatment of this condition.

Attacks of swelling of the internal organs complicated by intermittent constriction of the blood vessels to different organs can result in chest pain, attacks of sharp central pain (stabbing severe pain in the chest or abdomen), and changes in voice (suddenly developing a temporary "chipmunk" type of voice change). The sharp, stabbing, central pain can be helped with treatment with medications such as anticonvulsant (Tegretol or Neurontin).

The use of catheters adjacent to the sympathetic chain such as in the lumbar sympathetic chain can help prevent repeated needle infection for sympathetic nerve blocks. However, because of the congestion of the internal organs the catheter may irritate the sympathetic nerve branches causing constriction of the blood vessels to the spinal cord with temporary paraplegia. As soon as the weakness of extremities develops, the catheter should be removed. Not heeding to this ominous sign can result in paralysis of the lower extremities and incontinence.

The same congestion of internal organs can also cause hypersensitivity to smell and aversion to taste of certain foods.

H. Hooshmand, M.D.

RSD PUZZLE #35
"MY RSD IS FOUR YEARS OLD. I HAVE RECENTLY STARTED HAVING FREQUENT INFECTIONS AND MY DOCTOR TELLS ME THAT MY IMMUNE SYSTEM IS NOT FUNCTIONING PROPERLY. CAN RSD CAUSE THIS PROBLEM?"

The sympathetic and parasympathetic systems in coordination provide the following functions:

1. Control of vital signs (blood pressure, pulse, and respiration).

2. Controlling and up regulating and down regulating the immune system.

3. Control the body temperature.

The over acting of the sympathetic system results in cold skin and hot deep structures of bone, muscle, and viscera. The parasympathetic system does the opposite.

Professor B. G. W. Arnason of University of Chicago has demonstrated the reciprocal feedback of the nervous and immune systems. As he puts it "if the nervous system talks to the immune system via the sympathetic nervous system and possibly via the parasympathetic nerves, it seems likely that the immune system talks back". He has proven that the sympathetic system increases the CD8 T-cells function and the parasympathetic system does the opposite. In acute stage, stimulation of the sympathetic system up regulates the CD8 (killer) T-cells and the parasympathetic system does the opposite.

In the first few months of abnormal sympathetic function (acute RSD during the first 6 months) the sympathetic and parasympathetic systems show a significant plasticity and can adjust their activities to preserve the immune system. However, after two to three years, (chronic RSD) this power of plasticity and ability to fluctuate the balance of the immune system disappears. As a result, the immune function is thrown off balance with resultant development of frequent infections, and in the long run, development of a tendency for cancer. Certain treatments influence the plasticity and balance of the two systems positively or negatively. The following are the treatments that deteriorate the immune function.

1. Unnecessary surgery. This is especially true in the case of RSD involving the hand or foot causing inflammation and swelling at the wrist or ankle mimicking the picture of carpal tunnel or tarsal tunnel syndrome. Surgery at the wrist or ankle in such patients aggravates the condition tremendously and weakens the immune system. Sticking needles and giving injections in the swollen areas of carpal tunnel or tarsal tunnel also accelerates the deterioration of the immune system and should be avoided.

2. Intake of drugs such as alcohol, addicting narcotics, and addicting BZs.

3. A continuous distress due to the legal entanglements related to the trauma that initially caused RSD.

4. Inactivity and lack of exercise.

5. Certain, so-called foods such as hot dogs. It has been shown that children who eat more than four hot dogs a week have five times higher incidence of suffering from cancerous brain tumor than the children who do not eat hot dogs.

6. Use of ice on the extremity accelerates the constriction of the blood vessels, and aggravates the RSD hastening the development of the disturbance of the immune system.

Obviously, any measure taken in the opposite direction will prevent deterioration of the function of the immune system.

Treatments in the form of nerve blocks, antidepressants that provide natural REM sleep (such as Trazodone), and physiotherapy along with application of moist heat prevent the disturbance of the immune system.

Once the patient develops the picture of disturbance of the immune system, treatment with medications such as I.V. Immunoglobulin or ACTH may be helpful. Even though corticosteroids such as Prednisone or Decadron slow down the abnormal function of the immune system, they have a tendency to result in atrophy of the adrenal glands. The atrophy of the adrenal glands aggravates the function of the immune system.

ACTH in judicious doses does not cause atrophy of the adrenal glands and have beneficial effects both on the immune system and on reducing the swelling of the soft tissues secondary to RSD.

One of the complications of RSD, especially after the immune system is disturbed, is development of frequent attacks of neurodermatitis, skin rash, and break down of the skin.

Medications such as Zonalon (topical Sinequan) TAC (Tetracaine-adrenaline-cocaine) are helpful in counteracting the skin break down in such patients. Another similar topical lotion called LAT (Lidocaine-adrenaline-tetracaine) is similarly effective. The use of magnesium sulfate and hot water in some cases in more acute stages, the use of Capsaicin may be helpful. Clonidine Patch at any stage of RSD is a very effective, helpful treatment, not only for the skin dysfunction, but also as a sympathetic nerve block agent.

H. Hooshmand, M.D.

RSD PUZZLE #36

"A FEW YEARS AGO WHEN I STARTED SUFFERING FROM RSD, I KEPT LOSING WEIGHT. NOW AFTER 5 YEARS I HAVE A PROBLEM WITH GAINING WEIGHT REGARDLESS OF HOW MUCH I DIET OR EXERCISE."

As mentioned in the RSD Puzzle regarding the immune system disturbance, in acute stages of RSD there is plasticity and power of adjustment of up and down regulation and attempt at correction of the sympathetic dysfunction by the sympathetic nervous system. However, after a few years (usually 2-3 years), the ability of the sympathetic system to be capable of fluctuation and adjustment (plasticity) is lost. As a result, an exhaustion of the immune system as well as exhaustion of other functions of the sympathetic system develops. In the acute stage, the patient usually loses weight due to the increased body metabolism and caloric consumption. In the chronic stage, as the immune system becomes defective, the patient also develops a tendency to gain weight.

Other therapy factors also play a role. Long term treatment with tricyclic antidepressants (especially Elavil or Tofranil-Amitriptyline or Imipramine) results in obesity, aggravation of chronic fatigue, and slowing down of the body metabolism. The aggravation of chronic fatigue and insomnia result in generalized weakness and secondary increase in oral intake of food.

Switching the treatment from the tricyclic antidepressants to SSRI antidepressants, especially Prozac or Paxil counteracts the problem of weight gain. Paxil has the best potential of counteracting the fatigue.

Following the 4-F diet (fresh fruit, fresh vegetable, fish, and fowl) never skipping any of the four meals a day (breakfast, lunch, supper, and snack at bedtime), exercising and making certain to avoid any butter, margarine, mayonnaise, vegetable oil, and red meat of any kind, are very effective in counteracting the fatigue and obesity

H. Hooshmand, M.D.

RSD PUZZLE #37

"EVER SINCE I WAS AFFECTED BY RSD I HAVE SUFFERED FROM IRREGULAR, PAINFUL, AND HEAVY MENSTRUAL PERIODS. CAN THIS BE RELATED TO RSD?"

There are two main factors that RSD results in painful and irregular and heavy menstrual periods.

1. The visceral inflammation which is typical of RSD. In RSD the surface skin is cold and as the skin becomes deprived of proper blood flow, the blood flowing deep structures (bone, muscle, guts, and pelvis) increases with resultant inflammation of the deep structures. This is manifested in symptoms such as abdominal pains, pelvic inflammatory disease (PID), irregular, heavy, and painful menstruation, and attacks of persistent coughing. In the later stages of RSD, spontaneous attacks of abdominal pain, diarrhea, and weight loss may develop as well.

2. Sympathetic nerve blocks, especially over the lumbar sympathetic chain, or over the coeliac ganglion are quite effective to correct these problems. The alpha II blocker, Yohimex (Yohimbine) which is used for enhancement of erection, functions by reversing the cold and pale skin circulation, and as a result, the visceral deep structure inflammation is alleviated. Other sympathetic blocks such as Clonidine skin patch and oral Ketamine may be quite helpful. The attacks of diarrhea may be relieved with the help of the use of elixir of paregoric and tincture of belladonna. The later complication of attacks of diarrhea and weight loss is especially seen among patients who have been the victim of lightning strike.

H. Hooshmand, M.D.

RSD PUZZLE # 38
Use and Abuse of Butorphanol (STADOL)

According to the Physician Desk Reference, Butorphanol Tartrate is a synthetic opioid mixed agonist/antagonist analgesic. Of the three endorphin receptors in the brain (mu, theta, and kappa receptors), Stadol has more affinity to mu and theta receptors. In therapeutic doses, the kappa receptor which is a larger sized receptor is not filled with the ligand Stadol, and as a result, the endorphin fills the kappa receptor.

This explains the fact that in therapeutic doses, the Stadol does not stop the formation of the endorphin in the brain and in the spinal cord, and as a result, the patient does not develop withdrawal (rebound) phenomenon which is the main problem with opioid agonists such as Morphine, etc. However, if the dosage is increased above the therapeutic dose, then the Stadol floods all the receptors, and the brain and the spinal cord stop forming natural endorphin. This explains the agonist as well as antagonist effect of Stadol in regard to pain relief.

Opioid antagonists (Stadol, Nubain, or Buprenex) in therapeutic dosage do not cause any serious problem. The problem comes when an opioid agonists such as morphine, methadone, etc... is added to it, then the patient gets dry mouth, has drowsiness, and has nausea. In addition, in high doses, the Stadol is as habit forming as morphine family of drugs. This causes Stadol addiction and abuse (dependence). In this regard, the opioid antagonist acts similar to Antebuse. They are supposed to make the patient very sick if the patient takes opioid agonists along with it.

The above neuropharmacological principle explains the complexity of the agonist/antagonist analgesics.

H. Hooshmand, M.D.

RSD PUZZLE #39

"YOUR RSD IS IN REMISSION. IF YOU HAVE SHAKING AND MUSCLE SPASMS, THIS HAS NOTHING TO DO WITH RSD. YOUR SOFT TISSUE SWELLING IS DUE TO MYOFASCIAL PAIN. YOU DON'T NEED ANY MORE TREATMENT FOR RSD."

The misconception of RSD being considered as nothing but a burning pain and the lack of understanding that RSD manifests itself in other symptoms such as movement disorder, tremor, spasm, inflammation, skin rash, insomnia, depression, and agitation has resulted in the idea of "RSD in remission".

It is true that even after RSD is totally under control, one can detect abnormalities on tests such as thermography or bone scan which persist long after the successful treatment. This does not mean that the RSD is in remission. Most of the successfully treated RSD patients don't go into remission, but get over the disease and go back to their normal life. Not every one of them has future flare-ups. So, they are not in a true "remission" but they have been successfully treated.

On the other hand, calling a patient in remission just because the pain has been successfully controlled while the patient has movement disorder, insomnia, agitation, depression, and swelling or skin rash, is abuse of the term "remission". It sounds more like "a touch of pregnancy".

The treatment should be aimed at counteracting other manifestations of RSD such as movement disorder (with the help of muscle relaxants especially Baclofen), insomnia, agitation, and depression (with the help of newer antidepressants), and inflammation and swelling (as outlined in RSD Puzzle #33).

H. Hooshmand, M.D.

RSD PUZZLE #40

"YOUR SWELLING OF THE JOINTS IS NOTHING BUT ARTHRITIS, YOUR SKIN SWELLING IS NOTHING BUT FIBROMYALGIA, AND YOUR SKIN RASH IS FROM SKIN DISEASE AND HAS NOTHING TO DO WITH RSD."

Of these four principle manifestations of RSD (pain, movement disorder, inflammation, and insomnia) the inflammation manifests itself in several different forms. This may be in the form of simple swelling of the extremities, joint pain, skin rash, blotching or cyanosis, trophic changes such as hair loss or fingernails degeneration, black and blue spots without any trauma to the skin, bleeding under the skin, and persistent itching.

The inflammatory aspect of the RSD is just as disabling as the pain or movement disorder. It requires the same assertive aggressive treatment as well. The treatment can be achieved by the following measures:

1. Anti-inflammatory medications. It is best to use anti-inflammatory medications that are less likely to enhance bleeding. Medications such as Oruvail, Lodine, and similar other newer forms of anti-inflammatory medications are quite helpful.

2. Physical therapy in the form of a combination of moist heat and epsom salt, the use of gloves and stockings to counteract progressive accumulation of edema or even elephantiasis, as well as massage and exercise are quite effective.

3. Brief and temporary use of corticosteroids both in the form of proximal somatic nerve blocks and by mouth may be effective. In this regard, Prednisone has a tendency to increase water retention. On the other hand, on the maximal 2-3 weeks duration, treatment with Dextromethasone (Decadron) can be helpful.

4. Treatment with ACTH which is not a corticosteroid but a polypeptide hormone extracted from the hypothalamic region of the brain is quite effective and has far less side effects than corticosteroids. This should be given in a small dose of 40 units ACTH IM 3 times a week accompanied by the intake of antacid medications such as Zantac or Axid. It is an effective form of treatment for inflammatory manifestations of RSD.

5. Topical cream and medications can be quite affective.

One form of the topical cream that has been recently available and very effective is called Zonalon Cream which is a cream containing the antidepressant Sinequan. This is a very effective treatment in both reducing the inflammation and controlling the pain. Other topical corticosteroid creams can also be helpful.

6. For other more disabling manifestations of inflammation such as vascular headaches, persistent diarrhea, nausea and vomiting, and abdominal pain, other forms of treatment may be affective such as treatment with alpha blockers, elixir of paregoric, tincture of belladonna, and potable aloe liquid

H. Hooshmand, M.D.

RSD PUZZLE #41
THEY HAVE TRIED EVERY KIND OF NERVE BLOCK, NARCOTIC, ANTIDEPRESSANT WITH NO SUCCESS. WHAT SHOULD BE DONE NOW?

Dear Dr. Hooshmand:

My name is Brenda and I am writing you about my RSD.

The disease started after a right ankle injury to multiple ligaments and nerve injury on March 10, 1994. Since then I have had physical therapy, and all sorts of blocks: lumbar sympathetic blocks, epidurals, BIER blocks, Popliteal blocks, epidural Duramorph (single shot), and IV Lidocaine. These blocks last practically 4 hours and then I 'm back where I started.

I have been treated with Tricyclic antidepressants, Hytrin, Mexetill, Neurontin, Motrin, narcotic, etc., with no success.

My RSD has now spread from my right hip and buttock down to the right leg and foot. I have tendency for developing attacks of cellulitis around the areas of epidural shots. My doctor says that my immune system is shot.

Sincerely,

Brenda

Dear Brenda:

Your RSD has been diagnosed relatively early and it has been treated with the best of intentions with multiple blocks. However, by virtue of the fact the blocks last no more than 4 hours, most likely the main substance used has been local anesthetic such as Lidocaine. You need to have other chemicals such as Celestone or Depo-Medrol® added to the lidocaine.

The main problems that you are facing at this stage are the spread of the disease and the strong tendency for inflammation which is part and parcel of RSD.

The following suggestions may help bring the condition under better control:

1. Avoidance of ice during physical therapy.

2. Intermittent exercise of frequent nature, independent of the physical therapy. Learn from your heart. It beats 60 times a minute and never takes a vacation for 90 years. That's because half a second it works and half a second it rests. So, from morning to night, it is resting and exercising. Don't rely on the physical therapist to do the exercise for you.

3. For the problem of the inflammation, treatment with ACTH which is not a corticosteroid but stimulates the formation of endorphins, may be quite helpful to just calm down your inflammatory reaction. This can be done in conservative doses such as 40 units IM 3 times a week to be taken along with one Zantac a day.

4. Frequent Epsom salt and hot water baths help contain the inflammatory reaction.

5. Discontinuation of the addicting narcotics and replacing them with non-addicting pain medication such as Stadol, Nubain, or less addicting medications such as Talacin. It is true that Stadol has been abused by some patients but that is not the same addiction, and the use of the above mentioned Morphine antagonists helps the body build up its own endorphines and endo-BZ's.

6. After the above is done, you will notice the disease subsiding and becoming more focalized to the lower extremities. Then repetitive nerve blocks through the insertion will help a lot. These nerve blocks should have more than just local anesthetic in it.

7. I have noticed one major omission of medication in your treatment of RSD. You have not been on NEWER ANTIDEPRESSANTS. Obviously, the tricyclic antidepressants have not done anything for you. You need to be on medications such as Trazodone 150 to 300 mg at bedtime, or Fluoxetine 20 to 40 mg in the mornings. These antidepressants are analgesic of choice for chronic pain. Unless you take such antidepressants on a long-term basis, your pain will never get better. In RSD instead of no pain, no gain, the formula is no pain is all gain.

With best wishes,

H. Hooshmand, M.D.