

WHAT IS RSD?

WHAT IS SYMPATHETIC ANYWAY?

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REFLEX SYMPATHETIC DYSTROPHY (RSD)

To define Reflex Sympathetic Dystrophy (RSD) one should understand the terminology of sympathetic and parasympathetic nervous systems. There are two different types of nervous systems controlling the body. One is the so-called "somatic" nervous system which has clear-cut anatomical structures and is controlled by the cerebral cortex (cerebral hemispheres) in a relatively volitional manner. We see, hear, touch, taste or smell something, and volitionally and knowingly respond positively or negatively towards the stimulus. We can influence our response through the judgment of higher centers of the brain (cerebral hemispheres). This is the "somatic" system which is strongly influenced and controlled by our conscious mind.

The other system is the so-called autonomic system. The name implies that it is autonomous (kind of having a mind of its own). It is almost autonomous but it can be influenced to a certain extent by conscious brain as well. This system is quite primitive and old (from evolutionary standpoint). Even a worm has an autonomous nervous system. If one does an experiment by warming up one end of a fish tank water and cooling the other end of the; the worm will go from one extreme to the other and eventually will reside halfway between the two extremes of temperature in the mid portion of the fish tank. The worm does not need a brain to decide where to retire. The autonomic system does the job for it.

The autonomic nervous system concerns itself with preservation and protection of the "Internal Environment". For example, in warm blooded animals the autonomic nervous system keeps the temperature inside the body around 99° Fahrenheit (37 °C).

To protect the internal environment, the autonomic nervous system has two main components.

1. The sympathetic system.

2. The parasympathetic system.

The sympathetic system is a fight component of the "Fight and Flight" reflexes of the autonomic nervous system. On the fight end part of it the sympathetic system increases the internal temperature, raises the blood pressure, strengthens the protective function of the skin, makes the skin cold so that there would be no waste of temperature, makes the skin sweat excessively (so that there would be no extreme increase of the internal temperature); and increases muscle metabolism, bone circulation, circulation of the brain and guts. The end result is the animal is ready to fight. On the other hand, the parasympathetic system that is the balancer of the other end of the autonomic system (the flight system), drops the blood pressure, slows down the pulse, relaxes the muscles, and preserves energy by cutting down the calories burned in the body. As such, the parasympathetic system works the opposite of the sympathetic system. The two systems are totally independent and one cannot give a patient parasympathetic enhancing medication in the hope of cooling the sympathetic and vice-versa.

THEN WHAT IS RSD?

RSD is one form of disturbance of the function of the autonomic nervous system. Simply having a hyperactive sympathetic nervous system does not make RSD. The disturbance of the autonomic system comprises several diseases, some acquired, some genetic, some metabolic, some traumatic, etc.

Some examples of dysautonomias "disturbances of the autonomic nervous system" are attacks of hypotension (low blood pressure), congenital absence of sweating, and neuropathic pain syndrome.

The latter category of chronic neuropathic pain syndrome refers to the conditions that are not exactly necessarily reflex sympathetic dystrophy, but have an abnormal sympathetic component to them. They share sympathetically maintained pain (SMP) with RSD but they are not RSD.

Examples of such chronic neuropathic pain are postherpetic neuralgia (pain accompanying and following shingles), neuropathic diabetic neuropathy, acute neuropathic pain accompanying bee stings, snake bites or spider venom stings as

well as involvement of the sympathetic nerves due to the systemic AIDS infection.

The neuropathic pains are not at all synonymous with RSD. They do not even in any way resemble RSD. In some cases, however, they can end up with RSD.

The chronic neuropathic pain syndrome is far more common than the true clinical picture of RSD.

CAN RSD BE MORE CLEARLY DEFINED?

RSD is a definitive chronic pain syndrome called by several different names such as reflex sympathetic dysfunction, (stage I), reflex sympathetic dystrophy (stage II), reflex sympathetic atrophy (stage III), late stage RSD complicated by disturbance of immune system, suicidal and fetal tendency, cancer, heart attack or stroke (stage IV), complex regional pain syndrome (CRPS) which encompasses all different forms of RSD as well as the causalgic RSD, causalgia (burning, stabbing, constant pain, acting more like an epileptic seizure), shoulder-hand syndrome, sudeck's atrophy (circa 1900), traumatic vasospasm of Lehman (1934), mimocausalgia (1973), minor causalgia (1940), and half a dozen other names.

In medicine there is a trend. When a disease becomes confusing, the physicians become desperate and give it new names. Each of the above almost dozen names reflect some features of RSD.

Merskey and Bogduk in January of 1994 defined the syndrome as follows (IASP Press, Seattle classification of chronic pain 2nd edition)[1]: CRPS TYPE I is a syndrome that usually develops after an initiating noxious event, is not limited to the distribution of a single peripheral nerve, and is apparently disproportionate to the inciting event. It is associated at some point with evidence of edema, changes in skin blood-flow, abnormal sudomotor activity (sweating) in the region of the pain, or allodynia or hyperalgesia. They also clarify in the main features of CRPS (RSD). "The symptoms and signs may spread proximally or involve other extremities. Impairment of motor function is frequently seen". They clarify associated symptoms and signs and specify "atrophy of the skin, nails, and other tissues, alterations in hair growth, and loss of joint mobility may develop. Impairment of motor function can include weakness, tremor, and in rare instances, dystonia. Symptoms and signs fluctuate at times. Sympathetically maintained pain may be present and may be demonstrated with pharmacological blocking or provocation techniques. Affective symptoms of disorders occur secondary to the pain and disability. Guarding of the affected part is usually observed".

This is a long but relatively comprehensive definition of RSD. Building on the basis of this comprehensive definition of RSD, one can come to the conclusion that RSD is a syndrome with multiple manifestations which require the following minimal symptoms and signs for the condition to be called RSD (CRPS).

1. Pain: constant, burning pain, and in some forms at times during the course of the disease, stabbing type of pain (causalgic). The pain is relentless and is invariably accompanied by allodynia (even simple touch or breeze aggravating the pain) and hyperpathia (marked painful response to even a simple stimulation).

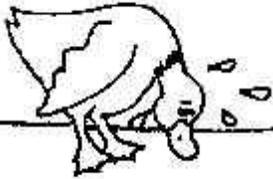
2. Spasms in the blood vessels of the skin and muscles of the extremities. The spasms in the blood vessels result in a cold extremity. The muscle spasms result in tremor, movement disorders such as dystonia, flexion spasm, weakness and clumsiness of the extremities, and tendency to fall.

3. RSD is accompanied by a certain degree of inflammation in practically all cases. This inflammation may be in the form of swelling (edema), skin rash (neurodermatitis), inflammatory changes of the skin color (mottled or purplish, bluish or reddish or pale discolorations), tendency for bleeding in the skin, skin becoming easily bruised, inflammation and swelling around the joints as well as in the joints (such as wrists, shoulders, knee, etc.) which can be identified on MRI in later stages, and secondary freezing of the joints.

4. The fourth component and pre-requisite of diagnosis of RSD is insomnia and emotional disturbance. The fact that the sympathetic sensory nerve fibers carrying the sympathetic pain and impulse up to the brain terminate in the part of the brain called "limbic system". This limbic (marginal) system which is positioned between the old brain (brainstem) and the new brain (cerebral hemispheres) is mainly located over the temporal and frontal lobes of the brain. The disturbance of function of these parts of the brain results in insomnia, agitation, depression, irritability, and disturbance of judgment. Insomnia is an integral part of an untreated RSD. So are problems of depression, irritability and agitation.

So the clinical diagnosis of RSD is based on the above four principles rather than simply excluding RSD and finding some other cause for the patient's pain.

The Four Duck Principle

<p>Principle 1: Afferent: Allodynia, hyperpathia.</p>	<p>Principle 2: Efferent: Muscle spasm, cold extremities, paresis, tremors.</p>
	
<p>Principle 3: Inflammation: Edema, ulcers, skin rash, MRSA.</p>	<p>Principle 4: Limbic System: Dysfunction, poor memory and judgment, insomnia, depression.</p>
	

Illustrations By: Mary Trent

Other laboratory and ancillary tests are helpful in confirming the diagnosis but if the results are negative they don't rule out RSD. For example, bone scan has 55 to 65% sensitivity of positive results in RSD so the other 35% of the patients still have RSD in the face of negative bone scan.

The thermography may be positive and helpful in confirming the diagnosis of RSD but at least 10% of RSD patients do not have the positive thermographic test. This is because of symmetrical involvement of RSD in both extremities. Thermography also has the handicap of tests such as MRI in that it can show false-positive results showing temperature changes in the absence of the symptoms and signs of RSD.

Phentolamine IV nerve block test is probably the most sensitive test to confirm RSD in the earlier stages (stages I and II within the first two to three years of the disease). However, as the disease becomes chronic, the longstanding constriction of the blood vessels causes disturbance of the circulation in the peripheral somatic nerves with resultant involvement of the somatic sensory nerves as well. As a result, the patient does not show a pure sympathetically mediated pain (SMP) but can show sympathetically independent pain (SIP) with no relief from Phentolamine in late stages of RSD.

In conclusion, RSD is a clinical bedside diagnosis. Not every hyperpathic pain is RSD. Not every SMP is RSD. SMP can be due to a simple post-herpetic neuralgia or diabetic neuropathy but that does not make RSD.

"Now that RSD has been diagnosed by the above criteria, what is the nature of the illness, manifestations, and treatment of RSD?"

RSD, as defined above, usually develops after a minor trauma. There are precipitating factors that enhance the development of RSD. These consist of immobilization of the extremity with cast or brace, application of ice, and inactivity due to strong addictive narcotics and tranquilizers.

The application of ice plays a major role. In experiments regarding the conductivity of the nerve impulse to the nerves, cold has shown to play a major role. If the temperature of the extremity drops from 37°centigrade to 10° centigrade, then the larger somatic sensory nerves stop conducting electricity through the nerve fibers. It takes the temperature to drop to 0° centigrade (freezing temperature) for the sympathetic nerves which are small thin fibers to stop conducting. The reason is the smaller the nerve fiber the less fatty sheath of myelin (insulator) surrounds the nerve. The fat in the myelin freezes more readily with the drop of temperature and stops the conduction of the nerve impulse.

What the above implies is the fact that once the extremity is cooled down with ice to 10° temperature or lower, the normal conduction of the somatic nerves (touch, vibration, and position sense) is blocked off and the extremities left with purely the sympathetic nerve fibers function (constant burning pain).

The above clearly indicates that cooling of the extremity that had minor injury will tilt the scale towards the hyperactive sympathetic burning pain function at the expense of excluding the normal good senses of touch, vibration and position senses.

The somatic and sympathetic sense counteract each other. The somatic sensory system inhibits and stops the provocation of the exaggerated burning pain at the spinal cord level. Cooling of the extremity selectively knocks out the inhibition of the good sensory input (somatic) and causes sympathetically mediated pain (SMP) to be the sole modulation of the sensation in the RSD extremity.

It becomes obvious that not only application of ice aggravates RSD but plays a major role in the development of RSD as well.

The application of brace type cast, wheelchair and crutches also reduces the proper sensory input and results in immobilization of the extremity. The extremity that has become immobile loses its surface temperature. The body considers that extremity inactive and not needing blood on the surface so the body constricts the surface blood vessels to preserve heat and not to waste it on an immobile extremity. This second factor of immobilization aggravates the first factor of burning pain by increasing the degree of constriction of blood vessels to the skin of the extremity.

The use of addicting narcotics and benzodiazepines (tranquilizers), results in withdrawal pain every 4 to 5 hours. The use of such addicting medications puts an end to the brain manufacturing its own narcotics and BZs. As a result, 4 to 5 hours after the administration of such medications, even though the patient does not have a new injury affecting his body, feels withdrawal pain because of the lack of endorphines and endoBZs.

The combination of the above three factors, use of ice, immobilization and addicting drugs, exaggerates the SMP (sympathetically maintained pain) due to the original minor trauma.

As the condition becomes chronic, the other aspects of this syndrome complete the clinical picture. Inflammation develops, insomnia, agitation and depression affect the patient's diurnal cycle, deprive the patient of resting well and sleeping normally at night and the end result is the full-blown picture of RSD.

The original injury may involve any part of the body, but the most common areas affected by this type of sympathetic pain (i.e. constant burning pain, accompanied by severe anxiety, depression and muscle spasm) are the hand, elbow, knee, and foot.

In the United States, over five million patient suffer from this extremely painful and disabling illness called by many names, including reflex sympathetic dystrophy (RSD), sympathetic dysfunction syndrome (SDS), and causalgia, to name a few. This unusual, but severe painful condition is caused by disturbance of the function of the sympathetic nervous system (SNS).

Normally, the sense of pain is perceived through two separate channels in the body. The most common type of pain is transmitted through the nerves that end up in the cortex of the brain, over the vertex of the head (parietal lobe). This somatic pain is temporary, clear-cut and focalized. As the area of nerve damage is healed, the pain disappears.

In contrast to the somatic pain, the sympathetic pain terminates in a more primitive part of the brain, called the limbic system [2]. This is the area of the brain at the margin of brain stem and neocortex (frontal and temporal lobes). It controls emotion, memory, and judgment. The end result is insomnia, agitation, irritability, poor judgment and depression (Tables 1 and 2).

Table 1 Pain Perception
1. Somatic (simple common pain) Parietal Cortex
2. Sympathetic (Neuropathic)
3. Bilateral Limbic System / Anterior Frontal Temporal Lobes [2]

Table 2 RSD Components
1. Sensory: Burning Pain
2. Motor: Cold Extremity Tremor/Flexor Spasm [3,4]
3. Inflammation: Swelling, Skin Rash, Bruising of Skin, Osteoporosis, Fractures, Fluids in Joints
4. Limbic System Dysfunction (Emotional Control Centers of Temporal and Frontal Lobes): Insomnia, Agitation, Depression, Poor Judgment

As the condition becomes more persistent and chronic, the physician unknowingly perpetuates and aggravates the condition by the good intention of trying surgical procedures in the area affected by RSD.

The disease was first recognized by the Civil War surgeon from Atlanta (Dr. S.W. Mitchell); however, even a century later, this condition remains enigmatic, and is often diagnosed too late for successful treatment; not infrequently, it is complicated by unnecessary surgical operations, along with addicting narcotic and tranquilizer treatment. Any operation in the injured area can result in new scar formation and therefore a new source of pain. The treatment with narcotics and tranquilizers deprives the brain of its own endogenous hormones (endorphins); this causes a new source of pain due to drug dependence. The scar of surgical procedure, added to drug dependency causes rapid deterioration; at this point, the treatment becomes the source of the disease.

Eventually, the four major manifestations of RSD evolve (see RSD Components, Table 2). The combination of a constant burning pain, which is instigated by even a simple touch (allodynia, muscle spasm and tremor, anxiety, depression and agitation, may result in a confusing, confounding and frustrating clinical picture, making the diagnosis and proper treatment quite difficult.

As a result, the source of this condition is mistaken for "psychological causes; e.g., malingering, hysteria and hypochondriasis.

In 1992, Doctor Mary E. Lynch had found no significant pre-existing psychopathology in RSD patients [5].

As the disease becomes chronic, it goes through four stages; these stages are not distinctive or clear-cut, but develop in an overlapping fashion. Stages I and II are the earliest and easiest to treat. If the disease is diagnosed and properly treated in the first six months, then stages III and IV shall be aborted. Any attempt at proper treatment, in the form of physical therapy or sympathetic block, will prevent serious complications seen in stage III and IV. (Table 3) As a result, because the classical advanced complications are not seen in partially treated patients, the clinician does not arrive at the proper diagnosis of RSD.

The staging of RSD is not as critically done on the basis of dysfunction (stage I) meaning abnormal function of the sympathetic system in the extremity, dystrophy (stage II , meaning trophic and inflammatory changes, skin changes and other signs of inflammation), atrophy (stage III, meaning usually minimal degree of atrophy in the involved muscles), or stage IV (meaning disturbance of the immune system, suicidal attempts, stroke, heart attacks, intractable hypertension and chest pain, and in some cases development of cancer). The prognosis is more related to the temporal development of the above four stages.

Table 3: Stages of RSD

Stage I: A sympathetic dysfunction with dermatomal distribution of the pain. The pain may spread in a mirror fashion to contralateral extremity or to adjacent regions on the same side of the body. In stage one the pain is usually SMP in nature.

Stage II: The dysfunction changes to dystrophy manifested by edema, hyperhidrosis, neurovascular instability with fluctuation of livedo reticularis and cyanosis causing change of temperature and color of the skin in matter of minutes. The dystrophic changes also include bouts of hair loss, ridging, dystrophic, brittle and discolored nails, skin rash, subcutaneous bleeding, neurodermatitis, and ulcerative lesions (Fig. 5). Due to the confusing clinical manifestations, the patient may be accused of self-mutilation and even "Münchhausen syndrome." All these dystrophic changes may not be present at the same time nor in the same patient. Careful history taken is important in this regard.

Stage III: Inflammation becomes more problematic and release of neuropeptides from c-fiber terminals results in multiple inflammatory and immune dysfunctions. The secondary release of substance P may damage mast cells and destroy muscle cells and fibroblasts.

Stage IV: Identifies the final stage of RSD / CRPS manifested by:

1. Failure of the immune system, reduction of helper T-cell lymphocytes and elevation of killer T-cell lymphocytes. This is in contrast to the opposite, up-regulated function of immune system in early stages.
2. The hypertension in early stages due to alpha-1 up-regulation reverses to eventual exhaustion and to the common occurrence of orthostatic hypotension in this late stage. Blood pressure changes in this stage are due to autonomic failure. The failure of the sympathetic system exaggerates the response to drugs that lower or raise the blood pressure.
3. Intractable generalized extremity and visceral edema involving the abdomen, pelvis, lungs, and extremities due to long-standing disturbance of sodium-potassium and calcium magnesium pumps usually left untreated for several years. The same organs may eventually be subject to multiple abscesses due to failure of immune function.
4. Ulcerative skin lesions.
5. High risks of cancer and suicide.
6. Multiple surgical procedures seem to be precipitating factors for development of stage IV.

This late stage is almost the flip side of earlier stages, and points to exhaustion of sympathetic and immune systems. Ganglion blocks in this stage are useless and treatment should be aimed at improving the edema and the failing immune system.

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The one extreme in the case of major causalgia with significant damage to the sympathetic nerve, the status 1 through 3 can develop in a matter of weeks or a few months.

On the other extreme, for example in the case of heart attack or stroke causing RSD, the stages evolve in a very slow fashion and it may take a few years before stages III and IV are seen.

The faster the stages develop, the more severe the RSD and the treatment should be applied in a more aggressive fashion.

On the other hand, even a partial or minimal treatment of chronic stages of RSD can change the clinical picture of stage III (atrophy) back to stage II or stage I. That is a good sign, but that does not mean that the patient's condition is mild just because the patient is in stage I. The more chronic the disease the more likely the persistence of the symptoms and complications even though the bed side examination shows reversal to stages I and II from stages III and IV.

If properly treated with extensive physical therapy, heat, mobilization, exercise and sympathetic nerve blocks, the success rate for full recovery in the first six months (stages I and II) is better than 80% [6].

SPREAD OF THE DISEASE

Even in the early stages, laboratory tests such as triphasic bone scanning or thermography show a spread of the disease from one side to the other. The headache and facial pain becomes bilateral the facial pain is complicated by dizziness, tinnitus (buzzing in the ear). In the later stages of the disease, the spread is both horizontal and vertical (e.g., from right arm to left, down the legs). The reason for this spread is the anatomical structure of the sympathetic nervous system. The majority of the Sympathetic nerve fibers do not follow the standard somatic nerve fibers, but follow the wall of the blood vessels and end up in the chain of clumps of the nerve cells, called "sympathetic ganglia", which are present on each side of the spine. Input of pain into any part of these chains of nerves causes the impulse to spread both vertically and horizontally [7]. This is the main reason sympathectomy or removal of the ganglia is fraught with an extremely high percentage of failure.

This complex clinical picture of the spread of this disease has played a major role in confusing and delaying the proper diagnosis and treatment of RSD. to begin with, the injury causes such a small scar that may be barely visible. This is followed by a constant burning pain, severe neck pain and headache, spread of pain to the opposite side, followed by dizziness, fatigue, insomnia, agitation and irritability.

On this background, the patient may develop pain in the arm, tremor of the hand, may have trouble walking, spasms in the legs, and may end up in a wheelchair. It is obvious that such a patient may be viewed as a neurotic, depressed, and a hypochondriac.

The condition is further compounded by the fact that the patient has normal MRI, CAT scans, and x-rays. The pain is a physiological phenomenon, due to the disturbance of small sympathetic nerve fibers; CAT scan and MRI will not show such an abnormality. An individual, who suffers a heart attack and goes to the emergency room, has a normal CAT scan and MRI in the face of a potentially fatal disease. By the time MRI is abnormal, showing fluid in the involved joints or damage to the bone due to increased circulation in the deep structures, the disease is quite advanced and easy to diagnose.

Eventually, the spasm may spread to the truncal muscles; as a result, an accordion-like jamming effect of the vertebrae evolves, with resultant bulging of the lower lumbar discs noted on MRI. This may result in unnecessary surgical operation. The RSD causes constriction of blood vessels to the hands; in this clinical picture, the patient is often mis-diagnosed as carpal tunnel syndrome, tardy ulnar nerve palsy, or thoracic outlet syndrome.

Not infrequently, such patients undergo multiple operations over the arms and cervical and lumbar spine regions, with rapid acceleration and deterioration of the RSD.

The worst risk factor, and the cardinal sin, is amputation of a limb. If the patient has any sympathetic mediated pain, amputation is going to multiply the disease by several times, due to the fact that the stump of the Amputation causes scar formation at the cut ending of the nerves with marked exacerbation of RSD in the most severe form, called "Causalgia".

Even without application, surgical procedure over the inflamed area of involvement of the RSD (such as ankle, knee or wrist), is a major aggravator of RSD which overnight changes as stage I RSD to stage III or later.

The Three areas that are most commonly and unfortunately operated on are:

1. Wrist according to the diagnosis of carpal tunnel syndrome. This has already discussed in the RSD quiz related to carpal tunnel syndrome. Simply put, carpal tunnel syndrome rarely causes RSD but in rare cases of RSD the inflammation of the soft tissue at the wrist results in a clinical picture identical to carpal tunnel syndrome (CTS). This form of carpal tunnel syndrome is the effect not the cause. It is the result of RSD rather than being the cause of RSD. In this type of CTS, the entire hand, wrist and forearm are quite hypopathic, allodynic and sensitive to touch in contrast to the true somatic carpal syndrome. In this form of RSD, treatment with nerve blocks and anti-inflammatory medications Baclofen, moist heat and Epsom salt clears up the symptoms and signs of CTS. On the other hand, surgery ends up with disastrous results.

2. The same is true in the case of so-called tarsal tunnel syndrome which is over-diagnosed and over-treated universally by the podiatrists and surgery over the tarsal tunnel on the ankle rapidly deteriorates the already existing RSD.

3. The same is true with a lot of minor injuries to the knee with full-blown clinical picture of cold extremity, flexion, weakness and atrophy of the muscles around the knee, difficulty with weight bearing, severe constant burning pain, all of which prompt the surgeon to explore the knee with obvious disastrous results.

Opposite to the prevalent notion of "treat the source" the proper principle should be treat the RSD and then see what happens to the so-called source.

Even nerve blocks should not be done in the area of inflammation of RSD (over the wrist, ankle, and dorsum of the foot, dorsum of the hand or the knee). Instead the nerve blocks which are very effective in treatment of RSD should be done in axial area (over the spine) where the nerves enter and exit from the spinal cord to the involved part of the extremity.

Simply said, do not needle, amputate, or operate the area of inflammation of the RSD.

DIAGNOSIS

The diagnosis is achieved by the physician's familiarity with the triad of RSD, as mentioned above. Confirmatory tests, such as beneficial effect from sympathetic nerve block, as well as the use of bone scan or thermography are quite helpful. None of the above tests yield 100% positive diagnostic proof. Even sympathetic nerve block is positive and relieves the pain in early stages of the disease. In the later stages, there has been enough damage - due to vasoconstriction - in the involved area, that non-sympathetic pain contaminates the clinical picture and the patient does not receive 100% relief from the nerve block.

TREATMENT

Early diagnosis in the first six months to maximal two years is the key to successful treatment [6]. Surgical procedures have no place in treatment of RSD. Sympathectomy or removal of a part of the chain of sympathetic ganglia on the side of the spine) has an extremely high rate of failure. It has been reported to help the war type of RSD, which is quite different from civil type. In the war type, the soldier is a young teenager who responds favorably to treatment, regardless of the mode of treatment. The war time RSD is due to high velocity damage to the nerves in the proximal parts of the extremities and sympathectomy, even in these cases, has a high rate of failure in the long run. The civil type of RSD is due to a small damage of the sympathetic nerves in the central or peripheral nervous system.

If the patient lives longer than five years, the rate of failure from sympathectomy is over 80%. The scar of the surgical procedure becomes a new source of RSD. Removal of a part of sympathetic ganglia does not prevent spread of the disease in the areas of the body where the sympathetic nerves have been removed. This is due to the fact that the adjacent sympathetic nerves eventually compensate for the lack of sympathetic function due to surgery.

SPINAL STIMULATORS

Insertion of epidural spinal stimulators has been quite vogues. In our comparison of 41 RSD patients treated with spinal stimulators vs. 40 non-RSD chronic pain patients who received the same treatment, the RSD group of patients had only maximum three and one half to four months relief of pain. Afterwards, the stimulator acted as a foreign body and became a new source of RSD. The reason may be due to the fact that the stimulators are digital in nature with predicted rhythmic stimulation, whereas the pain of RSD is practically constant and analog (variable) in nature.

No single physician is smart and potent enough to treat RSD. Successful treatment requires a teamwork of physical medicine, anesthesiology, and neuropharmacology physicians. The keys to successful treatment are early diagnosis, early mobilization and extensive physical therapy, and early detoxification of the patient from addicting narcotics, alcohol and addicting tranquilizers.

The anesthesiologist should interrupt the sympathetic hyperactivity by doing repetitive, successive (six) nerve blocks, combined in the same day with physical therapy and exercise. Discontinuation of ice and all other assistive devices, such as wheelchair, brace, cast, walker, etc., is essential.

The addicting drugs should be replaced with the treatment of choice for chronic pain in the form of newer-generation antidepressants, Paxil, Zoloft, and Trazodone, which are not tricyclic antidepressants. Trazodone is the treatment of choice to replace the tranquilizers and sleeping pills. It provides normal sleep, as well as prevention of chronic pain. The patient can be detoxified quickly and easily by discontinuation of the narcotics and replacement with nonaddicting ones, such as Stadol. The use of muscle relaxant, Baclofen, which selectively works on the spinal cord, counteracts the spasm, clumsiness and tremor.

INFUSION PUMP

In severe brain or spinal cord injuries that result in severe crippling spasticity, the infusion pump can be very effective in the treatment of spasticity. Unfortunately, not enough infusion pumps are being used for treatment of severe head and spinal cord injuries. With the use of Baclofen in the pump, such victims of severe crippling spasticity can become mobilized and can be spared from life-threatening inactivity and bed sores.

The infusion pump has been used for treatment of severe pain. In our studies of over 400 cases of advanced RSD, over three dozen patients have been treated with infusion pump, with close to 90% success rate. Through the pump, a drip irrigation form of pain medication is introduced to the spinal fluid. The pain medication given in one month is equivalent to the amount of medication given in two to three days by mouth or by IM injection.

Infusion pump should not be mistaken with other forms of narcotic administration. The infusion pump is totally different and practically opposite to administration of narcotics in the muscle (IM), IV, by skin patch, or simply in the epidural space.

The intrathecal infusion pump (administration of the medication directly to the spinal fluid that surrounds the brain and spinal cord), provides direct access to the brain and spinal cord bypassing liver, kidney and other organs. As a result, the patient requires only 1/20 monthly dose of the narcotic to provide complete relief of pain. In addition, when the pain is optimally achieved by use of as little as 1 to 7 mg a day (usually 3mg a day) of Morphine sulfate, if the patient for other reasons has pain (such as drinking alcohol or simultaneously taking other addicting narcotics by mouth), increasing the dosage of Morphine in the spinal fluid over and above 9mg per day, causes recurrence of severe pain. This is because the system is so flooded by such a strong dose of narcotics that the brain does not form its own endorphines, and the large doses of Morphine in the pump only causes drowsiness, causes the patient respiratory trouble, but does not completely control the pain.

The above phenomenon emphasizes the importance of optimal small dose of narcotic infusion in the spinal fluid which is obviously non-addicting. When distress is violated by increasing the dosage of medicine in the pump, then it becomes like any other form of addicting narcotic administration. The patient develops severe pain due to the fact that the brain cannot form its own endorphines. If the brain manufactures its own endorphine in the face of large doses of narcotic applied in the spinal fluid, then the patient faces the risk of dying from arrest of respiration. So it becomes obvious that the infusion pump is not just another form of giving addicting medications. It works because a very small amount (usually 1/20 to 1/30 dose) of pain medication is given in the form of drip irrigation directly in the spinal fluid with complete control of pain and complete relief of symptoms and signs of RSD.

DIET

The use of proper diet, with avoidance of chocolate (phenyletholamine), hot dog, liver and sausage, and alcohol is essential in management of RSD.

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