Reflex Sympathetic Dystrophy (RSD)/CRPS/ SUDECK does not exist

"Eppur si muove" G. Galilei.

Introduction

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This essay was prompted by a letter from Professor Mennen, Editor of Ezine, asking me to update the Editorial I wrote in the Journal of Hand Surgery European Volume.

This paper is about my extreme scepticism in the existence of the Sudeck atrophy, also known as reflex sympathetic dystrophy (RSD) or complex regional pain syndrome (CRPS). The name for this diagnosis varies depending on the age of the surgeon, their background, and their country, but they all describe the same: an abnormal painful response after trauma or surgery, accompanied by vasomotor changes, at least in the early stages, and the lack of a plausible cause for its development.

I was emotionally moved, when Professor Mennen wrote: "Your "message" is very important because so many patients are unfairly labelled [with the diagnosis of CRPS1] often because of doctors' slapdash diagnosis-making, ignorance or incompetence" (see also: IFSSH Ezine, Volume 29, February 2018, Editorial, "Checklist for Holistic Management"). I cannot agree more with this statement and the important truths it bears on our profession. In many cases diagnosing a patient with CRPS is tantamount to labelling them as a pariah. It is known that some doctors will refuse to see any patient with this diagnosis. Even worse, are the iatrogenic and psychological consequences of this diagnosis to the patient: [2-4] nocebo effect, medicalization, addiction to opioids and psychotropic drugs, personality changes, low self-esteem, catastrophic thoughts, etc. Many of these medications and therapies make it increasingly difficult to reverse the process. My second concern about this diagnosis is that it often is the end result of poor doctoring; in most cases rigorous physical exam and listening to the patient will elucidate the true mechanical or physical source of their pain.

In this report I will describe the process which drove me into playing this Quixotesque role of placing my reputation on the line at every meeting for the sake of debunking this diagnosis once and for all. As a matter of fact, by the time this writing comes out I may have already been burnt on a stake, as I will have delivered my Invited Keynote Speech before the American Society for Surgery of the Hand on my findings on RSD. However, like Galileo, who when before the Inquisition Tribunal and pressed to renounce his thoughts publicly, mumbled ""Eppur si muove" (yet it moves)", I too will stand and say out loud: RSD/CRPS1 does not exist.

THE BEGINNING OF THE END:

Preliminaries

I consider the whole condition named RSD-CRPS1-Sudeck, to be a complete fabrication. In my view, it is a very convenient "trashcan" diagnosis, where all pain complaints that we do not understand can be placed. The patient with this "syndrome" is sent away from the surgeon to Physical Therapy (and later to the Pain Clinic). In this manner, we surgeons, get rid of the problem, of the patient, and we can continue sleeping happily feeling we could not have prevented the problem. In other words, the source of chronic post-operative pain can be blamed on RSD, and, of course, on the patient, who is the ultimate person responsible for being so sickly that they develop this condition. Let's stress from the beginning that many of us think that there is something very suspicious about the whole concept. However, since everybody accepts it, we play what in psychology is called the bystander effect: "if nobody is willing to do anything, why should I?"[5]

The origin of CRPS is confusing, but it began 150 years ago when Silas Mitchell, a neurologist, described the burning pain and vasomotor changes soldiers had in their limbs after sustaining major nerve trunk injuries. The condition was named causalgia and it had an evident inciting pathology - a nerve injury. This initial concept was distorted by Paul Sudeck in 1900, who extended the condition to cases with similar clinical picture but caused by minor or even a non-traumatic event (minor causalgia). Later, Leriche and Policard would attribute over activation of the sympathetic nervous system as for the pathology resulting in the unusual clinical picture. It is difficult to determine when and how this totúm revolutum ended up in what we know today as RSD/CRPS1/Sudeck. But even with today's medical advances,[6,7] this "condition" has no consistent clinical picture, no specific diagnostic tests, an unknown pathophysiology, and lacks pragmatic curative treatment. Surprisingly, with such a meagre pedigree, it commands a major place in the medical literature. It is backed by no fewer than 6000 papers in a simple PubMed search and hundreds of chapters in reference books.

Indeed, it is remarkable that now, in the current trend of precise scientific virtuosity, when there is adoration for statistics versus observation; now, when anything published in any of the "reputed" journals has to be proved, doubly tested, doubly blinded, and doubly wrapped in numbers; precisely NOW, it so happens that this condition survives, as concocted more than 100 years ago, purely thanks to our blind adherence to long-established tenets, with no scientific foot to stand on. Is this not concerning?

To be fair, the condition has been not totally static since its inception by Mitchell, Sudeck and Leriche. Some years ago, the terminology and taxonomy of reflex sympathetic dystrophy were revised in order to dodge the lack of sympathetic system involvement.[8] The new terms are complex regional pain syndrome type 1 (CRPS type 1) and CRPS type 2.[9] Both CRPS 1 and 2 shared symptoms and signs, but while in type 2 there is an injured nerve, in CRPS1 no nerve injury can be recognized. Thus, reflex sympathetic dystrophy (RSD) and Sudeck is parenthetically retained for CRPS type 1 and causalgia is in turn maintained for CRPS type 2.

Although in this paper I will refer more to CRPS1, I will unavoidably discuss both, as CRPS 1 and 2 are often mixed in the literature, share core diagnostic features, and hence, make the distinction quite elusive at times.[10,11] From the beginning I should remark that true CRPS 2 (causalgia) is a different animal from CRPS1: it has an underlying pathology (a damaged nerve),

surgical treatment, and often a cure.[12,13] Conversely, CRPS1, which is (still) mainly considered a sympathetic mediated problem, has a medical approach with variable success and low-quality evidence to support any of the recommended treatments: [14] stellate ganglion blocks (repeated as needed),[15] sympatholytic drugs, opioids and drugs for neuropathic pain (anticonvulsants/antidepressants), bisphosphonates, steroids, free-radical scavengers, among many others. [6,7,15-20]

I should stress, that despite the fact that the condition has a main place in medicine, prominent surgeons and neurologists had already expressed their concerns on the overuse of CRPS as a diagnosis.[8,21-24] Championed by Dr Kasdan, some realized that behind many CRPS cases were the so-called psychogenic-hand.[25,26] Zhu, Jupiter, and Jones[12,13,27] pointed out that some forms of causalgia (presently known as CRPS 2) could be treated surgically offering a solution to patients with excruciating pain where a nerve had been insulted.[10,28]

As early as 1962, Stein[29] linked some forms of Sudeck to compression of the median nerve in the wrist. Later on, several studies favor the same approach for patients who have positive neurophysiological studies.[11,30-32] Finally, Dr. Dellon deserves a main seat among these pioneers, and although his body of work has been devoted to the lower limb,[24,33] his focus on the nerve as the root cause of this condition has also been applied in the upper limb.[34]

Is the literature always right?

We are all influenced by the literature and what has been written weighs heavily on any change. No one wants a rumpus in their Journal. Not surprisingly in my Editorial in JHSE,[1] the Editor-in-chief added a note saying that the journal view was to accept CRPS as a condition.[35] However, one of my mentors, Ian Jackson (a top craniofacial surgeon at the Mayo who, to my surprise and fortune, pupiled me very early on in my career), took me aback by saying: "Only stupid people believe everything that has been written". He was so right! There are plenty of examples even in recent medical history. Let's remember that the highest impact journals, i.e. New England Journal of Medicine, Lancet, JAMA, Annals of Surgery and many others, have supported the benefits of vagotomy for peptic ulcers for generations, yet we all know that the whole idea was "fake news".

For CRPS the literature has undergone constant adaptation to the results which demonstrated that a previous theory explaining and justifying CRPS was wrong. Just think for a moment that the role of sympathetic system in RSD was dismissed, once well-performed randomized studies proved that a placebo was just as good as the sympatholytic drugs.[8,21] Aggressive treatments such as phenol, were met with at best no effect or at worst the painful sympathalgia.[36] The promising spinal cord stimulation[37] could not stand the test of time,[38] and, so on, for the steroids, or any drug you can imagine. Even in recent reviews of the topic on RSD: [6,7,15,16,18,20,39] "could", "should", "perhaps", "at times", "often", "frequently", "seems", "may", "guess", "suppose", "theorize", "surely", "accept", "classically", "commonly", "probably", "suggest", "speculate"…"often are, but also may" are the strongest scientific terms the different authors use to support their conclusions. Could the diagnosis be a fabrication? (Figure 1).

The recognition that the Pain Clinics are graveyards

Again, many clues come by chance. I had a patient who, having been in the Pain Clinic with the diagnosis of RSD for 10 years following a minor crush to his thumb, came for another opinion. I found he had a glomus tumor, which I treated and cured. As I used to live in a small town where everybody bumps into each other, when I ran into the pain doctor whom I told, quite excitedly, that I had successfully treated a patient of his who had been diagnosed with CRPS. He replied, somewhat offended: "My role is not to know what the patients have but to ease their pain"….and he was right, but he also gave me a most important clue for my future: once you are in the Pain Clinic nobody is going to help you to get out of there.

The lack of knowledge/CRPS ratio

After this very first patient I began to see more and more patients diagnosed with RSD and, in most, I was able to find the cause and treat them successfully.

This was astonishing as by definition a patient who has been labelled as CRPS has no treatable cause for their pain. I also noticed that most cases came from surgeons whom I knew not to be the most knowledgeable (Figure 2). Without doubt, behind these alarming numbers there was a need to get rid of "annoying" patients who would be condemned to the Pain Clinic - to the graveyard- with no hope in sight.

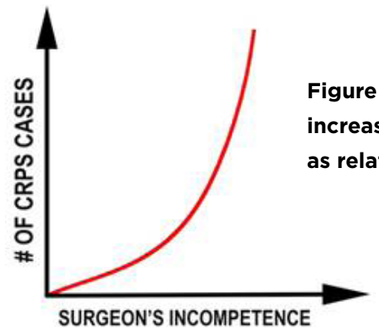

Figure 2. Exponential increase of CRPS cases as related to ignorance

Table 1. Budapest Criteria for CRPS (Harden at al., 2010)

AT THE TIME OF EXAMINATION THE PATIENT MUST REPORT:		
1. Continuing pain	disproportionate to inciting event	
2. Symptoms	at least 1 in 3 of the following 4 categories:	
	Sensory	hyperaesthesia/allodynia
	Vasomotor	temperature/ colour changes - assymetry
	Sudomotor	oedema/sweating changes - assymetry
	Motor / Trophic	deceased ROM. weakness tremor/dystonia trophic changes in skin, hair or nails
3. Signs	at least 1 in 2 of the following categories:	
	Sensory	hyperaesthesia to pin prick Allodynia to light touch
	Vasomotor	evidence of temperature/colour assymetry
	Sudomotor	evidence of oedema/sweating assymetry
	Motor / Trophic	evidence of deceased ROM, weakness tremor/dystonia, trophic changes to skin, hair or nails
4. No other diagnosis explaining symptoms and signs		

Weaknesses of the Budapest criteria

But why was it possible for a surgeon to label a condition as being CRPS if it had a treatable cause? How could we make a diagnosis that has such an enormous impact on a patient's well-being so frivolously? The reason was, without a doubt, that the criteria applied to diagnose CRPS were extremely indistinct and biased: anything would fit (Table 1).[40] Specifically, pain (criteria 1) was subjective, and criteria 2 and 3 were shared by trauma, inflammatory conditions, ischemia, etc, and were thus quite unspecific. However, the most unfair of all diagnostic criteria, and the one which later proved to be the main "sinkhole", was item 4. Criteria 4 left to a doctor's discretion which patient they felt had an unknown condition. As I already pointed out in my former editorial, "The only person who should reliably state that there is not an overt organic cause for a patient's pain should be the specialist in the field i.e. a Hand Surgeon". The rest do not have the knowledge and understanding to label a patient with CRPS. But even in the realm of hand surgery, do we all know the same?

THE IMPORTANCE OF IRRITATIVE CTS

The recognition that I could successfully treat patients labelled as having RSD by fellow surgeons/doctors, and the direct relationship to their lack of knowledge, triggered in my mind the following "anti-establishment" thought: "If only we all knew all the facts, RSD/CRPS/Sudeck would not exist". I, hence, became obsessed trying to understand what these patients really had. It was clear that some had factitious issues, others had sustained a nerve trauma (Causalgia/CRPS2), and a large group had suffered from bad-doctoring. But, what about the rest?

I am not sure exactly how and when I realized that the complaint of worsening of symptoms at night reported by some CRPS patients was the clue in the remaining cases. The only condition that I knew to produce this was compression of the median nerve in the carpal tunnel. However, these patients labelled as CRPS had a very different picture from a classic carpal tunnel syndrome (CTS): pain and numbness away from the median nerve territory, swelling, stiffness, burning pain, etc (Figure 3).

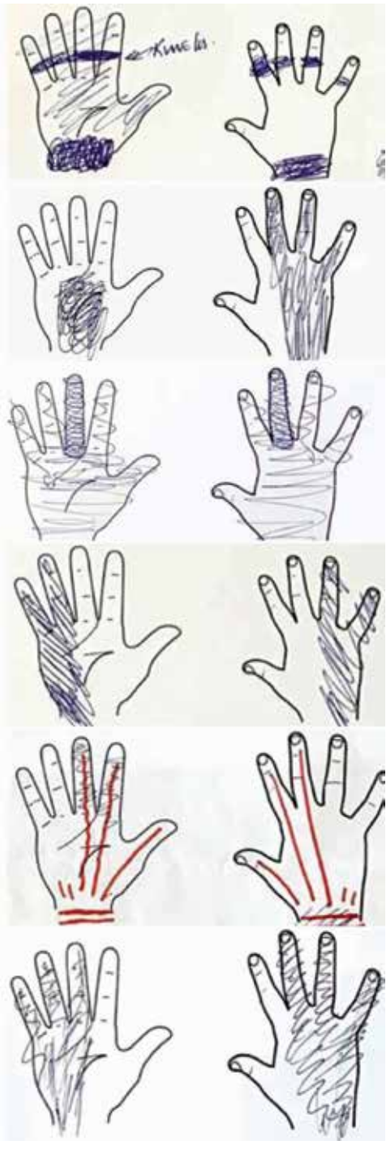

Figure 3. A sample of charts of my ongoing study, showing the area of allodynia-dysesthesias in the hand reported by the patients (marked). Note that most had more symptoms in the ulnar nerve territory, some in a glove distribution and few in the median nerve territory.

As always, most responses could be found in the literature. It has been long known that brachialgia or even more proximal pain, can be originated by compression of the median nerve in the carpal tunnel, and that by releasing the ligament the pain disappears.[41,42] Swelling has not been considered a prominent feature in CTS, yet Burke et al. have proved it to be a most prevalent symptom in idiopathic CTS.[43] The works of Ochoa and others opened my eyes and helped me to understand that the median nerve could cause pain in the ulnar nerve territory, proximally or even simulate an acute myocardial attack.[10,44] Furthermore, Bennett and Xie[45] demonstrated in rats that nerve compression triggered a clinical picture similar to a CRPS1. This assured me I was very much on the right track: the median nerve. Nevertheless, I still needed more evidence.

Currently, any ground-breaking change in our practices, or any new condition, is accepted only if accompanied by objective data, statistics and "evidence". Albeit good, we should not forget that observation has contributed enormously to the progress of Medicine. Through observation, Fleming realized that penicillium had a bactericidal effect. Through observation, syndromes such as "partial thenar atrophy" and "acroparesthesia" (derogatively known, as it affected mostly women, as "hysterical nocturnal paresthesias" – these were certainly pre #metoo days) were linked together, giving birth to a new condition: the ultrafamous carpal tunnel syndrome (CTS).[46] It is astonishing to realize that the most common operation a hand surgeon performs today, carpal tunnel release (CTR), was unknown until 1950. Thousands of patients have benefited from this important understanding - simply based on observation.[47,48] Before this linkage several "treatments" for idiopathic CTS patients had been recommended: quinine, iron, arsenic, morphine, strychnine, henbane, galvanization of the hands with interrupted and continuous current, Phenobarbital and Bromide combined with a vasodilator, and rib resection, among many others.[46] To sum up, all these advances, and so many more in the History of Medicine, were thanks to observation, presently reviled by the scientific methodology.

Through observation we noticed that a group of patients who had "compression" of the median nerve, did not display the typical CTS signs and symptoms. Chief complaints were pain and tingling in the median nerve distribution (but often not limited to it), worsening of the symptoms at night, swelling, and, above all, inability to make a full fist (Figure 4). Those were exactly the same signs and symptoms a very different group of patients displayed. The latter were all patients with previous trauma to their hands and who had been labelled and treated for CRPS prior to referral. Both groups were indistinguishable clinically and both were treated with carpal tunnel release (CTR). In no case were continuous axillary blocks, sympatholytic medication, or stellate ganglion blocks given after the surgery. Light painkillers (paracetamol orally) was prescribed as per our usual protocol for patients who have idiopathic CTR. The response and outcome to surgery was indistinguishable. We named this new condition irritative carpal tunnel syndrome, as the median nerve seemed to be irritated rather than

Figure 4.

compressed. I should stress that these patients were operated disregarding their nerve conduction studies which were negative in 2/3 of those whom were asked.

Figure 4. Severe form of bilateral irritative carpal tunnel syndrome. In both pictures, the patient is being asked to make a tight fist. On the left, he is shown preoperatively, on the right he is shown in the OR after bilateral CTR under local anesthesia. No antecedent trauma could be recalled.

THE SERIES: 100 patients with the diagnosis of RSD

Irritative carpal tunnel syndrome was the missing link I was looking for to be able to come full circle!!!

To confirm that I was right, I studied a cohort of 100 consecutive patients who had come to the office with the diagnosis of RSD. (It took me more than 4 years to gather the first 25 patients, but once I had appeared on the national news dealing with this topic, I gathered the remaining 75 in less than a year). Briefly, the preliminary data shows that ninety-five percent had been diagnosed with RSD by a physician (surgeon or rehabilitation doctor) and the rest by a therapist. Ninety percent had been treated in a pain clinic. Ninety-three percent were on opioids and/or psychotropic medication, one for more than 15 years. The remaining were on painkillers and steroids. All had pain, sense of stiffness and/or limited range of motion.

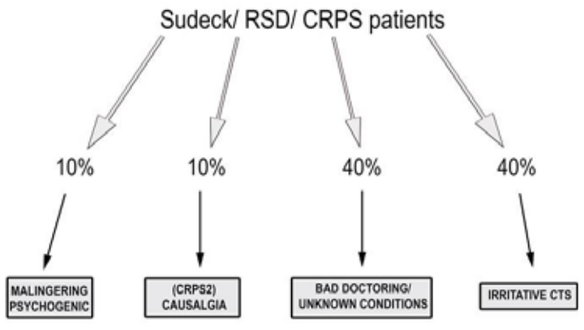

Figure 5: True allocation of the patients diagnosed with Sudeck's in this study.

The general results can be summarized in the chart below (Figure 5). Note than in nearly half, bad doctoring was behind the scenes: ignorance or overt malpractice masked by the CRPS/RSD acronyms!!

The psychogenic-hand patients were advised to go to a psychiatric consultation with little success. Five patients did not require any surgical treatment, but support in PT and were weaned off drugs. I open here a parenthesis to stress that it is not uncommon for some patients to develop, after surgery or trauma, what I was taught to be a "flare reaction". The clinical picture appeared some weeks after the trauma/surgery and shared the same symptoms and signs as a CRPS1. The problem cleared up with PT and reassurance. Now, due to abuse of the diagnosis of CRPS and the laxity of the criteria,[18,40] some flare reactions can be misinterpreted as CRPS - dragging the patient into the pernicious effects of such a misdiagnosis.[2-4]

Nearly half dismissed any operation on the grounds: "my doctor advised me against surgery", "my doctor told me you are only interested in operating on me to make money" and "the literature is against surgery", yet they had treatable conditions. The oversighted pathology was at times embarrassing (Figure 6).

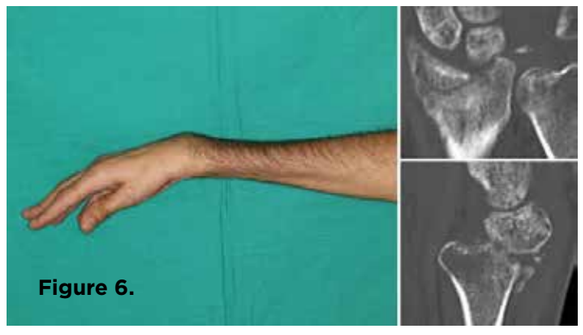

Figure 6. This 34-year-old male diagnosed with Sudeck had neuropathic pain, anesthesia in the median nerve territory, and a frozen hand. He had sustained a motorbike accident which required a week of ICU admission for abdominal and head trauma. The wrist was placed in a (tight?) cast. The CT scan shows the status of the wrist at his very first visit, 3 months after the accident. The malunion was aggravated by a concurrent Volkmann and intrinsic muscles contracture. Surgery was advised but the patient declined, arguing that his treating doctor had stressed to him that any surgery in a Sudeck patient would harm him. Instead, a 4-month course of PT and psychotropic medication were recommended. He was lost to follow up after this "confrontation".

Those who had surgery had their problem solved all bar one. This specific patient remained unhappy due to stiffness. Her pain was rated during physical therapy as 8 over 10 in a VAS (0-10). Nevertheless, her pain at night and daytime dropped from 10 to 0 at the six month follow up. This particular patient had a metastatic lung cancer and was undergoing chemotherapy. She developed shoulder and wrist pain after having been operated on for a wrist fracture. It is worth stressing that those with Irritative CTS were operated on disregarding their nerve conduction studies which were negative in a significant number of those whom were asked.

To summarize, by keeping the categorization chart in mind, no patients were to be labelled as RSD/CRPS1/ Sudeck.

THE AFTERMATH

TThe birth, or the death, of any condition or assumed treatment is, inevitably, surrounded by controversy. Recently, we have witnessed the death of vagotomy in peptic ulcer management, despite a wealth of supportive literature in high-ranking journals.

In my Quixotesque role to kill this condition, I am not alone - I am standing on the shoulder of giants, surgeons and neurologists,[8,10-13,21-34] whose keen eyes have been cornering the condition: psychogenic hand, causalgia (CRPS2) were but a part of the picture. But this was not enough, there were some patients left out who had symptoms and signs that could be assigned to CRPS. My background as a wrist and microsurgeon allowed me to spot structural bad-doctoring as the root cause of many CRPS cases. The final piece to the jigsaw was noting that the median nerve could be irritated without being compressed, thus frequently the standard neurological studies were non-contributory. More than 40 patients have been operated on for irritative CTS (Figure 7) and all except one, aforementioned, responded. Whether she had an "unknown condition", a psychogenic hand, or a grievance because of her underlying medical condition, requires further study. In this respect, patients who do not improve after a rigorous search for the cause or after failed surgery, might need to go to the Pain Clinic. This must always be under the supervision of the treating surgeon. For sure, the Pain Clinic should not be a graveyard of discarded patients, but the place where patients are to be treated temporarily, while the root cause of the problem is sought out and solved.

Figure 7. This patient was seen 8 months after sustaining a DRF treated in a cast. No malalignment existed on the X-rays. Nevertheless, she developed pain and swelling that required intensive PT and treatment in the Pain Clinic (see patient's picture on the left at 5 months with persisting swelling). On her first visit, despite being on gabapentin, lidocaine patches/opioids she rated her pain as 8 (on a VAS:0-10) and could not make a fist. Five weeks after CTR under local her pain was 0. She returned to work as a housekeeper in a hotel.

But do not get me wrong, the problem is not solved by simply releasing the carpal ligament. In fact, doing so, can even be detrimental: there are many

factors in play. Just as when recognizing a factitious injury you get to know all possible answers,[25,26,49,50] the same applies for bad-doctoring. In about 40% of the cases there was a major medical error behind them (including inadequate carpal tunnel release in 15%). Co-existent shoulder pain and established stiffness interfere in the recovery and has to be addressed. Besides this, surely the percentages and root causes of CRPS cases vary in each surgeon's practice. As an example, this may be more a nerve problem in Dr. Dellon's office, more a mechanical cause in mine, and a mixed etiology in others'. We are currently looking for a test that could help everyone to spot a patient with a treatable condition. We are studying the small fibers which are the afferents for pain, burning sensation, and which dominate vasomotor effectors (Aδ fibers -small myelinated- and C fibers -unmyelinated). These fibers cannot be recorded by the standard neurophysiological tests (which study large –fast-conducting- myelinated fibers). Perhaps the so-called Sympathetic Skin Response test may prove useful.[51-52] Another avenue is functional brain MRI. With the latter, we want to sort out who has real pain, and how different this is in irritative carpal tunnel syndrome versus idiopathic carpal tunnel syndrome.

My final words go to the suffering patients who need our love and compassion. They have been wandering from one doctor to another and resemble zombies more than normal people. Very few are looking for a secondary gain, and indeed most are under the effect of multiple drugs and receiving no empathy. (I recently had, as a patient, an ICU doctor, who could not stop cursing the incomprehension she had been through before we tackled her problem). I cannot deny that there are patients whose characteristics may make us repudiate them from the very outset. But let's be rational. First of all, as they have already been subjected to a failed treatment, they will regard the new surgeon with suspicion and lack of confidence. Furthermore, those who have been in the Pain Clinic for a long period, have become addicted to several drugs, with personality implications (low self-esteem, passive-aggressiveness, uncoordinated discourse, among others). Typically, all of them have a very low threshold for pain and, because of their long use - and abuse - of drugs, they self-medicate setting their own rules much to our despair. However, after several days or weeks, once they start to feel better, they place their regained trust in their treating doctor and become the most cooperative, thankful and willing to help others that you have ever had. Love them, they need our love and care.

CONCLUSIONS: The amplifier effect.

If I were to condense all my research into a very simple theory, I should name it the "Amplifier effect". The term stresses that, much like the amplifier of a music system which multiplies the sound picked up by the needle of the turntable, the "damaged/irritated" nerve will likewise multiply the final pain reading in our cortex to the power of "n" (Figure 9). This theory may appear as unproven as others that have attempted to explain the physiopathology of CRPS. However, there is an important difference - this approach has pragmatically proved successful.

Another conclusion of this study is that CRPS1 and 2 are the same condition, the former with an "irritated" nerve (RSD, Sudeck), and the latter with a structurally damaged nerve (causalgia). Nevertheless, both are nerve mediated pathologies. Therefore, a more appropriate term for the whole clinical picture could be "Neural amplified pain". Patients without a nerve issue, have to be categorized as psychogenic hand, malingerers, victims of bad doctoring, and some early cases could fall into the so-called "flare reaction" pigeonhole. Finally, a minority may be ascribed to unknown conditions (1 in 100 in my series). If one adheres to these understandings, there will be no more mystery hidden in CRPS. More importantly, the patient will be properly allocated to a real condition, not to CRPS. Consequently, this will prevent unnecessary suffering and medicalization in all patients,[2-4] and above all in those most vulnerable.[53,54] I foresee that in contradiction of the current trend of rocketing numbers of CRPS1 cases (50,000 new cases per year in the USA),[55] removing the niche will reduce the incidence of the problem drastically in few years, as has already occurred in other unsubstantiated epidemics in our field.[56,57]

In closing, I beseech you to help me to wipe out this fabrication that leaves our suffering patients stranded in Pain Clinics around the globe. Our endeavor is paved with resistance, not only has the existence of RSD been engraved in stone in our minds, but stubborn opposition to new ideas is part of the human nature. I confessed in my Editorial[1] that I dreamed about the eradication of this pseudo-condition. Now that the enlightenment is so close, I admit that too often I awaken in the middle of the night and it takes me hours to fall sleep again because of the excitement of knowing that if I succeed I will have done more good than anything else I have produced in my life. Please help the progress of science by placing all your medical knowledge at the service of your next CRPS patient.

As a final caveat: It takes a small leap of imagination, that if I can debunk the whole CRPS concept in my terrain, the rest of the body will be open for others to deliver the final blow (a job already started by prominent giants).[13,33,58]

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