NEUROPATHY/NEUROPATHIC MYOFASCIAL PAIN

MYOFASCIAL PAIN SYNDROMES ARE A LARGE AND DIVERSE GROUP OF PAINFUL CONDITIONS THAT OCCUR IN THE MUSCULOSKELETAL SYSTEM.

THEY AFFECT **MUSCLES AND THEIR CONNECTIVE TISSUE ATTACHMENTS** IN **ANY PART OF THE BODY**, AND ARE CUSTOMARILY NAMED ACCORDING TO THE LOCATION OF THE PAINFUL PART (LATERAL EPICONDYLITIS, ACHILLES TENDONITIS, FROZEN SHOULDER, BICIPITAL TENDONITIS, LOW BACK PAIN, ETC.)

THEY ARE PUZZLING BECAUSE THEY SEEM TO **ARISE AND PERSIST IN THE ABSENCE OF ANY DETECTABLE INJURY OR INFLAMMATION**.

MYOFASCIAL PAIN SYNDROMES ARE OFTEN DIFFICULT TO TREAT BECAUSE MEDICATIONS AND THE **COMMONLY AVAILABLE PHYSICAL THERAPIES GIVE ONLY TEMPORARY RELIEF**.

INNUMERABLE PATIENTS, THEREFORE **WANDER FROM PROVIDER TO PROVIDER IN A VAIN SEARCH FOR RELIEF**.

THE TERM **MYOFASCIAL PAIN SYNDROME** IS **USED IN A VAGUE AND INDETERMINATE WAY** TO DENOTE **ANY REGIONAL MUSCULOSKELETAL PAIN SYNDROME WITHOUT REGARD TO ITS SOURCE OR CAUSE**.

CAREFUL EXAMINATION OF THESE SYNDROMES OFTEN REVEALS THEM TO BE THE EFFECTS OF **NEUROPATHY** APPEARING IN THE MUSCULOSKELETAL SYSTEM.

THE INITIAL AND UNDERLYING PROBLEM IS **MALFUNCTION OF THE PERIPHERAL NERVOUS SYSTEM**, AND **PAIN IS** JUST ONE POSSIBLE BUT **NOT INEVITABLE** DOWNSTREAM PRODUCT OF THE **NEUROPATHY**.

THE KEY TO SUCCESSFUL MANAGEMENT OF THIS IMPORTANT AND WIDESPREAD CATEGORY OF CHRONIC PAIN IS TO UNDERSTAND **NEUROPATHY**, HOW IT CAN CAUSE PAIN, AND RECOGNIZE IT IN ITS **MANY GUISES**.

THE **DEFINITION OF PAIN** GIVEN BY **THE INTERNATIONAL ASSOCIATION FOR THE STUDY OF PAIN**: **"AN UNPLEASANT SENSORY AND EMOTIONAL EXPERIENCE ASSOCIATED WITH ACTUAL OR POTENTIAL TISSUE DAMAGE OR DESCRIBED BY THE PATIENT IN TERMS OF SUCH DAMAGE."**

ALTHOUGH PAIN ***MAY*** BE LINKED CAUSALLY TO TISSUE INJURY, IT ISN'T NECESSARILY SO.

INJURY DOES NOT ALWAYS GENERATE PAIN, NOR DOES PAIN ALWAYS SIGNAL INJURY.

PERSISTENT PAIN CAN OCCUR IN THE PRESENCE OF:

- ONGOING NOCICEPTION (E.G., AN UNHEALED FRACTURE) OR PERSISTENT INFLAMMATION
- PSYCHOLOGICAL FACTORS SUCH AS SOMATIZATION DISORDERS, DEPRESSION OR ADVERSE OPERANT LEARNING PROCESSES
- ABNORMAL FUNCTIONING IN THE NERVOUS SYSTEM

PAIN CAN ARISE FROM **NONNOXIOUS INPUT** OR FROM WITHIN THE BODY WHEN THERE IS SOME **FUNCTIONAL DISTURBANCE IN THE NERVOUS SYSTEM** (SUCH AS PERIPHERAL **NEUROPATHY**).

NEUROPATHIC PAIN IS GENERALLY USED TO REFER TO **ANY ACUTE OR CHRONIC PAIN SYNDROME IN WHICH THE MECHANISM THAT SUSTAINS THE PAIN IS INFERRED TO INVOLVE ABERRANT SOMATOSENSORY PROCESSING IN THE PERIPHERAL NERVOUS SYSTEM OR CENTRAL NERVOUS SYSTEM**.

IN ***NEUROPATHIC*** MYOFASCIAL PAIN, STRUCTURAL FACTORS EXIST AS WELL, SUCH AS THE FOLLOWING THAT CONTRIBUTE TO THE PAIN:

- MUSCLE SHORTENING
- DEGRADED, WEAKENED COLLAGEN
- TROPHIC CHANGES

C. CHAN GUNN PROPOSED THE CONCEPT OF **NEUROPATHIC** PAIN WHEN IT BECAME EVIDENT FROM CLINICAL OBSERVATIONS AND RESEARCH THAT:

PAIN IS NOT ALWAYS A SIGNAL OF INJURY, BUT CAN BE A PRODUCT OF **ABNORMAL NERVE FUNCTION**.

HIS EXAMINATION OF PATIENTS WHO HAD BACK PAIN BUT NO SIGNS OF INJURY SHOWED THAT THOSE WHO WERE DISABLED FOR LONG PERIODS HAD TENDERNESS OVER MUSCLE **MOTOR POINTS** IN AFFECTED MYOTOMES.

TENDER MOTOR POINTS ARE SENSITIVE **INDICATORS OF RADICULAR INVOLVEMENT [IRRITATION AT THE NERVE ROOT]**.

TENDER POINTS DIFFERENTIATE A SIMPLE MECHANICAL LOW BACK STRAIN WHICH HEALS QUICKLY FROM ONE THAT IS SLOW TO IMPROVE.

NEXT, A STUDY OF PATIENTS WITH **TENNIS ELBOW** SHOWED THAT TENDER POINTS AT THE ELBOW WERE **SECONDARY TO CERVICAL SPONDYLOSIS** AND **RADICULOPATHY** [**NEUROPATHY ORIGINATING AT THE NERVE ROOT**].

TREATING THE **NECK**, **NOT** THE **ELBOW**, WAS ABLE TO PROVIDE RELIEF.

A STUDY OF **PAIN IN THE SHOULDER** LIKEWISE IMPLICATED **NEUROPATHY AT THE CERVICAL SPINE**.

A PATTERN EMERGED:

PATIENTS WHO HAVE **PAIN BUT *NO SIGNS OF INJURY*** GENERALLY HAVE SENSORY, MOTOR AND AUTONOMIC MANIFESTATIONS OF **PERIPHERAL NEUROPATHY**.

PERIPHERAL NEUROPATHY MAY BE DEFINED AS **A DISEASE THAT CAUSES DISORDERED FUNCTION IN THE PERIPHERAL NERVE**.

ALTHOUGH SOMETIMES ASSOCIATED WITH STRUCTURAL CHANGES IN THE NERVE, A **NEUROPATHIC** NERVE **CAN DECEPTIVELY APPEAR NORMAL**.

IT CONDUCTS NERVE IMPULSES, SYNTHESIZES AND RELEASES TRANSMITTED SUBSTANCES AND EVOKES ACTION POTENTIALS AND MUSCLE CONTRACTION.

FOLLOWING **NEUROPATHY** AND **DENERVATION**, MANY DIVERSE PAIN SYNDROMES OF APPARENTLY UNRELATED CAUSATION MAY BE ATTRIBUTED TO **SUPERSENSITIVE RECEPTORS** [NOCICEPTORS] AND **HYPERREACTIVE CONTROL SYSTEMS** AT ***INTERNUNCIAL POOLS***.

THE FOLLOWING ATTRIBUTES ARE GENERALLY ASSOCIATED WITH ***NEUROPATHIC*** PAIN:

- PAIN WHEN **NO ONGOING TISSUE-DAMAGING PROCESS EXISTS**
- **DELAY IN ONSET AFTER PRECIPITATING INJURY**, GENERALLY TAKING **APPROXIMATELY 5 DAYS FOR SUPERSENSITIVITY TO DEVELOP**.
- **DYSESTHESIA, UNPLEASANT BURNING OR SEARING SENSATIONS, OR DEEP, ACHING PAIN** THAT IS MORE COMMON THAN DYSESTHETIC PAIN IN MUSCULOSKELETAL PAIN SYNDROMES.
- **PAIN FELT IN A REGION OF SENSORY DEFICIT**.

- **NEURALGIC PAIN, PAROXYSMAL BRIEF SHOOTING OR STABBING PAIN**.
- **SEVERE PAIN IN RESPONSE TO A NOXIOUS STIMULUS** [HYPERALGESIA].
- **SEVERE PAIN IN RESPONSE TO A STIMULUS** THAT ***IS NOT NORMALLY NOXIOUS*** [**ALLODYNIA**].
- **PRONOUNCED SUMMATION AND AFTERREACTION WITH REPETITIVE STIMULI**.

NEUROPATHIC PAIN IS PERCEIVED AS ***BEGINNING*** WITH **PERIPHERAL SENSITIZATION**.

IN PERIPHERAL SENSITIZATION INCREASED TRANSDUCTION SENSITIVITY OF NOCICEPTORS IS ASSOCIATED WITH ALTERATION OF IONIC CONDUCTANCES IN THE PERIPHERAL TERMINAL.

SENSITIZATION CAN OCCUR FOLLOWING TISSUE INFLAMMATION OR DAMAGE TO A PERIPHERAL NERVE.

INFLAMMATORY CELLS PRODUCE GROWTH FACTORS AND CYTOKINES THAT CONTRIBUTE TO THE INCREASED SENSITIVITY OF NOCICEPTORS, BUT **DAMAGE TO THE PERIPHERAL NERVE** IS MOST COMMONLY CAUSED BY **SPONDYLOSIS** AT THE **ROOT LEVEL** [**NEUROPATHIC** PAIN] WHEN ALL FIBERS OF THE PERIPHERAL NERVE CAN BE DAMAGED AND CAN LEAD TO ANY OR ALL OF THE FOLLOWING:

- **MOTOR**: MUSCLE SHORTENING IS **THE MOST SIGNIFICANT FEATURE** OF **RADICULOPATHY**; PAIN CAUSED BY THE **MECHANICAL EFFECTS OF MUSCLE SHORTENING**.
- **AUTONOMIC**: INCREASED **VASOCONSTRICTION**; **HYPERHYDROSIS**; **TROPHEDEMA**; AND **CAUSALGIC PAIN**, **REFLEX SYMPATHETIC DYSTROPHY** [**COMPLEX REGIONAL PAIN SYNDROME**].

- **TROPHIC**: **DERMATOMAL HAIR LOSS**; **COLLAGEN DEGRADATION** AND **FRAILTY** LEADING TO **ENTHESOPATHIC TENDONS**.

MINOR DAMAGE TO THE NERVE ROOT ISN'T CLINICALLY OBVIOUS.

RADICULOPATHY IS UNIVERSAL BUT USUALLY UNSUSPECTED IN **PRESPONDYLOSIS** WHEN **PAINLESS MUSCLE SHORTENING PRECEDES PERIPHERAL SENSITIZATION**.

DAMAGED PRIMARY AFFERENT FIBERS DEMONSTRATE:

- SPONTANEOUS ACTIVITY
- EXAGGERATED RESPONSE TO STIMULUS
- SENSITIVITY TO CATECHOLAMINES

THESE ARE EXPLAINED BY **CANNON AND ROSENBLUETH'S LAW OF DENERVATION** BUT IS SELDOM CITED TO EXPLAIN **NEUROPATHIC** PAIN.

IT POINTS OUT THAT THE NORMAL PHYSIOLOGY AND INTEGRITY OF ALL INNERVATED STRUCTURES ARE DEPENDENT ON THE ARRIVAL OF NERVE IMPULSES VIA THE INTACT NERVE TO PROVIDE A REGULATORY OR TROPIC EFFECT.

WHEN THIS FLOW [PROBABLY A COMBINATION OF **AXOPLASMIC FLOW** AND ELECTRICAL INPUT] IS BLOCKED INNERVATED STRUCTURES ARE DEPRIVED OF THE TROPHIC FACTOR WHICH IS VITAL FOR THE CONTROL AND MAINTENANCE OF CELLULAR FUNCTION.

A-TROPHIC STRUCTURES BECOME HIGHLY IRRITABLE AND DEVELOP **ABNORMAL SENSITIVITY** OR **SUPERSENSITIVITY** ACCORDING TO **CANNON AND ROSENBLUETH'S LAW OF DENERVATION**: **"WHEN A UNIT IS DESTROYED IN A SERIES OF EFFERENT NEURONS AN INCREASED IRRITABILITY TO CHEMICAL AGENTS DEVELOPS IN THE ISOLATED STRUCTURE OR STRUCTURES, THE EFFECT BEING MAXIMAL IN THE PART DIRECTLY DENERVATED."**

ALL **DENERVATED** STRUCTURES DEVELOP **SUPERSENSITIVITY**, INCLUDING:

- SKELETAL AND SMOOTH MUSCLE
- SPINAL NEURONS
- SYMPATHETIC GANGLIA
- ADRENAL GLANDS
- SWEAT GLANDS
- BRAIN CELLS

CANON AND **ROSENBLUETH'S** WORK WAS BASED ON **TOTAL DENERVATION OR DECENTRALIZATION** FOR SUPERSENSITIVITY TO DEVELOP.

ACCORDINGLY, THEY NAMED THE PHENOMENON **DENERVATION SUPERSENSITIVITY**.

NOW IT'S KNOWN THAT **PHYSICAL INTERRUPTION AND TOTAL DENERVATION AREN'T NECESSARY**.

ANY CIRCUMSTANCE THAT **IMPEDES THE FLOW OF MOTOR IMPULSES FOR A PERIOD OF TIME** CAN ROB THE EFFECTOR ORGAN OF ITS EXCITATORY INPUT AND CAUSE ***DISUSE*** **SUPERSENSITIVITY** IN THAT ORGAN AND IN **ASSOCIATED** ***SPINAL REFLEXES***.

THE IMPORTANCE OF **DISUSE SUPERSENSITIVITY** CAN'T BE OVEREMPHASIZED IN THAT **WHEN A NERVE MALFUNCTIONS** THE **STRUCTURES IT SUPPLIES BECOME SUPERSENSITIVE AND BEHAVE ABONORMALLY**.

THESE STRUCTURES **OVERREACT TO INPUTS** INVOLVING:

- **CHEMICAL**
- **STRETCH**
- **PRESSURE**

SUPERSENSITIVE MUSCLE CELLS CAN GENERATE **SPONTANEOUS ELECTRICAL IMPULSES** THAT TRIGGER **FALSE PAIN SIGNALS** OR PROVOKE **INVOLUNTARY MUSCLE ACTIVITY**.

SUPERSENSITIVE NERVE FIBERS **BECOME RECEPTIVE TO CHEMICAL TRANSMITTERS AT *EVERY POINT ALONG THEIR LENGTH*** INSTEAD OF ONLY AT THEIR TERMINALS.

SPROUTING MAY OCCUR AND **DENERVATED NERVES** ARE **PRONE TO ACCEPT CONTACTS FROM OTHER TYPES OF NERVES INCLUDING AUTONOMIC** AND **SENSORY** NERVE FIBERS.

SHORT CIRCUITS ARE POSSIBLE **BETWEEN SENSORY AND AUTONOMIC [VASOMOTOR] NERVES** AND MAY **CONTRIBUTE TO *COMPLEX REGIONAL PAIN SYNDROME***.

MANY DIVERSE PAIN SYNDROMES OF APPARENTLY UNKNOWN CAUSATION MAY BE ATTRIBUTED TO THE DEVELOPMENT OF HYPERSENSITIVE RECEPTOR ORGANS AND SUPERSENSITIVITY IN PAIN SENSORY PATHWAYS.

INSTEAD OF NOCICEPTION THERE CAN BE SEVERE PAIN IN RESPONSE TO A **NOXIOUS STIMULUS** [**HYPERALGESIA**] OR SEVERE PAIN IN RESPONSE TO **A STIMULUS THAT IS NOT NORMALLY NOXIOUS** [**ALLODYNIA**].

RADICULOPATHY IS **NEUROPATHY *AT THE NERVE ROOT*** **DUE TO SPONDYLOSIS**.

SPONDYLOSIS IS **THE STRUCTURAL DISINTEGRATION AND MORPHOLOGIC ALTERATIONS THAT OCCUR IN THE INTERVERTEBRAL DISK WITH PATHOANATOMIC CHANGES IN SURROUNDING STRUCTURES**.

THE **SPINAL NERVE ROOT** [BECAUSE OF ITS VULNERABLE POSITION] IS NOTABLY PRONE TO INJURY FROM **PRESSURE**, **STRETCH**, **ANGULATION** AND **FRICTION**.

SPONDYLOSIS INCREASES WITH AGE, THEREFORE, **SPONDYLOTIC PAIN** IS MORE COMMON IN MIDDLE-AGED INDIVIDUALS WHO HAVE ACCUMULATED AN ***INJURY POOL*** [**AN ACCUMULATION OF REPEATED MAJOR AND MINOR INJURIES TO A SEGMENT LEADING TO UNRESOLVED CLINCAL RESIDUALS THAT MAY OR NOT PRODUCE PAIN**].

MAJOR *OR* MINOR DAMAGE TO JOINT CONNECTIVE TISSUE CAN PROVOKE, EVOKE OR INVOKE ***FIBROSIS*** [SCAR TISSUE MULTIPLIES IN A VICIOUS CYCLE, **CONTRACTING WITH AGE**].

PROGRESSIVE JOINT FIBROSIS CAUSES **JOINT DEGENERATION** [**SPONDYLOSIS**] AND **NEUROPATHY**, SOMETIMES PROGRESSING INTO BONY **ANKYLOSIS**.

THIS LIMITS JOINT MOBILITY **PREVENTING ACTIVATION OF CHONDROCYTES** AND COMPRESSES THE JOINT **PREVENTING IMBIBITION**.

THE ABOVE CAUSES PROGRESSIVE JOINT CONNECTIVE DEGENERATION OR DEGRADATION [**SPONDYLOSIS**] WITH ACCOMPANYING **NEUROPATHY** THROUGH **LACK OF JOINT CARTILAGE PRODUCTION AND HYDRATION OF THE JOINT**.

IMPULSE CAVITATION ADJUSTING DISRUPTS THE SCAR TISSUE, REINSTATES JOINT MOBILITY, ALLOWS THE JOINT TO REGENERATE AND REVERSES THE **SPONDYLOSIS** AND RESULTANT **NEUROPATHY**.

SPONDYLOSIS ORDINARILY FOLLOWS A GRADUAL RELAPSING AND REMITTING COURSE THAT IS **SILENT UNLESS AND UNTIL SYMPTOMS**

ARE PRECIPITATED BY AN INCIDENT *OFTEN SO MINOR THAT IT PASSES UNNOTICED BY THE PATIENT*.

ALL GRADATIONS OF SPONDYLOSIS CAN EXIST, BUT **EARLY OR INCIPIENT SPONDYLOTIC CHANGES**, EVEN WHEN UNSUSPECTED, CAN NEVERTHELESS **IRRITATE AND UPSET FUNCTION IN THE SEGMENTAL NERVE**.

ACUTE INJURY TO A HEALTHY NERVE HAS NO PROLONGED DISCHARGE OF PAIN SIGNALS, WHEREAS THE SAME INJURY TO A **NEUROPATHIC** NERVE **CAN CAUSE A SUSTAINED DISCHARGE**.

FOR PAIN **TO BECOME PERSISTENT** AFFECTED FIBERS **MUST BE PREVIOUSLY IRRITATED OR DEFECTIVE**, WHICH IS WHY SOME PEOPLE **DEVELOP SEVERE PAIN AFTER AN APPARENTLY MINOR INJURY** AND WHY **THAT PAIN CAN CONTINUE BEYOND A REASONABLE PERIOD**.

MANIFESTATIONS OF **NEUROPATHIC** DYSFUNCTION ARE:

- **MOTOR**
- **SENSORY**
- **AUTONOMIC**

BRIEF TRANSIENT MOTOR MANIFESTATIONS ARE THE FIRST TO APPEAR AND **RADICULOPATHY *CAN OCCUR WITHOUT PAIN***.

MUSCLE SHORTENING IS **AN EARLY AND REGULAR FEATURE OF RADICULOPATHY** BECAUSE LARGE DIAMETER NERVE FIBERS AT THE NERVE ROOT [AXONS OF MOTONEURONS AND MYELINATED PRIMARY AFFERENTS-MUSCLE PROPRIOCEPTORS] ARE THE FIRST TO SUFFER PHYSICALLY.

PAINLESS REVERSIBLE TIGHT **MUSCLE KNOTS** CAN BE FELT IN MOST INDIVIDUALS.

PAIN ISN'T THEREFORE A FEATURE OF **RADICULOPATHY** UNLESS NOCICEPTIVE PATHWAYS ARE INVOLVED.

MANY **NEUROPATHIES** ARE PAIN FREE, SUCH AS:

- SUDOMOTOR HYPERACTIVITY IN **HYPERHYDROSIS**
- **MUSCLE WEAKNESS** IN VENTRAL ROOT DISEASE

DEGRADATION OF COLLAGEN

NEUROPATHY CONTRIBUTES TO DEGENERATIVE CONDITIONS, INCLUDING **SPONDYLOSIS**.

NEUROPATHY **DEGRADES THE QUALITY OF COLLAGEN CAUSING IT TO HAVE FEWER CROSS-LINKS, MAKING IT MORE FRAIL THAN NORMAL COLLAGEN**.

THE AMOUNT OF COLLAGEN IN SOFT AND SOFT AND SKELETAL TISSUES IS ALSO REDUCED AND BECAUSE COLLAGEN LENDS STRENGTH TO LIGAMENT, TENDON, CARTILAGE AND BONE, **NEUROPATHY** CAN EXPEDITE DEGENERATION IN WEIGHT-BEARING AND ACTIVITY-STRESSED PARTS OF THE BODY, INCLUDING THE SPINE AND JOINTS, BECOMING A SOURCE OF PAIN.

ENTHESOPATHIC THICKENING IN TENDONS ARE POSSIBLY CONPENSATIONS FOR THIS WEAKNESS.

CENTRAL MECHANISMS IN NEUROPATHIC PAIN

INTERACTIONS BETWEEN **PERIPHERAL AND CENTRAL MECHANISMS** OCCUR TO PRODUCE POSTINJURY HYPERSENSITIVITY AND **NEUROPATHIC** PAIN.

THE SPINAL CORD ISN'T JUST A PASSIVE CONVEYER OF PERIPHERAL SENSATION TO THE BRAIN BECAUSE IT ***CAN MODIFY OR AMPLIFY INCOMING SIGNALS***.

CENTRAL SENSITIZATION [**A STATE OF HYPEREXCITABILITY OF THE DORSAL HORN NEURON**] CAN OCCUR AFTER DAMAGE TO A PERIPHERAL NERVE [SPONDYLOSIS] OR LOW-FREQUENCY REPETITIVE C FIBER NOICEPTOR INPUT [FROM PERIPHERAL TISSUE INFLAMMATION, AS IN ARTHRITIS].

CENTRAL SENSITIZATION IS FROM:

- INCREASED SPONTANEOUS ACTIVITY OF DORSAL HORN NEURONS
- INCREASED RESPONSE TO AFFERENT INPUT
- EXPANSION OF RECEPTIVE FIELD SIZE
- REDUCTION IN THRESHOLD
- PROLONGED AFTERDISCHARGES

CENTRAL SENSITIZATION LEADS TO ***A CASCADE OF MOLECULAR EVENTS*** SUCH AS:

- ACTIVATION OF THE N-METHYL-D-ASPARTATE [NMDA] CHANNEL
- INCREASE IN INTRACELLULAR CA^{2+}
- WIND-UP/WIDE DYNAMIC RANGE [WDR] NEURON SENSITIZATION
- OTHER PHENOMENA

ALTERED SENSITIVITY IN THE DORSAL HORN

CENTRAL SENSITIZATION MAY BE MAINTAINED BY ONGOING PRIMARY AFFERENT INPUT, ALTERED CENTRAL NEURAL CIRCUITRY OR BOTH.

WOOLF IDENTIFIED FOUR STIMULUS-PROCESSING STATES:

- **NORMAL**: TOUCH IS PERCEIVED AS INNOCUOUS AND HIGH-INTENSITY STIMULATION AS PAIN.
- **SUPPRESSED**: HIGH-INTENSITY STIMULATION ISN'T PAINFUL BECAUSE OF INHIBITION FROM SEGMENTAL INHIBITION OR DESCENDING INHIBITION FROM HIGHER CENTERS

- **SENSITIZED**: LOW-INTENSITY STIMULATION IS PERCEIVED AS PAINFUL MECHANICAL **ALLODYNIA** AND HIGH-INTENSITY STIMULATION WHICH IS NORMALLY PAINFUL AND LEADS TO HYPERALGESIA.
- **REORGANIZED**: POSSIBLE STRUCTURAL CHANGES AND REORGANIZATION OF DORSAL HORN CIRCUITRY IN WHICH INAPPROPRIATE SYNAPSES MAY FORM BY **SPROUTING** CAUSING LOW-THRESHOLD AFFERENT INPUT TO BE MISINTERPRETED AS PAIN. A-β AFFERENTS ACQUIRE THE CAPACITY AFTER INFLAMMATION TO PRODUCE CENTRAL EXCITABILITY, WHICH THEY CAN'T NORMALLY DO.

EXPANSION OF RECEPTIVE FIELD SIZE

PAIN ***RADIATING SEVERAL SEGMENTS ABOVE AND BELOW THE LEVEL OF NOCICEPTIVE STIMULATION*** MAY BE EXPLAINED BY DISPERSION OF THE PRIMARY AFFERENT INPUT THROUGH PROPRIOSPINAL CONNECTIONS IN ADJACENT LAYERS 5 AND 6 OF THE DORSAL HORN.

THIS AREA ALSO CONTAINS **WDR NEURONS** [CALLED THIS BECAUSE THEY CAN ENCODE A RANGE OF STIMULI FROM LIGHT TOUCH TO INTENSE PAIN].

THE WDR RECEPTIVE FIELD IS IMMENSE COMPARED WITH THAT OF THE PRIMARY AFFERENT NEURON AND ANY INCREASE IN NOCICEPTIVE STIMULATION CAN LEAD TO THE RECRUITMENT OF MANY MORE WDR NEURONS.

PROLONGED AFTER DISCHARGES

PERCEIVED PAIN CAN OUTLAST THE STIMULUS.

IN NEUROPATHY DISCHARGE FROM PAIN FIBERS GENERATES PROLONGED ACTIVITY OF WDR NEURONS BECAUSE NORMAL INHIBITORY EFFECTS OF NERVE ACTIVITY IS LOST.

WIND-UP

WIND-UP PHENOMENA IS ***FREQUENCY DEPENDENT***.

LOW FREQUENCY INPUT [0.1 HZ] GIVES A CONSTANT RESPONSE FROM DORSAL HORN NEURONS.

FREQUENCIES GREATER THAN 0.5 HZ CAN GIVE RISE TO HYPEREXCITABILITY LASTING ***MANY MINUTES AFTER THE STIMULUS***.

C FIBERS RELEASE CHEMICAL SUBSTANCES ONTO DORSAL HORN NEURONS.

THERE ARE TWO TYPES OF RECEPTORS AT THE DORSAL HORN:

- NEUROKININ
- NMDA FOR AMINO ACIDS

BINDING OF AMINO ACIDS TO THE NMDA RECEPTOR DEPENDS ON ITS PRIOR ACTIVATION BY THE BINDING OF SUBSTANCE P TO THE NEUROKININ RECEPTOR SO RELEASE OF SUBSTANCE P MAY LEAD TO RECRUITMENT OF ***A SECOND RECEPTOR TYPE*** [NMDA] AND **EXAGGERATED RESPONSE TO FURTHER STIMULATION**.

THE SENSITIZED CELL UNDERGOES OTHER BIOCHEMICAL CHANGES AS INDICATED BY EXPRESSION OF THE **GENE** ***c-fos***.

PRODUCTS OF ***c-fos*** EXPRESSION ARE INVOLVED IN **REGULATION OF NEUROTRANSMITTER AND NERVE GROWTH FACTOR SYNTHESIS**.

CHALLENGES IN DIAGNOSIS AND TREATMENT

DIAGNOSING PAIN AND DYSFUNCTION CAUSED BY **RADICULOPATHY DEPENDS ALMOST ENTIRELY ON** THE EXAMINER'S **CLINICAL EXPERIENCE AND ACUMEN**.********

- HISTORY GIVES LITTLE ASSISTANCE.

- **PAIN OFTEN ARISES SPONTANEOUSLY** WITH **NO HISTORY OF TRAUMA** OR ELSE ***THE DEGREE OF REPORTED PAIN FAR EXCEEDS THAT CONSISTENT WITH THE INJURY***.
- LAB AND IMAGING ARE GENERALLY NOT HELPFUL.
- THERMOGRAPHY REVEALS **DECREASED SKIN TEMPERATURE IN AFFECTED DERMATOMES**, AN INDICATION OF **NEUROPATHY**, BUT DOESN'T NECESSARILY SIGNIFY PAIN.
- **RADICULOPATHIES** ARE ***DIFFICULT TO DOCUMENT WITH ROUTINE NERVE CONDUCTION STUDIES*** WHICH MEASURE ONLY THE FEW FASTEST CONDUCTING AND LARGEST FIBERS AND TAKE NO ACCOUNT OF THE MAJORITY OF SMALLER FIBERS.
- IN ***FOCAL*** **NEUROPATHY** NERVE CONDUCTION VELOCITIES REMAIN WITHIN THE WIDE RANGE OF NORMAL VALUES, BUT F-WAVE LATENCY MAY BE PROLONGED.
- ELECTROMYOGRAPHY ISN'T SPECIFIC.

PHYSICAL SIGNS OF **NEUROPATHY** ARE DISTINCTIVE AND DIFFERENT FROM WELL-KNOWN ONES OF OUTRIGHT DENERVATION, SUCH AS **LOSS OF SENSATION AND REFLEXES**.

THEY ARE IMPORTANT TO LOOK FOR BECAUSE THEY INDICATE EARLY NEURAL **DYSFUNCTION FOR WHICH NO SATISFACTORY LAB OR IMAGING TEST EXISTS**.

CAREFUL INSPECTION FOR SIGNS OF MOTOR, SENSORY AND **SUTONOMIC** [**VASOMOTOR**, **SUDOMOTOR** AND **PILOMOTOR**] DYSFUNCTION IN THE **SKIN** AND AFFECTED MUSCLES IS NECESSARY.

VASOCONSTRICTION DIFFERENTIATES **NEUROPATHIC** PAIN FROM INFLAMMATORY PAIN: IN **NEUROPATHIC** PAIN, AFFECTED PARTS ARE **PERCEPTIBLY COLDER**.

THERE MAY BE INCREASED SUDOMOTOR ACTIVITY AND THE PILOMOTOR REFLEX IS OFTEN HYPERACTIVE AND VISIBLE IN AFFECTED DERMATOMES AS **GOOSE BUMPS**.

THERE CAN BE INTERACTION BETWEEN PAIN AND AUTONOMIC PHENOMENA:

- A STIMULUS SUCH AS **CHILLING** [STIMULATES THE PILOMOTOR RESPONSE] **CAN PRECIPITATE PAIN**.
- CONVERSELY, PRESSURE ON A TENDER MOTOR POINT CAN PROVOKE PILOMOTOR AND SUDOMOTOR REFLEXES.

INCREASED PERMEABILITY IN BLOOD VESSELS CAN LEAD TO LOCAL **SUBCUTANEOUS TISSUE EDEMA** [NEUROGENIC EDEMA OR TROPHEDEMA], SEEN AS ***PEAU D'ORANGE*** SKIN AND CONFIRMED BY THE **MATCH STICK TEST**.

TROPHEDEMA IS **NONPITTING TO DIGITAL PRESSURE**, BUT WHEN A BLUNT INSTRUMENT SUCH AS **THE END OF A MATCH STICK** IS USED **THE INDENTATION PRODUCED IS CLEAR-CUT AND PERSISTS FOR MANY MINUTES**, A QUICK AND SIMPLE TEST THAT CAN DEMONSTRATE **NEUROPATHY** EARLIER THAN ELECTROMYOGRAPHY.

TROPHIC CHANGES SUCH AS **DERMATOMAL HAIR LOSS** MAY ALSO ACCOMPANY **NEUROPATHY**.

NEUROPATHIC CHANGES ARE **PRIMARILY IN MUSCLE** AND EVEN WHEN SYMPTOMS APPEAR TO BE IN JOINTS OR TENDONS, SIGNS IN **MUSCLES ARE THE MOST CONSISTENT AND RELEVANT**:

- INCREASED MUSCLE TONE
- TENDERNESS OVER MOTOR JOINTS
- **TAUT AND TENDER, PALPABLE CONTRACTURE BANDS**
- **RESTRICTED JOINT RANGE**

EACH CONSTITUENT MUSCLE MUST BE PALPATED AND ITS CONDITION NOTED.

PALPATION REQUIRES DETAILED KNOWLEDGE OF ANATOMY AND CLINICAL **SKILL** ***COMES ONLY WITH PRACTICE***.

BECAUSE MANY **PARASPINAL MUSCLES** ARE COMPOUND [SUCH AS THE LONGISSIMUS] AND EXTEND THROUGHOUT MOST OF THE LENGTH OF THE VERTEBRAL COLUMN, ***THE ENTIRE SPINE MUST BE EXAMINED*** **EVEN WHEN SYMPTOMS ARE LOCALIZED TO ONE REGION**.

MUSCLE SHORTENING FROM CONTRACTURE

MUSCLE CONTRACTURE IS A FUNDAMENTAL FEATURE OF MUSCULOSKELETAL PAIN AND OF ALL STRUCTURES THAT CAN DEVELOP SUPERSENSITIVITY, THE MOST WIDESPREAD IS **STRIATED MUSCLE**.

CONTRACTURE CAN PHYSICALLY GIVE RISE TO PAIN BY ***ITS RELENTLESS PULL ON SENSITIVE STRUCTURES***.

CLASSIC CONTRACTURE REFERS TO THE EVOKED SHORTENING OF A MUSCLE FIBER **IN THE ABSENCE OF ACTION POTENTIALS**.

ACCORDING TO **CANNON** AND **ROSENBLUETH** SKELETAL MUSCLE CAN BECOME SUPERSENSITIVE IN SEVERAL WAYS:

- **INCREASED SUSCEPTIBILITY**: LESSENED STIMULI THAT DON'T HAVE TO EXCEED A THRESHOLD CAN PRODUCE RESPONSES OF NORMAL AMPLITUDE
- **HYPEREXCITABILITY**: THE THRESHOLD OF THE STIMULATING AGENT IS LOWER THAN NORMAL
- **SUPERREACTIVITY**: THE CAPACITY OF THE MUSCLE TO RESPONSE IS AUGMENTED
- **SUPERDURATION OF RESPONSE**: THE AMPLITUDE OF RESPONSE IS UNCHANGED BUT ***ITS TIME COURSE IS PROLONGED***

SUPERSENSITIVE SKELETAL MUSCLE FIBERS OVERREACT TO A WIDE VARIETY OF CHEMICAL AND PHYSICAL INPUTS, INCLUDING **STRETCH AND PRESSURE** AND HAVE A LOWERED THRESHOLD TO ACETYLCHOLINE, ITSELF INCREASED FROM REDUCED LEVELS OF ACETYLCHOLINESTERASE.

ACETYLCHOLINE SLOWLY DEPOLARIZES SUPERSENSITIVE MUSCLE MEMBRANE, INDUCING AN ELECTROMECHANICAL COUPLING IN WHICH **TENSION DEVELOPS SLOWLY WITHOUT GENERATING ACTION POTENTIALS**.

IN **NORMAL MUSCLE** ACETYLCHOLINE ACTS ONLY AT RECEPTORS THAT ARE SITUATED IN THE NARROW ZONE OF INNERVATION.

IN **NEUROPATHY** IT ACTS AT **NEWLY FORMED EXTRAJUNCTIONAL RECEPTORS** [**HOT SPOTS**] THAT **APPEAR THROUGHOUT THE MUSCLE**.

TREATMENT

PHARMACOLOGIC MANAGEMENT OF **NEUROPATHIC** PAIN IS DIFFICULT AND ***THERE IS NO LONG-TERM DATA TO SUPPORT THE EFFECTIVENESS OF ANY DRUG IN TREATING THIS CONDITION***.

PHYSICAL MEDICINE

NEUROPATHIC PAIN IS **A SUPERSENSITIVITY PHENOMENON** AND ITS TREATMENT REQUIRES DESENSITIZATION.

SUPERSENSITIVITY AND OTHER FEATURES OF DENERVATED MUSCLE CAN BE REVERSED BY ELECTRIC STIMULATION.

PHYSICAL MEDICINE ALSO ACHIEVES ITS EFFECT BY STIMULATION.

LOCAL THERAPY EXCITES RECEPTORS IN **SKIN** AND **MUSCLE**, FOR EXAMPLE:

- **MASSAGE** ACTIVATES TACTILE AND PRESSURE RECEPTORS
- **EXERCISE**, **MANIPULATION** AND **DRY NEEDLING** STIMULATE MUSCLE SPINDLES AND GOLGI ORGANS
- **HEAT** AND **COLD** ACT ON THERMAL RECEPTORS

THESE ARE SENSED BY SPECIFIC RECEPTORS, TRANSDUCED INTO NERVE IMPULSES AND RELAYED TO THE DORSAL HORN.

ALL FORMS OF PHYSICAL MEDICINE, INCLUDING **DRY NEEDLING ARE EFFECTIVE ONLY WHEN THE NERVE** TO THE PAINFUL PART **IS STILL INTACT**.

REMOVING THE CAUSE OF NEUROPATHY IS KEY TO TREATMENT

SPONDYLOSIS IS BY FAR THE MOST COMMON CAUSE OF ***RADICULOPATHY***.

LOCAL TREATMENT SUCH AS ***MANIPULATION***, MASSAGE AND DRY NEEDLING SHOULD BE GIVEN TO ALL TENDER AND SHORTENED MUSCLES IN AFFECTED MYOTOMES, ***INCLUDING PARASPINAL MUSCLES***.

OUTCOMES OF TREATMENT DEPEND ON **APPROPRIATENESS OF TYPE OF THERAPY** USED AND **SKILL OF THE PRACTITIONER**.

FINE, FLEXIBLE ACUPUNCTURE NEEDLES USED IN INTRAMUSCULAR STIMULATION FIND AND RELEASE CONTRACTURES AND DEEP CONTRACTURE CAN ONLY BE RELEASED BY **PROBING WITH A NEEDLE**, WHICH **TRANSMITS FEEDBACK INFORMATION ON THE NATURE AND CONSISTENCY OF THE TISSUES ITS IS PENETRATING**.

WHEN PENETRATING NORMAL MUSCLE IT MEETS WITH LITTLE RESISTANCE, WHEN PENETRATING A ***CONTRACTURE*** THERE IS ***FIRM***

RESISTANCE AND **THE NEEDLE IS *FIRMLY GRASPED* BY THE MUSCLE**, CAUSING THE PATIENT TO **FEEL A PECULIAR CRAMPLIKE OR GRABBING SENSATION** FROM THE **SHORTENED MUSCLE** [WHICH **GRADUALLY EASES OFF DURING TREATMENT AS MUSCLE SHORTENING IS RELEASED** IN ***MINUTES***] REFERRED TO IN **ACUPUNCTURE LITERATURE** AS THE ***DEQI*** OR ***TEH CH'I*** **RESPONSE**, WHICH RESPONSE IS AN IMPORTANT FINDING, A SIGN OF MUSCLE CONTRACTURE CONFIRMING THE STATUS OF **NEUROPATHY**.

THE MUSCLE IS SHORTENED AND **NEUROPATHIC**.

ACCORDING TO FIELDS, THE STRANGE QUALITY OF **NEUROPATHIC** PAIN [A **PECULIAR CRAMPLIKE QUALITY**] PROBABLY RESULTS FROM DISRUPTION OF THE SENSORY APPARATUS SO THAT A NORMAL PATTERN OF NEURAL ACTIVITY IS NO LONGER TRANSMITTED TO THE PERCEPTUAL CENTERS.

ALLOWING THAT **NEUROPATHIC** PAIN PROBABLY ACTIVATES NOCICEPTIVE NEURONS BECAUSE THE MESSAGE THAT GETS THROUGH TO THE PERCEPTUAL CENTERS IS CLEARLY UNPLEASANT, BUT ***PATIENTS DISTINGUISH THEM FROM NORMAL PAIN SENSATIONS***.

CHRONIC MYOFASCIAL PAIN SENSATIONS ARE NOT NORMAL IN THAT THEY ARE ASSOCIATED WITH ***RECEPTORS THAT*** **SENSE MUSCLE SHORTENING** [**PROPRIOCEPTORS**].

PROGRESSIVE TACTILE HYPERSENSITIVITY

MA AND **WOOLF** DESCRIBED PROGRESSIVE TATILE HYPERSENSITIVITY, FINDING THAT REPEATED **LIGHT TOUCH** TO **AN INFLAMED AREA** PRODUCES **CUMULATIVE ALLODYNIA** PERSISTING FOR ***HOURS***, DEMONSTRATING THE CAPACITY TO PRODUCE **WIND-UP** OF SPINAL CORD NEURONS.

THIS IS DISTINCT FROM **CENTRAL SENSITIZATION** INDUCED BY STIMULATION INDUCED IN **NONINFLAMED TISSUE** LASTING ONLY FOR ***MINUTES***.

CUTANEOUS NERVES CONTRIBUTE BY **HILTON'S LAW OF PHYSIOLOGY** BECAUSE **THE SAME NERVES CONTROL BOTH**.

IN CHRONIC PAIN **FIBROSIS** EVENTUALLY **BECOMES A MAJOR FEATURE ON THE CONTRACTURE** AND DRY-NEEDLE TREATMENT IS MUCH LESS DRAMATIC.

THE EXTENT OF FIBROSIS DOESN'T CORRELATE WITH CHRONOLOGIC AGE AND ***SCARRING*** CAN OCCUR AFTER INJURY OR SURGERY.

THE TREATMENT OF **EXTENSIVE FIBROTIC CONTRACTURES** NECESSITATES **MORE FREQUENT AND EXTENSIVE NEEDLING** TO RELIEVE PAIN IN ALL TENDER BANDS, AND IT'S UNCOMMON TO ENCOUNTER A MUSCLE THAT IS TOTALLY FIBROTIC AND CANNOT BE RELEASED BY **VIGOROUS NEEDLING**.

FOR LONG-LASTING PAIN RELIEF AND **RESTORATION OF FUNCTION**, IT'S ESSENTIAL TO **RELEASE SHORTENED PARASPINAL MUSCLES** THAT MAY BE ***COMPRESSING A DISK*** AND **DISPERSE FIBROTIC TISSUE** THAT IS ***ENTRAPPING A NERVE ROOT***.

SUMMARY

- MANY **NEUROPATHIC** CONDITIONS ARE PAIN FREE.
- BECAUSE PAIN IS A MANIFESTATION OF **NEUROPATHY**, THERAPY SHOULD AIM AT THE CAUSE OF THE **NEUROPATHIC** CONDITION.
- **SPONDYLOSIS** IS BY FAR THE MOST COMMON CAUSE OF **RADICULOPATHY**.
- MYOFASCIAL PAIN SYNDROMES ARE ALMOST INVARIABLY **SEGMENTAL**, FOUND IN ***DERMATOMES***, **MYOTOMES** AND

SCLEROTOMES AND EXAMINATION **MUST ALWAYS INCLUDE THE SPINE**.

- LOOK FOR **VASOCONSTRICTION** AND **TROPHEDEMA** AS SIGNS OF **NEUROPATHY** AS DISTINGUISHING IT FROM **DENERVATION**.
- **NEUROPATHIC** PAIN HAS A **PROPRIOCEPTIVE** COMPONENT AND **CANNOT EXIST WITHOUT MUSCLE SHORTENING**: TENDER, SHORTENED MUSCLES IN MYOTOMES.
- **NEUROPATHIC** PAIN IS OFTEN THE UNSUSPECTED CAUSE OF MANY OTHER CONDITIONS SUCH AS **TENSION HEADACHE**, **FROZEN SHOULDER**, **TENNIS ELBOW** AND **LOW BACK PAIN**. MUSCLE SHORTENING **UPSETS JOINT ALIGNMENT** AND INCREASES PRESSURE ON **ARTICULAR SURFACES**, **DEGRADING** THE QUALITY OF **COLLAGEN** AND CONTRIBUTING TO DEGENERATION IN WEIGHT-BEARING AND ACTIVITY-STRESSED PARTS OF THE BODY.
- **RADICULOPATHY** IS PERPETUATED WHEN **SHORTENED PARASPINAL MUSCLES DRAW ADJACENT VERTEBRAE TOGETHER TO *COMPRESS THE DISC*** AND ***IRRITATE THE NERVE ROOT*** AND THE VICIOUS CYCLE ***MUST BE TREATED AT THE SPINE***.
- PROPERLY TREATED, THE SIGNS DISAPPEAR, OFTEN WITHIN ***MINUTES***.

EXPLORATORY LAPAROTOMY OR LAPAROSCOPY MAY STRETCH, AVULSE, OR OTHERWISE DAMAGE EITHER THE ILIOINGUINAL OR ILIOHYPOGASTRIC NERVES OR BOTH NERVES.

A **NEUROPATHY** MAY RESULT AND **DEVELOP AFTER VARIABLE INTERVALS**.

THE PAIN MAY BEGIN DAYS, WEEKS OR EVEN MONTHS ***AFTER*** THE INJURY AND IT'S COMMON THAT THERE IS A ***GRADUAL ESCALATION OF PAIN OVER TIME***.

THIS TYPE OF **NEUROPATHIC** PAIN IS USUALLY BURNING OR STABBING AND **MAY INCAPACITATE THE PATIENT** AND MAY INVOLVE ***CUTANEOUS NERVE ENTRAPMENT*** OR **INJURY** IN DIAGNOSIS OF CHRONIC LOWER ABDOMINAL PAIN FOLLOWING SURGICAL EXPLORATION.

SUCH AN INJURY CAN ALSO FOLLOW **TRAUMA**, **AUTO ACCIDENTS** OR **EXERCISE**.

OTHER DESCRIPTIVES INCLUDE **STABBING**, **CRAMPY**, **BURNING** OR **ACHING** PAIN WHICH **MAY OR NOT RADIATE** TO **UPPER THIGH**, **LABIA** OR **SCROTUM**.

EXAMINATION INCLUDES THE DIRECT PALPATION OF THE LOCAL AREA **WITH THE BLUNT END ON A PENCIL OR PEN** WITH PRESSURE DISTRIBUTED DIRECTLY VERTICAL DOWNWARD.

IMMEDIATE RELIEF IS THE USUAL REACTION TO DRY NEEDLING.

ILIOINGUINAL AND ILIOHYPOGASTRIC NERVE DISTRUBANCES OFTEN HAVE SURGICAL OR OTHER TYPES OF **TRAUMA** IN THE AREA OF THE **LOWER ABDOMINAL WALL**.

THE GENESIS OF THE PAIN ISN'T KNOWN, BUT SUSPECTED RETRACTION PLACED ON ***NERVES AROUND THE INCISION LINE*** THAT MAY RESULT IN **OVERSTRETCHED** AND **AVULSIVE-TYPE** ***NEURAL INJURIES***.

GENITOFEMORAL NERVE DISORDERS ARE **NEUROPATHIES** WITH REPORTS OF LOW ABDOMINAL PAIN OR BACK PAIN THAT HAS

MIGRATED TO THE FRONT OF THE BODY DESCENDING INTO THE SCROTAL OR LABIAL AREA.

TENDER POINT **DRY NEEDLING** USUALLY PROVIDES PAIN RELIEF.

- CHRONIC PELVIC PAIN DUE TO **NEUROPATHY**
- PUDENDAL **NEUROPATHY**
- ILIOINGUINAL AND ILIIOHYPOGASTRIC **NEUROPATHY**
- GENITOFEMORAL **NEUROPATHY**
- HYMENAL **NEUROPATHY**
- SYMPATHETIC PELVIC **NEUROPATHY**
- SCIATIC AND OBTURATOR **NEUROPATHY**
- INTERCOSTAL **NEUROPATHY**
- LOWER EXTREMITY **NEUROPATHY**
- UPPER LIMB NECK PAIN **NEUROPATHY**
- PARASYMPATHETIC **NEUROPATHY**

NEUROPATHIC PAIN **IS A COMPLEX BIOLOGICAL PHENOMENON WITH MANY COMPONENTS**

ABNORMAL AFFERENT BARRAGES THAT PROPAGATE INTO THE ***CENTRAL NERVOUS SYSTEM*** DIRECTLY **WILL ELICIT PARAESTHESIAS**, **DYSESTHESIAS** AND **PAIN**, **BURNING PAIN** AND **INTERMITTENT SPONTANEOUS BURSTS**.

PAINFUL SCARS CAN BE SURROUNDED BY TOUCH-EVOKED **ALLODYNIA** RADIATING TO VARIOUS AREAS DUE TO **CENTRAL SENSITIZATION** FROM THE AREA OF THE MOST SEVERE **NEUROPATHIC** PAIN.

NERVE BLOCK/DRY NEEDLING OF THE SCAR FROM WHICH THE NOXIOUS INPUT ORIGINATED AND **TRIGGERED A SPINAL CENTRAL**

SENSITIZATION ELIMINATES THE ALLODYNIA, SCAR PAIN, DYSESTHESIAS AND PARESTHESIAS.