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WIDENING OUR TREATMENT OPTIONS: UNDERSTANDING THE ROLE OF CONNECTIVE TISSUE IN POST MASTECTOMY PAIN.

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INTRODUCTION.

Human design: We started our lives as hunter-gatherers many years ago. To survive out there we had to make a few evolutionary adaptations to turn us into the ultimate survival machine. Moving from a tree-dwelling species to life on the open planes as hunters, survival depended on changes in the way man could move and develop the capacity to use tools. We started running around on 2 legs. This is much more economic than running around on 4 and uses less energy. It freed our arms and hands to manipulate tools, drink, and feed while running and to manipulate our environment. Our higher posture, mobile necks, and front facing eyes, made scanning our environment a lot more effective to see danger coming and to hunt. The culmination of man's evolution was Homo sapiens. Straight and tall, muscular, hardened and practical, modern man became the ultimate predator after 4 million years of evolution. Intelligent, economic movement became our exclusive successful evolutionary adaptation for survival.

Survival on the planes was tough**.** If you don't move, you don't survive. The basic functional design of the human body is therefore to move in order to survive. Our entire anatomy and physiology functions towards this end. Any injury or disease that compromises our ability to move is perceived as a threat to survival, and must be attended to as quickly and effectively as possible. Our entire wound healing process and capacity to recover is well designed to get us back to optimal functioning. **Pain** is the body's early warning system of damage or potential tissue damage and functions to help us make the necessary adjustments to survive. If dysfunction or poor repair interferes with movement, the neural system will constantly be in a state of alert, warning us of impending danger. This is often perceived as chronic pain or neurogenic pain.

Even as the modern domesticated Homo sapiens sapiens, we still depend on our exceptional movement ability and capacity to survive. Our modern workplace is alien to our design. Our constant bad movement habits, the uneconomical use of our working environment, as well as the fact that another subspecies – Homo sedens – is developing, puts the body under constant strain. This leads to a diminished movement capacity, triggering the neural warning systems ending as a painful dysfunctional body with the danger of not surviving in the world out there.

The body shouts: *"Give me back my ability to move, and I will reward you greatly with less pain!'*

What does all this have to do with breast cancer treatment? EVERYTHING! Treatment for breast cancer is an unscheduled, forced interference with our ability to move the body. If we cannot move well after the treatment, our very survival as an individual is at risk. This is the way we have been genetically wired since the day we started as a species. Anxiety and fear influences the way we deal with the potential threat to survival, and therefore influences the way we deal with the pain in the short as well as the longer term. This becomes even more critical when the pain becomes long term or chronic in nature.

POST MASTECTOMY COMPLAINTS

INTRODUCTION

Complaints after surgery and treatment for breast cancer are a lot more common than what is generally known as the Post Mastectomy Pain Syndrome (PMPS). In the literature relating to post breast cancer surgery, the most common complaints seem to be lymphoedema, impaired shoulder motion, muscle weakness, arm pain, and altered skin sensations. (Kelley 1998) Lymphoedema is a problem in 15-20 % of patients. (Chau 2002)

POST MASTECTOMY PAIN SYNDROME.

When dealing with the Post Mastectomy Pain Syndrome, the definition is based on three criteria: character of the pain, location of the pain, and timing of the pain. (Smith et al 1999) The pain should be typical of neuropathic pain with unpleasant and peculiar sensations described in the categories of numbness, pins and needles, burning or stabbing; it should be located in the axilla, arm shoulder, or chest wall of the side of the surgery; and should persist beyond the normal healing time of three months. (Smith et al 1999, Grady 2001) Various studies have put this syndrome at between 20 and 72 percent. (Stevens et al 1995; Carpenter et al 1998; Smith et al 1999; Guttrup et al 2000)

The time of onset of symptoms of PMPS is variable, and the frequency of symptoms range from continuous to a painful spell once per month. A large variety of terms are used to describe the pain which include tingling, stabbing, numbness, pins and needles, like threads pulling, and burning. The pain is aggravated by straining, sudden movements, tiredness, clothes rubbing, cold weather, by coughing, overuse, lack of movement or rest, and by lying on or putting pressure on the arm. Sufferers of this syndrome report medication, rest, heat, ice, exercise or movement, elevation, massage, physical support, holding the arm, or miscellaneous alternatives such as prayer, drinking alcohol or not wearing a brassiere as alleviating factors for their pain. (Smith et al 1999; Carpenter et al 1998)

The pain can be debilitating and affect activities of daily living, relationships, and sleep. The most striking risk factor for PMPS is age, decreasing from 65% in the youngest age group to 26% in the over 70 year olds. Patients reporting PMPS were more likely to have received pre-operative chemotherapy and post-operative radiotherapy than those who did not report PMPS. (Smith et al 1999)

OTHER COMPLAINTS

Outside this definition there are other recognised neuropathic pains occurring after surgery for breast cancer like phantom breast pain, scar pain, painful neuromata, and less specific neuropathic pain caused by radiotherapy effect on nerves. Associated pain such as adhesive capsulitis of the ipsilateral shoulder, carpal tunnel syndrome, painful arm oedema can also occur. (Grady 2001) The extent of the tissue injury cannot totally explain certain other painful syndromes such as headaches, neck-aches, backaches and varying degrees of suffering experienced by patients.

One of my main concerns about the treatment of patients with post mastectomy complaints is how often the literature in medical texts refers to physical modalities as a treatment option. They name them to include exercise, immobilisation, transcutaneous electrical nerve stimulation, acupuncture and the use of superficial heat, cold, massage or vibration. They then add that "these non-invasive techniques are easily taught, MAY (my emphasis) help patients to relax, relieve muscle spasm or distract them from their pain, and provide a means for patient-family participation". (MacDonald et al 1998)

In a report by the Maher Committee reporting on the experience of members of the Radiation Action Group Exposure (RAGE), some of their answers to a questionnaire raised concern. Of the 60% of women that have received Physiotherapy, only 20% found it effective. 25% received acupuncture with only 10% finding it effective. Medication was equally ineffective with 50% getting no relief at all. 10% sometimes get relief and only 40% found their medication effective (these tended to be the women on opiates). (Maher Committee 1995)

Damage to the patient is done on several levels:

- Mechanical damage by destruction of anatomical structures involved in movement. Important movement planes for function are damaged
- Damage to the vascular bed between fascial layers. This regenerates but takes time and may even stay compromised
- Neural damage to afferents from the skin and fasciae involved in amongst others proprioception
- Damage to bigger vascular vessels due to ligation during surgery

- Radiation further damages vascular beds in fasciae, muscle and skin (even in normal areas)
- This gives rise to fibrosis in supporting structures compromising neural feedback and function
- Freedom of normal movement diminishes due to compromised elasticity in muscles, scarring, and destruction of movement planes.

Treatment for this kind of damage is more effective on the level of physical modalities rather than on the pharmacological. For this reason I propose a movement model upon which to base clinical reasoning and treatment planning when faced with post mastectomy pain and dysfunction.

NEW MOVEMENT MODEL

The concept of grouping anatomic structures into layers is not new in surgery or anatomy. This technique aids the surgeon or anatomist by identifying safe tissue planes that allow atraumatic dissection or surgical approaches to deeper structures. It seems a logical step to also recognise these tissue planes functionally within their role in movement and movement quality. This is a natural progression from our basic anatomical model of bone, muscle, joint, ligament, and nerve that has become inadequate to understand the complexity of normal (and pathological) human movement. The concept of "movement planes" and the grouping of anatomical structures by fascial sheets and planes lead to a better understanding of the interrelationships of structures and their differing role in normal movement. Understanding the ***interrelationship*** of structures, and the role of fasciae in controlled movement is the base upon which the Movement Model is built.

Rotation

All movement in the human body is rotation around a movement axis in a joint or group of joints at all times. The ligamentous and capsular supporting structures around a joint guide this rotation in turn. It is, however, not the integrity of the joint and its supporting structures that determine the **quality** of the movement, freedom to rotate around an axis is determined by **ALL** the soft tissue structures around the joint. The **soft tissue** determines the quality of the movement and therefore the ultimate function of the unit.

Movement planes develop early in embryonic life – almost like concentric mesodermal rings around the central notochord. This layered system of myofascial compartments and planes form the basis for movement throughout life. Anything interfering with the freedom of these layers to glide on each other during movement be it adhesions, thickening, or shortening, will compromise the quality of movement. This in turn contributes to the development of pathology and pain within the movement apparatus. Movement is freedom. Our job is to restore movement and function to our patients and to give them back their freedom to the best of our abilities.

Five tissue layers intervene between the surface and the underlying skeleton in most parts of the body (Tobias p35, 1977). These are:

- **The skin,** consisting of the epidermis (the epithelial layer), and the dermis (the underlying connective tissue layer).
- **The superficial fascia** (tela subcutanea), which binds the skin to deeper structures.
- **Deep fascia,** a dense layer of connective tissue between superficial fascia and the muscles.
- **Muscle.**
- **Periosteum,** the fibrous sheath of the bones.

The one tissue that has greatly enhanced the understanding of movement over a larger plane has been a better understanding of connective tissue in the body. (Especially the connective tissue represented by fascial structures.)

Connective tissue (CT) does not move bones or initiate movement, it merely controls the quality and sets the limits of the movement taking place. The CT Bed provides connections between muscle layers as well as between adjacent muscles. It is not only the integrity of the joint that determines the quality of movement, all the soft tissue

structures around a joint, and even a great distance away from the joint, will determine the quality of the movement and therefore the ultimate function of the unit.

Connective tissue is not just an inert structure within the body with a lesser function than the other tissues; it is alive in the sense that it responds to stimulus. It has certain physical laws that it lives by. Living tissue is capable of changing its structure in response to changing environmental or functional demands. It requires nourishment to survive and is subject to disease processes, injury and the effect of aging. Directional pull and the stresses on the system as a whole determine its fibre content and direction as well as its ultimate function.

The connective tissues may be defined as the group of elements derived largely from the embryonic mesoderm. Muscle, bone, blood and the urogenital system also have a mesodermal origin. Connective tissue varies in terms of the physical nature of its intercellular matrix and in the number and density of its fibres. In descriptive terms, this means that some is harder or softer, some more elastic or more rigid. Connective tissue is continuous throughout the body from head to toe. It has no beginning and no end. In a broad sense, connective tissue literally connects and supports. It forms the structure of the body. It supports the organ, nerve and vascular systems. Muscle tissue is enfolded within the fascia: the combination is called myofascia. Movement is the outcome of embedded muscle tissue acting on the surrounding connective tissue. Structure is thus the result of movement. (Schultz 1996)

Connective tissues play several essential roles in the body, both structural (because of the special mechanical properties of the extra-cellular elements) and defensive (because of its cellular basis e.g. the macrophage or reticulo-endothelial system). Connective tissues are conventionally divided into “ordinary” types, which are distributed widely through the body, and special types, namely cartilage and bone. (Warwick 1973)

As Physiotherapists, we are concerned with the ordinary connective tissue that comprises the superficial and deep fascia, as well as the nerve and muscle sheathes, ligaments and tendons.

Vessels and Nerves of Connective Tissue

The blood vessels of connective tissue itself are very few. Fascia mostly derives its blood supply from the vessels running between the fascial layers.

Lymphatic vessels are very numerous in most forms of connective tissue, especially in the loose tissue beneath the skin and the mucous and nervous surfaces. They also occur abundantly in the sheaths of tendons as well as in the tendons themselves.

Nerves are found ending in dense connective tissues. Pacinian and ruffini’s receptors were found in the thoracolumbar fascia (Yahia et. al. 1992) and may be concluded that they will probably be present in fascia in general. Free nerve endings are found in all types of connective tissue, including the dermis, fascia, ligaments, tendons, and sheaths of blood vessels, meninges, joint capsules, periosteum, perichondrium, Haversian systems of bone, and the endomysial spaces of all types of muscle. These different fibres are both myelinated and non-myelinated, but always of small diameter and low conduction speeds, being of the group III sensory afferent type. (Warwick 1973) Some authorities regard the small diameter afferent fibre system as constantly monitoring the fluctuating “general state” of the body tissues rather than constituting a system of specific “pain afferents”.

A critical relationship exists between the neural components of joints and the surrounding ligamentous and fascial structures. Current research suggests that all of these CT structures receive a supply of small-calibre, primary afferent fibres, typical of those involved in nociception. Sensitisation of this small-calibre, primary afferent fibres system, along with sensitisation of their central connections in the dorsal horn of the spinal chord, appears to play a critical role in the evolution of chronic painful conditions.

In post mastectomy pain complaints, several factors lead to the sensitisation of these primary free- and high threshold nociceptive nerve endings. Primary damage done by surgery and reconstruction, secondary and further damage due to radiation and chemotherapy as well as the effects of posture, emotions, and myofascial dysfunction all contribute to the final mix of a dysfunctional and highly sensitised nociceptive system within the surrounding soft tissues.

Deciding on which structure is the **cause** of pain is a waste of time – but trying to decide on which structures are **involved** is constructive. It is not enough to only identify the structures involved, but also what causes their involvement, such as poor posture, habits, or previous injury. We must not look for post mastectomy pain coming from a single structure or source. Pain can come from any source or from an accumulation or summation of several sources.

Pain is both a somatic and psychic experience. Suffering may be much more intense when pain is experienced in association with other troublesome symptoms or feelings such as fatigue, anxiety, insomnia, depression, isolation, fear, anger and uncertainty. All of these will compound suffering and must be addressed as part of a comprehensive approach to pain management. (McDonald 1998)

POST MASTECTOMY PAIN. (PMP)

Understanding the connective tissue, the "movement plane" model, and how damage during treatment for primary breast cancer compromises function, still does not give sufficiently clear understanding of the pain patterns patients suffer from after treatment, and long after they should have recovered. At present, the pain model for PMP is a model of nociceptive pain due to tissue damage during surgery and radiation. This pain should clear within a reasonable time and the patient should be pain free to continue unhindered with her life.

If the pain goes beyond the 3 month expected time of settling down, and especially if it goes beyond a year, the nociceptive pain model suddenly does not fit the neurophysiology of the complaint. The emphasis of the model then shifts to a neurogenic and even a strong central psychogenic pain model. (Jung, 2003). These last two models are approached with pharmacological intervention models. (Mac Donald, 1998). The relevancy of our physiotherapy interventions is often sidelined as ineffective and purely palliative.

Professionals who deal with pain must understand the biology and pathophysiology of the whole pain phenomenon. They must have the ability to diagnose pain, or at least categorise pain and make clinical decisions related to the categories.

PAIN

The pain sciences revolution started a good 30 years ago and despite the years, there is still little evidence to show that patients are benefiting. The scientific revolution has not turned into a much-needed clinical revolution. The taxonomy committee of the International Association for the study of pain considered definitions of pain and concluded: "Pain is and unpleasant sensory and emotional experience associated with actual of potential tissue damage, or described in terms of such damage". (Wall, 1989).

The key words are **experience, emotional**, and the concept of **potential tissue damage**. Inputs such as anxiety, fear, and frustration affect the same clusters of neurones in the central nervous system (CNS) as do inputs from damaged tissues. This creates a perceptual **experience** of pain. Often there is a lack of close correlation between injury and pain. The brain modules, past experiences, knowledge, beliefs, and culture (Gifford 1997) determine much of this.

A second key issue is appreciating the difference between acute and chronic pain. Most acute pains are seen as being the result of physiologic events that serve a clear biologic purpose: to call on bodily adaptive measures to stop the pain and protect the injured tissues. Nociceptive messages have two roles – first, to inform subconscious brain systems in order to promote a co-ordinated physiological healing response, and second to inform consciousness via the medium of "pain" in order to change behaviour(Gifford 1997). Chronic pain, on the other hand, is regarded as a neurologic disease state. Many chronic pains, however, may be

advantageous in that they protect weakened or diseased tissues that are incapable of complete recovery.

Pain is a multidimensional experience, and should be considered as such in the clinic. The **sensory dimension** is the awareness of the intensity, location, quality and behaviour of pain. The **cognitive dimension** relates to thoughts about the problem, influenced by experience and previous knowledge. Finally, the **affective dimension** is the emotional response, usually negative, that motivates of governs responses to pain (Gifford 1997). The pain experience leads to altered levels of activity and specific or general altered movement patterns. This is where the altered movement patterns seen in tight connective tissue states due to damage or tightness, and the altered movement patterns due to the pain experience overlap. In many cases it is impossible to separate the two problems. Ongoing pain states are often of great concern to the patient, especially if the problem has not been validated or has been unsuccessfully managed by clinicians. The cognitive and emotional dimensions of pain generally get more involved, the longer the problem persists.

In PMP, the ongoing pain and dysfunction pattern is well documented due to the nature of the damage to the tissues after surgery and radiation. If we only apply the pain mechanisms as they are documented in literature directly to PMP, with the exclusion of the connective tissue model described earlier, treatment will at best only be partially effective.

Before presenting a pain model for a better understanding of a possible origin of PMP, a short explanation of the different categories of pain mechanisms. The mechanisms may co-exist and overlap extensively in pain states. These categories are nociceptive, peripheral neurogenic, central, affective/cognitive, and autonomic/motor mechanisms. Each term relates to a physiologic/pathophysiologic process that can give rise to pain in sensory, cognitive, and emotional dimensions.

Nociceptive pain: (Pain originating from target tissue origin). This pain is the easiest to understand. It is the result of mechanical and physiologic processes in injured tissues that stimulate high-threshold primary afferent C and A delta fibres. Nociceptive pain is generally linked to injury, inflammation, and repair. It frequently has a clear stimulus/response relationship.

Nociceptive pain primarily relates to acute pain. It may be present in adaptive chronic pain states where poorly conditioned tissues need protection from potentially damaging movements and forces, or disease states that maintain abnormal tissue biology such as inflammation. This pain usually eases as the injury settles. It improves naturally or in response to various treatments.

There are three basic nociceptive pain patterns. With **mechanical nociceptive pain,** scar tissue or abnormal pressures from tissues may mechanically distort nerve endings. Movement increases distortion of nerve endings, causing increased pain. **Ischaemic nociceptive pain** occurs as a result of ischaemia altering the physical and chemical environment of tissues. This results in increased excitation and sensitisation of nociceptors. Ischaemic tissue become more acidic, contains less oxygen and is rich in nociceptor activity enhancing chemicals. **Inflammatory nociceptive pain** relates to inflammatory processes in the injured or diseased tissues. The inflammation causes a decrease in the response threshold in afferent fibres and some even fire spontaneously. Just a small amount of movement or gentle pressure may evoke pain that takes some time to settle. Feeling worse in the mornings and reporting morning stiffness is a frequent complaint in this type of nociceptive pain. “Silent nociceptors” – those that are initially insensitive to any type of stimuli, become responsive to mechanical and/or thermal stimuli after repeated noxious stimulation or chemical irritation (Greenspan 1997). Thus, some populations of nociceptors my only become active after injury

Fortunately, in the overwhelming majority of cases, pain is short-lived. It subsides within seconds or minutes if the stimulus has produced no irreversible damage. Even if there is damage that outlasts the insult that produced it (bruise or burn) it heals within days and the

pain subsides. There are, however, circumstances in which the pain persists, or may even worsen with time. (Fields, 1990).

Neuropathic pain: (Pain from peripheral neural tissue origin.) The general term"neuropathic" is used to refer to pain due to abnormalities of nervous function. Neuropathic pain syndromes have certain characteristic features (Fields 1990):

- Pain occurs in the absence of a detectable ongoing tissue –damaging process.
- Abnormal or unfamiliar unpleasant sensations frequently having a burning and/or electrical quality
- Delay in onset after precipitating in jury
- Pain is felt in a region of sensory deficit
- Paroxysmal brief shooting or stabbing component.
- Mild stimuli are painful
- Pronounced summation and after-reaction with repetitive stimuli

Nociceptive pain usually resolves as damaged tissues heal, whereas pain from neuronal dysfunction can persist indefinitely. Jung et al. distinguishes four different types of chronic neuropathic pain following breast cancer surgery (Jung 2003) i.e. Phantom breast pain (versus non-painful phantom breast sensation), intercostobrachial neuralgic, neuroma pain, and pain from other nerve injuries (medial and lateral pectoral nerves, the long thoracic nerve, or the thoracodorsal nerve).

For the clinician, this means that a segment of nerve can become a source of pain. Mechanical forces, chemical (catecholamines) or metabolic changes such as ischaemia may evoke this.

Central sensitisation (pain related to altered central nervous system circuitry and processing). Central nervous system (CNS) cells change their response properties when subjected to high threshold input (i.e. nociceptor input). While an increase of CNS sensitivity is of great adaptive value to promote protective motor activity and healing behaviour, sometimes this enhanced excitability state persists long after peripheral tissues have healed to the best of their abilities, and the dominant source of pain shifts to the CNS (Gifford 1997).

Therapists may consider the symptoms weird and wholly inappropriate to the history. There is rarely a physical test that does not hurt in some way, and rarely a test where the patient reports an improvement in symptoms.

This short look at nociceptive, neuropathic and central pain mechanisms is by far not a full or comprehensive cover of the pain patterns seen in patients suffering from post mastectomy pain. Neuropathic and central patterns seem to be more often diagnosed and described when PMP is discussed. (Stevens 1995: Gottrup 2000; Jung 2003)

With PMP often seen in woman well beyond the normal recovery time for nociceptive pain it is natural to assume that the sources of pain beyond 3-12 months could be neuropathic, central or affective in origin. I am of the opinion that there are a large number of patients with PMP that has an ongoing nociceptive type of pain pattern of mechanical origin.

The observed patterns of dysfunction seen in patients after treatment for breast cancer can mostly be traced back to damage within the connective tissue and fascial movement planes. As seen in our "movement plan" model earlier, any interference with the freedom of fascia, fat pads and areolar tissue to glide and slide over each other during movement due to connective tissue tightness, thickening or adhesions and scarring will produce dysfunctional movement patterns. A vital function of the nervous system is to provide information that concerns injury and the threat of injury. The sensation of pain contributes to this function. Highly specialised types of sensory fibres provide information to the central nervous system not only about the environment, but also about the state of the organism itself.

One class of receptor with a relatively high threshold to adequate stimulus is the nociceptor. (Type IV nerve endings). They respond preferentially to noxious (injurious of potentially injurious) stimuli. These nociceptors are found distributed throughout the body, although their

concentrations may vary from tissue to tissue. The are found in all types of connective tissue. (Warwick 1973) This includes periosteum, fasciae, tendons, aponeuroses, deep and superficial joint capsules, fat pads (intra-articular and others) dura mater, and even intervertebral discs. (Wyke 1981; Oliver 1991; Campbell 1989; Willard 1997)

The nerve supply of the lumbar region has been extensively studies, and it has been established that the entire connective tissue stocking of this area receives a small-calibre, primary afferent fibre innervation. (Willard 1997; Oliver 1991; Yahai 1992). Several recent observations suggest that the population of sensory neurones innervating connective tissue is dynamic and can respond to changing states of the tissue. It can therefore be assumed that most if not all of the soft tissue structures of the shoulder girdle and breast would be amply supplied by primary afferent nerve endings (nociceptors) to constantly monitor the state of the area.

Once tissue damage occurs, a cascade of events results in enhanced pain to natural stimuli (Campbell 1989). This is due to nociceptor sensitisation, which is defined as a lowered threshold; and an increased responsiveness of the nerve endings to stimulation. (Greenspan 1997). Numerous chemical agents produced and released at an injury site are capable of sensitising nociceptors (e.g. prostoglandins and leukotrienes). Other chemical (brandykinin, substance P, cytokines, and serotonin) can both activate and sensitise nociceptors to subsequent stimuli.

In the lumbar spine, this sensitised nociceptive system slowly develops over time due to dysfunction or misuse of the region. This may probably be precipitated by previous injuries (even minor repeated insults). Many of these nociceptive nerve endings are capable of secreting pro-inflammatory neuropeptides from their distal processes. This sets off a vicious cycle of events of which the final result is tissue inflammation and oedema - neurogenic inflammation. Thus, the elements are present in the musculoskeletal system to facilitate chronic inflammatory processes, leading to tissue degeneration and chronic pain syndromes (Willard 1997).

After initial healing of the acute tissue damage has taken place, the nociceptive element of pain should stop, and normal activity should be resumed. Unfortunately, damage to the connective tissue due to treatment for breast cancer (surgery and radiotherapy) already leaves the primary afferent nerve endings sensitised to even minor noxious stimuli (Jung 2003, Gottrup 2000). With tightness due to scarring and tissue damage in the movement planes, dysfunctional movement patterns of the upper quarter keeps the sensitised primary afferent system (nociceptive) chronically stimulated.

Their primary role as monitors of the state of the organism now become a continuous flow of noxious stimuli to the central nervous system triggering all the other pain mechanisms (neuropathic, central, affective, autonomic) to become involved as well. Within this final mix of pain mechanisms after breast cancer treatment, the emphasis on the neuropathic has dominated. The fact that the nociceptive activity seems to be ongoing in PMP, may partially explain why the treatment for these pain syndromes have been of such mixed results. (Maher Committee).

SIMPLYFIED PAIN MODEL

Taking the foregoing discussion on the establishment of a working model, based on the freedom to move within fascial planes, how treatment damages this movement model, and our short discussion on pain mechanisms into account, I propose a simplified pain model for post mastectomy pain syndromes. This should guide our treatment interventions for this group of patients.

With the role of CT in guiding the quality of human movement becoming clearer, and the fact that it is so richly innervated by primary afferent nerve endings, restoring the integrity of the CT system after treatment damage should become the primary focus of therapy. All mechanical restrictions within the facial layers needs evaluation as far as freedom to move is concerned. Because of the sensitised nociceptive system with its lowered threshold to react

to stimuli, even slight fascial restrictions may become the focus of a noxious stimulus reaching the CNS. Because the CNS is sensitive to dysfunction and its potential interference with our ability to survive, it cannot ignore the constant nociceptive input. The patient therefore becomes so focussed on her problem, that the central, affective and emotional components may dominate, clouding our ability to diagnose the source of the pain.

Treatment directed towards restoring and maintaining normal, pain free movement should be the primary aim of any program following breast cancer treatment. Normal, or as close as we can get to it, movement of the fascial planes will reduce the noxious input of the sensitised nociceptors due to mechanical irritation of the system. It will also reduce the development of dysfunctional movement patterns in other areas (neck, back and shoulder) that may lead to secondary pain patterns and syndromes becoming a new source of anxiety to the patient.

This model guides me in my evaluation of the problem and gives me a clearer indication of the site at which therapeutic intervention should theoretically be targeted. With a simplified movement/pain model we can divert therapy at the dysfunction even if the "source" of the problem is not fully evident or clear. In many cases pain may be unalterable, but our therapeutic intervention to improve function may vastly diminish the patients suffering and disability.

Having a clear model to base our reasoning on, no matter how inadequate it may seem at present, empowers me, the therapist, to give the patient an adequate explanation of therapy, explanation of the problem and attention to the patients anxiety, job satisfaction and fear of pain. (Gifford 1997)

Management of post mastectomy pain requires therapists to take on pain in all its dimensions and the new opportunities it brings. It is a professionally empowering necessity. Pain provides a common link with many medically related professions. It provides a common language, it stimulates research, and it must direct therapy! We must begin to change our ways now, before current therapies are either embarrassing or are rejected. These are exciting times. Our role is to make sure they are exciting for our patients as well.

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