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BESTPRACTICE

Best Practice Guidelines for Diagnosing Muscle Imbalance:

Chiropractic versus Physiotherapy

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Abstract: Professor Vladimir Janda, an accomplished physiotherapist and neurologist, was a key figure in the 20th Century rehabilitation and manual therapy movement. Janda founded the rehabilitation department at Charles University Hospital in Prague, Czechoslovakia.

He was one of the seminal members of the Prague school of manual medicine and rehabilitation that expanded its influence throughout Central and Eastern Europe. Janda published over 16 books and more than 200 publications about muscle function, and has had a major influence over the physical therapy profession around the world. A review of Janda's published works demonstrates the breadth of his clinical interest and influence. His published papers varied greatly in their focus: from pediatrics to geriatrics, in addition to the lasting effects of pediatric conditions upon the adult, from postural to neurologic disorders, and from ankle conditions to obscure facial pain. His 1964 college thesis paper was on the association between sacroiliac pain and *gluteus maximus* weakness. (Janda, 1964)

In addition to publishing several texts in Czech, Janda subsequently published books in German and English. Janda's approach has been discussed in many text books, often in chapters that he authored. Many years ago, Janda published a manual muscle testing book in English that is now out of print. (Janda, 1983) Many leaders in the manual therapy world, like Drs. Chaitow and Liebenson, have depended upon the work of Vladimir Janda for their concepts of muscular imbalances and the use of the manual muscle test (MMT). These leaders interact and write in one another's books spreading the Janda-model far and wide.

This is unfortunate because of 5 PROBLEMS in Dr. Janda's view of muscle inhibitions … and fortunate as well because it has increased the worldwide understanding of the significance of MMT's diagnostic potential of muscle imbalances in human health.It may be that an entire generation of manual therapists has abandoned the diagnostic gold-mine of the MMT in part because of Janda's approach to the assessment of 'muscular imbalance'.

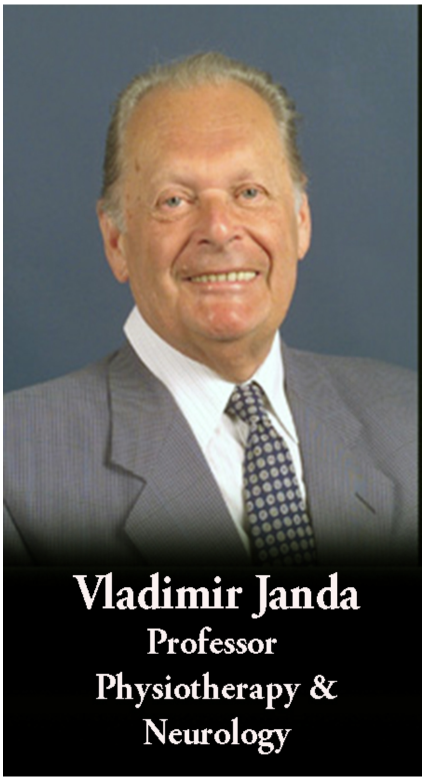
This presentation will explore this contention.

Indexing Terms: manual muscle test; muscle imbalance; Applied Kinesiology (AK); diagnosis; physical therapy profession; Goodheart; Janda.

Introduction

George J. Goodheart Jr, DC (1918- 2008) and Vladimir Janda, MD (1923-2002) influenced generations of practitioners spanning many disciplines. One difference between Goodheart's approach (a chiropractor) and Janda's (a physical therapist) is that muscle inhibitions are identified and treated first with chiropractic manipulative therapy (CMT). In agreement with the literature cited in my previous articles, muscle inhibition is seen as an etiological factor and/or common co-factor in neck, low back, and extremity pain and dysfunction. (Cuthbert 2009, a, b, c, d)

'… Cuthbert provides an exhaustive review of the two main approaches to the diagnosis of muscle imbalance: the physiotherapy approach of Janda and the chiropractic approach of Goodheart. The chiropractic approach is more refined and provides deeper clinical information through therapy localisation and challenge…'



There are 5 questionable physiotherapy propositions by Janda about muscular imbalance. I present these then follow with chiropractic clarifications given by Goodheart.

Janda's questionable propositions of physiotherapy are:

1. In the Janda/Physiotherapy/Czech School approach to muscular imbalance disorders, muscular hypertonicity and 'spasm' are considered the etiological factors of joint and soft-tissue dysfunction, and are treated first.
2. Janda stated frequently that postural muscles tend to be short, tight, and hypertonic when they are in dysfunction.
3. Exercise (physiotherapeutic sensorimotor training) is the treatment of choice for muscle inhibition and imbalance.
4. In the Janda assessment of postural disorders, 'visual and palpatory diagnosis is the most reliable form of assessment for muscular imbalance.' (Liebenson, 2007)
5. According to Janda: (Janda et al., 2007)

   '*Evaluation of muscle imbalance in a patient with an acute pain syndrome is unreliable and must be undertaken with precaution. A precise evaluation of tight muscles and movement patterns can be performed only if the patient is pain-free or almost pain-free.*'

Chiropractic reply to Janda's proposition 1

Muscle inhibition (not hypertonicity) is the primary characteristic of painful muscles

The manipulative treatment of muscle imbalance physiology was first described by George Goodheart (1964-1998) and was also considered a fundamental characteristic of postural and spinal imbalances by Vladimir Janda. (1983, 1964) The study of Goodheart's and Janda's development of the concepts of muscle imbalance (and their diagnostic and therapeutic approaches) provides fascinating comparisons.

The voluntary skeletal muscle system is the largest single organ in the body. It measures in at over 40% of the body mass and is maintained in a sophisticated state of balance and co-ordination throughout a wide range of postures and activities. The muscles are at once the source and the recipient of the greatest neural activity in the body. This includes sensory and motor activity, segmental and cerebral pathways, plus autonomic activity in relationship to the metabolic, visceral, and circulatory demands required during human life and movement.

The focus of treatment for muscular imbalance is where the Goodheart and Janda models divide. Goodheart and Janda agreed that the muscles are in fact '*the most exposed part of the nervous system.*'

As presented in the *Journal of Bodywork and Movement Therapies* (Cuthbert, Rosner, McDowall 2011) the evidence now shows with greater clarity that inflammation or injury produces inhibited muscles that may be specifically identified with the MMT. These authors showed that a symptomatic group of patients with mechanical neck pain (MNP) demonstrated significantly increased MMT findings in the form of reduced strength levels compared to a control group (n=248). This evidence suggests that the MMT is potentially a sensitive and specific test for evaluating cervical spine muscular impairments in patients with MNP.

For group 1 (Patients with Mechanical Neck Pain)

One-hundred and thirty-nine of 148 patients reporting MNP showed inhibition on MMT in at least one or more of the four tests (MMT of the *sternocleidomastoid*, *anterior scalene*, *upper trapezius*, and cervical extensor muscles), yielding a sensitivity of 93.9%. Weaknesses were broadly and to a large extent equally distributed (32.4%-43.2%) across the four muscle groups tested. (Table 1)

If the 148 MNP patients in this cohort were truly representative of the overall patient population, then it would be possible to compute a confidence interval. We chose the Clopper-Pearson two-sided interval, the methodology being appropriate for binomial (yes/no) data and making no assumptions about any data distributions being normal or approximately normal. To arrive at the confidence interval, we used the *binom.test* function from the 'R' statistical program (www.r-project.org).

Under these circumstances and using a 95% confidence interval, we would estimate that between 88.8% and 97.2% of all patients have positive MMT findings in one or more of the four muscle pairs tested. (Figure 1)

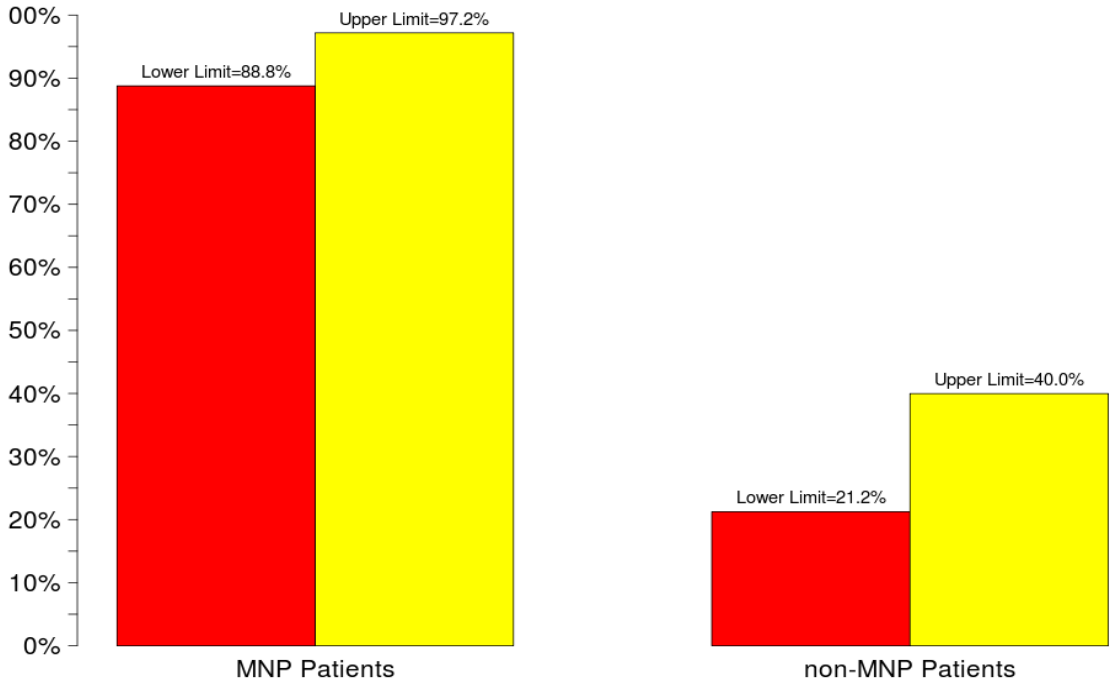
For group 2 (Patients without Mechanical Neck Pain):

Thirty of the 100 patients without MNP showed positive results in one or more of the four MMT tests, yielding a specificity of 70%. The total number of positive MMT findings in the control group was 37. However, there were only 30 patients with positive MMT findings, because several patients had positive results from more than one test. In this instance, positive findings were generally confined to the sternocleidomastoid and anterior scalene muscles. (Table 1) Using the assumptions discussed above, the 95% confidence interval for this group would be between 21.2% and 40.0%. (Figure 1)

Table 1: Number and Percentages of Patients with Positive MMT findings, by Muscle Group

	Control Group (100 patients)	Mechanical Neck Pain Group (148 Patients)
Sternocleidomastoid	18 (18%)	61 (41.2%)
Anterior scalene	13 (13%)	49 (33.1%)
Upper trapezius	4 (4%)	64 (43.2%)
Cervical extensors	2 (2%)	48 (32.4%)

Figure 1: Comparison of Positive MMT Findings Between MNP and Non-MNP Groups



The apparently wide gap of confidence intervals between those patient cohorts with or without MNP is noteworthy, keeping in mind that (1) the specificity of the MMT in patients without MNP was 70%, and (2) the sensitivity of the MMT in patients with MNP was 93.7%.

The '*sensitivity*' of the MMT proved to be high for subjects in group 1. Sensitivity indicates the likelihood of receiving a positive MMT result in one or more of the cervical muscles tested when MNP was truly present. The '*specificity*' of the MMT was not as high but still significant for group 2. Specificity indicates the likelihood of receiving a negative MMT result when MNP was not present. Under these circumstances, our data as shown in Figure 1 suggest that MMT was a sensitive and moderately specific test for differentiating the two groups of patients with and without MNP.

Controlled clinical studies have shown that dysfunction and pain specifically in the cervical spine will produce inhibited muscles. These data indicate that the body's reaction to injury and pain is not increased muscular tension and stiffness; rather muscle inhibition is often more significant as measured by several different methods of testing.

In 1920 Cyriax first described the relationship between muscle weakness (detected with a manual muscle test, or MMT) and headaches. (Cyriax 1920)

In 2008 an important literature review on neck muscle strength by Dvir (in a special issue of JMPT called '*Cervical Outcome Measures: State of the Art*') confirms that '*overall studies indicate that compared to normal subjects patients suffering from neck-related disorders present with significant reduction in cervical strength.*' (Falla et al. 2004; Falla et al. 2003; Vernon et al 1992; Silverman et al. 1991)

Muscle imbalance (the '*hypertonicity is primary model*') as conceived by Janda was mainly embraced by the physiotherapy and professional massage community, though in recent years it has lost some of its popularity to the concept of core function and motor control. (Jull et al 2019, 2008; Lederman 2010; Chaitow et al, 2008)

Because the Janda approach avoids the MMT for the evaluation of patients with muscular imbalances who are simultaneously in pain, it is usually supposed in this world-view that the tight and tense muscles (the most easily palpable signs of muscle imbalance phenomena and the antagonists to the inhibited muscles) are responsible for muscle weakness.

In Janda's model '*tightness weakness*' develops when a muscle is chronically shortened and eventually loses strength (i.e., the psoas). Janda has reported that even when a muscle appears to be tight or stiff, some decrease in muscle strength occurs. Brooks confirms that chronically contracted muscles are weaker than muscles with a normal length. (Brooks, 1986)

Leahy, the founder of chiropractic's *Active Release Technique®* says it simply: '*When a muscle is tight it tends to weaken and when a muscle is weak it tends to be tight*.' (Leahy 1999)

'*Stretch before strengthening*' is another fundamental law in the Janda-Rehabilitation program. '*If a movement pattern is faulty, the general rule of thumb is to initiate rehabilitation by treating tight muscles related to the faulty pattern*.' (See Janda's movement pattern assessments below)

'*Once tight muscles are addressed then facilitation and training of the "weak link" can proceed. The reason for this is if muscle tightness is present, then strength training will typically reinforce 'trick' movements, thus perpetuating the muscle incoordination.*' (Janda 2007)

In the Janda worldview Sherrington's law of reciprocal innervation operates primarily in one direction as a neuromusculoskeletal law of nature, i.e. it is the hypertonic muscle that creates the phenomenon of inhibited muscles, not the other way around. This, however, is not the correct interpretation of Sherrington's law of reciprocal innervation.

Sherrington's Law of reciprocal innervation states that muscle inhibition usually generates hypertonicity/tightness in antagonist muscles, and that the relationship between weak and tight muscles is reciprocal, with inhibition producing the same influence on their antagonist muscles as tightness. Sherrington advises that '*Knowledge of reflex inhibition equally with that of reflex excitation is essential for the study of nervous co-ordination*.' (Sherrington 1913) Abnormal muscle inhibition is as neurologically important as over-facilitation in patients with pain and dysfunction … in fact, as will be seen later, muscle inhibition is the primary long-term consequence of pain. (Mense & Simons 2001)

Lund (Lund et al. 1991) and many others have comprehensively confirmed Sherrington's early insights showing that inhibition is frequently found in muscles resulting from injury, inflammation or pain and that the inhibition or weakness leads to reciprocal facilitation of its antagonist(s) and aberrant behaviour of its synergist(s). (Cuthbert, 2009 a, b, c, d) It is also true that hypertonicity in a muscle also leads to reciprocal inhibition of its antagonist(s) and aberrant behaviour of its synergist(s). (Janda 1983) This is the reciprocity of Sherrington's Law, with due respect paid to both models of diagnosis and treatment for muscular imbalance (Goodheart's and/or Janda's).

Goodheart's model is the one used by members of the professions who employ the MMT for diagnosis of neuromusculoskeletal dysfunction. (Cuthbert et al. 2018; Cuthbert et al 2022; Leaf 2010; Kendall et al. 2005; Garten 2004; Gerz 2001; Walther 2000; Maffeton, 1999; Goodheart, 1998; Walker 1996) No other system of physical diagnosis in chiropractic has so extensively described the muscular etiologies and the corrective methods for neck muscle impairments as applied kinesiology (AK).

The AK MMT examination detects weak muscles, inhibited muscles, compensatory movement patterns, antalgic movement patterns, synergist substitution, timing and endurance impairments, and muscles recruited in an abnormal sequence. (Schmitt & Cuthbert 2008) These dysfunctions help to identify which muscle or muscles are in trouble and what may be causing the problem. The weakness of muscles in the distribution of the motor nerve must by distinguished from the dysfunctional patterns of weakness induced by micro-avulsions and enthesopathy, trigger points, acupuncture meridian problems, cranial-sacral problems and other strains related to functional muscle groups, regardless of innervation.

The broad scope of '*causes*' that have so far been identified using applied kinesiology methods have been described. (Cuthbert & Walther 2018; Rosner & Cuthbert 2012) An inclusive assessment should be made to those components of dysfunction that best meet the patient's current needs. The AK method of evaluation for muscular inhibition helps identify the most appropriate and timely intervention.

Figure 2: Required actions by manual physicians

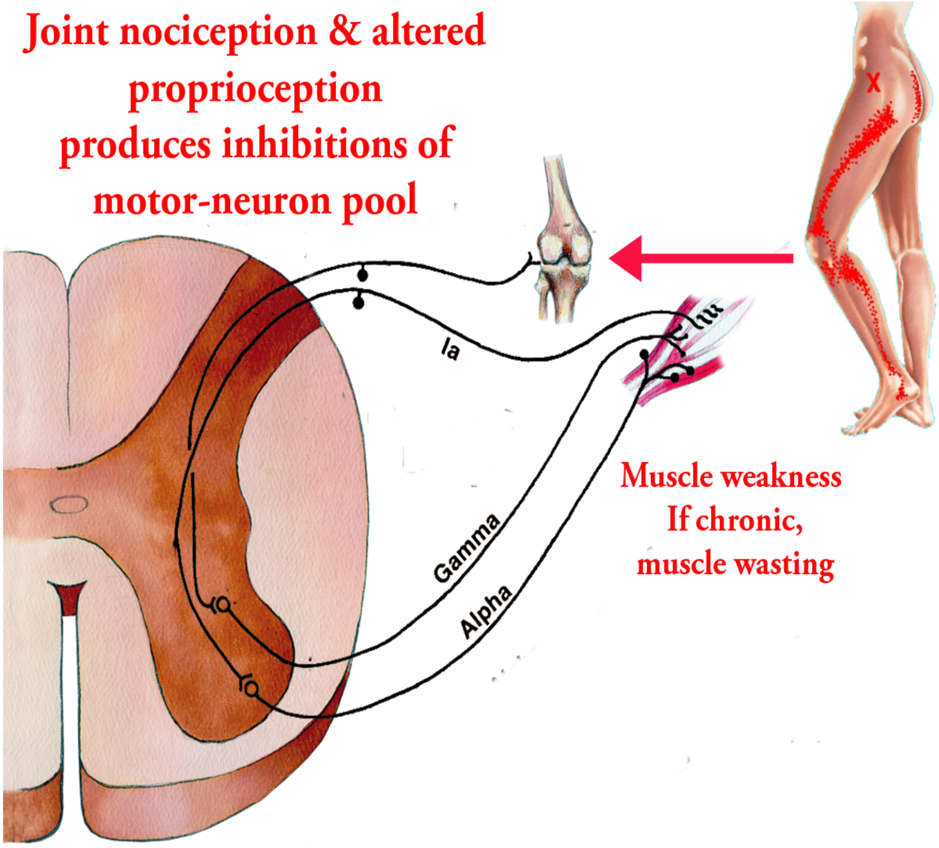
There is no suggestion implied here that Janda's approach does not also seek causes, however, the principles of AK demand a primary attention to context and the processes involved in symptom manifestation, e.g. postural and structural imbalance, lowered vitality, organ dysfunction, nutritional imbalance, toxicity, meridian imbalances, etc., as well as the more obvious etiological features of any given condition.

Examples of manual medical approaches that offer short-term gain, without consideration of the context out of which the symptoms have emerged, can be described for almost all professions and modalities. This is the case when they are applied in isolation, outside of a comprehensive contextual evaluation of the patient's broader symptoms and needs.

With the complexity of symptoms on display in the typical patient, including pain and dysfunctional tissues, joints, etc., where would it be most appropriate to initiate treatment? The MMT (with the addition of therapy localization and challenge) identifies the dysfunctional tissue and allows for the identification of the precise articular, soft-tissue, biochemical, or psycho-emotional correction that will change that finding.

This suggests that much that is currently done in chiropractic, osteopathic, manual medicine, physical therapy and massage therapy settings may fail to meet the basic AK requirements in dealing with the whole person and the causes of their problems.

Figure 3: Inhibited muscles found in the applied kinesiology clinical setting are capable of spontaneous strengthening when the inhibitory reflex is identified and remedied (most commonly through chiropractic subluxation correction or soft tissue manipulation). (Cuthbert & Walther, 2018)



Because in Janda's model muscle hypertonicity/tightness/spasm is the primary cause of inhibition in its antagonists, spasm is treated first.

However … modern pain research has demonstrated that one of the most prominent features of all chronic pain is the presence of localized areas of soft tissue dysfunction which promote pain, muscle imbalance, distress and muscle weakness in local and distant structures. (Chaitow & DeLany 2008; Melzack and Wall 1988) These are loci which are known as myofascial trigger points, (MTrP) the focus of enormous research effort and clinical treatment.

According to Travell and Simons and the now well-established '*integrated myofascial trigger point hypothesis*', an active trigger point will inhibit the function of the muscle in which it is housed as well as those which lie in its target zone of referral. (Simons et al. 1999)

'*Although weakness is generally characteristic of a muscle with active myofascial trigger points, the magnitude is variable from muscle to muscle, and from subject to subject. EMG studies indicate that in muscles with active trigger points the muscle starts out fatigued, it fatigues more rapidly, and it becomes exhausted sooner than normal muscles.*' (Mense & Simons 2001)

It must be repeated that MTrPs are considered a hallmark finding of muscle pain syndromes and within clinical practice are claimed to be a common source of musculoskeletal pain and dysfunction in people presenting for manual therapy. (Blanco et al. 2006) Because muscles with MTrPs are almost always inhibited with movement and/or exertion, the Janda hypothesis that hypertonic/tight muscles are the etiological cause of musculoskeletal dysfunction ignores the findings of Travell, Simons, Kendall, Goodheart and many other researchers.

Figure 4: Reproduced with kind permission of Simons & Travell

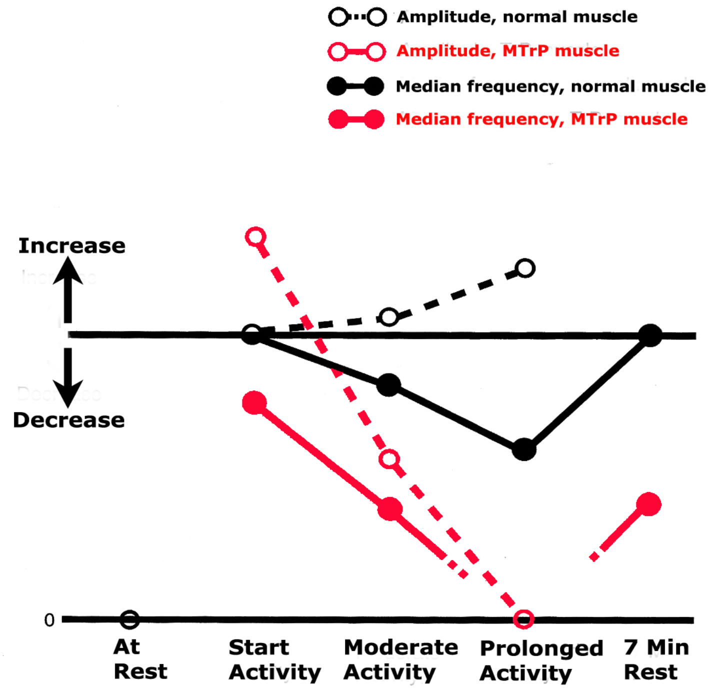


Figure 2.3. Comparison of surface electromyographic response to fatiguing exercise of normal muscle (*black lines*) and muscle with active myofascial trigger points (*red lines*). The averaged amplitude (*open circles*) and mean power frequency (*solid circles*) of the electromyographic record from the muscle with trigger points start out as if the muscle is already fatigued and show that the muscle reaches exhaustion more quickly (and is slower to recover) than normal muscle. These changes are accompanied by accelerated fatigue and weakness of the muscle with trigger points.

In Janda's approach hypertonic muscles are treated with physiotherapeutic means such as massage, stretching, proprioceptive neuromuscular facilitation, electrotherapy and other methods that do not usually include chiropractic or medical manipulative therapy. (Liebenson 2019, 2007; Page et al. 2010; Chaitow & DeLany 2008) In Janda's classic text on MMT there is no mention of spinal or other joint manipulation options for any muscle inhibitions, and no correlations are observed between manipulative corrections (nor cranial, meridian, nutritional, or psychological treatments) and specific muscle inhibitions. (Janda 1983)

Briefly, the following findings emerges from the literature concerning the muscular inhibitions co-present in patients with chronic low back pain (CLBP):

- Reduced force of contraction: Force losses in trunk muscles occur in acute and CLBP patients (Cuthbert & Walther 2018; Cuthbert 2009 , b, c, d; Hides et al 1994; Ng et al 1998; Shirado 1995a)
- Reduced range of motion FREQUENTLY DUE TO muscle inhibition: Loss of flexion-relaxation response in the spinal muscles during flexion in patients with CLBP. Extensor activation prevents full forward bending. (Liebenson 2019; Simons & Travell 1999; Shirado 1995b) Individuals with high pain-related fear had smaller excursions of the lumbar spine for reaches to all targets at 3 and 6 weeks, but not at 12 weeks following pain onset. (Thomas et al. 2008) Smaller stride length. (Lamoth et al. 2008)
- Reduced velocity of muscle contraction: Reduced velocity of trunk movement during induced back pain. (Zedka et al. 1999) Individuals with high pain-related fear had smaller peak velocities and accelerations of the lumbar spine and hip joints, even after resolution of back pain. (Thomas et al. 2008) Walking velocity significantly lower in LBP patients. (Lamoth et al. 2008)
- Decreased endurance of muscles: Increased fatiguability of trunk muscles in patient with CLBP. (Suter & Lindsay 2001; Shirado et al. 1995a, b; Roy 1989)

- Alterations in timing of muscular contraction: Impaired postural control of the lumbar spine is associated with delayed trunk/abdominal muscles response times in CLBP patients. (Thomas et al. 2008; MacDonald et al. 2006; Radebold et al. 2001; Hodges et al. 1999) Increase in trunk co-contraction in CLBP patients. (Cholewicki et al. 2005) Increase co-contraction in trunk during walking and additional cognitive demands. (Lamoth et al. 2008)
- Impaired coordination control of muscles: Lumbar spine-hip joint coordination altered in back pain subjects. (Shum et al. 2005) Dis-coordination in pelvis-thorax coordination in LBP. (Lamoth et al. 2006)
- Impaired balance and decreased postural stability: Changes in postural control in CLBP. (Popa et al. 2007) Impaired postural control of the lumbar spine associated with delayed muscle response times in CLBP patients. (Radebold et al. 2001) Changes in postural control unrelated to pain in CLBP. (della Volpe et al. 2006) Post spinal surgery postural control changes both in pain and pain-free subjects. However, more evident in the symptomatic subjects. (Bouche et al. 2006) Hip strategy for balance control in quiet standing is affected in CLBP. (Mok et al. 2004) The elegant experiments of Zampagni et al. (2009) demonstrated the effect of the AK shock absorber test upon hip muscle control as well. Experimental muscle pain changes feed-forward postural responses of the trunk muscles. (Hodges et al. 2003)
- Impaired reaction time: Compared to healthy controls, persons with LBP exhibited a reduced ability to adapt trunk-pelvis coordination and spinal muscle activity to sudden changes in walking velocity. (Lamoth et al. 2006) Slower reaction time in LBP patients.

Similar lists of contemporary research showing the muscle inhibitions accompanying most of the other physical disorders experienced by patients who visit chiropractors and physiotherapists around the world is available. (Cuthbert et al. 2018; Cuthbert 2009a, b, c, d.)

We now have a conundrum.

With all these accepted 'facts' about muscle imbalance, why does the Janda approach always seek out the hypertonic/overactive muscle, and consider it the primary muscular consequence of neuromuscular dysfunction?

We must ask why is there a fundamental error in The Janda/Physiotherapeutic/Prague school approach to the diagnosis and treatment of muscular imbalance?

It appears that hypertonicity is the keystone to the Janda muscular imbalance phenomenon because it is the most easily palpated and visualized, the modus operandi has therefore become the *modus vivendi* of this system of diagnosis.

However we know palpation itself can have poor reliability, and the entity palpated under the skin (particularly for the determination of the strength, weakness, or responsiveness of the muscle under the palpating hands) is not easily identified by any examiner's sense of palpatory touch! (Rathbone et al. 2017; Mense & Simons 2001; Troyanovich et al. 1999; Keating et al. 1990)

Chiropractic reply to Janda's proposition 2

The constant variability of a muscle's adaptation to dysfunction

It is the clinical experience of manual muscle testers that muscle imbalances relate primarily to the individual patient's adaptations to specific injuries and stresses, rather than to any a priori properties of these muscles to be either hypotonic or hypertonic as a law of nature.

AK essentially sees muscle function as a '*transcript of the central integrative state of the anterior horn motoneurons, summing all excitatory and inhibitory inputs*' being experienced by the patient at the time of the examination. (Schmitt & Yanuck 1998) If a muscle becomes hypotonic or hypertonic, this occurs as a result of a life-time of adaptive neurological events in a patient's history, and not because a particular muscle is '*postural*' or '*phasic*' as a law of nature.

Generally, Janda's conception that postural muscles tend to be tight and phasic muscles tend to be weak is imprecise and inaccurate simply by virtue of the multitude of studies that show postural muscles are inhibited, slower to contract, show decreased endurance and reduced performance in painful physical disturbances. (Cuthbert & Walther 2018)

The fact that postural muscles frequently show inhibition on MMT assessment is more in line with the very common impression that pain makes muscles difficult to use and less powerful. (Mills 1983)

For instance there is considerable variability in the changes of muscle activity between individuals with neck and low back pain, as demonstrated by the large standard deviation of EMG data. (Jull et al. 2019; Cuthbert & Walther 2018; Cuthbert 2009 b; Falla et al. 2004) On this point Travell and Simons cited Edinger as well as Strong who showed long ago that individuals with leg length inequality who placed their feet in a normal position a few inches apart showed substantial individual variations in the standing EMG in identical muscles. (Strong et al. 1967; Edinger et al. 1957) This evidence has been consistently reiterated in the scientific literature since that time.

It has also been demonstrated that every task or movement we perform will never exactly repeat itself, (Bartlett et al. 2007) variability is neurologically built into our body's response to life! It is now suggested that such variability is an essential and healthy aspect of biological systems and that during injury and disease processes this variability tends to be diminished. (Stergiou et al. 2006) Further, muscle recruitment will vary considerably from one task to another. (McGill et al. 2003)

For example the trunk muscles will display completely different activation patterns during standing, walking, reaching to the sides or forward, bending or lifting or any other imaginable movement. (Andersson et al. 1996) For this reason, the MMT's usefulness while the patient is in differing postures is a critical, real-world addition to the MMT, and an unexplored approach in Janda's writings on the use of the MMT.

Furthermore, even within the same task, changes in the underlying movement parameters and many other additional factors will influence the complex recruitment of muscles. Most importantly for patients who see physicians, the experience, anticipation, or fear of pain will influence the muscle recruitment patterns dramatically, (McGill et al. 2003) making the predictability of the '*Janda Postural Syndromes*' dependably shaky.

No single pattern of muscle recruitment dominates movement (otherwise it would be impossible to move freely). This has important implications for diagnosis and treatment and reinforces the necessity of an integrative and dynamic approach. It suggests that patients should be evaluated in a variety of positions and that these should be similar to the positions that the patient uses in daily life. (Seidler 2004)

Janda emphasizes that prime movers and synergists are tested with the MMT, not individual muscles, and emphasizes that the usefulness of the MMT is minor compared to the visual assessment of total body movements during activities of daily living. (Janda 1983) (See reply to Janda's proposition 4 below)

However it should be pointed out that every muscle is a prime mover in some specific direction and action. In the search for that action, one is led into the field of precise, individual MMT. Manual muscle tests are designed to replicate the primary vector of motion of a muscle while minimizing the contributions of secondary mover muscles.

During an individual MMT, the designated primary mover muscle should have the highest level of activity compared with the secondary mover or synergist muscles. When any one muscle in the body is inhibited in its strength or action, stability of the part is impaired or some exact movement is lost to some extent. When inhibition of a muscle results in the inability to hold the test position or perform the test movement ascribed to that muscle, the usefulness of the individual muscle test is substantiated. Each of the body's muscles moves the organism in a unique direction and is definitely a part of every patient's total movement pattern.

Figure 5: Manual muscle tests are designed to replicate the primary vector of motion of a muscle while minimizing the contribution of secondary mover muscles. There is an ideal starting position and vector of testing force that places the cervical muscle being tested as the prime mover and the synergists at a disadvantage during the test. Accurate MMT must be done with a high level of anatomical and physiological knowledge.

Proprioceptive acuity also depends on the intactness of the sensing apparatus (mechanoreceptors and their peripheral to central pathways) and the intactness of central integration/processing of sensory information. Generally in musculoskeletal injury the damage is to the proprioceptive apparatus in the periphery. Later it may be accompanied by adaptive central re-organization. Proprioceptive changes in peripheral musculoskeletal injuries usually manifest as diminished acuity in position and movement sense, diminished strength, velocity and endurance of movement. (Cuthbert et al. 2018; Cuthbert 2009 a, b, c, d) These changes together with nociception often result in unrefined motor control, wherein the '*predictable postural syndromes*' predicted by Janda are rarely found except in the textbooks on '*the Janda model*'.

In the Goodheart model, if muscle inhibition is caused by a manipulable articular or soft-tissue disorder then the inhibited muscle's response to the proper manipulative therapy will be immediate and the tight antagonist muscles will relax. This brings about postural balance on visual inspection and corrects the positive MMT findings, both of which are evidence of the muscle imbalance phenomena.

A reproduction of lecture notes given by Dr. Janet Travell may be useful in this comparison. Dr. Travell indicates that there is no '*universal law*' of muscle imbalance for any muscle, and that every single muscle has a '*mind*' and a physiological history of its own.

Figure 6: Historical snippet

An After-dinner Talk by Dr. Janet Travell

"I think that I've never seen two people with exactly the same problem, and if you try to analyze the problem in a cookbook style according to a recipe you don't get good results. There are nearly 500 different muscles in the body and multiple factors affecting them -- everybody's different -- no two people have the same thumbprint -- not even identical twins -- nobody in the world. If you take the 26 letters of the alphabet -- how many words can you make out of them? A dictionary three inches thick! And one person is like the French language, one is like the English language, another is like the Spanish, and so on. The real solution is to approach every problem completely fresh with an unbiased mood and with the concept that it's going to be something new. It's very hard to teach doctors that you look at every single person as something entirely individual and unique. You will then solve problems and you find that you leave your mind open and you get clues and cues - listening, watching, looking, and you say, "oh my goodness, I haven't seen that in ages."

Chiropractic reply to Janda's proposition 3

Is Exercise the Best Treatment for Patients with Pain and Muscle Inhibition?

In Janda's physiotherapeutic model of muscle imbalance, the inhibited (weak) muscles are treated with exercise, rocker-boards, wobble-boards, balance-shoes, and mini-trampolines among others. (Page et al. 2010; Morris 2006) The principles of this physical therapy approach to muscular imbalances were based on the work of Bobath and Bobath (1964) who developed physiotherapy programs for children with cerebral palsy.

It should be of major concern that patient compliance, adherence, and participation is quite poor for exercise programs generally, even when the individuals felt the effort was producing benefits. (Liebenson 2019, 2007; Lederman 2010; Chaitow 2008) Most rehabilitation, stretching, and exercise programs report a reduction in patient participation (even when the individuals felt that the effort was producing benefits). (Lewthwaite 1990) It should be noted that no study to date has demonstrated that core stability exercises will reset onset timing of muscle contractions in chronic low back pain patients.

Leading physiotherapists Falla and Hodges have been quite frank in their review of the protocols of using exercise for spinal pain: '*Exercise is the (sic) most effective treatment for the management and prevention of spinal pain; yet on average, it delivers small to moderate treatment effects, which are rarely long lasting.*' (Falla & Hodges 2017.)

For the *Journal of the American Medical Association*, Goertz et al (2018) also found that physical therapy and medical care, when chiropractic care was added, was more effective than the usual physical therapy and medical care combination for low back pain intensity (mean difference, -1.1; 95% CI, -1.4 to -0.7), disability (mean difference, -2.2; 95% CI, -3.1 to -1.2), and satisfaction (mean difference, 2.5; 95% CI, 2.1 to 2.8) at three military medical sites where the comparisons were conducted. (Goertz et al. 2018)

Wigers et al. (1996) found that 73% of patients failed to continue an exercise program when followed up, although 83% felt they would have been better if they had done so. Correcting muscle inhibitions with remedial exercise is also time-consuming, and patients are remarkable in how incorrectly they can perform their exercises! (Liebenson, 2019) The use of advanced smartwatches, smartphone apps, and online software for monitoring physical activity has become increasingly common because of this non-compliance with programmed-exercise issue.

Despite this experts such as Liebenson (20019) and Lederman (2010, 1997) highlight the need to move as rapidly as possible from passive (operator controlled) to active (patient controlled) methods. The rate at which this happens depends in their model largely on the degree of progress, pain reduction and functional improvement.

However the question must be asked: if home exercises are needed for muscles that are still inhibited after in-office assessment and treatment, how effective are the hands-on manipulative treatment modalities being used, if they are being used at all?

Although exercise can increase muscle strength, (Liebenson 2019; Jull et al. 2019; Bearne et al. 2002) few studies have investigated whether this results in an improvement of functional performance and decreases disability. Moreover, these research studies usually involve prolonged and labor-intensive rehabilitation regimes - often patients are required to attend three exercise classes per week for twelve weeks - making them expensive and clinically impracticable, though quite common in our over-priced, under-serviced medical utopias in the western world these days.

An important study by Korthals-de Bos et al. in the *British Medical Journal* (2003) showed that patients who received care from general practitioners for neck pain were randomly allocated to receive 1) manual therapy (spinal mobilization), 2) physiotherapy (mainly exercise) or 3) general practitioner care (counseling, education and medication). Throughout this 52-week study, patients rated their perceived recovery, intensity of pain and functional disability. Manual therapy proved to be the most effective treatment for neck pain. The clinical outcome measures showed that manual therapy resulted in faster recovery than physiotherapy and general practitioner care. While achieving this superior outcome, the total costs of the manual therapy-treated patients were about one third of the costs of physiotherapy or general practitioner care.

As well as being effective, rehabilitation regimes must also be safe. It has been suggested that exercise of inflamed joints might cause joint damage by hypoxic reperfusion injury, (Blake et al. 1989) whereby contraction of muscles acting across inflamed joints raises the intra-articular pressure above the perfusion pressure, precluding the blood supply to the synovium, which becomes hypoxic. The implication is that exercise, even common physical activities, may be inappropriate, dangerous and contraindicated for patients with inflammatory joint conditions, i.e. patients who are in pain and most likely to visit clinicians for treatment.

Although chiropractors employ exercise and rehabilitation programs in their treatment of patients, they focus their unique training and skills on providing structural, nutritional, and psychosocial corrections for the neurological inhibitions found. Furthermore joint dysfunctions that produce muscle inhibition in patients will not be effectively addressed with exercise, stretching, and other non-manipulative modalities. (Dishman et al., 2008)

Chiropractic reply to Janda's proposition 4

Visual Diagnosis of muscular dysfunction – the 'skinvelope' problem

Goodheart, Walther, Kendall, Chaitow and DeLany, Liebenson, Janse, Sutherland, Magoun (Goodheart, 1964-1998; Walther 2000; Janse, 1976; Kendall et al. 2005; Chaitow & DeLany 2008; Liebenson 2019, 2007; Sutherland 1998; Magoun 1976) and many others have written extensively about the closed kinematic chain of the body. As an example, when the foot is in contact with the ground, the foot, leg, thigh, and pelvis make up a modified closed kinematic chain. Imbalance in any part of the chain will cause change in function of the remote portions of the chain; thus extended pronation puts torsion into the leg, thigh, and pelvis, which would not ordinarily be present. Because foot malfunctions lead to instability during gait, compensation patterns emerge that have body-wide implications.

Dananberg (1997) and Simons et al (1999) report that a cascade of myofascial conditions are likely to emerge in the patient with disturbances in foot structure and function, including pain in the low back, thigh, neck and shoulder, knee and foot.

The visual diagnosis of a specific joint or muscle impairment in the foot and simultaneously its relationship to a specific joint or muscle impairment in the hip, shoulder, neck or jaw is fraught with difficulty. (Lederman 2010)

The different elements within the chain of events that a patient performs in front of the examiner occur within a fraction of a second; far too rapidly to be accessed individually in the absence of laboratory tools. Therefore what is actually observed by the examiner who depends upon visual diagnosis of these muscle-joint interactions is the grand total of how rapidly and smoothly a person's global posture moves between two activities – it is almost impossible to make a diagnosis of a specific muscle or joint dysfunction on this basis.

By way of comparison, the AK MMT permits a specific challenge to a muscle or joint in the foot to be immediately followed by another MMT to a distant muscle, thereby making evident to both the physician and the patient the measurable and dynamic interactions going on between two distant structures. The MMT as used in AK makes the diagnosis of these interactions between the joints and muscles of the foot and remote muscles throughout the body far easier.

Dananberg also reminds us that the visual diagnosis of muscular imbalances during gait is difficult. (Dananberg 1997) What is actually observed by the examiner is the big picture of how rapidly and smoothly a person can change between two activities - inaccurate, but for many clinicians in the Janda-universe, apparently good enough. The addition of the AK MMT approach for dysfunction in the strength and movement of the great toe, and testing the muscles that move the great toe during the stance position of gait, as well the influence of *functional hallux limitus* upon remote muscle function throughout the body, is a great help in diagnosing this subtle but critical disorder. (Cuthbert et al. 2018)

In reviewing Janda's writings, I cannot find any instances where manipulation of the foot is recommended to correct its dysfunctions. In fact, throughout his classic textbook on the MMT, Janda does not mention relationships between the muscles found inhibited on MMT and the manipulative approaches that might correct this finding. In his discussion of rehabilitation approaches for patients with foot and leg disorders, sensory-motor stimulation approaches are the primary treatments offered.

However Page et al in their excellent book on *The Janda Approach* cite the large number of manipulative approaches now present in the modern world for muscle imbalance phenomena. Janda's students are making up for their founder's manipulative shortfall.

The term sensory-motor system is very important as Janda (like Goodheart) understood that to split the function of the musculoskeletal system from the central nervous system is wrong. Both parts function as one inseparable functional unit and cannot be sundered. However, the manipulative approach (particularly high-velocity, low-amplitude manipulation) in order to strengthen muscular inhibitions associated with these specific disorders, is underplayed and essentially unspoken in Janda's writing and rehabilitative system of therapeutics.

In the examination of muscular dysfunctions, Janda also points to the existence of oculo-pelvic and pelvi-ocular reflexes. This means that a change in pelvic orientation alters the position of the eyes and visa versa, and to the fact that eye position modifies muscle tone – visual synkinesis – particularly involving the suboccipital muscles (look upward and the extensors strengthen, look downward and flexors prepare for activity, etc.). The implication of modified eye position due to altered pelvic position therefore becomes yet another factor to be considered when unraveling chain reactions of interacting adaptive elements.

'*These examples*' Janda says, '*serve to emphasize that one should not limit consideration to local clinical symptomatology but that we should always maintain a general view*'. This approach is identical to the one developed in applied kinesiology, with the essential addition of a reliable tool for analysis of the effect of '*oculo-pelvic*' and '*pelvic-ocular*' reflexes upon skeletal muscle function. (Already in 1979, Goodheart developed methods for testing these interactions, called '*oculo-basic*' in AK, nearly a decade before Janda described these interactions.) (Goodheart 1979) As mentioned, Goodheart found a diagnostic solution to the visual reflex problems Janda elegantly surmised. (Cuthbert 2006)

Figure 7: AK MMT methods can evaluate whether the sensory inputs coming from the upper cervical spine, the muscles of the neck and the rest of the body are congruent with those coming from the eyes and inner ears.