Integrative Dry Needling for the Upper Extremity and Hand

Pre-course Reading

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INTEGRATIVE DRY NEEDLING

Pre-Course Reading and Review (Foundation Course)

Note: This course has both a written and practical examination on the last day. To receive course credit the participant must achieve an 80% or better on both the written and practical examinations. Each participant is required to complete this pre-reading manual prior to the first day of the course.

Course description:

This specialty course is designed for licensed healthcare professionals who treat neuromusculoskeletal conditions in the upper extremity and hand. The participant will learn IDN's neurologic model of dry needling and how to integrate this physical agent modality into their daily clinical model of treatment. This lab-based course is focused on using dry needling as a tool to manage pain, restore function and to maximize soft tissue healing and elasticity. IDN's peripheral nerve mapping model provides the framework for the clinician to address common neuromusculoskeletal conditions encountered in daily practice such as: tendinopathy, epicondylalgia, tenosynovitis, intrinsic and extrinsic tightness, shoulder rotator cuff/impingement syndromes, nerve entrapments, radicular pain, and OA. The course is designed for clinicians with and without prior dry needling training or experience to attend. The main focus is for the clinician to obtain safe and effective dry needling skills using sound clinical decision-making.

Why Modern Dry Needling is not Traditional Chinese Acupuncture.

By Dr. Yun-tao Ma

Modern dry needling (DN) is not traditional Chinese acupuncture (TCA) because DN practice is based on the laws of modern medicine, while TCA is based on the laws of Traditional Chinese Medicine (TCM).

Modern DN has developed on the foundation of modern Western medicine, which consists of biology, chemistry and physics. Each scientific field may offer DN the information from many sub-fields, such as molecular biology, physiology, pathology, anatomy, kinesiology, and more. Medicine is an applied biology and obeys the laws of chemistry and physics. Science advances when new information obtained is different from the old, the law of science may change, so the laws of modern medicine may change; DN is advancing with science and the new laws of medicine. Thus, DN is dynamically advancing and will not stagnate in its evolution.

Traditional Chinese acupuncture was developed at least 2,500 years ago in ancient agricultural Chinese society. Ancient Chinese believed that there were universal laws that govern the universe, nature, social structure (from family to politics), human body and human medicine and diseases. These laws are in fact a product of the Chinese agricultural civilization and philosophy. The first law of traditional Chinese medicine (TCM) is the Yin-Yang. The second law is the interrelation of five elements. Then there are numerous minor laws that govern the TCM and TCA. The third law is the Qi (vitality, energy) that exists in every entity. If the new information contradicts with those universal laws, the information must be modified to fit into the universal laws. The TCA community believes that Chinese medicine is different from modern medicine and it does not obey modern scientific laws, and the scientific method cannot be applied to TCA. However, the “laws” of Chinese medicine are just human-formed historical concepts, not natural laws. Thus, acupuncture theories, like the central concept of meridians, have been regarded as fact, not theory, and have been humanly kept in its antique form for thousands of years.

DN techniques are based on the laws of modern medicine. Those laws are rules nature must live by. The “laws” that format TCM and TCA are human-created concepts representing ancient human thoughts and bias. This is the fundamental difference between modern DN and TCA though both DN and TCA are clinically effective.

Current research and clinical data have modified the hypothesis and clinical techniques of modern DN. The result: Neurologic Dry Needling (NDN).

1) Research demonstrates the clinical outcomes of needling “official” acupoints and non-acupoints are equally effective. This falsifies the uniqueness of meridians and acupoints.

2) Trigger point hypothesis: Trigger points cause myofascial pain. Based on this hypothesis it is important to identify and deactivate the trigger points. This approach can be clinically effective. However, a non- trigger point approach, such as superficial needling or needling of distant points can also reduce or heal myofascial pain even though the trigger points were untouched during treatment. *Note: Various research sources cite an overlap of the named trigger points and acupoints, ranging from 80% to 100%, claiming they are the same points. Thus, the same scientific discovery in (1) can be applied to the trigger point hypothesis.

3) The clinical evidence demonstrates that healing can be achieved by using both local symptomatic points and distant points.

Based on research and clinical data since the 1960’s, we have falsified the meridian theory, and now we are improving the trigger point hypothesis. The results: IDN / NDN are developed by synthesizing all the DN scientific and empirical data.

Evolution of Dry Needling Models

Empirical Model type	Physiologic features of the model	Weakness of the model	Historical notes
Ancient model	• The model reveals systemic and non-specific effects of needling physiology. • A system of accumulation of ancient and modern clinical data.	• Model development is disadvantages by its philosophy. • Modern medical understanding is irrelevant (some improvement in modern versions). • Complicated out of date theories and unnecessary clinical procedures.	Empirical development: Chinese agricultural civilization at least 2,500 years ago.
Trigger point model **1st generation of dry needling**	• Local muscle patho-histology and patho-physiology of trigger points are emphasized. • Local gross anatomy is emphasized.	• Systemic physiology of needling effect is ignored. • Pain physiology of sensory nerve is underestimated. • Anti-inflammatory physiology of needling is neglected.	Empirical development: 1930s: J Kellgren 1940s: J Travell 1970-2010: J Travell & D Simon
Gunn approach **2nd generation of dry needling**	• Spinal segmental physiology of needling stimulation is emphasized. • Concept of soft tissue dysfunction is considered.	• Non-segmental physiology of needling effect is ignored.	Empirical development: 1970s: Dr CC Gunn
Neurologic Dry Needling **3rd generation of dry needling**	• Integration of all known models. Systemic, segmental, and symptomatic needling is emphasized. • Pain physiology of nervous system and soft tissue dysfunction are inter-connected. • Pain of neurogenic origin is emphasized. • Clinical procedure is comprehensive but simple. • Preventive effect of needling is emphasized. • Quantitative analysis as a prognostic is used.		Empirical development: 1970s: HC Dung 1990s: HC Dung & YT Ma 2000s: YT Ma 24 Homeostatic points.

The Laws of Dry Needling: The reality of dry needling therapy. - Dr Yun-Tao Ma.

There are different modalities of DN and this diversity, in fact, promotes the advancement of DN therapy. As science philosopher Karl Popper indicates - disagreement advances science. However, this diversity often confuses both instructors and students of DN. For example, some instructors believe that only their way is correct and other techniques are not supported by scientific data. This creates uncertainty with students trying to understand which technique is correct when facing different DN paradigms. These laws will help explain the diversity of DN and clarify the confusion.

a. All needling models clinically work. All models are partially truthful models.

b. All theories are tentative and subjective to change as science advances. Differentiation of facts from theories is needed in understanding the therapy.

c. If any scientific researches support one model, in fact, the research will support all models.

d. If any scientific researches denies/falsifies the theory of particular model, it applies to that theory, not the clinical techniques of the model.

e. Each model has its unique benefits and unique limitations.

f. Physiologically all models do not conflict with each other. Thus, it is possible to integrate all models into a new model with new theories.

IDN Conceptual Model of the 3 S's and the 3 I's - Dr Frank Gargano

**I wrote this section as a simple way to describe how we view pain and dysfunction as a systemic issue and how neurologic dry needling can be utilized to address it.

The 3I's describe the "problem" with musculoskeletal pain and dysfunction, ***Inflammation***, ***Irritation***, and ***Inhibition***. The 3I's do not occur in isolation, they are coupled with one another in an injurious situation. Each of the 3I's occur ***locally (Symptomatic)***, ***Segmentally***, and ***Systemically***. If pain and dysfunction are treated solely at the site of the symptoms "you may get to the point but will miss the system," as we like to say. The human body works as an integrated organism and must be viewed and treated in this way. The human body shares neurology and circulation and to believe that an injury remains local to the site, absent an effect on the rest of the body, is limiting your treatment effect. This reminds me of the "guru" based treatment models of the 1980's and 90's. Clinicians would utilize a specific manual therapy paradigm and would become cult-like in their treatment approach, and if it failed the patient was labeled "not fixable". Since that time, the PT profession has become more eclectic in viewing the body as greater than the sum of its parts. This eclectic view is in perfect alignment with the IDN system of dry needling.

The days of focusing on a point as the source of pain and dysfunction are numbered and it is time to move toward a more reliable and global assessment and comprehensive treatment. The global thinking of the 3I's and the 3S's is the model that sums up both the injury of the system and a comprehensive treatment. The dilemma lies in the fact that we do not fully understand the experience of pain, dysfunction or the physiological mechanisms of dry needling. This is today's reality and if we can embrace it, we can continue to move our thinking forward.

The more you read about ***Inflammation*** the more it becomes apparent it is centered at the root of pain and dysfunction in the human body. This creates ***Irritation*** of peripheral nerves that can create the sensory experience of pain and the motor effects that drive neuromuscular ***Inhibition***. If inflammation could be managed more effectively, we may be better able to mitigate its effect on both pain and motor dysfunction. This is not a revelation but a basic fact of treating most musculoskeletal conditions.

Following injury, the ***inflammation*** will reside in ***local*** tissues however, it will quickly become widespread ***(systemic)*** and involve multiple tissues but specifically the nervous system, such as is seen in chronic conditions. ***Neurogenic inflammation*** results from bioactive chemicals activating sensory neurons, which in turn activates the release of sensitizing chemicals from peripheral nerve terminals ***(irritation)***. This bi-directional process from local peripheral tissue to the CNS causes a more widespread inflammatory process. The ***inflammation*** that produces pain and dysfunction can be ***local***, ***segmental*** and or ***systemic***, which is what creates the challenge.

This may explain why the modern continuing education seminars now place more focus on assessing and treating the body as a whole, with less focus on identifying the specific tissue that is at fault. The tissue specific diagnoses that to aim identify the "involved" structure is faulty reasoning and should be reconsidered. Sizer et al.'s paper on sound clinical reasoning outlines the need for a multifactorial construct in encouraging innovative practice (Sizer et al., 2016). Acknowledging the lack of diagnostic accuracy in clinical testing, palpation and even patient report makes treatment design challenging to say the least. That may have led some to attempt to create a cookbook style of treatment where it is assumed that a common grouping of signs and symptoms will all respond to a specific treatment regimen. We all know how that worked out, and essentially lead back to the "not fixable" conclusions for patients that did not fit or respond favorably to the treatment mold they were put into.

IDN's 3S's concept of treatment provides no preconceived notions of the source of the 3I's, instead the 3S's and 3I's provide a foundation upon which to build a treatment plan.

• ***Symptomatic (local)*** - This is certainly the most obvious type of pain and dysfunction to treat as the patient tells you it hurts "here". This is usually an acute to sub-acute injury and the area may be swollen with a loss of motion.

• ***Segmental*** - Manual therapy clinicians understand that when treating musculoskeletal pain and dysfunction the spinal component cannot be ignored. They have been trained to first "clear" the spine to reduce the likelihood of missing a segmental problem based on a peripheral complaint. The ***segmental*** effects of needling help to reduce the symptoms of the ***local (symptomatic)*** points.

• ***Systemic*** - This is where the most confusion and even misunderstanding of mechanism is experienced. In the human body there is shared neurology, circulation and physiology that we cannot separate into pieces or parts. We base our ***systemic*** treatment on homeostatic points. Homeostatic points are key neurological areas in the body that have stronger therapeutic signaling to the CNS and are present in reproducible locations and patterns. The innervation zones of homeostatic points are extensions of major peripheral nerves that are present in consistent locations around the body based on the predictable anatomy of the peripheral nervous system. This is in stark contrast to locating the highly variable myofascial trigger points.

In some patient presentations (acute symptoms) treating just symptomatic points (local) may be all that is needed to get the desired effect. As you move from the acute patient to the sub-acute and into the chronic, the need to expand the treatment methods becomes empirically evident by the reduced clinical results. Assessment tools, such as quantitative sensory testing, may be used to identify the possible central mechanism driving the symptoms. We believe it is relevant to ***address the 3 S's together***, because clinically this approach has a better chance ***to address the 3 I's of pain and dysfunction***.

Determining Dosage of Dry Needling Treatment - Dr Frank Gargano

The current literature is lacking on a definitive and scientific answer to the question of how dry needling dosage is clinically determined. Currently, the answer relies mostly on empirical and historical knowledge. In general, our definition of needling dosage is based on the number and extent of the therapeutic lesion(s) produced from the needle insertions. Therapeutic lesion is defined as the neurophysiological response of the body to the needle penetrations; the more therapeutic lesion(s) produced, the higher the needle dosage delivered. The extent of a therapeutic lesion(s) is not solely dependent on the number of needles used, but also on the gauge of the needle and how it is manipulated while in situ. Based on that description it is understandable that we do not have a standardized nomenclature to quantify the amount of therapeutic lesion required for optimal clinical effect. This is in contrast to exercise or medication prescription that can have a very specific and easy to follow prescription. For example, the always popular, but highly questionable 3 sets of 10 for exercise or take 2 pills 3 times per day. The lack of a standardized system to measure needle dosage leaves us with vague statements such as continue needling "until it stops twitching", "until the energy flows", 'until the muscle relaxes", or the intuitive "until the pain goes away?" This highlights one of the major limitations in dry needling research, lack of identification of the needle dosage used to obtain the clinical effect.

When deciding on the treatment dosage for dry needling there are several patient factors to consider. For the purpose of this paper, I would suggest the following short list of considerations related to needle dosage:

- Age and health status of the patient
- State of the condition being treated (acute/chronic)
- Patient experience with dry needling especially within the last 6 months
- Healing potential based on the IDN Quantitative Analysis

When the subjective and physical examinations have been completed, the decision to utilize dry needling has been made, and patient consent obtained, the next step is determining location and dosage of needling. On the first day of treatment it is recommended to "talk more and needle less." By this we refer to educating the patient about dry needling while also reducing the potential for significant post-needle soreness. Determination of the patient's tolerance and response to needling is a process so being conservative initially is prudent. With that said, the first treatment should have a therapeutic effect but not at the expense of dogma that states the mission is to "eliminate" knots or points at all costs.

There are several techniques of dry needling that can be integrated into each treatment session, each can be used separately or can be used in combination. The following describes the most common clinical applications utilized:

1. **Superficial needling:** The needle is inserted into the dermal and epidermal layers and not into the muscle tissue. This type of needling is the most conservative and very unlikely to cause pain or post-treatment soreness. A minor therapeutic lesion is still produced allowing the patient to receive the neurological and physiological benefits.

2. **Deep Needling:** The needle is inserted directly into the muscle tissue. There are various levels of needle penetration from inoculating just the outer layer of muscle or needling through the entire thickness of the muscle. Depending on how deep the needle is placed will determine the neurophysiological responses that result because penetrating deeper provides greater stimulation to the additional soft tissue and neurological levels. When the needle is set without needle manipulation it would be considered a basic deep needling technique of low dosage but is a progression of dosage over superficial needling.

3. **Needle manipulation:** The amount of dosage (therapeutic lesion) obtained is also based on the variables of speed, amplitude and intensity of the needle manipulation or movement. This is a progression from basic deep needling and has several manipulation techniques that can be utilized depending on the specific goals you are trying to accomplish. In a subsequent paper, I will describe in detail these different techniques and their specific uses:

- Needle rotation - The in-situ needle is rotated until a definitive endpoint is reached signifying the soft tissue has completely "wound" around the needle shaft inducing additional therapeutic lesion.
- Needle tenting - When the soft tissue is wound tightly around the needle it is pulled up causing a traction or stretch of the tissue inducing additional therapeutic lesion.
- Needle pistoning - A high velocity up and down conical movement of the needle. There are various levels of intensity of pistoning that are dependent on the speed, and amplitude of the needling. The higher the speed and amplitude the larger the therapeutic lesion produced. Pistoning is considered the most aggressive needling technique and produces the greatest post-needling soreness.

4. **Electrical Needle Stimulation (ENS):** ENS delivers a mild electrical stimulation to soft tissue via attaching alligator type clips onto the solid monofilament needles. ENS is a progression of dosage of manual needling by inducing rhythmic vibrations (of non-contractile tissue) and repeated muscle contractions. Because the needle is in situ the repeated muscles contractions will induce additional lesion.

5. **Time in situ.** Empirically, the length of time a needle is left in situ (without manipulation) does not influence the dosage, if we define dosage as amount of therapeutic lesion produced.

Dosage determination: Research does not currently exist that provides a definitive guideline of needling dosage for specific diagnoses or conditions. Dry needling is a non-specific treatment that relies on the body's ability to self-heal the needle induced therapeutic lesions via multiple physiological processes. Because each patient's situation, injury and condition are unique, trying to determine the correct dosage is complicated to say the least. Each clinician has their opinion on proper dosage, based primarily on their clinical experience and empirical evidence. This is important information, but it is not easily quantifiable to other patients or for use by other clinicians. We propose a clinical model that quantifies needling dosage into more general categories of low, moderate or high. Each category has an increasing number of needles and progressively more aggressive needling techniques that generate larger therapeutic lesions.

Low Dosage: Applies to the initial treatment session for all patients. Also, patients with compromised physical conditions, low healing potential or are at an advanced age require a cautious start. The following is recommended for this category:

- Superficial needling techniques
- Basic deep needling technique (no needle manipulation)
- Number of needles: 5-10 needles

Moderate Dosage: Applies to patients that have had prior needling treatment (low dosage) with a positive reaction within the last 6 months. They are in relatively good physical condition and have been assessed to have good healing potential.

- Deep needling can now include needle manipulation techniques of moderate intensity, which may include pistoning, rotation and or tenting techniques.
- ENS can be introduced and provided for up to 5 minutes of active muscle contraction.
- Number of needles: Up to 20 needles

High Dosage: Applies to patients that have had significant experience with dry needling treatment and are likely healthy, active individuals with excellent healing potential.

- Deep needling with high intensity needle manipulations, which may include pistoning, rotation and or tenting techniques.
- ENS treatment time can be extended up to 10+ minutes and multiple areas can be treated in one session.
- Number of needles: 21+ needles

Summary: In this short paper we provided general categories for the prescription of dry needling dosage focusing only on the induced therapeutic lesion. What needs to be addressed in a subsequent paper is the patient's perception and response, which are linked to dosage and ultimately the therapeutic outcome.

The current guidelines are intended to help all clinicians, but specifically clinicians new to dry needling treatment, with the clinical decision-making related to the original question of "How many needles should be used?" These guidelines were never intended to be the definitive answer as it is not currently possible to specifically quantify the non-specific and systemic modality of dry needling. As research progresses and physiological healing processes are better elucidated, we may be better able to quantify the amount of therapeutic lesion required to get the desired treatment effect.

Quantitative Sensory Testing and the Efficacy of Dry Needling Treatment

Quantitative Sensory Testing (QST) is the most widely used paradigm to assess central hypersensitivity, it consists of the application of a standardized stimulus to a peripheral (nerve) tissue and recording the patient's response.[1] The paradigm relies on the assumption that a non-painful stimulus, when applied to a non-injured tissue, will evoke pain only if central nociceptive pathways are hypersensitive. Determining if the hypersensitivity (neuropathic pain) is caused by a peripheral mechanism, central mechanisms, or a combination of the two, can be difficult clinically due to their interdependence.

Neuropathic pain is defined as *"Pain caused by a lesion or disease of the somatosensory nervous system*".[2] The somatosensory nervous system provides information about the body including skin, musculoskeletal, and visceral organs, which has clinical relevance for the clinicians providing neuromusculoskeletal treatment, specifically dry needling treatment. Neuropathic pain may be spontaneous or evoked, as an increased response to a painful stimulus (hyperalgesia) or a painful response to a normally nonpainful stimulus (allodynia).[3] This definition provides a broad concept and shares common features of chronic musculoskeletal pain with other pain conditions e.g. neuropathic, and visceral.

It is an ongoing clinical challenge to make a prognosis on the potential outcome of treatment and certainly the time required to achieve a treatment result. QST has shown some promise in assisting clinicians, specifically neuromusculoskeletal practitioners, in estimating these difficult prognoses.[4] In this paper an overview of a novel bedside (IDN) QST exam is provided, and how it can be used as a part of comprehensive evaluative plan in estimating if dry needling would be of clinical value as a part of the treatment plan.

Application: The stimulus applied in QST can be chemical, thermal, electrical, vibrational or physical (pressure) to evoke a response. In the IDN bedside QST exam the application of a physical pressure is the most readily available stimulus that can be trained and practiced achieving an acceptable level of intra-tester reliability.[1] The IDN bedside QST exam involves applying a consistent pressure to 16 standardized assessment points in two peripheral nerve pathways, one in the upper extremity and one in the lower extremity. In the IDN system, homeostatic neuro-trigger points are defined as areas of increased sensitivity that are universal in all humans, and are proposed to be of central origin based on meeting the following criteria:

1. Homeostatic neuro-trigger point sensitivity must be present bilaterally and symmetrically
2. Homeostatic neuro-trigger points develop in predictable locations and in a predictable sequence in all humans. The sequence of peripheral sensitization is the result of anatomical, physiological, biomechanical and behavioral factors
3. Homeostatic neuro-trigger points develop slowly over time based on intrinsic and extrinsic factors, which may represent the patient's health condition and healing potential
4. Homeostatic neuro-trigger point sensitization occurs linearly along nerve trunks in the limbs and occurs as an area or patch on the torso and face.
5. The sensitivity and specificity (size/area) of homeostatic neuro-trigger points are highly dynamic.

The Radial and the Saphenous nerves were chosen to be assessed because they are the first peripheral nerves to become sensitized in the upper and lower extremities respectively.[5,8] Once the proper application of the stimulus is taught[6], the examiner applies the stimulus (pressure) down the standardized nerve pathways (homeostatic points) in all four extremities for a total of 16 possible positive findings.

Because the IDN bedside QST exam is both an objective tactile interpretation by the examiner, and a subjective report of discomfort by the patient, a positive finding requires both. All positive findings are calculated with the range being from 0 to 16. The greater the number of positive findings the greater the correlation to the sensitization being centrally mediated.[5,7-9]

To make the IDN bedside QST exam of clinical utility the following was considered:

1. Applicable to every patient
2. Reproducible with any patient for any condition
3. Able to be administered by all clinicians who are properly trained
4. Testing is of short duration
5. Quantitative results that can assist in clinical decision making
6. Efficacy of dry needling treatment

The difficulty in assessing pain, and certainly the measurement of it, relates to its complexity and subjective nature. When we measure pain clinically, we invariably target subjective pain because it is perceivable and easy to find. Objective pain is not perceivable, patients that have objective pain will not know they have it unless it is triggered. Objective pain is, by definition, a passive neuro-trigger point sensitivity that is only perceivable when palpated. It is objective pain that is assessed in the IDN bedside QST exam because it is relatively stable in intensity and location (radial and saphenous nerve pathways). For this discussion objective pain is directly related to the level of sensitivity being maintained within the homeostatic points via central processes.[7] In other words, homeostatic points are objective pain, which are centrally mediated. The greater the number of sensitized homeostatic points a patient carry is directly related to their degree of central sensitization, which in turn influences the manageability of their symptoms and efficacy of the dry needling treatment.[5,7,9]

Interpretation:
As is the case in many bedside clinical tests, it is not possible to make definitive statements, this certainly applies to making estimates on the presence and degree of central sensitivity. That said, central sensitivity has been extensively investigated in humans.[4] When exaggerated pain responses and expansion of pain areas occur after limited tissue damage it is reasonable to infer that it is the result of enhanced nociceptive processes within the central nervous system[1,7,10,11] Unfortunately, we cannot say much more. For instance, clinicians are still unable to say whether, or to what extent pain hypersensitivity is the result of psychosocial factors.

QST is primarily a measure of pain sensitivity and does not allow conclusions to be drawn on the causes, mechanisms or location (peripheral or central) of the underlying hypersensitivity. For example, if a patient with shoulder pain did a vigorous run the morning of the IDN bedside QST exam they would experience exercised induced inflammation, which sensitizes the peripheral nervous system leading to false positive QST findings. Recognizing the limitations, and understanding the intent is to only discover centrally mediated findings, the IDN bedside QST exam was modified as follows:

- Patients should only be assessed when at a resting baseline of physical activity to minimize acute inflammation influencing the findings.
- Pain medications and chemicals affecting consciousness, such as alcohol or marijuana, may influence the accuracy of the patient's response.
- The assessment of both upper and lower extremities provides a better systemic view of patient condition.
- Assessment of multiple points linearly down the nerve path gains insight into the degree of sensitivity.

The interpretation of the IDN bedside QST exam is empirically based on tens of thousands of patient examinations and treatments dating back from the 1970's to today.[5,7,8] Founded in the premise that when the human body is under stress from injury, disease, surgery or other physiological events, inflammation increases causing peripheral sensitization and likely central effects. In patients presenting with chronic pain, this peripheral sensitization profoundly spreads, represented clinically as inflamed sensory nerves (homeostatic

points). [6,7] The number of positive findings (peripheral sensitization) discovered during the IDN bedside QST exam is an indicator of the severity of inflammation in the body. The greater the number of positive findings is an empirical estimate of how many dry needling treatment sessions are needed to reduce the systemic inflammation and how long the pain relief can last.

Research

QST has demonstrated potential benefits when compared with traditional neurological diagnostic tools. For example, around 80% of the peripheral nervous system consists of small nerve fibers[12] however, traditional diagnostic methods (e.g. electromyography, nerve conduction velocity, and evoked potential), primarily focus on the large nerve fibers. [11,12] Deep tissue pain sensation transmits through small caliber A-delta (group III), and C fibers (group IV). [13] QST can target these fibers by using frequencies that target small fibers (e.g., current perception threshold and vibratory perception threshold) or sensory stimuli (e.g., pain and temperature) that are preferential to these fibers. This lends itself to bedside assessment using pain, generated by the examiners pressure, as the measure. Potential disadvantages are that the specificity of these responses has not been adequately demonstrated, and this testing is not completely objective because pain is subjective by nature requiring the patient to provide a voluntary response. Considering the currently used methods of visual analog scales and pain questionnaires, the IDN bedside QST exam may provide a more objective measurement of neural sensitivity.

In the literature there is strong evidence for central hypersensitivity (abnormal pain response) being a prognostic factor for poor outcomes in chronic musculoskeletal pain.[4,14] The evolution of pain theory and evidence of a central component of post-injury pain hypersensitivity implicate central sensitivity in musculoskeletal pain mechanisms.[10] Involvement of the central nervous system in musculoskeletal pain mechanisms (specifically in chronic or maladaptive pain) is emerging as a new target area for treatment. Interestingly, acute and chronic pain have similar mechanisms and the temporal relationship may be more related to central mechanisms in making the transition from acute to chronic pain. This suggests that early detection might allow clinicians to make a more accurate prognosis for their patients.

Clinically, a patient who has been injured in a motor vehicle accident (MVA) is a good example of persistent pain complaints lasting well past the timeframe that tissue healing is expected to be complete. There is research to show this persistent pain, specifically in post MVA's, to be a function of neurologic sensitivity being maintained within the nerve structure secondary to the early vascular changes resulting from the injury.[11,15] Patients present with cervical and upper extremity symptoms that are not localized to specific dermatomes or myotomes. A likely explanation for these diffuse symptoms is the development of central sensitivity driven by the persistent nociceptive barrage into the dorsal horn. [14,16] Treatment that focuses solely on the peripheral driver misses the complexity of the systemic involvement.

A systematic review and meta-analysis[4] demonstrated a predictive relationship between baseline QST, a measure of pain hypersensitivity, and musculoskeletal pain and disability at follow-up. These studies showed this predictive relationship across multiple musculoskeletal conditions (OA, LBP, WAD, post-operative pain) affecting different anatomical sites (knee, hip, low back, neck, shoulder), and across different QST modalities and study contexts (cohort studies and RCTs). This review demonstrated that pain hypersensitivity predicts prognosis and that QST might help identify patients who could most benefit from interventions aiming to improve pain and disability. Clinically identifying which patients might be at particular risk of a poor outcome is important in order to identify those who are most likely to benefit from treatment. At a minimum this allows the clinician to have an informed discussion with the patient about the expected prognosis and potential treatment outcome prior to initiation of care.

Clinical Relevance:

QST is a safe, simple and useful bedside tool to determine the potential benefit of engaging in a treatment plan and the potential treatment outcome. The IDN bedside QST exam is not intended to differentiate diseases or to investigate the mechanism of diseases. The goal of the IDN bedside QST exam is to provide the information necessary for evaluating the self-healing potential of a patient and the projected efficacy of dry needling treatments. IDN bedside QST exam accompanied by a full patient history, orthopedic examination, diagnostic

testing, and the clinician's experience and intuition provides a more inclusive view of patient condition and prognosis.

Future studies are needed to determine the reliability of specific QST approaches and establish clinically meaningful thresholds in specific pathologies in order to validate QST as a clinical decision aid for neuromusculoskeletal conditions.

Dr. Frank Gargano, PT, DPT, CIDN, MCTA, CMP

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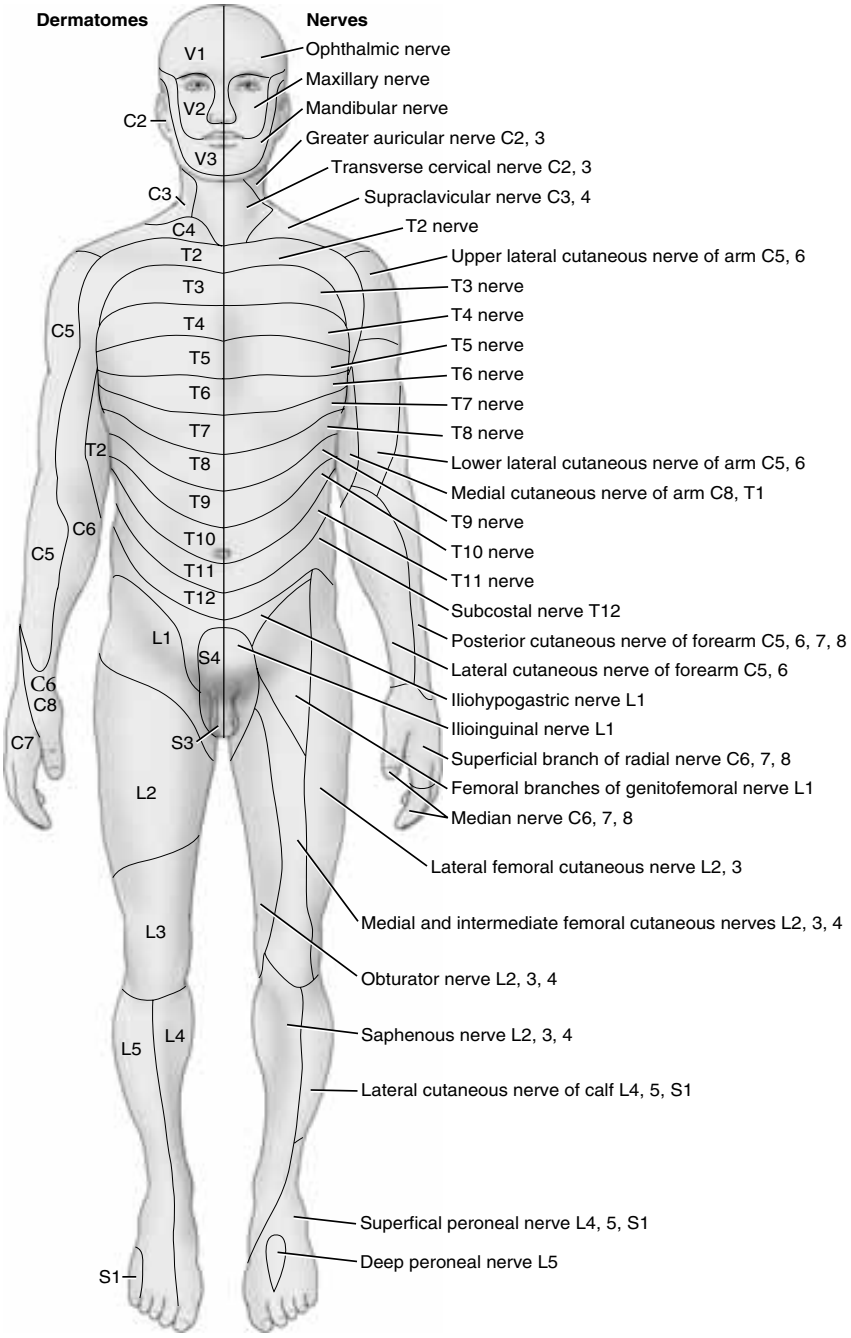
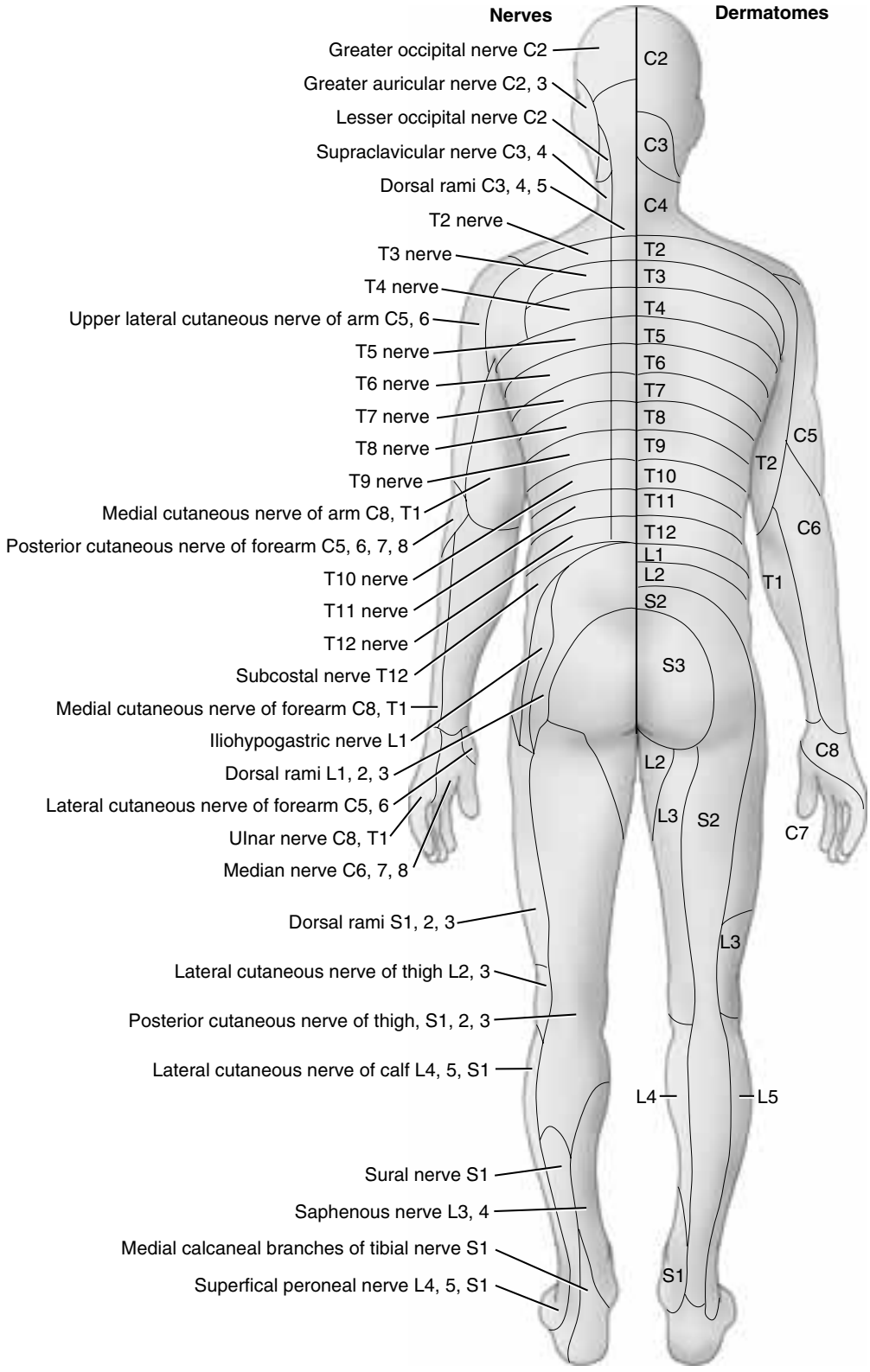
Review the named nerve distributions



Review the named nerve distributions



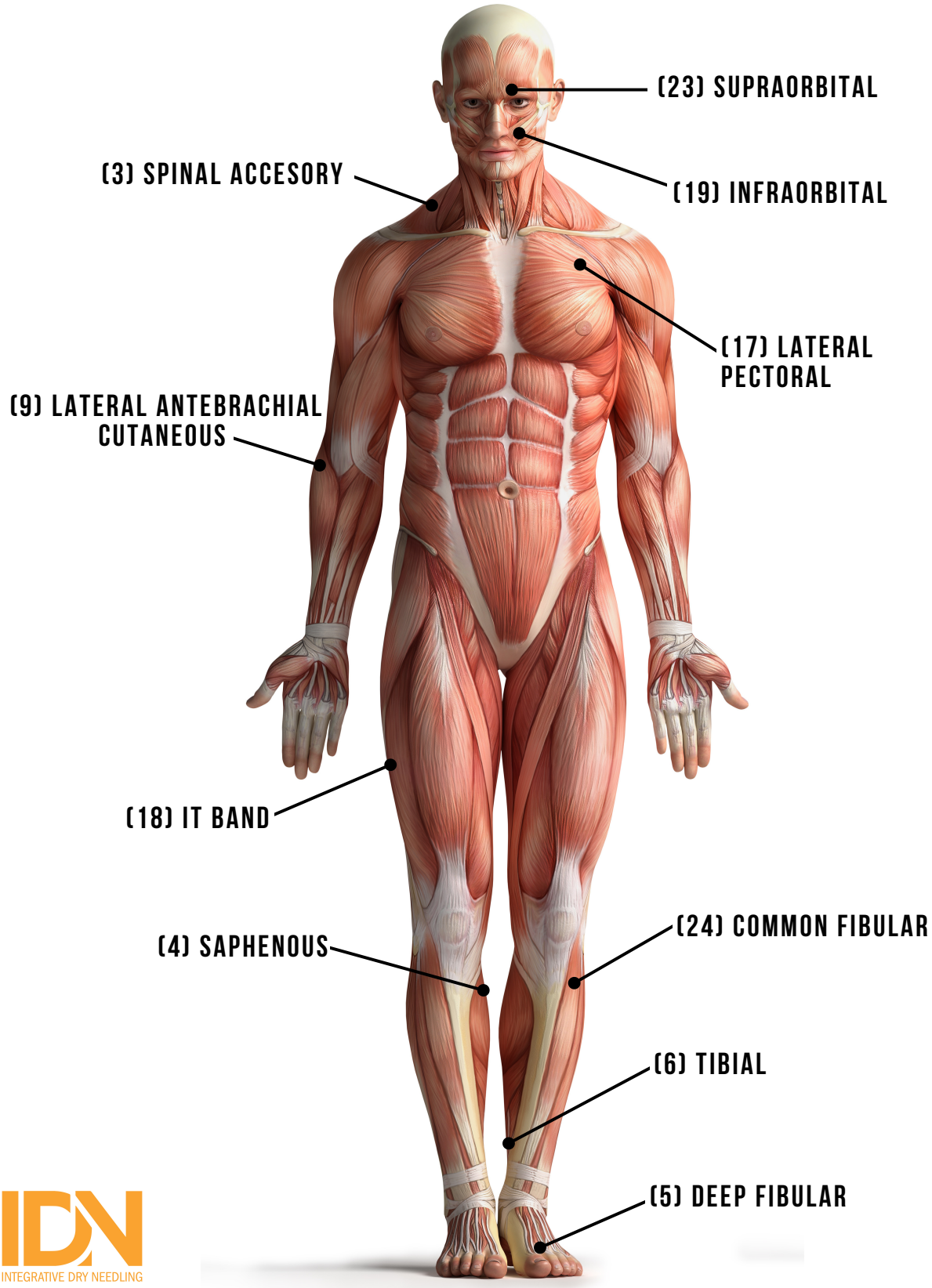
Upper Extremity Homeostatic Neuro-Trigger Points(HNTrP)

KEY: There is a total of 24 homeostatic neuro-trigger points located throughout the body- below are the relevant upper extremity/shoulder girdle homeostatic neuro-trigger points that will be covered in this course. The length and depth of the needle penetration is variable based on patient size and the intended therapeutic result. In the extremities, different needling techniques can be safely utilized from superficial to deep (including to the depth of bone). This decision is based solely on the specific need of the patient and your therapeutic goal for the treatment.

1. Deep Radial	The deep radial nerve homeostatic point is 2-finger widths distal to the lateral epicondyle in the groove under the extensor bulk.
Needle direction	Perpendicular to skin toward the radius
Needle depth	*Depth is variable dependent on patient size and clinical intent.
Special notes	Upper extremity Quantitative Analysis point.
3. Spinal Accessory	Mid-point between the acromion and 7th cervical vertebra on the anterior aspect of the upper trap
Needle length	25-50mm / 1-2 inch
Needle direction	Posterior to anterior, or anterior to posterior, slightly cephalic and always directed toward the clinician's palpating finger
Needle depth	Insert needle into muscle bulk until detected by palpating finger on opposite side
Special notes	LUNG FIELD SAFETY. Must hold muscle between thumb and fingers while needling. Needle IN/OUT +/- pistoning. Release muscle AFTER needle removed.
4. Saphenous	Located in a 'box' below the tibial plateau and on the medial side of the tibial shaft. Palpate for the most tender spot within the box.
Needle direction	Perpendicular to skin
Needle depth	*Depth is variable dependent on patient size and clinical intent.
Special notes	Lower Extremity Quantitative Analysis point.
8.Suprascapular (Infraspinatus)	Bracket the medial border and lateral border of the scapula with your thumb and middle finger; center your index finger between them. (Center of the scapular fossa)
Needle length	25-50mm / 1-2 inch
Needle direction	Perpendicular to skin
Needle depth	As deep as necessary, may go to the bone
Special notes	Care must be taken to verify that you are over the scapula and not medial to it!
9. Lateral Antebrachial Cutaneous	HNTrP located on the lateral elbow crease medial to the extensor bulk lying over the radial head.
Needle direction	Insert the needle medial to extensor muscle bulk on the lateral aspect of the elbow crease perpendicular to the skin toward the radial head
Needle depth	*Depth is variable dependent on patient size and clinical intent.
Special notes	
12. Superficial Radial	Located between the 1st and 2nd metacarpals at the midpoint of the interosseous muscle bulk
Needle length	25mm / 1inch
Needle direction	Perpendicular to skin.
Needle depth	As deep as necessary, until detected by palpating finger on palmar aspect of hand.
Special notes	Use clinician's fingers to palpate palmar aspect of hand between 1st and 2nd MC while inserting.

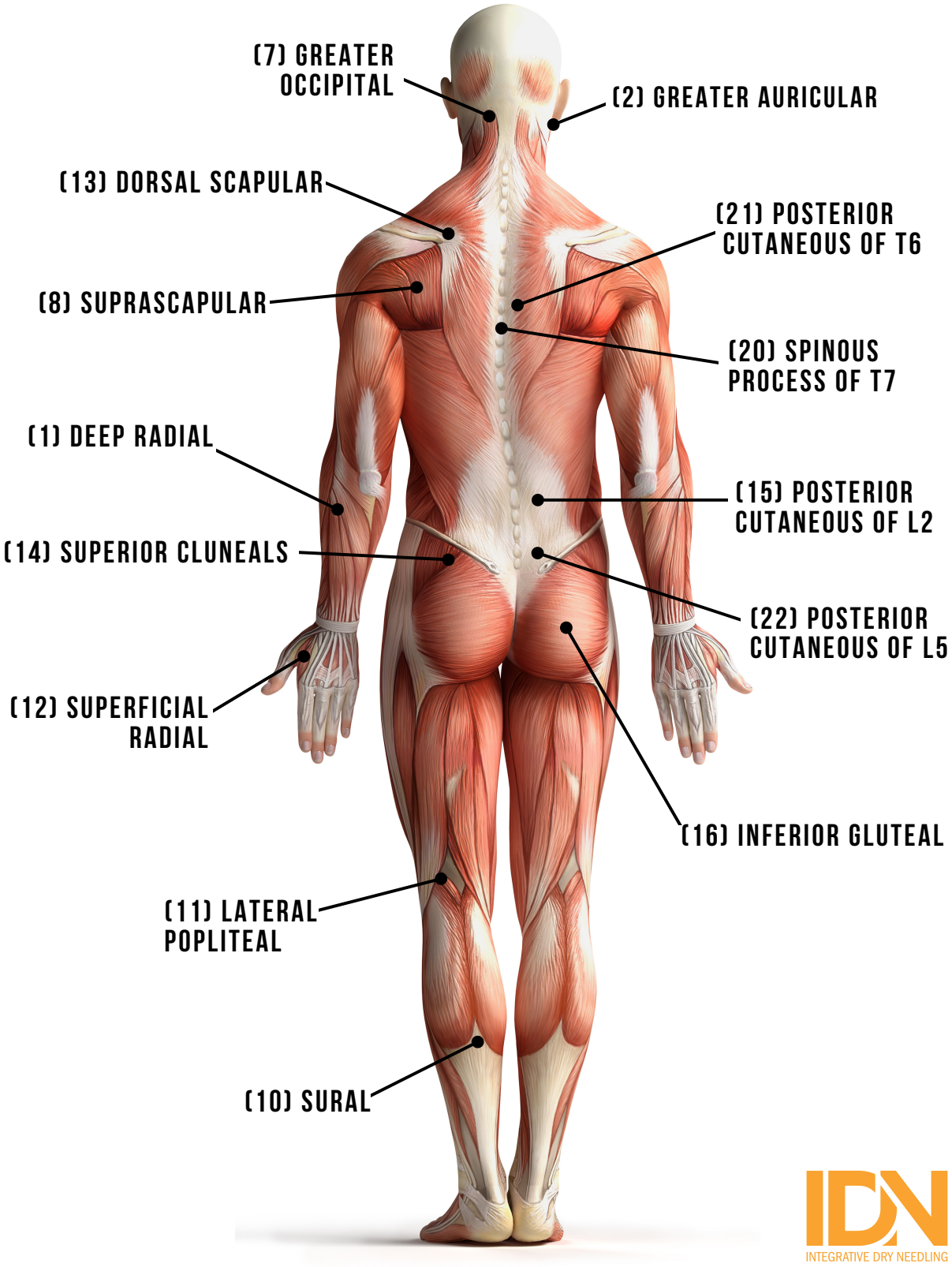
13. Dorsal Scapular	Locate the superior angle of the scapula, the HNTrP is in the levator scapulae insertion.
Needle length	Up to 25mm / 1inch
Needle direction	The needle should be directed from the medial aspect of the superior angle of the scapula moving laterally away from the thorax, assuring the needle point is over the bony backdrop of superior angle of the scapula.
Needle depth	Assure the needle point is directed toward the bony backdrop of the superior angle of the scapula. It is also acceptable to grasp the soft tissue and lift awayfrom the thorax and needle as described above.
Special notes	LUNG FIELD SAFETY. Needle IN/OUT and verify that you are needling from medial to lateral toward the superior angle of the scapula.
17. Lateral Pectoral	HNTrP is located 2 finger widths inferior and perpendicular to the center of the clavicle (anatomical point only-not directly needled instead using innervation field)
Needle length	50mm / 2inch
Needle direction	Medial to lateral direction targeting the lateral third of the pectoralis major (innervation field) aiming the needle tip toward the clinician's palpating fingers.
Needle depth	Until the tip of the needle is detected by the palpating fingers under pectoralis major. It is not necessary to needle the anatomical point of the lateral pectoral homeostatic point.
Special notes	LUNG FIELD SAFETY. Must hold pectoralis muscle between thumb and fingers while needling, always identify the rib cage and needle parallel, never perpendicular, to it. Needle IN/OUT +/- pistoning. Release muscle hold AFTER needle is removed. Care must be taken when IMPLANTED DEVICES (tissue or other device) are present and may be prudent not to perform dry needling.

HOMEOSTATIC NEUROTRIGGER POINTS



IDN
INTEGRATIVE DRY NEEDLING

HOMEOSTATIC NEUROTRIGGER POINTS



IDN
INTEGRATIVE DRY NEEDLING

Blood Borne Pathogens and Universal Precautions

The information in this lecture has been drawn from the Centers for Disease Control and Prevention (CDC), World Health Organization (WHO) and Occupational Safety and Health Administration (OSHA). This information has been adaptedto the specific practice requirements of dry needling therapy. We suggest that you periodically consult the websites of the CDC and OSHA for new information, recommendations and/or requirements.

Table of Contents

Part 1: Knowledge of Bloodborne Pathogens

1.1 Introduction: Concept of infection

The following information is related to clinical practice of dry needling therapy. The human body is constantly exposed to infectious agents, on the skin, on mucous membranes of the eyes, in the mouth, respiratory passageways, urinary tract, respiratory passageways, urinary bladder, and colon. In healthy persons, these infectious pathogens do not cause infection as they are called normal flora. But these agents can cause diseases if they are introduced to other tissues or the immune system is ineffective in controlling the infectious agents. In addition, if a person is intermittently exposed to virulent bacteria or viruses from outside the body, specific infections like pneumonia, streptococcal and staphylococcal infections may occur.

Evolving with these pathogens, our body has established natural barriers to prevent infection. The barriers include intact skin, healthy respiratory mucosa, which expel inhaled pathogens, stomach acid that kills swallowed bacteria, cleaning effect of tears, urine, and acidity of vaginal secretions. Microbes can enter the body through a cut or wound of the skin, orifice (mouth, nose, urethra, etc.). There are many potential sources of infection in a clinic office, such as body secretions (sweat, nasal fluid, saliva, blood), dust, clothing, furniture, etc. Patients can get infected in a clinic office through two pathways, autogenous infections and cross-infections.

Autogenous infections

The infectious agents the patient is carrying cause this pathway of infection. It may happen in two ways in dry needling therapy. Firstly, if the same needle is used in two locations A and B, the bacteria on location A will be transferred to location B, which may cause an infection on B location. To prevent it, always use single-use and disposal needles. Secondly, for example, if the abdominal needling punctures the peritoneum and the intestine, the bacteria normal to intestine now will invade the abdominal cavity potentially causing an infection, such as life-threatening peritonitis. To prevent this occurring the guidelines of needling safety should be exactly followed.

Cross-infections

The infection is acquired from other sources, such as from other patients or from practitioner or by the environment. Infectious agents can travel from one host to another in a variety of ways, the media to transfer the agents can be bodily fluids, dust or droplets of moisture in the air or on the surface of office furniture, or personal contact. If the density of an infectious agent is high enough, it may cause infection even in healthy persons. The most serious infection acquired in a medical office is hepatitis B virus and HIV. Tuberculosis (TB) is another concern.

To prevent cross-infection, the proper guidelines will be provided in the next section. But as a rule, dry needling should not be used in patients with infectious diseases, especially patients with serious infections. Before treatment, a precise medical history should be obtained.

1.2 Bloodborne pathogens

Bloodborne pathogens are infectious microorganisms in human blood that can cause disease in humans. These pathogens include, but are not limited to, hepatitis B (HBV), hepatitis C (HCV) and human immunodeficiency virus (HIV).

Part 2 Bloodborne Pathogens: Hepatitis

2.1 Hepatitis: CDC Recommendation

The CDC states that the highest risks occur during the professional training period; therefore, vaccination should be completed while in school. [1] When an employee hired for a position of high risk of infection to hepatitis refuses to be vaccinated, this employee should be required to complete and sign a document stating that he or she understands the risk of not being vaccinated and is refusing the vaccination despite the risk to acquire Hepatitis.

2.2 Hepatitis

Hepatitis means the inflammation of the liver. There are five types of hepatitis viruses: A, B, C, D, and E. Hepatitis A and E are transmitted through fecal-contaminated water and food. The others are transmitted by blood and sexual contact. For our practice, we will focus on hepatitis B and C, which are known to be blood-borne infections. For information on the other types of hepatitis consult the CDC website. (www.cdc.gov)

2.3 Hepatitis B (HBV)

Hepatitis B virus (HBV) is one of the bloodborne pathogens affecting office practice. HBV can cause lifelong infection, cirrhosis, cancer of the liver, and liver failure. Chronic infection is more likely to develop in persons infected as infants or young children; adults may have higher rates of new infections and acute infection. Health care professionals can have HBV immunity if they receive the HBV vaccine.

Transmission of HBV

HBV is contagious through contact with contaminated blood and body fluids, such as sharing living/working space and daily life utilities like eating utensils, toothpaste, and participating in high risk behaviors (unprotected sex, etc.).

It is estimated that there are 1.25 million Americans chronically infected with HBV, of whom 20-30% were infected since childhood. When treating patients, the medical history should be thoroughly recorded. Some HBV patients have more virus in their blood (source patients) so they are more likely to transmit diseases. Healthcare professionals have significant risk of exposure to HBV. The risk of HBV infection in the office is primarily related to the degree of contact with blood of the source patients. Healthcare professionals are at a higher risk of HBV infection than the public if their work involves occupational exposure to blood and body fluid. It should be noted that the unintentional injury of a healthcare worker from a needle stick or cut by a contaminated instrument could be the mode of transmission from the source patient.

HBV Infection Process

After initial infection, the incubation period for HBV is 50 to 180 days. During this period, the infectious virus appears in the blood, saliva, urine, feces, semen, tears and even sweat. The carrier during this period has no symptoms but can spread the virus. The early symptoms begin with mild flu-like signs, fever, chills, nausea, general fatigue, insidious onset of anorexia, joint pain or abdominal pain, skin rash and diarrhea. These symptoms may last two to six weeks. After the initial symptoms, extreme fatigue and depression may follow for several months. Approximately 30% of carriers have no signs or symptoms. Fully 70% of HBV patients that have recovered from the symptomatic stage are still infectious for more than three months after the symptoms have subsided. If a healthcare worker is infected, he may unknowingly transmit HBV to patients or office staff. The infected clinicians should not work but consult with a physician for treatment until fully recovered.

Precautionary Measures

High standards of office hygiene and clean needle technique should be strictly followed. For dry needling therapists, patients with any type of bloodborne pathogen should not be treated with dry needling and the patients should be referred a physician. Single-use sterile disposable needles should always be used and after use should be disposed of properly according to specific state guidelines.

Treatment of HBV

There are established pharmaceuticals to treat hepatitis B. The treatment of acute stage is mainly symptomatic. HBV patients should be evaluated by their physician for liver disease.

2.4 Hepatitis C (HCV)

Hepatitis C virus (HCV) infection is the most common chronic bloodborne viral infection in the United States. A national survey of the civilian, non-institutionalized U.S. population found that 1.8% of Americas have been infected with HCV, and 75-85% of them are chronically infected. Many of those infected are not aware of their infection resulting in chronic liver disease that may not become apparent for 10-20 years. Those with the highest risk of HCV infection are drug users. Sixty percent of individuals with a history of injection drug use are infected. 15% are infected through sexual intercourse. About 10% of those infected have no recognizable source of infection. There is a risk of occupational exposure for HCV if the healthcare workers are exposed to large amounts of blood (surgery, hemodialysis). The incubation period of HCV is 20 -90 days, with most cases occurring 5-10 weeks after being infected. After infected, the period of communicability can extend from one week through the chronic stage. The onset is insidious, and the symptoms may

include anorexia, vomiting, jaundice, and general fatigue. The course is similar to HBV but lasts longer.

Treatment of HCV

Therapy for HCV is changing rapidly. Treatment is based on liver enzyme levels, genotype of infected virus, and liver conditions. Usually the treatment may extend from 6 months to 2 years.

2.5 Other Types of Hepatitis

Other types of hepatitis, hepatitis A (HAV), hepatitis D (HDV), and hepatitis E (HEV) are not bloodborne infections. These conditions are not discussed here. Their information can be obtained from the CDC website. (www.cdc.gov)

2.6 Chronic Carriers of Hepatitis

Chronic carriers of HBV and HBC can spread the virus through bodily fluid and excretions. They are classified into two categories: Chronic persistent and chronic active. The former is asymptomatic or has minimal symptoms but can infect others. The latter shows progressive symptoms such as malaise, general fatigue, anorexia, jaundice and weight loss. Five to ten percent of HBV and 75-85% of HCV patients develop the chronic condition. Thus, as a general precaution, chronic carriers of hepatitis should not be treated with dry needling.

Table 1: Brief Summary of Hepatitis Characteristics

Hepatitis	Incubation	Transmission	Onset	Chronic
B	50-180 days	Bloodborne	Insidious	Depends on age group
C	20-90 days	Bloodborne	Insidious	60-70%
A	15-50 days	Fecal-oral	Abrupt	No
D	Unknown	Unknown	Unknown	Unknown
E	15-60 days	Fecal-oral	Abrupt	No

Table 2: Key Facts of HBV and HCV

	Hepatitis B	Hepatitis C
Number of US cases	Estimated 1.2 million people living with chronic Hepatitis B. About 19,800 new cases/year	Estimated 3.2 million living with chronic Hepatitis C. About 29,700 new cases/year
Key facts	About 2 in 3 people do not know they are infected by HBV	About 50% of infected do not know they are infected.
How long does it last?	From a mild illness, lasting a few weeks to a serious life-long or chronic condition.	From a mild illness, lasting a few weeks to a serious life-long infection. Most infected become chronic Hepatitis C.
How is it spread?	Exposure to blood, semen, other body fluids of the infected. Sharing personal items. Outbreaks from poor infection control in healthcare facilities.	Exposure to blood or body fluids of the infected. Sharing personal items. Outbreaks from poor infection control in healthcare facilities.
How serious is it	15-25% of infected patients develop chronic liver disease, cirrhosis, liver failure, or liver cancer	75-85% of infected develop chronic infection. 5-20% develops cirrhosis. 1-5% will die from cirrhosis or liver cancer.
Treatment	Medication and supportive care Some chronic patients are treated with antiviral drugs.	Acute: antiviral and supportive care. Chronic: antiviral drugs

Reference: http:// www.cdc.gov/ncidod/ diseases/ hepatitis

Part 3 Bloodborne pathogens: Human Immunodeficiency Disease (HIV)

3.1 HIV Basics

HIV virus can lead to acquired immunodeficiency syndrome, or AIDS. Unlike some other viruses, the human body cannot get rid of HIV. That means that once you have HIV, you have it for life. HIV spreads through body fluids that affect specific cells of the immune system, called CD4 cells, or T cells. Over time, HIV can destroy so many of these cells that the body can't fight off infections and disease.

Approximately 34,800 new HIV infections occurred in the United States in 2019. Annual infections in the U.S. have been reduced by more than two-thirds since the height of the epidemic in the mid-1980s. Further, CDC estimates of annual HIV infections in the United States show hopeful signs of progress in recent years. CDC estimates show new HIV infections declined 8% from 37,800 in 2015 to 34,800 in 2019, after a period of general stability.

At year-end 2019, an estimated **1.2 million people in the United States aged 13 and older had HIV in the U.S.**, the most recent year for which this information is available. (HIV.gov)
According to the latest CDC data:

- About **13% of people with HIV in the U.S. don't know it and so need testing**. Early HIV diagnosis is crucial. Everyone aged 13-64 should be tested at least once. People at higher risk of acquiring (or exposure to) HIV should be tested at least annually. Sexually active gay and bisexual men may benefit from more frequent testing (e.g., every 3-6 months).

- According to another CDC report, of the estimated 1.2 million people with HIV (diagnosed and undiagnosed) in 2019, **about 65.9% received some HIV care, 50.1% were retained in care, and 56.8% were virally suppressed or undetectable**. Having a suppressed or undetectable viral load protects the health of a person living with HIV, preventing disease progression. There is also a major prevention benefit. A person living with HIV who takes HIV medicine daily as prescribed and gets and stays virally suppressed can stay healthy and has effectively no risk of sexually transmitting HIV to HIV-negative partners.

  Deaths: In 2019, there were 15,815 deaths among adults and adolescents with diagnosed HIV in the United States and 6 dependent areas.

3.2 HIV Transmission

In the United States, HIV is spread mainly by sexual intercourse or sharing drug injection equipment such as needles with a HIV infected person. Only certain fluids—blood, semen, pre-seminal fluid, rectal fluids, vaginal fluids, and breast milk—from an HIV infected person can transmit HIV. These fluids must come in contact with a mucous membrane or damaged tissue or be directly injected into the bloodstream (from a needle or syringe) for transmission to possibly occur. Mucous membranes can be found inside the rectum, the vagina, the opening of the penis, and the mouth.

In the United States, HIV is spread mainly by:

Having sex with someone who has HIV:

- Anal sex is the highest-risk sexual behavior.
- Vaginal sex is the second highest-risk sexual behavior.
- Having multiple sex partners or having other sexually transmitted infections can increase the risk of infection through sex.
- Sharing needles, syringes, rinse water, or other equipment (works) used to prepare injection drugs with someone who has HIV.

Less commonly, HIV may be spread by

- Being born to an infected mother. HIV can be passed from mother to child during pregnancy, birth, or breastfeeding.
- Being stuck with an HIV-contaminated needle or other sharp object. This is a risk mainly for health care workers.
- Receiving blood transfusions, blood products, or organ/tissue transplants that are contaminated with HIV. This risk is extremely small because of rigorous testing of the US bloodsupply and donated organs and tissues.
- Eating food that has been pre-chewed by an HIV-infected person. The contamination occurs when infected blood from a caregiver's mouth mixes with food while chewing and is veryrare.
- Being bitten by a person with HIV. Each of the very small number of documented cases has involved severe trauma with extensive tissue damage and the presence of blood. There is norisk of transmission if the skin is not broken.

- Oral sex—using the mouth to stimulate the penis, vagina, or anus.
- Contact between broken skin, wounds, or mucous membranes and HIV-infected blood or blood-contaminated body fluids. These reports have also been extremely rare.
- Deep, open mouth kissing if the person with HIV has sores or bleeding gums and blood is exchanged. HIV is not spread through saliva. Transmission through kissing alone is extremelyrare.

3.3 HIV Infection Process

A person can transmit HIV to others during any of these stages:

Acute infection: Within 2 to 4 weeks after infection with HIV, you may feel sick with flu-like symptoms. This is called acute retroviral syndrome (ARS) or primary HIV infection, and it's the body's natural response to the HIV infection. (Not everyone develops ARS, however—and some people may have no symptoms.) During this period of infection, large amounts of HIV are being produced in your body. The virus uses important immune system cells called CD4 cells to make copies of itself and destroys these cells in the process. Because of this, the CD4 count can fall quickly.

Your ability to spread HIV is highest during this stage because the amount of virus in the blood is very high. Eventually, your immune response will begin to bring the amount of virus in your body back down to a stable level. At this point, your CD4 count will then begin to increase, but it may not return to pre-infection levels.

Clinical latency (inactivity or dormancy): This period is sometimes called asymptomatic HIV infection or chronic HIV infection. During this phase, HIV is still active, but reproduces at very low levels. You may not have any symptoms or get sick during this time. People who are on antiretroviral therapy (ART) may live with clinical latency for several decades. For people who are not on ART, this period can last up to a decade, but some may progress through this phase faster. It is important to remember that you are still able to transmit HIV to others during this phase even if you are treated with ART, although ART greatly reduces the risk. Toward the middle and end of this period, your viral load begins to rise and your CD4 cell count begins to drop. As this happens, you may begin to have symptoms of HIV infection as your immune system becomes too weak to protect you.

AIDS (acquired immunodeficiency syndrome): This is the stage of infection that occurs when your immune system is badly damaged, and you become vulnerable to infections and infection-related cancers called opportunistic illnesses. When the number of your CD4 cells falls below 200 cells per cubic millimeter of blood (200 cells/mm3), you are considered to have progressed to AIDS. (Normal CD4 counts are between 500 and 1,600 cells/mm3.) You can also be diagnosed with AIDS if you develop one or more opportunistic illnesses, regardless of your CD4 count. Without treatment, people who are diagnosed with AIDS typically survive about 3 years. Once someone has a dangerous opportunistic illness, life expectancy without treatment falls to about 1 year. People with AIDS need medical treatment to prevent death.

3.4 Risks to Healthcare Workers

Special precautions should be taken when working with HIV patients because they may have other pathogens that are contagious such as tuberculosis, staphylococcal infection, hepatitis, and herpes virus. It is imperative to practice universal precautions when working with patients who may be HIV seropositive.

Part 4. CDC's Universal Precaution Recommendations

4.1 Definition: Last updated by NIOSH September 2016

Universal precautions, also termed standard precaution, refers to certain infection-control steps that medical professionals take to reduce the risk of transmitting HIV and other infectious diseases. The scientific basis of universal precautions is that individuals should treat any blood or bodily fluid as though it contains hepatitis, HIV or another infectious agent. In other words, universal precautions assume that all bodily fluids are dangerous and tell medical professionals to treat them accordingly.

This not only protects caregivers, but also, by applying the same procedures to everyone, removes the stigma that might otherwise be associated with glove-and-mask use around highly infectious patients.

4.2 OSHA Mandated the Use of Universal Precautions

OSHA mandated the use of universal precautions as a form of infection control in the early 1990s, after it became clear that HIV spread through exposure to blood and certain other bodily fluids. One of the most interesting aspects of the mandate is that the 1987 CDC document on which OSHA standards are based explicitly acknowledges the fact that medical history and examination are not reliable methods of identifying bloodborne illnesses in all patients. This is still true, particularly during the early weeks of HIV infection, even though the HIV test has improved. It is also a problem for several other illnesses.

4.3 Summary of CDC's Universal Precaution Recommendations (CDC.gov)

The following recommendations are provided, and the clinicians should follow the recommendations wherever applied.

1 Wash hands before and after each medical procedure (may use a waterless hand cleaner).
2 Wear gloves whenever there is a possibility of coming in contact with blood or otherpotentially infectious materials (body fluids, synovial fluid, wound exudates, etc.).
3 Wear full-body gowns whenever there is a possibility of blood splashing onto thepractitioner.
4 Wear face masks and eye protection whenever there is a possibility of blood splashing intothe practitioner's face.
5 Dispose of all contaminated sharp objects in an appropriate puncture-proof container.
6 Dispose of all contaminated personal protective equipment in an appropriate containermarked biohazardous.
7 Spills of blood or blood-containing body fluids should be cleaned up using a solution of bleach (sodium hypochlorite) solution (1:100) for smooth surface and 1:10 for porous surface. The solution should be freshly made before use.

Part 5. Clean Needle Procedures

5.1 Skin Preparation Prior to Needling Insertion (Clinicaltrials.gov)

At present, based on the available evidence, the World Health Organization (WHO) and The Centre for Disease Control (CDC) do not recommend the use of alcohol swabs for preparation of the skin prior to vaccination, unless the skin is grossly contaminated or dirty. (1) Skin that currently has an active lesion should not be used for needle insertion. These areas often carry higher risk for infection.

Risk assessment of potentially contaminated skin should be conducted to maintain appropriate cleaning of the skin where required. If you believe that the patient's skin is soiled; it should be cleaned prior to needle insertion. There is no clear evidence that skin cleansing with soap and water, alcohol

swabs, or antibacterial substances like chlorhexidine is better or worse than the other options. If the insertion site is cleaned with an alcohol swab, it should be allowed to dry prior to needle insertion.

Conclusion: If the skin is clean, the needling area does not necessarily need to be swabbed with alcohol.

Special Note for Dry Needling: All the research on skin cleaning mentioned above were done with injection of medication like vaccination injection (wet needling). Dry needling is an effective treatment for tissue inflammation (pain) and swelling. Clinically, we insert dry needles into painful areas, which are usually inflamed or swollen. If the skin area is clean according to your assessment, dry needling is safe to be used in these areas. Skin areas with open wounds should never be needled.

5.2 Recommendations

The following information is provided for your reference. A summary of the recommendations is cited but the original research paper can be consulted. (1)

1. Insert needles into clean, intact skin.
2. For most patients, especially those with intact immune systems, skin preparation with antiseptics is unnecessary and may be disadvantageous by creating an imbalance betweennormal resident bacteria and pathogens.
3. While definitive studies of the effects of the practitioner touching the needle shaft remain to be done, needling characteristics, proper hand washing, and hand drying minimize the risk ofpatient infections and justify the continued practice of touching the needle shaft.
4. For patients with compromised immune systems, skin preparation with chlorhexidine-alcoholor providone-iodine scrubs is superior to 70% isopropyl alcohol.
5. Universal blood and body fluid precautions (universal precautions) should be followed. When treating patients with high risk of being infectious, the practitioner should protect himself or herself by using appropriate barriers, such as gloves or finger cots.

(1) Allen McDaniels, MD and Donna Pittman, MD: Is Skin Preparation Necessary Before Needling? A Review. Medical Acupuncture, Volume 23, Number 1,2011. Pp. 7-10.

5.3 Basic Procedures

1. Always wash hands between patients, and before and after needling.
2. Always prepare a clean field before performing dry needling. Here the clean field means bothclean skin area for needling and clean office environment such as table surface for storing needles, etc.
3. Use only sterile single-use needles as instructed in the lecture.
4. Always use gloves or finger cots to handle needles before and after insertion.
5. If blood drop appears after removing needles, sterile cotton swab should be used to clean theblood and the swab should be properly disposed of according to specific state and federal laws.
6. Always immediately isolate used needles in special sharp's disposal containers.

Definition of Anti-Microbial Procedure:

Antiseptic: Product used to reduce the density of microbial life on living tissue, particularly on the skin area.

Aseptic techniques: Techniques for preventing infection during invasive procedures such as surgical operation or dressing wounds. Dry needling procedure does not need this technique

Clean field: For dry needling, it means a clean skin area where the needling will be performed and a clean environment such as the table surface to store needles.

*Note: A clean field is not the same as a sterile field.

Disinfectants: The chemicals used in disinfection such as hypochlorite dilution (bleach), which should not be used on living tissue. (Not to be confused with antiseptics).

Disinfection: The use of disinfectants with proper procedures to destroy or reduce the number of pathogens on inanimate objects such as equipment and clinic surfaces. Some bacteria, spores, and viruses may resist the often-lethal effects of some disinfectants.

Sterilization: The use of specific procedures that destroy all microbial life, including viruses.

Part 6. Needle Stick

Needle sticks occur frequently in the healthcare setting; so risk of a needle stick is always a concern. In most cases, needle stick injuries occur because of unsafe needle handling practices and negligence on the part of the healthcare worker. Despite the frequency of needle sticks occurring in healthcare settings, the risk of transmission of microorganisms is very small (King 2022). The risk of transmission increases when the individual has been exposed to a higher quantity of blood and was stuck with a larger-bore needle. Allied health professionals that practice dry needling or acupuncture are also at risk of sustaining a needle stick. Fortunately, dry needling treatment utilizes a small gauge monofilament needle and the quantity of blood they are exposed to makes the risk of transmission even less likely, but not impossible. It is the responsibility of healthcare institutions and providers to set up policies and procedures for safe needle handling and post-needle stick management. Consider consultation with infectious disease specialists to assist in setting up the proper program for your situation. (*King KC, Strony R. Needlestick. [Updated 2022 Jul 1]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK493147/)*

Part 7. Indication and Contraindication of Dry Needling

Dry needling is a non-pharmaceutical procedure that restores local and systemic homeostasis through multiple physiological mechanism. Dry needling is a non-specific therapy that does not conflictwith any pharmaceutical or surgical procedures used in conventional medicine. Physiologically dry needling can be incorporated into many fields of conventional medicine as supplementary and supportive therapy. In physical medicine, dry needling is used to accelerate natural healing of soft tissuepain and neuromusculoskeletal dysfunction. However, from a clinical perspective, dry needling should never be used in the following cases:

1. Patients with bloodborne pathogens (BBP) as explained in lecture, regardless if the patient is active or latent, acute or chronic. As medical history may not reveal the BBP condition in some patients, universal precautions should be applied when treating these patients.
2. Other blood diseases like hemophilia.

3. Patient is allergic to the metals in the needle.
4. Cancer tissue area. Dry needling is effective in reducing cancer pain in patients with chemo or radiation therapy as NIH panel (1997) shows. But needling should be performed away from site of the cancer tissue.
5. Patients that have a needle phobia.

Part 8. Disposing of Biohazardous Waste

Any solid waste or liquid waste that may impose a threat of infection to humans, including human tissues, human blood and fluid, discarded dry needles, table paper or cotton balls that contain human blood are classified as medical, or biohazard waste. Note that OSHA has determined that a cotton ball containing enough blood that it can be wrung out must be classified as biohazardous waste, less than that amount on a cotton ball means that it is considered trash. OSHA has enacted specific rules concerning the handling and disposal of biohazardous waste to prevent exposure of employees, patients, and the public to disease-causing agents. These rules require that discarded biohazardous sharps must be packaged in impermeable, red, polyethylene or polypropylene bags and sealed. The discarded sharps must be separated from all other wastes and be collected by professional personnel. OSHA regulations contain minimum standards established by the federal government. However, state and local laws should be strictly followed for disposal of biohazardous waste.

Finally, all safety procedures and precautions instructed in the lecture should be strictly observed during the lab practice.

Join us on Social Media

Info@Integrativedryneedling.com
Dave@Integrativedryneedling.com
Sonya@Integrativedryneedling.com
TJ@Integrativedryneedling.com
Nina@integrativedryneedling.com
Frank@Integrativedryneedling.com
Nick@Integrativedryneedling.com
Stephanie@Integrativedryneedling.com
Mark@Integrativedryneedling.com

Frank Gargano-@DrFrankGargano
David Griswold-@DGrizPT
Nick Sanders-@NickSandersPT
Sonya Worth-@SonyaWorth

linkedin.com/Integrative dry needling

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