The WG Transeva's position in the modern world and its affect on the equine.

BSET Academy

Stacey Arnold

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Introduction

The Winks Greene Transeva Machine is one of the many forms of electrotherapy in the world today. Electrotherapy is defined as the application of electrical stimulation transmitted through the body for therapeutic purposes (Gallo 2004). The Transeva was first manufactured in the early 1900's before the war (Strong 1967). After being destroyed in the destruction of the war, the machine was rebuilt again and followed by countless success stories (Strong 1967). Even though the WG Transeva has been used to treat countless different cases over the years, not much is known about the science behind the WG Transeva and what makes it unique. As years passed and technology evolved many machines have been developed with the aim of treating or aiding in pain. While they have proved helpful to a certain degree, the WG Transeva is still proving to have long-last, effective results. To fully understand the WG Transeva and other electrotherapy machines, the physiology of the muscle and how it responds to stimuli will also be discussed.

CHAPTER 2: THE PHYSIOLOGY OF THE MUSCLE

There are three types of muscles in the equine body namely: cardiac muscles, skeletal muscles and smooth muscles (Starkebaum 2015). While the cardiac muscles are around the walls of the heart and smooth muscles around the rest of the organs, both are under involuntary control (Starkebaum 2015). Regardless of which type of muscle it is though, all muscle tissues have four basic properties:

1. Excitability – ability to respond to stimuli
2. Extensibility – ability to continue contracting over a range of resting lengths
3. Elasticity – ability to successfully rebound to the original length post contracting
4. Contractability – the ability to shorten actively and exert a pull / tension which can be harnessed by connective tissue (Tallitsch 2009.)

For the purpose of this research I will focus on skeletal muscles' structure, their fibres types, fibre distribution and how they contract.

2.1. Muscle structure

A muscle is made up of various layers starting with the actin and myosin fibres arranged in parallel lines and surrounded by sacroplasmic reticulum, known as sacromeres (Valberg, MacLeay n.d.; Krans 2010; Tallitsch 2009). The thousands of parallel sacromeres come together to form one myofibril (Valberg, MacLeay n.d.). Transverse-tubules run the length of myofibrils deep into the tissue (Tallitsch 2009). Various myofibrils then form a single muscle fibre (Valberg, MacLeay n.d.). These muscle fibres attach to the bone via tendinous insertions and are surrounded by a thin sheet containing the blood vessels and nerves which

feed the fibres information and nutrients (Valberg, MacLeay n.d.). Each nerve branch communicates with one muscle fibre at the motor end and ultimately stimulates all the muscle fibres under its control (Valberg, MacLeay n.d.).

2.2. Types of muscle fibres

Muscle fibres are categorised into two main types according to their function; type 2 being locomotive or type 1 being postural (Hyytiäinen, Mykkänen, Hielm-Björkman, Stubbs, and McGowan 2014). Type 1 fibres are also know as slow twitch fibres due to them being able to hold a titanic twitch for long periods of time without fatigue (Valberg, MacLeay n.d.). The resistance to fatigue is a result of high mitochondria density which results in a high aerobic or oxidative capacity, highest lipid stores, highest density of capillaries and the lowest glycogen stores (Valberg, MacLeay n.d.).

Type 2 fibres are further divided into type 2A fibres and type 2B fibres (Valberg, MacLeay n.d.). Contrary to type 1 fibres, type 2B fibres possess the following properties; fastest contractile speed, largest cross-sectional area, highest glycogen stores and lowest oxidative capacity making the fibres ideal for short burst of power (Valberg, MacLeay n.d.). Furthermore type 2A fibres have intermediate properties and contractile speed between type 1 and type 2B fibres (Valberg, MacLeay n.d.). Research has shown that the deeper regions of muscles are suited for a postural function and lower level, but longer duration activity while superficial muscles are more involved with short bursts of power (Hyytiäinen et al 2014).

2.3. Fibre distribution

Fibre distribution has been widely studied in an attempt to find a pattern regarding the fibre distribution and size. While certain concepts prove correct, it is important to remember that each horse is an individual and that training also has an effect on the muscle fibres. Generally speaking Arabians, which are bred for long distance, have more type 1 fibres in their muscles (Hyytiäinen et al 2014). Quarter horses or thoroughbreds on the other hand prove to have more type 2 fibres (Hyytiäinen et al 2014). Samples were taken from both the left and right sides of the horse and no significant difference were found between the two sides (López-Rivero, Serrano, Diz, Galisteo 1992). Superficial and hind limb muscles consisted of more type 2 fibres while deep muscles had relatively equal type 1 and type 2 fibres (Hyytiäinen et al 2014). Muscles such as the psoas group, bicepts femoris, glutes and longissimus showed a composition of mainly type 2 fibres (Hyytiäinen et al 2014).

Studies have also proved that type 1 muscle fibres were larger in the deep regions and varying sizes of type 2B fibres in the superficial regions (López-Rivero et al 1992). Samples taken from the gluteus medias had larger type 2 fibres (López-Rivero et al 1992).

2.4. How muscles contract to create movement

The nervous system is responsible for the initial stage of muscle contraction as all voluntary muscle contractions are initiated at the cerebrum which is part of the central nervous system (T.Gore, P.Gore, and Giffin 2008). The spinal cord, which is also part of the central nervous system, has an intermediary function between the brain and other parts of the body (Hadden 2005).

All conscious actions require sensory input as well as a host of other cognitive processes that allow the most appropriate motor output for the given circumstances (Knierim 1997). Ultimately the final output is commands sent to a certain set of muscles (Knierim 1997). The cell bodies of the motor neurons are located in the anterior horn of the gray matter of the spinal cord, and are cells which act directly upon the activated portion of the body (Floeter 2010; Hadden 2005). Afferent neurons carry impulses to the central nervous system and efferent neurons carry them away from the central nervous system (Hadden 2005). The neurons which innervate skeletal muscle and cause muscle contractions that generate movement are large cells known as alpha motor neurons (Knierim 1997). Gamma / fusimotor neurons are smaller and innervate one or more than three types of fibres within the muscle spindle (Floeter 2010). Lastly there are neurons known as skeleton-fusimotor neurons / beta motor neurons which innervate both intra- and extrafusal fibres (Floeter 2010). Motor neurons are responsible for releasing the neurotransmitter acetylcholine at a synapse known as the neuromuscular junction (Knierim 1997). As the motor axons near the target they split into branches which lose their myelin sheaths in the process (Floeter 2010). When the neurotransmitter binds to the receptors on the muscle fibre an action potential is initiated triggering the contraction of the muscle (Knierim 1997). Motor neurons also control the amount of force that is exerted by muscle fibres (Knierim 1997). Small and large motor neurons innervate different muscle fibres as follows: small motor neurons innervate slow-twitch fibres, intermediate-sized motor neurons innervate fast-twitch fatigue-resistant fibres and large motor neurons innervate fast-twitch fatigable muscle fibres (Knierim 1997). Muscle fibres are innervated through nerve impulses. Nerve impulses travel through the synapse, which is a chemical combination of sodium, chloride, calcium and potassium (Hadden 2005). In basic terms the impulse travels from the sensory nerve cell to the sensory nerve itself, then to the spinal cord and motor nerve, ending at the underlying muscle (Hall 1998). The axon from one cell binds to the dendrite of another cell (Hadden 2005).

The process by which muscles contract is known as the sliding filament theory. When a muscle is relaxed the actin and myosin filaments partially overlap, and when the muscle contracts they overlap entirely (Krans 2010). During muscle contraction it was observed that the thick myosin filaments remain central and constant in length while the thin actin filaments change in length, hence the sacromeres shortening as

well (Krans 2010). According to Floeter 2010 the sacromeres is the smallest unit of contraction and is referred to in figure 1.

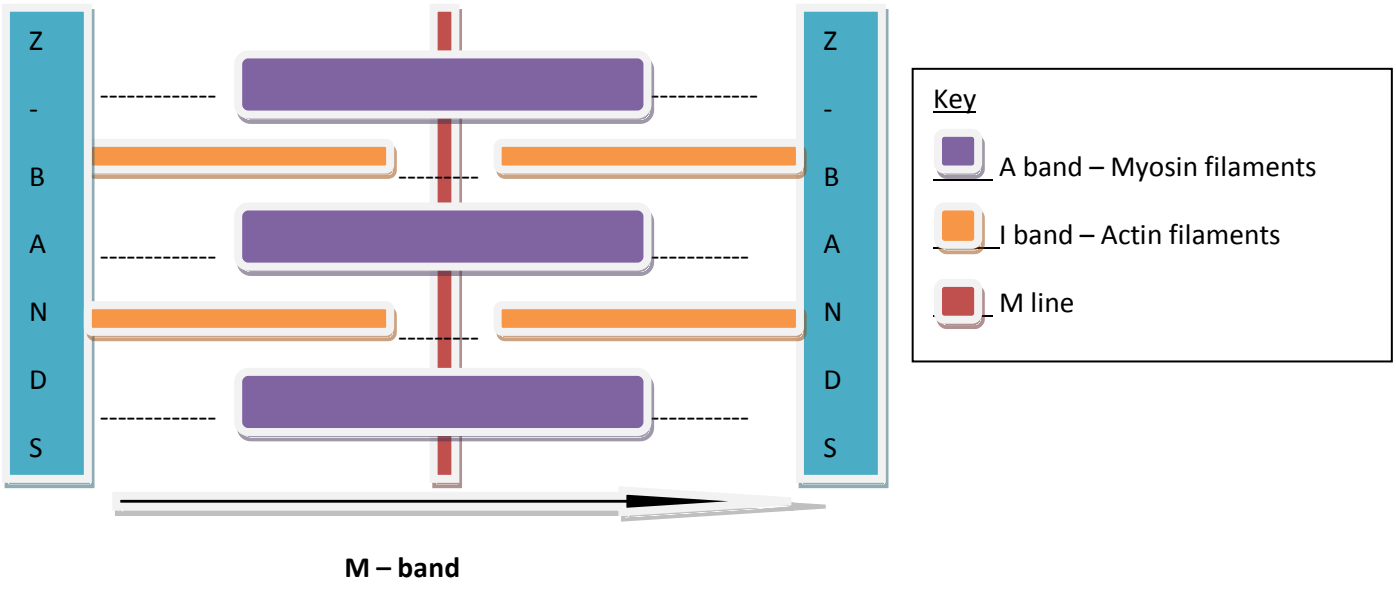


Figure 1 structure of a sacromeres in a muscle fiber

As previously mentioned this process is initiated by a nerve impulse. The nerve impulse travels to the neuromuscular junction stimulating the nerve terminal to release a neurotransmitter acetylcholine (Valberg, MacLeay n.d.). This neurotransmitter binds to the motor-end plate of the muscle fibre initiating depolarization of the muscle membrane (Valberg, MacLeay n.d.). Calcium is then released into the myoplasm via the T-tubules which penetrate deep into the muscle from the sacroplasmic reticulum where it is stored (Valberg, MacLeay n.d.; Floeter 2010). ATP is hydrolysed to form ADP and phosphate which provides the necessary energy for the formation of cross-bridges (Krans 2010). When the calcium is released it binds to the protein troponin, uncovering the binding site on the actin filaments (Floeter 2010). The globular head of the myosin filament then binds itself to the available binding site (Krans 2010). For the myosin head to detach, it is depolarized causing the myosin filament to bind to the next available binding site (Floeter 2010). This movement from one binding site to the next pulls both ends of the myofibrils to the middle causes tension, resulting in the sacromeres shortening / muscle contracting (Valberg, MacLeay n.d.; Floeter 2010).

For the muscle to relax, calcium must be pumped back through the T-tubules into the sacroplasmic reticulum where it is stored until the next impulse (Valberg, MacLeay n.d.).

Looking at how a muscle contracts in more detail emphasises the importance of available calcium and sufficient ATP in the body. If the supply of ATP is depleted, the myosin head remain bound to the actin binding sites resulting in rigor mortis (Krans 2010).

CHAPTER 3: THE INJURED / STRESSED MUSCLE

Muscle fatigue is the leading cause for muscle injuries such as sprains and tears due to the inability of the muscle cells to continue to contract efficiently (King and Mansmann 2005).Muscle fatigue can also be defined as a decrease in the capacity to perform a physical activity at a given intensity (Marlin 2007; Enoka and Duchateau 2008). Hence the higher the intensity of the activity, the quicker fatigue will set in (Marlin 2007). Various factors can influence the onset of fatigue including fitness, age, body condition, intensity, duration, and even environmental conditions such as difficult terrain and heat to name a few (Marlin 2007; King and Mansmann 2005). However, fatigue occurs because one or more of the physiological processes that enables the generation of force becomes impaired (Enoka and Duchateau 2008).

Example 1

Reduction in activation of the muscle by the nervous system (Enoka and Duchateau 2008). Various studies show that some of the fatigue experienced during repetitive and long-duration contractions can be caused by a lack of muscle activation through the nervous system (Enoka and Duchateau 2008). Painful stimuli from the muscles or joints may also cause a negative effect on the horse's willingness to move forward (Marlin 2007).

Example 2

ATP supports all cellular based activity, including muscle contraction (Marlin 2007). However, when the low amount of ATP stored in the muscle is depleted, it is seen as a possible cause of fatigue (Marlin 2007).

The anaerobic breakdown of glycogen creates the by-product lactic acid, and the anaerobic metabolism involves hydrolysis of creatine phosphate to creatine and inorganic phosphate (Westerblad, Allen, Lännergren 2006). Up until recently it was believed that lactic acid was the cause for muscle fatigue and delayed onset muscle soreness, however, recent studies have challenged this theory (Marlin 2007; Hiskey 2010; Westerblad et al 2006). Lactic acid is known to stay in the system for only a few hours post exercise, making it clear that it cannot be the cause for delayed onset muscle soreness as that only occurs 24-72hours post exercise (Hiskey 2010). Instead, the cause is thought to be damage to the myofilaments, specifically at the Z-discs and muscle's connective tissue, caused by eccentric contractions (Hiskey 2010; Page 1995). Structural damage was found both immediately and 3 days after eccentric exercise (Page 1995). Once these structure have been damaged inflammation occurs and the pain receptors become more sensitive (Hiskey 2010). Symptoms include pain, decreased motion and decreased force production (Page 1995). Initial studies showed a correlation between lactic acid build-up, reduction of force

production and shortening velocity (Westerblad et al 2006). However, in recent studies it was noted that force sometimes recovers more rapidly than the pH diminished by the lactic acid after the end of fatiguing contractions, proving that there is no constant correlation between the two (Westerblad et al 2006). It was rather proven that temperature may play a bigger role in muscle fatigue (Westerblad et al 2006). It has also been suggested that lactic acid build-up decreases performance by inhibiting calcium release from the sacroplasmic reticulum (SR) ultimately decreasing the degree of activation of the contractile machinery (Westerblad et al 2006). This was also discounted when it was discovered that lactic acid has no direct effect on depolarization-induced SR calcium released (Westerblad et al 2006). Through all of these studies it is made clear that if lactic acid has any affect on the fatigued muscle it would not be a direct effect.

Now that lactic acid has been discounted, one can look into the concept of inorganic phosphate being the cause for fatigue (Westerblad et al 2006). It is proposed that inorganic phosphate is released in the transition from low-force, weakly attached states to high-force, strongly attached states during cross-bridge action (Westerblad et al 2006). Based off of this statement it is implied that fewer cross-bridges would be in high-force states and as inorganic phosphate increases the force production would decrease during fatigue development (Westerblad et al 2006). Various studies in mammalian muscle tissue have shown an increased inorganic phosphate level at rest and no significant accumulation at fatigue, proving its direct effect on force depression (Westerblad et al 2006). Increased myoplasmic inorganic phosphate may have a direct action on the cross bridge function, hence decreasing force production during fatigue (Westerblad et al 2006). Inorganic phosphate is also likely to cause reduced myofibrillar calcium sensitivity which reduces force (Westerblad et al 2006).

When a muscle reaches the point of fatigue it is more susceptible to muscle spasms or fibrillations, and injury such as muscle sprains and tears.

Muscle spasm can be defined as involuntary, excessive or prolonged muscle contractions involving one or more muscles (Vorvick 2014; King and Mansmann 2005). Spasms can be caused by various factors such as overuse/injury, dehydration, incorrect mineral levels or even cold temperature (Vorvick 2014; King and Mansmann 2005). When a muscle goes into spasm the connective tissue within and around it registers the stretching of the fibers hence registering pain (King and Mansmann 2005). A spasm shortens the muscle ultimately preventing it from releasing (King and Mansmann 2005).

Unfortunately, information on muscle fibrillations is limited and few studies were carried out to back all the information. Muscle fibrillation is defined as independent, local muscular contractions in response to spontaneous activation of single muscle fibers (Pond, Marcante, Zanato, Martino, Stramare, Vindigni, Zampieri, Hofer, Kern, Masiero, and Piccione 2014). These spontaneous contractions are a defining characteristic of motor nerve disruption to the muscle fibers as well as spinal cord injury and peripheral denervation (Pond, Marcante et

al 2014). Numerous reports have demonstrated that it is mainly the loss of electrophysiological stimulation which initiates the fibrillation (Pond, Marcante et al 2014). It was concluded that these irregular contractions arose from localized changes in sodium conductance (Pond, Marcante et al 2014). One theory behind fibrillation is the shift to more negative potentials by 10 mV is able to cause this. Other studies revealed intracellular calcium levels increase post denervation while the rate of calcium uptake into the sacroplasmic reticulum decreases, suggesting that this increased calcium uptake may result in increased release of calcium from the sacroplasmic reticulum (Pond, Marcante et al 2014). Another factor that may play a role is neurotrophic factors; which refers to the chemicals that are released from neurons and have a regulatory effect on muscle physiology (Pond, Marcante et al 2014). Numerous studies have shown that an increased sensitivity to acetylcholine shortly follows denervation and travels to the muscle fibers themselves (Pont, Marcante et al 2014). This increased sensitivity is thought to have an effect on fibrillation activity (Pond, Marcante et al 2014). It is also believed that fibrillation causes the increase in lactate oxidation post denervation (Pond, Marcante et al 2014). Finally, fibrillation has proved helpful in preventing a more serious degree of muscle atrophy and is sometimes used to assess the degree of neural injury and reinnervation that has occurred (Pond, Marcante et al 2014).

More severe exercise induced injuries include muscle sprains and tears. The basic characteristic of sprains and tears are shown in table 1 found below.

Table 1 cause and location of muscle sprains and tears (King, Mansmann 2005)

	Muscle Sprains	**Muscle Tears**
Cause	Overexertion during work	Sudden movement
Location of damage	Belly of the muscle	Tendinous portion at the end of the muscle

Acute / excessive muscle sprains can result from direct trauma causing a contusion at the site of impact or indirect trauma causing a disruption in the myofibers (Page 1995). The speed, strength and type of contraction also has an effect on how severe the sprain is (Berro 2002). Predisposing factors include two-joint muscles, muscles contracting eccentrically and muscles with a higher percentage of type 2 fibers (Page 1995).

Table 2 below briefly explains the different levels of muscle sprains.

Table 2 degrees of muscle sprains (Page 1995)

First degree sprain	**Second degree sprain**	**Third degree sprain / tear**
Minute separation of muscle fibers	Partial tearing of some fibers	Complete rupture or tendinous avulsion

When there is tearing of fascia or tendinous tissue that attaches the muscle onto the bone it is classified as a muscle tear (King, Mansmann 2005; Page 1995).

Stating it basically, the progress of a muscle problem starts with trauma / strain which leads to tightening and creation of spasms or muscle tears, ending in muscle malfunction (Berro 2002). It is during the cooling down period after a strenuous workout that the muscle is most susceptible to spasm or sprain as there is insufficient oxygen reaching the fibers (Berro 2002).

Regardless of the type of injury the repair process for any muscle injury consists of the following overlapping phases:

1. destruction/degeneration
2. regeneration
3. fibrosis (T.Järvinen, T.Järvinen, Kääriäinen, kalimo, M.Järvinen 2005; Prisk and Huard 2003).

3.1 Destruction/degeneration phase

The destruction/degeneration phase occurs from the instant the muscle is injured (T.Järvinen et al 2005; Prisk and Huard 2003). The sacroplasm of the ruptured muscles stumps are torn causing the myofibers to retract forming a gap (T.Järvinen et al 2005; Prisk and Huard 2003). Due to the fact that skeletal muscles are richly vascularised, any capillary injury leads to a hematoma being formed in this gap (Prisk and Huard 2003).

Along with this damage the myofibers also become denervated through destruction of the intramuscular nerve branches (Prisk and Huard 2003).

Due to the damage of the blood vessels; activated macrophages, borne inflammatory cells and growth factors gain direct access to the injury site (T.Järvinen et al 2005; Prisk and Huard 2003). Macrophages are believed to play a role in various processes including the phagocytosis and removal of cellular debris during the destruction phase (Prisk and Huard 2003). Macrophage phagocytosis is a specific necrotic material process; the necrotized parts of the injured myofibers which survived the injury serve as a scaffold inside to begin the formation of new myofibers (T.Järvinen et al 2005).

3.2 Regeneration phase

Regeneration consists of two phases which occur almost simultaneously, namely the repair and remodelling phases (T.Järvinen et al 2005). During these simultaneous phases the disrupted myofibers regenerated and the formation of scar tissue occurs (T.Järvinen et al 2005). In response to the injury the damaged cells proliferate, differentiate into myoblasts and then join to each other to form multinucleated myotubes (T.Järvinen et al 2005;

Prisk and Huard 2003). These newly formed myotubes fuse with the remaining part of the injured myofibers (T.Järvinen et al 2005). Eventually these regenerating myofibers acquire their mature form (T.Järvinen et al 2005; Prisk and Huard 2003). The myofibers of the survived muscle stumps form multiple branches on both sides of the connective tissue scar while trying to pierce through the scar separating them (T.Järvinen et al 2005).

Early granulation tissue consisting of blood derived fibrin and fibronectin which acts as a scaffold and anchorage site for the invading fibroblasts (T.Järvinen et al 2005). This newly formed tissue is able to provide the wound tissue with the initial strength to withstand applied contraction forces (T.Järvinen et al 2005). The relative proteins are then synthesized to restore the integrity of the connective tissue framework (T.Järvinen et al 2005). With time the initially large granulation tissue condenses into a small connective tissue mass composed mainly of type 1 collagen (T.Järvinen et al 2005). In rare cases where excessive proliferation of fibroblasts takes place, the formation of dense scar tissue within the injured muscle occurs creating a mechanical barrier that delays or restricts the regeneration of myofibers (T.Järvinen et al 2005).

The restoration of vascular supply to the injured muscle is the first sign of regeneration and is crucial for recovery of the injured muscle (T.Järvinen et al 2005). New capillaries sprout from the surviving trunks of the blood vessels towards the centre of the injured area. ultimately providing the area with the oxygen supply required for aerobic energy metabolism (T.Järvinen et al 2005). This aerobic metabolism constitutes the principal energy pathway for the multinucleated myofibers in the final stages of regeneration (T.Järvinen et al 2005).

It is proven that myofibers regeneration continues regardless of innervations, but atrophy occurs if reinnervation is not accomplished (T.Järvinen et al 2005). In case of neurogenic denervation which occurs when the axon is ruptured, reinnervation requires the re-growth of the new axon distal to the rupture (T.Järvinen et al 2005). However, nerve-muscle contact is re-established fairly rapidly due to the fact that axons are usually ruptured within or next to the muscle (T.Järvinen et al 2005).

Various growth factors are released during the inflammatory phase, each with their own roles (Prisk and Huard 2003). Is has been proven that a number of these growth factors have significant roles to play during the regeneration and fibrosis stages, such as influencing the proliferation and differentiation of myoblasts and muscle stem cells (Prisk and Huard 2003).

3.3 Fibrosis

The fibrotic tissue consists of type 3 and type 1 collagen which replaces the hematoma in the necrotic muscle gap as early as the third day post injury (Prisk and Huard 2003). In the early stages of post injury this tissue provides support for the ruptured myofibers, but in the later stages 7-14 days post injury, it becomes increasingly dense restricting the regenerative growth of myofibers (Prisk and Huard 2003). The dense fibrotic tissue which develops

in the later stages of severe injury is known to prevent the myofibers stumps from rejoining as well as new axons from reaching muscle fibers to create neuromuscular junctions, hence causing possible atrophy following denervation (Prisk and Huard 2003).

CHAPTER 4: ALTERNATIVE THERAPIES

4.1 ALTERNATIVE THERAPIES USED

4.1.1. Hwave Therapy

Information on this therapy is limited and their technique guarded, leaving only basic information readily available to the public with very little scientific research and / or testimonials. H-wave is based on the well established fact that fluid shifts and pressures are essential in the process of tissue healing and homeostasis balance in an injured area (Hwave 2013). Through not only muscle stimulation, but neurological stimulation as well as the circulation and the healing process is enhanced (New Market Equine Physiotherapist). This technology makes use of a bipolar exponential decaying waveform allowing for deep stimulation without causing fatigue to the muscles (New Market Equine Physiotherapist). Inflammation has been successfully treated as during treatment circulation is improved and fluid shifts enhanced (Hwave 2013). According to Hwave (2013) not only have there been good results regarding inflammation, but range of motion can be increased significantly with repetitive use. There are two settings this machine can be used on; 2Hz or 60Hz (New Market Equine Physiotherapist). The 2Hz therapeutic mode causes visible muscle contractions in turn increasing circulation and activating the lymphatic system (New Market Equine Physiotherapist). The 60Hz option, on the other hand, shuts down pain signals and encourages the production and release of endorphins which are the body's natural painkillers (New Market Equine Physiotherapist).

4.1.2. Therapeutic Ultrasound

This technique has the ability to heat deep tissue without causing a significant rise in skin temperature which in turn promotes healing (Brooks 2010). How heat facilitates healing is shown in figure 1 below.

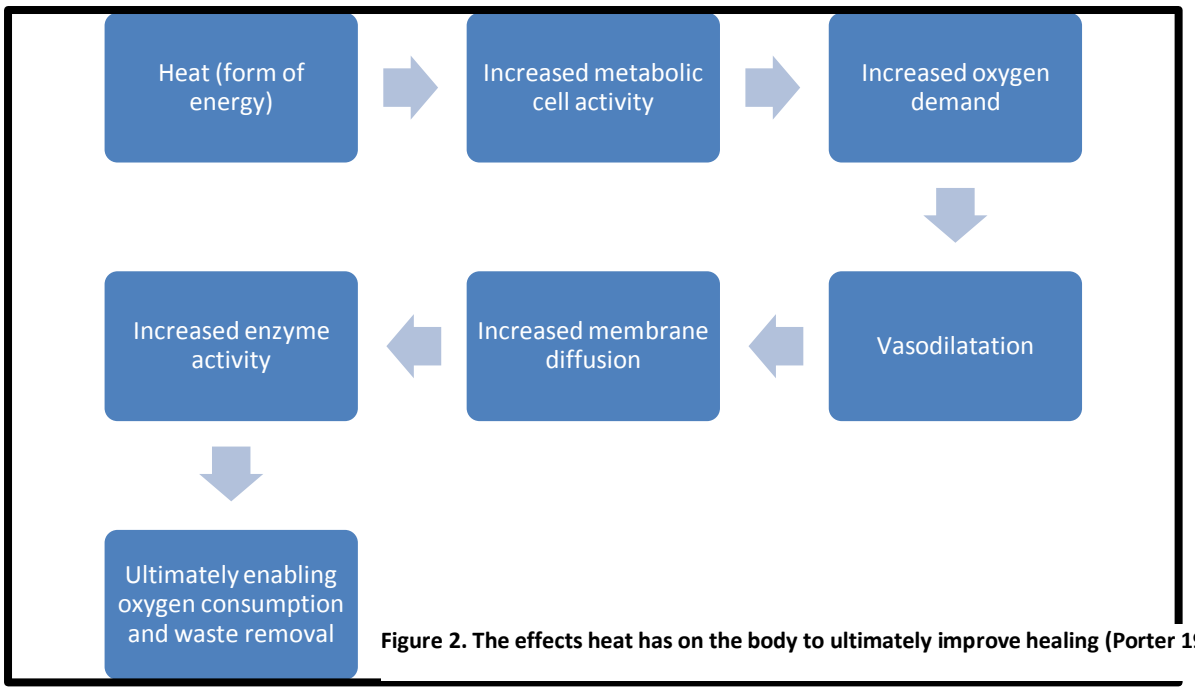


Figure 2. The effects heat has on the body to ultimately improve healing (Porter 1998).

During treatment electrical energy is converted into high frequency sound waves as large as 20000Hz through the transducer in the head of the device (Brooks 2010; Porter 1998). Any frequency higher than 18000Hz is inaudible to the human ear (Porter 1998). One million hertz becomes one megahertz (Porter 1998). Therapeutic ultrasound units have a small range of 0.8MHZ to 3MHZ with 1MHZ being ideal for sound absorption in the body (New Market Equine Physiotherapist).

The wave patterns transmitted are effective as they are moving in a straight line from the source with little divergence making it more powerful (Porter 1998). These waves can cause two effects; thermal and non-thermal (Brooks 2010). When making a thermal effect the tissue temperatures rise making it ideal for pre-stretching of tight muscles whereas non-thermal effects decrease inflammation and are ideal for promotion of tissue healing (Brooks 2010).

It has been very effective in the following cases:

- Joint mobility
- Tendon extension
- Scar tissue
- Bony growth
- Pain relief
- Muscle spasm relaxation
- Reduction of oedema

- Wound healing (Brooks 2010)

4.1.3. Electro Acupuncture

Electro acupuncture is, as the name suggests, a subsection of acupuncture hence being the only therapy that falls under Traditional Chinese Medicine (Boldt 2007). The treatment of electro acupuncture involves inserting sterile acupuncture needles into specific point in the body (Acupuncture Today). A device is then connected via clips between needles which sends continuous electric pulses into the targeted points (Acupuncture Today). Stimulation can be achieved and adapted to the injury by varying the frequency, intensity and even pulse (Boldt 2007). The effects of acupuncture / electro acupuncture are a series of interactions between the nervous system, endocrine system and the immune system (Boldt 2007). When inserting the needles micro trauma is caused resulting in a local inflammatory response, increasing circulation and ultimately resulting in muscle relaxation (Boldt 2007).

Depending on the injury or reason for treatment, a high or low frequency stimulation is used (Cefar). High frequency stimulation (15-200Hz) has a fast, brief effect and is used with local needles for acute or sub-acute pain (Cefar). Alternatively, low frequency stimulation (<10Hz) has a slower, long lasting effect causing visible muscle contractions and enhancing the effects of acupuncture (Cefar).

Use of this form therapy has shown promising results when used to relieve arthritis, improve blood flow, reduce postsurgical pain and restore damaged nerves (Cefar).

4.1.4. Transcutaneous Electrical Nerve Stimulation (TENS)

TENS units are pocket sized, battery powered devices which send electrical signals to control pain by having an effect on the nervous system (Tens Pain Relief Therapy 2016). The equipment activates the descending inhibitory pathway from the brain to the spinal cord, ultimately supporting the gate-control theory which states that when the gate is closed or blocked, pain signals cannot reach the brain (Transcutaneous Electrical Nerve Stimulation). The TENS machine can be applied at a low <10Hz or high >50Hz frequency accordingly (DeSantana, Walsh, Vance, Rakel, Sluka 2008). The pulse width, frequency and intensity can be altered on the unit, but the two channels are typically set equally (Harding 2015). This device can be used on two settings; high and low pulse rates (Harding 2015). A high pulse rate has a current of 90-130Hz and is based on the gate control theory of pain (Harding 2015). The theory states that the mechanism between the central nervous system and the brain is open when signals are sent to the brain (Harding 2015). When the gate is closed no pain signals can reach the brain (Harding 2015). TENS complies to this theory by stimulating certain non pain carrying nerves hence closing the gate through having the brain receive faster pulses versus slower painful pulses (Harding 2015). The low pulse rate is between 2-5Hz and is responsible for stimulating endorphins which act as a morphine to block the pain signals (Harding 2015). Due to

these settings it can be used to treat chronic or acute pain such as arthritis or even muscle pain (Harding 2015). Pads are stuck onto the affected area and connected to the device via wires (Transcutaneous Electrical Nerve Stimulation). The electrical impulse is then sent from the device, through the wires, to the pads and under the skin to the sensory nerves initializing the gate-control theory (Transcutaneous Electrical Nerve Stimulation). This form of therapy has proven that regardless of frequency or intensity, the central mechanism is activated producing analgesia (DeSantana et al 2008). Much success have been seen when used in cases of arthritis and various other musculoskeletal injuries (DeSantana 2008).

4.1.5. Electrical Muscle Stimulation (EMS)

Electrical muscle stimulation / neuromuscular electrical stimulation is delivered to the muscle at a high intensity in a static condition to evoke visible muscle contractions (Maffiuletti, Minetto, Farina, and Bottinelli 2011). Pads are placed on or around the affected muscle (Gordin 2007-2016). When a pulse is delivered all the motor neurons in the treated area are activated simultaneously, causing an isometric, uncoordinated contraction (Francis 2006). When a muscle contracts naturally the red and intermediate fibres shorten their recruitment rates in response to white fibre recruitment *following* the contraction; EMS reverses this recruitment order resulting in the response to white fibre recruitment coming into play *before* the contraction (Francis 2006). The machine has the potential to serve strength training, rehabilitation and preventive tool, testing tool or even post exercise recovery tool (Maffiuletti et al 2011).The maximum stimulation pattern goes as follows; 10 reps of 10 seconds with a 50 second rest period between contractions (Francis 2006). The duration of the rest period is important as that is the minimum time the muscle needs to ensure a maximal contraction to be followed (Francis 2006). Ultimately, through controlling so many aspects of the contraction it is possible to improve the speed of motor unit activation as well as the muscle memory (Francis 2006).

This form of therapy has proven to be successful in the following ways:

- Relaxation of muscle spasms
- Prevention of atrophy
- Increased circulation
- Muscle re-education
- Increasing / maintaining range of motion (Gordin 2007-2016)

There are only two main limitations with this type of therapy; discomfort and the relatively incomplete muscle recruitment (Maffiuletti et al 2011).

CHAPTER 5: THE WINKS GREENE TRANSEVA MACHINE

The origin of the Winks Greene (WG) Transeva started with Sir Charles Strong in the early 1900's when he developed what was known as the Strong Box (Strong 1967). It was first built before the war, but had to be rebuilt again after the destruction of the war (Strong 1967). Strong had developed a faradic machine which could be strapped around ones waist and was connected to the vehicle for power (Strong 1967). The complete apparatus consisted of the main apparatus, the remote control and the special roller (Strong 1967). The remote control had two-pin plugs to connect the main apparatus to the source of energy supply, which in that case was a vehicle (Strong 1967). The operating dials were also located on the remote control which allowed Charles Strong to alter the pulse and intensity (Strong 1967). It was not until the year 1939 when it was suggested to him to include the equine in his practises (Strong 1967). This Transeva had shown miraculous results in treating sprains and strains in both the human and the equine whereby Strong was knighted by the Queen of England (Strong 1967). In 1951 a South African woman named Desiree M. Greene, more commonly known as Winks Greene, shadowed Strong in England for 18months before returning to South Africa with a Strong Box and the Transeva technique well studied. Thirty-two years later in 1984 a racehorse in Summerveld named Gondolier, referred to Winks Greene by veterinarian Dr. Brian Baker, had sustained an injury to his near hind psoas group roughly a year before the Durban July. Gondolier arrived at the Natal Equine Rehabilitation Centre in September 1984 (Sunday times, July 1985) and stayed there for treatment over a period of 6months. According to Winks Greene's stable records 1985, Gondolier returned to Clairewood after 6 months of treatment where he went into full training. After a number of treatments by the Strong Box Gondolier had gone sound and carried on to win the Durban July 1985. This was the first big success of many success stories to come. When Strong died at the age of 79 years, he had only made a handful of the Transeva machines and had left one to Winks with a dying wish that she would improve the design. During Strong's era a battery was put into the machine to make it more user friendly as well as removing the danger of cords connected to a vehicle for power. In 1999 under Winks Greene's direction a technician, Nicoras, created extra movements in the pulse train to make it more comfortable for the equine (Nicoras 1999). The screen which is on the modern day WG Transeva was added in just before Wink's death in June 2010. Today, Winks's last advocate, Beth Shaw, is based in the Karkloof and continuing her legacy as guardian of the Equine Transeva Technique (ETT). Beth Shaw teaches ETT at Beth Shaw Equine Therapist (BSET) Academy and is constantly working on developing and improving ETT. Beth and her team have helped create 5 more Vodacom Durban July winners since Beth's established practise.

The WG Transeva was designed with the following specifications listed in the table 1:

Table 3 WG Transeva specifications

Voltage	0-374V
Average power consumption	5 watts
Pulse duration	0-255 µs
Pulse rate	0.5-2 seconds
Pulse train	14 movements consisting of 7 twin peak actions. 5milliseconds in between movements hence 70milliseconds in total.
Hertz	200 cycles
Amps	0-59.93 milliamps

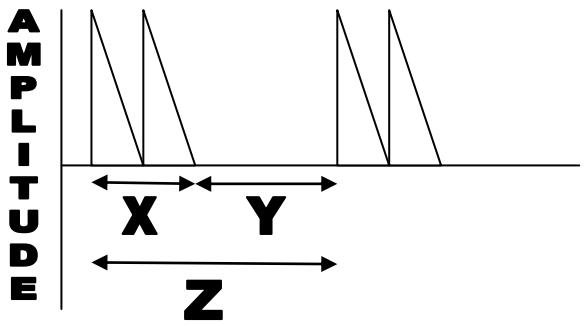
In Strong's early scripture it was stated that the Strong Box used a faradic current. However, the modern WG Transeva is known to have a pulsating current which makes use of twin peaks. The characteristics of high voltage pulsed current include twin spikes in a monophasic waveform, a higher voltage than 150V, total current of 1.5mA and a duration of 5-100µs (Sandoval, Ramirez, Camargo, Salvini 2010). This type of waveform is known as an isolated unit of bi- / uni-directional movement of charged particles which periodically cease for a finite amount of time, whereas faradic waveforms are continuous bi-directional flows of charged particles (Gallo 2004; Rennie 2014). Looking at these definitions prove the WG Transeva uses a twin peak pulse as it goes back to zero for a finite amount of time. Skin and the length of the cords have proven to cause some resistance in the flow of electricity while the subcutaneous tissue acts as a conductor (Gallo 2004; Rennie 2014). The law for flow of currents is current (I) = voltage ÷ resistance (Gallo 2004). Sensory and motor nerves are the target tissues proving that the WG Transeva stimulates the nerves (Gallo 2004; Rennie 2014). Many cases have proven that this high voltage current allows for relative comfort and avoids tissue damage while stimulating deep tissues through the dynamic ability to change the current when needed (Anap n.d).

When meeting with technician Les Fleming, to analyze the WG Transeva current and waveform in Hillcrest, Natal on 12 March 2016, the following facts were noted using a TPS 2024 four channel digital storage Oscilloscope:

- It is an AC current.
- Both a positive and negative charge is needed to create a comfortable pulse. Without a negative charge there would be no rhythm.

- The positive charge has a more powerful charge than the negative charge hence more energy is required in the negative charge of the pulse train. Refer to figure 2 for an image of the Transeva waveform.
- If working on a quicker pulse the positive charge has more power.
- If working on a slower pulse the negative charge has more power.
- When assessing the WG Transeva current without the machine connected to a patient there was no consistency between the starting pulse being negative or positive.
- When assessing the WG Transeva current with the machine connected to a patient there was a consistent positive movement in the pulse train to start.

Basic Twin peak pulse



KEY

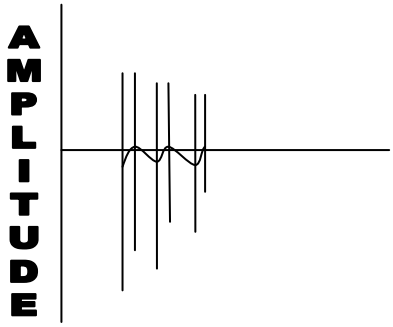
X – Pulse duration

Y – Pulse interval

Z – 1 cycle (X+Y)

Figure 3 Twin Peak action found in the Transeva current

Basic WG Transeva Waveform



Note:

- Twin peaks are present

- The negative current proves more powerful than the positive current

Figure 4 basic Transeva waveform

The two main advantages of the WG Transeva include the moveable hand piece and the immediate adjustability of the current in both intensity and rate. These unique factors allow for the practitioner to pin point the exact location of the lesion in the muscle which is causing the weakness or restriction. Through recognizing the

“tectonic state” of the muscle, the practitioner can move their hand accordingly to bring the muscle back online (Shaw 2016). The tectonic state or tectonic contraction refers to the muscle tone, the value of the muscle contraction, the irritability of the muscle and the elasticity of the muscle (Shaw 2016). Through the rhythmic contraction The WG Transeva is also capable of penetrating the deep seated skeletal muscles due to the pulse rate of two seconds. Based on the research done on the alternative therapies discussed in chapter 4, it was made evident that unlike the WG Transeva they do not target the source of the pain, but rather treat only the pain (Hwave 2013; Brooks 2010; Boldt 2007; Harding 2015; Francis 2006) Due to this factor, when these forms of electro-stimulation are used regularly, only then do they show results. Although these previously mentioned factors do make the WG Transeva more effective, it is the Equine Transeva Technique (ETT) technique which contributes largely to the significant results.

A session with the WG Transeva last between 45 and 60 minutes. The procedure for setting up a horse for treatment goes as follows:

i. Two teaspoons of course salt is dissolved into one litre of warm water

ii. wet the equine patient over the withers with a sponge

iii. The soaked towel is placed over the withers

iv. One negative plate is placed on either side of the wither over the towel

v. The two negative plates are joined via connecting wire

vi. A surcingle over the plates, around the trunk of the horse and with care, is fastened tighly

vii. The negative wire plugged into the WG Transeva is then attached to the negative plate the side which the therapist is working

viii. The positive hand piece is placed on the horse with the machine’s intensity on zero and turned off.

Once all these steps have been completed the treatment can begin. The electrical impulse emitted by the WG Transeva travels through the hand piece to create rhythmic muscular contractions. These rhythmic contractions are thought to have the ability to increase circulation, improve lymph flow, correct muscle function and break down scar tissue. The movable hand piece allows for more effective treatment as it allows for free movement to locate the lesion in the muscle and work dynamically to release the restriction.

CHAPTER 6: CASE STUDIES

6.1 Case Study 1

The WG Transeva has been involved with countless Vodacom Durban July horses over the years including names such as Power King (2015 winner), Pomodoro (2012 winner), Big City Life (2009), Eye of the Tiger (2006) and Dynasty (2003) just to name a few (Vodacom Durban July Roll of Honour). All these horses presented musculoskeletal pain which was corrected and worked through using the WG Transeva weeks before the big race. However, the WG Transeva has made its way into every discipline over the years. (Shaw 2003-2015)

6.2 Case Study 2

The following case study regards a horse Secret who came to the Natal Rehabilitation Centre, Karkloof Natal, after injuring her right hind superficial digital flexor tendon during a polo game. She was severely lame in the walk and reluctant to put full weight on her leg when she arrived. While on box rest she was given light treatments to improve circulation. In the initial treatments she was very accepting of the pulse. As we continued she became more and more sensitive as we started breaking down the scar tissue that had been building in the surrounding muscles. After treating her ishiums and gastrocnemius muscles she became more lame again. The lameness persisted regardless of treatments so we decided to stop the treatments for a few weeks. During those few weeks the tendon had settled and she was sound in the walk. When investigating along her ilium with the WG Transeva a large amount of nerve involvement was found. The following treatment showed nerve involvement at the right hind sacro-iliac joint and later on at her hip as well. On days where nerve involvement was found she would be more lame post treatment and then more sound the following day. She also started exhibiting interesting sweat patterns at night. When returning to the barn around 7pm she would have sweat patches along both her scapula spines, right hind hip and lumbar. This persisted for a week after she first exhibited nerve involvement under the WG Transeva. She is now sound in the walk and 2/5 lame in the trot. Unfortunately she will not be able to return to high goal polo, however, she is significantly more sound and comfortable with her right hind. (Arnold 2015-2016)

6.3 Case Study 3

Another interesting case was a race horse, Warrior. He came in after tripping and falling during a race resulting in him pulling up lame right front. He had also sustained a sublux to the near hind; however, we were not informed as to how and when it occurred. It was clear that this horse could run, but had developed incorrect muscling causing restriction. The worst of the restriction was defiantly in front although the weakness was behind. A large amount of stress was visible through his right front shoulder. He would go on and off lame right front and it took

time before he stopped tripping as much. During one treatment it was noted that Warrior had nerve involvement at his left hind sacro-iliac joint. After working through that his right front shoulder was treated. Following the stress lines over the point of shoulder and into his bicepts brachii Warrior exhibited more nerve involvement and sensitivity. Immediately after wetting Warriors right hind hip, the horse collapsed to the ground and started rolling frantically, rubbing his right hind hip. For a number of treatments following that incident Warrior showed sensitivity and nerve involvement over his hip. Soon after, Warrior's tripping improved and he started truly using his hind quarters. (Arnold 2014-2015)

6.4 Case Study 4

Smudge, the head shaker, had started with a slight head throw which eventually progressed to the point where he could no longer be ridden. Smudge had received plenty of treatments with the WG Transeva. During treatment of the neck at C_3 on both sides, Smudge would start head shaking and snorting. In the next treatment, more sensitivity and tension the neck was found on the left side along the accessory nerve and at C_7 T_1. However, it was at Smudge's off hind hip where most nerve involvement was found this time i.e head tossing and snorting. (Arnold 2016)

6.5 Case Study 5

The WG Transeva has also had brilliant results when used to treat humans. A client, Cobus, struggled with minor scoliosis. After a day of heavy lifting Cobus would be in significant pain and be left unable to bend over. Cobus's spine was slanted slightly to the right. Upon initial treatment the left side of Cobus's back was found to be much more tense. There were many areas of restriction throughout the back and so the treatment was focused on simply loosening up the back muscles. The following day there was a large amount of pain relief. During the second treatment the area of restriction were focused on and worked through. The following day Cobus's back felt stiff. Due to the fact that the pain was caused by a skeletal factor what proved most helpful was alternating between light and hard treatments once a week. (Arnold 2014-2015)

6.6 Case Study 6

Another client, Shaun, was in a car accident resulting in a fracture on the left ilium. Pins were inserted to keep the pelvis in place while healing. After weeks of crutches Shaun had started walking with his left food facing outward and had regained some movement in that hip. Under the Transeva the rectus femoris muscle gave a good test, slightly pulling up to his pelvis. When moving towards the pectineus muscle over the sartorius the contractability and elasticity decreased. Once that movement was improved over those muscles the pulse pulled down Shaun's leg affecting the foot. The main sensitivity was found gracilis and psoas system. While treating these muscles under rhythmic contractions the exact location of where the muscles where cut during surgery were clear. Immediately after Shaun's first treatment the pain in the groin area had disappeared and with conscious effort,

Shuan could walk straighter with his leg and foot. Three days after treatment the muscles had tightened up again. The gracilis and psoas system had much better contractability in the following treatment allowing for the use of a deeper pulse and higher intensity. The irritability of the pectineus and sartorius muscles had also improved substantially; allowing a immediate pull into the foot. Post treatment Shaun was stiff for that evening and the following day. Two days post treatment Shaun had felt a large improvement in pain level and range of motion. (Arnold 2015)

Conclusion

Although the muscle fiber composition will differ with each equine, their response to stimuli remains the same. All of the electrotherapy machines discussed have the ability to affect the nervous system, ultimately affecting the muscles. While they have shown success in regard to alleviating pain they typically do not have long term affects. This is due to the fact that other machines do not target the source of the pain, but rather treat symptomatically. Based on Strong's books and notes, the WG Transeva's waveform and current has since been widely accepted by the equine patient. While the WG Transeva has shown good result, the evidence is inconclusive and extensive studies need to be done to back up these findings.

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