

Student's Guide

The Pathomechanics of Degenerative Joint Disease: A One Health Comparative Case Study Approach

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I) Case Synopsis and Classification

Synopsis

The most common disease affecting man and animals is degenerative joint disease (DJD, osteoarthritis). Pathomechanical forces are induced by how an individual structurally interacts with its environment and directly cause joint injury. Therapeutics based upon identification of the sources of mechanical stress are critically needed as treatments focused only on controlling pain and tissue pathology mostly fail to prevent disease progression. Static postural analysis (SPA) is a well-established technique requiring no specialized equipment that can be used to identify the pathomechanical causes of joint pain and damage. It is a physics-based functional anatomical approach that can also explain why a particular joint is painful even when lesions are not visible. To perform SPA, free-body diagrams are used to analyze the normal and pathomechanical forces and torques acting on an individual in various static and freeze action postures. For this case study, comparative SPA analyses of the common forms of DJD in humans, horses and dogs will be performed. 2D models will be used to highlight vulnerabilities that are both shared and unique between humans and animals. This type of functional analysis illustrates *why* a patient has DJD, and can be used by practitioners to educate clients and formulate individualized therapies. The comparative approach emphasizes that the causal relationship between pathomechanical forces and DJD is based upon the same principles across species, allows a better understanding of the shared susceptibility to a very common disease and facilitates the transfer of therapeutic approaches between human and veterinary medicine.

Learning Goals and Objectives

After this comparative case study, students will have trained their minds to consider mechanical explanations for DJD and their eyes to interpret movements and postures that may be leading to these disease processes. Thus, the main focuses of this study are: (1) although sites of DJD vary across species, the response of tissues to mechanical stress and an underlying susceptibility to DJD is shared by humans and other animals because they share a vertebrate *bauplan* (body plan). (2) Acquired postural weaknesses from habitual postures, activities, or training regimens create asymmetrical forces within the body that predispose humans and other animals to DJD. (3) These weaknesses and asymmetries can be observed through observation of postures and movements.

This comparative analysis includes three cases that are designed to show how both underlying anatomic susceptibilities and habitual behaviors that lead to acquired postural weaknesses predispose animals and humans to mechanically induced DJD in similar ways even if the exact location of damage varies. Thus, the first case focuses on a small animal (the dog; Case 1), the second a large animal (the horse; Case 2), and the third a human (Case 3). Cross species comparisons are made in all 3 cases and the depth and complexity of the analysis varies to illustrate how static postural analysis can be utilized in both clinical and research settings.

- Case 1 is an introduction and demonstrates through static postural analysis how the shape, size, and varying proportions of the vertebrate body (so well-displayed in the vast differences seen in dog breeds) can predispose an animal to mechanical issues that eventually lead to disease.
- Case 2 demonstrates how static postural analysis can be used in a clinical setting to identify the aberrant biomechanics that are causing DJD in a specific patient. It also emphasizes enthesis organs and their importance in understanding the effects of mechanical forces on joints.
- Case 3 demonstrates in more detail how to test pathomechanical hypotheses using the physics-based method of free-body force analysis, as would be done in research.

General Objectives

1) Students will be able to use the pathomechanical paradigm to answer important questions about degenerative joint disease such as:

- What is the root cause of DJD and why does it get worse?
- Why doesn't the severity of lesions always correlate with pain or lameness?
- If DJD is caused by 'wear and tear', why does exercise help patients regardless of its severity?
- Why do the current treatments (painkillers, anti-inflammatories, cartilage repair) fail?

2) Students will be able to explain why the common sites of degenerative joint disease are both similar and different across species.

3) Students will be able to describe the role of enthesis organs in dispersing mechanical stress and why they are the common sites of damage in degenerative joint disease.

4) Students will be able to describe how habitual motion and acquired postural asymmetries can predispose an individual of any species to the development of degenerative joint disease.

5) Students will be able to describe how static postural (free body) functional analysis can be used to identify the aberrant biomechanics that induce lesions of degenerative joint disease in individual patients.

6) Students will be able to describe how static postural (free body) functional analysis can be used in a research setting to test hypotheses about the pathomechanics of degenerative joint disease.

General Case Descriptions

Case 1: Degenerative lumbosacral stenosis and spondylosis in dogs (with comparisons to humans and horses)

This case has the simplest analysis and is a good introduction to pathomechanics and the concept of static (free body) postural analysis. It should be used to emphasize that mechanically –induced lesions are very common, especially in the spine, and many (ie: spondylosis) are not always associated with pain or dysfunction. Rather they represent tissue responses to chronic mechanical stress that restored stability. This case also illustrates that body size and shape, as well as physical activities, have a big impact on mechanically–induced degenerative joint disease. The point that full body assessment of each patient is needed to identify the source of mechanical dysfunction in an individual should be emphasized.

Case 2: Navicular disease in a horse (with comparisons to humans)

In addition to providing a pathomechanical perspective on the pathogenesis of navicular disease, this case can be also used to teach students about enthesis organs, which are specialized adaptations to mechanical stress and are found in many joints. Although they are the sites of many of the lesions, their role in the pathogenesis of degenerative joint disease has been relatively neglected (see references for a good review). In addition, this case, along with case 3, emphasizes that pathomechanical loading and tissue damage can be induced by habitual posture, and provides a simple way of assessing that loading through tracing and analysis of static positions.

Case 3: Forward neck posture in humans (with comparison to horses)

The forward neck posture and the pain and dysfunction associated with it, is something that many people can personally relate to and thus can be used to stimulate discussion. This case has the most extensive analysis, which includes how to set up and solve the equations used. It is presented in a way that can be used to guide students through the calculations and includes a detailed appendix that can be used as a guide for self-directed individual or small group activities. This case can also be presented in the simpler format used in the other cases.

II) Background: Understanding Chronic Degenerative Joint Disease (DJD, Osteoarthritis)

Objectives:

- 1) Students will be able to describe the importance of mechanical forces in the pathogenesis of degenerative joint disease at the gross, tissue and molecular level.
- 2) Students will be able to describe the importance of pain as an indication of both ongoing and pending tissue damage.

Importance of Paradigms and Definitions

Paradigms in medicine represent consensus ways of thinking about the causes and progression of disease and thus determine how diseases are treated. Since they provide the best current explanation of disease they also define the direction of research. As research progresses and technology advances, a paradigm's weaknesses become apparent, especially in its failure to answer important questions and lead to the development of effective treatments. *Over time, as knowledge accumulates, these paradigms will fail* and new ones, that provide better explanations/treatments, will be developed. This process is called a paradigm shift and should be embraced, as it is critical to the advancement of both science and medicine. Under the predominant paradigm, DJD is defined as structural and functional failure of the joints through a process of inflammation and 'wear and tear' in which damage exceeds repair. Symptoms are pain, swelling, lameness and loss of function. The lesions include damage to cartilage and bone, inflammation and proliferation of bone around the joints. Treatment is based upon the idea that inflammation and tissue lesions are the primary sources of pain and dysfunction and is thus primarily focused on painkillers, anti-inflammatories and surgery. *The signs that a paradigm is failing are in its inability to answer important questions.*

Important unanswered questions in DJD:

- What is the root cause of DJD and why does it get worse?
- Why doesn't the severity of lesions always correlate with pain or lameness?
- If DJD is caused by 'wear and tear', why does exercise help patients regardless of its severity?
- *Why do the current treatments (painkillers, anti-inflammatories, cartilage repair) fail?*

Mechanics and DJD: Why Do the Lesions Form?

What we understand about disease is based upon how we study it. Traditionally, anatomy and pathology have been taught through dissection of cadavers, which leads to a stationary and limited understanding of how the musculoskeletal system works and why DJD lesions

form. Understanding how the musculoskeletal system functions and has adapted to the mechanical stress associated with movement provides a much better basis for both understanding the pathogenesis of DJD and the development of more effective treatments.

- *Muscles: Buffering Mechanical Stress*

Muscles do much more than create the forces needed to extend and flex joints, which is all that has been traditionally taught in anatomy courses. Muscles are also critical for the buffering of the mechanical forces on the joints. They do this by absorbing very large amounts of energy and are thus critical to preventing overloading and to the stabilization of joints. *This function is so important that the energy produced by normal walking would tear all the ligaments in the knee if it were not absorbed by muscular activity!* Think about the implication of this. *It means that joint stress and injuries can be predicted based on assessment of muscle function.* This allows prevention strategies to be developed before there is serious damage to the joint aka: prehabilitation, which is widely used in training human athletes. It also means that a muscle may be overdeveloped and/or hyper-contracted because of a weakness or loss of function in another muscle, in which case, simply trying to release a contracted muscle without considering whether it is compensating for dysfunction in another muscle group could further imbalance and stress the affected joints. (For discussion see Brandt KD et al. Ann Rheum Dis. 2006 Oct;65(10):1261-4).

- *Enthesis Organs: Transmitting Mechanical Stress*

All of the often-massive forces generated by muscles are transmitted to bone through a structurally continuous gradient from slender tendons to bone at specialized structures called entheses. Entheses are also present at other regions of high mechanical stress such as where ligaments and joint capsules attach to bone. These various attachment sites are often interconnected and highly adapted (enthesis organs), which both allows dissipation of mechanical forces and explains why the source of pain may not be at the exact sites where tendons are attaching to bone. These attachment sites are where mechanical forces are transferred between two tissues of very different physical properties (ie: soft, flexible tendons, ligaments and joint capsules to hard bone) and, although many are highly adapted (enthesis organs), they are vulnerable to failure, a situation well known to mechanical engineers. As these entheses are sites of mechanical stress, they are often where lesions of DJD (ie: osteophytes – which are proliferations of bone around joints) occur. Both the equine navicular apparatus in horses and Achilles complex in humans are highly specialized functional enthesis organs (see Case 2).

The enthesis organ concept provides the best explanation for degenerative arthropathies. It highlights the fact that concentration of mechanical stress at an attachment site involves not only the focal attachment site but also the surrounding tissues. Stress is therefore dissipated away from the actual enthesis to joint capsules, ligaments and the adjacent dense fibrous connective tissue, which explains why they are usually also affected in degenerative joint disease. In addition, it has been recently recognized that in many cases ‘chronic arthritis’ is primarily a degenerative rather than a primary inflammatory disease

and that *mechanical stress directly induces inflammation*. This has led to a reconsideration of the role of anti-inflammatories as a primary or sole treatment for degenerative joint disease. (For reviews see: Shaw H.M. and Benjamin M. Scand J Med Sci Sports 2007; 17: 303–315; Benjamin M et al. J. Anat. 2006, 208, pp471–490; Felson D.T. Osteoarthritis Cartilage . 2013 January ; 21(1): 10–15. doi:10.1016/j.joca.2012.09.012; Zhang L.Z. et al Arthritis Care & Research Vol. 64, No. 7, July 2012, pp 960–967; and Waller C et al Knee Surg Sports Traumatol Arthrosc (2011) 19:1823–1829).

Thus mechanical destabilization caused by muscular weakness, overloading and/or asymmetrical loading will directly impact and potentially damage enthesis organs and if chronic and/or severe, induce the lesions of DJD.

- *Mechanical Damage to Bone and Cartilage:*

Bones can sense and, given time, remodel to adapt to biomechanical stresses, as evidenced by the fact that patterns made by the bony trabeculae line up with the mechanical forces on the bone. In order to adapt to changes in mechanical stress, the bone must be able to sense it. Osteocyte processes pass through small canals in the bone to interconnect with each other and it is thought that they sense mechanical stress through changes in the fluid pressure in the canals. These canals can be injected with dye to see the connections. With continual mechanical damage to the bone, such as occurs in DJD, this network is disrupted and repair processes are inhibited. *At the cellular level mechanical signals are directly converted into biochemical responses through mechanotransduction and, when they are excessive or otherwise abnormal (i.e.: pathomechanical), they directly induce degeneration and the release of inflammatory mediators from all the tissues they impact.* In addition, extensive molecular cross-talk between articular cartilage and subchondral bone allows them to function and respond to mechanical stress as a unit, which emphasizes that repair of cartilage without addressing the mechanical stress on the underlying bone is unlikely to be successful (For reviews see: Chen & Ingbar D. Osteoarthritis and Cartilage (1999) 7, 81–94; Vincent T.L. Current Opinion in Pharmacology 2013, 13:449–454; Zhang L.Z. et al Arthritis Care & Research Vol. 64, No. 7, July 2012, pp 960–967).

- *Mechanics and Pain*

Many believe that tissue lesions cause pain and they do, but ***pain***, especially in the musculoskeletal system, has an important preventive function, in that it ***is a signal for pending as well as ongoing tissue injury***. The importance of this is emphasized by the fact that people born without pain receptors seriously damage themselves, especially their limbs. That pain is a warning system of pending injury should not come as a surprise, as an injury to a limb is invariably fatal in the wild. Mechanical pressure receptors are all around joints and tendons. Pain as a signal for pending musculoskeletal injury explains why pain and lameness can occur without evidence of tissue injury, and was recently very well explained by a doctor who treats ballet dancers and musicians (Clin Rheumatol. 2013 Apr;32(4):425-34). This concept explains why painkillers alone fail to stop progression of

the tissue injury and eventually fail to be effective. Unfortunately it is poorly understood as painkillers are often the sole treatment offered for DJD. From a pathomechanical perspective, simply prescribing painkillers without correcting the mechanics that are inducing the tissue injury will ensure that the joints are further damaged, thus higher and higher, and potentially addicting doses will be needed and that the drugs will eventually become ineffective.

Emerging New Paradigm: DJD is a Disease of Mechanics

The role of aberrant mechanical forces in the pathogenesis of DJD is increasingly being recognized (for reviews see: Felson D.T. Osteoarthritis Cartilage . 2013 January ; 21(1): 10–15. doi:10.1016/j.joca.2012.09.012; Egloff C et. Al. Swiss Med Wkly. 2012;142:w13583). Although the term *pathomechanics* has been used in the current paradigm under which tissue damage is considered to cause aberrant biomechanics, the term is better used to describe the aberrant biomechanics that directly damage the joints. Under this emerging new paradigm, pathological forces induce joint damage through excessive compression, overloading and instability. Correcting and perfecting joint function is the basis for training athletes and has dramatically increased levels of performance. It is also the basis of sports medicine, physical therapy and rehabilitation programs. But how well does the pathomechanical paradigm answer the important questions?

- What is the root cause of DJD and why does it get worse?
 - Almost all forms of DJD have a mechanical component
 - Mechanical disruption alone, but not inflammation alone, can induce DJD
 - Mechanical disruption directly induces inflammation
 - *Pathomechanics overwhelms all other factors in the progression of DJD*

- Why doesn't the severity of lesions always correlate with pain or lameness?
 - The lesions are NOT, as has been assumed, always the primary source of the pain
 - They form *in response to* mechanical overloading and joint instability
 - Mechanical correction may not resolve the lesions, but in many cases it can relieve the pain & allow return to function
 - Some damage is permanent, but how much repair is possible once the mechanics have been corrected has not been well investigated under the current paradigm

- If DJD is caused by 'wear and tear', why does exercise help patients with DJD regardless of its severity?
 - Pain relief results from stabilization and de-compression of the affected joints

- This comes from relief of overloading and the correct development of the muscles affecting the joint (ie: the wrong exercise will further de-stabilize the joint and make DJD worse)
- Why do the current treatments (painkillers, anti-inflammatories, cartilage repair) fail?
 - Painkillers, anti-inflammatories relieve pain but do not prevent further mechanical damage
 - Cross talk between tissues means that attempts to repair cartilage will fail as long as the mechanical environment is pathological
 - Bone and joint tissues have the capacity to repair damage, but only *if the mechanical forces are corrected*

Summary: Pathomechanics and DJD

- Pathomechanical loading directly induces bone and joint damage, inflammation and pain
- Pain is a signal of pending as well as ongoing tissue injury
- The ability of muscles to buffer mechanical forces is critical to joint stability
- Many of the lesions in DJD are in the enthesis organs, which transfer mechanical stress from soft tissues to bone, and are formed to compensate for the stress
- No therapies will be truly effective unless the individual's pathomechanics are corrected
- Exercise designed to restore proper muscle function and joint mechanics *relieves symptoms at all stages of OA and slows or stops progression of DJD*
- *It is increasingly being realized that correction of the pathomechanical forces is both therapeutic and preventative (Prehabilitation) and will likely make more invasive treatments unnecessary*

Clinical Applications:

Why understanding the importance of pathomechanics in degenerative joint disease is critical to helping patients and clients.

Patient, or for animals: client, understanding and engagement are critical for the successful prevention and treatment of a variety of diseases and are especially important in degenerative joint diseases. The understanding that chronic postural asymmetries and aberrant movements are often both the root cause of degenerative joint disease and the reason for disease progression is as unfamiliar to patients/clients as it is to many doctors. This is especially unfortunate given the fact that once made aware of the simple concept that: "how you move can lead to injury", the patient/client is often best able to identify the movement that is creating the abnormal stress, and to develop ways to correct it. For example, sometimes correction of pathomechanical stress and the consequent pain relief is as simple as increasing the use of the less dominant hand for carrying objects or repetitive tasks (see example in case two). In addition, if patients/clients are educated to think

pathomechanically about their musculoskeletal pain they will take an active rather than a passive role in their therapy, and are much more likely to have a positive outcome.

To be most effective, patient/client education should begin at the time of initial diagnosis and the doctor should be emphasizing that identifying and correcting the underlying biomechanical abnormalities is *more effective than the painkillers prescribed, or in many cases, surgery*, even if he/she cannot identify exactly what is wrong and is referring the patient for physical therapy. The physical therapist is thus empowered by the medical doctor's endorsement, and an effective health professional team is established for the patient. This kind of team cannot be truly effective if the doctor does not really understand, why correcting pathomechanics is so important in the treatment of degenerative joint disease, and cannot adequately emphasize to the patients/clients that they must be engaged and consistently work to improve their, or their animal's, musculoskeletal health. An excellent example of a doctor using a pathomechanical approach to very effectively treat ballet dancers and musicians was recently described (Clin Rheumatol. 2013 Apr;32(4):425-34).

The Consequences of a Failed Paradigm:

The widespread belief in both human and veterinary medicine that painkillers are the primary therapy for chronic degenerative joint disease has had tragic consequences, especially as more effective painkillers have been developed. In human medicine, opioid painkillers like OxyContin have been increasingly prescribed to treat chronic musculoskeletal pain, despite the fact there is little evidence of their efficacy in chronic nonmalignant diseases. The result has been an epidemic of drug addiction and poisoning that has contributed to an increase in the mortality rate of middle-aged white Americans (<http://www.nytimes.com/2015/11/08/opinion/sunday/how-doctors-helped-drive-the-addiction-crisis.html>; Proc Natl Acad Sci U S A. 2015 Dec 8;112(49):15078-83).

Similarly, powerful painkillers are used to block the pain induced by musculoskeletal injuries in race and show horses. It has been estimated that 24 horses die each week on American racetracks, many of which were running on powerful prescription painkillers. The veterinarian's role in this tragedy was facilitated by the belief that allowing horses to run on painkillers would make racing 'safer and more humane', in reality the painkillers masked serious, often chronic injuries, which made the development of more biomechanically-optimized training methods a low priority, and resulted in more catastrophic breakdowns. (<http://www.nytimes.com/interactive/2012/04/30/us/breakdown-horses-series.html>).

Since non-specialists write many of the prescriptions for painkillers to treat chronic musculoskeletal disease, the best approach for preventing these unintended and tragic consequences is a better understanding of the critical role of pathomechanics in chronic musculoskeletal injury by both medical doctors and veterinarians. The purpose of the following cases is to facilitate this understanding.

III) Understanding the Mechanics of Degenerative Joint Disease and Helping Patients: Characterizing Pathomechanical Forces through Static Posture (Free Body) Analysis

Free-body Analysis: What is it?

- In any given position, forces and torques acting on a particular skeletal element balance one another in a state of static equilibrium.
- Forces and torques can be resolved graphically (to identify sources of pathomechanical loading in an individual) and mathematically (to test hypotheses).
- Although the resulting analysis represents a static condition or an infinitesimally small instant within a movement, comparing the changes in forces and torques throughout several instants of a movement (i.e., phases) is useful for understanding how forces change during motion.

Free-body Analysis: The Basics

Force: any interaction that, when unopposed, will change the motion of an object

- Magnitude: given as a scalar (single number)
- Direction: given as an angle with degrees.
 - In 2D as two components (horizontal and vertical; or x and y)
 - In 3D as three components (x, y and z)
- So, each force is a vector with a magnitude and a direction.
- Each force is analyzed separately.
- In a state of static equilibrium, the sum of all horizontal forces equals zero and so does the sum of all vertical forces. In other words, all horizontal force components and all vertical force components are balanced.

Torque: the tendency of a force to rotate an object about an axis, fulcrum, or pivot

- Given as a magnitude and a sign (generally "+" for counterclockwise; "-" for clockwise)
- Each torque, like each force, is analyzed separately in relation to its center of rotation.
- To obtain the torque of a particular force: the magnitude of the related force vector is multiplied by its torque arm (the distance from the center of rotation to the point at which the force acts; in 2D a torque arm is drawn at a right angle to the line of each force vector; in 3D a torque arm is drawn directly to the point on which the force acts).
- In a state of static equilibrium, the sum of all torques equals zero or, in other words, all clockwise and all counterclockwise torques are balanced.

The Use of Statics in Understanding Motion

- Analyzing consecutive postures (e.g., phases in a step) and the accompanying change in the force regime is vital for understanding motion.

Applying Free Body Analysis to Understand the Pathogenesis of DJD: A Cross Species Approach

Free body analysis was used to characterize the pathomechanics in cases of degenerative lumbosacral stenosis and spondylosis in dogs (case 1), navicular disease in horses (case 2) and the forward neck posture in humans (case 3). The analysis increases in complexity from case 1 to case 3, and illustrates how free body analysis can be used to understand and research the anatomic, functional and individual susceptibility to the mechanical tissue damage that characterizes degenerative joint disease. Most importantly, it can also be used to educate patients and clients as well as develop targeted mechanically-based therapies. Cross species comparisons stressing similarities and differences in pathomechanical – induced injury in humans and animals are made in each case.

Case 1: Dog with Degenerative Lumbosacral Stenosis

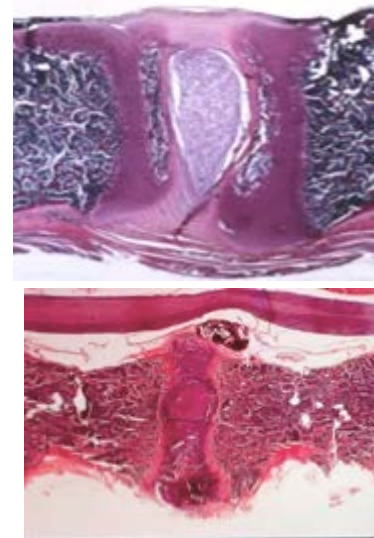
Human Comparative Aspect - Psoas Muscle Complex

Objectives:

- 1) Students will compare the position of the body during two phases of movement.
- 2) Students will identify the forces acting within a biological system.
- 3) Students will analyze these movements and postures in order to identify areas that are under mechanical stress and how they correlate with the location of lesions.

Case History and Clinical Findings:

An 8 year old castrated male Doberman that performed in agility competitions presented with a history of refusing to jump obstacles and having difficulty rising and lying down. Clinically he had lumbosacral pain, hyperesthesia of the lumbosacral region, difficulty rising, unilateral hindlimb lameness and posterior paresis. He also had pain on hyperextension of the caudal lumbar spine with lumbosacral pressure (lordosis test). Radiographs revealed collapse of the intervertebral disc space, sclerosis of the vertebral end plates, telescoping in the caudal aperture of L7, ventral subluxation of S1 and ventral spondylosis. Based upon these findings a diagnosis of degenerative lumbosacral stenosis was made (Meij B.P. et al 2010. Vet Clin Small Anim 40:983).



A normal disc from a young dog (top) and a ruptured disc with compression of the spinal cord by degenerate remnants of nucleus pulposus in 12-year-old Dachshund (bottom). Images courtesy of J. C. Woodard

Degenerative Lumbar Spinal Disease in Dogs and Humans: Biomechanical Considerations

Lower back pain is one of the most common medical problems, affecting between 70-85% of the human population at some point in their lives; 90% of people over the age of 50-60 have degenerative changes in their intervertebral discs (Biyani A. et al. 2004. J Am Acad Ortho Surg 12:106). Degenerative lumbosacral stenosis is the canine equivalent of lumbar disc herniation in people and is also characterized by lumbosacral and limb pain induced by compression of the nerve roots (da Costa R.C. 2014. Vet J 202:201). It primarily occurs in large breed dogs and is especially common in German Shepherds (Oliver JE et al. 1978. JAVMA173: 207). As in humans, degeneration of the intervertebral disc plays an important role in degenerative lumbosacral stenosis in dogs. In both diseases mechanical stress is considered to be a critical factor in degeneration of the intervertebral disc as the intervertebral joint is the most important structure for the maintenance of vertebral stability (Meij B.P. et al 2010.Vet Clin Small Anim 40: 983).

The intervertebral disc comprises the annulus fibrosus, the nucleus pulposus and the cartilaginous end plate. The annulus fibrosus connects adjoining vertebrae and provides tensile strength. It is composed of type I collagen arranged in well-organized lamellar layers. The jelly-like nucleus pulposus distributes the load over the vertebral end plates and acts as a shock absorber when the spine is subjected to compressive forces. The cartilaginous end plate covers the ends of the vertebrae. It is semipermeable and allows nutrients to diffuse into the disc (Meij B.P. et al 2010.Vet Clin Small Anim 40: 983).

Although humans are bipedal and dogs are quadrupedal, the axial compressive forces on the vertebral column are similar (Zimmerman M.C. et al 1992. Spine.17:213–20). There are other biomechanical similarities between the canine and human lumbar spines, even though humans have 5 lumbar vertebrae while dogs have 7 (Smolders LA. et al. 2012 Eur Spine J 21:1692). Canine spines have considerably less axial rotation than human spines, but the intersegmental differences in flexion-extension, axial rotation and lateral bending are similar. The absolute mobility in the L1-L2 and the L2-L3 segments in flexion and extension are comparable, although the dog has more range of lateral bending in this area. The flexion-extensional mobility of the L6-L7 segment of the canine spine is comparable to that of the L4-L5 segment in humans. The lateral bending mobility of the dog L7-S1 region is similar to that of the human L5-S1, but it is more mobile in flexion and extension. While there are differences in the absolute mobility of different segments, overall the canine lumbar spine resembles the human spine biomechanically (Smolders LA. et al. 2012 Eur Spine J 21:1692).

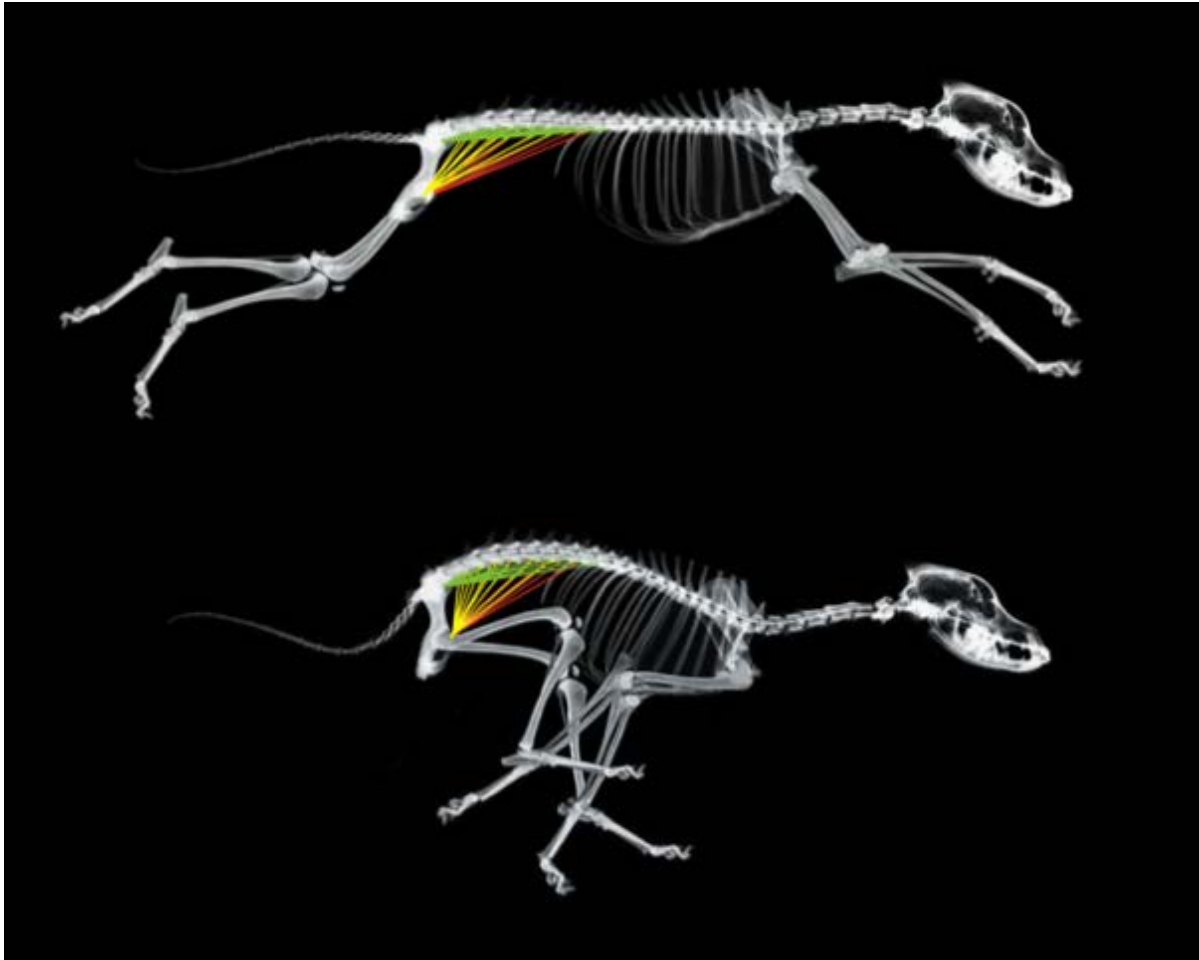
Ventral spondylosis (spondylosis deformans) in the lumbar region is a very common lesion in dogs as well as humans, and was found in 61-75% of dogs presented for necropsy (Levine G.L. et. Al. 2006. JAVMA 228:96). However, it is very often incidental and is not always associated with degeneration of the intervertebral disc. In the dog the area with the most extensive and highest incidence of spondylosis lesions is from T13-L5 and the lumbosacral joint (Levine G.L. et. Al. 2006. JAVMA 228:96).



Spondylosis, especially in the cranial lumbar spine is one of the most common, usually incidental, lesions found at necropsy and in canine paleopathological specimens. The top specimen is from a Pre-Columbian dog while the bottom specimen is from a modern case.

The distribution of bridging spondylosis is best explained by a consideration of how the spine functions when a dog runs.

Question 1: In the images of the running dog, what region of the dog's spine has the most range of movement? What are the muscles attaching to the lumbar spine and what do they do?



Answer:

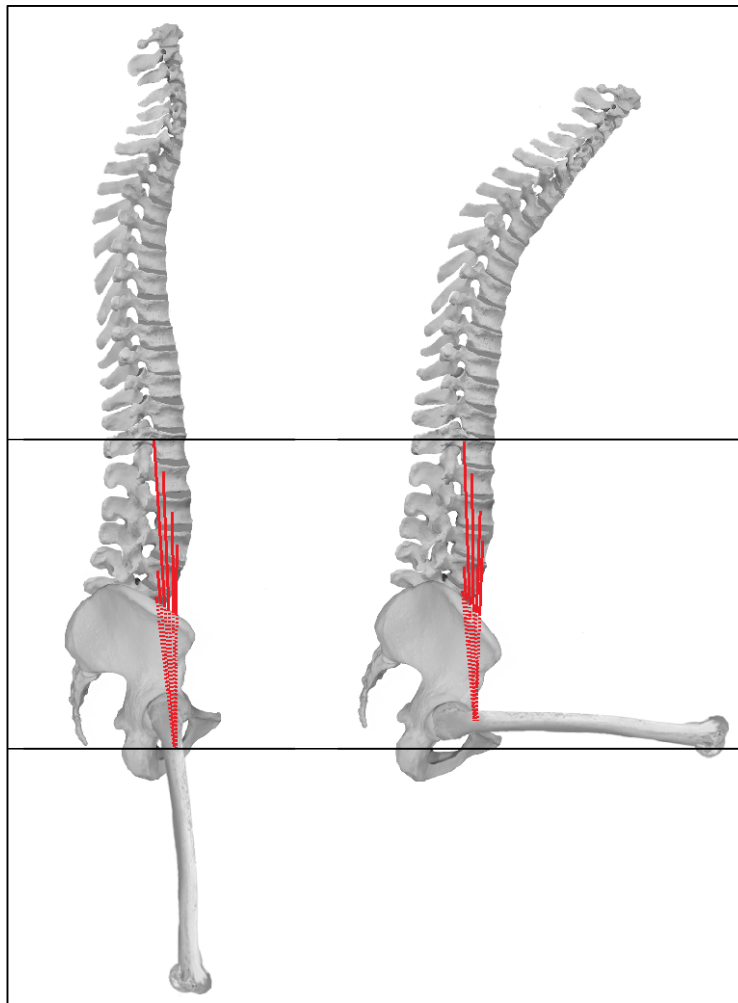
Increased or aberrant repetitive mechanical stress induced by movement will especially impact the areas of the spine that are the most mobile, which are the areas most affected by spondylosis and degenerative lumbosacral stenosis. When pathomechanical forces increase instability, there is hypertrophy and ossification of the soft tissues of the affected joint's enthesis and, over time, bone can bridge the joint (ventral spondylosis) to re-stabilize the joint.

Degenerative Lumbosacral Stenosis: Pathomechanical Considerations

The proposed pathogenesis of degenerative lumbar spinal disease in dogs is that chronic abnormal motion of the lumbosacral junction, caused by repetitive stress and/or genetic or congenital abnormalities (the most likely being related to conformation) induces degeneration of the L7-S1 disc (Meij B.P. et al 2010.Vet Clin Small Anim 40: 983). Intervertebral disc degeneration begins with degradation of proteoglycans, which leads to dehydration, loss of size and further degeneration. The instability induced by the disc degeneration combined with the resultant changes in mechanical loading can eventually lead to subluxation of the sacrum and the development of spondylosis at the lumbosacral joint (Meij B.P. et al 2010.Vet Clin Small Anim 40: 983).

Dogs with degenerative lumbosacral stenosis have a reduced range of lumbosacral flexion-extension and can present with subluxation of S1 (Meij B.P. et al 2010.Vet Clin Small Anim 40: 983). This potentially results from strain injuries caused by over-stretching of the iliopsoas (psoas major and iliacus) muscle complex and increased stress on the intervertebral joints. Over-contraction of these muscles, both to compensate for the instability and in response to the pain, can induce misalignment and even subluxation of the sacroiliac joint (Caban Q. et. al. 2013. Compend Contin Educ Vet 35(5):E2).

Comparative Considerations: The Human Psoas Muscle Complex



In the human, the psoas muscle complex is similar to that of the dog in location and, like the dog, it flexes the hip and affects the curve of the lumbar region. However, whereas the spine of the dog deals with compressive forces in the longitudinal axis as its hindquarters move forward during movement (watch the video of the running Dachshund to see this: <https://www.youtube.com/watch?v=OpXw3oMraUI>), the human spine constantly deals with the compressive forces in the longitudinal axis caused by gravity. These compressive forces are increased when the hip is flexed and the trunk bent over, as when one is sitting with the forward head posture. Maintaining this position for long periods of time will eventually shorten the muscles and their related connective tissues (see case 3). To counteract this position when standing (i.e., to stand up straight) an individual must extend their spine, which has become flexed- so the individual must compensate more to remain upright and straight. These actions can induce strain of the psoas muscle complex and instability of the lumbar joints and lead to disc degeneration, instability and spondylosis (Plastaras, C.T.; et al. 2007. *The Lumbar Degenerative Disc. In Interventional Spine: An Algorithmic Approach* ed. Slipman, C.W. et al. Saunders Elsevier.)

Thus, even though the exact movements causing excessive and destabilizing forces on the lumbar and sacral areas of the spine in humans and dogs are different, the pathomechanical effects on the intervertebral joints and pathological changes are very similar.

The Breed Effect: Why is Intervertebral Disc Disease More Common in Chondrodystrophic Breeds?

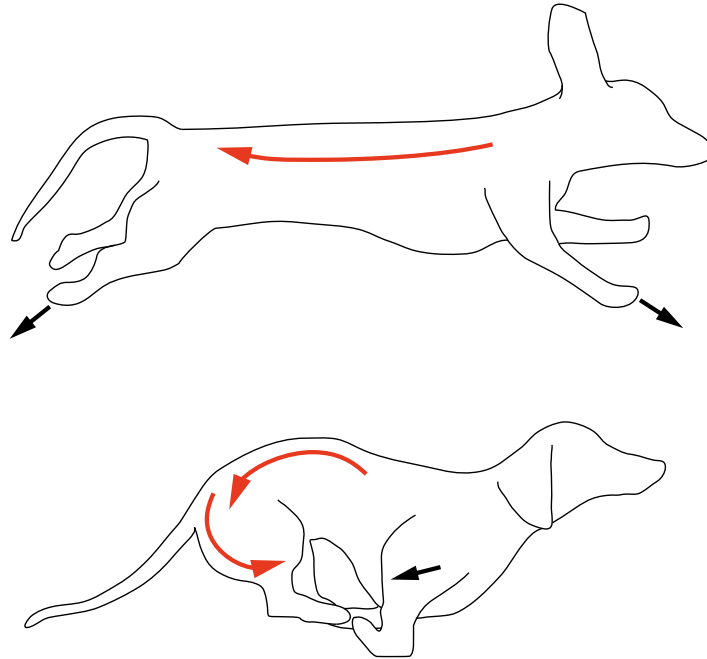
The domestic dog has the widest range of phenotypes of any mammal, there being a 200X time size difference between the largest and smallest breeds. This wide variation in musculoskeletal features must be considered in pathomechanical analysis of chronic degenerative joint disease.

The exact areas under biomechanical stress in dogs are breed specific, since there is an extremely wide range of body types, sizes, and proportions. Lesions found in the cervical and thoracic regions of the modern dogs are likely to have additional causes such as overzealous breeding for more extreme body sizes and shapes, and diet (especially those leading to obesity) (Kranenburg H.J.C. et al. 2011 Vet J 190:e84; Levine G.L. et al. 2006. JAVMA 228:96).

The long body short-legged phenotype of many of the chondrodystrophic dogs impacts the biomechanics of the spine. For example, chondrodystrophic dogs have a larger range of axial rotation in their spines and the biomechanical effects of decompressive spinal surgery are different than for non-chondrodystrophic dogs (Smolders LA. et al. 2012 Eur Spine J 21:1692). Interestingly, the intervertebral discs in chondrodystrophic dogs degenerate at a very young age. This degeneration is characterized by cartilage metaplasia and mineralization of the nucleus pulposus and is similar, although accelerated, to that observed with aging in humans (Smolders LA. et al. 2012 Eur Spine J 21:1692). While genetic changes related to the chondrodystrophic phenotype may also be inducing rapid degeneration of the intervertebral discs, excess mechanical stress on the spine related to the long body, short-legged conformation is likely to also be contributing.

How do Dachshunds run and where is the mechanical stress on their spines?

Watch the following video in slow motion (.25 speed) to better understand the movements: <https://www.youtube.com/watch?v=OpXw3oMraUI>



Top figure: As the Dachshund runs, it kicks off the ground. Its hind legs and pelvis are thrown back and the spine is extended. Bottom figure: In the next phase of the movement, the spine is flexed and the pelvis tucks under the body- the hind legs overlap with the front legs as the Dachshund prepares to unfurl. Flexion and extension occur in the lumbar spine. Compare the lumbar flexion and extension in this dog with that of the non-chondrodystrophic running dog.

Because the domestic dog has such a wide range of phenotypes, breed and individual characteristics must be considered in pathomechanical analysis of chronic degenerative joint disease in dogs.

Comparative note: The horse moves with minimal flexion and extension of the lumbar spine compared to dogs and humans and lumbar spondylosis and intervertebral disc disease are comparatively very rare in horses.

Clinical Applications: What a Clinician Can Do

- Conceptualize the lesions as a result, rather than the cause of the problem.
- Explain to the patient/client how the spine moves in themselves or the species of interest and why specific areas are susceptible to biomechanical damage
- Help the patient/client identify individually specific ways of moving that may be leading to aberrant biomechanical forces and potential ways to change these movements to relieve the pathomechanical stress.

Case 2: Horse with Navicular Disease

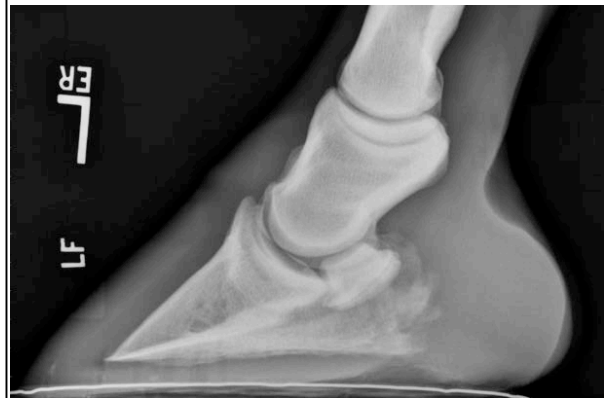
Human Comparative Aspect - Achilles Enthesis Organ and Handedness

Objectives:

- 1) Students will compare and contrast the functional entheses organs of two different species.
- 2) Students will identify the pathomechanical forces acting within an individual and how they correlate with lesions of degenerative joint disease.
- 3) Students will be able to describe how forces in one area can cause damage in another.

Case History and Clinical Findings: An 11-year-old Trakhener gelding that had been worked in dressage developed a chronic left front limb lameness. Clinical examination and nerve blocks revealed he was also lame in the right front, and the lameness was characterized by short strides, stumbling, unevenness on turns and heel pain. Radiographs confirmed a diagnosis of navicular disease, which is characterized by degeneration of the navicular bone and damage to the surrounding soft tissue. What is the navicular apparatus and why is it so subject to injury?

<p style="text-align: center;"><i>How Do Horses Actually Move?</i></p> <p><u>Hind legs:</u> generate primarily forward, with some upward, thrust</p> <p><u>Spine:</u> Channels the propulsive forces; back muscles protect the spine from the effects of gravity and the forces generated by the legs. Forward thrust is passed forward and loads the front legs</p> <p><u>Front legs:</u> Manage the loading forces by pushing up. Like a pogo stick, if too much force is applied, the muscles and elastic recoil are overwhelmed and the force compresses the bones and joints.</p>



Premiere Enthesis Organs: The Human Achilles and the Equine Navicular Apparatus

The human Achilles tendon attachment has been characterized as being the premiere functional entheses organ (Benjamin et. al. 2004 Arthritis & Rheumatism 40:3306). The equine navicular apparatus, although an anatomically different structure, has all of the same basic structural adaptations to mechanical stress as are found in the human Achilles entheses, an observation that should not be surprising given their shared mammalian musculoskeletal heritage (Bentley V.A. et. al. 2007 J. Anat. 211:662). At both sites, tensile mechanical stress, especially that created by ***dorsiflexion of the foot***, is dissipated away

from the osteotendinous junction by contact of the tendon with bone proximate to the actual attachment site. This bony contact takes place on the superior tuberosity, which is a protuberance of bone on the calcaneus, in humans, and the navicular bone, a sesamoid closely attached to the distal phalanx, in the horse. For both structures, this contact allows the tendon to bend to its insertion site, like a wrap around pulley, thus ensuring that the angle of attachment is maintained throughout various movements of the foot. It also acts as a fulcrum, which provides both the Achilles and equine deep digital flexor tendon a long lever arm and thus a mechanical advantage at their action on the calcaneus and third phalanx respectively (interestingly the navicular bone is fused to the distal phalanx in rhinoceroses, which provides an even stronger fulcrum for the deep digital flexor tendon). Finally, this contact allows for the transfer of compressive forces from the tendon to bone (ie: superior tuberosity on the calcaneus in humans or the navicular bone in horses) before the actual insertion site. If these compressive forces are excessive, damage to the bone, as is especially well characterized in navicular disease, will occur.

Other features of functional entheses shared by both the Achilles and the navicular apparatus include: a transition from the dense fibrous tissue of the tendon, to uncalcified and calcified fibrocartilage to bone; the presence of fibrocartilage at the site of bending of the tendon round the bone; the presence of a bursa between the bone and the tendon, and the presence of a fat pad (Benjamin et. al. 2004 *Arthritis & Rheumatism*, 40:3306;). The presence of fibrocartilage, both within the tendon and on the periosteal surface of the bone where it contacts the tendon, is an adaptation to withstand compression and shear, as the aggrecan-rich extracellular matrix of the fibrocartilage allows for a high water content which resists compression of the tendon as it passes over the bone. The bursa acts to minimize the frictional forces on the fibrocartilage as the tendon passes over the bone to induce movements of the foot. The presence of a fat pad plays a role in minimizing pressure changes in the bursa during movements of the foot. It also contains pain and pressure receptors that can monitor overloading of the enthesis.

The navicular enthesis does have a feature that is present in other enthesis organs, but is not very prominent in the Achilles, which is that the deep digital flexor tendon attaches in a pit or depression within the third phalanx. This type of attachment also facilitates the dissemination of mechanical stress away from the tendon attachment site similar to that just described, in that it provides a ridge of bone as a pulley or fulcrum right before the insertion. The presence of two specialized structures for the dissemination of mechanical stress within the enthesis organ of the deep digital flexor tendon indicates it is under a very high level of mechanical stress.

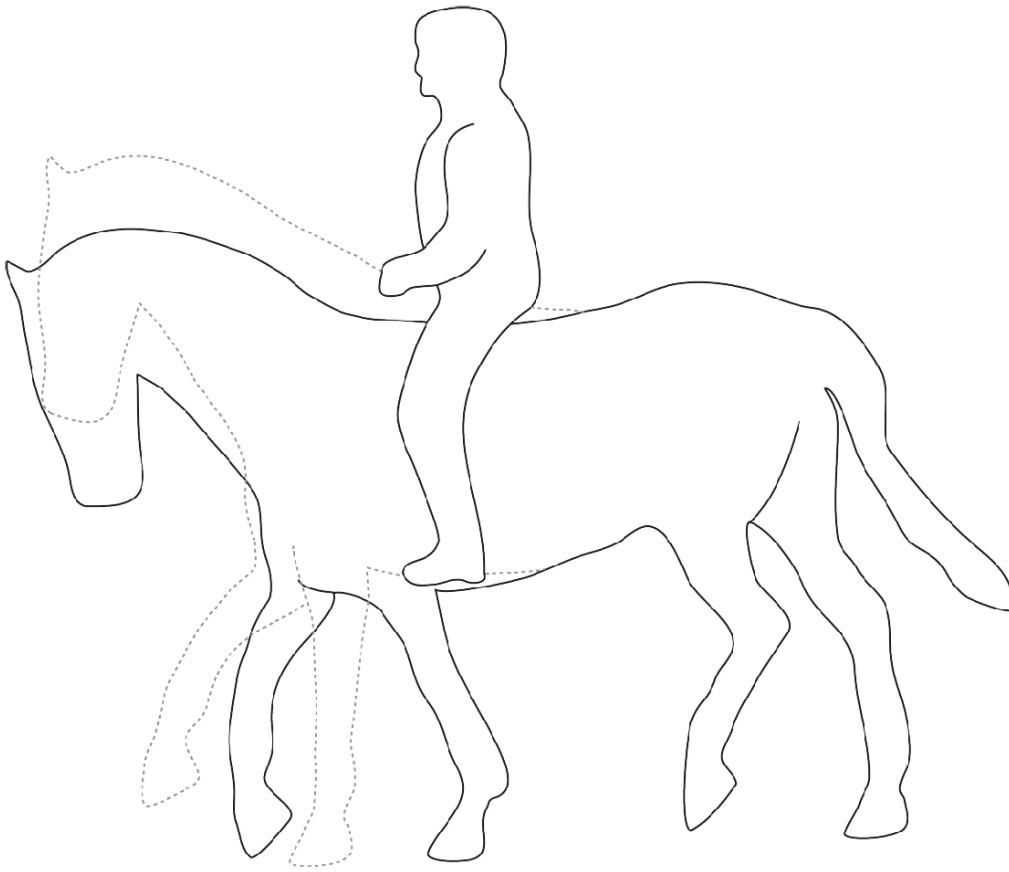


The human Achilles and the equine navicular apparatus are anatomically different structures but both are functional enthesis organs and have the same structural adaptations to manage mechanical stress. One feature is that both the Achilles and deep digital flexor tendon (tan in the images) bend around and contact bone, the superior tuberosity of the calcaneus and the navicular bone (green in the images), before their insertion sites. The bone acts a fulcrum and helps maintain the constant insertion angle of the tendon throughout the movements of the foot. Compressive forces are also transferred to the bone. The images of the human foot were modified from www.bestfootdoc.com. The images of the horse's foot were taken of plastinated sections of the foot of a horse with navicular disease and an area of hemorrhage is apparent right below where the tendon passes around the navicular bone (Specimen image from Dr. Christopher von Horst: www.plastinate.com).

Pathomechanical Analysis: Horse with Navicular Disease

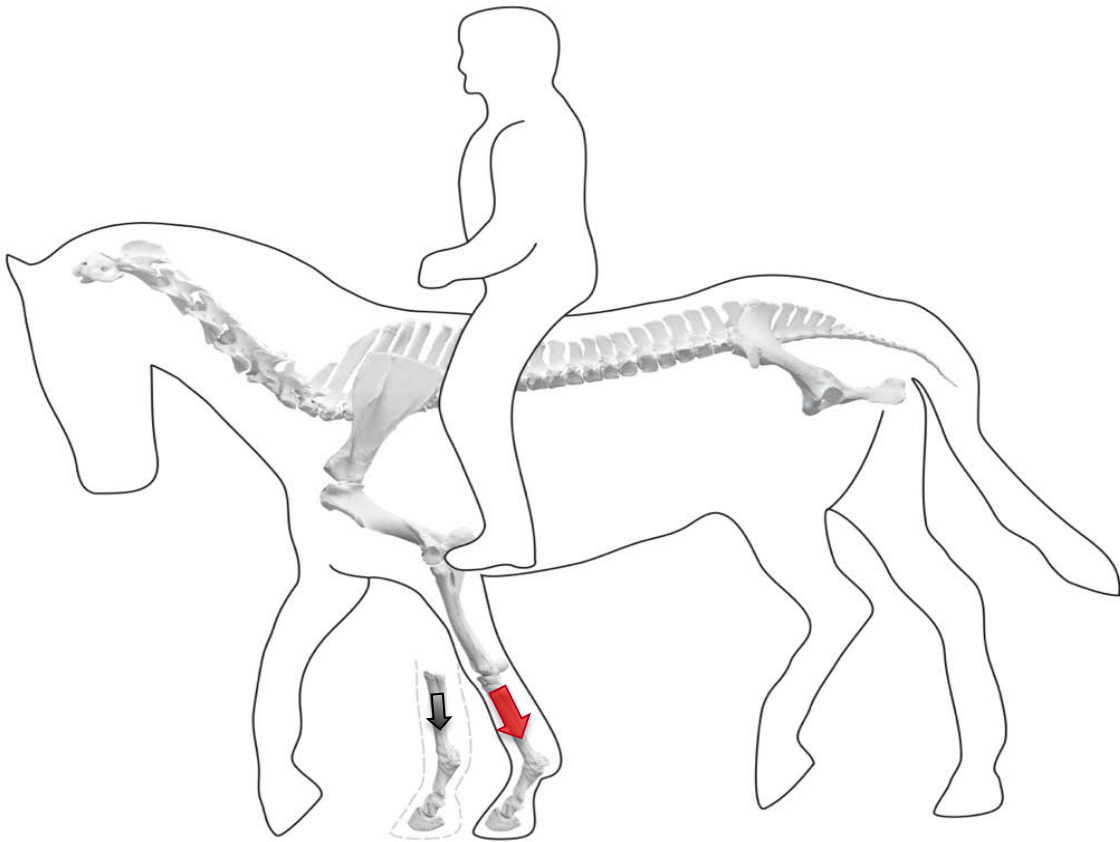
- **Functional Analysis:** An outline of this horse at the trot taken from video footage is shown below.

Question 1: How is this horse's position different from the dotted outline?

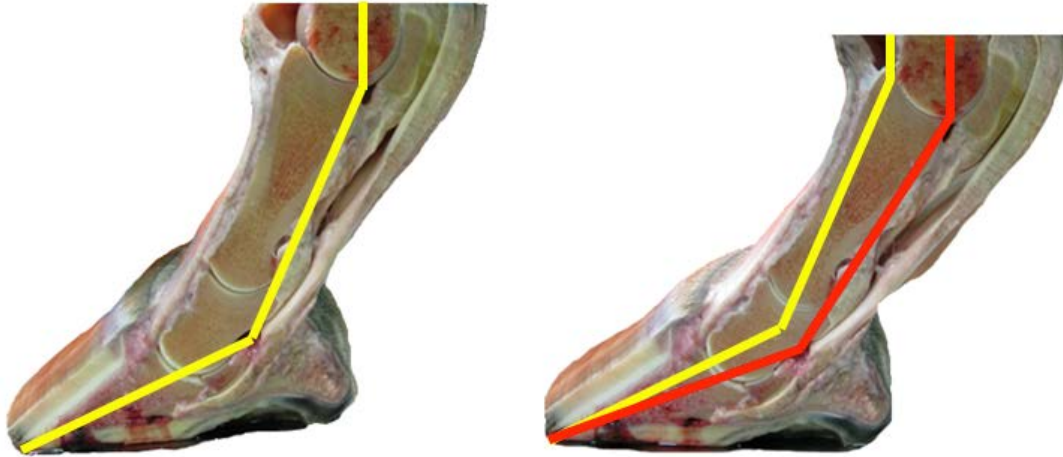


Answer:

Question 2: How is the overloading impacting the front leg?



Answer:



The normal forelimb kinematic is shown on the left and the axis of the foot is indicated in yellow. The overloading of the forelimb and the resulting dorsiflexion of the distal and proximate interphalangeal joints with compression of the foot axis, as observed in the horse with navicular disease, is shown on the right. Note the compression of the navicular entheses and heel.

- **Free-body Force Analysis:**

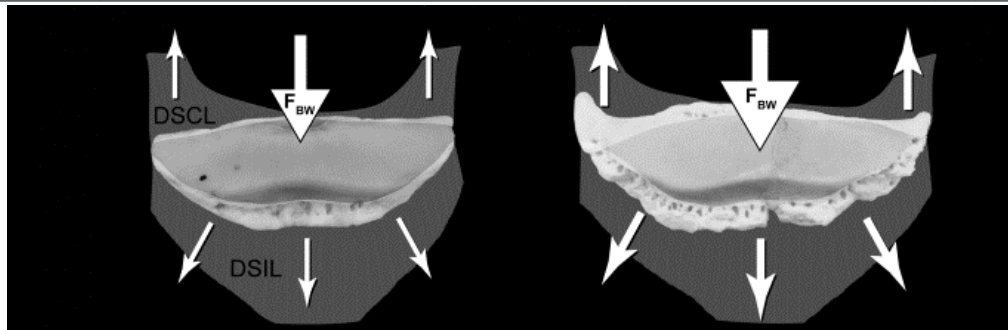
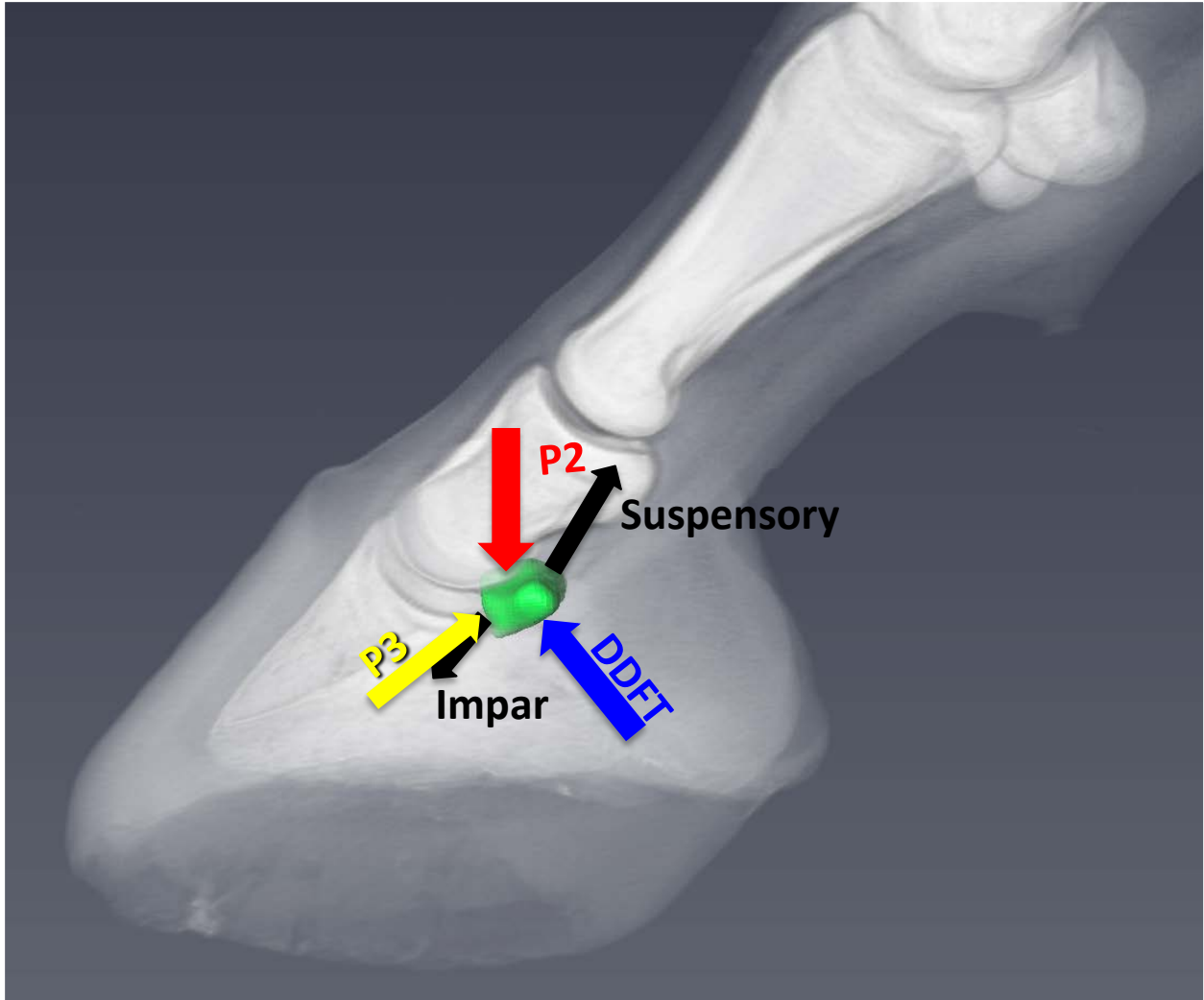
In a healthy foot position the forces acting on the navicular bone come from contact with P3 (the coffin bone), P2 (the short pastern bone) and the deep digital flexor tendon. The ligaments connect the bones and provide stability to the joints. When the foot is dorsiflexed, as illustrated above, the forces on the navicular entheses increase and the bone is compressed and damaged. The ligaments also become engaged and increase their tensile forces on the navicular bone. If the forces are chronic, bone forms where the soft tendon attaches to the bone to strengthen the entheses (osteophytes or enthesophytes). The mathematical resolution and graphic representation of the 3D force analysis of the navicular apparatus was completed by Kaitlyn Carrick Ruff, a senior in biological engineering at the University of Georgia.

Navicular Force Analysis Equations (see Case 3 for details on how force analysis is done):

- $\Sigma F_y = 0$: Sum of forces in the y direction
- $\Sigma F_z = 0$: Sum of forces in the z direction
- $\Sigma M = 0$: Sum of the moments/torques

-
- DDFT force was a known value found in literature
 - Forces from P2 and P3 were calculated using a force analysis and above equations

-
- The force from P2 is greater than the force from P3.
 - P2 directly over Navicular Bone – bone being impacted
 - Navicular bone abutted by P3

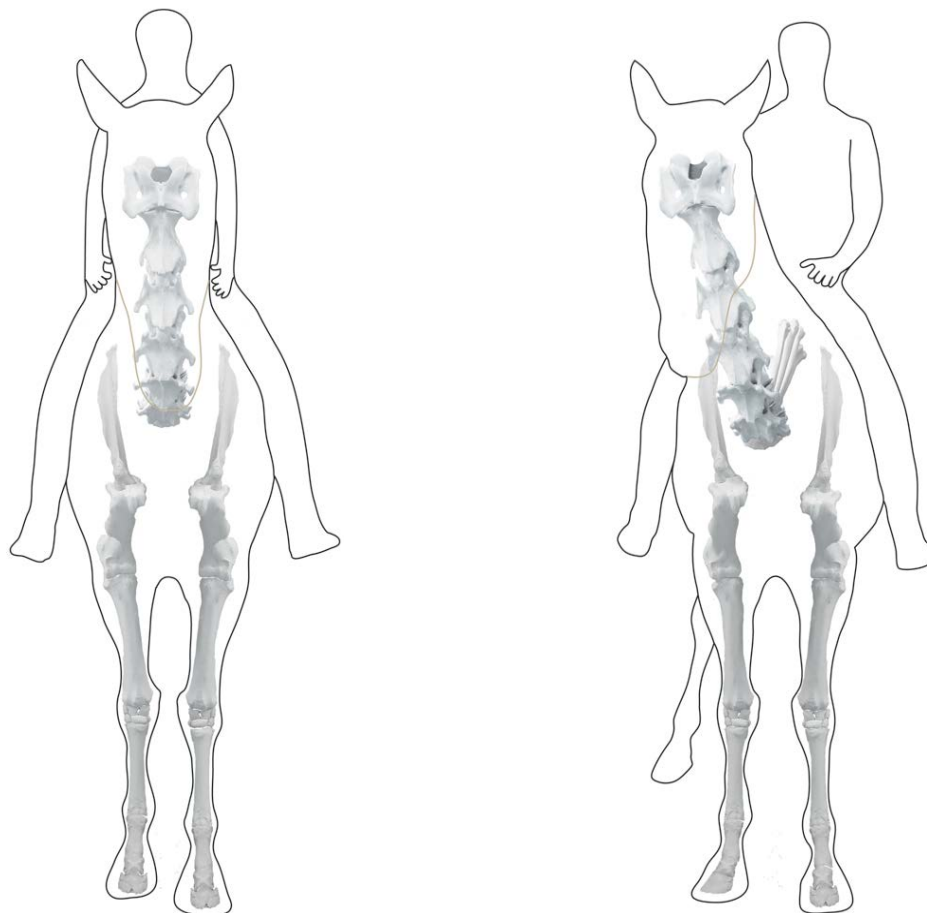


Forces acting on the navicular bone during dorsiflexion of the interphalangeal joints. Dorsiflexion increases the tensile forces from the ligaments, which, if chronic, results in bone being laid down to increase the strength of the attachment sites (enthesis) as illustrated in the bottom figure. Note: the CT scan of the foot was done in a relaxed not a dorsiflexed position.

The horse described in this example, had a bilateral and asymmetrical lameness, which is a common characteristic of navicular disease. In this case, the asymmetrical loading of the front legs can be explained by the presence of a persistent transversal rotation of the spine. Lateral bending is always associated with transversal rotations of the spine, which are localized to the first 11 thoracic vertebrae and involve several vertebrae (Denoix JM.1999 Vet Clin North Am Equine Pract. 15:27). Transversal rotations can be either in the same direction as the lateral bend of the spine (correct) or opposite to it (inverted). The rider straddles the dorsal spinous processes of a horse's thoracic vertebrae. When an inverted transversal rotation is present, the rider's seat is pushed to one side, and the perception is that the horse 'fell on its shoulder'. This perception is correct in that the inverted transversal rotation has asymmetrically loaded the shoulder and front leg.

An outline with skeletal elements added was taken from photographs of horse with a persistent transversal rotation, similar to that in the horse with navicular disease. The horse is shown standing square on the left and in his more habitual posture on the right.

Question 3: Which front leg is overloaded in the position illustrated on the right?



Answer:

Case Outcome:

The horse in this case example was rehabilitated by Jean Luc Cornille (Scienceofmotion.com). The rehabilitation focused on recreating the proper balance, and correcting the transversal rotation by symmetrical development of the spinal muscles. The horse became sound after three months of rehabilitation, returned to training and performed in dressage until fourth level. He remained sound for 3 years when he was lost to follow up.

Comparative Considerations: Acquired Postural Asymmetries

Persistent transversal rotations in horses can result from acquired postural asymmetries in the spinal muscles and, although they are transversal displacements of the spine rather than the lateral ones (scoliosis) common in humans, they are comparable to spinal deviations induced in humans by repetitive asymmetrical activities.

Question 4: What common trait in humans can lead to lateral spinal asymmetries shown?

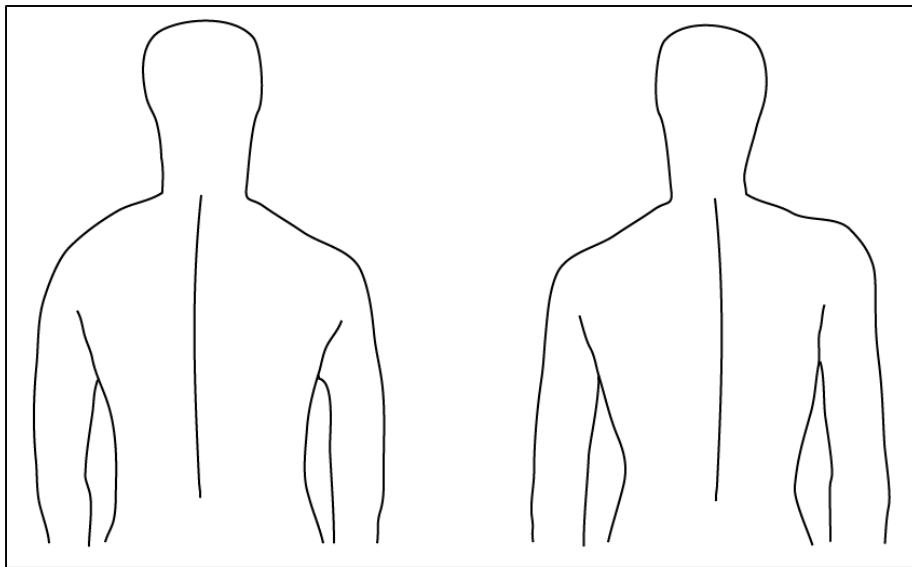
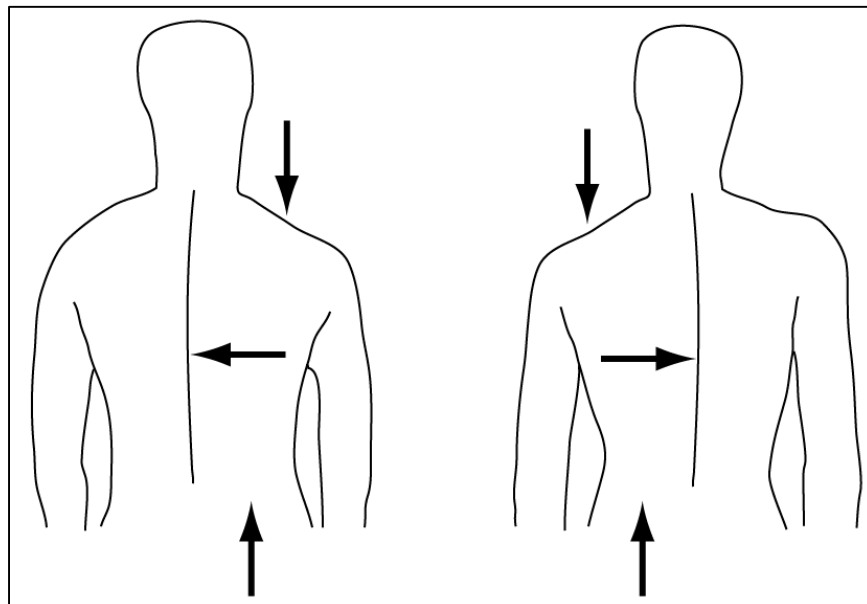


Figure is based off of image in Kendall et al. 2005. Muscles: Testing and Function with Posture and Pain

Answer:

Acquired postural weaknesses resulting from asymmetrical activities are most commonly due to handedness in humans. In right-handed individuals the muscles of the upper body that tend to show evidence of acquired postural weakness are the left lateral trunk muscles, right hip abductors, and the right hip lateral rotators (Kendall et al. 2005 Muscles: Testing and Function with Posture and Pain). These asymmetrical weaknesses continue in the lower half of the body in the right peroneus longus and brevis, left tibialis posterior, left flexor hallucis longus and the left flexor digitorum longus (Kendall et al. 2005. Muscles: Testing and Function with Posture and Pain), demonstrating that an activity seemingly focused in one region impacts the entire body. Left-handed individuals have the opposite pattern, although the weaknesses are not as common as in right-handed people, likely because left-handed individuals must use their less dominant hand more to function in environments set up for the right-handed.

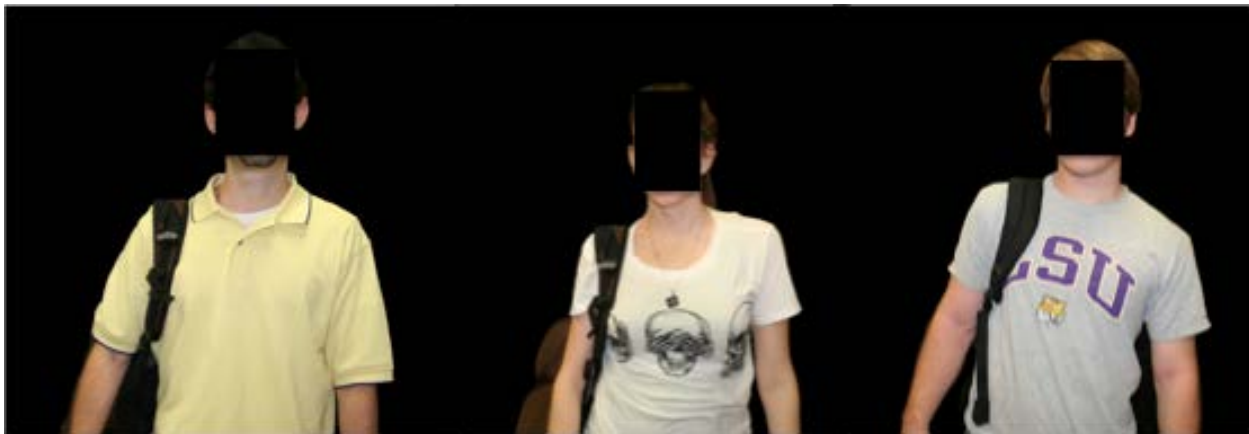
Question 5: Based on this information, which of the following individuals is likely right-handed and why?



Answer:

The carrying of external loads can enhance the pathomechanical stress associated with acquired postural asymmetries. For horses, this external load is usually the rider, and as can be seen in the figure for question 3, which shows that for a horse with a right vertebral transversal rotation, the rider has been shifted to the left, which increases the loading of the left front leg. Although people can carry objects in many different ways including: in the hands, on the head, over a shoulder, on a balanced stick or in a bag, each method has its own benefits and drawbacks (Datta SR & Ramanathan NL. 1971 Ergonomics, 14:269-278). In our modern Western society, we frequently carry heavy items like briefcases in one hand, or we overload our purses and backpacks and carry them on one shoulder. This results in individually variable ways of compensating for the extra weight. However, if poorly compensated, prolonged or excessive, compressive forces caused by the asymmetrical overloading of a shoulder can lead to clinical disease, such as thoracic outlet syndrome (TOS), in which the nerves of the lower root of the brachial plexus are compressed (Mackinnon SE & Novak CB. 2002 Curr Prob Surg 39:1070-1145). As is often the case in pathomechanical injuries, the symptoms of TOS manifest at sites distant from the primary problem and include pain or numbness in the fingers and forearm, weakness of the hand, neck pain and headache (https://my.clevelandclinic.org/services/heart/disorders/hic/Thoracic_Outlet_Syndrome). Other biomechanical causes of TOS include: repetitive arm and shoulder movements and poor, forward head posture (see Case #3 for information about forward head posture).

Question 6: In the following image of three right-handed individuals holding the same weighted backpack on their right shoulder, who is handling the weight best and why do you think this? Describe the compensations of the other two individuals.



“Physical therapy that addresses postural abnormalities, neural mobility, and muscle imbalance relieves the symptoms of most (TOS) patients.” (Mackinnon SE & Novak CB. 2002 Curr Prob Surg 39:1070-1145). This “physical therapy” may be as simple as, in the case of the heavy backpack on the shoulder, taking off some of the weight.

Clinical Applications: What a Clinician Can Do

- Educate the patients/clients about the impacts of postural asymmetries on overloading joints and the potential that damage created by the resulting compressive forces may result in pain at distant sites.
- Encourage clients or patients to evaluate how their own posture and behaviors may be inducing pathomechanical injury.
- Refer patient/clients to physical therapists with a good understanding of why correction of the pathomechanics is critical to effectively treating their pain and dysfunction.
- DO NOT simply prescribe painkillers without some plan for addressing the primary pathomechanical problem.

Case Study #3 Human Forward Head Posture: Including Extended Analysis

Comparative Considerations: Effects of Straightening the Curvature of the Neck

Objectives:

- 1) Students will compare the mechanics of a healthy and an un-healthy posture
- 2) Students will analyze these postures in order to identify areas that are dealing with too much force.
- 3) Students will hypothesize the differences in the force regimes between the healthy and un-healthy postures.
- 4) Students will test their hypotheses by creating free-body force diagrams for these postures.

Patient and Symptoms

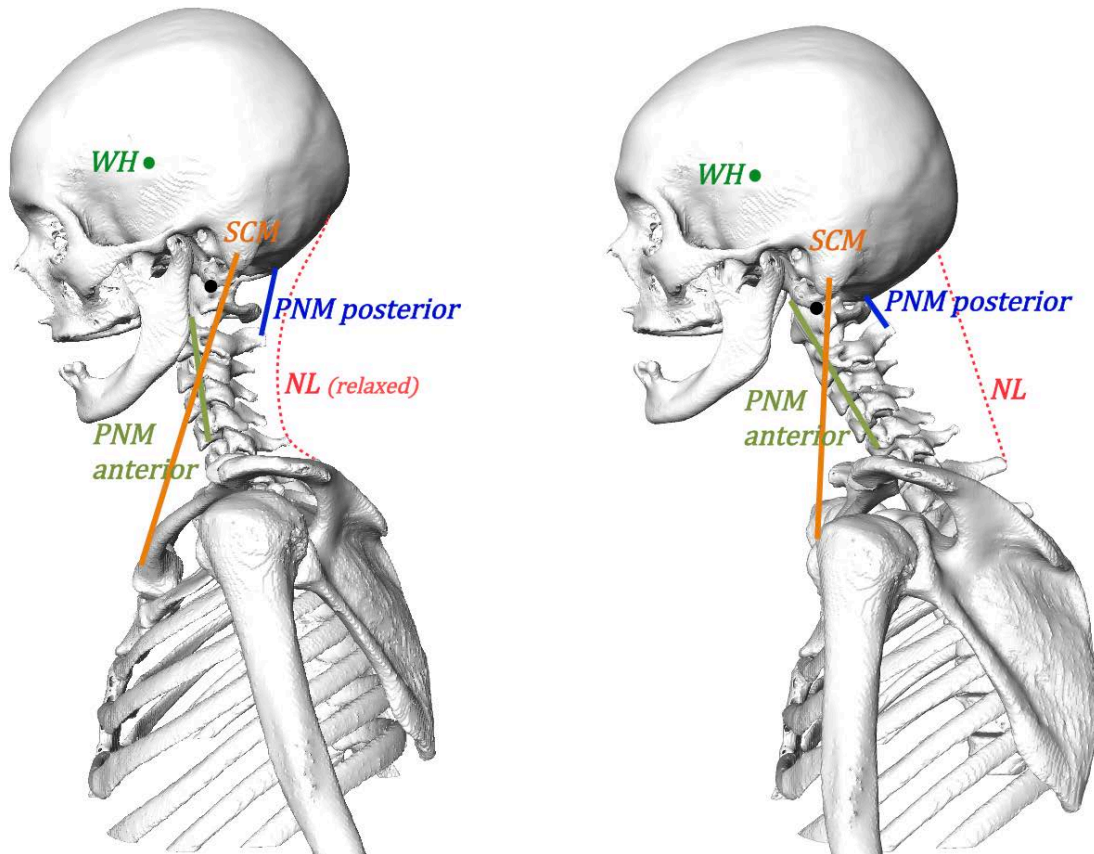
The patient is a 37 year-old female in generally good health. She is married with one child (6 years old). She is currently a full-time student and does secretarial temp work on the side. Her exercise consists of the activities she does with her son, including non-competitive bike riding and soccer. She complains of: constant headaches at the front of the head (wonders if they might be migraines); shoulder and back of neck stiffness or pain; pain at the base of the head (suboccipital pain); burning between the shoulder blades; not sleeping well at night, possibly from sleep apnea. A previous doctor prescribed an anti-inflammatory, which helps somewhat, but the problems persist and seem to have gotten worse- she often leaves work early or misses classes because of the headaches. Previous scans have shown no abnormal vertebral lesions or disc disease.

Diagnosis via Pathomechanical Analysis

Before ordering a series of expensive tests to help in identifying a treatment for the patient, you notice that she does not have good posture and seems hunched whether she is sitting or standing. Thus, you consider the biomechanics of her system and wonder whether her bad posture (i.e., forward head posture) could be the cause of her issues.



Question 1: How are the two postures different in length of muscles, and orientation of skeletal features and muscles, and what do these differences mean in regards to pain or discomfort for the patient?

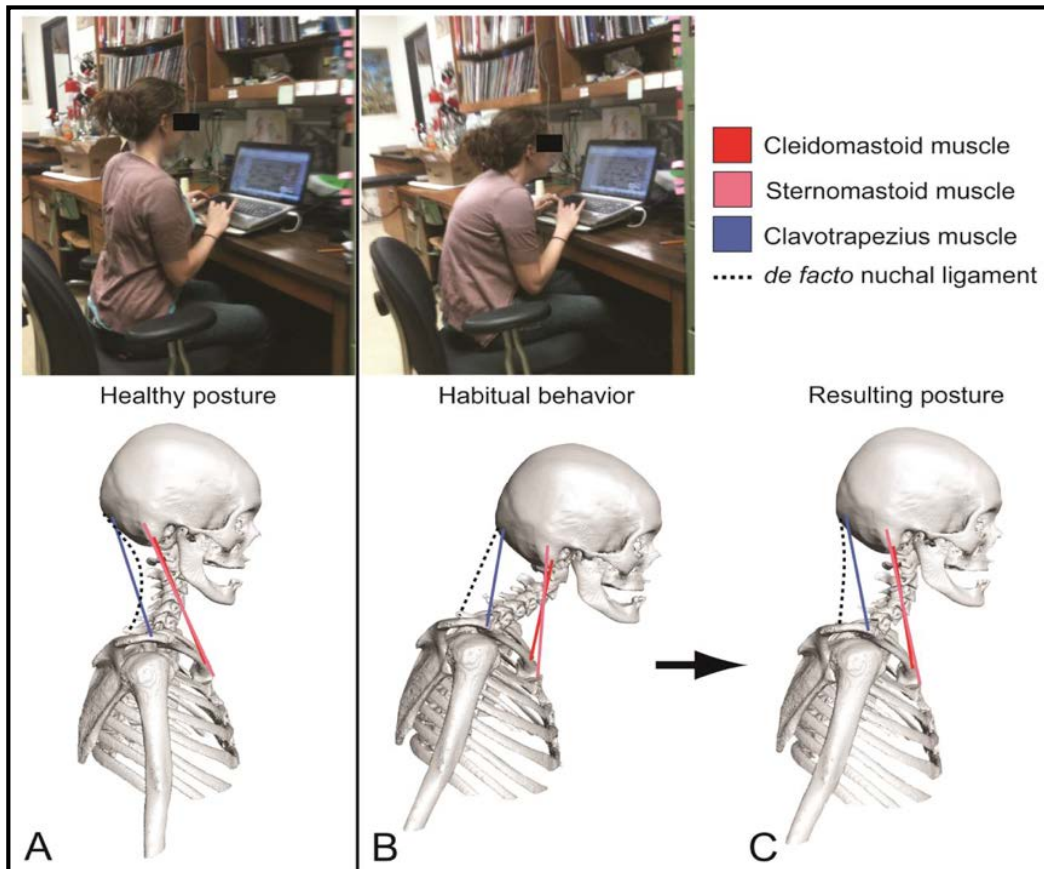


Illustrations of the mechanical forces acting in the upright versus forward neck postures. WH = weight of the head; PNM anterior and posterior = the forces of the paravertebral muscles (multifidus, semispinalis capitus and others); NL = forces from the nuchal ligament, trapezius (mostly the upper portion where it attaches to the skull), clavotrapezius, and splenius muscles; SCM = Sternocleidomastoid muscles

Answer:

Question 2: If this posture is habitual, what will happen over time?

Answer:



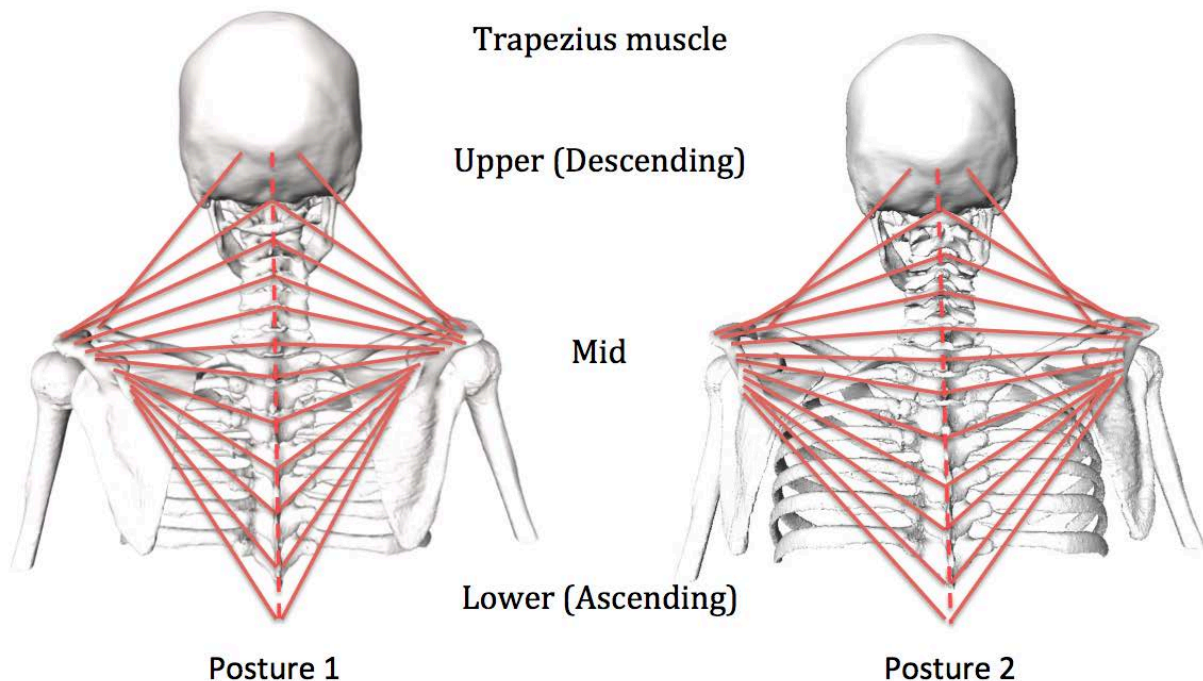
From Osborn, 2013: The forces created by habitual activities lead to noticeable structural changes. **A:** The normal, healthy posture of a human. **B:** The habitual craning of the neck and poor posture while working (e.g., on a computer) of a human. **C:** Resulting change in the configuration of the skeletal and muscular elements of the shoulder suspension apparatus even when the individual is relaxed. All images of the “Visible Human Female” are used with permission from the National Library of Medicine’s Visible Human Project®.

More generally speaking, certain behaviors, over time, (e.g., sitting with a forward head posture at a desk, 8 hours a day for 10 years) will affect your resting posture if no preventative steps are taken. Thus, your postures are a direct reflection of behaviors and movements (or lack thereof). This is true if you are practicing a bad posture (leading to pathological changes) or if you are practicing a good posture (alleviating the pain caused by those pathologies).

In addition to computer work, the use of smart phones is producing an increase in the number of people with forward head postures described as the 'iHunch', iPosture' or 'Text Neck'. When chronic it can result in the 'dowager's hump' in which the upper back is frozen into a forward curve. Although this condition was usually associated with the elderly, it is increasingly being seen in teenagers. The forward slouched position has also been found to have effects on productivity, mood and memory (http://www.nytimes.com/2015/12/13/opinion/sunday/your-iphone-is-ruining-your-posture-and-your-mood.html?_r=0).

Question 3: Why the burning between the shoulders?

Answer:



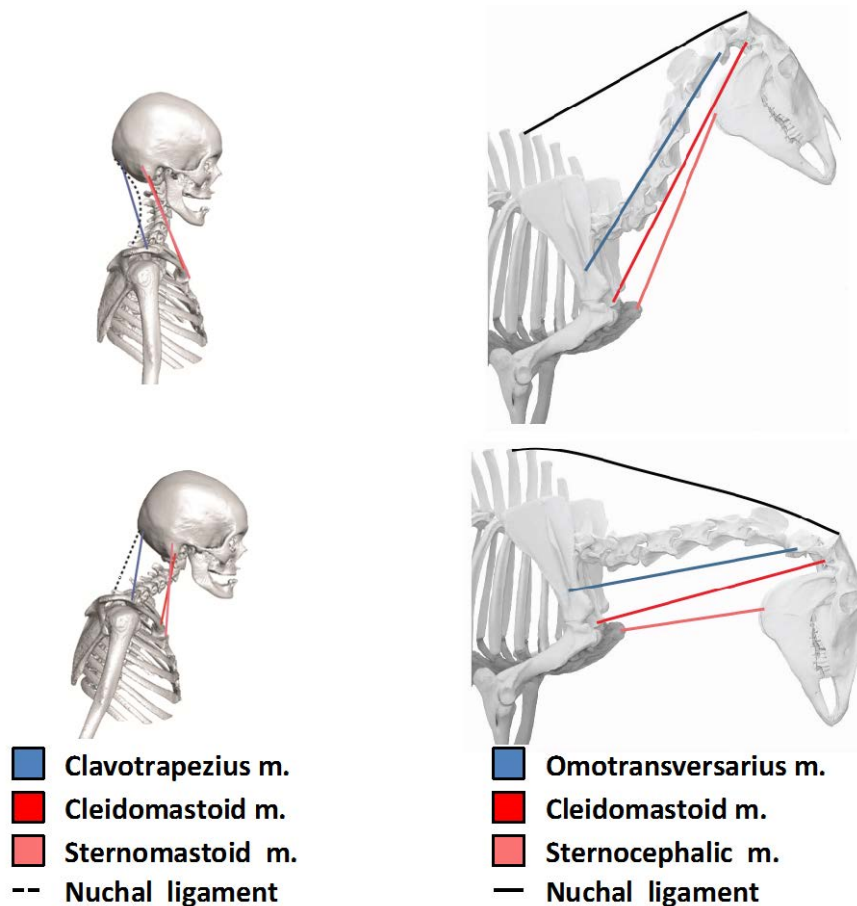
Results/Outcome

Because the patient took an active interest in her posture, she consistently went to therapy and practiced the recommended exercises for her weak or too-tight neck muscles. She also became more aware of her posture, especially when she was at work. She found some sources online and began working on finding a good posture that allowed her to work without pain- this required some experimentation. She discovered through her own research that her forward head posture was just the beginning of her postural problems- the rest of her spine was also compromised from her regular sitting posture- and is now working to address these issues.

Comparative Considerations: The Issue of Straightening the Curvature of the Neck

For every inch of Forward Head Posture, it can increase the weight of the head on the spine by an additional 10 pounds.”

- Kapandji, I.A. 1974. The Physiology of the Joints. Volume 3. The Trunk and the Vertebral Column. 2nd Ed. Churchill Livingstone, Edinburgh. Pp.251.



The forward head posture and the accompanying straightening of the neck is linked to pain and headaches in humans (Watson D.H. & Trott P.H.. 1993. Cervical Headache: An Investigation of Natural Head Posture and Upper Cervical Flexor Muscle Performance. Cephalalgia 13:272-284; Nagasawa A. et al. 2005. Roentgenographic Findings of the Cervical Spine in Tension-Type Headache. Headache: The Journal of Head and Face Pain 33:90-95; Fernandez de las Penas C., et al. 2006. Myofascial Trigger Points, Neck Mobility and Forward Head Posture in Unilateral Migraine. Cephalalgia 26:1061-1070). Like the human in good posture, the horse in high collection also has an s-shaped curvature of the neck. Also as in the human, this curvature straightens out as the neck is lowered. Whereas the skull balances atop a curved neck in humans with no help from the “nuchal ligament” the well developed nuchal ligament of the horse balances the head to keep it atop a curved neck and is under increased tension when the head is lowered.

Clinical Applications: What a Clinician Can Do

- Educate clients or patients that bodies respond to pathological force and adapt to it, but can also recover when these pathological forces are relieved.
- Educate clients that changes in the skeletomuscular system take time and consistency.
- Encourage clients or patients to evaluate their own behaviors that may be causing pathomechanical forces, and to come up with potential ways to change these behaviors.
- Work to change the common patient/client expectation that drugs and surgery alone will effectively treat degenerative joint disease.

Example of Free-body Analysis on a Simplified Biological System: Healthy and Forward Head Postures of the Human

Background information

In this case, you will compare the force regime acting on the skull of a human individual exhibiting a healthy posture of the head, neck, and shoulders to that of a human exhibiting a forward head posture. This forward head posture is extremely common and anyone who sits at a computer, microscope, or bent over a book, watches TV, or looks at a cell phone will be familiar with the ease in which his/her head and neck fall forward and shoulders hunch.

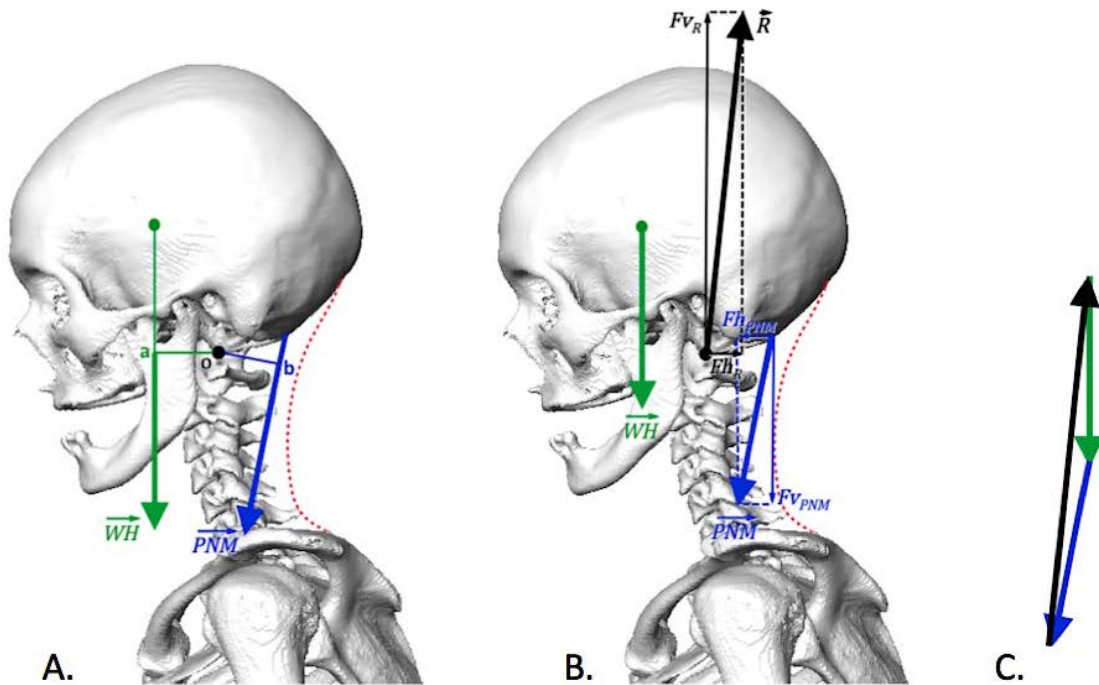
To understand why the change in posture has such an effect, we must consider the functional anatomy of this region and forces that are acting on this system (in both the healthy and pathologic conditions). We present here a highly simplified, but understandable example of a free-body analysis. We will consider the following structures:

- The skull and the related weight of head (WH)
- The cervical vertebral column
- The shoulders (clavicle, scapula, and humerus)
- The paravertebral muscles (including, but not limited to, the multifidus and semispinalis capitis muscles) – here they are simplified as one force (PNM).
- The nuchal ligament and its relationship to the trapezius muscle (mostly the upper portion that attaches to the skull, the clavotrapezius muscle) and the splenius muscle- here they are simplified as one force (NL).

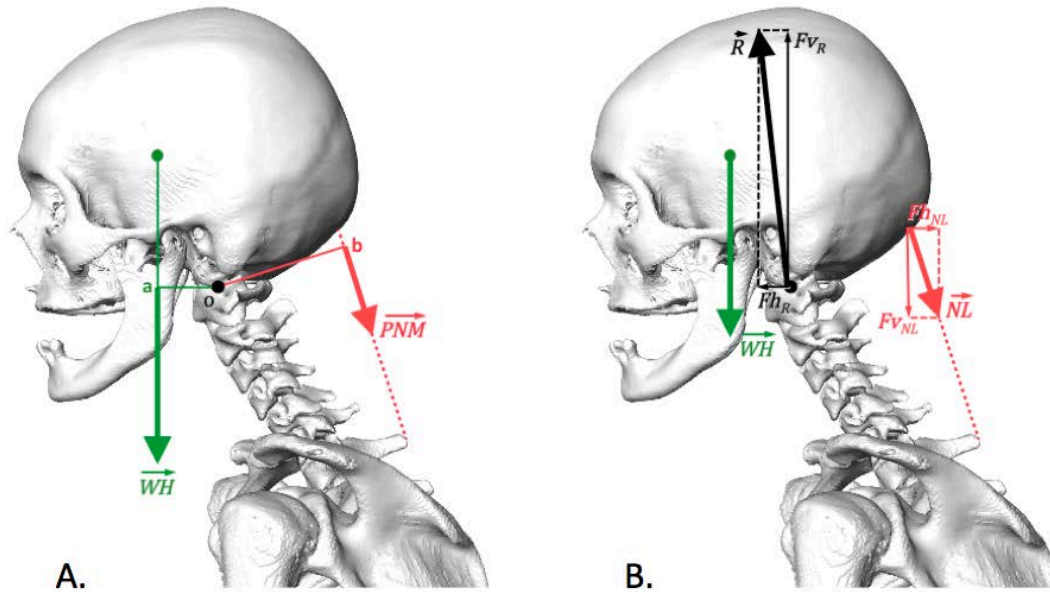
General Instructions to resolve mathematically (see Appendix A for details)

1. Identify the skeletal element of interest.
 - a. The skull
2. Identify the relevant muscles or structures that are generating forces that act on the skeletal element.
 - a. The weight of the skull: \overline{WH}
 - b. A group of deep, core, postural neck muscles (simplified here as one force): \overline{PNM}
 - c. The reaction force at the atlanto-occipital joint: \vec{R}
3. Identify the known (or given) information.
 - a. Magnitude of the weight of the head in Newtons: WH
 - b. Center of gravity for \overline{WH}
 - c. Direction of lines of force
 - i. In 2D, forces may be purely horizontal, vertical, or oblique (with horizontal and vertical components). See the following steps for how to analyze these forces.
 - ii. \overline{WH} is purely vertical (it has no horizontal component)
 - iii. \overline{PNM} is oblique (or at an angle), so it has both a vertical (F_{vPNM}) and a horizontal (F_{hPNM}) component. If the magnitude of the force is known, the magnitudes of the vertical and horizontal components can be solved for with the sin and cosin of the angle of \overline{PNM}

- d. Center of rotation at the atlanto-occipital joint: \mathbf{o}
- e. Torque arms
 - i. \overline{WH} [\mathbf{oa} ; distance from \mathbf{o} to the line of force of \overline{WH} (\mathbf{a})]
 - ii. \overline{PNM} [\mathbf{ob} ; distance from \mathbf{o} to the line of force of \overline{PNM} (\mathbf{b})]
- 4. Mathematically and graphically resolve the analysis by balancing the forces and torques.
- 5. Follow the same steps for the next posture/position.
- 6. Compare the relationship of forces and any directional changes.



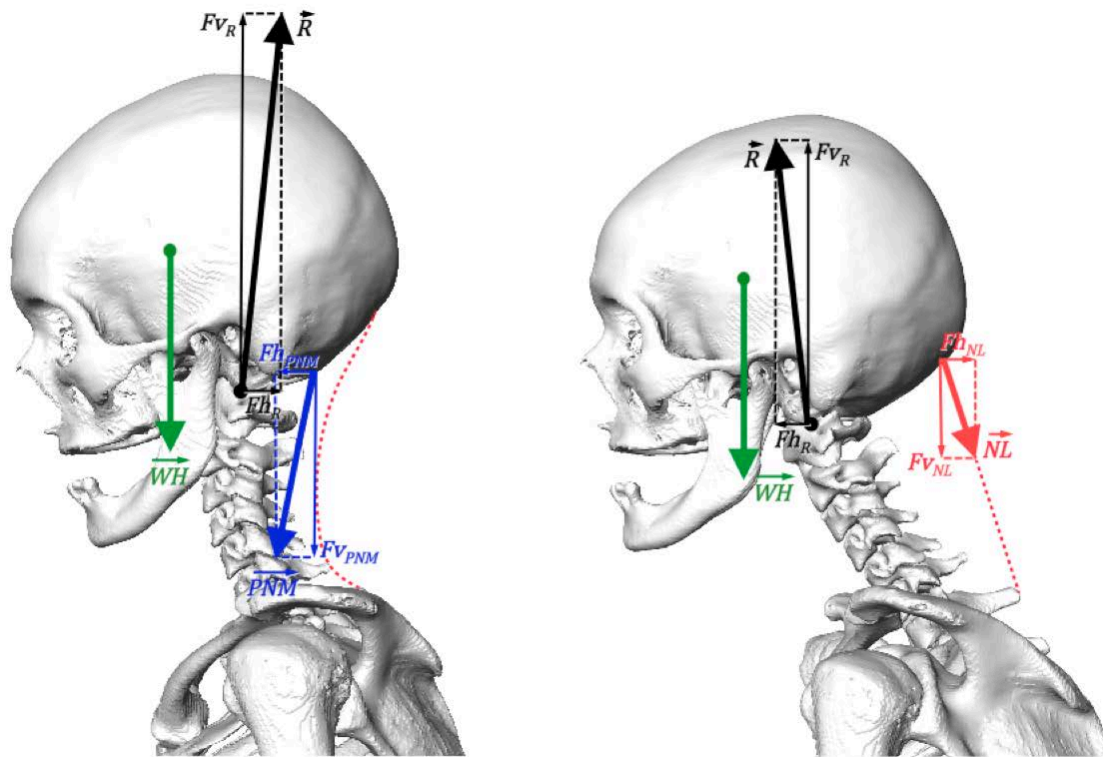
Free-body force analysis of Posture 1 shows the balanced system. **A:** Torque analysis. **B:** Force analysis. **C:** Graphic resolution of forces.



A. Free-body force analysis of Posture 2 shows the balanced system. **A:** Torque analysis. **B:** Force analysis.

Compare and contrast the two postures based on the analysis of the simplified system

Position 1 vs. Position 2



In Position 1 (the healthy posture) the deep, core paravertebral muscles of the anterior and posterior neck are acting to stabilize the vertebral column and hold the head and neck upright by many, small contractions that constantly “fix” these skeletal elements in place. In other words, these muscles are “postural” muscles. The composition of the fiber types of these postural muscles support this function: the cervical paravertebral neck muscles consist mostly of Type I slow twitch fibers, which are characterized by their high endurance abilities (Wharton et al. 1996; Boyd-Clark et al. 2001). Notice that this group of muscles has been simplified as one force (*PNM*). In this position, the actions of *PNM* counterbalance the force generated by the weight of the head, while the nuchal ligament (*NL*) is relaxed and its constituent muscles in the neck (the clavotrapezius and splenius capitis muscles) are free to move the head and shoulder.

In Position 2 (the forward head posture) some of the deep, core paravertebral muscles are weak, while others are working too much (see below) and the nuchal ligament is pulled taut and placed under tension as it works to suspend the head. It is important to note here that the nuchal ligament of the human is not the same as the nuchal ligament of the horse, cow, or dog. Instead, in the neck it is an intersection of muscle fibers of two superficial posterior neck muscles, specifically the upper and middle trapezius muscle fibers, and those of the muscle just deep to the trapezius, the splenius capitis muscle. Thus, in this

position, the trapezius and splenius capitis muscles suspend the head, acting as postural muscles, instead of movers and shakers (as in Position 1). The postural function for the clavotrapezius muscle (and likely the splenius capitis muscle) is not supported by the composition of its fiber type. Although the middle and lower portions of the trapezius muscle do have a predominance of type I slow twitch fibers, the portion of the upper trapezius that attaches to the skull (i.e., the clavotrapezius) has a predominance of type II fast twitch fibers, which are characterized by their high force, power, and speed production, but low endurance (Lindman et al., 1990; 1991).

Deeper Thinking: Think holistically and beyond the site of the issue

This simplified version of the head, neck and shoulders of the human provides an example and the ability to practice the method of free-body force analysis when analyzing the kinetics of a skeletomuscular system within a biological organism. However, biological systems are much more complex than what we have depicted here and, thus, an accurate, un-simplified free-body analysis is also much more complex. But, the principles learned in doing this simple analysis can be applied to the evaluation of a patient even when time precludes you from doing the full, mathematical analysis.

Compare again the first and second posture, but consider in a little more detail the muscles in this region.

Recommendations for those interested in learning more about musculoskeletal balance and optimization in humans:

- Muscles: Testing and Function with Posture and Pain (Kendall et al., 2005)
- Alexander Technique: <http://www.alexandertechnique.com/>
- Feldenkrais Method: <http://www.feldenkrais.com/>
- Gokhale Method: <http://gokhalemethod.com/gokhale-method>
- Myoskeletal Alignment Techniques:
<http://erikdalton.com/media/published-articles/>
 - Of particular relevance for this case study:
 - <http://erikdalton.com/media/published-articles/forward-head-posture/>
 - <http://erikdalton.com/media/published-articles/bike-body/>
 - <http://erikdalton.com/finding-weak-key-link/>

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Case 1: Degenerative lumbosacral stenosis and spondylosis in dogs

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