Joint stability in an aging perspective

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2 Introduction

As we age, our physiology undergoes a wide range of changes. When we start noticing it, the transformation has been in progress for years. Some of the changes are obvious; the hair turns gray and wrinkles start showing in the eye area. As we turn 50, the decrease in function, strength, and vitality is recognizable. Consider people, above the age of 50, who plays soccer and compare them with people in their 20's. The older group is more restricted in their movements, a lot slower, less powerful and you can add clumsy. In contrast, the younger group moves effortless, smooth, faster and they seem to bounce around like springs. The differences are noticeable, but what is the underlying physiological explanation?

In the following, we look at some of the physiological changes in the musculoskeletal system during aging. We will have our attention directed towards joint stability seen in an aging perspective. Changes in joint stability are of utmost importance for movement, coordination, and speed. Based on the theory, we will look at the transformation of different tissue types responsible for joint stability and see if physical exercise can accommodate the changes and help our patients.

2.1 Synopsis

Does joint stability change during aging, what are the consequences and how can we use physical exercise to prevent or postpone degeneration of the tissues responsible for joint stability?

3 Method

In answering the synopsis, I will use literature and review research papers covering this topic. It is important to understand the theoretical foundation upon which joint stability rests on. From a theoretical perspective, I will define joint stability and describe the function and the degeneration of the various tissues responsible for joint stability. Lastly; search for scientific documentation, which can give us training recommendations to prevent or postpone the consequences of aging in relation to joint stability. In trying to grab relevant literature and studies, I have used keywords like "joint stability and aging", "bones and aging", "muscles and aging". I found several review articles that were relevant to this assignment. The studies show that some joints are better described and examined than others. I have allowed myself to take a more generalizing position in relation to the interpretation of the research results. More precisely, the results of a study of age-related changes in mechanical proprioceptors or other properties in a knee are likely to apply to other joints in the body. It could be interesting to document the degree of age-related changes in joint stability as a function of increasing age. This has proved difficult, in part because many of the studies have been performed on human cadavers. In my search, it has not been possible to find data that covers the entire range of age.

3.1 Included

Within this scope, we will look at bones, cartilage, ligaments, muscles, tendons, and the nervous system. In our effort to help our patients, the attention is directed towards physical training.

3.2 Not included

The visual and vestibular system, even though it plays a role in this topic, falls outside the scope of this study. The same concerns nutrition and osteopathic treatment, although it probably has a positive impact on the loss of joint stability associated with aging.

4 Theory

4.1 Definition joint stability

Joint stability is a result of several subsystems working together to ensure the stability of a joint. These are the passive, active, and neural subsystems. In essence, this system provides resistance to a joint enhanced by musculoskeletal tissues (1).

4.2 Passive system

4.2.1 Bones and cartilage

The bony part of a joint is composed of substantia compacta and substantia spongiosa. The bones are lined with periost, which is highly vascularized as bones and contains many nerves. Where the bones come together the surface of the bones is covered with hyalin cartilage, which gives the joint minimum friction and a great ability to endure pressure. Bone cells are referred to as osteocytes, and continuously remodeling is taken place. To ensure healthy bones, two different cells, osteoclaster, and osteoblaster, respectively decomposes and built up new bone cells (2).

Hyalin cartilage is unique in the sense of its properties. It has the ability to yield because of its elastic resilience and in cooperation with synovial fluid containing hyaluron, to make sure that the joints move smoothly. The cartilage relies only on getting fluid and nutrition from the synovial fluid and the subchondral bone. Because of the resilience of the cartilage, it does not contain any vascular or nerve supply (2). Human chondrocytes (HC) derives from normal human articular cartilage, where they produce and maintain the extracellular matrix of cartilage, including type II collagen.

4.2.2 Ligaments

Ligaments connect bone to bone and thus stabilize, guide, and restrict joint motions (3-7). Like tendons, ligaments function to resist tensile load (6). Ligaments are composed of collagen type I (70% dry weight), elastin fibers, proteoglycans, and other minor collagens (8). The collagen fibers transfer the force within the ligaments. The multiple collagen fiber bundles are interdigitated and function together to maintain normal joint motion. Ligaments can be classified either as intra-articular or extra-articular. Like muscles and tendons, the ligaments are carrying mechanoreceptors, The central nervous system is through these receptors able to follow joint position, velocity, and distance to the barrier of the joint. With motion in a joint, the ligament will be stretched and thereby activate the mechanoreceptors within the ligament (2).

4.3 Active system

4.3.1 Muscles

Muscles, also referred to as striated muscles, has the capability to transfer chemical energy to strength and movement. The initiator of this process is the nervous system sending a nerve impulse to the muscle, which transforms chemical energy into biomechanical energy. Built in the muscles are the mechanoreceptors, which is located in both the muscles (muscle spindle) and the tendons (golgi tendon organ). Muscle spindles register the length of the muscles, while the Golgi tendon organs register muscle power. A muscle, to move a joint, expand from one bone to another through a tendon which is the continuity of the muscle. The attachments to the bones are referred to as origan and insertion. Each muscle is covered by muscle fascicles, which consist of muscle fibers and each of those fibers contains a bunch of myofibrils. They then again contain the myofilaments, which is the contractile unit in the muscle. The muscles are richly supported by a vascular system (2).

4.3.2 Tendons

Tendons are composed of different types of collagen fibers, proteoglycans, glycosaminoglycans, and elastin. They are also referred to as a part of the connective tissue. Unlike ligaments, they are more regularly arranged connective tissue. Their role in the musculoskeletal system is to transfer force from muscle to bone. The tendons are a highly oriented structure, which enables them to resist tensile stress in the direction of their fiber orientation due to collagen and to resist compressive forces due to proteoglycans.

The development of the tendon increases from birth through maturity and then decreases towards old age.

During development tendons are supported with a high level of vascularization. Tendons receive vascular supply from junctions between muscle and tendon (musculotendinous) and between bone and tendon (osseotendinous). They also receive vascular supply from various surrounding tissues such as paratenon, mesotenon, and vincula.

Areas of friction, torsion, and compression, and junctional zones can compromise the tendon vascularity.

There are differences in how much blood tendons receive. The supply can differ from one tendon to another; for example, the supraspinatus tendon and the achilles tendon have reduced tendon vascularity (9). Within a specific tendon, we see differences in blood supply. In the supraspinatus tendon, there is an area referred to as the Codman's critical zone which is hypovascular. It is caused by pressure from the humeral head, and when the pressure is removed, the vascularity increases (10). Similar to the achilles; 2-7 cm from the insertion of the tendon to the heal bone, there is an area of decreased vascularity (11).

4.4 Neural system

The nervous system consists of two parts; a central system and a peripheral system.

4.4.1 Central nervous system

The central system is the brain and the spinal cord. The brain is divided into different areas with different tasks. The areas responsible for motion are the cerebral cortex, basal nuclei, thalamus, primary somatomotor cortex, somatosensory cortex, and cerebellum. Of special interest in this study is the primary somatosensory cortex, which receives the information from the mechanoreceptors. The spinal cord, also referred to as medulla spinalis, receives sensory information from the muscles and joints through thick afferent fibers entering the dorsal horns and travels up to medulla oblongata, crosses to the opposite side, synapses with thalamus, and ends at the primary somatosensory cortex. Voluntary movements travel from the somatomotor cortex, 80 percent crosses at the medulla oblongata and travels down to the segment involved, and reaches the ventral horns. The uncrossed nerve fibers cross at the same segment and unite with the rest of the nerve fibers (12).

4.4.2 Peripheral sensory system

The peripheral system controlling our joints are similar to the central nervous system, divided into a sensory- and a motor part. The sensory part is again divided into other components; the mechanoreceptors, embedded in muscles, joints, cutaneous stretch receptors, and the sensory feedback

from the eyes and ears (2). In this study, we focus on the mechanoreceptors and their integration with the central nervous system.

Proprioception plays a critical role in movement control by providing inputs to internal models that couple sensory signals and motor commands (13). Feedback from joint receptors (ligaments) appears to provide information restricted to an extreme joint position (14) and is therefore unlikely to play a large role in postural control. There is a general agreement that muscle spindle receptors provide the primary source of proprioceptive information for postural control.

Furthermore, proprioceptive inputs trigger the rapid, automatic, and coordinated postural responses to the unexpected movement of a support surface. The large afferent fibers (Ia afferents) are critical for the timing of automatic postural responses to ensure coordinated control of the body center of mass and balance after unexpected disturbances of the support surface (15). In contrast, the timing of automatic postural responses was unaffected by loss of vestibular information after bilateral labyrinthectomy, even when vision was absent (16).

The muscles are enervated in units and referred to as the motor unit (MU). It is the link between the nervous system and muscle, which enhances the firing and activation of the muscles. Type II muscle fibers are organized in larger motor units and therefore able to generate much more power than type 1 muscle fibers, which on the other hand have more endurance capacity. As humans, we have approximately half type I and half type II with some individual differences (2).

5 Review of studies in respect to joint stability and aging

5.1 Bone and cartilage

Cellular aging has been shown to increase in human chondrocytes from the age of 1 to 87 years, with a concomitant decrease in telomere (small extensions at the end of all chromosomes) erosion and reduced mitochondria (17). It has been linked to an age-related decrease in the chondrocyte's responsiveness to anabolic growth factors such as IGF (18) and synthesis of smaller matrix molecules (e.g. aggrecan and link protein), with oxidative damage causing mitochondrial degeneration

Age-related changes in the subchondral bone, which often undergoes significant remodeling (19), would probably affect the oxygen tension in the joint, as would increasing calcification of the cartilage itself, which has also been associated with advancing age.

Mutations in the mitochondrial DNA of chondrocytes may also contribute to the age-related changes in articular cartilage (20). They can affect several pathways, including defective chondrocyte biosynthesis and increased apoptosis. It can lead to reduced anabolic activity and synthesis of proinflammatory cytokines and degradative enzymes and an impaired response to the mechanical and inflammatory insults to the cartilage. (21).

An intriguing "calcification paradox" has been suggested, where increased calcification of the vasculature occurs while the bone becomes less calcified with increasing age (22).

5.2 Ligaments aging

In many studies, the ACL and PCL seem to be the most investigated ligaments in humans. The review below reflects that.

A histologic cadaveric study showed that collagen fiber disorientation was the most prevalent change that occurred earliest (23) and ligamentous sheath inflammation increased with age. Another cadaveric study with special emphasis on the ACL, PCL, and cartilage supports it and demonstrated that only 6 % of the intra-articular ligaments were classified as normal (24).

ACL cell maturity decreases in metabolic activity, collagen production and response to platelet-rich plasma occur along with an increase in apoptosis (25).

A study demonstrated that the matrix and organization of the ligament to bone insertion changes at the insertion site with increasing age. Younger people resembling articular cartilage while the adult interface resembled fibrocartilaginous tissue. There were marked differences in collagen fiber orientation that became more pronounced with age (26).

Normal aging results in decreased numbers and altered morphology of mechanoreceptors in the ACL, which correlates positively with the deficits in proprioception associated with aging (27).

An evaluation of the structural properties of the femur-ACL-tibia complex in younger (22-35 years), middle-aged (40-50 years), and older (60-97 years) knees and found that linear stiffness, ultimate load, and energy absorbed decreased significantly with specimen age (28).

5.3 Muscles and aging

With increasing age, the force-generating capacity (strength) in the muscles is reduced (29,30). Research indicates that the observed loss of strength in older people is primarily linked to the result of muscle atrophy and alterations in the percentage of contractile tissue within the muscle (30-33) rather than deficits in muscle activation (motor unit (MU) recruitment and firing rates) (34-36).

Muscle fiber number and size in skeletal muscle cross-sectional area (CSA) decreases with age (30, 31, 33). Sarcopenia is the term used to describe a reduction in fiber size, fiber number, or a combination of the two.

Microscopic evaluation of cross-sections from whole human vastus lateralis muscles suggests a reduction in the total number of fibers within a muscle is the primary source of sarcopenia, although a small reduction in fiber size may occur (31). Researchers have also demonstrated that the muscles of older people (65–83 years of age) contain less contractile tissue and more non-contractile tissue when compared with the skeletal muscle of younger people (26–44 years of age) (33). Noncontractile tissue is composed of fat and connective tissue. A greater percentage of noncontractile tissue results in a decreased force production capacity.

In reviews of aging and muscle morphology, some authors have concluded that the size of type I (slow) fibers does not change substantially with age, but that type II (fast) fibers undergo selective atrophy (29, 37). Although the consensus is that only type II fibers are greatly reduced in size with aging (in people without impairments), the number of type I and type II fibers appears to decline similarly with age (29).

5.4 Tendon aging

The number of people experiencing tendon injuries correlates with patient age, but investigation and research of the cellular changes associated with age are less clear.

To keep the tendons healthy, there is a need for normal vascular supply and mechanotransduction to maintain tendon development, homeostasis, healing, and degeneration (38, 39).

A study has demonstrated that neovascularity was seen in patients with Achilles tendinopathy in the form of hypervascularity of the tendon with unevenly distributed thick-walled vessels as compared with healthy tendons (40). Another study of patients with achilles tendinopathy showed that 97,3 % of the tendons has evidence of neovascularization and 55,6 % of the tendons had neovascularization at the location of the tendon thickening (41).

There is some evidence that blood supply to tendons decreases with aging. A in vivo study looking at the vascularity of the rotator cuff tears with ultrasound showed that there was a significant decrease in blood flow in the intra tendinous region in elderly subjects compared with younger subjects but no differences in the bursal blood flow (42). Another study confirms it by finding a significant decrease in blood flow in the supraspinatus tendon in patients older than age 40 compared with younger patients after exercise (43).

5.5 Neural system aging

5.5.1 Sensory

During a fall, individuals prepare for the impact based on sensory information, which would be mainly of proprioceptive origin at the fall onset (44). Therefore, alterations within the proprioceptive signal likely increase the risk of falls and impede the ability to reduce fall-related injury.

The diameter of the spindles decreases with age (45) and there is an age-related decrease in the total number of intrafusal muscle fibers and chain fibers in postmortem human muscle spindles (46).

Looking at the innervation of muscle spindles, an early study (47) performed on human cadavers (aged between 16 and 82 years) reported that in the anterior tibial nerve of the foot the total number of nerve

fibers drastically decreased with age, with a significant decrease in the proportion of large fibers in the older subjects. As Ia afferents are the largest peripheral axons, this suggests an age-related decrease in the amount of muscle spindle afferents, especially the Ia afferents. The dynamic sensitivity of the muscle spindle decreases with aging (48), which leads to a lower discharge frequency in response to muscle stretch. The dampening of muscle spindle sensitivity seen with aging may be accounted for by the morphological changes.

5.5.2 Spinal synaptic integration

The number of small neurons in the intermediate zone of the ventral horn decreased with aging. As those small neurons are thought to be mostly interneurons (49), these results suggest a decreased complexity in the spinal network that may alter the integration of the afferent signal.

5.5.3 Supraspinal integration

Although little is known about the role of aging in the integration of the proprioceptive signal at the supraspinal level, there is evidence that older adults experiencing mobility impairment are more likely to have underlying alterations in the structure and function of the brain (50).

Efficient integration of different sensory inputs in the brain might be compromised because of age-related declines in white (51) and gray (52) matter integrity.

The decrease in brain gray matter thickness in pre-and postcentral gyrus areas (53), which are related to the sensorimotor regions of the brain, may also result in poorer proprioceptive integration. Age-related decline in the brain stem structure partly accounts for the decline in postural control (54).

Studies have shown greater activity in the supplementary motor area, motor area, premotor cortex, and putamen of older adults during motor imagery of upright standing in various conditions compared with young adults (55). This engagement of additional cortical areas most likely reflects a compensatory mechanism for the age-related sensorimotor decline (56, 57). Changes in brain neurochemistry that occur with aging can also induce motor deficits (58). Furthermore, impairments in perceptual processing of sensory signals contribute to prolonging muscle response delays during perturbed upright standing in older adults (59).

One potential consequence of these structural and functional changes is an increase in neural noise within the sensorimotor system that may impair the neural signal (60). The neural noise hypothesis rests on the assumption that the effective signal-to-noise ratio decreases (61) because of increased spontaneous/baseline neural spiking activity (62), which disrupts the fidelity of neural signals. A lower signal-to-noise ratio should require greater processing that may partly explain the greater brain activation observed in older adults.

5.5.4 Motor units

Aging is accompanied by a loss of motor units (63, 64), changes to the morphology and properties of existing motor units (65). There is an indication that MU firing rates decrease with age (66). Other studies suggest that MU firing rates do not decrease (67,68), but may become more variable (68). The variability in the MU firing rates of older people might be due to preferential denervation of type II fibers and subsequent reinnervation through collateral sprouting by neighboring MUs normally associated with type I fibers (29). Increased variability in MU firing rates may lead to deficiencies in motor control and force production.

Bones and cartilage		Decrease in telomere (small extensions at the end of all chromosomes), erosion and reduced mitochondria	Increasing calcification of the cartilage	Mutations in the mitochondrial DNA of chondrocytes may also contribute to the age-related changes in articular cartilage	"Calcification paradox" - increased calcification of the vasculature occurs while the bone becomes less calcified	
Ligaments		Collagen fiber disorientation and ligamentous sheath inflammation increases. Linear stiffness, ultimate load, and energy absorbed decreases significantly	Matrix and organization of the ligament to bone insertion changes at the insertion site	ACL cell maturity decreases in metabolic activity, collagen production and response to platelet-rich plasma occur along with an increase in apoptosis	Decreased numbers and altered morphology of mechanoreceptors in the ACL	In older (60-97 years) knees linear stiffness, ultimate load, and energy absorbed decreased significantly with specimen age
Muscles		Muscle atrophy and alterations in the percentage of contractile tissue	Muscle fiber number and size in skeletal muscle cross-sectional area (CSA) decreases (sarcopenia)	Reduction in the total number of fibers within a muscle is the primary source of sarcopenia	Only type II fibers are greatly reduced in size – both type I and II decreases similar in numbers	
Tendons		The amount of people experiencing tendon injuries correlate with patient age	Blood supply to tendons decreases with aging			
Neural	Sensory	Decrease in intrafusal muscle fibers and chain fibers in muscle spindles	Decrease in the amount of muscle spindle afferents, especially the Ia afferents	The dynamic sensitivity of muscle spindles decreases with age		
	Spinal synaptic integration	The number of small neurons (interneurons) in the intermediate zone of the ventral horn decreases with aging				
	Supraspinal integration	Declines in white and gray matter integrity with aging	Increased activity in the supplementary motor area, motor area, premotor cortex, and putamen of older adults during upright standing	Changes in brain neurochemistry can also induce motor deficits	Impairments in perceptual processing of sensory signals contribute to prolong muscle response delays during perturbed upright standing in older adults	Signal-to-noise ratio decreases, which disrupts the fidelity of neural signals. A lower signal-to-noise ratio should require greater processing (brain activation)
	Motor units (MU)	Loss of motor units	Changes to the morphology and properties of existing motor units	Increased variability in MU firing rates may lead to deficiencies in motor control and force production		

Table 4.1 Tissue types responsible for joint stability from the perspective of aging in relation to changes in quality and properties

6 Training

How do older people respond to training and what type of training is the most efficient to slow down the age-related degeneration of the systems responsible for joint stability?

Training of muscle is typically divided into 2 major categories: endurance and strength training. Endurance training refers to exercise directed at improving stamina (the duration that a person can maintain strenuous activity) and aerobic capacity ($\dot{V}$ o2max). Strength training refers to exercise directed at improving the maximum force-generating capacity of muscle. Appropriate exercise can alter, slow, or even partially reverse some of the age-related physiological changes that occur in skeletal muscle, including sarcopenia, reduced lean muscle mass, decreased force production (29, 69, 70, 71,72–74). Progressive resistance training (PRT) programs or high-intensity training programs – both examples of strength training are used in several studies. High-intensity resistance programs usually consist of 2 to 6 sets of 8 repetitions at approximately 80% of a person's one-repetition maximum (1-RM) (75, 74, 76) A common misconception is that older people need to "take it easy" when performing exercise. Researchers suggest that older people who are healthy respond to strength and endurance training in a similar fashion to younger people (29, 75, 77). Thus, to get optimal results, health professionals need to train their older patients at intensities that are optimally suited to induce the desired training effects.

6.1 Effect on Oxidative Capacity and Muscle Capillarization

According to Coggan and colleagues (69) muscle capillarization (muscle fiber to capillary ratio) increased in older individuals (60–70 years of age) who engaged in an endurance training program that consisted of walking or jogging. The capillary densities (capillaries per square millimeter) of these participants increased by 20%, whereas the number of capillaries per muscle fiber increased by 25% (69). These findings imply that new capillaries were generated in the muscle. Other studies of the peripheral effects of endurance training on 65-year-old men demonstrated that the oxidative capacity of the older men's muscles increased by 125% (78). Also, Frontera and colleagues (74) found that a high-intensity strength training program increased participants' capillary per muscle fiber ratio by 15%. These results indicate that training can have a profound effect on the oxidative capacity of an elderly person's skeletal muscle.

6.2 Effects on Muscle Fiber Characteristics

Both endurance and strength training can limit the extent of sarcopenia in elderly people (79, 69,72,73). Older people (aged 60–97 years) who perform regular resistance training have been shown to have increases in force production and muscle fiber CSA (72,73,80). Studies have reported force gains (48%-174%) with associated moderate gains in CSA (15%-17%) with high-intensity training programs (81,76) In these studies, researchers demonstrated that high-intensity training can reduce the decrease in muscle force and CSA in older people.

6.3 Effects on Motor Unit Characteristics

Several researchers (72,73, 81, 76) have demonstrated that, although gains in both CSA and force production occur with training, the observed gains in force are generally much greater than the gains

observed in muscle CSA. Increases in force production is a result of increased skeletal muscle CSA (hypertrophy), but also a result of training-related neural adaptations – recruitment and firing rates of MUs (31,72). Neural adaptations are the primary source of force production gains in the first 8 weeks of training, whereas increases in muscle CSA are the primary source of the force production gains after 8 weeks (75).

6.4 Other Effects Related to Training and Skeletal Muscle

The skeletal muscle of older people is damaged more easily with the loading that occurs during training when compared with the skeletal muscle of younger people (74, 82–85). Older people who regularly train may have higher dietary protein requirements than younger people who perform a similar exercise (82). To gain more muscle mass, a dietary protein supplement, may be useful for older people who are doing a strength training exercise. Osteoporosis is a common health disorder in older people, and several studies have demonstrated a relationship between osteoporosis and sarcopenia (86-88). Both running and strength training help to prevent or slow osteoporosis due to the relationship between sarcopenia and BMD (86-91). Repetitive loading of bones during muscle contraction that occurs with strength training can maintain or increase BMD (86-88).

Vascular	Muscle fiber	Motor units (MU)	Bone mass density (BMD)
Muscle capillarization (muscle fiber to capillary ratio) increases	Both endurance and strength training can limit the extent of sarcopenia in elderly people	Neural adaptations are the primary source of force production gains in the first 8 weeks of training, whereas increases in muscle CSA are the primary source of the force production gains after 8 weeks	
Oxidative capacity increases in both endurance and high-intensity strength training	Regular resistance training increases force production and muscle fiber CSA		The repetitive loading of bones during muscle contraction that occurs with strength training can maintain or increase BMD

Table 5.1 Exercise effect on the vascular system, muscle fibers, motor units, and bone mass density in the elderly population exposed to aerobic exercise or strength training.

7 Conclusion

The musculoskeletal system is a dynamic environment consisting of a variety of tissue types made up of several different cellular components within complex arrangements of matrices. The interaction of each tissue type is essential for optimal functioning of the skeleton as a whole, and as such, each region and tissue type has the potential to significantly affect the others. As we age, the alterations in normal biological responses lead to impaired tissue function, manifest in features we recognize as aging.

We can conclude, to answer the synopsis, that joint stability alters during aging. The review has demonstrated that aging is accompanied by altered inputs from peripheral, spinal, and supraspinal centers. Ultimately, motor performance is impaired and its variability is greater with advanced age. We have seen muscle fibers exposed to sarcopenia, where especially the decrease in total numbers of muscle fibers is an important factor. The vascularity in the tendon decreases as well, which seems to play a role in the degeneration of the tendon.

On the positive side and backed up by research, we can reduce and postpone the negative effect of age-related degeneration of the joints and the surrounding tissue types responsible for joint stability. Training, both aerobic and strength training, has proven to be effective by increasing the capillarization in the region exposed to training. Furthermore; loss of muscle mass (CSA) is reduced and optimizing of the neural system are important factors in preventing alteration of joint stability. To achieve the positive outcome of training and get the adaptation in the musculoskeletal system, the intensity is crucial and needs to be high.

As the text shows, the properties of the tissues change and generally become more fibrous. Muscle tissue is transformed into connective tissue and cartilage and ligaments become more fibrotic. Overall, it reduces the mobility of the joints. Inability to bring the joints into a biomechanically optimal position due to reduced mobility will reduce the stabilizing ability of the joints. It may be interesting to examine how mobility training will affect our joint stability.

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