Musculoskeletal Weeks 4-6 Review

Lecture 26: The Healthy Joint; Dr. Clare Yellowley
Qs

1. Do all joints have a fluid filled joint space?

No, synovial joints are the only joints that have a fluid filled cavity.

2. What is the purpose of synovial fluid?

The purpose of synovial fluid is to provide nutrients to the avascular cartilage as well as provide lubrication for the articular surfaces so they can slide smoothly over each other.

3. Does the synovial membrane cover intra-articular structures such as ligaments and menisci?

No, the synovial membrane does not cover intra-articular structures such as ligaments and menisci.

4. Can a torn meniscus heal?

A torn meniscus can heal, the outer edge has a blood supply because it is attached to the capsule.

5. Does the meniscus heal better than articular cartilage?

Yes, the meniscus has blood supply from the capsule that supplies the outer 1/3 of the meniscus making it a better candidate for healing than avascular cartilage.

6. Is movement at a joint dictated by the contours of the articular surfaces only?

No, movement of a joint is dictated by the attachment of ligaments, muscles, surrounding bones/structures as well as the contours of the articular surfaces.

Lecture 27: Joint Response to Injury and Pathology; Dr. Natalia Vapniarsky

Summary of the effects of injury on joint function:

Joint part	Subchondral bone	Cartilage	Synovium
Damage	Edema/Contusion	Disrupt superficial collagen alignment	Hyperemia and edema -> exudate
Mechanism	+/- sclerosis +/- resorption/sequestration	Decreased proteoglycans leading to softening Chondrocyte death Eburnation	Joint swelling (effusion/edema) Synovial fluid viscosity
Result	Reduced ability to bear and transmit forces	Reduced ability to transmit forces	Decreased fluid viscosity leading to reduced lubrication

*Damage to the subchondral bone and synovium result in pain due to vascular innervation

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Lecture 28: Joint Fluid Analysis; Dr. Bill Vernau

Characteristic	Normal	Hemarthrosis	Degenerative	Inflammatory
Appearance	Clear to Straw colored	Red, cloudy or xanthochromic	Clear	Clear to Cloudy
Protein	<2.5 g/dl	Increased	Variable	Normal to increased
Viscosity	High	Decreased	Normal to decreased	Normal to decreased
Mucin clot	Good	Normal to poor	Normal to poor	Fair to poor
Cell count (/µl)	<1,500 <3,000 (stifle)	Increased RBCs	Normal to <10,000	Normal to >100,000
Neutrophils	<5% (often 0%)	Relative to blood	< 10%	>10% to 100%
Mononuclear cells	>95%	Relative to blood	> 90%, often with increased cytoplasmic volume and foaminess	0% to <90%

Lecture 29: Gait Analysis SA; Dr. Barbro Filliquist

• Movement should be effortless!
• Gait analysis is performed at a walk and a trot
• Look for asymmetric movement and assess which leg is in contact with the ground less time – affected limb
• Can be hard to assess when bilateral lameness is present
• Subjective assessment used mainly in clinics but kinetic +/- kinematic assessment can help significantly

Qs
2 symmetric gaits of the dog and cat: Walk, Trot
3 Categories of abnormalities that can lead to lameness: Musculoskeletal, Neurological, Mechanical
Phases of a gait: Swing, Stance

Lecture 38

Neuromuscular Junction Steps

1. Terminal depolarization due to a nerve impulse causing L-type calcium channels to release calcium. Vesicles fuse with the membrane and Ach is released into the synaptic cleft
2. Ach binds to the Ach receptor which opens ion channels allowing Na+ influx and local membrane depolarization

3. Sodium channels open and the plasma membrane depolarizes
4. T-tubule Ca2+ channels open
5. SR Ca2+ release

Muscle contraction

1. AP reaches T-tubular system causing depolarization
2. L-type voltage-sensitive Ca2+ channels change conformation which induces the ryanodine-sensitive Ca2+ channels to open and release the calcium stored in the terminal cisterna.
3. Ryanodine-sensitive Ca2+ channels open and release Ca2+ from the sarcoplasmic reticulum into the sarcomere
4. The release of Ca2+ binds to troponin C and integrates contraction by regulating myosin-actin interaction
5. Ca2+ dependent ATPase mediates the return of Ca2+ to the sarcoplasmic reticulum (Ca2+ binds to calsequestrin)