Preconference Form

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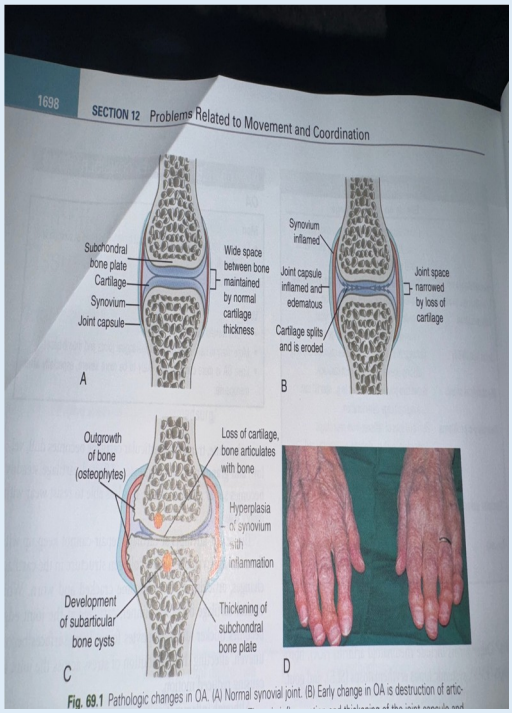
Medical Diagnosis/Disease: Osteoarthritis

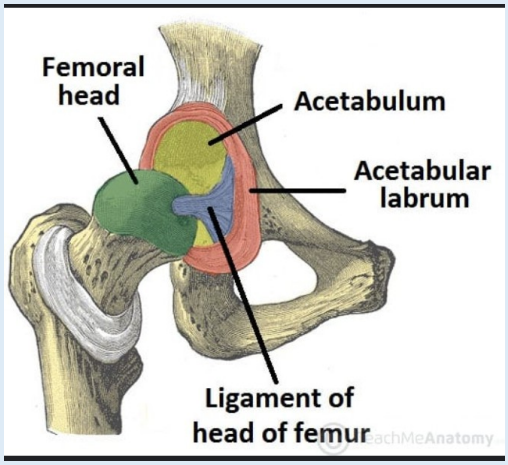
NCLEX IV *(8)*: Physiological Integrity/Physiological Adaptation **NCLEX IV**

***(7)*: Reduction of Risk**

Anatomy and Physiology

Normal Structures:





- The hip joint is a ball and

Pathophysiology of Disease:

- Osteoarthritis is a slowly progressive noninflammatory disorder of the diarth\rodial (*synovial*) joints.
- This process involves the gradual loss of articular cartilage with formation of bony outgrowths (spurs or osteophytes) at the joint margins.
- Cartilage destruction likely begins between the ages of 20 and 30.
- Various genetic traits as well as known/unknown events can cause OA so there is no specific cause.
- The development of OR includes genetic, metabolic, and

Anticipated Diagnostics

Labs:

- No laboratory tests can be used to diagnose OA

Additional Diagnostics:

- X-ray to determine the degree of OA.
- Synovial fluid analysis to distinguish OA from other types of inflammatory

socket joint that is the point of articulation between the head of the femur and the acetabulum of the pelvis.

- The hip joint connects the lower extremities with the axial skeleton.
- Due to the depth of the acetabulum, it can encompass almost the entire head of the femur. There is additional fibrocartilaginous collar surrounding the acetabulum, the acetabular labrum, which provides the following functions:
  - Load transmission
  - Negative pressure maintenance (i.e., the "vacuum seal") to enhance hip joint stability
  - Regulation of synovial fluid hydrodynamic properties
- The capsular ligaments include

local factors interacting to cause cartilage deterioration from damage at the level of the chondrocytes.

- Generally smooth translucent articular cartilage becomes dull, yellow, and granular as OA progresses.
- The body's attempts at cartilage repair cannot keep up with the destruction of OA. As the collagen structure in the cartilage changes cartilage becomes thinner, cartilage at the joint edges becomes thicker and osteophytes form. Joint surfaces become uneven, affecting the distribution of stress across the joint causing reduced motion.
- Inflammation is not typical of OA, however secondary synovitis may occur when phagocytes try to rid the joining of small pieces of cartilage torn from the joint surface.

arthritis.

the iliofemoral ligament (T ligament of Bigelow), and the pubofemoral and ischiofemoral ligaments.

- The iliofemoral ligament is the strongest ligament in the body and attaches the anterior inferior iliac spine (AIIS) to the intertrochanteric crest of the femur.

NCLEX II *(3)*: Health Promotion and Maintenance **NCLEX IV *(7)*: Reduction of Risk**

Contributing Risk Factors:	Signs and Symptoms:	Possible Therapeutic Procedures	Prevention of Complications
❖ Being above the age of 65 ❖ Specific trauma or mechanical event leading to breakdown of articular cartilage. ❖ Obesity	❖ Joint pain ❖ Join stiffness that occurs after periods of rest or an unchanged position (*gelling phenomenon*) ❖ Early morning stiffness is common, often resolved in 30 minutes (this distinguishes OA from inflammatory joint disorders such as RA.	Non-surgical: ❖ NSAIDS ❖ Topical treatment (Capsaicin cream) ❖ Intraarticular corticosteroid injections Surgical: ❖ Total Hip Arthroplasty (THA): ❖ Also known as a total hip replacement, provides significant relief	(What are some potential complications associated with this disease process) ❖ Limited mobility ❖ Joint pain

		of pain and improved function for patients with joint deterioration from OA, RA, and other conditions. ❖ In THA the prosthesis (implant) replaces the ball-and-socket components can be cemented in place with polymethyl methacrylate, which bonds to the bone.They may be inserted without cement (cementless) cementless THA may provide longer	

		stability by enabling growth of new bone tissue into the porous surface coating off the prosthesis. Cementless devices are better for younger, more acreage patients and patients with good one quality.	

NCLEX IV *(6)*: Pharmacological and Parenteral Therapies

Anticipated Medication Management:

- OA has no cure so management is heavily characterized by managing pain and inflammation, preventing disability , and maintaining and improving

NCLEX IV *(5)*: Basic Care and Comfort

Non-Pharmacologic Care Measures:

- Teaching a cardiac diet to reduce obesity risk providing relief for the joint pressure.
- Exploring exercises / stretches that promote hip

NCLEX III *(4)*: Psychosocial/Holistic Care Needs

What stressors might a patient with this diagnosis be experiencing?:

- Fear of losing independence
- Pain
- Management of

joint function. ❖ Drug therapy is based on the joint affected and symptoms severity ❖ NSAIDs are the first-line treatment for OA. They inhibit the production of cyclooxygenase.	mobility	symptoms

Client/Family Education **NCLEX I *(1)*: Safe and Effective Care Environment**

List 3 potential teaching topics/areas	Multidisciplinary Team Involvement
❖ Teaching the client safe ambulation, whether they may need a 1:1 assist or just need help with their ambulatory devices. ❖ Teaching the client to stay physically active (to their best ability) because staying	(Which other disciplines do you expect to share in the care of this patient) ❖ Orthopedic ❖ Surgery

stationary will make it worse ❖ Depending if this education is post-op infection prevention could be highlighted to keep the THA site from getting infected.		