

Rheumatoid Arthritis: Primarily an Autoimmune Disease

Event Type

Live Online 

ACPE Expiration Date

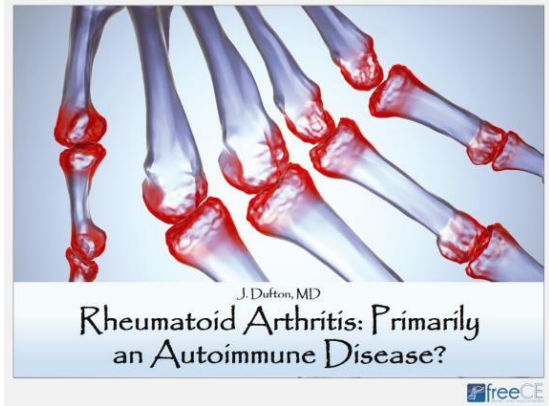
10/29/2016

Credits

1 Contact Hour

Target Audience

Nurses, Pharmacists, Pharmacy Technicians



Program Overview

Rheumatoid arthritis (RA) is an inflammatory type of arthritis characterized by severe joint pain, inflammation and loss of mobility -- particularly within the hands of middle age to elderly women. Symptoms often come and go and can affect other body parts, such as the eyes, mouth and lungs. The causes of RA is unknown, although it's categorized as an autoimmune disease because it appears that the immune system is attacking its own tissues. Genetics, hormones, environment, diet and gastrointestinal health are all related factors. An estimated 1.5 to 2 million American adults currently suffer from RA. There are natural remedies, lifestyle changes and pharmaceutical treatments that help reduce the symptoms of RA and slow its progression.

Nurse Educational Objectives

- Describe the pathophysiology, frequency and implications of rheumatoid arthritis (RA)
- Outline the non-pharmacological methods used to treat RA
- Compare and contrast the most common pharmaceuticals used to treat RA, including mechanism of action and potential side effects

Pharmacist Educational Objectives

- Describe the pathophysiology, frequency and implications of rheumatoid arthritis (RA)
- Outline the non-pharmacological methods used to treat RA
- Compare and contrast the most common pharmaceuticals used to treat RA, including mechanism of action and potential side effects

Pharmacy Technician Educational Objectives

- List signs and symptoms of a Rheumatoid Arthritis (RA)
- List pharmacological and non-pharmacological treatments for RA

Activity Type

Knowledge

Accreditation

Nurse	N-859
Pharmacist	0798-0000-13-252-L01-P
Pharmacy Technician	0798-0000-13-252-L01-T



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Faculty



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Medical Writer, Wellness Partners

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J. Dufton, MD

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Accreditation
Pharmacists: 0798-0000-13-252-L01-P
Pharmacy Technicians: 0798-0000-13-252-L01-T
Nurses: N 859

Faculty
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Wellness Partners

CE Credit(s)
1.0 contact hour(s)

Faculty Disclosure
Dr. Dufton has no actual or potential conflicts of interest in relation to this program.

Learning Objectives

- Describe the pathophysiology, frequency and implications of rheumatoid arthritis (RA)
- Outline the non-pharmacological methods used to treat RA
- Compare and contrast the most common pharmaceuticals used to treat RA, including mechanism of action and potential side effects

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OBJECTIVES:

- Describe the cause, frequency and implications of rheumatoid arthritis (RA).
- Outline the non-pharmacological methods used to treat RA.
- Compare and contrast the most common medications used to treat RA, including mechanisms of action and potential side effects.

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Arthritis Overview



- Latin meaning "inflamed joint"
- Major cause of disability for adults and even children
- About 22% of American adults have doctor-diagnosed arthritis (of some type) & prevalence is 50% at 65 years old and greater.
- Over 100 arthritis types known
- RA is **not** the most common type (OA is), but it's one of the most destructive and painful
- First recognized medical description of RA was made in 1800

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Rheumatoid Arthritis (RA)

- A chronic inflammatory disorder that typically affects the joints of the hands / feet, although widespread systemic inflammation (affecting many tissues and organs) is not uncommon
- Affects the lining of joints (synovium), causing severe swelling and pain that eventually leads to bone erosion / joint deformity
- Very different from OA -- a degenerative condition that slowly destroys cartilage, connective tissue and bone within joints
- At least **1.5 million** American adults have RA -- mostly between the ages of 30-60 -- much more common in women
- The cause is not fully understood
- Classified as an autoimmune disease without cure



RA Pathophysiology

- Involves inflammatory response of synovium, secondary to:
 - synovial cell swelling (turgescence)
 - excess synovial fluid production
 - "pannus" development in synovium
- Inflammation thickens synovium, which eventually destroying cartilage and bones /in w/in joints -- ankylosis
- Tendons / ligaments weaken & stretch -- joint becomes unstable / misaligned
- Affects other organs **15-25%** of time
- Can inflame lungs (pleura), eye (sclera), heart (pericardium), upper C-spine, and subcutaneous tissue (nodes)



Chronically Elevated Immune Response

- Abnormal interaction between lymphocytes (B-cells / T-cells)
- B-cells present antigens to T-cells -- triggers production of rheumatoid factor (RF) & antibodies to citrullinated peptides (ACPA)
- Inflammation is driven either by B-cell or T-cell products stimulating release of TNF and other cytokines (interleukins)
- Disease progression can be slowed down by blockage of **TNF-alpha** and/or depletion of B-cells
- Other immune cells that are relevant at the site of inflammation include plasma cells -- derived from B-cells



What is an Autoimmune Response?

- Within the medical model, certain cells of the immune system are thought to malfunction due to:
 - genetic factors that affect lymphocyte production / function directly
 - genes that impact connective tissue type or hormone balance
- In RA, it appears lymphocytes attack otherwise healthy synovium tissue, which lines the insides of synovial joints
- Chronic response / inflammation persists since self antigens can't be removed from the body in a "self vs. self" situation
- Often classified as **Type III** immune complex disease (when RF present), but sometimes as a Type IV cell-mediated disease when there's no RF present

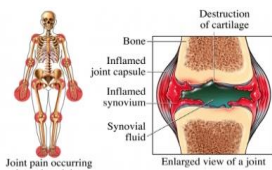


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Most Common Signs & Symptoms

- Severe joint inflammation, pain, redness and warmth
- Joint stiffness
- Reduced range of motion (ROM), loss of joint function and muscle pain
- Starts in smaller joints (bilaterally) of hands / feet, then spreads to larger joints (knees, shoulders, hips), then may involve organs
- Associated w/ fatigue, low fever, anemia & weight loss
- Periods of increased symptoms ("flares") alternate w/ periods of relative remission
- Firm lumps (rheumatoid nodes) grow beneath the skin



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Complications of RA

- Inflammation of the glands of the eyes and mouth can cause dryness and is referred to as **Sjögren's syndrome**
- Pleuritis causes chest pain, shortness of breath, and coughing
- Pericarditis causes chest pain and increases risk of heart attack
- RA can reduce RBCs (anemia) and WBCs
- Rheumatoid nodules / nodes can put pressure on tendons, ligaments, and nerves, and trigger carpal tunnel syndrome
- Longstanding RA is associated w/ **vasculitis**, which impairs blood supply to tissues and leads to tissue death (necrosis) in the hands and feet
- RA can inflame spinal joints and make them unstable

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Risk Factors

- **Gender:** RA is **2-3X** more common in women
- **Genetics:** **50%** of RA risk is believed to be genetic -- associated w/ inherited antigen HLA-DR4 and genes PTPN22 (all races) and PADI4 (only Asian descent)
- **Smoking:** most significant non-genetic risk: heavy smokers (especially men) are **2.5X** more likely to develop RA
- **Age:** most cases seen in adults between the ages of 35-55
- **Infections:** epidemiological studies have confirmed a potential association with herpes infections (Epstein-Barr virus & HHV-6)
- **Environmental factors:** unknown by mainstream medicine



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Alternative Theories of Causality

- **Bacterial infections:** "auto-antibodies" are merely antibodies generated in response to pathogenic bacteria that have been destroyed
 - Dental infections (cavitations) are implicated
- **Unacknowledged pathogens:** mycoplasma, viruses, fungi, fragments
- **Leaky gut syndrome:** intestinal damage (from allergic reactions, gluten, toxins) leads to inflammation and increased porosity -- undigested protein enters bloodstream and deposits in joints/tissues, which triggers chronic immune response
- **Weakened immunity:** immune system is not overactive or faulty, but overwhelmed and suppressed from stress/ poor diet/ over-medication
- **Vitamin D deficiency:** common in those with RA and may be causally associated -- reduced immunity, hormone imbalance

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Diagnosing RA

- **Medical history and physical exam:** to establish possible genetic links / risks & check for joint inflammation and typical signs
- **Blood tests:** elevated erythrocyte sedimentation rate (ESR) w/ RA and immunological studies look for:
 - rheumatoid factors (non-specific antibodies), but negative RF does not rule out RA -- **15-20% are seronegative**. RF is usually negative early in disease, plus it's found in 10% of healthy population and other conditions: Sjögren's syndrome, Hepatitis C and some other chronic infections
 - other tests look for the presence of anti-citrullinated protein antibodies (ACPAs) -- positive in 67% of RA cases, but rarely positive if not present, so specificity is high at 95%
- **Imaging:** X-rays eventually show loss of joint space, soft tissue swelling, and characteristic deformity; ultrasonography can depict early cartilage erosion, synovium swelling and vascular changes



Classifying & Diagnostic Criteria

- The American College of Rheumatology developed a system for classifying RA based upon X-ray appearance of joints.
 - **Stage I:** no damage seen on X-rays, but symptoms present
 - **Stage II:** some bone thinning, slight cartilage erosion, limited mobility, some muscle atrophy
 - **Stage III:** cartilage and bone erosion, some joint deformity, muscle atrophy, soft tissue nodes
 - **Stage IV:** cartilage and bone damage, osteoporosis, joint deformity, ankylosis, extensive muscle atrophy, soft tissue nodes
- Newer criteria (2010) based on a point system from 0-10, with a score of **6 or higher** indicating unequivocal RA.
- Point system based on how many joints involved, what joints involved, positive serological markers, positive ESR and CRP values, and duration of symptoms.



RA on X-Ray

Comparing normal hand (left) to RA hand (right):



< inflammation, cartilage and bone erosion in joint, general ulnar deviation, Boutonniere deformity of thumb, possible "swan neck" deformities in other fingers.



Incidence / Prevalence of RA

- About 41 per 100,000 Americans are diagnosed w/ RA each year
- Incidence increases w/ age: 8.7 per 100,000 among those aged 18-34 compared with 54 per 100,000 among those aged ≥ 85 years
- Incidence peaks between 65-74 years of age, with majority of sufferers are women between the ages of 35-55.
- From 1995 to 2007, rates increased by 2.5% yearly among women, but decreased by 0.5% among men.
- Lifetime risk: **4-5%** for U.S. women and **2-3%** for men
- In the U.S., Native Americans have highest prevalence rates (6%).
- RA most prevalent in Russia, Eastern Europe and South America and lowest in S.E. Asia and parts of Africa.



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Impact of RA

- Patients w/ RA have significantly worse functional status than those w/ OA or most other forms of arthritis
- 40% more likely to report poor health, 30% more likely to need help with personal care and twice as likely to experience activity limitation compared to people w/out arthritis
- Report more losses in function than people w/out arthritis in every activity including work, leisure, and social relations
- Reduces the lifespan of patients by anywhere from **3-12 years**
- Accounts for about 22% of all deaths due to arthritis



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Natural Approaches Overview

- **Lifestyle changes:** gentle exercise may help reduce symptoms, but weight-bearing exercise often triggers flares. Regular exercise improves muscle strength, reduces stiffness, and lowers risk of osteoporosis / disability..
- **Physical treatments:** acupuncture, physiotherapy, chiropractic, massage, cryotherapy and hydrotherapy may help with RA symptoms, but typically more effective for OA.
- **Devices:** wrist / hand splints and custom made foot orthotics or orthopedic shoes can alleviate pain from RA. Magnets may help also.
- **Acupuncture:** effective for pain and inflammation control.
- **Herbal remedies:** some herbs are good anti-inflammatories / analgesics.
- **Other natural compounds:** omega-3 fats, GLA, vitamin D, vitamin C, selenium, MSM.

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Herbal Remedies

- **Thunder god vine:**
 - Root portion used for centuries in Chinese medicine
 - Good anti-inflammatory & suppresses immune response
 - Reduced symptoms of RA in human & animal studies
 - Taken orally or applied topically
 - Oral doses range: 180-570 mg daily for up to 20 weeks
 - High rate of side effects (GI and infection risk)
- **Boswellia resin:** aka frankincense
 - anti-inflammatory and immune system effects in laboratory and animal studies
 - The most biologically active component is called AKBA.
 - Extract doses range from 250-1,000 mg daily (+ results after 1 week)
 - Safe to take long term



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More Herbal Remedies

- **Turmeric root:**
 - Contains curcumin, a strong anti-inflammatory & analgesic
 - Performs as well as ibuprofen and diclofenac in studies, but w/out side effects
 - Extract doses range from 300-600 mg 3X daily
 - Also a blood thinner
- **Devil's claw:**
 - Anti-inflammatory & analgesic
 - Studies indicate that treatment (8-12 weeks) reduces joint pain and improves function (spine, hips, and knees)
 - Extract doses range from 100-200 mg 2X daily
 - Also a blood thinner & vasodilator



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More Herbal Remedies

- **Ginger root:**
 - blocks COX-2 production and reduces inflammation much like Celebrex / Vioxx
 - At least 2,000 mg daily (divided doses) to be helpful for RA
 - Also a blood thinner
- **Willow bark:** contains salicin
 - Salicin slower for pain relief than ASA, but safer and effects last longer
 - Extract doses range from 300-500 mg 3X daily
 - Also a blood thinner
- **Capsaicin:**
 - Derived from cayenne peppers and usually applied as a cream
 - Short-term reduction in substance-P; chemical that contributes to joint pain.
 - Decreased pain after at least 3 days
 - Doesn't improve joint swelling or function



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Dietary Supplements

- **Omega-3 fats:** includes ALA, EPA and DHA
 - strong anti-inflammatory properties and aid in tissue repair
 - EPA & DHA found in fish oils; ALA in flaxseed, hemp & walnuts
 - 1,000 mg 2-4X daily for noticeable anti-inflammatory effects
- **Borage seed oil:** richest source of gamma-linolenic acid (GLA) -- other sources include black current seed and evening primrose oils.
 - GLA inhibits leukotrienes and demonstrates anti-inflammatory effects
 - dosage for RA: 1-3 grams daily for at least 24 weeks
 - may lower threshold for seizures, so epileptics need to be cautious
- **Vitamin D:** RDA between 600-800 IU -- far too low
 - a significant % of RA patients are vitamin D deficient
 - acts more like a hormone -- essential for strong / normal immunity
 - D3 safest form, even up to 40,000 IU daily -- sun exposure important

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Dietary Supplements

- **Vitamin C:** RDA between 70-120 mg -- far too low
 - megadoses of least 1,000 mg 3-5X daily
 - needed for tissue (collagen) repair and acts as mild anti-inflammatory
- **MSM:** methylsulfonylmethane
 - source of organic sulfur for chondroitin sulfate production
 - good anti-inflammatory & mild analgesic
 - 3,000 - 4,000 mg 2X per day can impact arthritic pain after a few days
- **Selenium:** RDA is 55 mcg -- more than 200 mcg may cause issues
 - strong antioxidant and needed by immune system for normal function
 - stimulates WBC production and works in tandem w/ zinc
 - people w/ RA (and cancer) often have low levels of selenium, but researchers not sure if it's a cause or an effect of the disease.

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Leaky Gut & Dietary Changes

- If leaky gut syndrome (LGS) is associated w/ autoimmune conditions such as RA, then changes in diet are important
 - **Dr. Alessio Fasano**, a researcher at the University of Maryland School of Medicine, believes that 3 main factors underpin most, if not all, autoimmune diseases:
 - 1) a foreign substance is recognized by the body
 - 2) the immune system overreacts to the foreign substance
 - 3) the intestines are unusually too permeable -- dietary factors
- With LGS, abnormally large spaces develop between the cells of the duodenal wall, which allow entry of undigested protein, infectious microorganisms and various toxins
- **Gluten** (plant protein found in grains such as wheat, barley, rye) is arguably the most common trigger for LGS
- Toxins found in **beans** and **root vegetables** may be important factors



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Drug Therapy Overview

- Goal: to minimize the pain & swelling, to prevent bone destruction & joint deformity, and to maintain day-to-day function
- Treatment options:
 - OTC NSAIDs
 - Rx NSAIDs -- COX-2 inhibitors
 - steroidal anti-inflammatories
 - disease-modifying anti-rheumatic drugs (DMARDs)
 - older immune suppressants
- Medications are not curative and some actually accelerate joint destruction w/ long-term use



OTC NSAIDs

- Most common: **ibuprofen** (Advil®, Motrin®), **naproxen** (Aleve®), and **ASA** (Bayer® Aspirin, Anacin®)
- Help relieve pain & inflammation of RA, but have high incidence of side effects (stomach irritation, ulcers, bleeding)
 - Risk of serious stomach problems: ASA > naproxen > ibuprofen
 - Most NSAIDs increase risk of heart attack, but NOT naproxen
- ASA is non-selective cyclooxygenase (COX) inhibitor --more COX-1
- Ibuprofen & naproxen non-selective COX inhibitors too -- more COX-2
- Dosages for RA: ASA 1,000 mg 3X daily; ibuprofen 400-800 mg every 6 hrs (3,200 mg max); naproxen 250-550 mg 2X daily (1,650 mg max)
- Some studies show that long-term use of NSAIDs actually accelerate cartilage erosion, connective tissue break down and joint destruction
- Capsaicin (Theragen®) & salicylate (BenGay®, Salonpas®) creams



Rx NSAIDs

- COX-2 inhibitors (such as **Celebrex®**) selectively inhibit only COX-2, which makes them much easier on the stomach
- Higher risks of MI & stroke, which prompted FDA to remove 2 from the U.S. market: Vioxx® (2004) & Bextra® (2005)
- Expensive compared to other Rx NSAIDs such as diclofenac, diflunisal, indomethacin, naproxen, etc.
- Celebrex® doses for RA are typically 100-200 mg 2X daily
- **Diclofenac** (Voltaren®, Cataflam®) is an acetic acid derivative: oral dosage for RA is between 150-200 mg daily in 2-4 divided doses
- **Diflunisal** (Dolobid®) is a carboxylic acid derivative: oral dosage for RA is between 500-1,000 mg daily in 2 divided doses
- **Ketoprofen** (Orudis®) was available OTC in the U.S., but now Rx: dosage for RA 200 mg extended release capsule once daily
- Ketoprofen based Powergel (2.5%) available for topical application



Corticosteroids

- Strong anti-inflammatories that mimic adrenal hormones (such as **cortisone**, **prednisone**, & **prednisolone**). Also suppress immunity, which is helpful for autoimmune conditions such as RA
 - Cortisone injections are more common for OA, but RA is systemic, so pills, creams and intranasal sprays more common
 - Injections are limited to about 2-4 per year, depending on the joint, effectiveness & side effects
 - For RA, low-dose corticosteroids (7.5 mg daily) are used as "bridge therapy" until slower acting DMARDs become effective
 - Larger doses (25 mg daily) are used short-term to treat RA flares
- Side effects include: edema, weight gain, increased BP, & eye pressure (glaucoma), mood swings, fatigue, slow healing, increased risk of infections, thin skin, easy bruising, and osteoporosis



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DMARDs

- DMARDs (disease-modifying anti-rheumatic drugs) work slowly to modify course of RA -- promote remission & prevent progressive joint destruction.
- Patient likely to be prescribed a DMARD early in the course of their disease -- about a dozen drugs to choose from.
 - Most common DMARD is **methotrexate** (Rheumatrex, Trexall, Folex).
 - Adults w/ RA usually start by taking 7.5 - 10 mg (3-4 pills) once per week.
 - The dose may be increased up to 25 mg a week over time if needed -- time to effect can be w/in 3 weeks, but more often closer to 12 weeks.
 - Methotrexate also available as a liquid or injection.
 - Methotrexate increases liver enzymes in about 15% of patients and it's teratogenic, so not appropriate for women planning a pregnancy.
- DMARDs often used in conjunction w/ NSAIDs and/or steroids.
- If conventional DMARDs not effective after 12 weeks, then other "biological agents" considered.



DMARD's: Biologics

- Biologics (biologic response modifiers) are the newest category of meds used for RA and are a subset of DMARDs -- must be injected.
- Biologics block specific steps in the inflammation process, so they don't wipe out the entire immune response as some other RA drugs do.
 - TNF-alpha inhibitors** block a cytokine called tumor necrosis factor-alpha (an inflammatory substance) and include Cimzia, Enbrel, Humira, Remicade and Simponi.
 - Kineret blocks a cytokine called interleukin-1 (IL-1); Actemra blocks interleukin-6 (IL-6); Orencia blocks the activation of T-cells; Rituxan blocks B-cell activation.
- Biologics have shown great effectiveness in controlling inflammation and preventing the joint erosion & deformity that characterize RA, but they are expensive and induce many side effects including severe nausea, diarrhea, hair loss and greater risk of serious infections (TB).



DMARDs: JAK & SYK Inhibitors

- A newer subset of DMARDs is **JAK inhibitors**: they block Janus kinase pathways, which are involved in the body's immune response and can function abnormally w/ RA.
- Unlike biologics, JAK inhibitors can be taken orally.
 - The newest (2012) JAK drug is tofacitinib (Xeljanz) and is being compared favorably to biologics.
 - Another new drug, SB1578, inhibits JAK-2 pathways and can normalize cytokine levels, at least in animal studies.
- SYK inhibitors** block the spleen tyrosine kinase pathway.
 - The SYK inhibitor fostamatinib works particularly well w/ methotrexate for reducing RA symptoms.



Older Immune Suppressants

- Older immune suppressants tame or block immunity in a global fashion and are actually the original DMARDs.
- However, global immune suppressants significantly increase risk of serious infections & impair healing.
- Examples include:
 - azathioprine (Imuran, Azasan)
 - cyclosporine (Neoral, Sandimmune)
- Corticosteroids also globally suppress.
- Rarely prescribed for RA these days DMARDs work better side effects.



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Fighting RA w/ Antibodies

- An active area of RA research focuses on antibodies -- proteins made by the immune system that identify disease-causing agents.
- Several antibodies are already approved by the FDA for use in RA treatment, but new ones are being studied for effectiveness.
 - Denosumab (Prolia) targets a molecule called RANKL, a cytokine that plays an inflammatory role.
 - Gevokizumab inhibits IL-18, a cytokine that triggers inflammation.
 - Ofatumumab (Arzerra), already approved for treating leukemia, is an anti-CD20 monoclonal antibody now being studied for RA use.
 - Other monoclonal antibodies include: Simulect, Zenapax and Orthoclone.
- Antibody treatments are typically given as injections or infusions.



Prosorba Column

- Prosorba column therapy involves pumping blood drawn from a vein into an apheresis machine (cell separator), which separates the plasma from the blood cells.
- The Prosorba column is a plastic cylinder that contains a sand-like substance coated with a material called **protein A**.
- Protein A binds unwanted antibodies from the plasma (that promote RA) and removes them.
- The Prosorba column is indicated for moderate to severe RA that hasn't responded to DMARDs or for patients that are allergic to such meds.



Surgical Options

- If disability from RA is significant and conservative management is ineffective, various surgeries are available for weight-bearing joints.
 - In early phases of RA, an **synovectomy** may be performed -- consisting of removing the inflamed synovia, which helps prevent destruction.
 - Regular arthroscopic surgery (trim damaged cartilage and clean out joint space) is not especially effective for symptoms.
 - Severely affected joints may require joint replacement surgery (**arthroplasty**), which often reduces pain and increases function.
 - Osteotomy (realignment) another option, depending on joint destruction.
 - Arthrodesis is surgical fusion of joints, usually in the spine.
 - Tendon repair surgery for trigger finger, swan neck deformity.
- Surgery carries a risk of bleeding, infection and pain.



Notes:

