# Medical Reference Manual

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Section I: Chagas disease

Introduction - Chagas disease, also known as *American trypanosomiasis*, was discovered in 1909 by Carlos Chagas in Brazil. The discovery of this disease is unusual, in that it presents the anomaly that the vector was discovered first, then the etiological agent, and finally the human consequences. Chagas affects the endothelial system and muscular fibers in particular, especially the stretch fibers found in the heart.

The disease is caused by the parasitic flagellate *Trypanosoma cruzii*. Reduviid bugs, or kissing bugs, are blood-sucking insects that transmit the disease by biting individuals infected with the parasite. Subsequent individuals may become infected by coming into contact with feces from the reduviid bugs, receiving contaminated blood transfusions, or the disease may be passed in utero from an infected mother to fetus.

Life cycle
**Epidemiology**
The disease is indigenous to the Americas and is distributed from the southern part of North America to every country in Central and South America. Worldwide, it is estimated that 16 to 18 million people are infected with Chagas disease; of those infected, 50,000 will die each year (CDC website).

The reduviid bugs tend to live in cracks and holes of substandard housing, and thus housing made with non lasting materials such as wood, mud, palm leaves and cardboard tend to be at higher risk for infestations. The prevalence in humans is especially high in rural areas, where up to 15% of the population may be infected with this parasite (Chinchilla, 2002)

*Trypanisoma cruzii* is a parasite that has adapted itself to many different animals, and has also been identified in dogs, foxes, marsupials, rats, and racoons. The parasite has a two different cycles: one in the jungle and another around human establishment. The disparate nature of these two locations suggests there may be a mediator animal between both of them, thus forming an intermediate reservoir. The *Trypanisoma cruzii* is very sensitive to environment changes. However, despite this fragility, it can survive a few days in refrigerated blood, making the serology study for *Trypanisoma cruzii* obligatory.

**Clinical and pathologic aspects**
There are three stages of Chagas disease: acute, intermediate, and chronic:

1. **Acute stage**: Approximately 1% of the people infected with *T. cruzi* will develop symptoms. In these individuals, the characteristic sign for a Chagas infection is the Romaña sign, in which the person's eye on one side of the face swells, usually at the bite wound or where feces were deposited or accidentally rubbed into the eye. Other non-specific symptoms seen in association with Chagas disease include:
   - Swollen lymph nodes
   - Fever
   - Enlarged liver or spleen
   These symptoms may last from 4-8 weeks and then resolve, or a patient may progress to the intermediate and chronic stages.

2. **Intermediate stage**: Typically occurs 8 to 10 weeks following initial infection. People at the intermediate stage of infection rarely demonstrate symptoms. This stage may last for several years.

3. **Chronic stage**: In 20-30% of the infected individuals, the disease will progress to its chronic form. Over time, the body produces antibodies against the *T. cruzi* parasite. In a select number of individuals, these antibodies also recognize epitopes found on healthy cells, leading to an autoimmune reaction. The chronic stage produces two different pathological aspects:
   - Cardiac problems: enlarged heart, arrhythmias, cardiac insufficiency, heart failure
   - Development of “megas”: Destruction of nervous fibers around hollow organs, such as the GI tract. Over time, the elastic condition of those organs will be lost, causing decreased peristalsis and excessive expansion of them. This results in megacolon and megaesophagus in affected individuals.
**Diagnosis, Treatment and Prevention**

During the **acute** stage, *Trypanosoma cruzii* may be diagnosed by observing the organism in the peripheral blood, detectable by direct observation under a microscope or with Wright or Giemsa stains. If the **chronic** stage of Chagas is suspected, diagnosis can be made through blood tests, clinical diagnosis, and EKG.

If Chagas is detected during the acute stage, it is possible to treat with **benznidazole** or **nifurtimox** (under an Investigational New Drug protocol from the CDC Drug Service). In the chronic stage of this parasite, there are no effective treatments currently approved for use, though a few products are currently being researched. Prevention consists in eliminate reservoirs, fumigation, and in generalized improvement in the social-economic conditions of an affected area.

**Section II: Leishmaniasis**

**Introduction** - Leishmaniasis is a parasitic infection caused by the bite of a sandfly infected with the intracellular protozoa *Leishmania*. There are three forms of Leishmaniasis: **cutaneous**, **mucocutaneous**, and **visceral**.

- The **cutaneous** form of the disease causes skin lesions all over the body, including the arms, legs, and face.
- The **mucocutaneous** form causes potentially disfiguring lesions of the mucous membranes of the nose, throat, and mouth.
- The **visceral** form affects the internal organs of the body (including the spleen, liver, and bone marrow).

![Sandfly](image)

![Sandfly bite](image)

![Body diagram](image)

Figure: Leishmaniasis is capable of causing a variety of symptoms, including cutaneous lesions or attack of the visceral organs, depending on the species.

Currently, there are 3 main species of *Leishmania* that have been identified: *L. donovani*, *L. tropica* and *L. braziliensis*. *L. donovani* produces mainly the visceral form of Leishmaniasis, with a tendency to locate itself in the spleen, liver, bone marrow and lymph nodes (reticuloendothelial system). *L. tropica* and *L. braziliensis* tend to cause the mucocutaneous and cutaneous forms of Leishmaniasis.

**Epidemiology** - Leishmaniasis is currently found in over 88 countries, placing approximately 350 million people at risk for acquiring the disease. Because the sandfly tends to be susceptible to colder climates, the disease is found primarily in tropical and subtropical locations. Current World Health Organization estimates of people affected by Leishmaniasis approaches 12 million.
Approximately 500,000 cases of visceral Leishmaniasis occur annually, with 90% of these cases found in five countries: India, Bangladesh, Nepal, Sudan, and Brazil. Cutaneous Leishmaniasis affects mainly Afghanistan, Brazil, Iran, Peru, Saudi Arabia, and Syria. Mucocutaneous Leishmaniasis appears mainly in Bolivia, Brazil, and Peru.

The distribution of visceral Leishmaniasis.

The insects that transmit this parasite are phlebotomine sand flies. Disease transmission occurs when the flies bite an infected animal or human. The reservoirs for the parasite are different types of wild rodent. The insects tend to be most active in the evening hours, and are about a third of the size of a mosquito. 

**Lifecycle**

**Sandfly Stages**

1. Sandfly takes a blood meal (injects promastigote stage into the skin)
2. Promastigotes are phagocytized by macrophages
3. Promastigotes transform into amastigotes inside macrophages
4. Amastigotes multiply in cells (including macrophages) of various tissues
5. Sandfly takes a blood meal (ingests macrophages infected with amastigotes)
6. Ingestion of parasitized cell
7. Amastigotes transform into promastigote stage in midgut
8. Divide in midgut and migrate to proboscis

**Human Stages**

1. Sandfly takes a blood meal (injects promastigote stage into the skin)
2. Promastigotes are phagocytized by macrophages
3. Promastigotes transform into amastigotes inside macrophages
4. Amastigotes multiply in cells (including macrophages) of various tissues
5. Sandfly takes a blood meal (ingests macrophages infected with amastigotes)

▲ = Infective Stage
▲ = Diagnostic Stage

http://www.cdc.gov/dpdx
Clinical and pathological aspects

Cutaneous Leishmaniasis: lesions occur in places where the sandflies have fed, usually within a few weeks of the initial bite. Over time, the sores take on a volcanic appearance, with a rounded shape, raised edges and a central crater area. Examinations of the crater area may reveal papilla. Swollen lymph nodes may also be present near the site of the lesions.

Mucocutaneous Leishmaniasis: lesions found on the mucous membranes of the mouth, nose, and throat. Over time, these lesions can cause significant disfiguration. The muco-cutaneous lesions can destroy the nasal bone, as well as the soft palate of the mouth, causing deformations in nose and skin around the lips. Due to these complications, Leishmaniasis is sometimes mistaken for leprosy (Hanson’s Disease).

Visceral Leishmaniasis: This form of the disease is also called kala azar. It develops several months after the initial infection, and is characterized by fever, anemia, weight loss, and enlargement of the spleen and liver. Left untreated, visceral Leishmaniasis has a mortality rate of almost 100%. This form of the disease has also recently emerged as a growing problem, as it is increasingly being identified as an opportunistic infection in individuals infected with HIV. In immunosuppressed individuals, such as those with HIV, the disease progresses rapidly to the visceral form. Visceral Leishmaniasis also appears to accelerate the onset of AIDS in HIV positive individuals.

Diagnosis, Treatment and Prevention

Diagnosis of this disease is made via biopsy and subsequent observation of the parasite under microscope, after treating the specimen with Wright, Giemsa or Leishman staining. Further confirmation is sometimes needed through cell culture or animal inoculation. Another laboratory test is the Montenegro reaction, which consists of injecting Leishmaniasis antigen into the dermis of a person suspected to be infected with the parasite. The skin is then examined 48 hours following subdermal injection. Positive results are defined as erythematous (reddened) and swollen area surrounded the site where the antigen was injected.

Treatment is antimony or arsenic products, such as Reprodal and Glucatyme. Both of these drugs are extremely toxic to the liver, and thus therapy must be followed closely by a physician.

Prevention of this disease comes from avoiding exposure to sand flies. Insect repellent, mosquito nets, and insecticide applied to living and sleeping areas can help decrease the risk of exposure. Treatment of patients with Leishmaniasis will also help decrease the number of human reservoirs, as well as eliminate the risk of spread of the disease from blood-borne exposure to infected individuals.

Section III: Malaria

Introduction - Malaria is an infection that affects red blood cells and is caused by different species of Plasmodium parasite. From Italian, the name malaria literally means “mal aire” (bad air). Patients suffering from malaria experience periodic attacks of chills, fever, and sweating. Because outbreaks of malaria are associated with areas containing stagnant water, in the past it was assumed that the foul odor of the water was the causative agent of the disease. However, in 1880 Charles Louis Alphonse Laveran, a French army surgeon stationed in Constantine, Algeria, noted the presence of the Plasmodium parasite in a blood smear from a patient suffering from malaria.

Currently, there are two reservoirs present in nature for this parasite: humans and the Anophele mosquito. Malaria is transmitted primarily from human to human transmitted the bites of infected female Anophele mosquitoes. The mosquitoes carry the Plasmodium parasite within its salivary glands and inject parasites into humans when it feeds. Transmission may also occur through congenital acquisition or through the transfusion of infected blood products.

Epidemiology - Approximately 41% of the world’s population lives in areas endemic for malaria. Because the Anophele mosquito and the plasmodium parasite require warmer temperatures to survive, malaria is typically found in tropical and subtropical areas. Current figures from the CDC website (www.cdc.gov) estimate that between 700,000 and 2.7 million people die each year from malaria, 75% of them Africa children. Currently, the area most affected by this disease is Sub-Saharan Africa.
The non-human vector responsible for transmission of this disease is the *Anopheles* mosquito. There are different species of the *Anopheles*, in Central America the most common are *A. albimanus* and *A. punctimacula*. These vectors live in swamphy places and some are capable of biting two or three times a day, depending on the feeding habits of a particular species. Both the *Anopheles* mosquito and the plasmodium parasite are susceptible to climate changes. The malaria parasite is unable to grow and reproduce inside the mosquito vector if temperatures are lower than 16°C or higher than 33°C.

In endemic areas, children are more susceptible to become infected and morbidity among this age group is high. The infectivity rate of malaria decreases proportionally to age increment. In non-endemic areas, the whole population is equally susceptible of getting infected. Occasionally epidemic episodes may be caused by environment or climatic changes, increasing longevity and density of the vector.

Four different species of the Plasmodium parasite cause malaria: *P. vivax*, *P. malariae*, *P. ovale*, and *P. falciparum*. *P. vivax* and *P. falciparum* cause the majority of infections in the world, especially in Africa. *P. falciparum* causes more disease than other species of plasmodium, and tends to have a higher incidence of drug resistant strains. *P. vivax* infection is uncommon among blacks, as their red blood cells have evolved to eliminate a surface antigen known as the Duffy factor, thus making them resistant to infection.

*Life Cycle*
Malaria is spread by mosquitoes of the *Anopheles* family. (next page)
The malaria parasite life cycle involves two hosts. During a blood meal, a malaria-infected female *Anopheles* mosquito inoculates sporozoites into the human host 1. Sporozoites infect liver cells 2 and mature into schizonts 3, which rupture and release merozoites 4. (Of note, in *P. vivax* and *P. ovale* a dormant stage [hypnozoites] can persist in the liver and cause relapses by invading the bloodstream weeks, or even years later.) After this initial replication in the liver (exo-erythrocytic schizogony 5), the parasites undergo asexual multiplication in the erythrocytes (erythrocytic schizogony 6). Merozoites infect red blood cells 5. The ring stage trophozoites mature into schizonts, which rupture releasing merozoites 6. Some parasites differentiate into sexual erythrocytic stages (gametocytes) 7. Blood stage parasites are responsible for the clinical manifestations of the disease.

The gametocytes, male (microgametocytes) and female (macrogametocytes), are ingested by an *Anopheles* mosquito during a blood meal 8. The parasites’ multiplication in the mosquito is known as the sporogonic cycle 9. While in the mosquito’s stomach, the microgametes penetrate the macrogametes generating zygotes 10. The zygotes in turn become motile and elongated (ookinetes) 11 which invade the midgut wall of the mosquito where they develop into oocysts 12. The oocysts grow, rupture, and release sporozoites 13, which make their way to the mosquito’s salivary glands. Inoculation of the sporozoites 14 into a new human host perpetuates the malaria life cycle.

**Clinical and pathological aspects** - Fever, anemia and splenomegaly are the fundamental clinical and pathological aspects of malaria. Other associated symptoms of malaria include fatigue, dizziness, gastrointestinal symptoms, muscle pains, and dry cough. These symptoms usually result from the release of inflammatory factors in response to the parasite presence.
Once the parasites have entered the bloodstream, they invade the red blood cells, replicate, and eventually cause the red blood cells to rupture. The febrile attacks characteristic of malaria are caused when the red blood cells rupture, releasing the new crop of parasites. These attacks tend to display a sequential pattern that occurs within a 4-6 hour period. Intense, shaking chills (the cold stage) are followed by high fever to 41 degrees or higher (the hot stage), then profuse sweating (the sweating stage), and a final period of remission. The pattern of fever is particular to different species and is cyclic in nature. Cycles of invasion and rupture occur every 48 hours (or 72 hours for *P. malariae*) and may be repeated multiple times. Between fever attacks, the patient may experience fatigue, but is otherwise asymptomatic.

The various species of plasmodium also invade different types of red blood cells (RBCs), producing diverse clinical pictures in the patient. *P. vivax* and *P. ovale* preferentially invade reticulocytes (immature red blood cells composing approximately 1% of total blood volume), while *P. malariae* favors mature RBCs. *P. falciparum* is the most non-discriminatory of the 4 species, invading all types of RBCs. As a result of this preferential cell invasion, *P. malariae* and *P. falciparum* tend to produce anemia.

*P. falciparum* tends to produce the most serious form of malaria, and has even been known to cause death within 24 hours of infection in some cases. In these severe cases, the *P. falciparum* parasite may invade up to 3-5% of the RBC volume. Further complications from infections of *P. falciparum* arise from the ability of this parasite to cause RBCs to sequester and adhere to one another within capillaries and postcapillary venules. As a result of this, patients may experience a number of complications, including:

- Cerebral malaria
- Hemolytic anemia
- Cardiac dysrhythmias
- Hyperpyrexia
- Pulmonary edema
- Renal failure

Malaria is often found in areas endemic for HIV as well, though current studies indicate that malaria does not function as an opportunistic infection. However, pregnant woman are the largest at-risk population for malaria infection, though the exact mechanism of this susceptibility is poorly understood.

![Malaria Cycle Diagram](image)

**Diagnosis, Treatment and Prevention** - Diagnosis of malaria is based on microscopic evaluation of blood smears and clinical history. Normally, with the exception of *P. falciparum*, only approximately 2% of the cells will be infected with the malaria parasite.
Treatment of malaria is dependent on geography. P. malariae, P. vivax, and P. ovale are all sensitive to the chloroquine. However, P. falciparum has developed resistance to chloroquine. Identified areas with these strains of Plasmodium include Africa, Central America, areas south of the Panama Canal, South America, India, and South East Asia. Thus, in areas with chloroquine-resistant P. falciparum, treatment options include quinine, artemether, mefloquine, or pyrimethamine/sulfadoxine. In many cases, these drugs must be combined to treat malaria. Patients on these medications must be monitored closely, as they are toxic to the liver.

**Prevention of malaria** - The best prevention is the elimination of stagnant water, the use of insect repellent and mosquito nets.

### Section IV: Dengue fever

**Introduction** - Within the past 20 years, Dengue has emerged as the most important of the arbovirus diseases (diseases transmitted by mosquitoes, ticks, etc.) due to its rapidly expanding incidence in tropical and sub-tropical regions of the world. Dengue is a mosquito-borne virus that belongs to *Flaviviridae* family. Dengue produces a wide spectrum of diseases in infected human hosts, ranging from non-specific viral symptoms to the potentially fatal Dengue hemorrhagic fever (DHF). The virus has four different, antigenically distinct subtypes: DEN-1, DEN-2, DEN-3 and DEN-4. Once people are infected with a particular subtype, long-term immunity is created. Thus, for persons living in endemic areas, it is possible to acquire Dengue up to four times within the span of a person’s lifetime. Over the past 20 years, the incidence of Dengue has increased substantially within the Americas (www.cdc.gov).

**Epidemiology** - Approximately 100 million people are infected with Dengue every year, and of these, several hundred thousand cases of DHF occur according to recent CDC estimates. The case-mortality for Dengue is currently approximately 5%, however with proper management of the disease, the mortality rate falls to less than 1%. Dengue epidemics have become larger and more frequent within the past 25 years, making proper management and vector control essential.

**World Distribution of Dengue - 2005**

Dengue is spread by the *Aedes* mosquito, most commonly the *Aedes aegypti* species. This mosquito prefers to feed during the day, and is responsible for the majority of Dengue transmission. The mosquito is often found near human habitations, and has two main feeding periods: once in morning after daybreak and in the afternoon shortly before dark.
Clinical Presentations - Clinical symptoms typically occur 4-7 days following exposure from an infected mosquito. The Dengue virus produces a wide spectrum of symptoms in the patient. Currently, there are four clinical syndrome definitions for patients presenting with Dengue:

1. Undifferentiated fever
2. Classic Dengue fever
3. Dengue hemorrhagic fever
4. Dengue shock syndrome

The majority of people infected with Dengue acquire a mild form of the disease, undifferentiated fever. A prospective study (DS Burke, et al. A prospective study of dengue infections in Bangkok. Am J Trop Med Hyg 1988; 38:172-80.) estimated that approximately 87% of people infected with Dengue are asymptomatic or have only mild viral symptoms. No rash is manifested in these patients.

Other patients present with non-specific viral symptoms, otherwise known as classic dengue fever. In this presentation of Dengue, patients experience sudden onset of fever (typically high, up to 105 degrees Fahrenheit), headaches, retroocular (behind the eye) pain, myalgia, chills and back pain. The joints are rigid and produce a particular gait in the patient known as “dandy gait”.

Dengue fever is characterized by:

- Fever
- Rash
- Muscle and joint pains

Aedes aegypti mosquito
The fever of Dengue tends to be biphasic in nature. Thus, the initial presentation of symptoms tends to last 3-7 days, followed by a brief period of remission. The patient then experiences another period of fever, which lasts hours to several days. During this time, the characteristic rash of Dengue also appears. The rash appears 3-5 days after onset of fever and can spread from the torso to the arms, legs, and face. It typically has a scarletiniform or macule-papular form.

The disease is usually self-limited, although convalescence can be prolonged. The patients may also have swollen lymph nodes, decreased white blood cell count and small hemorrhagic manifestations. Symptoms of classic dengue fever typically last about a week, and following this period will completely disappear.

The remaining two presentations of the virus produce severe, potentially fatal forms of Dengue known as dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS). Any four of the Dengue subtypes can produce these syndromes, however the DEN-2 is most commonly associated with petechial rash and gastro-intestinal hemorrhage. DHF and DSS mainly effects children. If recognized and treated properly, the mortality rate from dengue hemorrhagic fever is less than 1%.

The beginning of Dengue hemorrhagic fever (DHF) is exactly the same as that of classic dengue fever. The critical stage for hemorrhagic fever begins when the initial fever remits. Following the abatement of the fever, the patient begins to demonstrate signs of capillary fragility. Most patients demonstrate only mild symptoms, including small skin hemorrhages (petechiae) or microscopic hematuria (blood in the urine). However, some patients have more severe symptoms, including: bleeding gums, nose bleeds, vomiting of blood, and blood in bowel movements.

A selected number of patients will progress to dengue septic shock. Due to a high level of vascular permeability, patients with DSS rapidly progress to complete circulatory failure. Early signs of DSS are restlessness, cold clammy skin, rapid weak pulse, narrowing of pulse pressure, and hypotension. Progression of DHF to DSS may also be marked by: severe abdominal pain, vomiting, marked changes in temperature (from fever to hypothermia), and change in the mental status of the patient (either irritability or progression to non-responsiveness).

Fatality rates among those with DSS may be higher than 10%. DHF and DSS occur in children and adults. The prognosis for both depends on an early diagnosis and appropriate management, including maintenance of appropriate blood pressure and electrolytic balance in the patient.

Currently, the exam mechanism for the hemorrhagic symptoms in Dengue is unknown. Some subtypes of the Dengue virus contain envelope glycoproteins that are similar to a number of clotting factors within the body, including plasminogen. One proposed theory suggests that DHF is the result of an autoimmune reaction, due to the high level of homology between the virus envelope and the body’s coagulation system.

**Diagnosis, Treatment and Prevention** - The gold standard for diagnosis of Dengue is based on serologic tests. However, if serological testing is unavailable, clinical presentation and the tourniquet test may be applied. The tourniquet test is used to assess capillary fragility.

To perform the tourniquet test, a blood pressure cuff is placed on the upper arm of the patient and inflated for 5 minutes to a point midway between the systolic and diastolic pressure of the patient. The cuff is then removed and the skin is allowed to return to its normal coloring. The number of petechiae produced from the test are counted for over a one square inch (or 2.5 cm) area. If more than 20 petechiae are noted, the test is considered to be positive.
**Characteristic rash of Dengue**

Treatment depends on clinical syndrome:

1. **Classic dengue fever and undifferentiated fever**: requires fluids and use of antipyretic medications such as Tylenol. *Never use aspirin. Use of aspirin is associated with bleeding tendency and Reye’s syndrome in children.*

2. **Hemorrhagic dengue and/or dengue shock syndromes**: requires hospitalization, IV fluids (Dhaka solution or Ringer lactate), antipyretics and sometimes platelet transfusion.

Prevention is sustained in elimination of vector mosquito by avoiding stagnant water and fumigation. A vaccine for Dengue is still in the research stage of development.

**Section V: Evaluation and Treatment of Diarrhea**

Introduction - Diarrhea can present in a number of different manners in the patient, ranging from a mild, self-limited episode to a severe, life-threatening illness. Diarrhea is defined as an increase in stool weight (more than 200 g/day), or the passage of more than three loose, watery stools per day (World Health Organization definition). Clinically, a patient with diarrhea presents with increased stool frequency, liquidity of feces, increased sense of urgency, or fecal incontinence.

In evaluating the cause of the diarrhea, a number of different factors can be used during the evaluation including: duration of illness, severity of disease, presence of systemic symptoms (such as fever), and characteristics of the stool. However, because the number of etiological agents causing diarrhea is substantial, it is first useful to determine if the diarrhea is **acute** or **chronic**.

**Evaluating Diarrhea**

**Acute diarrhea** - Acute diarrhea is defined as lasting less than 3 weeks and is most commonly caused by infectious organisms or toxins. Over 90% of acute diarrhea tends to be mild and self-limited. Other details suggestive of an infectious cause for an acute presentation include similar recent illness in family members, ingestion of improperly prepared foods, or exposure to unpurified or contaminated water.

If the presenting case is determined to be acute in nature, another helpful distinguishing factor is if the diarrhea is **noninflammatory** or **inflammatory**.

**Non-inflammatory Diarrhea** - Etiological agents causing this type of diarrhea do not invade the gastrointestinal cells. Because of this, no fecal leukocytes or systemic symptoms (such as fever, headache, increased white blood cell count) occur. Patients with non-inflammatory diarrhea typically present with watery, nonbloody diarrhea and **lack of systemic symptoms**. Some non-invasive bacteria produce an enterotoxin, which may also cause nausea and vomiting to be present. Most cases of non-
inflammatory diarrhea originate in the small intestines and tend to be mild. However, bacteria such as cholera are capable of producing voluminous, life-threatening cases of acute, non-inflammatory diarrhea.

Causes of acute non-inflammatory diarrhea:
- Viral: Norwalk virus, Rotavirus
- Protozoal: Giardia, Cryptosporidium
- Bacterial: Staphylococcus aureus, Enterotoxigenic E. coli, Vibrio cholera

**Inflammatory Diarrhea** - Etiological agents that produce inflammatory diarrhea typically invade intestinal cells, producing cell death and a systemic inflammatory reaction. Patients present with fever and bloody diarrhea. Because the infectious agents invade the intestinal tissue, fecal leukocytes will also be present.

Causes of acute inflammatory diarrhea:
- Viral: Cytomegalovirus
- Protozoal: Entamoeba histolytica
- Bacterial: Enterohemorrhagic E. coli 0157, Vibrio cholera, Enteroinvasive E. coli, Salmonella, Neisseria gonorrhoeae, Yersinia enterocolitica, Listeria monocytogenes, Shigella, Chlamydia

**Chronic diarrhea** - Chronic diarrhea is defined as lasting more than 4 weeks and an infectious etiology is uncommon. To evaluate cases of chronic diarrhea, causes are grouped into six major categories: secretory diarrhea, osmotic diarrhea, inflammatory conditions, malabsorption syndromes, motility disorders, or chronic infections.

**Secretory diarrhea** - This type of diarrhea is caused by and abnormal ion transport across intestinal epithelial cells, resulting in decreased absorption and/or increased secretion. Abnormal transport results in decreased sodium chloride absorption or an increase in chloride secretion, resulting in water accumulation inside the intestinal lumen. Diagnostic clues for this type of diarrhea include production of large stool volumes that demonstrate little change in output with patient fasting.

A typical example of secretory diarrhea is cholera. Cholera is caused by a gram negative bacillus named Vibrio cholerae. Cholera is an acute infectious disease, non toxic and non invasive, capable of causing epidemics. The pathology of the disease is characterized by excessive, rapid loss of gastrointestinal fluids, saline depletion, acidosis and shock. The illness presents with vomiting, diarrhea, and abdominal cramps, liquid, voluminous and clear stools (known as rice water diarrhea). Up to 10 to 15 L of intestinal fluid can be lost per day, resulting in dehydration, metabolic acidosis and hypocalcemia. Mortality is over 60% in non treated patients, but is reduced to only 1% in those patients who receive adequate rehydration.

Other examples of secretory diarrhea include stimulant laxatives, intestinal resection, neuroendocrine tumors (Zollinger-Ellison syndrome, VIPoma, medullary carcinoma of the thyroid), medications, and bile salt malabsorption.

**Osmotic diarrhea** - Osmotic diarrhea is caused by the presence of poorly absorbed, osmotically active solutes in the lumen of the intestines. Typically, when stool leaves the colon, the estimated osmolality is approximately 290 mosm/kg. If the osmolality is markedly different from this expected value (>125 mosm/kg), this implies the ingestion or malabsorption of an osmotically active substance. Osmotic diarrhea resolves with fasting. The most common cause of osmotic diarrhea is lactose intolerance. Other causes include: medications (including laxative abuse, antacids, sorbitol) and factitious diarrhea (antacids, laxatives).

**Abnormal intestinal motility** - Abnormal motility of the intestinal tract is typically secondary to a systemic disease or following surgery. Two mechanisms may be the cause of abnormal intestinal motility:
1. Enhanced motility: results in rapid transit of material through the intestines and decreased contact between luminal contents and absorptive epithelial cells. Causes of Enhanced motility: some types of tumors, gastrectomy or in irritable bowel syndrome, hyperthyroidism
2. Slowed motility: stasis of material within the intestinal lumen can lead to bacterial growth, resulting in malabsorption and subsequent diarrhea. Causes of slowed motility: diabetes mellitus, blind intestinal loop.

Irritable bowel syndrome is probably the most common cause of chronic diarrhea.

**Inflammatory Conditions** - Patients with these diseases tend to present with bloody diarrhea and a variety of other symptoms including fever, abdominal pain, and hematochezia. Causes of inflammatory chronic diarrhea: Ulcerative colitis, Crohn’s disease, radiation enteritis, malignancy, or microscopic colitis.

**Malabsorption Syndromes** - Malabsorption syndromes are caused by a number of factors, including mucosal intestinal disease, intestinal resection, lymphatic obstruction, pancreatic disorders, and bacterial overgrowth. Patients with these diseases present with diarrhea, weight loss, osmotic diarrhea, and nutritional deficiencies. Causes of malabsorption syndrome include: small bowel mucosal disorders, pancreatic disease, bacterial overgrowth, lymphatic obstruction.

**Chronic Infections** - Certain parasitic infections may proceed to a chronic state, such as giardia, *Entamoeba histolytica*, and the intestinal nematodes. Immunocompromised patients (such as patients with HIV) are susceptible to chronic diarrhea from a number of different etiological causes. Common causes of chronic infection diarrhea in the immunocompetent host: *Giardia lamblia, Entamoeba histolytica*. Causes of AIDS-related chronic infection diarrhea:
- Viral: HIV infection, Cytomegalovirus
- Bacterial: *Clostridium difficile, Mycobacterium avium* complex
- Protozoal: Microsporidia, cryptosporidium, *Isospora belli*

**Section VI: Intestinal Parasites**

**Macroscopic Parasites (Worms)**
**Nematodes (roundworms)**
Nematodes are roundworms with bilateral symmetry that vary in size from 2mm to 1 meter. The digestive system includes one mouth, that occasionally may have hooks, one pharynx, one esophagus and one intestine that ends in the rectum. This parasite has well differentiated male and female sex. Females are capable of producing between one dozen and one million eggs per day.

**Ascariasis (roundworms)** - Ascaris lumbricoides is the most common intestinal helminth (worm) in human beings. Currently, an estimated 1 billion people are infected with Ascaris. Transmission of the disease occurs through the fecal-oral route, when humans ingest mature eggs in fecally contaminated food. Thus, Ascaris has a high incidence in areas with poor hygiene and sanitation or in areas in which human excrement is used as fertilizer.

**Life cycle** - Once a person ingests mature eggs, the eggs hatch in the small intestines and the larvae penetrate the intestinal wall. The larvae then travel in the blood stream until they reach the heart, where they burrow into the alveolar wall. The larvae mature in the lungs and then travel up the bronchus, where they are coughed up and swallowed into the esophagus. From the esophagus, the worms travel back down the intestinal tract to the small intestine. The life cycle is then completed in the small intestines, where the newly matured nematodes form eggs than then are passed in the feces of the infected human.
Adult worms live in the lumen of the small intestine. A female may produce approximately 200,000 eggs per day, which are passed with the feces. Unfertilized eggs may be ingested but are not infective. Fertile eggs embryonate and become infective after 18 days to several weeks, depending on the environmental conditions (optimum: moist, warm, shaded soil). After infective eggs are swallowed, the larvae hatch, invade the intestinal mucosa, and are carried via the portal, then systemic circulation to the lungs. The larvae mature further in the lungs (10 to 14 days), penetrate the alveolar walls, ascend the bronchial tree to the throat, and are swallowed. Upon reaching the small intestine, they develop into adult worms. Between 2 and 3 months are required from ingestion of the infective eggs to oviposition by the adult female. Adult worms can live 1 to 2 years.

**Signs and symptoms**

1. Abdominal pain
2. Anorexia
3. Nausea and vomiting
4. Diarrhea and/or constipation
5. Nocturnal cough

**Complications**

Complications can arise, depending on the area in the body in which the worms and larvae settle:

1. Petechial hemorrhages
2. Alveolar pneumonitis
3. Severe infections that may produce lethal bronchopneumonia
4. Eosinophilia
5. Uticaria

**Enterobiasis (pinworms)**

This disease is produced by Enterobius vermicularis. [Life cycle (next page):](http://www.dpd.cdc.gov/dpx)
Eggs are deposited on perianal folds. Self-infection occurs by transferring infective eggs to the mouth with hands that have scratched the perianal area. Person-to-person transmission can also occur through handling of contaminated clothes or bed linens. Enterobiasis may also be acquired through surfaces in the environment that are contaminated with pinworm eggs (e.g., curtains, carpeting). Some small number of eggs may become airborne and inhaled. These would be swallowed and follow the same development as ingested eggs. Following ingestion of infective eggs, the larvae hatch in the small intestine and the adults establish themselves in the colon. The time interval from ingestion of infective eggs to oviposition by the adult females is about one month. The life span of the adults is about two months. Gravid females migrate nocturnally outside the anus and oviposit while crawling on the skin of the perianal area. The larvae contained inside the eggs develop (the eggs become infective) in 4 to 6 hours under optimal conditions. Retroinfection, or the migration of newly hatched larvae from the anal skin back into the rectum, may occur but the frequency with which this happens is unknown.
Transmission

1. mouth-hand-mouth
2. Anus-mouth
3. Indirect
4. Inhalation
5. Retro-infection

Symptoms

1. Intestinal erosions
2. Intense anal itching and perianal ulcers
3. Appendicitis (rare)
4. Nervousness

Treatment - In both cases treatment is with albendazole.

Uncinariasis (hookworms)

Ancylostoma duodenale, Necator americanus are the hookworms that affect human beings. The only difference between the two of them is the geographic distribution and their size.

Life Cycle - The life cycle in humans begins when a larva penetrates the skin, especially through the feet or hands, then those larvae are transported to the lungs through the circulation. Once they mature there, the cycle is exactly the same as in Ascaris lumbricoides.
**Epidemiology** - The transmission of infectious larvae requires the eggs to be in well drained and humid land, so that the infection may be located in tropics and subtropics and the southern United States. Every female is capable of producing between ten and twenty thousand eggs in 4 to 8 weeks and the production can continue for 5 years.

**Signs and symptoms**
1. Local exanthema
2. Pneumonitis
3. Nausea, vomiting and diarrhea
4. Anemia (blood loss of 0,15 and 0,25 ml per day)
5. Secondary intestinal infections

**Diagnosis** - Feces sample is the simplest way to diagnose the infections caused by hookworms.

**Treatment** - The drugs of choice are Mebendazole or Albendazole and ferrous sulfate to treat anemia.

**Prevention** - Education (wear shoes), improve sanitary conditions and the adequate disposal of feces are the most important preventive measures.

**Nematodes of the tissues**
Trichinosis - Trichinella spiralis is the causal agent. Adults may live in duodenal mucosa of many mammals around the world. Infectious larvae are found in different muscles, but pigs are the domestic animals affected in most of the cases. The infection begins when infected meat is swallowed, then infectious larvae are freed in the small intestine where they mature into adult stages. One single female may produce more than 1500 larvae in the next 1 to 3 months. The larvae pass from the intestinal mucosa to the muscle fibers through circulation, and there they are transformed into cysts.

**Epidemiology** - Trichinosis is found worldwide and its prevalence is related to the use of pork as food. Besides pigs, many other mammals may be contaminated and host the microorganism, and they represent a potential source of infection for human beings.

**Signs and symptoms**
1. Mild diarrhea and flu-like symptoms in mild infections
2. Fever, gastrointestinal symptoms, eosinophilia, muscle pain and periorbital edema in more complicated infections
3. Bleeding under the nails may be a symptom of toxic migration of the larvae.
4. In very complicated cases neurologic symptoms and signs may appear, including psychosis, meningoencephalitis and strokes.
5. Death may occur within the next 4 to 6 weeks of the primary infection.

**Diagnosis, treatment and prevention** - Diagnosis is based on clinic symptoms, especially during epidemics. Muscle biopsy may also be done. 
**Treatment** - Treatment is symptomatic because there is no treatment to cure the larvae infection. During intestinal infection Albendazole may help. 
**Prevention** - Prevention is very important, especially the education of the population whose diet is based on pork or bear meat. Meat must be very well cooked until it changes into a gray color; microwaving or smoking the meat does not kill the parasite). Storage must be in a freezer with a temperature lower than – 40 °C.

**Filariasis (elephantiasis)**
**Life Cycle** -- The life cycle of filariasis will always require a vector that in most cases is an insect. In the insect the larvae evolve from the first to the third stage. Once the mosquito bites a human the stage three larva is transmitted. It circulates through the lymphatic system and will stay there for about one year, maturing into adult forms. These parasites are very susceptible to physiologic changes of the host (such as fever), and die easily.

Filariasis Bancrofi - Produced by Wuchereria bancrofti, affects mainly the lymphatic system and the connective tissue. It is transmitted by a mosquito called Culex pipiens.
Symptoms
1. Local inflammatory process
2. Obstruction of the lymphatic system
3. Fibrosis and calcification
4. Hypertrophy of subcutaneous tissue (elephantiasis) that may occur in between 1% and 20% of all cases.

Diagnosis - During the inflammatory process, eosinophilia may appear, nevertheless, it is necessary to demonstrate the presence of microfilariae in blood to make the definitive diagnosis. The blood sample must be taken at night in order to have a higher amount of filarias present.

Treatment - In cases of chronic lymphatic infection treatment is not really helpful and in those cases the drug of choice is dietilcarbamazine. Ivermectine is still in research. Surgery and support therapy are helpful and have esthetic utility.

Prevention - The vector control, use of insect repellent and adequate clothing are the basic preventive measures.

Oncocercosis
It is produced by Onchocerca volvulus which has a life cycle identical to that of Wuchereria bancrofti. It is transmitted by mosquito species of Simulium also known as “coffee sand fly”.
Signs and symptoms
1. Inflammatory process around the bite that can be followed by a fibrotic process.
2. The larvae will directly affect the eyes, where they arrive through the circulation. They can produce conjunctivitis, queratitis, iritis, corneal opacity and finally, blindness, caused mainly by an immunologic reaction that produces tissue destruction.

Diagnosis - Diagnosis is done by biopsy and a blood test.

Treatment
1. Surgical removal of the nodes
2. Antihistamines and steroids.

Trematodes
Trematodes are leaf shaped helminths, which vary in size from 0.5 mm to several centimeters. The organism’s digestive system is very simple, basically composed of one mouth, one esophagus and one excretory system. They do not have nervous or circulatory systems. They are hermaphrodites, except the Schistosoma type.

These parasites are divided into 4 different groups:
1. Group I circulatory system parasites, such as Schistosoma.
2. Group II digestive tract parasites, such as Fasciolopsis.
3. Group III liver parasites, such as Fasciola hepatica.
4. Group IV respiratory tract parasites, such as Paragonimus.

Schistosomiasis
This disease is produced by Schistosoma mansoni, a pathology which must be reported to the World Health Organization.

Life Cycle – Both male and female live in the mesenteric veins. The larvae pass through the intestinal wall to get to the lumen and circulate with the feces to the environment in order to serve as a parasite in a snail, where they will mature into sporocysts and cercaria, that travel to the environment again and penetrate the human host through the skin. Once there they reach the lymph nodes, lungs and heart,
from which they circulate to the liver in order to mature into adults. They then go to the mesenteric veins. It is transmitted through contaminated water.

**Signs and symptoms**

1. **Urticaria**
2. During migration and maturity symptoms are mild.
3. When they pass through the intestinal wall, necrosis and intestinal ulcers may appear, resulting in disentery and severe abdominal pain.
4. **Hepatomegalia.**
5. Occasionally larvae may invade bone marrow and anemia may develop.

**Cestodes (tapeworms)**

Their shape and constitution may vary, but all of them have very well developed muscles. They are constituted by 3 units:

- Escólex or primitive head.
- Neck or germinative part.
- Estrobile that forms most part of cestode’s body. It is at the same time formed by many different units called proglotides.

They are hermaphrodites

The proglotides are in different stages:

- Immature
- Mature with well developed sexual organs
- Grávido filled with many eggs.

They have a relatively developed nervous system.

**Life cycle** - Adults are found in the small intestine, where they reproduce and eggs are transformed into stage 1 larvae. They go to the environment with the feces where they serve as a parasite of a micro-snail, developing into stages 2 and 3 larva (infectious stage), that then pass to a second intermedial host in order to mature into stage 4 larva, the definitive infectious stage for humans.

**Teniasis**

This parasitic infection may be caused by two different species Taenia saginata and Taenia solium, the first one can measure even 12 meters and its intermediate host is the cow; the second one measures less than 5 meters and its intermediate host is the pig. Both species have a long longevity and there are reported cases of parasites living 25 years. Usually the infection is caused by only one parasite.
Signs and symptoms
1. Abdominal pain, diarrhea and/or constipation.
2. Anorexia
3. Intestinal obstruction
4. Appendicitis caused by migration of proglotides.

Diagnosis, Treatment and Prevention - Diagnosis is based on the direct observation of eggs or proglotides in feces. The treatment is Albendazole.

Cisticercosis
Cisticercosis syndrome produced when larvae of Taenia are developed in humans, especially T. solium. It happens when a human eats the parasite eggs, so intermediate hosts are not present and larvae pass through the circulation or lymphatic system to different organs, usually the brain. Those larvae cannot finish the life cycle, but they can produce pathology.
Signs and symptoms - Symptoms depend on where the larvae are located. But in any case, inflammatory processes, fibrosis, degradation of tissue and calcification may be found in the site of lesion.

The most common sites where cisticerci are located are:
1. Subcutaneous tissue (usually asymptomatic)
2. Brain, where the main symptoms are intense cefalea, nausea, vomiting, seizures and even death.
3. Eyes, where symptoms are produced by pressure.

Diagnosis, Treatment and Prevention - The diagnosis is made when larvae are observed, directly in tissue or indirectly by CT scan, X-rays or blood tests. At present, the only treatment known is surgical removal of larvae.

Section VI. b. Macroscopic parasites (Protozoa)

Protozoa are very small and unicellular animals that are capable of doing any vital process. They have two main parts: cytoplasm and nucleus. In the external part of the cytoplasm they can have motion structures such as cilia, pseudopodes or flagelli.

Amoebas - In the majority of cases, amoebas are simple guests and only in a few cases do they produce pathology.

Life Cycle - There are two different stages: a mobile one called trofozoite, that will change its shape to form a pre-cyst that will finally produce the cystic stage, that infects the intestine and produces the amoebas. Entamoeba type:
<table>
<thead>
<tr>
<th>Group</th>
<th>Specie</th>
<th>Infectious stage</th>
<th>Habitat</th>
<th>Transmission</th>
</tr>
</thead>
<tbody>
<tr>
<td>First</td>
<td>E. gingivalis</td>
<td>Trofozoite</td>
<td>mouth</td>
<td>Direct contact</td>
</tr>
<tr>
<td>Second</td>
<td>E. polecki</td>
<td>Uni-nucleus cyst</td>
<td>Large intestine</td>
<td>Ídem E. histolytica</td>
</tr>
<tr>
<td>Third (only in fish)</td>
<td>E.gadi</td>
<td>Bi-nucleus cyst</td>
<td>Intestine</td>
<td>???</td>
</tr>
<tr>
<td>Fourth</td>
<td>E. histolytica</td>
<td>Tetra-nucleus cyst</td>
<td>Large Intestine</td>
<td>Anus-land-food-mouth</td>
</tr>
<tr>
<td></td>
<td>E. dispar</td>
<td></td>
<td></td>
<td>Anus-hand-objects-mouth</td>
</tr>
<tr>
<td></td>
<td>E. hartmani</td>
<td></td>
<td></td>
<td>Anus-land-water-mouth</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Through flies and cockroaches</td>
</tr>
<tr>
<td>Fifth</td>
<td>E. coli</td>
<td>Octo-nucleus cyst</td>
<td>Large Intestine</td>
<td>Ídem E. histolytica</td>
</tr>
</tbody>
</table>

Taken from Chinchilla, M. Parasitologia Medica. San José: editorial EACM, 1999.

Entamoeba histolytica
This specific amoeba produces pathology only when it is found in the large intestine. There is another form known as guest amoeba that will never produce pathology.

Life Cycle - During the pathologic life cycle the following changes may be observed: in the large intestine the amoeba multiplies and produces a pre-cyst, the by binarian division it produces a cyst with many nucleus, in the large intestine or in the environment. The tetra-nucleus cyst is the infectious stage that evolves to a meta-cystic form and finally matures into an amoeba. Entamoeba histolytica may produce intestinal or extra-intestinal pathology if it invades liver or lungs.
Signs and symptoms

1. Necrosis of the intestinal mucosa, producing ulcers, hemorrhages and dysentery.
2. Intense abdominal pain, tenesmo, headaches, anorexia and generalized muscle pain.
3. Some complications are appendicitis, peritonitis or shorter intestinal lumen, due to scarring of ulcers.
4. When amoebas get into the circulation they may go to other organs, especially the liver where they produce an abscess and from there they can reach the lungs passing through the diaphragm.
**Diagnosis, treatment and prevention** - Diagnosis is based on finding the trophozoites in feces or in serologic tests. Treatment is made with imidazolic products such as metronidazole (Flagyl) or in some cases chloroquine may help. The basis of prevention is cleanliness, boiling water, cooking meat very well and hand washing.

**Giardiasis**
This parasite of the small intestine is widely distributed around the world, it is produced by Lamblia intestinales, which is easy to recognize in the microscope due to its tennis racket shape. The infectious stage is the tetra-nucleus cyst.

![Photo: Arabela Paniagua](image)

**Signs and symptoms**
1. Deficient intestinal absorption manifested in yellow, watery and sulphurous diarrhea.
2. Anorexia and abdominal cramps.
3. Headaches and myalgia.
4. In some cases, if the giardia migrates to the biliary canals, it can produce an obstruction, causing estatorrea associated with jaundice.

**Diagnosis, Treatment and Prevention** - Diagnosis is made through feces culture, at least three on three different days (one each day). Treatment is the same as with amebiasis (Metronidazole)
Since giardiasis is transmitted by contaminated water sources, the basis of prevention is boiling water, improving the sanitary conditions of stored water.

**Life Cycle – next page**
Section VII. Parasites of the skin

Scabies  *Sarcoptes scabei*  File photo: Arabela Paniagua

Produced by *Sarcoptes scabei*, it is distributed worldwide in all ages and socioeconomic strata. Other types of scabies may cause infestations in other mammals such as domestic cats, dogs, pigs, and horses. They may cause temporary itching in humans due to dermatitis but they do not multiply on the human host.

Lesion caused by *S.scabei*. Taken from http://www.cdc.gov/ image library

Signs and symptoms

1. The waste products produced by mites burrowing the skin cause an allergic reaction rash, mostly found on hands, especially between the fingers, wrist, elbow and/or knee.
2. A papular scabies rash may be seen on scapular region and abdomen.
3. In newborns it can appear on the face.
4. Secondary bacterial infection may result due to scratching
5. In immunocompromised patients, a severe form of scabies can be seen, it consists on vesicles and formation of thick crust over the skin. In these cases itch is minor.
**Life cycle** - When a person is infested with scabies mites for the first time, there is usually little evidence of infestation for the first month (range 2 to 6 weeks). After this time and in subsequent infestations, people usually become sensitized to mites and symptoms generally occur within 1 to 4 days.
**Diagnosis, Treatment and Prevention** - Diagnosis is based on the characteristics of the rash and its distribution in the body. Whenever possible a sample of skin may be taken in order to examine it under the microscope to find the mites or ova.

When one person of the family is infested, the whole family must be treated, even if they do not have symptoms or signs. There are different alternatives to treat scabies, the most effective one is ivermectine on oral solution or pills (with the disadvantage of the elevated price). Other common treatments are lotions such as benzyl benzoate or crotamin. Crotamin is preferable for use in small children because it is less toxic. It is important to note that the itching sensation (which is an allergic reaction) can continue for up to two weeks even after the treatment has been successful and the mites are dead. Therefore, antihistamines may be given for immediate relief of the itching sensation.

Since mites can survive for several days in sheets, around beds or in the floor, prevention is based on cleanliness of the area, boiling sheets and bedding and improving the sanitary conditions.

**Lice**

![Pediculus humanus](image)

Pediculus humanus. File photo: Arabela Paniagua

Lice infestation is caused by different species, depending on what part of the body they are located: Pediculus humanus capitis, produces head lice, Pthirus pubis produces pubic lice and Pediculus humanus corporis produces body lice. They are transmitted by close contact like play, embraces, and sharing a bed. Lice can also be transmitted by sharing infested combs, brushes, towels or pillows. The louse feeds on blood several times a day and resides close to the scalp or skin in order to maintain its body temperature. It is important to note that in order to live, adult lice feed on human blood. They cannot live for more than 2 days off of their animal host.

**Signs and symptoms**

1. The majority of infestations are asymptomatic
2. Tickling feeling of something moving in the hair
3. Itching
4. Secondary bacterial infection caused by scratching
5.

**Diagnosis, Treatment and Prevention** - The diagnosis of pediculosis is best made by finding a live nymph or adult louse on the scalp or in the hair of a person. Finding numerous nits within 6 mm of the scalp is highly suggestive of active infestation. Finding nits only more than 6 mm from the scalp is only indicative of previous infestation. In order to treat lice is necessary to apply a lice shampoo based on lindane or benzyl benzoate. When pubic lice are found it is necessary to shave the pubic area and improve the local and general hygiene.
VIII. **Fungal infections of the skin**

Fungal infections may appear on any part of the body, but occur most frequently on the scalp, the parts without hair, between the toes or fingers and in the groin. Many fungal infections grow in the form of a ring. They often itch. Moist skin encourages fungal infections. This means fungal infections are more likely when skin is not dried properly after sweating heavily or bathing, or when it is covered with a material that does not allow sweat to evaporate. Damage to the skin surface, such as a cut or graze, can also encourage fungi to grow.

Fungal infections inside the body can cause more serious health problems than those on the skin. These infections only affect people whose immune systems are not working properly as a result of another illness or treatments for cancer.

**Dermatophytosis:** A superficial infection of the skin caused by a fungus

**Candidiasis:** infection by fungi of the genus **Candida**. **Candida** a genus of yeastlike fungi which produce mycelia. This infection also called Thrush often looks like small white patches, which leave a red mark when rubbed off. In adults, vaginal thrush can cause itchiness and a thick, white discharge.
Tinea Versicolor: This condition causes increased dark patches on pale or untanned skin and light patches on tanned or darker skin. People with oily skin are most likely to be affected.

**Diagnosis** - Fungal infections are usually easy to diagnose from the appearance and location of the rash, eg athlete’s foot. If the doctor wants to make sure what is causing the symptoms, he or she may take a scraping of skin or a fragment of nail or hair and send it to the laboratory for analysis before choosing the treatment.

**Treatment** - Since most skin fungal infections are surface infections, they are usually anti-fungal treatments applied directly to the infected area. There are a variety of treatments available in the form of creams, lotions and medicated powders. Over the counter antifungal creams are ½ the strength of prescription antifungals and may require a longer period of treatment. Some infections become systemic or cover a large area of the body therefore requiring oral treatment as well.

As with the use of any treatment either topical or oral there can be allergic response. All patients should be instructed to return to their health care provider if any of the symptoms of allergic reaction occur.

**Prevention** - Taking these steps may help to reduce the risk of getting a fungal infection:

- Dry the skin carefully after bathing, swimming or after heavy sweating. Wearing tight fitting clothing can keep moisture on the body setting up a perfect environment for fungal growth.
- Do not share personal items such as combs and brushes with someone who has a known fungal infection.
• The use of clothing or shoes that do not allow the body to breath should be avoided. If that is not possible make sure the areas are dried well after use.
• People with diabetes need to keep good control of their blood sugar

Information for this section was provided throughout the CDC website and The British United Provident Association limited. Photo’s from Dermnet.com

IX. Eye diseases

Pterygium
A fleshy thickening on the eye surface that slowly grows out from the edge of the white part of the eye near the nose and into the cornea; caused in part by sunlight, wind, and dust. Dark glasses may help calm irritation and slow the growth of a pterygium. It should be removed by surgery before it reaches the pupil. Unfortunately, after surgery a pterygium often grows back again.

Folk treatments using powdered shells do more harm than good. To help calm itching and burning you can try using cold compresses. Or use eye drops of chamomile (well boiled, then cooled, and without sugar).

Conjunctivitis – ‘Pink eye’
This infection causes redness, pus, and mild ‘burning’ in one or both eyes. Lids often stick together after sleep. It is especially common in children.
Treatment - First clean pus from the eyes with a clean cloth moistened with boiled water. Then put in antibiotic eye ointment. Pull down the lower lid and put a little bit of ointment inside. Putting ointment outside the eye does no good.

Caution: Do not touch the tube against the eye.

Prevention - Most conjunctivitis is very contagious. The infection is easily spread from one person to another. Do not let a child with pink eye play or sleep with others, or use the same towel. Wash hands after touching eyes.
Cataracts - The lens of the eye, behind the pupil, becomes cloudy, making the pupil look gray or white when you shine a light into it. Cataracts is common in older persons, but it also occurs, rarely, in babies. If a blink person with cataracts can still tell light from dark and notice movement, surgery may let him see again. However, he will need strong glasses afterward, which take time to get used to. Medicines do not help cataracts (Now sometimes during surgery an artificial lens is put inside the eye so that strong eyeglasses are not needed.)

X. Iron-deficiency Anemia

Anemia is a condition where red blood cells are not providing adequate oxygen to body tissues. There are many types and causes of anemia. Iron deficiency anemia is a decrease in the number of red cells in the blood caused by too little iron.

**Causes, incidence, and risk factors** - Iron deficiency anemia is the most common form of anemia. Approximately 20% of women, 50% of pregnant women, and 3% of men are iron deficient. Iron is an essential component of hemoglobin, the oxygen-carrying pigment in the blood. Iron is normally obtained through the food in your diet and by recycling iron from old red blood cells. Without it, the blood cannot carry oxygen effectively -- and oxygen is needed for the normal functioning of every cell in the body. The causes of iron deficiency are too little iron in the diet, poor absorption of iron by the body, and loss of blood (including from heavy menstrual bleeding). It can also be related to lead poisoning in children. Anemia develops slowly after the normal stores of iron have been depleted in the body and in the bone marrow. Women, in general, have smaller stores of iron than men and have increased loss through menstruation, placing them at higher risk for anemia than men. In men and postmenopausal women, anemia is usually caused by gastrointestinal blood losss associated with ulcers, the use of aspirin or nonsteroidal anti-inflammatory medications (NSAIDS), or certain types of cancer (esophagus, stomach, colon).

**Treatment** - The cause of the deficiency must be identified, particularly in older patients who are most susceptible to intestinal cancer. Oral iron supplements are available (ferrous sulfate). The best absorption of iron is on an empty stomach, but many people are unable to tolerate this and may need to take it with food. Milk and antacids may interfere with absorption of iron and should not be taken at the same time as iron supplements. Vitamin C can increase absorption and is essential in the production of hemoglobin. Supplemental iron is needed during pregnancy and lactation because normal dietary intake rarely supplies the required amount.

The hematocrit should return to normal after 2 months of iron therapy, but the iron should be continued for another 6 to 12 months to replenish the body's iron stores, which are contained mostly in the bone marrow. --Information from the NLM Web Site
XI. Pharmacology

Introduction
Pharmacology can be defined as the study of substances that interact with living systems through chemical processes, especially when regulator molecules act or inhibit normal processes. Medical pharmacology is the science of studying substances utilized to prevent, diagnostic, or treat different diseases. Use of different pharmacological agents has been employed for centuries. The first written registers are from 2500 years ago, found in Egypt and China, and include some drugs that are still used. For many centuries, tentative efforts to include rational experimental design methods in medicine failed, in the late 17th century observation and research were initiated, and thus in European countries such as Great Britain, the principles of pharmacotherapy started to be developed. During the 19th century in France, François Magendie and Claude Bernard developed experimental methods to understand physiology and pharmacology in animals. In the 20th century, the concept of rational therapeutic design (especially that of clinical evidence-based medicine and target-driven therapies) helped to more accurately evaluate the therapeutic properties of different medications.

The Basic Function of Drugs
In general terms, a drug can be defined as any substance that produces changes in the biological function of the body through chemical actions. In most cases, the drug molecule interacts with a specific molecule in the biological system, leading to a regulated action. Drugs can be synthesised by the body (i.e. hormones) or can be chemical substances that are not synthesised in the organism. In order to interact with the receptor, a drug molecule must have the proper size, electric charge, shape, and atomic composition. Moreover, drugs are often administered in a different location in the body than where they are needed to function. Thus, a drug must also have the necessary properties to be transported to the site of the desired action within the body. Finally, an effective drug is one that must be deactivated or excreted from the organism with reasonable speed, in order to produce an appropriated result.

Drug reactivity and drug-receptor Interactions
A drug interacts with receptors through chemical forces that produce chemical bonds or interactions. There are three different types of chemical bonds: covalent, electrostatic, and hydrophobic. Covalent bonds are very strong and, in many cases, irreversible. Electrostatic bonds are more common and can vary from relatively strong unions between molecules with ionic charge (ionic enlaces), to weaker hydrogen bonding. Hydrophobic bonds are extremely weak, and because of this property, tend to be very important in the interaction of drugs that are soluble in lipids.

Drug shape - A drug interacts with its receptor through a mechanism similar to a lock-and-key analogy.

Interaction drug-organism - There are two different types of interactions between the body and a drug. The process by which a drug acts in the body is called pharmacodynamics. The process by which the body acts on the drug is called pharmacokinetics.

The process of pharmacodynamics involves the study of how a drug and its potential receptor within the body interact. Pharmacokinetics is the study of how drugs are absorbed, distributed, metabolized, and excreted from the body.

Anti-inflammatory drugs
Inflammation is a normal response to tissue injury caused by trauma, chemicals, or microbiologic agents caused by the organism’s effort to destroy invading micro-organisms or repair tissue. However, inflammation is sometimes inappropriately triggered and can cause progressive tissue injury. Anti-inflammatory or immunosuppressive drugs may be required in order to modulate the organism’s immune response.
Anti-inflammatory drugs may be classified into several categories, including: Non-steroidal anti-inflammatory drugs (NSAIDs), non-narcotic analgesics, drugs for arthritis, and drugs for gout.

In this review, we are going to focus on those medications we use in Costa Rica during the ISL clinics. We will provide summaries of the drugs regarding to drug function, side effects, and potential interactions with other drugs.

**NSAIDs**

**Aspirin:** acetylsalicylic acid is a weak acid that is unique among the NSAIDs, in that it irreversibly acetylates cyclooxygenase. As a result, aspirin has anti-inflammatory, antipyretic, and analgesic effects.

**Indications:** Aspirin is indicated in rheumatoid arthritis, mild pain and fever, prevention of thrombosis, and acute rheumatic fever.

**Adverse reactions:** Some adverse effects are tinnitus, nausea, occult bleeding, prolonged bleeding time, rash, urticaria, and Reye’s syndrome.

**Interactions:** In combination with other NSAIDs, aspirin may increase the risk of GI bleeding.

**Contraindications:** Aspirin is contraindicated in patients hypersensitive to the drug and patients with bleeding disorders.

**Dosages:** Aspirin is available in tablets of 100 mg and 500 mg daily.

**Ibuprofen:** is a propionic acid derivative and has anti-inflammatory, analgesic, and antipyretic effects. It is a reversible inhibitor of cyclooxygenase.

**Indications:** Ibuprofen is indicated in rheumatoid arthritis, mild to moderate pain and dysmenorrhea, fever, and juvenile arthritis.

**Adverse reactions:** In some patients, ibuprofen produces headache, epigastric distress, prolonged bleeding time, pruritus, and rash.

**Interactions:** It may interact with thiazide diuretics, decreasing the effectiveness.

**Contraindications:** Patients with a history of allergic reactions to Ibuprofen, patients with nasal polyps, or in patients with past histories of adverse reactions to other NSAIDs.

**Dosages:** Available in tablets of 200 mg, 400 mg and 800 mg P.O p.r.n.

**Diclofenac:** is also an inhibitor of cyclooxygenase.

**Indications:** diclofenac is approved for long-term use in the treatment of rheumatoid arthritis, osteoarthritis, ankylosing spondilitis. It can also be used as an analgesic and in the treatment of primary dysmenorrhea.

**Adverse reactions:** Associated with anxiety, insomnia, tinnitus, abdominal pain and cramps, GI bleeding. May also cause photosensitivity reactions.

**Interactions:** If used with anticoagulants (such as warfarin or aspirin), may cause excessive bleeding.

**Dosages:** Diclofenac is available in tablets of 50 mg P.O b.i.d or t.i.d, p.r.n.

**Ketoprofen:** Similar to ibuprofen, Ketoprofen is also a propionic acid derivative. Its interactions, side effects and uses are the same as ibuprofen. It is available in capsules 25, 50 and 75 mg.

**Non-narcotic analgesics**

This group of medications, unlike the NSAIDs, has little or no anti-inflammatory activity. These drugs also have a therapeutic advantage, in that they do not cause physical dependence or tolerance.

**Acetaminophen:** Acts by inhibiting prostaglandin synthesis in the central nervous system (CNS). Acetaminophen is a suitable substitute for the analgesic and anti-pyretic effects of aspirin, and does not have the potential side effect of excessive bleeding. However, it does not have the same anti-inflammatory effect of aspirin.

**Indications:** It is indicated for mild pain and fever.

**Adverse reactions:** Acetaminophen may cause hemolytic anemia, liver damage, jaundice, hypoglycaemia, rash, or urticaria.
**Interactions:** If combined with alcohol, acetaminophen may increase the risk of hepatic damage. The combination of acetaminophen with barbiturates (such as carbamazepine and rifampicin) may reduce its therapeutic effects and enhance the hepatotoxic side effects.

**Dosages:** It is available in tablets 500 mg t.i.d or q.i.d for adults and oral suspension 120 mg/5 ml, for children (the children dosage is 15 mg/kg).

**Drugs used for arthritis**

Chloroquine: the mechanism of the anti-inflammatory activity for this drug is uncertain. Besides inhibiting nuclear acid synthesis, the chloroquines are known to stabilize lysosomal membrane and trap free radicals. In treating anti-inflammatory disorders, they are exclusively used for rheumatic arthritis that is unresponsive to NSAIDs. This drug has been shown to slow progression of erosive bone lesions and may induce remission. In high doses, chloroquines may cause serious side effects, such as gastrointestinal upset, pruritus, headaches, and visual disturbances. It should be used cautiously in patients with liver dysfunction.

**Antimicrobial drugs**

These drugs are effective in the treatment of infections because in general they are able to kill an invading micro-organism without causing harm to the host cells. However, selective toxicity is relative, rather than absolute.

To select the appropriate antibiotic agent, it is important to consider the organism's identity, its sensitivity to a particular drug, the safety of the agent, and the profile of the individual patient.

Ideally, antimicrobial treatment is started only when the identity of the pathogenic organism has been determined. Nevertheless, in conditions where the microbial agent cannot be identified, it is necessary to institute empiric therapy based on the site of infection and the patient's history.

Antimicrobial drugs can be classified as inhibitors of metabolism, inhibitors of cell wall synthesis, inhibitors of protein synthesis, inhibitors of nucleic acid function, urinary tract antiseptics, and anti/protozoal medications.

**Inhibitors of cell wall synthesis:** this group of medications interferes with the synthesis of the bacteria cell wall, a structure that is unique to bacteria. Within this group, the β-lactam antibiotics are found. This diverse group of antibiotics includes the penicillins, cephalosporins, carbapenems, and monobactams.

**Amoxicillin:** this derivative of penicillin is indicated in the treatment of systemic infections, acute and chronic UTI caused by susceptible strains of gram-positive and gram-negative organisms, uncomplicated gonorrhea, and in endocarditis prophylaxis for patients undergoing dental, gastrointestinal (GI), or genitor-urinary (GU) procedures.

**Adverse effects:** includes hypersensitive reactions, lethargy, nausea, vomiting, diarrhoea, anemia, and dizziness.

**Interactions:** may decrease efficacy of oral contraceptives and increase the risk of rash if combined with allopurinol.

**Dosages:** It is available in 500 mg capsules for adults and 125 mg/5 ml oral suspension for children. Dosage in adults is 250-500 mg q 8 h for at least 72 hours and in children 10-20 mg/kg q 8 h.

**Cephalexin:** this cephalosporin is indicated in the treatment of respiratory and urinary tract infections, skin and soft-tissue infections, and bone and joint infections caused by gram-negative bacteria.

**Adverse effects:** It may produce dizziness, headaches, pseudo-membranous colitis, nausea, anorexia, genital pruritus, arthralgia, and hypersensitivity reactions.

**Interactions:** if used together with amino glycosides, such as gentamicin of streptomycin, may increase the risk of nephrotoxicity.

**Dosages:** Cephalexin is available in 500 mg capsules and 125 mg/5 ml oral suspension. Adults: 500 mg q 6-8 h at least 72 hours, children 10-25 mg/kg q 6-12 h.
**Inhibitors of metabolism:** Folic acid is required for the synthesis RNA and DNA, which are necessary for cellular growth and replication. The sulpha drugs are inhibitors of folic acid synthesis. The antibacterial spectrum treated by this drug class includes both gram-positive and gram-negative bacteria, except *P. aeruginosa* and enterococcus.

**Sulfamethoxazole-trimethoprim:** this drug is indicated in urinary tract infections (UTIs), otitis media in patients with penicillin allergies, upper respiratory tract infections (URTI), and traveller’s diarrhea. Adverse reactions: Associated with allergic reactions, headache, nausea, vomiting, muscle weakness, and *Steven-Johnson syndrome*.  
**Interactions:** May increase the serum levels of phenytoin (anticonvulsant drug) by decreasing its metabolism.

**Dosages:** TMT-SMX is available in 80/400 mg tablets and 16/80 mg/ml oral suspension. Adults' dosage is 1 tab q 12 h at least 72 hours, children dosage 1.5-3 mg/kg q 12 h P.O.

**Inhibitors of protein synthesis:** these types of antibiotics exert their effects by targeting the bacterial ribosome. The drugs are thought to exert their bacteriostatic effects by binding to the 30S and 50S ribosomal subunits of microorganisms, thus inhibiting protein synthesis.

**Doxycycline:** this tetracycline is indicated in infections caused by susceptible gram-positive and gram-negative bacteria (including those that produce Lyme disease), inhalation, and GI and oropharyngeal anthrax, gonorrhea in patients allergic to penicillin, primary or secondary syphilis in patients allergic to penicillin, uncomplicated UTI, and for prevention of malaria.  
**Adverse effects:** The most dangerous side effect is intracranial hypertension, but this is not a common side effect. Other adverse reactions include: anorexia, dysphagia, epigastric distress, bone growth retardation, photosensitivity, and discoloration of teeth.

**Interactions:** Antacids decrease the absorption of these antibiotics, as well as the absorption of iron. Doxycycline may decrease the effectiveness of oral contraceptives. It also may cause photosensitivity.

**Dosages:** The available forms are 50 and 100 mg tablets which dosage is 1 tab q 12 h P.O

**Quinolones:** these medications enter the cell by passive diffusion through water-filled protein channels, called porins, in the outer membrane of the bacteria. Once in the intracellular environment, they inhibit the replication of DNA.

**Ciprofloxacin:** this fluoroquinolone is indicated for mild to moderate UTIs caused by *Enterobacteracea*, mild to moderate bone and joint infections, severe respiratory tract infections, chronic bacterial prostatitis, and as empirical therapy in febrile neutropenic patients.  
**Adverse effects:** May cause headaches, fatigue, insomnia, nausea, diarrhea, dyspepsia, joint inflammation, and *Steven Johnson syndrome*. **Cipro should not be given to children or pregnant women.**

**Interactions:** In combination with NSAIDs, may increase the stimulation of the CNS

**Dosages:** Ciprofloxacin is available in 500 mg tablets, dosage 1 q 12 hours P.O

**Metronidazole:** This antibiotic is part of the nitroimidazole family, but is also useful as an antiprotozoal drug. It has a strong anibiotic activity against most anaerobic gram-negative bacteria, and some antiprotozoal effects against organisms like *Lamblia intestinalis*, *Entamoeba histolytica* and *Trichomona vaginalis*.  
**Indications:** Used in the treatment of abdominal and pelvic infections, as well as brain abscess caused by anaerobic bacteria.

**Adverse effects:** It may produce headaches, vertigo, insomnia, abdominal cramping pain, darkened urine, rash, and **transient leukopenia**.  
**Interactions:** If combined with cimetidine, may produce hepatic toxicity. Use in combination with alcohol can produce a disulfiram-like reaction, including nausea, vomiting, headache, and cramps and flushing.

**Dosages:** Metronidazole is available in 250 and 500 mg capsules and 125 mg/5 ml or 250 mg/5 ml oral suspension
Anthelmintic drugs
Anthelmintic drugs: Target metabolic processes found in parasites.

**Mebendazole**: this anthelmintic acts by binding to and interfering with the synthesis of the parasite’s microtubules, as well as decreasing uptake of glucose by the parasite.
Indications: Mebendazole is indicated in the treatment of pinworms, roundworms, whipworms, and hookworms.
Adverse effects: Though rare, mebendazole may cause seizures. The most common adverse effect reported in patients is transient abdominal pain.
Interactions: If combined with cimetidine, the plasma levels of mebendazole may increase.

**Dosages**: Dosage is 100 mg P.O as a single dosage, but must be repeated if parasitic infection persists.

**Albendazole**: this is a wide-spectrum anthelmintic that acts by inhibiting the microtubules synthesis in nematodes, which decreases the glucose uptake and at produces death in the parasite. As a result of these effects, the parasites die within a few days of treatment and will be expelled from GI tract. Albendazole also acts to kill larvae and ovum produced by the parasites.
Indications: This medication is used for the treatment of nematodes, cestodes and trematodes.
Adverse effects: epigastric discomfort, nausea, dizziness, insomnia, and headaches are the most common side effects, though only 6% of patients appear to experience these symptoms.
Interactions: see Mebendazole.

**Dosages**: Albendazole is available in 200 mg and 400 mg tablets. Dosage is 400 mg as a single dose in mild infestations but usually more than 1 dosage is needed, depending on the diagnosis, between 3 days and 3 month of treatment. Dosage in children is 15 mg/kg.

Avoid using anthelmintic drugs during pregnancy, children under the age of 2, or patients with peptic ulcers.

Antifungal drugs
Mycotic infections, infections caused by fungi, are quite common and mainly produce superficial infections of the skin in the general population. There are several types of fungi are capable of invading internal organs and producing systemic mycotic infections, which are often life-threatening. Nevertheless, because systemic fungal infections must generally be treated in the hospital under close supervision, this section of the review will only describe topical or oral antifungal medications used in primary care settings.

**Drugs for superficial Mycotic infections**: fungi that cause skin infections are called dermatophytes, and include the tinea family of infections. In addition to tinea, other fungi that cause skin infections include epidermophytum.

**Griseofulvin**: arrests fungal activity by disrupting the mitotic spindle structure within the fungal cells. Therapy must be continued for weeks or months, until normal tissue replaces the infected tissue.
Adverse effects: Symptoms include headaches, nausea, vomiting, and flatulence, and are often found during the initial stages of treatment. Hypersensitivity reactions can also occur.
Interactions: Increases the effectiveness of cumarin anticoagulants. If taken with alcohol, may cause tachycardia, diaphoresis, and flushing.

**Dosages**: The available form is 500 mg tablets, which dosage is 500 mg up to 1 g daily and the duration of therapy is 2 to 8 weeks.

**Nystatin**: is a polyene antibiotic. In general terms, nystatin produces pores in the bacterial cell wall, producing the leakage of electrolytes and small molecules from the cell into the external milieu, which then results in cell death.
Indications: Use is restricted to topical treatment because of its systemic toxicity. It is indicated for intestinal, oral, and vaginal candidiasis.
Dosages: Nystatin is available in vaginal suppositories 100,000 units. For vaginal candidiasis, 1 vaginal tablet should be inserted into vagina h.s for 5 days.

Other topical agents: Miconazole and clotrimazole are topically active drugs that interfere with fungal DNA replication by binding to sterols within the fungal cell membrane. This binding increases permeability of the membrane, resulting in the leakage of cell nutrients and cell death. Some side effects are rash, urticaria, peeling, blistering, and dyspareunia.

Antihistamines
Allergic reactions are produced by the liberation of cell mediators stimulated by IgE, created in response to a previous exposure to the same allergen (such as dust, pollen, medications, food). Mediators released by the mast cells include histamine, leukotriens, and chemotactic factors.

Chlorpheniramine: is a drug that competes with histamine for H1-receptor sites on effector cells. In preventing the binding of histamine to its receptor, chlorpheniramine mitigates the allergic response. However, it is only capable of preventing, not reversing, the histamine-mediated responses.

Indications: Indicated in rhinitis and allergy symptoms.
Adverse effects: Some adverse reactions include sedation, drowsiness, hypotension, epigastric distress, and urticaria.
Interactions: If administered with CNS depressants or alcohol, may increase sedation.

Dosages: It is available in 4 mg tablets that are administered 1 q 4-6 h P.O. Children dosage is 0.1 mg/Kg.

Brompheniramine: competes with H1 receptor sites on effector cells. It prevents, but does not reverse, the histamine-mediated responses to allergen exposure.

Indications: indicated in the treatment of rhinitis and allergy symptoms.
Adverse effects: may cause dizziness, tremors, insomnia, hypotension, anorexia, nausea, and urine retention.

Dosages: It is available in elixir 2 mg/5ml. Children dosage is 0.1 mg/kg q 6-8 hours.

Cough and asthma suppressants
Drugs may reach the lungs through inhalation, oral, or parenteral routes. Administration depends on how fast the therapy must be initiated. Inhalation is the best way to deliver drugs to the respiratory system, as it is direct, rapid, very effective, and produces the least number of systemic side effects.

Drugs to treat asthma
Asthma is a chronic disease characterized by periods of acute bronchoconstriction, resulting in cough, chest tightness, wheezing and rapid respiration.
Salbutamol: is a β-2 agonist that relaxes bronchial, uterine, and vascular smooth muscles.

Indications: salbutamol, also known as albuterol, is indicated in the treatment or prevention of reversible obstructive airway disease (asthma).
Adverse reactions: may produce tremor, nervousness, dizziness, hyperactivity, and tachycardia, dry, irritated nasal passages, or hypersensitivity reactions.
Interactions: If used with CNS stimulants, it may increase the effects.

Dosages: salbutamol is available in aerosol inhalators 90 mcg/metered spray. Adults and children 4 and older may do 1 or 2 inhalations q 4-6 hours.

Ipratropium bromide: is an anticholinergic agent that inhibits vagally mediated reflexes by antagonizing acetylcholine at muscarinic receptors on bronchial smooth muscle.

Indications: is indicated in treatment of bronchospasm in chronic bronchitis and emphysema. It is also indicated in asthmatic patients who are intolerant to adrenergic agents.
Adverse effects: may produce dizziness, headache, blurred vision, GI distress, dry mouth, and hypersensitivity reactions.
**Dosages:** It is available in 18 mcg/metered spray. Usually adults and children age 6 and older must do 2 inhalations q.i.d. Some patients may take additional inhalations p.r.n.

**Cough suppressants**

**Dextromethorphan:** an antitussive (anti-cough medication) that suppresses the cough reflex by direct action on the cough center in the medulla of the brain.

**Indications:** indicated in the treatment of non-productive cough.

**Adverse effects:** may cause drowsiness, dizziness, and stomach pain.

**Dosages:** available in syrup 5 mg/5 ml. Adults and children aged 12 and older may take 10 to 20 mg q 4-6 hours. Children dosage is 0.5 mg/kg.

**Guaifenesin:** increases production of respiratory tract fluids to help liquefy and reduce the viscosity of persistent, respiratory secretions.

**Indications:** indicated as an expectorant in the treatment of productive cough.

**Adverse effects:** may produce dizziness, headache, and rash.

**Drugs used to treat gastrointestinal disorders**

The secretion of gastric acid by parietal cells in the gastric mucosa is controlled by acetylcholine, histamine, prostaglandins E2 and I2, and gastrin. These chemical mediators bind a receptor on the surface of the parietal cells, activating the H⁺/K⁺ ATPase proton pump that secretes HCl (hydrochloric acid) into the lumen of the stomach. Though the pathogenesis of peptic ulcer disease is not well understood, there are three major factors associated with the illness: *H. pylori* infection, increased hydrochloric acid secretion, and inadequate mucosa defence against the presence of gastric acid.

**Cimetidine:** competitively inhibits action of histamine on the H2 receptors on the surface of parietal cells, thus decreasing gastric acid secretion.

**Indications:** indicated in the short-term treatment of duodenal ulcers, acute stress ulcers, and gastroesophageal reflux disease (heartburn).

**Adverse effects:** may cause dizziness, headache, mild and transient diarrhea, and hypersensitivity reactions.

**Interactions:** Antacids interfere with cimetidine absorption. If cimetidine is used with alcohol, the blood concentration of the drug may increase.

**Dosages:** Available in 300 mg tablets. Dosage in adults is 300-600 mg b.i.d P.O

**Ranitidine:** same mechanism as cimetidine.

Indications and interactions are similar to those described for cimetidine.

**Dosages:** ranitidine is available in 150 mg tablets. Adult dosage is 150 b.i.d or 300 mg h.s P.O

**Calcium carbonate:** an antacid that reduces total acid load in the GI tract, elevates gastric pH to reduce pepsin activity, strengthens the gastric mucosa barrier, and increases esophageal sphincter tone.

**Indications:** indicated as a calcium supplement and to treat overproduction of gastric acid.

**Adverse effects:** may be associated with headaches, irritability, nausea, and altered phosphate levels.

**Dosages:** in adults 350 mg to 1.5 g P.O 1 hour after meal or p.r.n.

**Drugs used to treat parasitic infections of the skin**

**Benzyl Benzoate:** used to treat scabies and is included in lice shampoos. This compound is used topically and kills the scabies mite by limiting the animal’s ability to take in oxygen, causing death by asphyxiation. This drug also kills the eggs of the parasite by destroying the outer membrane.

**Dosage:** Benzyl Benzoate is an extremely toxic substance and must be used with care.

For adults: Apply topically to the entire body, except the face, at night before going to bed for five days. Repeat the treatment again after a week.
For children: **BENZYL BENZOATE IN ITS NORMAL PRESENTATION OF 5% SOLUTION SHOULD NOT BE USED ON CHILDREN UNDER TWO YEARS OF AGE BECAUSE OF ITS HIGH TOXICITY.**
For small children it must be diluted, one part Benzyl Benzoate to one part water, and is applied topically for 5 days in the same manner.

***It is very important to educate patients about the necessity of washing all bed linens, clothing and pillows in boiling water. The scabies mite can live for a several days outside of the human body in beds, couches, linens, etc.

**CROTAMIN:** used to treat scabies. This drug is applied in the same way as benzyl benzoate. However, crotamin is less toxic than benzyl benzoate, and is thus preferred for use with small children because it can be applied without dilution.

**QWELL, QUITOSO, PIOJINA, etc.:** These shampoo brands are used in the treatment of lice. The active ingredient in all of these preparations is Benzyl Benzoate or permectrine. The shampoos are applied topically to the affected areas for 3-10 days consecutively, according to the brand and its active ingredient.

**XII. Home Cures and Popular Beliefs**

Everywhere on earth people use home remedies. In some places, the older or traditional ways of healing have been passed down from parents to children for hundreds of years. Many home remedies have great value. Others have less. And some may be risky or harmful. Home remedies, like modern medicines, must be used with caution.

<table>
<thead>
<tr>
<th>Try to do no harm.</th>
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<td>Only use home remedies if you are sure they are safe and know exactly how to use them.</td>
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*Home Cures that help* - For many sicknesses, time-tested home remedies work as well as modern medicines – or even better. They are often cheaper. And in some cases they are safer. For example, many of the herbal teas people use for home treatment of coughs and colds do more good and cause fewer problems than cough syrups and strong medicines some doctors prescribe.

Also, the “rice water”, teas, or sweetened drinks that many mothers give to babies with diarrhea are often safer and do more good than any modern medicine. What matters most is that a baby with diarrhea get plenty of liquids.

*The Limitations of Home Remedies* - Some diseases are helped by home remedies. Others can be treated better with modern medicine. This is true for most serious infections. Sicknesses like pneumonia, tetanus, typhoid, tuberculosis, appendicitis, diseases caused by sexual contact, and fever after childbirth should be treated with modern medicines as soon as possible. For these diseases, do not lose time trying to treat them first with home remedies only.

It is sometimes hard to be sure which home remedies work well and which do not. More careful studies are needed. For this reason:

| It is often safer to treat very serious illnesses with modern medicines – following the advice of a health worker if possible. |
Old Ways and New - Some modern ways of meeting health needs work better than old ones. But at times the older, traditional ways are best. For example, traditional ways of caring for children or old people are often kinder and work better than some newer, less personal ways.

Not many years ago everyone thought that mother’s milk was the best food for a young baby. They were right! Then the big companies that make canned and artificial milk began to tell mothers that bottle feeding was better. This is not true, but many mothers believed them and started to bottle feed their babies. As a result, thousands of babies have suffered and died needlessly from infection or hunger. For these reasons, breast is best.

Beliefs that can make people well - Some home remedies have a direct effect on the body. Others seem to work only because people believe in them. The healing power of belief can be very strong. For example: I once saw a man who suffered from a very bad headache. To cure him, a woman gave him a small piece of yam, or sweet potato. She told him it was a strong painkiller. He believed her – and the pain went away quickly. It was his faith in her treatment, and not the yam itself that made him feel better.

Many home remedies work in this way. They help largely because people have faith in them. For this reason, they are especially useful to cure illnesses that are partly in people’s minds, or those caused in part by a person’s beliefs, worries, or fears.

Included in this group of sicknesses are “bewitchment or hexing”, unreasonable or hysterical fear, uncertain “aches or pains” (especially in persons going through stressful times, such as teenage girls or older women), and anxiety or nervous worry. Also included are some cases of asthma, hiccups, indigestion, stomach ulcers, migraine headaches, and even warts.

For all of these problems, the manner of ‘touch’ of the healer can be very important. What it often comes down to is showing you care, helping the sick person believe he will get well, or simply helping him relax.

Sometimes a person’s belief in a remedy can help with problems that have completely physical causes. For example: Mexican villagers have the following home cures for poisonous snakebite…1) to use ‘guaco’ leaves 2) to bite the snake 3) to apply tobacco 4) to apply the skin of a poisonous lizard 5) to smear the snake’s bile on the bite.

In other lands people have their own snakebite remedies – often many different ones. As far as we know, none of these home remedies has any direct effect against snake poison. The person who says that a home remedy kept a snake’s poison from harming him at all was probably bitten by a non-poisonous snake!

Yet any of these home remedies may do some good if the person believes in it. If it makes him less afraid, his pulse will slow down, he will move and tremble less, and as a result, the poison will spread through his body more slowly. So there is less danger! But the benefit of these home remedies for snakebite is limited. In spite of their common use, many people still become very ill or die. As far as we know:

No home cure for poisonous bites (whether from snakes, scorpions, spiders, or other poisonous animals) has much effect beyond that of the healing power of belief.
For snakebite is it usually better to use modern treatment. Be prepared: obtain ‘antivenoms’ or ‘serums’ for poisonous bites – before you need them. Do not wait until it is too late.

Beliefs that can make people sick - The power of belief can help heal people. But it can also harm them. If a person believes strongly enough that something will hurt him, his own fear can make him sick. For example: Once I was called to see a woman who had just had a miscarriage and was still bleeding a little. There was an orange tree near her house. So I suggested she drink a glass of orange juice. (Oranges have vitamin C which helps strengthen blood vessels). She drank it – even though she was afraid it would harm her. Her fear was so great that soon she became very ill. I examined her, but could find nothing physically wrong. I tried to comfort her. But she said she was going to die. At last I gave her an injection of distilled (completely pure) water. Distilled water has no medical effect. But since she had great faith in injections, she quickly got better. Actually, the juice did not harm her. What harmed her was her belief that it would make her sick. And what made her well was her faith in injections!

In this same way, many persons go on believing false ideas about witchcraft, injections, dies, and many other things. Much needless suffering is the result. Perhaps, in a way, I had helped this woman. But the more I thought about it, the more I realized I had also wronged her. I had led her to believe things that were not true.

I wanted to set this right. So a few days later, when she was completely well. I went to her home and apologized for what I had done. I tried to help her understand that not the orange juice, but her fear had made her so sick. And that not the injection of water, but her freedom from fear had helped her get well.

By understanding the truth about the orange, the injection, and the tricks of her own mind, perhaps this woman and her family will become freer from fear and better able to care for their health in the future. For health is closely related to understanding and freedom from fear.

Many things do harm only because people believe they are harmful

Witchcraft – Black Magic – And the Evil Eye - If a person believes strongly enough that someone has the power to harm him, he may actually become ill. Anyone who believes he is bewitched or has been given the evil eye is really the victim of his own fears.

A ‘witch’ has no power over other people, except for her ability to make them believe that she has. For this reason: It is impossible to bewitch a person who does not believe in witchcraft. Some people think that they are ‘bewitched’ when they have strange or frightening illnesses (such as tumors of the genitals or cirrhosis of the liver). Such sicknesses have nothing to do with witchcraft or black magic. Their causes are natural. Do not waste your money at ‘magic centers’ that claim to cure witchcraft. And do not seek revenge against a witch, because it will not solve anything. If you are seriously ill, go for medical help.

List of “False Folk Beliefs”
These examples are from the mountains of Mexico, the area that I know best. Perhaps some of the beliefs of your people are similar. Think about ways to learn which beliefs in your area lead to better health and which do not.

Example: When people think someone is bewitched, that he will get well if his relatives harm or kill the witch. False! No one is ever helped by harming someone else.

--Is it true that when the ‘soft spot’ on top of a baby’s head sinks inward this means the baby will die of diarrhea unless he gets special treatment? This is often true. The ‘soft spot’ sinks because the baby has lost too much liquid. Unless he gets more liquid soon, he may die.

--Is it true that if the light of the eclipsing moon falls on a pregnant mother, her child will be born deformed or retarded? This is not true! But children may be born retarded, deaf, or deformed if the mother does not use iodized salt, if she takes certain medicines, or for other reasons.
Is it true that mothers should give birth in a darkened room? It is true that soft light is easier on the eyes of both the mother and the newborn child. But there should be enough light for the midwife to see what she is doing.

Is it true that a newborn baby should not be bathed until the cord falls off? True! The stump of the cord should be kept dry until it falls off. But the baby can be gently cleaned with a clean, soft, damp cloth.

How many days after giving birth should a mother wait before she bathes? A mother should wash with warm water the day after giving birth. The custom of not bathing for weeks following childbirth can lead to infections.

Is it true that traditional breast feeding is better than ‘modern’ bottle feeding? TRUE! Breast milk is better food and also helps protect the baby against infection.

What foods should women avoid in the first few weeks after childbirth? In the weeks following childbirth, women should not avoid any nutritious foods. Instead, they should eat plenty of fruit, vegetables, meat, milk, eggs, whole grains, and beans.

Is it a good idea to bathe a sick person, or will it do him harm? It is a good idea. Sick people should be bathed in warm water every day.

Is it true that oranges, guavas, and other fruits are harmful when one has a cold or fever? NO! All fruits and juices are helpful when one has a cold or fever. They do not cause congestion or harm of any kind.

Is it true that when a person has a high fever, he should be wrapped up so that the air will not harm him? NO! When a person has a high fever, take off all covers and clothing. Let the air reach his body. This will help the fever go down.

Is it true that tea made from willow bark will help bring fever down and stop pain? True. It helps. Willow bark has a natural medicine in it very much like aspirin.

Sunken Fontanel or Sofá Spot
The fontanel in the soft spot on the top of a newborn baby’s head. It is where the bones of his skull have not formed completely. Normally it takes a year to a year and a half for the soft spot to close completely. Mothers in different lands realize that when the soft spot sinks inward their babies are in danger. They have many beliefs to explain this. In Latin America, mothers think the baby’s brains have slipped downward. They try to correct this by sucking on the soft spot, by pushing up on the roof of the mouth, or by holding the baby upside down and slapping his feet. This does not help because... A sunken soft spot is really caused by dehydration.

This means the child is losing more liquid than he is drinking. He is too dry – usually because he has diarrhea, or diarrhea with vomiting. Treatment:
1. Give the child plenty of liquid: Rehydration Drink, breast milk, or boiled water.
2. If necessary, treat the causes of the diarrhea and vomiting. For most diarrheas, medicine is not needed, and may do more harm than good.

Note: If the soft spot is swollen or bulges upward, this may be a sign of meningitis. Begin treatment at once and medical help.

Ways to tell whether a home remedy works or not
Because a lot of people use a home cure does not necessarily mean it works well or is safe. It is often hard to know which remedies are helpful and which may be harmful. Careful study is needed to be sure. Here are four rules to help tell which remedies are least likely to work, or are dangerous. (Examples are from Mexican villages).

1. THE MORE REMEDIES THERE ARE FOR ANY ONE ILLNESS, THE LESS LIKELY IT IS THAT ANY OF THEM WORKS. For example, in rural Mexico there are many home remedies for goiter, none of which does any real good. Here are some of them:
   1) to place a crab on the goiter 2) to rub the goiter with the hand of a dead child 3) to smear the brains of a vulture on the goiter 4) to smear human feces on the goiter.
Not one of these many remedies works. If is did, the others would not be needed. When a sickness has just one popular cure, it is more likely to be a good one. For prevention and treatment of goiter use iodized salt.

2. FOUL OR DISGUSTING REMEDIES ARE NOT LIKELY TO HELP – AND ARE OFTEN HARMFUL. For example: 1) the idea that leprosy can be cured by a drink made from rotting snakes 2) the idea that syphilis can be cured by eating a vulture. These two remedies do not help at all. The first one can cause dangerous infections. Belief in remedies like these sometimes causes delay in getting proper medical care.

3. REMEDIES THAT USE ANIMAL OR HUMAN WASTE DO NO GOOD AND CAN CAUSE DANGEROUS INFECTIOUS. NEVER USE THEM. Examples: 1) Putting human feces around the eye does not cure blurred vision and can cause infections 2) Smearing cow dung on the head to fight ringworm can cause tetanus and other dangerous infections. Also, the droppings of rabbits or other animals do not help heal burns. To use them is very dangerous. Cow dung held in the hand, cannot help control fits. Teas made from human, pig, or any other animal feces do not cure anything. They can make people sicker. Never put feces on the navel of a newborn baby. This can cause tetanus.

XIII. Medicinal Plants

Many plants have curative powers. Some of the best modern medicines are made from wild herbs. Nevertheless, not all curative herbs people use have medical value...and those that have are sometimes used the wrong way. Try to learn about the herbs in your area and find out which ones are Worthwhile. CAUTION! Some medicinal herbs are very poisonous if taken in more than the recommended dose. For this reason it is often safer to use modern medicine, since the dosage is easier to control. Here are a few examples of plants that can be useful if used correctly.

**ANGEL’S TRUMPET**: The leaves of this and certain other members of the nightshade family contain a drug that helps to calm intestinal cramps, stomach-aches, and even gallbladder pain. Grind up 1 or 2 leaves of Angel’s Trumpet and soak them for a day in 7 tablespoons (100 ml) of water. Dosage: Between 10 and 15 drops every 4 hours (adults only) WARNING! Angel’s Trumpet is very poisonous if you take more than the recommended dose.

**CORN SILK**: (the tassels or ‘silk’ from an ear of maize) A tea made from corn silk makes a person pass more urine. This can help reduce swelling of the feet especially in pregnant women. Boil a large handful of corn silk in water and drink 1 or 2 glasses. It is not dangerous.

**GARLIC**: A drink made from garlic can often get rid of pinworms. Chop finely, or crush, 4 cloves of garlic and mix with 1 glass of liquid (water, juice, or milk). Dosage: Drink 1 glass daily for 3 weeks.

**CARDON CACTUS**: (Pachycerius pectin-aboriginum) Cactus juice can be used to clean wounds when there is no boiled water and no way to get any. Cardon cactus also helps stop a wound from bleeding, because the juice makes the cut blood vessels squeeze shut. Cut a piece of the cactus with a clean knife and press it firmly against the wound. When the bleeding is under control, tie a piece of the cactus to the wound with a strip of cloth. After 2 or 3 hours, take off the cactus and clean the wound with boiled water and soap.

**ALOE VERA**: (Sabila) Aloe vera can be used to treat minor burns and wounds. The thick, slimy juice inside the plant calms pain and itching, aids healing, and helps prevent infection. Cut off a piece of the plant, peel back the outer layer, and apply the fleshy leaf or juice directly to the burn or wound. Aloe can also help treat stomach ulcers and gastritis. Chop the spongy leaves into small pieces, soak them in water overnight, and then drink one glass of the slimy, bitter liquid every 2 hours.

**PAPAYA**: Ripe papayas are rich in vitamins and also aid digestion. Eating them is especially helpful for weak or old people who complain of upset stomach when they eat meat, chicken, or eggs. Papaya
makes these foods easier to digest. Papaya can also help get rid of intestinal worms, although modern medicines often work better. Collect 3 or 4 teaspoons (15-20 ml.) of the ‘milk’ that comes out when the green fruit or trunk of the tree is cut. Mix this with an equal amount of sugar or honey and stir it into a cup of hot water. If possible, drink along with a laxative. Or, dry and crush to a powder the papaya seeds. Take 3 teaspoon mixed with 1 glass water or some honey 3 times a day for 7 days. Papayas can also be used for treating pressure sores. The fruit contains chemicals that help soften and make dead flesh easier to remove. First clean and wash out a pressure sore that has dead flesh in it. Then soak a sterile cloth or gauze with ‘milk’ from the trunk or green fruit of a papaya plant and pack this into the sore. Repeat cleaning and repacking 3 times a day.