**Dental Caries & Periodental Disease***Dental caries*-progressive decalcification of the enamel and dentin of a tooth
*Periodental Disease*- this is a gum disease related to dysbiosis of the mouth where the bacteria make the gums inflame and start eating away at the tooth surface below the gum and as well as the bones on which the teeth are anchored
Etiology: dental infection, oral dysbiosis, acid by products of bacterial metabolism, mineral deficiencies
Signs and Symptoms: sometimes no symptoms, pain, sensitivity to pressure or temperature, gums may appear swollen, gums may bleed (especially when flossing or brushing)
Pathophysiology: sugar and starches feed the bacteria, the bacteria combined with saccharides and salivary proteins to create plaque. The plaque becomes a target for the acid production. This leads to demineralization, cavities and receding gums. The microbes can also create abscesses in the tooth or any tissues that support the tooth.
Recommendations: p. 141-142
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**Herpes Simplex**
Virus that create vesicular eruptions on the skin
Etiology: human contact with it, can be transferred to baby through the mother’s birth canal
Signs and Symptoms: multiple small blisters on the lips, tingling on the lips
Pathophysiology: after an outbreak the virus remains dormant in the nerve ganglion. It often reoccurs during acute illness or in response to heat or stress.
Recommendations: p. 143 & p. 144
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**Hyperchlorhydria**Excess hydrochloric acid in the stomach
Etiology: stress stimulates the sympathetic nervous system which shuts down gastric acid secretion, as a result in some people, the vagus nerve (a parasympathetic cranial nerve) responds to stress by producing more HCL/any irritation of the gastric mucosa releases histamine and histamine stimulates receptors on the parietal cells which stimulate the release of HCL ie: H.pylori/ non-steroidal anti inflammatories/pancreatic tumour or hypercalcemia also can stimulate the gastrin production and therefore increase acid secretion
Signs and Symptoms: heartburn
Pathophysiology: depend on the etiology (see above)
Recommendations: p.145 & 146
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**Reflux Esophagitis and GERD**Acid from the stomach flows up the esophagus causing discomfort and sometimes damage to the esophagus.
Etiology: haital hernia, weakened cardiac sphincter, hypo and hyper chlorhydria, food sensitivities, dysbiosis of the stomach including H.Pylori and gastritis as well as physical stresses like obesity, overeating and pregnancy.
Signs and Symptoms: heart burn, indigestion, non cardiac pain radiating to the neck, jaw and arms/ occasionally asthma, cough, hoarseness, difficulty in swallowing or nocturnal regurgitation
Pathophysiology: depend on the etiology (see above), dysbiosis in the stomach can let microbes get out of hand and they produce gas and acidic by products (often the suffer will take antacids which will further suppress HCL and thus furthering microbial growth)
Recommendations: p. 148
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**Hypochlorhydria**Too little HCL
Etiology: eating on the run, acute or chronic stress, inefficient nutrients to keep parietal cells healthy will decrease HCL production (stress, nutrient poor diet, inability to digest and absorb nutrients from overworked pancreas compromising digestion or inflammatory mucus in the way compromising absorption), excessive protein stimulating and then exhausting HCL secretion and then leading to underactive stomach.
Signs and Symptoms: many signs see p. 150 in text
Pathophysiology: microbial overgrowth causing dysbiosis, fermentation and putrefaction in the stomach (with gas, bloating and acid reflux), protein deficiency, poor overall digestion, mineral and B12 deficiency
Recommendations: p. 151-153
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**Peptic Ulcer**
Crater-like erosion most often in the stomach mucosa (but can be anywhere in the gut)
Etiology: too much acid production and not enough production of protective mucous and insufficient bicarbonate secretion to neutralize the acid, or not enough acid with resulting in decreased mucus protection, reflux of bile causing ulceration, non steroidal anti-inflammatories irritate the gut/H.Pylor which increases acid production and reduces protective mucus
Signs and Symptoms: gnawing, burning epigastric pain that can begin within a half hour of eating and not relieved by eating
Pathophysiology: mucosal damage
Recommendations: p. 155
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**Dysbiosis**Microbial imbalance
Etiology:
1) *Death of good bacteria* antibiotics, diarrhea, starvation or other illness characterized by depleted nutritional status, compromised immunity and overgrowth of pathogenic bacteria, chlorine and steroids.
2) *Microbial overgrowth* due to overeating, compromised digestion, steroids
Signs and Symptoms: craving sugar and sweets, constipation, diarrhea, bloating, abdominal pain, fatigue, itchy skin, rashes, feeling vaguely unwell, re occuring vaginal, prostate or urinary infections, spaced out, difficulty concentrating, poor memory, PMS, menstrual irregularities, or sexual dysfunction
Pathophysiology: leaky gut, systemic Candida infection, food sensitivities, chronic inflammation causing the release of cortisol to bring down the inflammation and depleted the adrenal glands and increased susceptibility to degenerative diseases like bone thinning, type 2 diabetes and cardiovascular disease, overloaded liver making up for poor digestion and having to deal with toxic metabolic by products of pathogenic bacterial overgrowth
Recommendations: p. 157-178
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**Irritable Bowel Syndrome**Motility disorder of the entire GI tract (not autoimmune related) creating GI symptoms
Etiology: low fibre intake combined with poor digestion and lots of gas producing microbes in the gut resulting in a hypersensitive gut that doesn’t do well with digestive overload and also alternates between constipation and diarrhea.
Signs and Symptoms: abdominal pain and distension, flatulence, diarrhea, constipation and anxiety
Pathophysiology: mucus in the stool indicating inflammation, poor digestion indicates dysbiosis can progress to IBD which is a more severe form of degeneration, poor peristalsis due to lack of fiber narrowing of the gut, stress further causes narrowing because the muscles of the gut tighten in response to stress, transit time is lengthened and elimination delayed which creates of areas to dilate with fecal impaction (diverticuli or ‘blow outs’ of mucosa can occur and become inflamed, aggravated by smoking and caffeine because just like stress, stimulants shunt blood away from the gut and slow down digestion
Recommendations: p. 180 & 181
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**Celiac Disease**Malabsorption, weight loss and diarrhea resulting from immunological intolerance to dietary gluten containing foods, especially wheat, rye and barley
Etiology: inability to digest gluten causing damage to mucosal cells which become inflamed and swollen (the villi of the intestine flatten and break off severely compromising all digestion). Some research suggests that it is the dissacharide fragments attached to the gliadin that are causing the damage and the real underlying problem is failure to digest dissacharides-which explains why many don’t improve significantly by JUST eliminating gluten.
Signs and Symptoms: frequent diarrhea, occasional constipation, pale stools (greasy, foul smelling or even frothy due to fermentation), digestive upsets, bloating, gas, abdominal pain, irritable bowel, colitis, headache, allergies, schizophrenia, or other psychological manifestations
*Pathophysiology*: distended abdomens, thin limbs and slow growth in children, increased incidence of diabetes, arthritis, osteoporosis, calcification of the brain, epilepsy and thyroid disease
Recommendations: p. 182 & 183
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**Inflammatory Bowel Diseases (Crohn’s Disease and Ulcerative Colitis)**Inflamed bowel with malabsorption, severe dysbiosis and so much irritation and weakening of the immune system that an autoimmune condition has developed.
*Crohn’s*-inflammation of lower ileum, colon and sometimes other parts of the small intestine
*Ulcerative colitis*- chronic inflammation and ulceration of the colon mucosa only with bloody diarrhea
Etiology: Cause is unknown but possibly genetics, food sensitivities, infectious agents, environmental toxicity and dietary factors.
Signs and Symptoms: abdominal pain, bloody, watery diarrhea with mucus and pus, fever, weight loss, increased urgency to defecate sometimes up to twenty times a day, rectal bleeding and fissures, arthritis, anorexia, and sometimes liver, kidney, eyes and skin are affected. Often many food sensitivities (especially to dairy, wheat, yeast, vaccines and some drugs like salicylic acid).
Pathophysiology: severe dysbiosis, mutated pathogenic form of E.coli, exhausted goblet cells that produce protective mucous leave mucosa vulnerable to damage and ulceration
Recommendations: p. 185-186
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**Constipation**
Decrease in frequency of defecation accompanied by difficult or incomplete passage of stool and/or passage of excessively hard, dry stool
Etiology: low fiber diet, refined food, dysbiosis, low fluid intake, low calcium and magnesium intake, insufficient exercise, stress, irritable bowel syndrome, abdominal adhesions from a past injury or surgery, hypothyroid condition, laxative abuse and psychological issues
Signs and Symptoms: bowel movements once or less per day, they may be small in quantity, difficult to pass, hard and non continuous, bloating, abdominal pain, nausea or lack of appetite
Pathophysiology: the stool becomes hardened and the increased toxins formed by microbes have more time to be absorbed into the blood causing intoxication, diverticulosis can occur
Recommendations: p.189 & 191
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**Hemorrhoids**
Enlarged anal veins that have lost their elasticity (can be visible externally or invisible internally)
Etiology: caused and aggravated by sitting or standing for long, lifting heavy objects, constipation, straining, pregnancy, obesity, lack of exercise, liver damage, food sensitivity, low fibre intake or explosive diarrhea
Signs and Symptoms: bleeding during bowel movements, itching or irritation around the anus, pain, discomfort or swelling around anus, lump near anus which may be painful or sensitive and leakage of feces.
Pathophysiology: dilation of the venous plexus at the anal rectal junction, bleeding vessels due to irritation by bowel contents
Recommendations: p. 193 & 194
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