

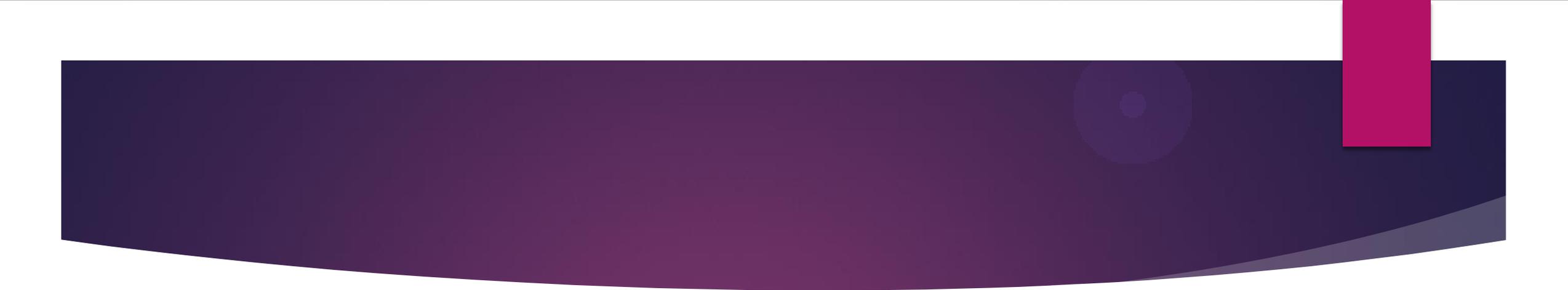
Gastrointestinal disease

FALACE, CHAPTER 11, 12

BURKET, CHAPTER 14

Liver Disease

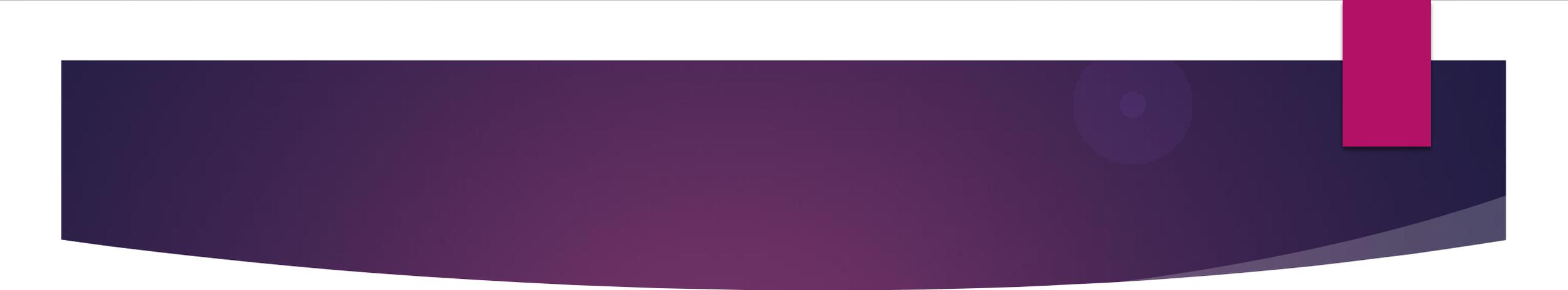
- ▶ Liver plays a vital role in metabolic functions, including secretion of bile needed for fat absorption, conversion of sugar to glycogen, and excretion of bilirubin, a waste product of hemoglobin metabolism.
- ▶ Impairment of liver function can lead to abnormalities in the metabolism of amino acids, ammonia, protein, carbohydrates, and lipids (triglycerides and cholesterol).
- ▶ Many biochemical functions performed by the liver, such as synthesis of coagulation factors and drug metabolism.
- ▶ Hepatitis is inflammation of the liver that may result from infectious or other causes.
- ▶ Viral hepatitis, infectious mononucleosis, secondary syphilis, and tuberculosis.

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- ▶ Noninfectious hepatitis can result from excessive or prolonged use of toxic substances (e.g., drugs [acetaminophen, alcohol, halothane, ketoconazole, methyldopa, methotrexate]; more commonly, alcohol).
 - ▶ Five distinct viruses—types A, B, C, D, and E—are associated with this disease.
 - ▶ Hepatitis A was called *infectious hepatitis*, and hepatitis B was referred to as *serum hepatitis*.
 - ▶ Hepatitis D (also known as *delta*) occurs only in association with hepatitis B.
 - ▶ Hepatitis C and hepatitis E were known as *non-A non-B hepatitis (NANB)*.
 - ▶ They were distinguished by the route of transmission.

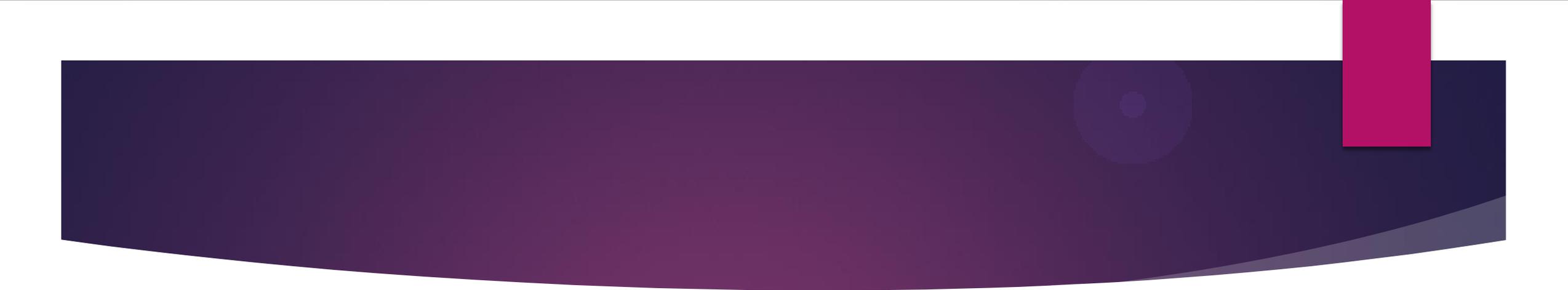
- ▶ *hepatitis non-A-E*: In up to 20% of cases of hepatitis, a standard virus cannot be identified, and the disease is not associated with toxic, metabolic, or genetic conditions.
- ▶ **Hepatitis A**: RNA virus of the Picornaviridae family that replicates in the liver, is excreted in the bile, and is shed in the stool.
- ▶ Serologic tests for HAV and its antibodies—anti-HAV, immunoglobulin M (IgM), and anti-HAV immunoglobulin G (IgG).
- ▶ Transmission of HAV occurs almost exclusively through fecal contamination of food or water, usually by traveling in an endemic region, or by direct contact with an infected person.
- ▶ Common sources include contaminated wells or water supplies, restaurants, and raw shellfish.

- ▶ The disease occurs primarily in children and young adults and is often asymptomatic.
- ▶ Hepatitis A tends to be of mild severity and self-limiting; it lasts a couple of weeks and often goes undiagnosed.
- ▶ Fatigue, fever, lymphadenopathy, gastrointestinal (GI) upset/nausea, night sweats, and possibly, icteric jaundice.
- ▶ HAV infection may be effectively prevented by administration of the HAV immune globulin prophylactically or within 2 weeks of exposure.
- ▶ **Hepatitis B:** This virus replicates predominantly in hepatocytes and to a lesser extent in stem cells in the pancreas, bone marrow, and spleen.

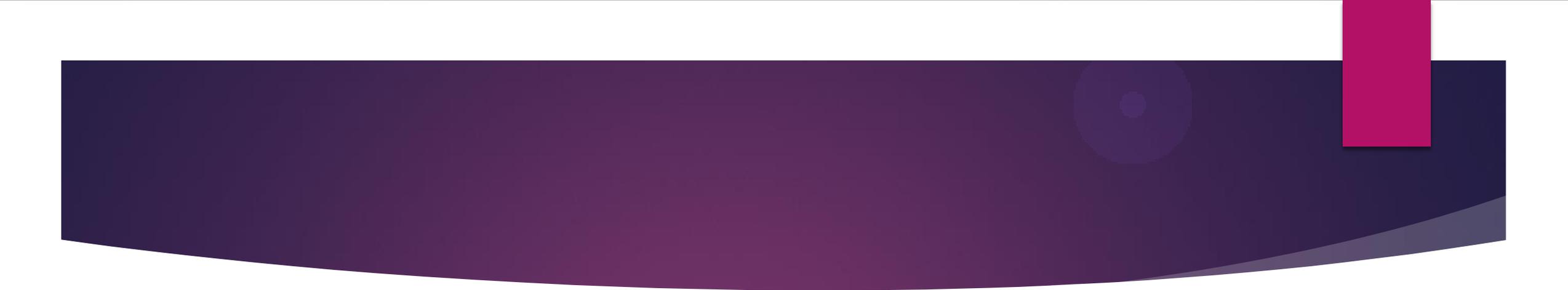
- ▶ The outer shell is the HBsAg, circulates in the blood for up to 6 months after infection.
- ▶ The antibody responsible for clearing the infection is anti-HBs.
- ▶ The inner core of the particle is the hepatitis B core antigen (HBcAg), with corresponding antibodies IgG anti-HBc and IgM anti-HBc (indicating recent infection).
- ▶ A third particle is the hepatitis B early antigen (HBeAg), an antigenic component derived from cleavage of the core antigen.
- ▶ It is related to hepatitis B infectivity. Its corresponding antibody is anti-HBe.
- ▶ Serologic tests are available for all these antigen/antibody systems, except the HBcAg, which is retained in hepatocytes.

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- ▶ Hepatitis B may be transmitted efficiently by percutaneous and permucosal exposure.
 - ▶ Exposures that may cause HBV infection include the following:
 - Direct percutaneous inoculation, transfusion of infective blood or blood products (serum, plasma, factor concentrates), needle sharing, tattooing, and body piercing.
 - Indirect percutaneous introduction of infective serum or plasma through minute skin cuts or abrasions.
 - Absorption of infective serum or plasma through mucosal surfaces of the mouth or eye.

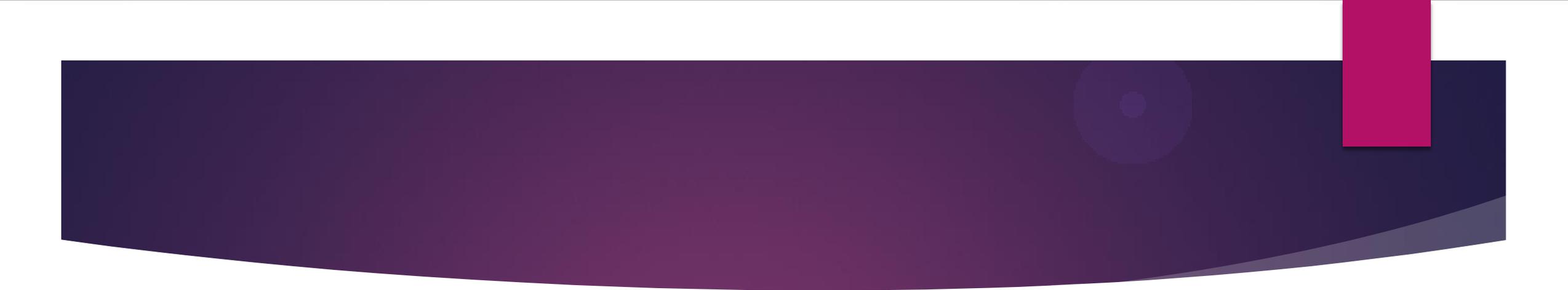
- Absorption of infective secretions, such as saliva or semen, through mucosal surfaces, as might occur following heterosexual or homosexual contact.
- Transfer of infective serum or plasma via inanimate environmental surfaces or possibly through vectors. Experimental data indicate that fecal transmission of HBV does not occur and airborne spread is not of epidemiologic importance.
- ▶ **Hepatitis C:** HCV is similar to HBV in terms of behavior and characteristics.
- ▶ Those at greatest risk for this disease are injection drug users and those with large or repeated percutaneous exposures.

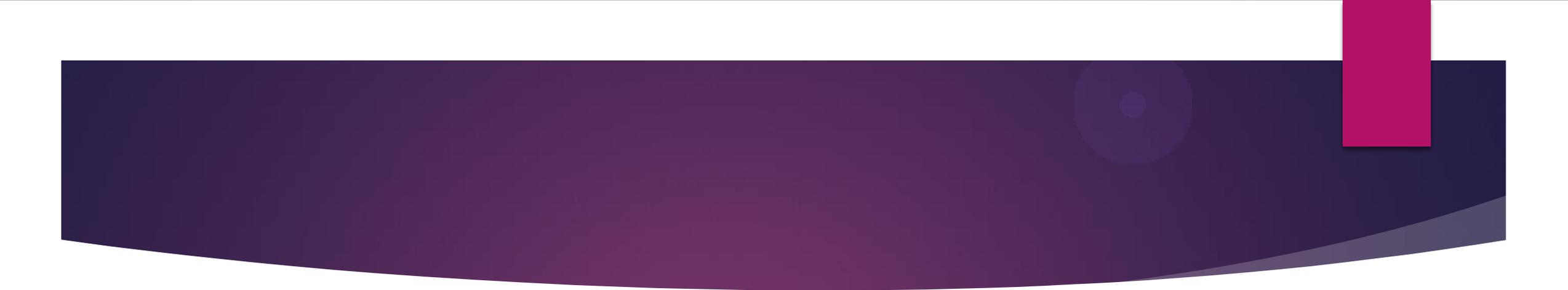
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- ▶ Others at increased risk are patients on hemodialysis, persons who have multiple sexual partners or who have sexual contacts with those who have chronic HCV, health care workers exposed to blood, and recipients of whole blood, blood cellular components, or plasma.
 - ▶ **Hepatitis D:** HDV requires HBsAg for its viral envelope and transmissibility, but once inside, a permissive cell replicates without the helper HBV.
 - ▶ HDV occurs only in patients with HBV infection, as a coinfection or a superinfection.
 - ▶ The hepatitis D antigen (HDAg) and its antibody (anti-HDV) can be detected with serologic testing.
 - ▶ HDV is transmitted parenterally and sexually, similar to HBV.

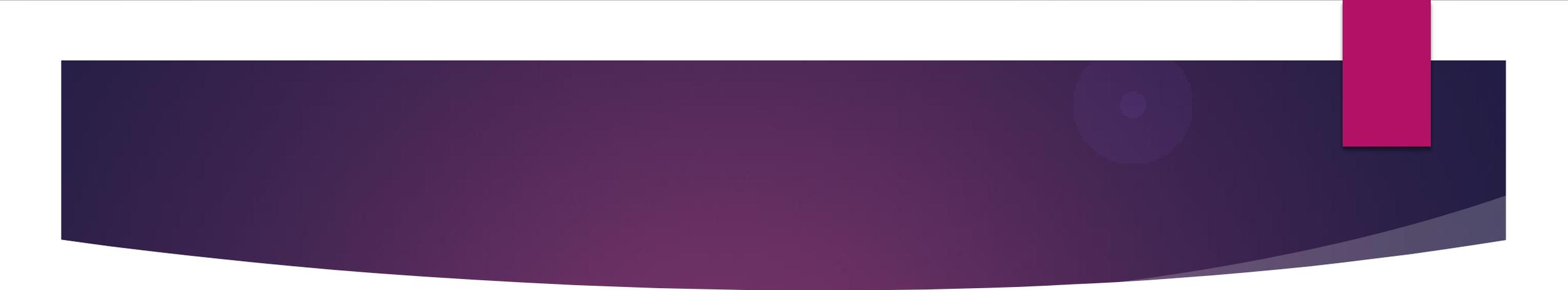
- ▶ **Hepatitis E:** This virus is responsible for enterically transmitted (formerly “NANB”) hepatitis, a disorder clinically similar to hepatitis A infection.
- ▶ **Hepatitis Non–A–E:** Cases of acute hepatitis that appear to have a viral origin but that cannot be attributed to any known virus.
- ▶ HCV is less infectious and less efficient in transmission when compared with HBV.
- ▶ The risk of contracting HBV is reported to range from 6% to 30%, with potential infectiousness correlating with HBeAg in the serum.
- ▶ The seroconversion rate of accidental blood exposure to HCV is between 2% and 8%.
- ▶ The risk of contracting HIV after a percutaneous or other sharp injury is 0.3%.

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- ▶ Commonly, acute viral hepatitis is characterized by ballooning degeneration and necrosis of liver cells.
 - ▶ The entire liver lobule becomes inflamed and consists of lymphocytes and mononuclear phagocytes.
 - ▶ Icterus (jaundice) is associated with hepatitis in approximately 70% of cases of HAV, approximately 30% of cases of HBV infection, and approximately 25% of cases of HCV and HEV.
 - ▶ This is caused by an accumulation of bilirubin in the plasma, epithelium, and urine.
 - ▶ Jaundice usually becomes clinically apparent when the plasma level of bilirubin approaches 2.5 mg/100 mL (normal is less than 1 mg/100 mL).

- ▶ **Fulminant Hepatitis:** A serious complication of acute viral hepatitis is fulminant hepatitis, which is characterized by massive hepatocellular destruction and a mortality rate of approximately 80%.
- ▶ This condition occurs more commonly among the elderly and those with chronic liver disease.
- ▶ **Chronic Infection:** Chronic infection (carrier state) is characterized by persistence of low levels of virus in the liver and serum viral antigens (HBsAg, HBeAg, and hepatitis C virus antigen [HCVAg]) for longer than 6 months without signs of liver disease.
- ▶ Individuals with this condition potentially are infectious to others.

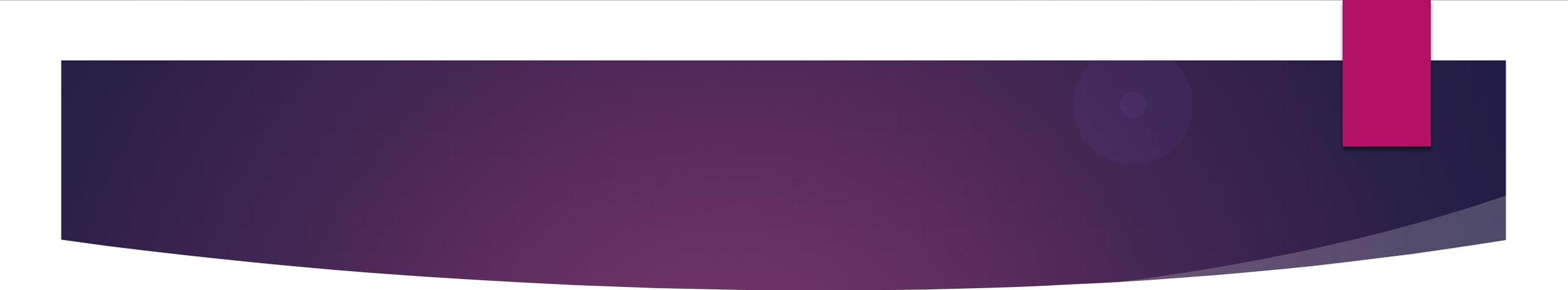
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- ▶ Chronic active hepatitis is characterized by active virus replication in the liver; HBsAg, HBeAg, or HCVAg in the serum; signs and symptoms of chronic liver disease; persistent hepatic cellular necrosis; and elevated liver enzymes for longer than 6 months.
 - ▶ The prodromal (preicteric) phase, which usually precedes the onset of jaundice by 1 or 2 weeks, consists of abdominal pain, anorexia, intermittent nausea, vomiting, fatigue, myalgia, malaise, and fever.
 - ▶ The icteric phase is heralded by the onset of clinical jaundice, a yellow-brown cast of the eyes, skin, oral mucosa, and urine.
 - ▶ Hepatomegaly and splenomegaly frequently are seen.

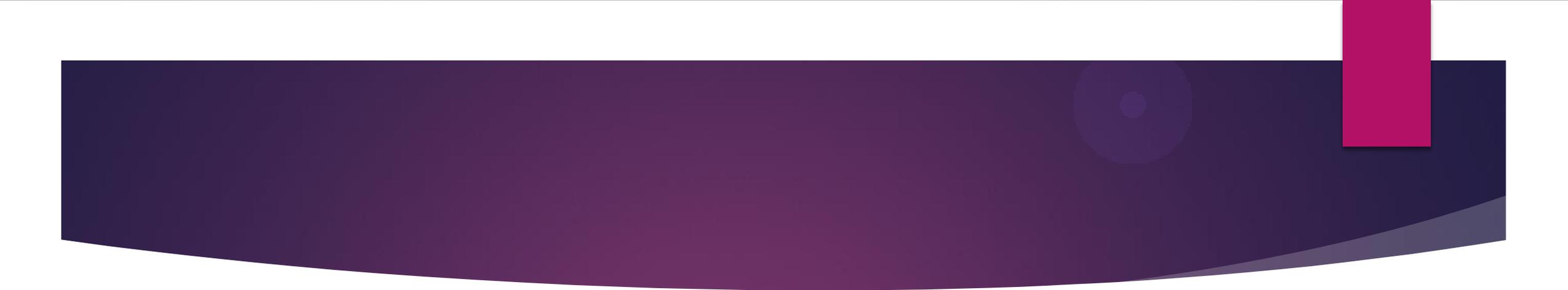
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- ▶ During the convalescent or recovery (posticteric) phase, symptoms disappear, but hepatomegaly and abnormal liver function values may persist for a variable period.
 - ▶ Laboratory Findings: AST, ALT, serum bilirubin, alkaline phosphatase, gamma-glutamyl transpeptidase, and lactate dehydrogenase, as well as increased white blood cell count and prothrombin time.
 - ▶ Treatment: Bed rest and fluids, high-calorie diet, Alcohol and drugs metabolized by the liver are not to be ingested. Viral antigen and ALT levels should be monitored for 6 months.
 - ▶ Standard therapy for patients with chronic hepatitis is interferon (IFN) alfa-2b (3 to 10 million units) administered three times weekly for 6 months to 1 year.

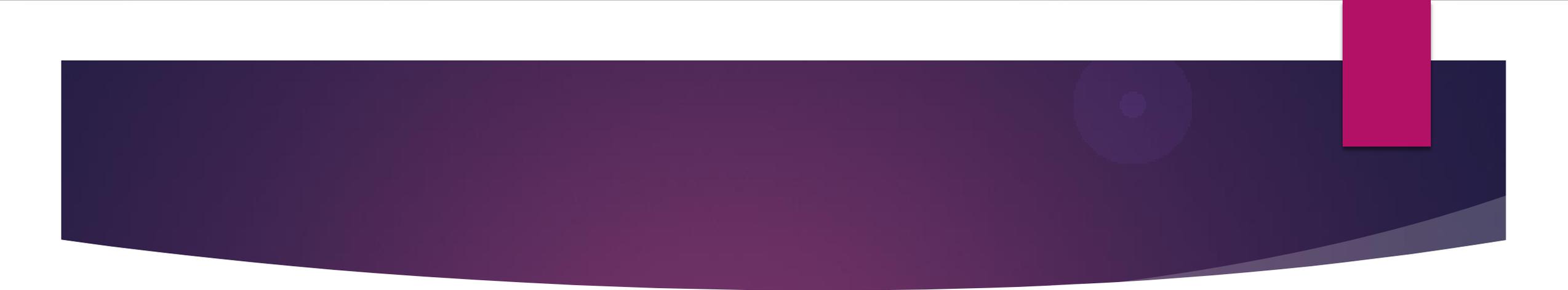
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- ▶ Lamivudine, ribavirin.
 - ▶ Corticosteroids are usually reserved for fulminant hepatitis.
 - ▶ Liver transplantation is a last resort for patients who develop cirrhosis.

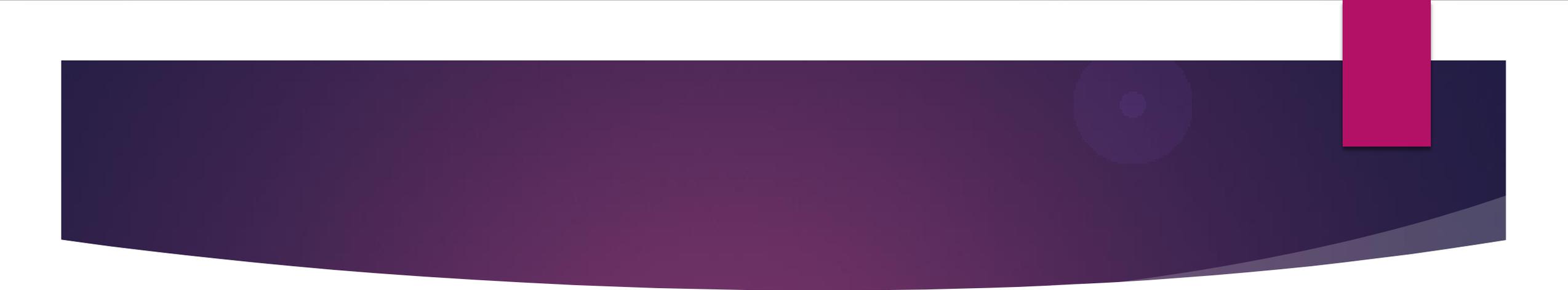
ALCOHOLIC LIVER DISEASE

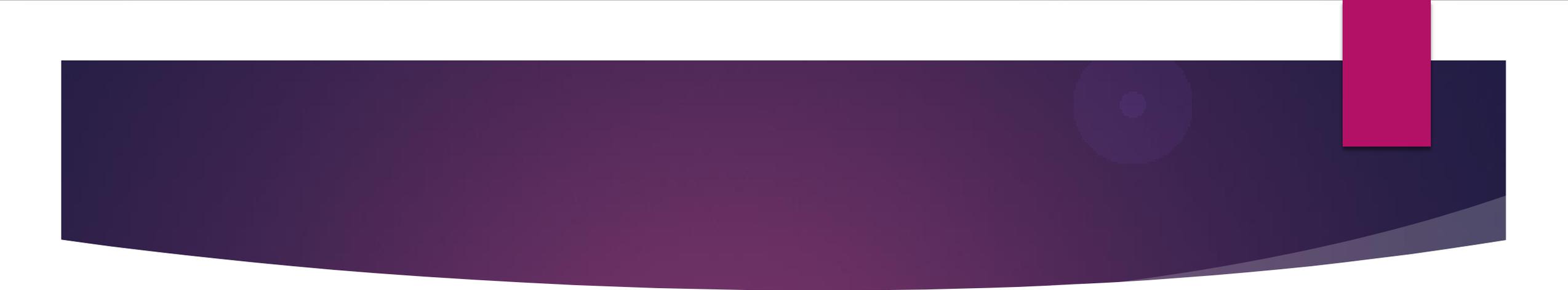
- ▶ Alcoholism is a chronic addiction to ethanol in which a person craves and uncontrollably consumes ethanol, becomes tolerant to its intoxicating effects, and has symptoms of alcohol withdrawal when the drinking stops.
- ▶ *DSM-IV* defines *alcohol dependence* as repeated alcohol related difficulties in at least three of seven areas of functioning:
 - Tolerance
 - Withdrawal
 - Ingestion of larger amounts of alcohol over longer periods than intended
 - Inability to control use
 - Giving up important activities to drink
 - Spending a great deal of time involved with alcohol use
 - Continued use of alcohol despite physical or psychological consequences

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- ▶ Alcohol consumption in large or chronic amounts contributes to disease and injury. In contrast, moderate consumption of alcohol (2 to 6 drinks per week) is associated with decreased mortality and cardiovascular disease rate.
 - ▶ Alcohol is hepatotoxic and its metabolite, acetylaldehyde, is fibrinogenic.
 - ▶ The earliest change seen in alcoholic liver disease is a fatty infiltrate. Hepatocytes become engorged with fatty lobules and distended, and the entire liver is enlarged. Usually, no other structural changes are noted.
 - ▶ A second and more serious form of alcoholic liver disease is alcoholic hepatitis. This is a diffuse inflammatory condition of the liver that is characterized by destructive cellular changes, some of which may be irreversible.

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- ▶ The third and most serious form of alcoholic liver disease is cirrhosis, which is generally considered an irreversible condition characterized by progressive fibrosis and abnormal regeneration of liver architecture in response to chronic injury or insult.
 - ▶ Hepatic failure is manifested by: esophagitis, gastritis, and pancreatitis.
 - ▶ Malnutrition, weight loss, protein deficiency (including coagulation factors), impairment of urea synthesis and glucose metabolism, endocrine disturbances, encephalopathy, renal failure, portal hypertension, and jaundice.
 - ▶ Accompanying portal hypertension is seen as the development of ascites and esophageal varices.

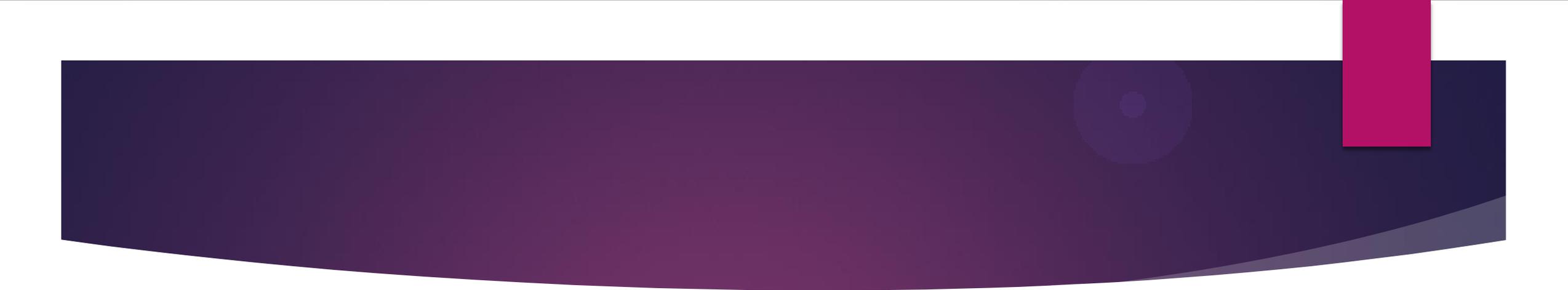
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- ▶ Bleeding tendencies are a significant feature in advanced liver disease. The basis for the diathesis is in part a deficiency of coagulation factors, especially the prothrombin group (factors II, VII, IX, and X).
 - ▶ In addition to these deficiencies, thrombocytopenia may be caused by hypersplenism caused by portal hypertension and bone marrow depression.
 - ▶ Anemia and leukopenia also may be noted because of the toxic effects of alcohol on the bone marrow and nutritional deficiencies.
 - ▶ The combination of hemorrhagic tendencies and severe portal hypertension sets the stage for episodes of gastrointestinal bleeding, epistaxis, ecchymosis, or ruptured esophageal varix.

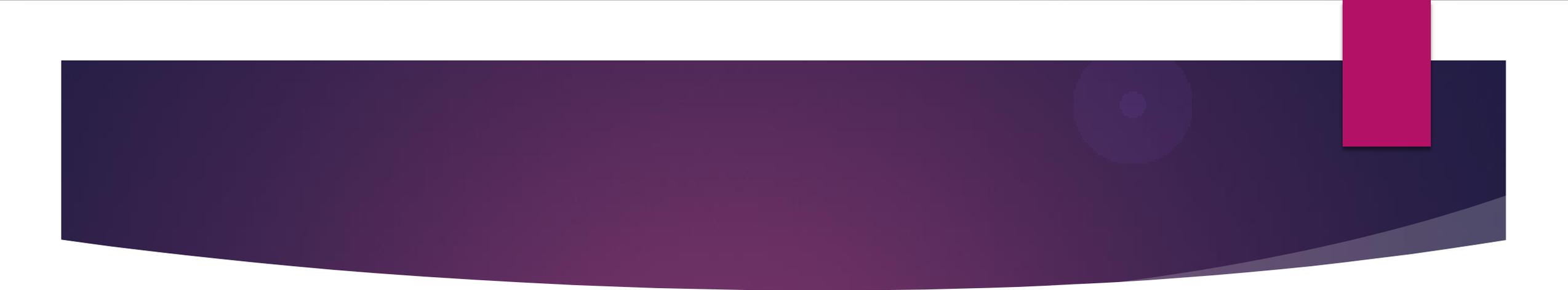
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- ▶ Ethanol abuse predisposes the individual to infection by several mechanisms.
 - ▶ The clinical presentation of alcoholic hepatitis often is nonspecific and may include features such as nausea, vomiting, anorexia, malaise, weight loss, and fever.
 - ▶ More specific findings include hepatomegaly, splenomegaly, jaundice, ascites, ankle edema, and spider angiomas.
 - ▶ **Laboratory Findings:** bilirubin, alkaline phosphatase, AST, ALT, gamma-glutamyl transpeptidase, amylase, uric acid, triglycerides, and cholesterol, CBC & diff.

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- ▶ Medical management:
 - ▶ The first is identification and intervention.
 - ▶ The second step is withdrawal from alcohol or, in cases of severe dependence, reduction in alcohol consumption.
 - ▶ The third step is to manage central nervous system depression caused by the rapid removal of ethanol.
 - ▶ Administration of a benzodiazepine, such as diazepam or chlordiazepoxide, gradually decreases drug levels over a 3- to 5-day period and alleviates alcohol withdrawal symptoms.

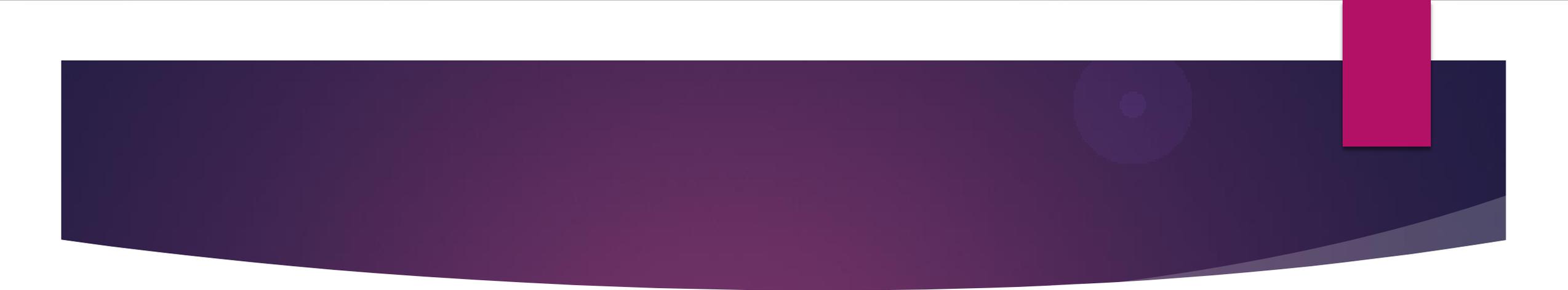
PEPTIC ULCER DISEASE

- ▶ A peptic ulcer is a well-defined break in the gastrointestinal mucosa that results from chronic acid/pepsin secretions and the destructive effects of and host response to *Helicobacter pylori*.
- ▶ The first portion of the duodenum is the location of most ulcers in Western populations, whereas gastric ulcers are more frequent in Asia.
- ▶ Infection is correlated with poor socioeconomic status, contaminated drinking water, and familial overcrowding, especially during childhood.
- ▶ Because of their ability to directly damage mucosa, reduce mucosal prostaglandin production, and inhibit mucous secretion, NSAIDs are associated with 15% to 20% of peptic ulcers.
- ▶ Ulcers caused by NSAIDs more often are located in the stomach than in the duodenum.

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- ▶ Risk with NSAID use increases with age older than 60 years, high-dosage long-term therapy, use of NSAIDs with long plasma half-lives (e.g., piroxicam) rather than those with short half-lives (i.e., ibuprofen), and concomitant use of alcohol, corticosteroids, anticoagulants, or aspirin.
 - ▶ Use of nitrogen-containing bisphosphonate drugs (alendronate, risedronate) for the treatment of osteoporosis and immunosuppressive medications such as mycophenolate is associated with esophageal and gastric ulcers.
 - ▶ Although many patients with active peptic ulcer have no ulcer symptoms, most develop epigastric pain that is longstanding (several hours) and sharply localized.

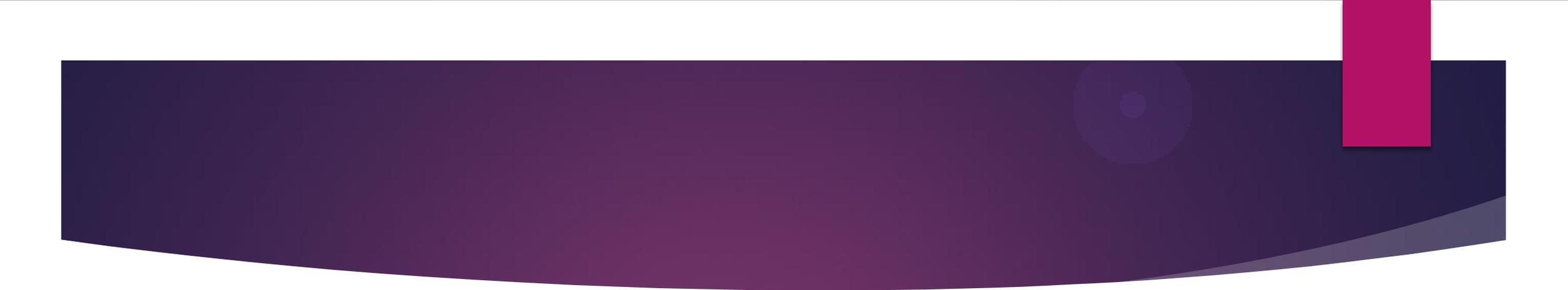
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- ▶ The discomfort of a duodenal ulcer manifests most commonly on an empty stomach, usually 90 minutes to 3 hours after eating, and frequently awakens the patient in the middle of the night.
 - ▶ Ingestion of food, milk, or antacids provides rapid relief in most cases.
 - ▶ Patients with gastric ulcers, however, are unpredictable in their response to food and may develop abdominal pain from eating.
 - ▶ Changes in the character of pain may indicate the development of complications.
 - ▶ Increased discomfort, loss of antacid relief, or pain radiating to the back may signal deeper penetration or perforation of the ulcer.

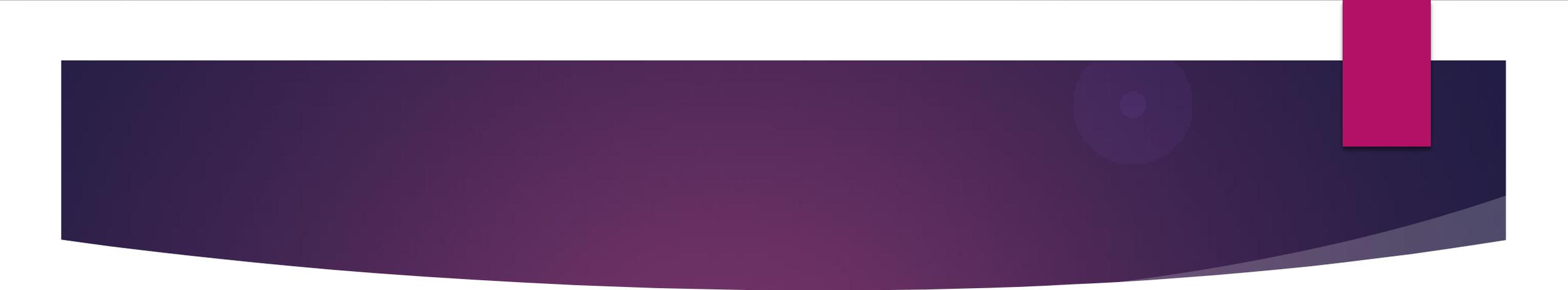
- ▶ Protracted vomiting a few hours after a meal is a sign of gastric outlet (pyloric) obstruction.
- ▶ Melena (bloody stools) or black tarry stools indicate blood loss due to gastrointestinal hemorrhage.
- ▶ Laboratory Findings: fiberoptic endoscopy and laboratory tests for *H. pylori*.
- ▶ MEDICAL MANAGEMENT: Combination therapy is recommended because antisecretory drugs, such as H₂ antagonists and proton pump inhibitors (PPIs), provide rapid relief of pain and accelerate healing, and antibiotics are efficient in eradicating *H. pylori*.
- ▶ In all patients who undergo peptic ulcer therapy, ulcerogenic factors (i.e., continued use of alcohol, aspirin, NSAIDs, and corticosteroids; foods that aggravate symptoms and stimulate gastric acid secretion; stress) should be eliminated to accelerate healing and limit the occurrence of relapse.

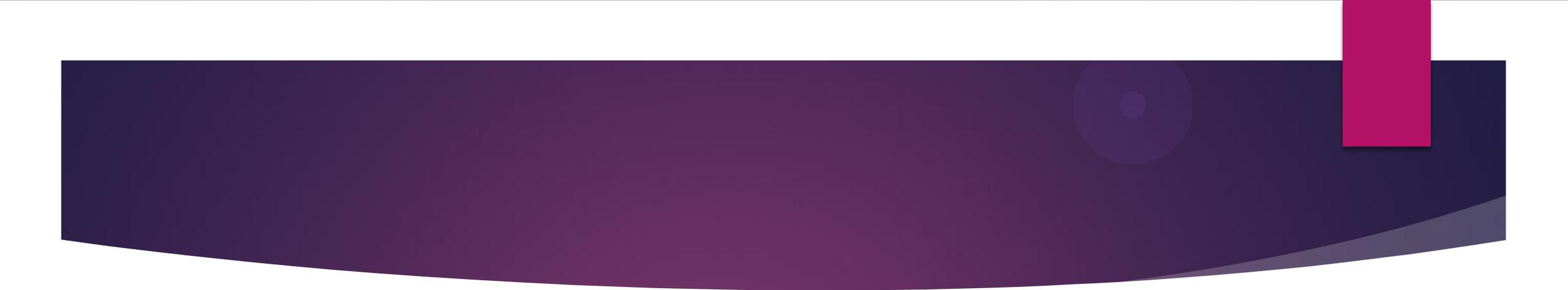
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- ▶ Patients appear to benefit from smoking cessation.
 - ▶ surgery is reserved primarily for complications of peptic ulcer disease such as significant bleeding, perforation, and gastric outlet obstruction.

INFLAMMATORY BOWEL DISEASE

- ▶ Ulcerative colitis and Crohn's disease.
- ▶ Ulcerative colitis is a mucosal disease that is limited to the large intestine and rectum.
- ▶ Crohn's disease is a transmural process (affects entire wall of the bowel) that may produce "patchy" ulcerations along any point of the alimentary canal from the mouth to the anus but most commonly involves the terminal ileum.
- ▶ Ulcerative colitis and Crohn's disease are inflammatory diseases of unknown cause that are generally thought to be associated with immune dysfunction in response to environmental factors in genetically susceptible persons.

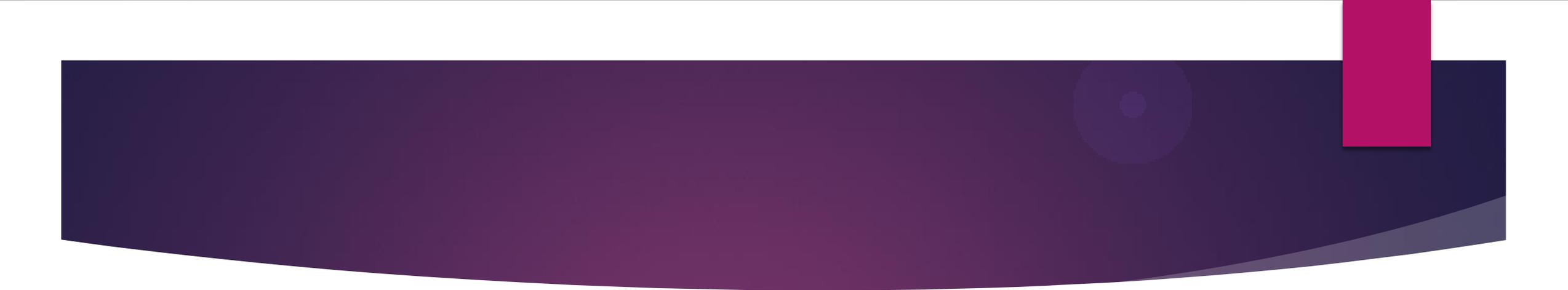
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- ▶ Patients with ulcerative colitis have three prominent symptoms: (1) attacks of diarrhea, (2) rectal bleeding (or bloody diarrhea), and (3) abdominal cramps.
 - ▶ Onset may be sudden or insidious, but the disease continues along a chronic, intermittent course in most patients.
 - ▶ Initial manifestations of Crohn's disease consist of recurrent or persistent diarrhea (often without blood), abdominal cramps, anorexia, and weight loss.
 - ▶ Intestinal complications from chronic inflammatory damage include transmural fibrosis, intestinal fissuring, fistulas, and abscess formation.

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- ▶ Malabsorption is an additional complication that can result in striking degrees of weight loss, growth failure, anemia, and clubbing of the fingers.
 - ▶ Reduced bone mineral density (i.e., osteoporosis) also results from malabsorption and chronic corticosteroid use.
 - ▶ The diagnosis of IBD is based primarily on clinical findings, results of colonoscopy and biopsy, and observations on histologic examination of intestinal mucosal, abdominal radiographs and stool examinations.
 - ▶ MEDICAL MANAGEMENT:
 - ▶ Anti-inflammatory medications (e.g., sulfasalazine, 5-aminosalicylic acid, corticosteroids) are generally first-line drugs.

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- ▶ Immunosuppressive agents and antibiotics are used as second-line drugs.
 - ▶ Third-line approaches for persons with Crohn's disease who are refractory to steroid treatment include monoclonal antibody (infliximab [Remicade]) against tumor necrosis factor and surgical resection to remove the diseased portion of the colon.
 - ▶ Supportive therapy that includes bed rest, dietary manipulation, and nutritional supplementation is often required.

PSEUDOMEMBRANOUS COLITIS

- ▶ Pseudomembranous colitis is a severe and sometimes fatal form of colitis that results from the overgrowth of *Clostridium difficile*.
- ▶ Overgrowth results from the loss of competitive anaerobic gut bacteria, most commonly through the use of broad-spectrum antibiotics, but it can also result from heavy metal intoxication, sepsis, and organ failure.
- ▶ Diarrhea is the most common symptom.
- ▶ In severe cases, diarrhea is bloody and is accompanied by abdominal cramps and tenderness and by fever.
- ▶ Severe dehydration, metabolic acidosis, hypotension, peritonitis are serious complications of untreated disease.

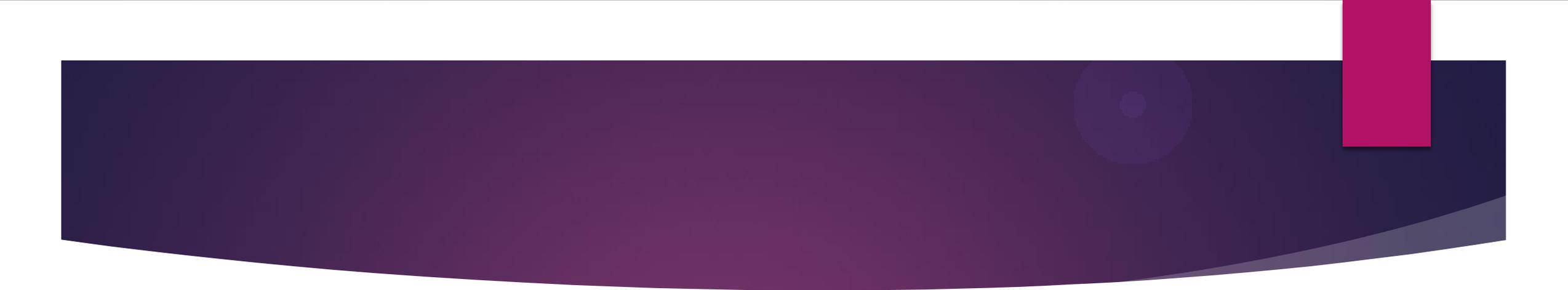


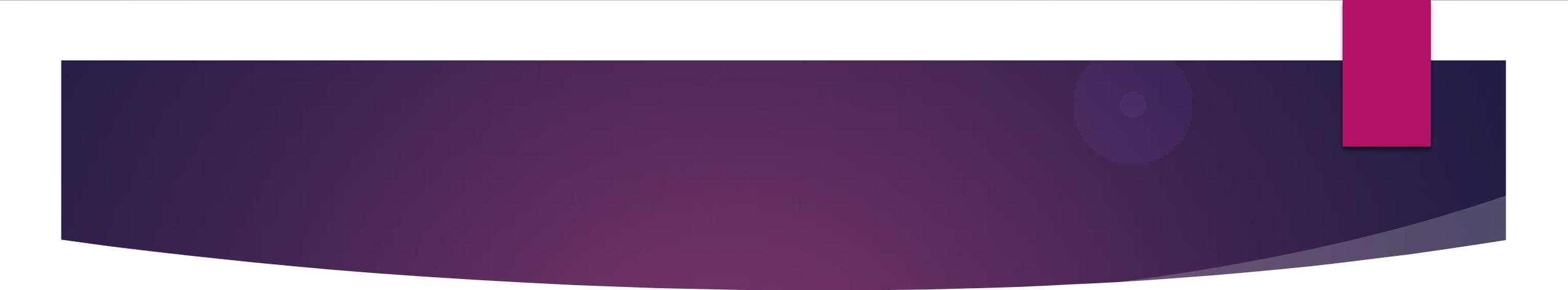
▶ MEDICAL MANAGEMENT:

- ▶ First-line treatment of pseudomembranous colitis involves discontinuing use of the antimicrobial agent.
- ▶ In more severe cases, oral metronidazole (Flagyl, 250 mg four times a day for 10 days) is the therapy of first choice.
- ▶ Vancomycin (125 to 500 mg four times a day for 10 days) is recommended for patients who are unresponsive to metronidazole.

Gastroesophageal Reflux Disease

- ▶ During gastroesophageal reflux, gastric contents (chyme) passively move up from the stomach into the esophagus.
- ▶ heartburn is the cardinal symptom of GERD and is defined as a sensation of burning or heat that spreads upward from the epigastrium to the neck.
- ▶ chest pain is another important symptom that is related to disorders of the esophagus. chest pain can mimic the symptoms of an acute cardiovascular.
- ▶ Barrett's esophagus is a variant of GERD in which normal squamous epithelium is replaced by columnar epithelium.

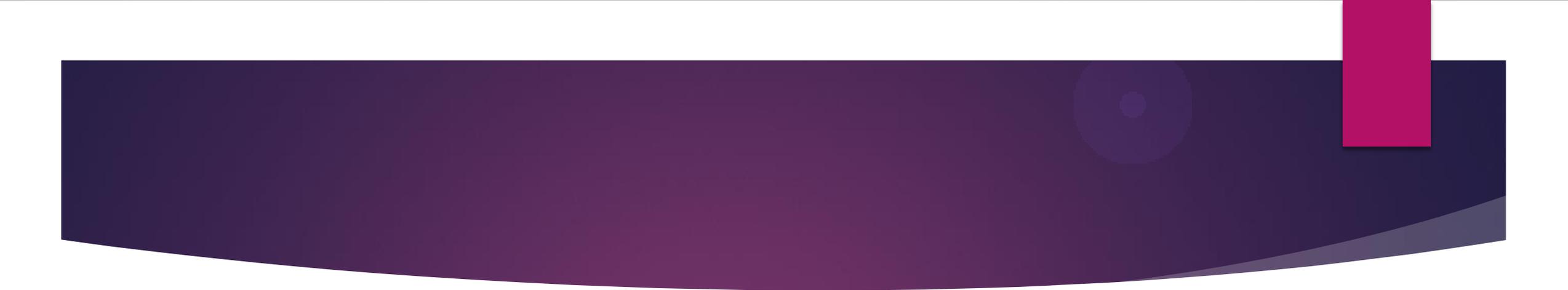
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- ▶ The relaxation of the lower esophageal sphincter for the purpose of relieving pressure in the stomach (from gas and the ingestion of food) is called the “burp” mechanism. this phenomenon is a normal process and occurs only when a person is in an erect posture; gastric contents are thereby prevented from flowing into the esophagus and possibly being aspirated.
 - ▶ The gastroesophageal junction, which prevents the regurgitation (retrograde or upward flow) of gastric contents, is composed of an internal lower esophageal sphincter. external pressure on the junction by the diaphragm also assists in this function.
 - ▶ when this barrier fails, gastric contents may make their way into the esophagus and cause symptoms.

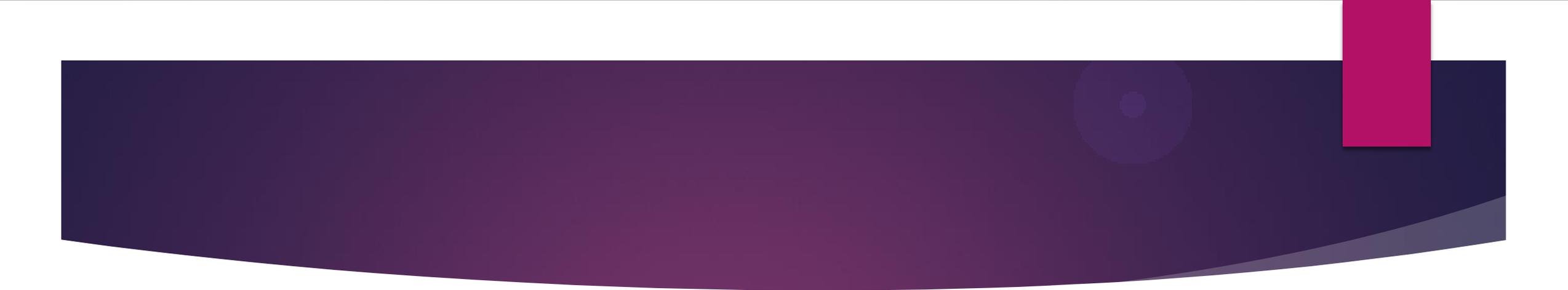
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- ▶ significant success in preventing or reducing the symptoms of GERD is seen with lifestyle modification. weight loss reduces the pressure difference between the abdomen and the thorax, thereby reducing reflux. smoking cessation will increase the production of saliva and therefore counteract the symptoms of GERD. fatty meals slow down gastric emptying.
 - ▶ proton pump inhibitors (PPIs) such as omeprazole, provide not only symptomatic relief but also resolution of signs, including those that involve significant ulcers and/or esophageal damage.
 - ▶ patients who experience GERD complain of dysgeusia (foul taste), dental sensitivity related to hot or cold stimuli, dental erosion, and/or pulpitis. Dental thermal sensitivity is generally due to erosion of enamel by gastric acid.

- ▶ Mild baking soda mouthrinses may be swished and expectorated to minimize dysgeusia due to acid reflux. Dental management should provide topical fluoride applications using custom-made occlusive tray delivery in order to ensure optimal dental mineralization and reduction of thermal sensitivity.
- ▶ patients taking cimetidine (tagament) or other H₂ receptor antagonists may experience a toxic reaction to lidocaine (or other amide local anesthetics) if the anesthetic is injected intravascularly.
- ▶ cimetidine also has been shown to inhibit the absorption and, therefore, the blood concentration ofazole antifungal drugs such as ketaconazole via the potent inhibition of the cytochrome p450 enzyme system.
- ▶ soft tissue changes such as esophageal stricture and fibrosis may complicate intubation if the patient requires general anesthesia for an oral maxillofacial procedure.
- ▶ Oral mucosal changes are minimal; however, erythema and mucosal atrophy may be present as a result of chronic exposure of tissues to acid. mild sodium bicarbonate rinses may again be useful if mild signs of stomatitis are present.

Hiatal Hernia

- ▶ The esophagus passes through the diaphragmatic hiatus and into the stomach just inferior to the diaphragm. the hiatus causes an anatomic narrowing of the opening into the stomach and thus helps prevent reflux of stomach contents into the esophagus.
- ▶ some patients have a weakened or enlarged hiatus, perhaps due to hereditary factors. it may also be caused by obesity, exercising (eg, weight lifting), or chronic straining when passing stools.
- ▶ when a weakened or enlarged hiatus occurs, a portion of the stomach herniates into the chest cavity through this enlarged hole, resulting in a hiatal.

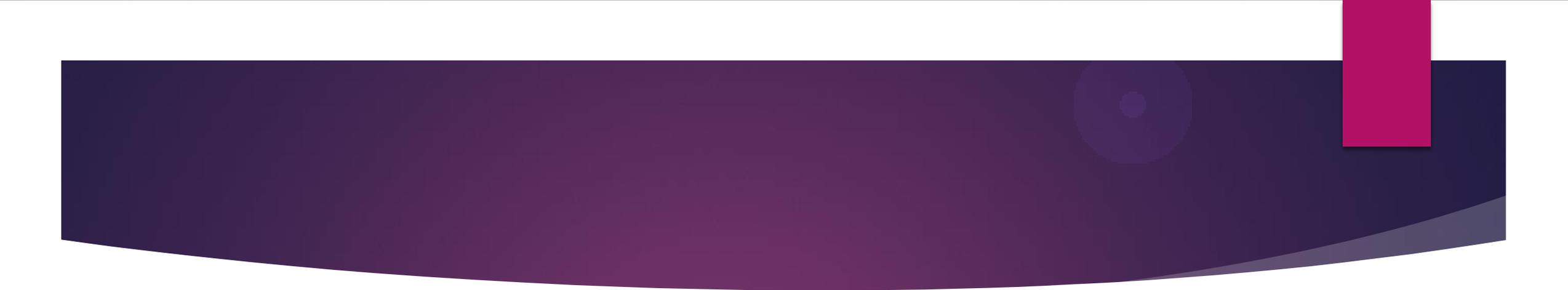
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- ▶ symptoms of hiatal hernia often include chest pain, which may radiate in patterns similar to those of myocardial infarction pain.
 - ▶ infants with hiatal hernia usually regurgitate blood stained food and may also have difficulty in breathing and swallowing.
 - ▶ adult patients with hiatal hernia may experience chronic acid reflux into the esophagus. chronic gastrointestinal reflux can erode the esophageal lining, causing bleeding, which may lead to anemia.
 - ▶ additionally, chronic esophageal inflammation may produce scarring, resulting in esophageal narrowing. this narrowing causes dysphagia, and because food does not pass easily into the stomach, patients experience an uncomfortable feeling of fullness or “bloating.”

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- ▶ adults typically present with heartburn that is exacerbated when bending forward or lying down. the pain may spread to the jaw and down the arms, similar to an attack of angina pectoris.
 - ▶ other symptoms include hiccups, a dry cough, and an increase in the contractile force of the heart.
 - ▶ The infant should sleep in a crib with the head raised and be given an altered diet consisting of food that has a thicker than normal consistency.
 - ▶ with adults, anything that will increase abdominal pressure and cause reflux, such as bending, abdominal exercises, and tight belts and girdles, should be avoided. Because obesity increases intraabdominal pressure, weight loss may be recommended to relieve symptoms. sleeping with the head elevated will also prevent the symptoms of hiatal hernia.

- ▶ patients should also eat smaller and more frequent meals and should have their main meal at lunchtime. this should be followed by a light supper, with nothing being consumed within 2 to 3 hours of bedtime.
- ▶ foods and habits that increase the reflux of acid should be avoided or significantly reduced. these foods or habits include nicotine (tobacco products), alcohol, caffeine, chocolate, fatty foods, and peppermint or spearmint oil flavorings.
- ▶ if a hiatal hernia is treated with medications that cause xerostomia (dry mouth), the dose or drug type may need to be altered by the patient's physician.
- ▶ various treatment modalities for dry mouth, such as artificial saliva, alcohol free mouthwashes, or increased fluid intake, may need to be prescribed.
- ▶ class v caries, or root caries, are sequelae of dry mouth, even in patients who have been relatively free of caries prior to developing the disease.
- ▶ if reflux into the oral cavity is present, oral manifestations that are the same as those of GERD may be present.

eating Disorders: anorexia and Bulimia

- ▶ anorexia involves individuals who intentionally starve themselves when they are already underweight. people suffering from this disorder have an intense fear of becoming fat, even when they are extremely underweight (defined as body weight that is 15% or more below the recommended levels).
- ▶ in contrast to those with anorexia, persons with bulimia nervosa consume large amounts of food during “binge” episodes in which they feel out of control of their eating. Bulimic individuals are also not as successful in dieting as are those with anorexia. they then try to prevent weight gain after such episodes by vomiting, using laxatives or diuretics, dieting, and/or exercising aggressively.
- ▶ to be diagnosed with bulimia nervosa, an individual must engage in bingeing and purging at least twice a week for 3 months; exhibit a feeling of lack of control over eating; regularly use self induced vomiting, laxatives, or diuretics to prevent weight gain; and exhibit a persistent excessive concern with body shape and weight.

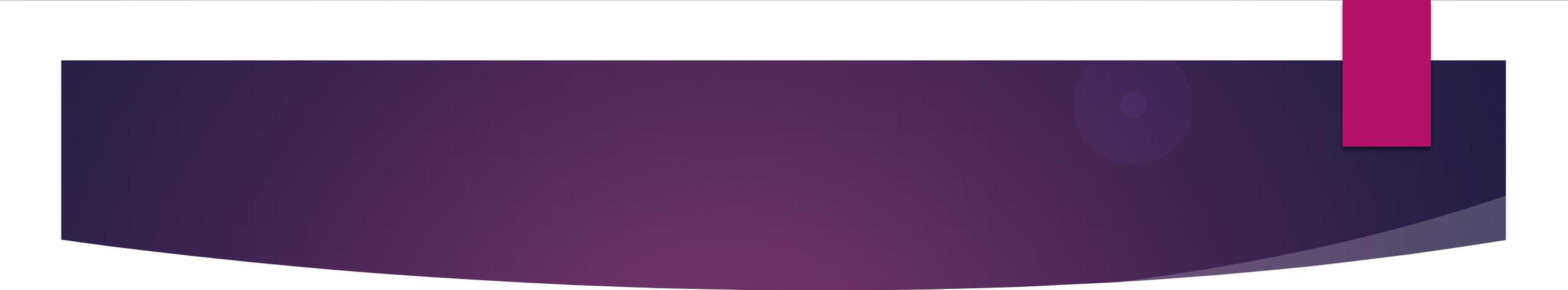
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- ▶ the cardinal oral manifestation of eating disorders is severe erosion of the enamel on the lingual surfaces of the maxillary teeth. acids from chronic vomiting are the cause.
 - ▶ mandibular teeth may be affected but not as severely as the maxillary teeth. parotid enlargement may develop as a sequela of starvation. rarely does one observe soft tissue changes of the oral mucosa because of trauma from gastric acids.

- ▶ gardner's syndrome consists of intestinal polyposis (which represents premalignant lesions) and multiple impacted supernumerary (extra) teeth.
- ▶ Plummer vinson syndrome: the hallmark of this disorder is dysphagia resulting from esophageal stricture, causing many patients to have a fear of choking. patients may present with a lemon tinted pallor and with dryness of the skin, spoonshaped fingernails, koilonychia, and splenomegaly. the oral manifestations are the result of an iron deficiency anemia.
- ▶ oral findings include atrophic glossitis with erythema or fissuring, angular cheilitis, thinning of the vermilion borders of the lips, and leukoplakia of the tongue. inspection of the oral mucous membranes will disclose atrophy and hyperkeratinization. these oral changes are similar to those encountered in the pharynx and esophagus. carcinoma of the upper alimentary tract has been reported in 10 to 30% of patients.

- ▶ peutzJeghers syndrome is characterized by multiple intestinal polyps throughout the gastrointestinal tract but primarily in the small intestine. malignancies in the gastrointestinal tract and elsewhere in the body have been reported in approximately 10% of patients with this syndrome.
- ▶ pigmentation (present from birth) of the face, lips, and oral cavity is a hallmark of this syndrome. interestingly, the facial pigmentation fades later in life, although the intraoral mucosal pigmentation persists. no specific oral treatment is necessary.
- ▶ cowden's syndrome (multiple hamartoma and neoplasia syndrome): facial trichilemmomas, gastrointestinal polyps, breast and thyroid neoplasms, and oral abnormalities. cowden's syndrome is considered to be a cutaneous marker of internal malignancies. pebbly papilloma like lesions and multiple fibromas may be found widely distributed throughout the oral cavity.

VIRAL HEPATITIS, TYPES B, C, D, AND E

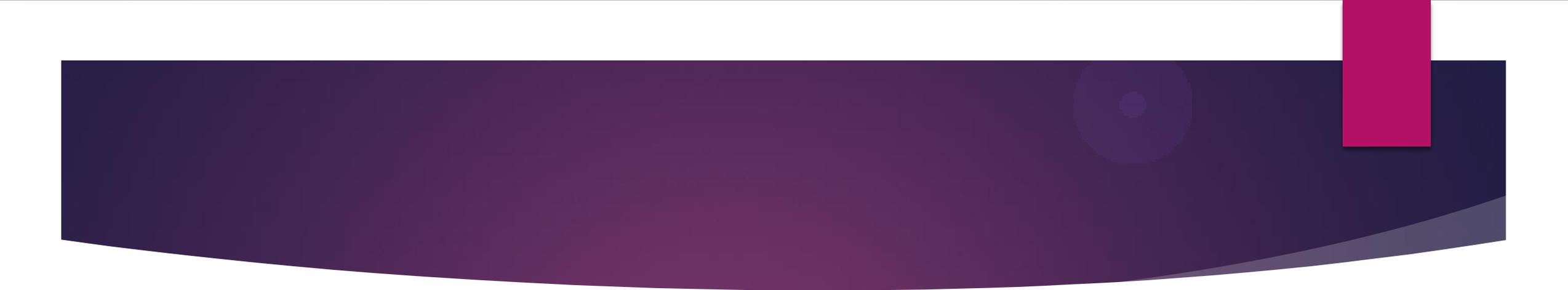
- Potential problem related to dental care:
 1. Hepatitis may be contracted by the dentist from an infectious patient.
 2. Patients or staff may be infected by the dentist with active hepatitis or who is a carrier.
 3. With chronic active hepatitis, the patient may have chronic liver dysfunction, which may be associated with a bleeding tendency or altered drug metabolism.
- Oral manifestation:
 - Bleeding
 - Lichenoid eruptions

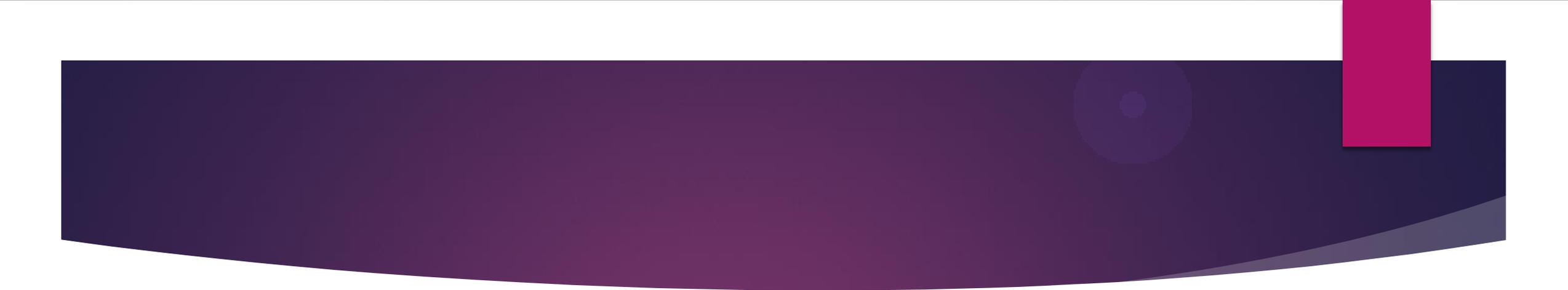


- Prevention of problem:

Caution: Because most carriers are undetectable by history, all patients should be treated with the use of standard precautions. risk may be decreased by the use of hepatitis B vaccine.

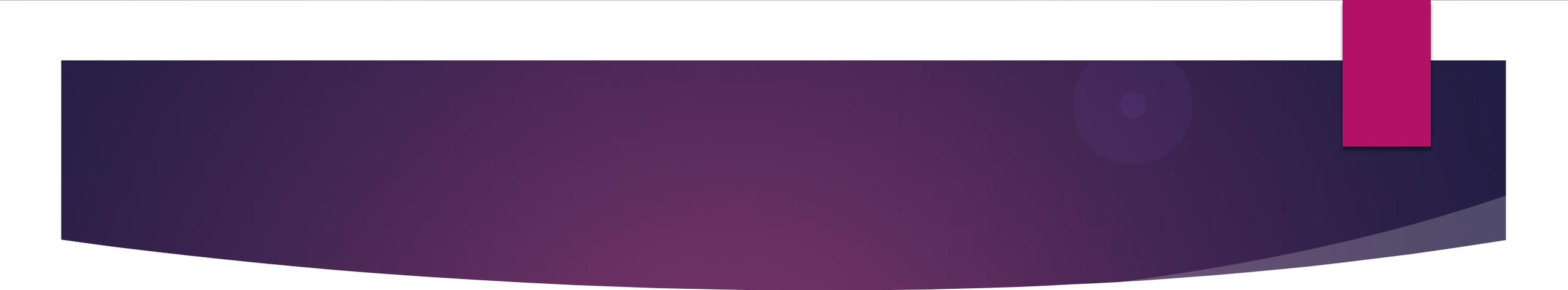
- For patient with active hepatitis, use the following procedures:
- Consult with the physician (to determine status).
- Treat on an emergency basis only.

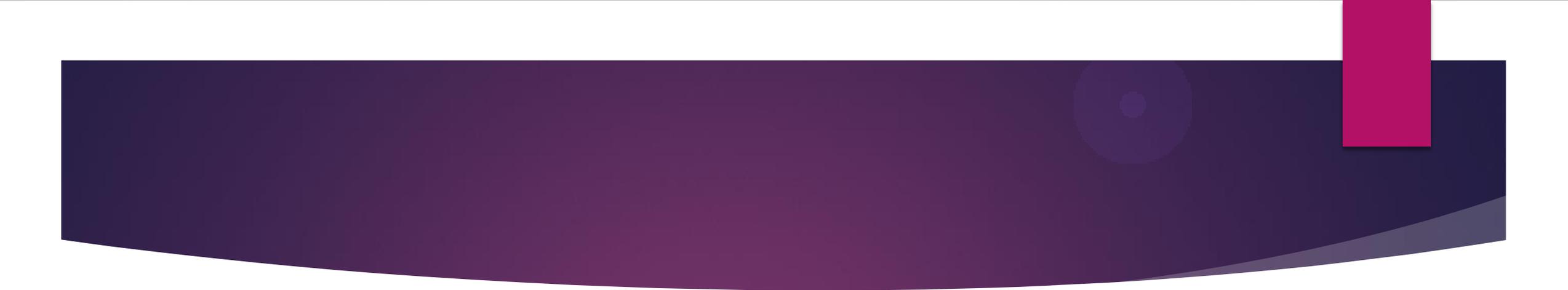
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- For patients with a history of hepatitis, use the following procedures:
 - Consult with the physician (to determine status).
 - Probable type determination:
 1. Age at time of infection (type B uncommon at younger than 15 years of age)
 2. Source of infection (if food or water, usually type A or E)
 3. If blood transfusion related, probably type C
 4. If type is indeterminate, assay for hepatitis B surface antigen (HBsAg) may be considered.

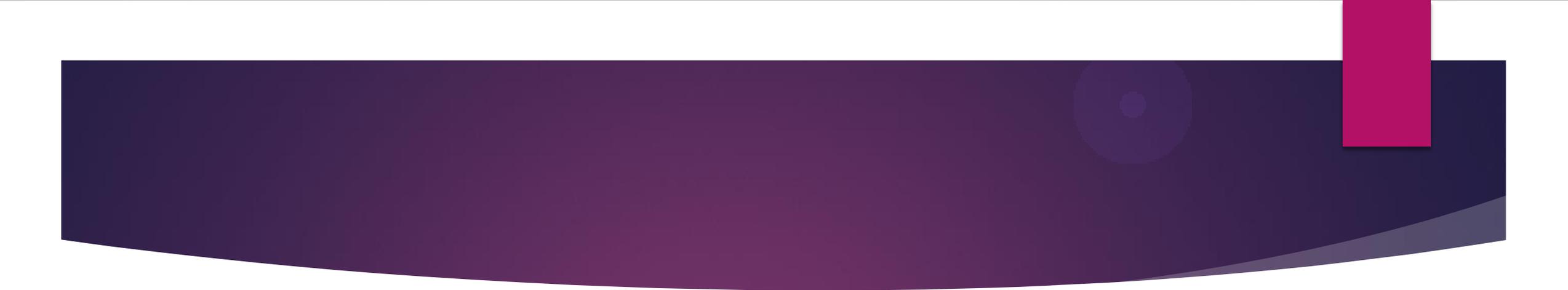
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- With patients in high-risk categories, consider screening for HBsAg or anti-hepatitis C virus.
 - If HBsAg or hepatitis C virus positive (carrier):
 - Consult with the physician to determine the status of liver dysfunction and/or recommendations for early treatment.
 - Minimize the use of drugs metabolized by the liver.
 - Monitor preoperative prothrombin time and bleeding time in chronic active hepatitis.
 - Needle stick:
 - Consult the physician.
 - Consider hepatitis B immunoglobulin.
 - Treatment planning modifications: none required

ALCOHOLIC LIVER DISEASE (CIRRHOSIS)

- Potential Problems Related to Dental Care:
 - Bleeding tendencies; unpredictable drug metabolism
- Oral manifestation:
 - Neglect, bleeding, ecchymoses, petechiae, glossitis, angular cheilosis, impaired healing, parotid enlargement, candidiasis, oral cancer, alcohol breath odor, bruxism, dental attrition and xerostomia

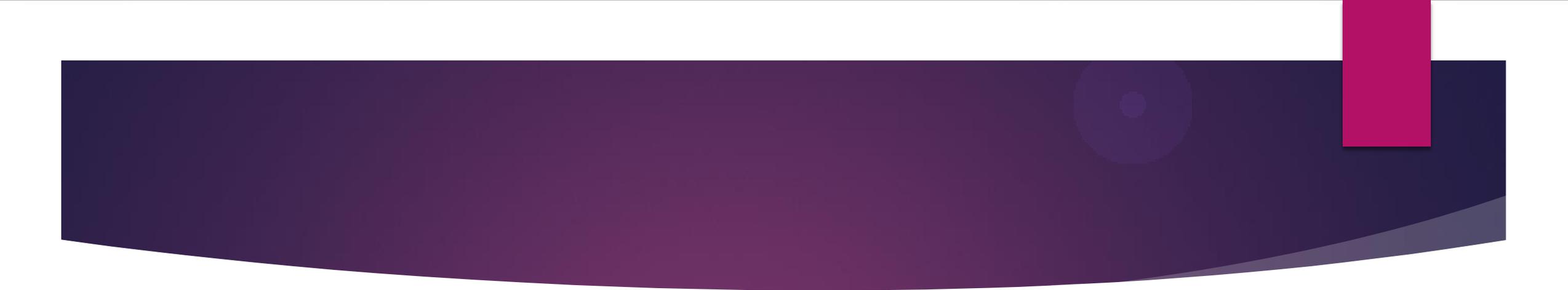
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- Prevention of problem:
 - Identify alcoholic patients through the following methods:
 - History
 - Clinical examination
 - Detection of odor on breath
 - Information from friends or relatives
 - Consult with the physician to determine the status of liver dysfunction.
 - Perform clinical screening with the CAGE questionnaire, and attempt to guide patients during treatment.

- 
- Laboratory screening should include the following:
 - Complete blood count with differential
 - Aspartate aminotransferase, alanine aminotransferase
 - Platelet count
 - Thrombin time
 - Prothrombin time
 - Minimize the use of drugs metabolized by the liver.
 - If screening tests are abnormal for surgery, consider antifibrinolytic agents, fresh frozen plasma, vitamin K, and platelets.
 - Defer routine care if ascites (encephalopathy), if present.

- 
- Treatment planning modifications:
 - Because oral neglect is commonly seen in alcoholic individuals, patients should be required to demonstrate interest in and ability to care for dentition before any significant treatment is rendered.

Peptic ulcer disease

- Potential Problems Related to Dental Care:
 1. Further injury to the intestinal mucosa caused by aspirin and NSAIDs
 2. Fungal overgrowth during or after systemic antibiotic use
- Oral manifestation:
 - Rare—Enamel dissolution associated with persistent regurgitation
 - Fungal overgrowth
 - Rare—Vitamin B deficiency (glossopyrosis) with omeprazole use

- 
- Prevention of problems:
 - Avoid aspirin and NSAIDs.
 - Avoid corticosteroids.
 - Examine oral cavity for signs of fungal overgrowth.

 - Treatment planning modifications:
 - Provide as stress free an environment as possible.

INFLAMMATORY BOWEL DISEASE

- Potential Problems Related to Dental Care:

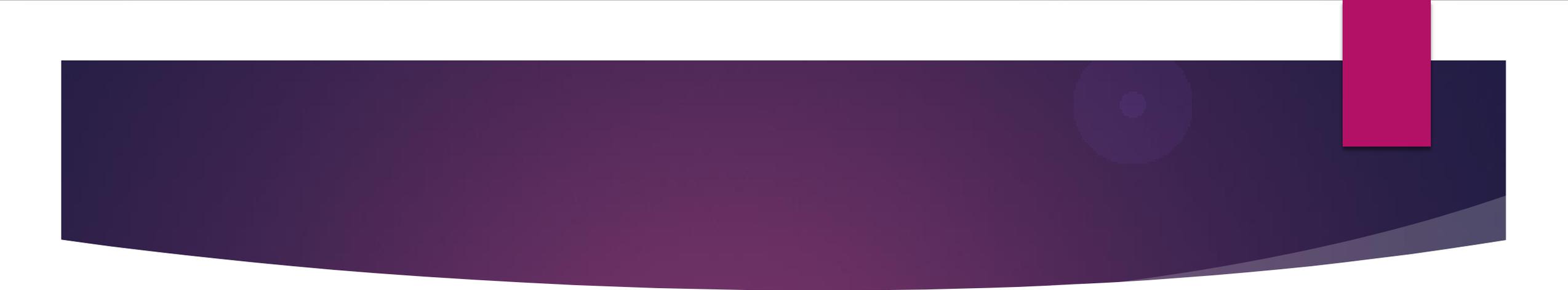
In patients who are being treated with steroids, stress may lead to serious medical problems.

- Oral manifestation:

- Cobblestone
- Aphthous lesions
- Pyostomatitis vegetans

- Prevention of problems:

- Additional steroids may be needed for surgical procedures.
- Complete blood count is needed to monitor toxic hematologic effects of drugs.

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- Treatment planning modifications:
 - Schedule appointments during remissions.

PSEUDOMEMBRANOUS COLITIS

- Potential Problems Related to Dental Care:

Fungal overgrowth during or after course of antibiotics

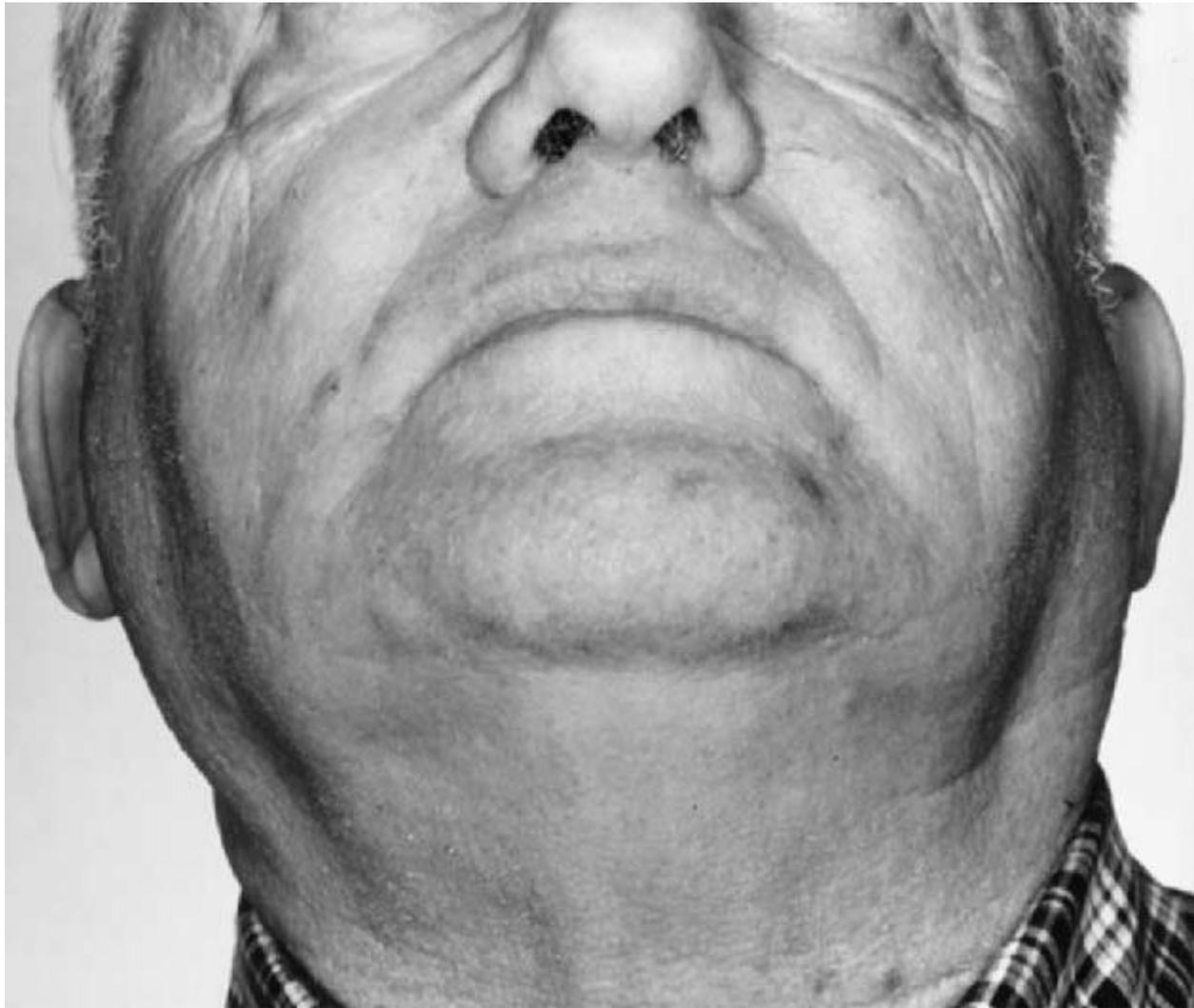
- Oral manifestation: Rare—Fungal overgrowth
- Prevention of problems:
 - Select appropriate antibiotic, dosage, and duration.
 - Take precautions with prolonged antibiotic use in the elderly and those previously affected.
- Treatment planning modifications:
 - Schedule appointments when the patient is free of disease.



Spider angioma.



Patient with jaundice.



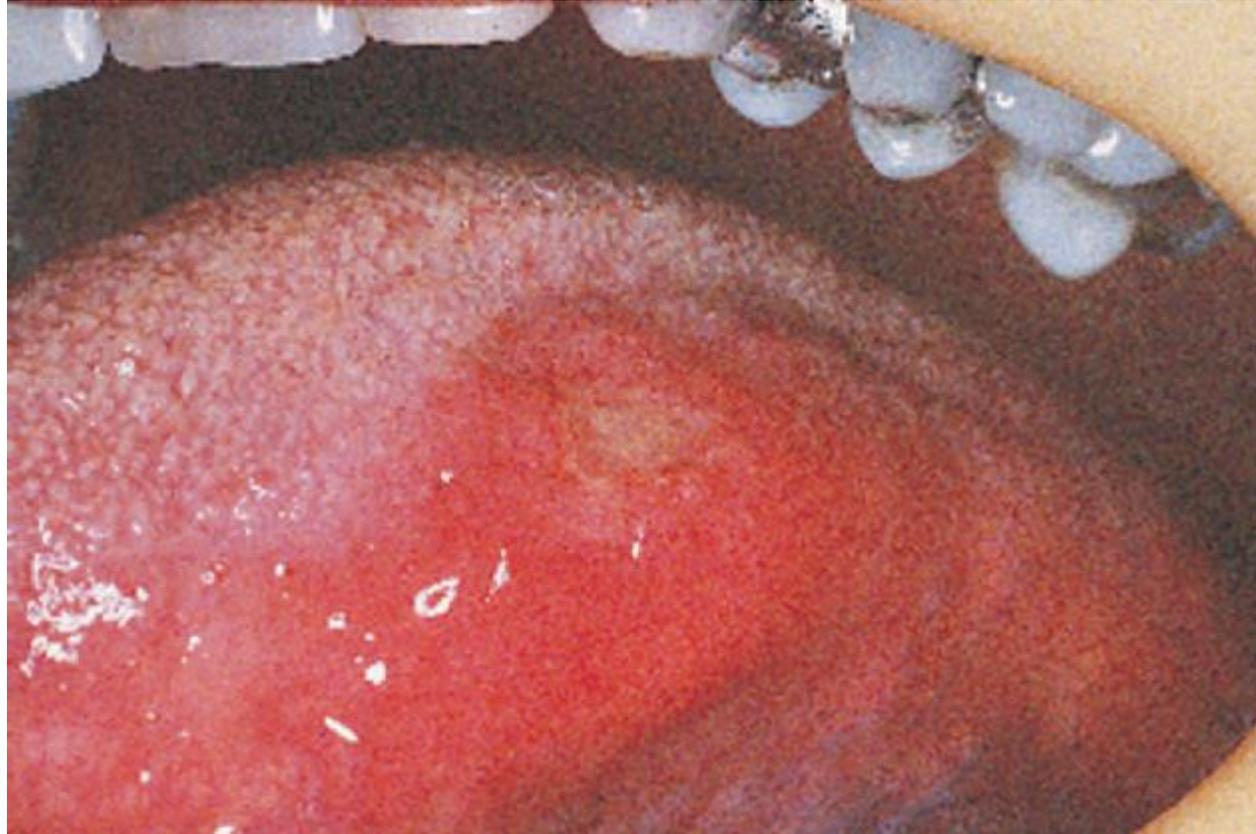
Painless enlargement of the parotid glands associated with alcoholism.



Median rhomboid glossitis caused by antibiotic use.



Perimylolysis. Destruction of palatal enamel of maxillary incisors in a patient with persistent regurgitation.



Oral ulceration associated with ulcerative colitis.



Pyostomatitis vegetans. Pustular raised lesions of palate in a patient with ulcerative colitis.