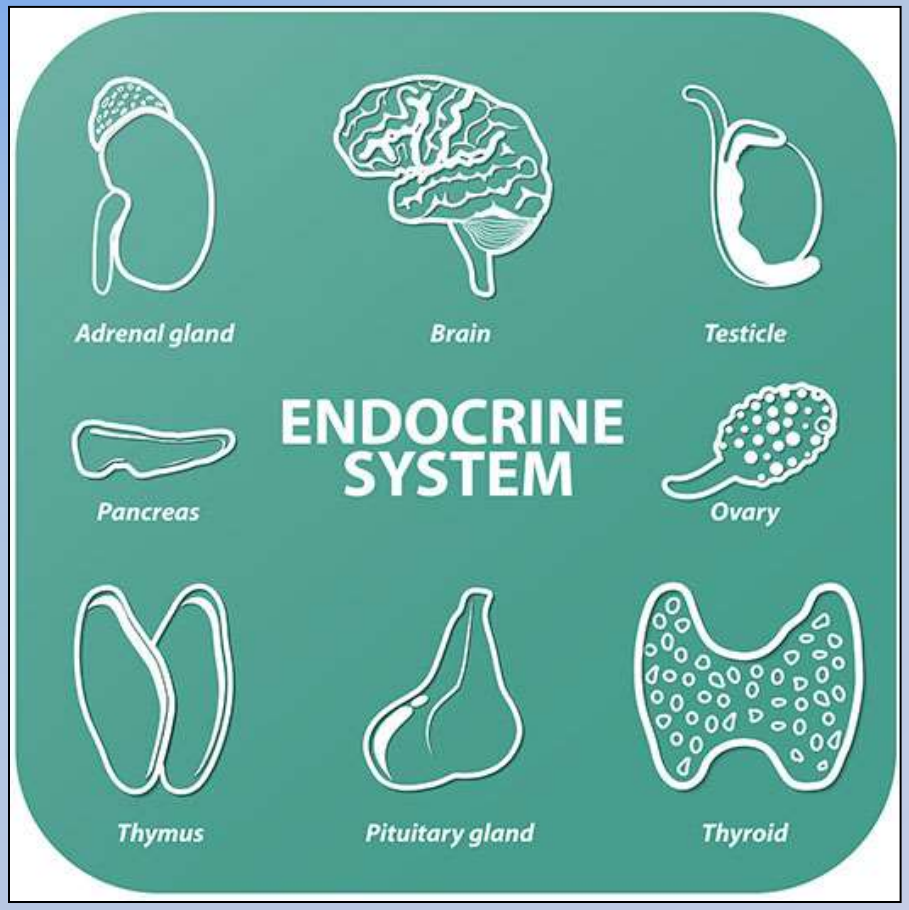


Endocrinal influence On Periodontal tissue

Dr. Sachin Bhagat

Contents

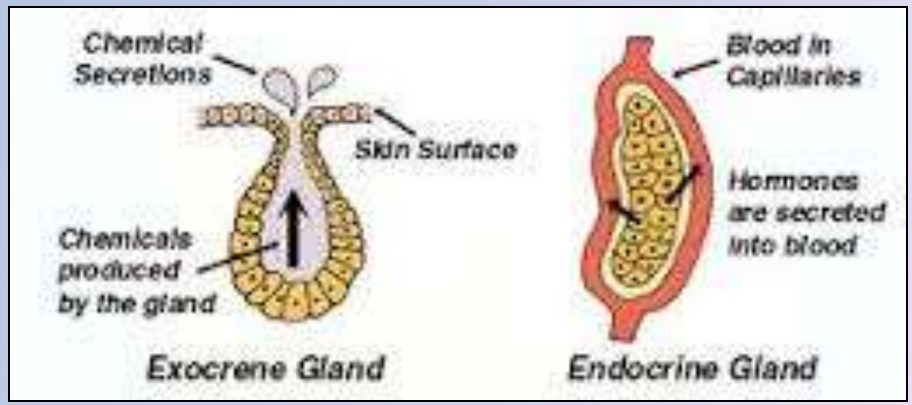
- Introduction
- Diabetes mellitus
- Female Sex hormones
  - Puberty
  - Menses
  - Pregnancy
  - Oral contraceptives
  - Menopause

- Hypopituitarism and Hyperpituitarism
- Hypothyroidism and Hyperthyroidism
- Hypoparathyroidism and Hyperparathyroidism
- Conclusion

Introduction

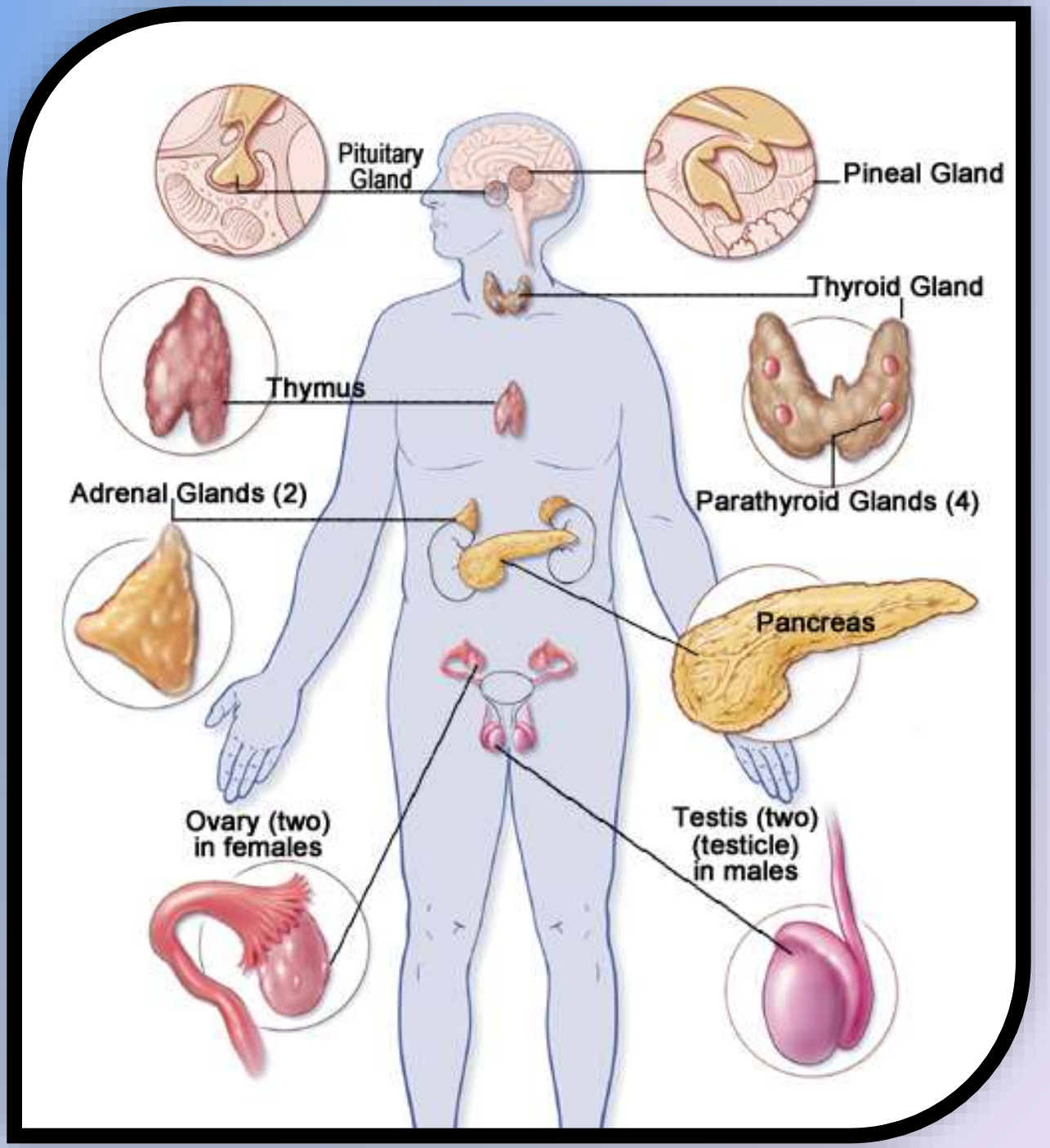
Endocrine word is derived from Greek language;
(endon – within, crinein – to set apart)

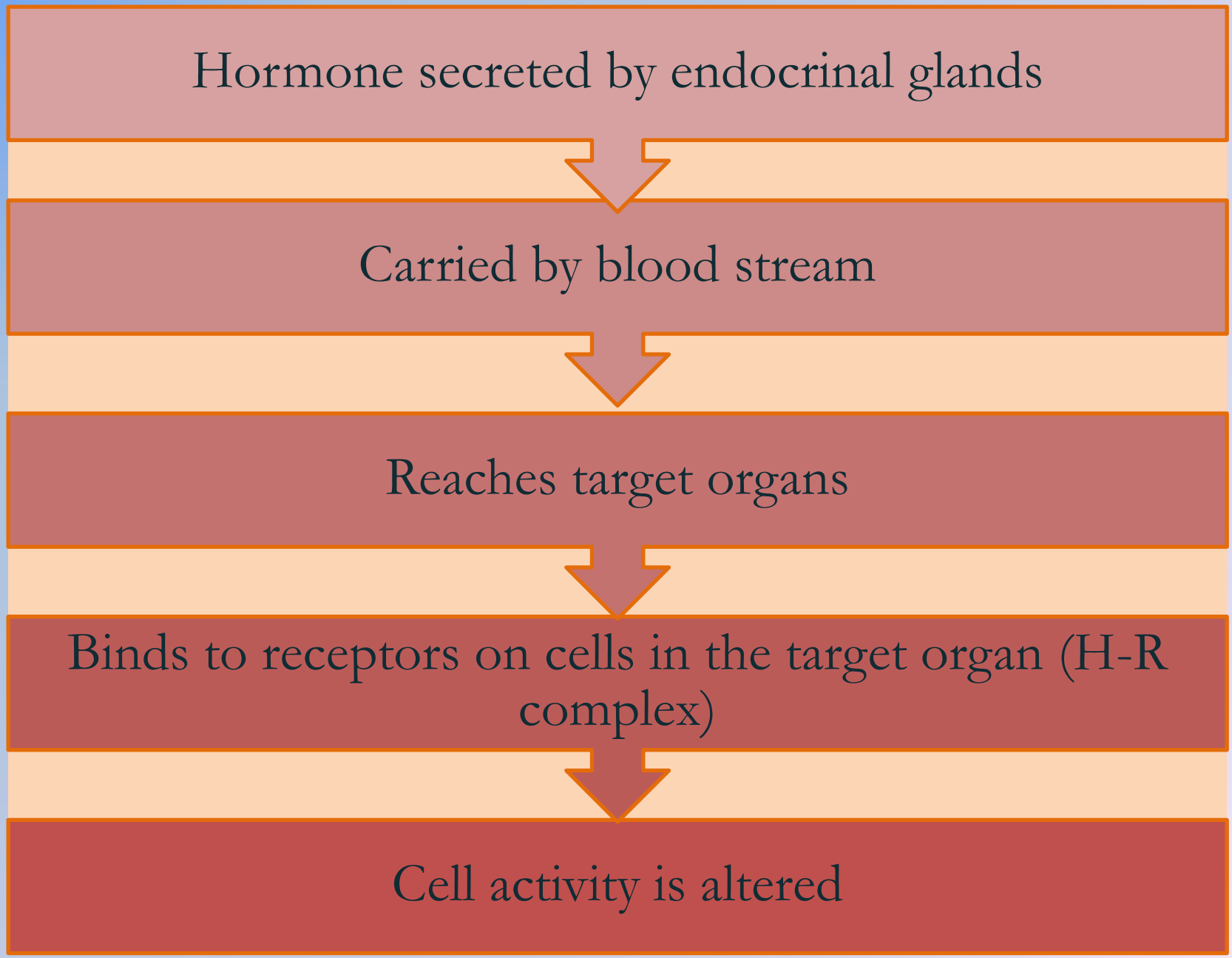
Endocrine e.g. pancreas
Exocrine e.g. salivary glands



Hormone

A chemical agent which is released from one group of cells and travel via the blood stream to affect one or more different groups of cells.


Pituitary Gland
Pineal Gland
Thyroid Gland
Thymus
Adrenal Glands (2)
Parathyroid Glands (4)
Pancreas
Ovary (two) in females
Testis (two) (testicle) in males


Hormone secreted by endocrinal glands
Carried by blood stream
Reaches target organs
Binds to receptors on cells in the target organ (H-R complex)
Cell activity is altered

Endocrine glands can overproduce (hyperfunction)

or underproduce (hypofunction) hormones.

Endocrine disorders may be caused :

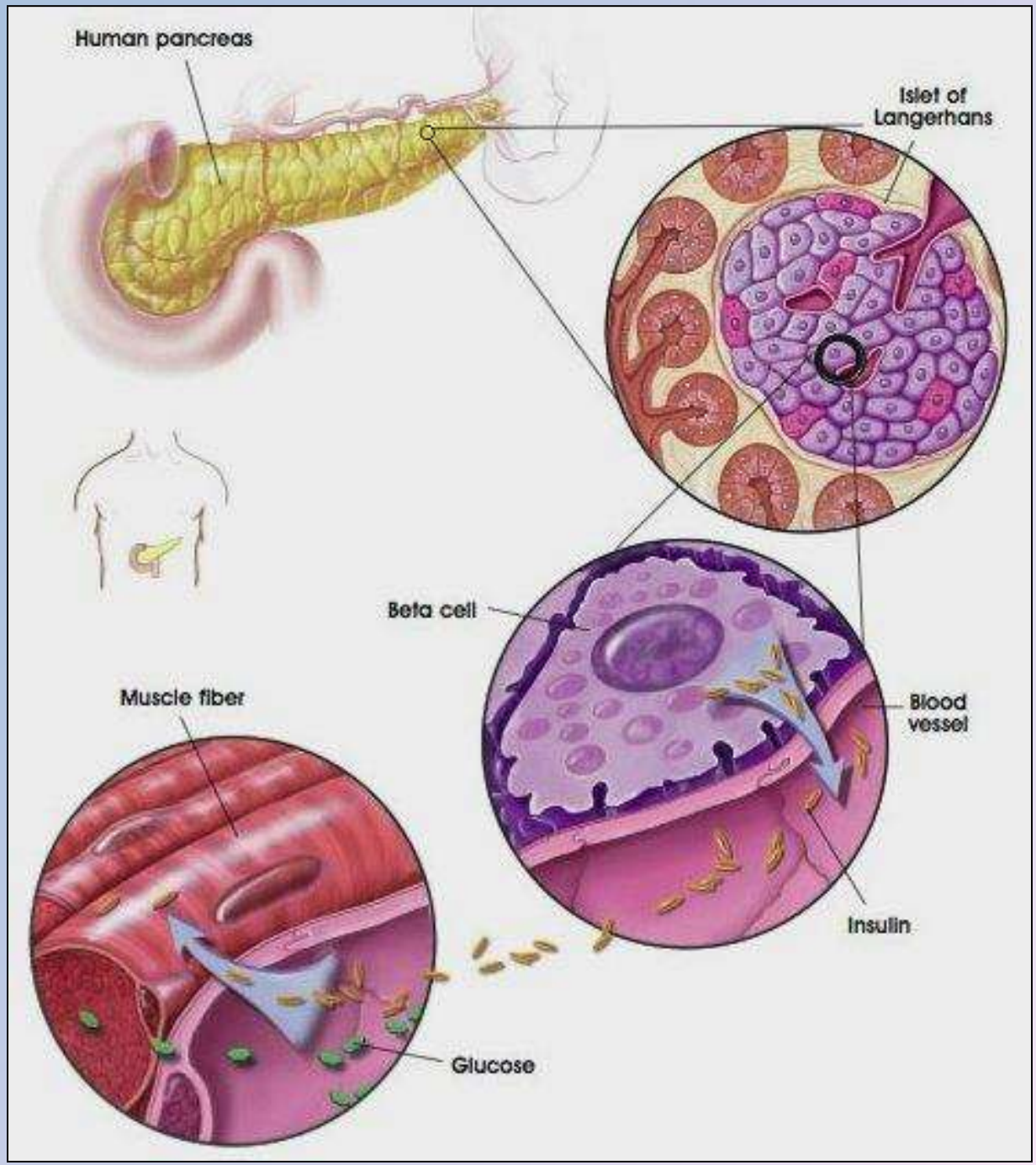
I. by hormone excess (e.g. primary hyperparathyroidism)

II. by diminished hormone release (e.g. hypothyroidism)

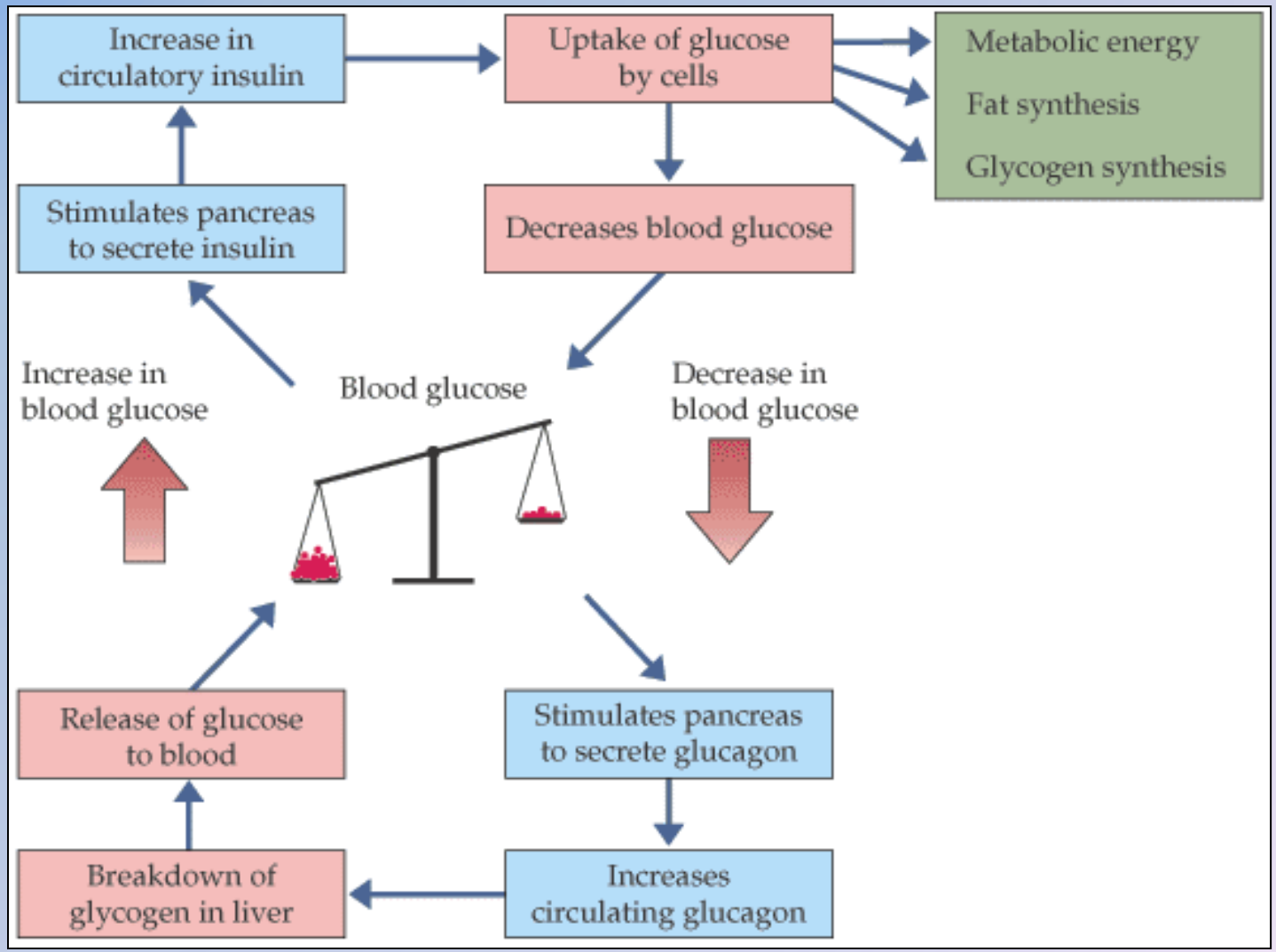
III. by hormone resistance (e.g. type 2 diabetes).

Diabetes mellitus

Insulin secretion

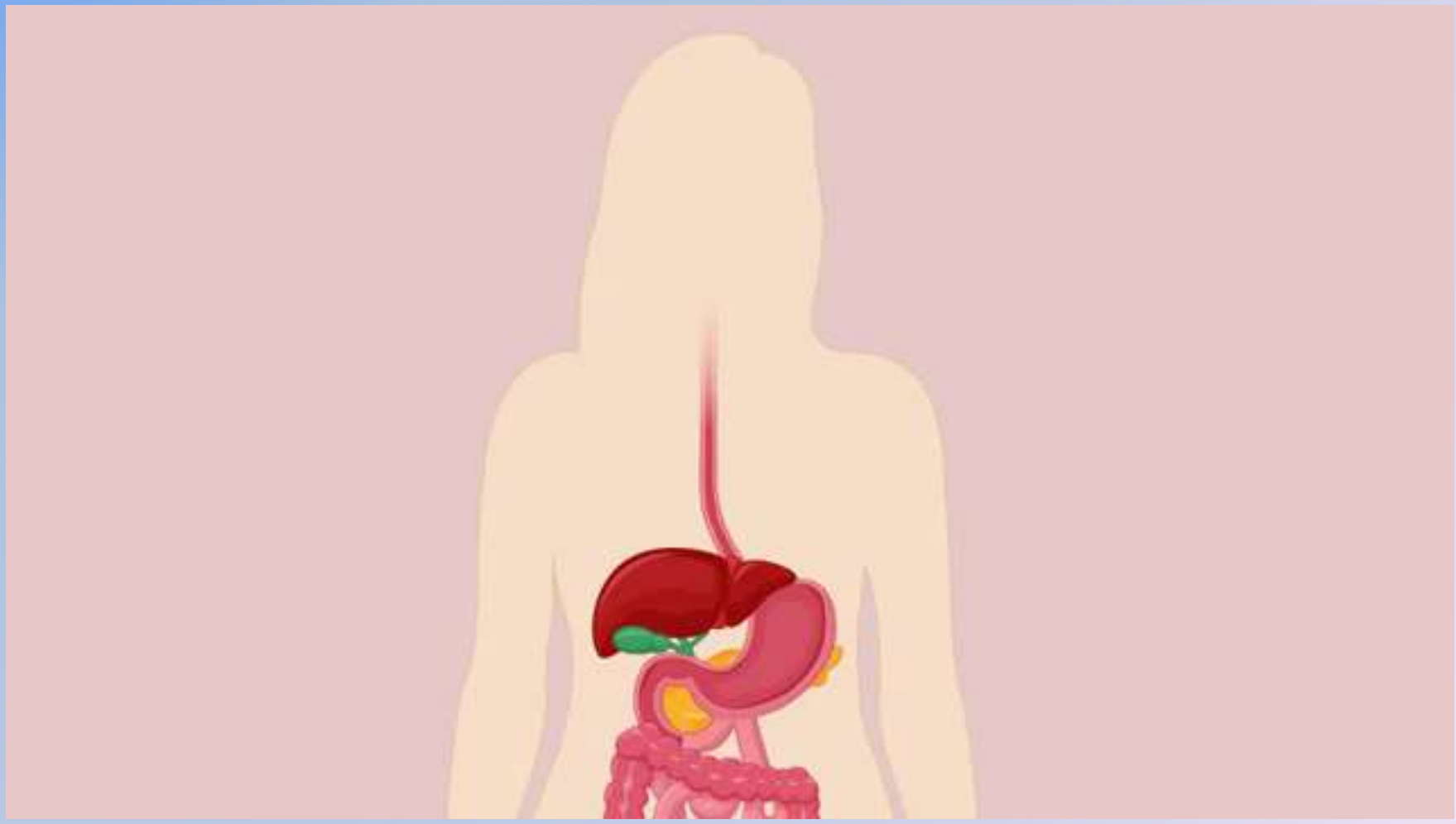


Blood glucose regulation



Diabetes mellitus

It is defined as a *complex metabolic disease*, characterized by chronic hyperglycemia caused by diminished insulin production, impaired insulin action or a combination of both, which results in the inability of glucose to be transported from blood stream into the tissues, which in turn results in high blood glucose levels.



TYPE 1 DM	TYPE 2 DM
Insulin dependent diabetes mellitus	Non-Insulin dependent diabetes mellitus
Autoimmune destruction of β cells	Resistance to insulin action Impaired insulin secretion
5-10%	90-95%
Children and young adults	Adults
Unstable and Difficult to control	Easy to control
Insulin injections	Diet and oral hypoglycemics
Ketoacidosis and coma	Ketoacidosis and coma - uncommon
Polydipsia, polyphagia, polyuria - evident	Polydipsia, polyphagia, polyuria – less severe form

Hyperglycemia secondary to diseases or condition

- Gestational diabetes
  - 2-10% of pregnancies
  - Increased risk of developing Type 2 DM

- Acromegaly
- Cushing's Syndrome
- Pancreactomy

Oral manifestations of Diabetes

- Mucosal drying
- Cheilosis
- Cracking
- Oral ulceration
- Burning mouth & tongue
- Diminished salivary flow
- Increased rate of dental caries
- Candida infection, mucormycosis
- Other bacterial infections involving hemolytic streptococci, and staphylococci

Periodontal Manifestation of Diabetes

- Frequent multiple periodontal abscesses – migratory abscesses
- Enlarged gingiva
- Polypoid gingival proliferations
- Severe gingival inflammation
- Deep periodontal pockets
- Rapid bone loss
- Poor healing


(C)


(D)


(E)

BACTERIAL PATHOGENS

Type 1 DM

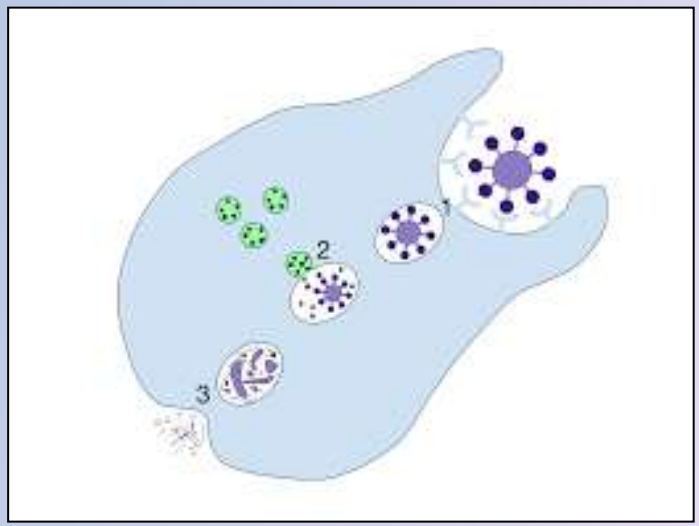
- Capnocytophaga
- Anaerobic vibrios
- actinomyces species

Type 2 DM

- P. Gingivalis
- P. Intermedia
- C. Rectus
- A. Actinomycetemcomitans

Neutrophil dysfunction

- Impaired chemotaxis
- Defective phagocytosis
- Impaired adherence



Primary defence in decreased

Increased bacterial proliferation

Altered collagen metabolism

Chronic Hyperglycemia

↓

Proteins undergo non enzymatic glycosylation

↓

Excess amounts of accumulated glycation end products (AGEs)

↓

Collagen becomes less soluble and less repair tendency

↓

More susceptible to pathogenic breakdown

Two-way relationship between periodontal disease and diabetes mellitus

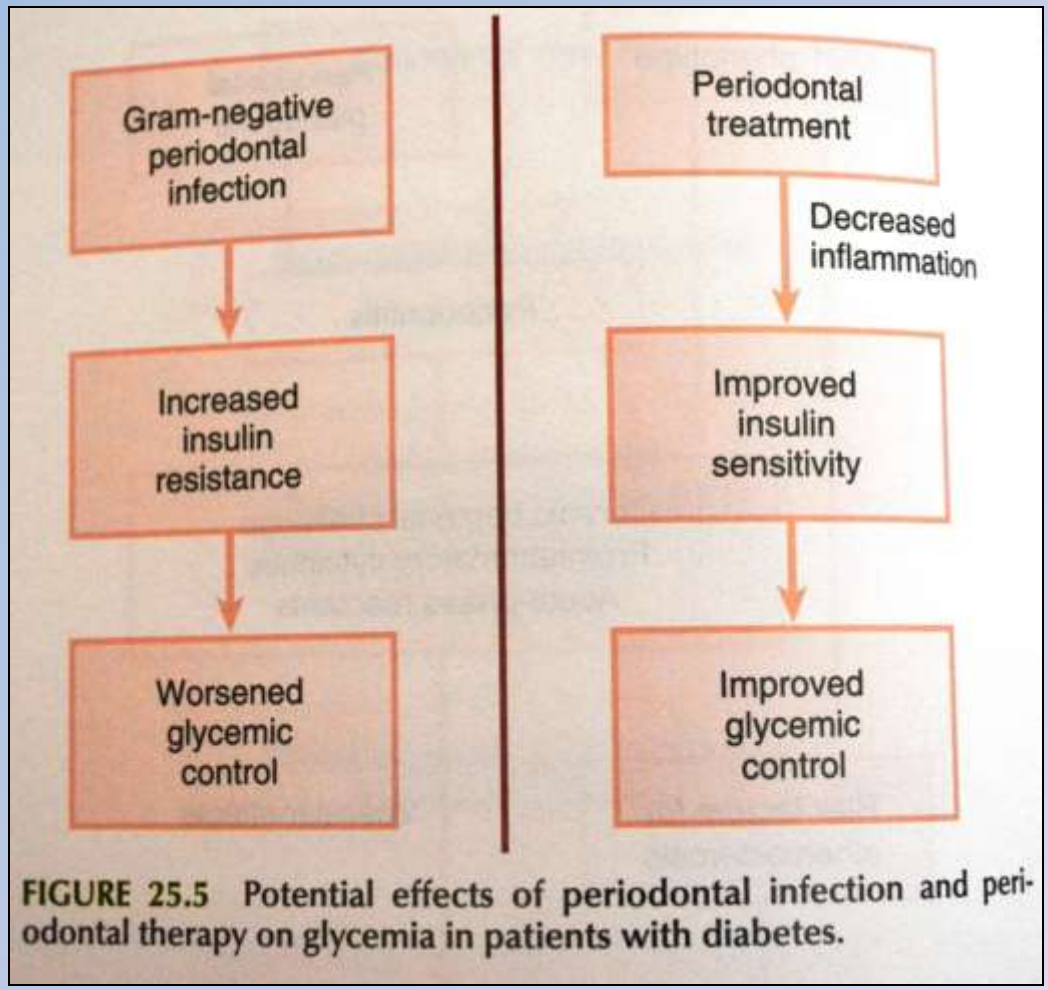


FIGURE 25.5 Potential effects of periodontal infection and periodontal therapy on glycemia in patients with diabetes.

COMPLICATION OF DIABETES MELLITUS

Microvascular complications

- Retinopathy - Blindness
- Nephropathy - Renal failure
- Neuropathy - Sensory disturbances

Macrovascular complications

- Cardiovascular (coronary artery disease)
- Cerebrovascular (stroke)

Altered wound healing

BOX 25.2 Complications of Diabetes Mellitus

1. Retinopathy
2. Nephropathy
3. Neuropathy
4. Macrovascular disease
5. Altered wound healing
6. Periodontal disease

From Löe H: Periodontal disease: the sixth complication of diabetes mellitus *Diabetes Care* 16(Suppl 1):329, 1993.

Diagnosis

BOX 30.2 Diagnostic Criteria for Diabetes Mellitus

Diabetes mellitus may be diagnosed by any one of three different laboratory methods. Whichever method is used, it must be confirmed on a subsequent day by using any one of the following three methods.

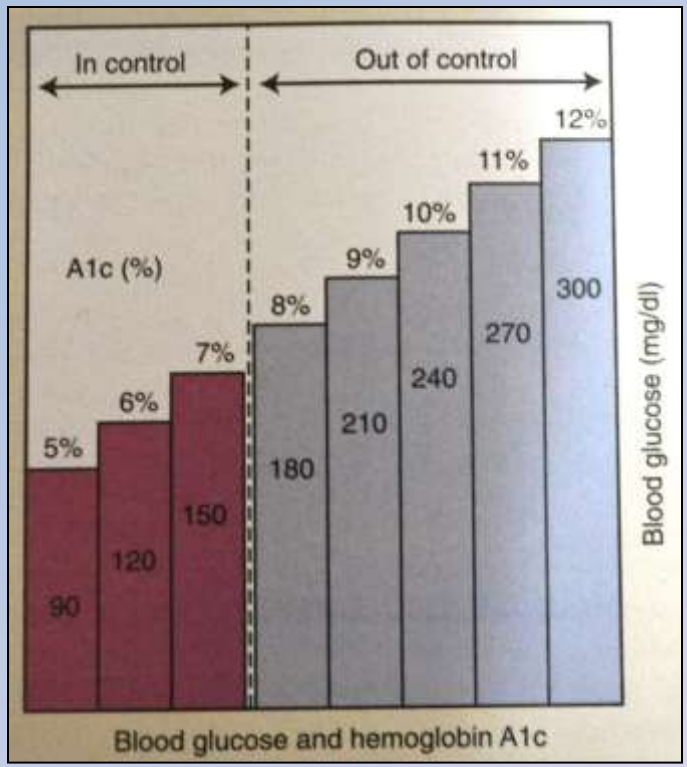
1. Symptoms of diabetes plus casual (nonfasting) plasma glucose ≥200 mg/dL. Casual glucose may be drawn at any time of day without regard to time since the last meal. Classic symptoms of diabetes include polyuria, polydipsia, and unexplained weight loss.
2. Fasting plasma glucose ≥126 mg/dL. "Fasting" is defined as no caloric intake for at least 8 h. (Normal fasting glucose is 70–100 mg/dL.)
3. Two-hour postprandial glucose ≥200 mg/dL during an oral glucose tolerance test.[a] The test should be performed using a glucose load containing the equivalent of 75 g of anhydrous glucose dissolved in water. (Normal 2-h postprandial glucose is <140 mg/dL.)

[a]The third method is not recommended for routine clinical use.

Data from American Diabetes Association: *Diabetes Care* 26(Suppl 1):5, 2003

BOX 30.3 Laboratory Evaluation of Diabetes Control: Glycosylated Hemoglobin (HbA1c) Assay[a]

4%–6%	Normal
<7%	Good diabetes control
7%–8%	Moderate diabetes control
>8%	Action suggested to improve diabetes control



Type 1 diabetes

- These patients have an absolute requirement of insulin for survival.

Short acting – Regular

Intermediate -- NPH(neutral protamine hagedorn)

Long acting – Levemir, Glargine

Rapid acting – Lispro, Aspart

Type 2 diabetes

Oral hypoglycemic agents currently available are

- Sulfonylureas
- Biguanides
- Glucosidase inhibitors
- Thiazolidinediones
- Meglitinides

Dental management of the diabetic patient

Key considerations include

- Physician’s consent
- Stress reduction,
- Diet modification,
- Antibiotic use,
- Changes in medication regimens, and
- Appointment timing.

Stress reduction

- Epinephrine and cortisol secretion - increased in stressful situations.
- elevate blood glucose levels and interfere with glycemic control.
- Reduce patient apprehension
- minimize discomfort
- preoperative sedation and analgesia.

Diet modification

- Never miss the meal and medication before the procedure
- Use glucometer to assess blood glucose levels
  - Sedation cases
  - Long procedures

Use of antibiotics

- Not required for controlled diabetic undergoing non surgical therapy
- Prophylactic Antibiotics recommended before surgical procedures

Changes in medication regimens

- Well controlled diabetic patients having routine periodontal treatment may take their normal insulin doses as long as they eat their normal meal
- Long procedures – dosage of the medication may be reduced
- Diet restrictions before surgery – conscious sedation - dosage of the medication may be reduced
- Diet restrictions after surgery - dosage of the medication may be reduced

Appointment timings

- greatest risk of hypoglycemia is usually during the time of peak insulin activity:

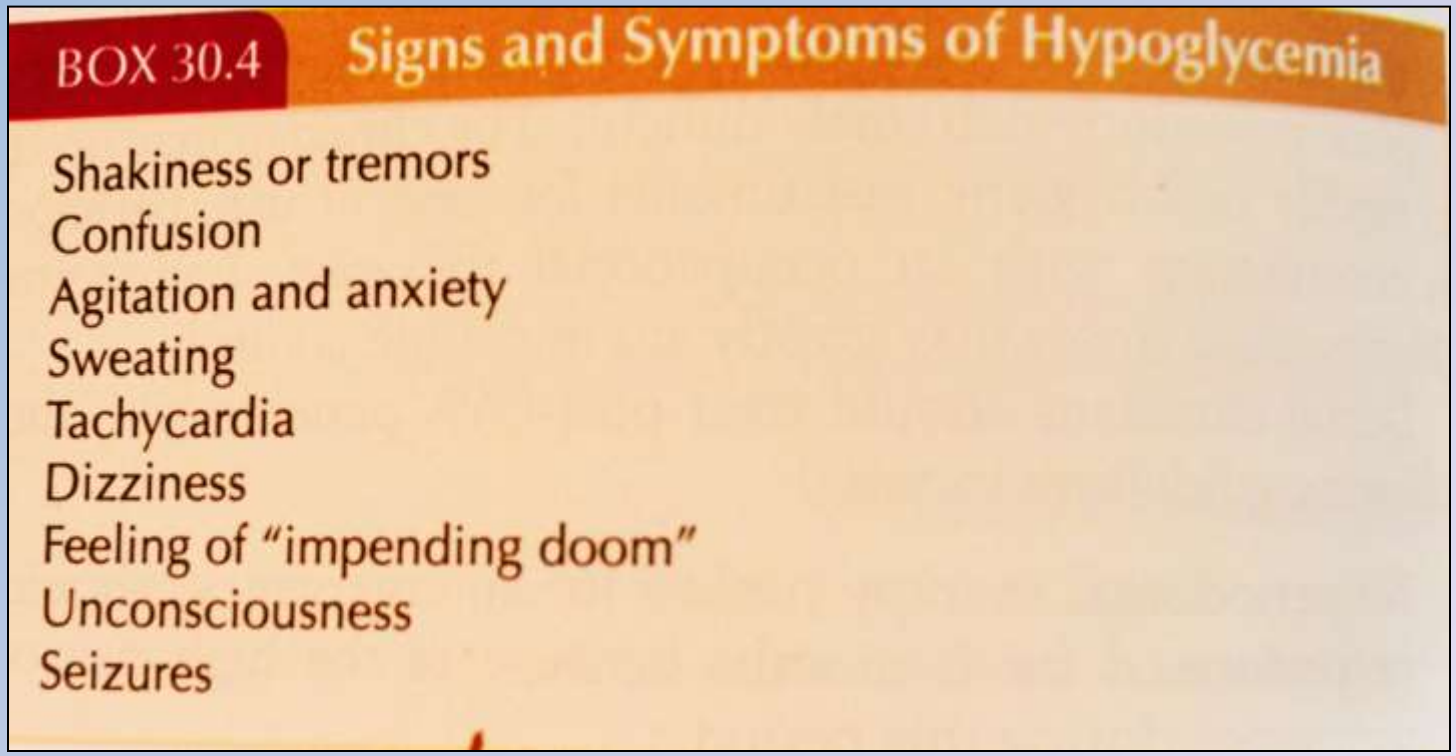
TABLE 37-6 Types of Insulin

Type	Classification	ACTIVITY		
		Onset	Peak	Duration
Lispro; Aspart; Glulisine	Rapid acting	15 minutes	30–90 minutes	<5 hours
Regular	Short acting	30–60 minutes	2–3 hours	4–12 hours
NPH	Intermediate acting	2–4 hours	4–10 hours	14–18 hours
Levemir	Long acting	1–2 hours	Relatively flat	up to 24 hours
Glargine	Long acting	6–8 hours	"Peakless" (has no peak in activity)	20–24+ hours

- plan dental treatment either before or after periods of peak insulin activity
- Limit the size of the surgical fields

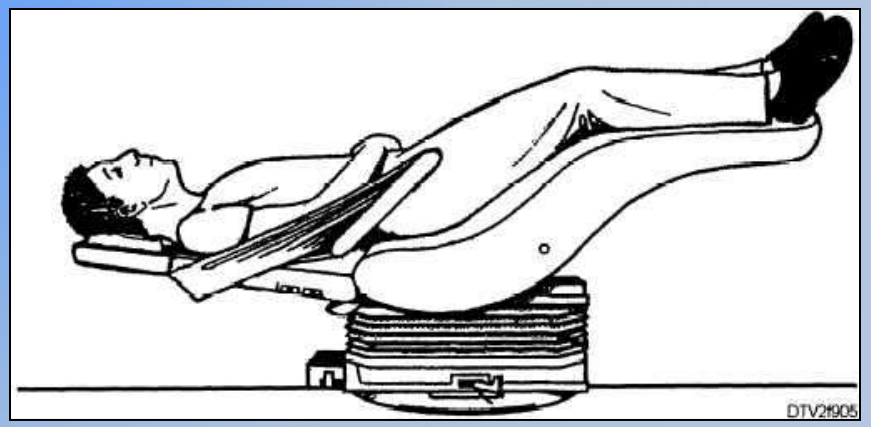
Hypoglycemia (Insulin Shock)

- Occcurs at BGL<60mg/dl



BOX 30.4 Signs and Symptoms of Hypoglycemia

Shakiness or tremors
Confusion
Agitation and anxiety
Sweating
Tachycardia
Dizziness
Feeling of "impending doom"
Unconsciousness
Seizures



Trendelenburg position

guidelines

1. Provide approximately 15 g of oral carbohydrate to the patient:
   a. 4–6 oz of juice or soda
   b. 3–4 tsp of table sugar
   c. Hard candy with 15 g of sugar
2. If the patient is unable to take food or drink by mouth, or if the patient is sedated:
   a. Give 25–30 mL of 50% dextrose intravenous (IV), which provides 12.5–15.0 g of dextrose, or
   b. Give 1 mg of glucagon IV (glucagon results in rapid release of stored glucose from the liver), or
   c. Give 1 mg of glucagon intramuscularly or subcutaneously (if no IV access is present).

Hyperglycemia (Ketoacidosis)

- Seen when BGL>180mg/dl
- Rarely seen in the dental office
- Takes days or weeks to develop
- Reduction of insulin or hypoglycemic drug

Signs and symptoms

- Increased thirst.
- Acetone breath/ fruity odour
- Headaches.
- Trouble concentrating.
- Blurred vision.
- Frequent urination.
- Fatigue (weak, tired feeling)
- Weight loss.

Assessment	Hypoglycemia	Hyperglycemia
History	• Too much of insulin • Little or no food	• Insufficient insulin • Excessive food consumption
Onset	Usually rapid onset Common in patients with good glycemic control	Usually slow onset Common in patients with poor glycemic control
Blood glucose levels	Usually less than 60mg/dl	Above 180mg/dl
Signs and symptoms	Shakiness, sweating, dizziness, weakness, fatigue, headache, irritable	Polydipsia, polyuria, polyphagia, nausea, blurring of vision, drowsiness
Glucose load	Improvement is seen	No improvement is seen

Important questions

LAQs

- Enumerate different endocrine systems affecting periodontium and describe diabetes mellitus in detail.
- Describe the different endocrine systems affecting periodontium

SAQs

- Differences between Type 1 and Type 2 DM
- Diagnostic tests for diabetes Mellitus
- Periodontal manifestations of DM
- Complications of DM

VIVA

- Signs and symptoms of hypoglycemia and hyperglycemia
- Periodontal microorganism seen in Type 1 and Type 2 DM
- Normal ranges of blood glucose level
- Significance of HBA1c test

THANK YOU