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ANESTHETIC CONSIDERATIONS IN DM

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Diabetes mellitus

results from an inadequate supply of insulin and/or an inadequate tissue response to insulin,

yielding increased circulating glucose levels

with eventual microvascular and macrovascular complications.

DIAGNOSIS

As per American Diabetes association (ADA)

A normal fasting plasma glucose is 70 to 100 mg/dL.

A random plasma glucose concentration of 200 mg/dL or higher, with classical signs and symptoms (polyuria,polydyspia,unexplained weight loss)

or

A fasting (no calorie intake >8hrs)plasma glucose concentration of 126 mg/dL or higher

or

An abnormal oral glucose tolerance test (OGTT), in which the glucose concentration is 200 mg/dL or higher 2 hours after a standard carbohydrate load (75 gm of glucose).

Impaired Fasting Glucose

- Any fasting glucose between 101 and 125 mg/dL
- Increased risk for DM
- Must educate regarding risks and need for lifestyle modifications

HEMOGLOBIN A1c

- provides a valuable assessment of long-term glycemic control
- Erythrocyte hemoglobin is nonenzymatically glycosylated by glucose
- HbA1c is stable glycosylated hemoglobin
- Its percentage concentration indicates average plasma glucose
- concentration during the preceding 60 to 90 days.
- Performed 2 times a year.
- The normal range for Hb A1c is 4% to 6%.
- Increased risk of microvascular and macrovascular disease begins at a Hb A1c of 6.5%.
- **Lowering HbA_{1C} Reduces Risk of Complications**

Gestational diabetes

- Hyperglycemia first diagnosed during pregnancy
- Occurs in 2-5% of pregnancies
- Occurs due to placental hormone changes that effect insulin function (greater resistance)
- Screening usually occurs during the 24^{th}-28^{th} week in high risk patients
- Criteria for diagnosis is different than for Type 1 and Type 2
- Dietary changes are initial treatment and insulin is the only BG lowering agent used
- Better maternal glycemic control lowers the incidence of neonatal hypoglycemia and hyper bilirubinemia
- Postpartum BG levels usually return to normal
- Women with a history of gestational diabetes have a 20-50% chance of getting type II DM within 5-10 years

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DRUG THERAPY IN DM

- Oral Diabetic Medications
  - Sulfonylureas-gliclazide,tolbutamide
    increases insulin release
    longer duration of action
  - Biguanides-(Metformin)
    potentiates insulin
    causes lactic acidoses in dehydrated pts
  - Alpha-Glucosidase (acarbose)
    Inhibitors-decrease absorption of glucose
  - Nonsulfonylurea Secretagogues
    - Meglitinide
    - D-phenylalanine derivatives
  - Thiazolidinediones (TZDs)-rosiglitazone

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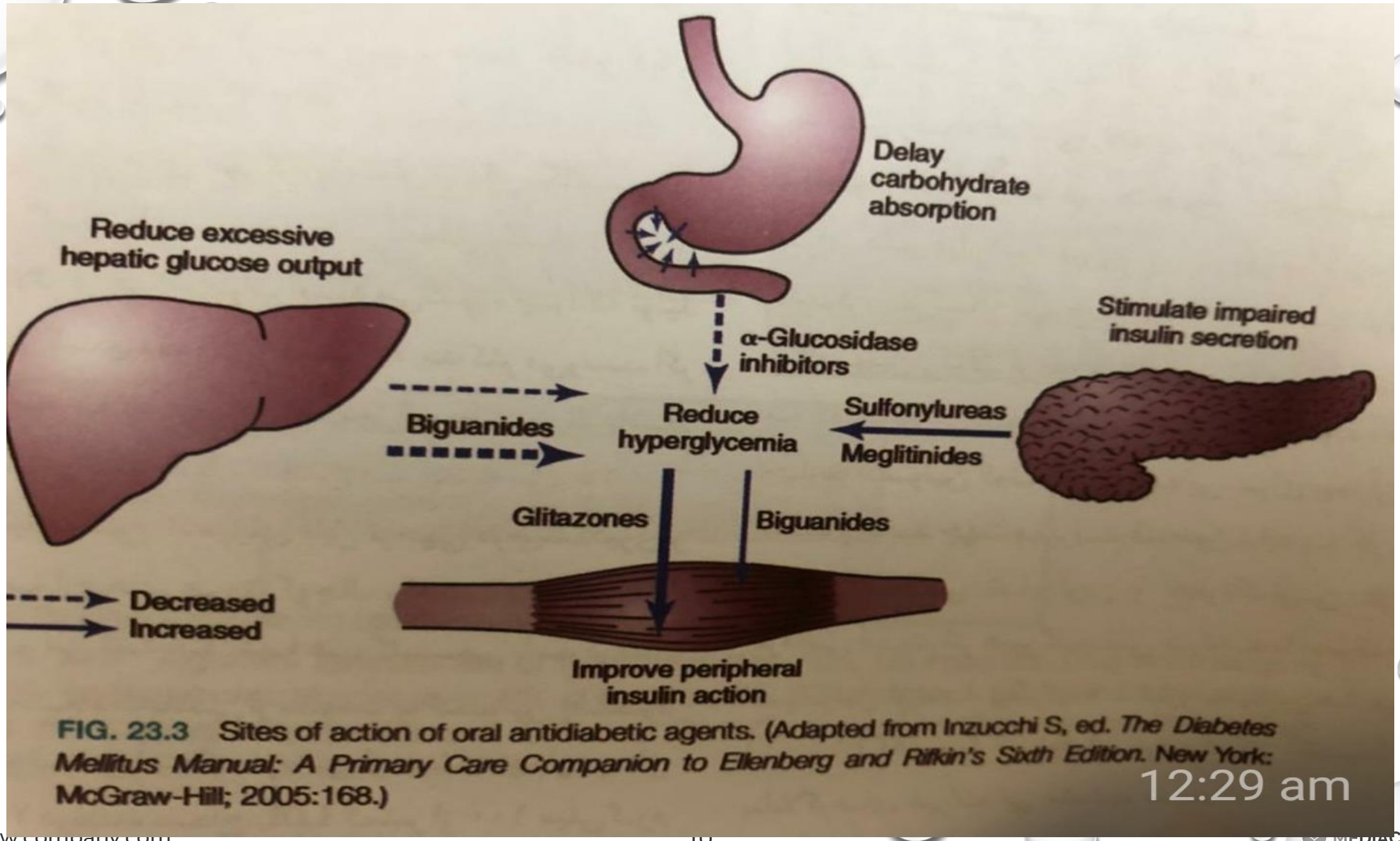

FIG. 23.3 Sites of action of oral antidiabetic agents. (Adapted from Inzucchi S, ed. *The Diabetes Mellitus Manual: A Primary Care Companion to Ellenberg and Rifkin's Sixth Edition*. New York: McGraw-Hill; 2005:168.)

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Complications of DM

- ACUTE
  - Metabolic
    - Diabetic ketoacidosis(DKA)
    - Hyperosmolar Hyperglycemic Nonketotic Syndrome(HHNS)
    - Hypoglycemia
  - Infections
- CHRONIC
  - Microvascular
    - Retinopathy
    - Nephropathy
  - Macrovascular
    - CAD
    - CVD
    - PVD
  - Neuropathies
  - Diabetic Foot Disorders
  - Psychosocial concerns

Diabetic Ketoacidosis

- **Definition** : Diabetic ketoacidosis (DKA) is a complex metabolic state of hyperglycemia, ketosis, and acidosis
- **Causes** :
  - Infection is the most frequent cause of diabetic ketoacidosis, particularly in patients with known diabetes
  - Missed insulin doses
- **Pathophysiology** :

  Hyperglycemia → Osmotic diuresis → loss of free water and electrolytes → Hypovolemia → tissue hypoperfusion and lactic acidosis

  The ketoacids (acetoacetate, beta-hydroxybutyrate, acetone) are products of proteolysis and lipolysis → KETOACIDOSIS

  Potassium is the most important electrolyte in patients with severe diabetic ketoacidosis (Hyperkalemia or Hypokalemia)

DIABETIC KETOACIDOSIS (DKA)

Signs and symptoms

- Hyperglycemia (>300mg/dl)
- Elevated serum ketones
- Acidosis as evidenced by pH of 6.8-7.3
- $HCO_3 < 18$
- Osmolarity <320
- Dehydration
- Electrolyte depletion
- GI distress (abdominal pain, N/V)
- Respiratory distress (Kussmaul's respirations and acetone breath odor)

TREATMENT OF DKA

Managing diabetic ketoacidosis (DKA) in an intensive care unit during the first 24-48 hours always is advisable.

- Correction of fluid loss with intravenous fluids
- Correction of hyperglycemia with insulin
- Correction of electrolyte disturbances, particularly potassium loss
- Correction of acid-base balance
- Treatment of concurrent infection, if present

Hyperosmolar Hyperglycemic Nonketotic Syndrome(HHNS)

- Four Clinical Features
  - Hyperglycemia (> 600-2000)
  - Mild or no ketosis
  - Hyperosmolality of plasma or serum (>340)
  - Profound dehydration
- 10-20% mortality rate with HHNS
- Risk factors include
  - Elderly with Type 2
  - Undiagnosed DM
  - Prolonged hyperglycemia
- Signs and Symptoms
  - Hypotension
  - Dehydration
  - Tachycardia
  - Decreased mentation
  - Neurologic abnormalities (focal)

WOUND HEALING & INFECTIONS

Wound healing is impaired in diabetic pts

Infections are the common cause of diabetic complications

- Alterations in leukocyte function-
  - ↓ chemostasis
  - Impaired phagocytic activity
  - Reduced intracellular killing
- Commonly seen infections
  - Cutaneous-furunculosis and carbuncles
  - Vulvovaginits
  - Cellulitis
  - UTI
  - Ear
- Must be treated aggressively

Gastrointestinal effects of DAN

- Affects 25% of diabetics
- impair gastric secretion and gastric motility, causing gastroparesis diabeticorum
- nausea, vomiting, early satiety, bloating, and epigastric pain.
- In DAN, the counterregulatory hormone responses to hypoglycemia are impaired and the warning signs eliminated, creating a dangerous situation of hypoglycemia unawareness.

Pre-op evaluation

Autonomic Neuropathy

- typical symptoms and signs of postural hypotension, gastroparesis, gustatory sweating, and nocturnal diarrhoea.
- It is worth assessing all diabetic patients for autonomic neuropathy.
- The easiest way is to assess heart rate variability. The normal heart rate should increase by over 15 beats/minute in response to deep breathing. Neuropathy is likely is there is less than 10 beats/minute increase

- Cardiovascular
- Diabetics are more prone to ischaemic heart disease (IHD), hypertension, peripheral vascular disease, cerebrovascular disease, cardiomyopathy and perioperative myocardial infarction.
- Ischaemia may be “silent” as a result of neuropathy. Routine ECG should be performed and appropriate stress testing if in doubt.
- Autonomic neuropathy can result in sudden tachycardia, bradycardia, postural hypotension and profound hypotension after central neuraxial blockade

Gastrointestinal

- Gastroparesis is characterised by a delay in gastric emptying without any gastric outlet obstruction. Increased gastric contents increase the risk of aspiration.
- Always ask about symptoms of reflux and consider a rapid sequence induction with cricoid pressure even in elective procedures.
- If available prescribe an H^2 antagonist such as ranitidine150mg plus metoclopramide 10mg, at least 2 hours preoperatively.

Renal

- Diabetes is one of the commonest causes of end-stage renal failure.
- Check urea, creatinine and electrolytes. Specifically check the potassium especially in view of the possible need for suxamethonium as a result of gastroparesis.
- If unavailable, proteinuria is likely to indicate kidney damage.
- Ensure adequate hydration to reduce postoperative renal dysfunction.

Anesthetic agents and diabetes mellitus

- etomidate inhibits adrenal steroid genesis and may induce a decrease in the glycemic response to surgery.
- alpha 2 agonist, clonidine, reduces sympathetic tone and the release of norepinephrine from nerve terminals. clonidine decreased circulating catecholamines & improved blood glucose control and decreased insulin requirements
- High doses of opiates induce hemodynamic ,hormonal and metabolic stability
- Avoid lactate containing solutions-cause hyperglycemia
- Diabetics less able to metabolise lipid emulsions-prolonged infusions of propofol should be avoided-can lead to hyperglycemia

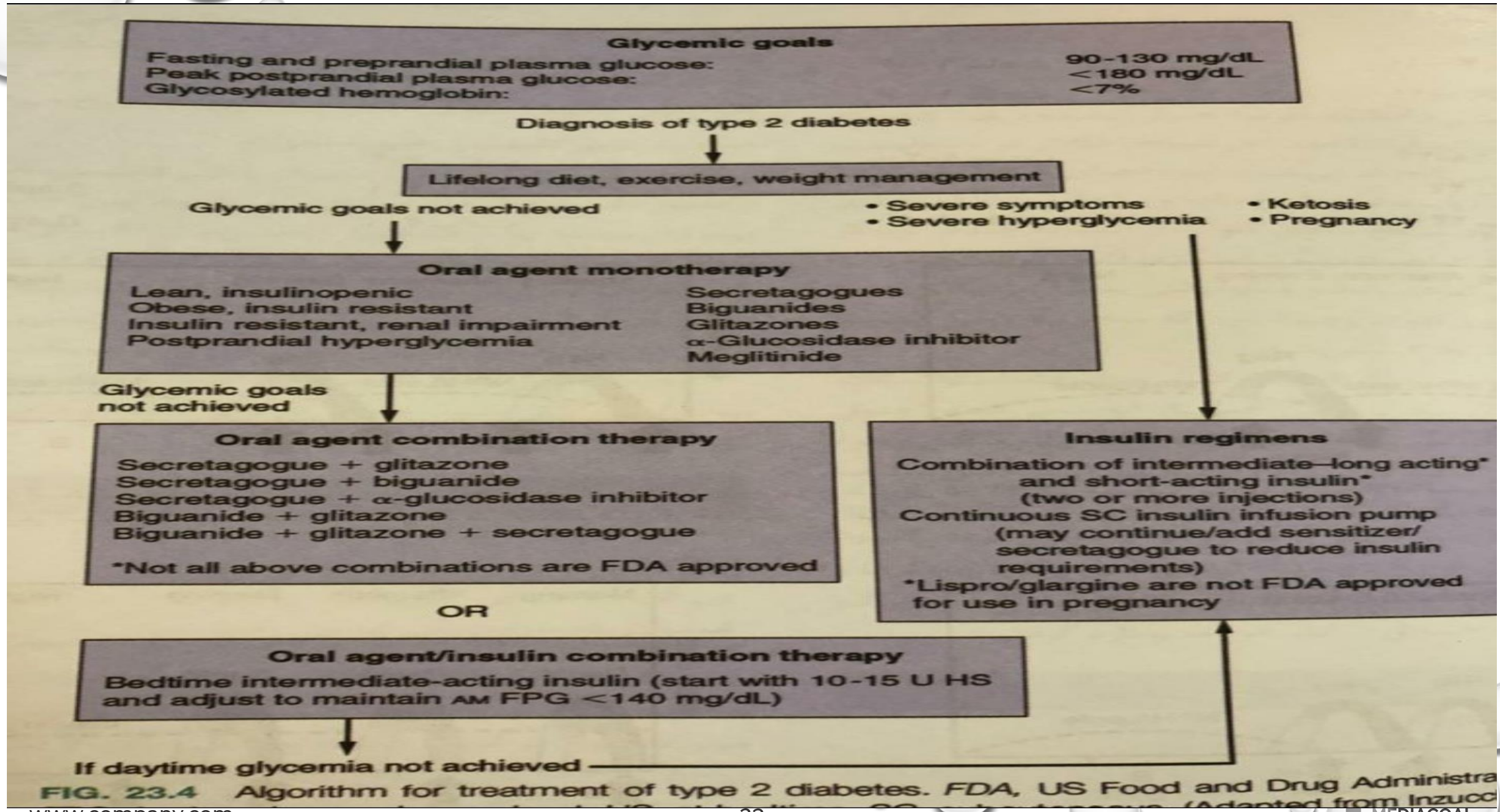

FIG. 23.4 Algorithm for treatment of type 2 diabetes. *FDA*, US Food and Drug Administra

Minor surgery, type 2 diabetes NOT on insulin (diet/tablet controlled)

- **1st on list**
- Preoperative blood sugar <180mg/dl Take normal medication inc. evening dose
- Preoperative blood sugar >180mg/dl Treat as if "Major" surgery
- Omit oral hypoglycaemic on morning of surgery
- Monitor blood glucose 1 hour preop; intraoperatively if over 1 hour; and 4hourly postop until eating.
- Recommence oral hypoglycaemics with first meal

Classic "Non-tight Control" Regimen

- Aim-to prevent hypoglycemia,ketoacidosis and hyperosmolar states
- protocol
  - NPO after midnight
  - 6AM –start i.v. dextrose 5% @ 125ml/hr/70 kgBW
  - ½ usual morning insulin dose s/c
  - Cont 5D intra op
  - Post op -monitor glucose and give insulin on sliding scale

Regional techniques

- are not contraindicated in the diabetic patient
- offer some potential advantages such as the avoidance of intubation, having an awake patient to warn of impending hypoglycaemia, and an earlier return to normal eating patterns.
- Document any existing motor/sensory neuropathies prior to performing any blocks
- look for evidence of autonomic neuropathy. If present, expect increased hypotension after neuraxial blocks.
- The chances of epidural abscess are also increased.

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