

Insulins

The oldest and most effective glucose-lowering therapy available, classified entirely by onset, peak and duration rather than by chemical target - the one glucose-lowering class with no absolute contraindication, because dose is titrated to the individual rather than fixed by the drug.

Online guide: <https://www.medguideglobal.com/pharmacology/endocrine-and-metabolic/insulins>

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Quick revision

One Liner: Insulins are classified entirely by pharmacokinetic profile - rapid, short/regular, intermediate (NPH) and long-acting - because they all activate the same insulin receptor; the basal-bolus concept pairs a background long-acting insulin with mealtime rapid or short-acting doses, and hypoglycemia is the universal dose-limiting risk across every formulation.

1. All insulins act on the same insulin receptor, a tyrosine kinase receptor; classification into rapid, short, intermediate and long-acting is entirely pharmacokinetic, not mechanistic. [7]
2. Rapid-acting analogs (lispro, aspart, glulisine) have an onset of about 5-15 minutes, peak around 30 minutes to 1-2 hours, and last 3-5 hours, matching them to mealtime (bolus) dosing. [7][1]
3. Regular human insulin has a slower onset (about 30 minutes) and longer duration (about 6-8 hours) than the rapid-acting analogs, and is the only insulin formulation that can be given intravenously, unlike the modern analogs. [7][5]
4. NPH is the intermediate-acting insulin: a cloudy suspension requiring resuspension before each dose, onset in 1-4 hours, peak around 4-8 hours (a median of about 6.5 hours in one label), typically dosed once or twice daily. [7][4]
5. Long-acting analogs (glargine, detemir, degludec) provide a relatively flat, peakless basal profile lasting roughly 24 hours (glargine) to more than 42 hours (degludec), intended to mimic background insulin secretion rather than cover a meal. [2][3]
6. A basal-bolus regimen pairs a long-acting insulin for background glucose control with a rapid- or short-acting insulin dosed before meals - the closest pharmacologic approximation of normal pancreatic insulin secretion. [7]
7. Hypoglycemia is described in every insulin product's own label as the most common adverse reaction associated with insulin therapy, and severe hypoglycemia can cause seizures, be life-threatening, or cause death. [2][1][3][4][5]
8. Unlike the oral and injectable non-insulin glucose-lowering drug classes, insulin has no absolute contraindication beyond hypersensitivity and active hypoglycemia in the labels reviewed here - dose is titrated to the individual patient rather than fixed by the drug. [7][2]

Overview

1. Insulin is the oldest hormone replacement therapy in modern medicine and remains the single most effective glucose-lowering treatment available - the ADA Standards of Care place insulin, alongside tirzepatide and select GLP-1 receptor agonists, among the agents producing the largest HbA1c reductions of any glucose-lowering class - with no ceiling on efficacy the way oral agents have - the dose can simply be increased until glycemic targets are met, within the limits set by hypoglycemia risk. Every insulin formulation activates the same insulin receptor and produces the same downstream effect: translocation of glucose transporters to the cell surface in skeletal muscle and fat, increased peripheral glucose uptake, and suppression of hepatic glucose production. Because the mechanism is identical across formulations, the entire classification system for insulin is pharmacokinetic rather than mechanistic - it is organised by how fast a formulation starts working, when it peaks, and how long it lasts. [7][8]
2. Rapid-acting insulin analogs - lispro, aspart and glulisine - are engineered to be absorbed faster than native human insulin, with onset in roughly 5 to 15 minutes, a peak around 30 minutes to 1-2 hours, and a duration of about 3 to 5

hours. This profile matches them to mealtime, or bolus, dosing. Regular human insulin, by contrast, is unmodified recombinant human insulin with a slower onset of about 30 minutes and a longer duration of roughly 6 to 8 hours; it is also the only insulin formulation in this comparison that can be administered intravenously, which matters specifically in acute inpatient settings such as diabetic ketoacidosis management, where the rapid-acting analogs are not the labelled option. [7][5]

3. NPH insulin (isophane insulin suspension) is the intermediate-acting formulation, created by adding protamine to regular human insulin to slow its absorption. It is a cloudy suspension that must be gently resuspended - rolled and inverted, not shaken - before every dose, with an onset of 1 to 4 hours and a peak effect described in one label as a median of about 6.5 hours. Long-acting analogs - glargine, detemir and degludec - sit at the other end of the spectrum, designed for a relatively flat, peakless profile that approximates background (basal) insulin secretion. Glargine's labelled duration is around 24 hours; degludec's ultra-long profile extends to at least 42 hours after the final dose, with a roughly 25-hour half-life at steady state, which is why it can be dosed once daily at any time rather than at a fixed clock time. [7][4][2][3]

4. The clinical logic that ties these formulations together is the basal-bolus concept: a long-acting insulin supplies a steady background level that covers glucose production between meals and overnight, while a rapid- or short-acting insulin is dosed before each meal to cover the glucose load from food. Premixed formulations, which combine a fixed ratio of an intermediate-acting and a rapid- or short-acting insulin in a single injection, trade some of this flexibility for a simpler regimen with fewer daily injections. Across every formulation, hypoglycemia is the universal, dose-limiting adverse effect - described in each product's own label as the most common adverse reaction associated with insulin therapy, capable of causing seizures and, in severe cases, death. [7][2][1]

Classification and drug examples

Intro: Insulins are classified by onset, peak and duration of action rather than by chemical target, since every formulation activates the same insulin receptor.

1. Name: Rapid-acting analogs

1. Note: Onset about 5-15 minutes, peak around 30 minutes to 1-2 hours, duration 3-5 hours; used for mealtime (bolus) coverage or in insulin pumps.

1. Name: Insulin lispro

Humalog

1. Route: Subcutaneous injection (intravenous use only under close medical supervision, per label)

1. Note: Faster onset and shorter duration than regular human insulin when given subcutaneously. [1]

2. Name: Insulin aspart

NovoLog

2. Route: Subcutaneous injection

2. Note: Comparable rapid-acting profile to lispro. [7]

3. Name: Insulin glulisine

Apidra

3. Route: Subcutaneous injection

3. Note: Comparable rapid-acting profile to lispro and aspart. [7] [7]

2. Name: Short-acting (regular) insulin

2. Note: Unmodified recombinant human insulin; onset about 30 minutes, duration 6-8 hours subcutaneously. The only insulin type in this comparison labelled for intravenous administration.

Name: Regular human insulin

1. Humulin R

2. Novolin R

Route: Subcutaneous injection or, under medical supervision, intravenous

Note: Can be given intravenously with close blood glucose and potassium monitoring, unlike the rapid- or long-acting

analogs. [5] [5]

3. Name: Intermediate-acting insulin (NPH)

3. Note: A cloudy protamine-containing suspension requiring resuspension before each dose; onset 1-4 hours, peak around 4-8 hours, typically dosed once or twice daily.

Name: NPH insulin (isophane insulin suspension)

1. Humulin N

2. Novolin N

Route: Subcutaneous injection only (not for intravenous use)

Note: Must be gently rolled and inverted, not shaken, to resuspend the cloudy formulation evenly before each dose. [4] [4][7]

4. Name: Long-acting analogs

4. Note: Designed for a relatively flat, peakless basal profile; once-daily dosing (glargine, degludec) or once/twice-daily (detemir).

1. Name: Insulin glargine

Lantus

1. Route: Subcutaneous injection only (not for intravenous use or insulin pumps)

1. Note: Median duration about 24 hours in clinical studies, with no pronounced peak; must not be diluted or mixed with any other insulin. [2]

2. Name: Insulin detemir

Levemir

2. Route: Subcutaneous injection

2. Note: Long-acting basal analog; not matched to a confirmed medicine_slug in this site's registry. [7]

3. Name: Insulin degludec

Tresiba

3. Route: Subcutaneous injection

3. Note: Ultra-long duration of at least 42 hours after the last dose, with a roughly 25-hour half-life at steady state, allowing flexible once-daily dosing at any time of day in adults. [3] [2][3]

5. Name: Premixed formulations

5. Note: Fixed-ratio combinations of an intermediate-acting and a rapid- or short-acting insulin in a single injection, trading dosing flexibility for a simpler regimen.

Name: Premixed insulin (e.g. 70/30 NPH/regular or lispro/lispro-protamine mixes)

1. Humulin 70/30

2. Humalog Mix 75/25

Route: Subcutaneous injection

Note: Combines a basal (protamine-suspended) component with a mealtime component in one pen or vial; not individually titratable the way separate basal and bolus insulins are. [7] [7]

Mechanism of action

Summary: Insulin lowers blood glucose by binding the insulin receptor, a transmembrane tyrosine kinase receptor present on skeletal muscle, adipose tissue and liver cells. Binding to the receptor's alpha subunits activates tyrosine kinase activity in the beta subunits, triggering an intracellular signalling cascade that causes glucose transporters (chiefly GLUT4) to translocate from intracellular stores to the cell surface, increasing peripheral glucose uptake. In the liver, insulin signalling suppresses gluconeogenesis and glycogenolysis, reducing hepatic glucose output. Insulin additionally inhibits lipolysis and proteolysis and promotes protein synthesis, effects relevant to its broader metabolic role beyond glucose alone. Every insulin formulation - rapid, short, intermediate or long-acting - shares this identical receptor-level mechanism; formulations differ only in how their chemical modification or suspension changes the rate of subcutaneous absorption, not in what the hormone does once it reaches the receptor.

Target: The insulin receptor, a tyrosine kinase receptor on skeletal muscle, adipose tissue and hepatocytes

Cidal Static: Not applicable - a hormone-receptor signalling effect, not a cytotoxic or cytostatic one

Kill Kinetics: Glucose-lowering effect follows each formulation's own onset/peak/duration profile and requires ongoing dosing to be maintained

1. Step: 1

1. Title: Insulin binds the alpha subunits of the insulin receptor

1. Detail: The insulin receptor is a transmembrane tyrosine kinase receptor found on skeletal muscle, fat and liver cells. [7]

2. Step: 2

2. Title: Receptor binding activates tyrosine kinase activity in the beta subunit

2. Detail: This activation triggers an intracellular signalling cascade. [7]

3. Step: 3

3. Title: Glucose transporters translocate to the cell surface

3. Detail: GLUT4 transporters move from the cytoplasm to the cell membrane in skeletal muscle and adipose tissue, increasing glucose uptake from the bloodstream. [7]

4. Step: 4

4. Title: Hepatic glucose production is suppressed

4. Detail: Insulin signalling in the liver reduces gluconeogenesis and glycogenolysis, lowering the amount of glucose the liver releases into the circulation. [7]

5. Step: 5

5. Title: Insulin also inhibits lipolysis and proteolysis and promotes protein synthesis

5. Detail: These broader anabolic and anti-catabolic effects are part of insulin's physiologic role beyond glucose disposal alone. [7]

Major clinical uses

1. Drug: Rapid-acting insulin (lispro, aspart, glulisine)

1. Organism: Not applicable

1. Indication: Mealtime (bolus) glycemic control in type 1 and type 2 diabetes; a mainstay of therapy in type 1 diabetes per current ADA guidance

1. Line: combined with a long-acting or intermediate-acting insulin in a basal-bolus regimen, or used in an insulin pump

1. Note: Given within about 15 minutes before a meal or immediately after, depending on the specific product's label. [1][7]

2. Drug: Regular human insulin

2. Organism: Not applicable

2. Indication: Type 1 and type 2 diabetes mellitus; the labelled intravenous option for acute inpatient glycemic management

2. Line: mealtime coverage subcutaneously, or continuous/bolus intravenous dosing under close monitoring in acute care

2. Note: The only insulin formulation reviewed here explicitly labelled for intravenous use. [5]

3. Drug: NPH insulin

3. Organism: Not applicable

3. Indication: Type 1 and type 2 diabetes mellitus, background (basal) glycemic control

3. Line: once or twice daily, often paired with a rapid- or short-acting insulin for mealtime coverage

3. Note: A cloudy suspension requiring resuspension before each dose, distinguishing its handling from the clear analog solutions. [4]

4. Drug: Long-acting insulin (glargine, detemir, degludec)

4. Organism: Not applicable

4. Indication: Background (basal) glycemic control in type 1 and type 2 diabetes; degludec approved from age 1 year and older
4. Line: once daily (glargine, degludec) or once/twice daily (detemir), typically combined with mealtime insulin in type 1 diabetes
4. Note: Designed for a flat, peakless profile rather than mealtime coverage. [2][3]
5. Drug: Premixed insulin
5. Organism: Not applicable
5. Indication: Type 2 diabetes, primarily, where a simpler fixed-ratio regimen is preferred over separate basal and bolus dosing
5. Line: typically twice daily before meals
5. Note: Trades individualised dose adjustment for fewer daily injections. [7]

Pharmacokinetics

1. Drug: Insulin lispro
1. Route: Subcutaneous (intravenous only under close medical supervision)
1. Absorption: Rapid subcutaneous absorption; onset about 5-15 minutes
1. Csf Penetration: Not applicable
1. Metabolism: Degraded like endogenous insulin, principally hepatic and renal
1. Elimination: Hepatic and renal insulin-degrading pathways
1. Dose Adjust: Renal or hepatic impairment can prolong insulin action and increase hypoglycemia risk; dose is individualised accordingly [1][7]
2. Drug: Regular human insulin
2. Route: Subcutaneous or intravenous
2. Absorption: Onset about 30 minutes subcutaneously (about 10-15 minutes intravenously)
2. Csf Penetration: Not applicable
2. Metabolism: Cleared by the same hepatic and renal insulin-degrading enzymes as endogenous insulin
2. Elimination: Hepatic and renal
2. Dose Adjust: Renal or hepatic impairment can prolong action; potassium is monitored closely with intravenous use [5]
3. Drug: NPH insulin
3. Route: Subcutaneous only
3. Absorption: Slower than regular insulin because of the protamine suspension; onset 1-4 hours, peak around a median of 6.5 hours
3. Csf Penetration: Not applicable
3. Metabolism: Degraded like endogenous insulin
3. Elimination: Hepatic and renal
3. Dose Adjust: Renal or hepatic impairment can prolong action and increase hypoglycemia risk [4]
4. Drug: Insulin glargine
4. Route: Subcutaneous only
4. Absorption: Slow, sustained absorption gives a relatively flat profile with no pronounced peak over 24 hours
4. Csf Penetration: Not applicable
4. Metabolism: Degraded like endogenous insulin
4. Elimination: Hepatic and renal
4. Dose Adjust: Renal or hepatic impairment can prolong action and increase hypoglycemia risk; must not be diluted or mixed with any other insulin or solution [2]
5. Drug: Insulin degludec
5. Route: Subcutaneous only

- 5. Absorption: Ultra-slow, sustained absorption; onset about 1 hour, glucose-lowering effect lasting at least 42 hours after the last dose
- 5. Csf Penetration: Not applicable
- 5. Metabolism: Degraded like endogenous insulin
- 5. Elimination: Hepatic and renal
- 5. Dose Adjust: Renal or hepatic impairment can prolong action; half-life is approximately 25 hours at steady state regardless of dose [3]
- 1. This page gives no dose regimens by design. Insulin dosing is individualised to the patient's weight, renal and hepatic function, activity level, diet and glycemic targets, and belongs entirely with the treating clinician and a current prescribing reference, not on this page.
- 2. The single pharmacokinetic property that most separates insulin as a class from the oral and other injectable glucose-lowering agents is that dose has no fixed ceiling - it is titrated up or down against the individual's measured glucose response, which is also the reason insulin has no absolute contraindication beyond hypersensitivity and active hypoglycemia. [7][2]
- 3. Regular human insulin is the only formulation reviewed here labelled for intravenous administration; the analogs (lispro, glargine, degludec) and NPH are all subcutaneous-only, which is a genuinely different administration-route distinction rather than a matter of degree. [5][2][4]

Adverse effects

- 1. Effect: Weight gain
 - 1. Detail: A recognised effect of insulin therapy generally, related to its anabolic actions and improved glucose utilisation. [7]
- 2. Effect: Injection-site lipodystrophy, including lipohypertrophy
 - 2. Detail: Repeated injection into the same site can cause localized fatty tissue changes that alter subsequent insulin absorption; rotating injection sites is the standard mitigation described in the pharmacology literature. [7]
- 3. Effect: Local injection-site reactions
 - 3. Detail: Redness, swelling or itching at the injection site can occur with any subcutaneous insulin. [2]
- 1. Effect: Hypoglycemia
 - 1. Detail: Described in every insulin product's own label as the most common adverse reaction associated with insulin therapy; severe hypoglycemia can cause seizures, may be life-threatening, or cause death, and the effect can be prolonged or delayed with longer-acting formulations.
 - 1. Action: Blood glucose monitoring and dose individualisation are the primary mitigation; patients and caregivers are counselled on recognising and treating hypoglycemia promptly. [2][1][3][4][5]
- 2. Effect: Hypokalemia
 - 2. Detail: Insulin drives potassium into cells alongside glucose, which is why intravenous regular insulin use requires close potassium monitoring in acute care.
 - 2. Action: Serum potassium is monitored, particularly with intravenous dosing or in patients on other hypokalemia-inducing drugs. [5][7]
- 3. Effect: Fluid retention and heart failure with concomitant thiazolidinediones
 - 3. Detail: Thiazolidinediones can cause dose-related fluid retention, particularly when combined with insulin, which may lead to or exacerbate heart failure.
 - 3. Action: Patients on both a TZD and insulin are observed for signs and symptoms of heart failure; dose reduction or discontinuation of the TZD is considered if it develops. [1]
- 4. Effect: Serious hypersensitivity, including anaphylaxis
 - 4. Detail: Severe, life-threatening generalized allergy, including anaphylaxis, can occur with insulin, though it is uncommon.
 - 4. Action: Discontinue and seek urgent care for signs of a serious allergic reaction. [3]

1. Drug: Regular human insulin

1. Effect: The only formulation reviewed here labelled for intravenous administration, which changes both its onset (10-15 minutes IV) and its monitoring requirements (close glucose and potassium checks). [5]

2. Drug: NPH insulin

2. Effect: Must be gently resuspended (rolled and inverted, not shaken) before every dose because it is a cloudy suspension rather than a clear solution; inadequate resuspension can cause dosing inconsistency. [4]

3. Drug: Insulin degludec

3. Effect: Its ultra-long, roughly 25-hour half-life at steady state allows once-daily dosing at any time of day in adults, but the same property means a missed-dose correction or dose change takes longer to be reflected in glucose control than with shorter-acting basal insulins. [3]

4. Drug: Insulin glargine

4. Effect: Must not be diluted or mixed with any other insulin or solution, and is not given intravenously or via an insulin pump, unlike regular human insulin. [2]

Contraindications, precautions and interactions

1. Use during an episode of hypoglycemia - labelled across the class (lispro, glargine, degludec, NPH, regular human insulin). [1][2][3][4][5]

2. Known hypersensitivity to the specific insulin product or its excipients. [1][2][3][4][5]

3. Beyond these two, insulin as a class carries no absolute contraindication in the labels and pharmacology literature reviewed here - a genuine structural difference from the oral and other injectable glucose-lowering classes, because dose is titrated to the individual rather than fixed. [7]

1. Renal or hepatic impairment can prolong the action of any insulin formulation and increase hypoglycemia risk, requiring closer monitoring and dose individualisation rather than a fixed contraindication. [7]

2. None of the insulin products reviewed here is intended for diabetic ketoacidosis via the subcutaneous route in the specific formulations discussed (rapid-acting analogs, glargine, degludec, NPH); regular human insulin given intravenously is the labelled option in that acute setting. [2][3][5]

3. Concomitant thiazolidinedione use raises fluid-retention and heart-failure risk and warrants monitoring. [1]

1. With: Other glucose-lowering drugs, particularly sulfonylureas and DPP-4 inhibitors or GLP-1 receptor agonists

1. Effect: Additive glucose-lowering effect increases hypoglycemia risk; concomitant drug doses are often reduced when insulin is added. [6]

2. With: Thiazolidinediones (TZDs)

2. Effect: Increase fluid retention risk when combined with insulin, which can precipitate or worsen heart failure. [1]

3. With: Beta-blockers

3. Effect: Can mask the adrenergic warning symptoms of hypoglycemia (tremor, palpitations), delaying recognition. [5]

Comparison tables

Title: Classified by time course, not by target

Caption: Every insulin activates the same receptor; the clinically important differences are onset, peak and duration, which determine whether a formulation covers a meal or provides background coverage.

1. Key: category

1. Label: Category

2. Key: example

2. Label: Example

3. Key: onset

3. Label: Onset

4. Key: duration

4. Label: Duration

5. Key: role

5. Label: Typical role

1. Category: Rapid-acting

1. Example: Lispro

1. Onset: About 5-15 minutes

1. Duration: About 3-5 hours

1. Role: Mealtime (bolus) coverage; insulin pumps [7][1]

2. Category: Short-acting (regular)

2. Example: Regular human insulin

2. Onset: About 30 minutes

2. Duration: About 6-8 hours

2. Role: Mealtime coverage; the only type labelled for intravenous use [7][5]

3. Category: Intermediate-acting

3. Example: NPH

3. Onset: About 1-4 hours

3. Duration: Peak around a median of 6.5 hours; dosed once or twice daily

3. Role: Background coverage, older regimens [4]

4. Category: Long-acting

4. Example: Glargine

4. Onset: About 1-2 hours

4. Duration: About 24 hours, relatively flat

4. Role: Background (basal) coverage [2]

5. Category: Ultra-long-acting

5. Example: Degludec

5. Onset: About 1 hour

5. Duration: At least 42 hours after the last dose

5. Role: Background coverage with flexible dose timing [3]

High-yield exam pearls

1. Pearl: Every insulin activates the same insulin receptor - classification into rapid, short, intermediate and long-acting is purely pharmacokinetic, describing absorption speed, not a different mechanism.

1. Why: A frequently tested distinction from receptor-targeted drug classes, where different subclasses often do have genuinely different molecular targets. [7]

2. Pearl: Regular human insulin is the only formulation in this comparison labelled for intravenous use - the rapid-acting analogs, NPH and the long-acting analogs are subcutaneous-only.

2. Why: This is why regular insulin, not a rapid-acting analog, is the labelled choice for intravenous glycemic management in acute care. [5][2][4]

3. Pearl: Insulin has no absolute contraindication beyond hypersensitivity and active hypoglycemia - unlike the oral glucose-lowering classes, dose is titrated to the individual rather than fixed by the drug.

3. Why: A classic single-best-answer contrast question between insulin and every other glucose-lowering class covered in this mechanism family. [7]

4. Pearl: NPH is a cloudy suspension that must be resuspended by rolling and inverting, never shaking, before each dose.

4. Why: Inadequate resuspension is a real, testable source of dosing inconsistency specific to NPH and premixed formulations, not the clear analog solutions. [4]

5. Pearl: Insulin degludec's roughly 25-hour half-life at steady state is what allows flexible once-daily dosing at any time of day in adults, rather than a fixed clock time.

5. Why: Connects a pharmacokinetic number directly to a labelled practical dosing feature, a common way exam pearls are built for long-acting insulins. [3]

Common exam traps

1. Trap: Assuming a rapid-acting insulin analog can be substituted for regular human insulin in an intravenous infusion.
1. Correction: Regular human insulin is the formulation labelled for intravenous administration; the rapid-acting analogs and the long-acting analogs in the material reviewed here are subcutaneous-only. [5][1][2]
2. Trap: Believing insulin has the same absolute contraindications as an oral glucose-lowering drug (renal thresholds, hepatic thresholds, hypersensitivity to a specific chemical scaffold).
2. Correction: Insulin's only absolute contraindications in the material reviewed are hypersensitivity and use during an active hypoglycemic episode; renal and hepatic impairment change the dose needed rather than ruling the drug out. [7]
3. Trap: Treating NPH like a clear, ready-to-inject solution.
3. Correction: NPH is a cloudy suspension requiring gentle resuspension (rolling and inverting) before every dose - shaking or skipping this step is a real source of dosing variability. [4]
4. Trap: Assuming all long-acting insulins have an identical duration of action.
4. Correction: Glargine's labelled duration is around 24 hours with a median that can range up to and beyond that in some patients, while degludec's ultra-long profile lasts at least 42 hours after the final dose - a meaningfully different pharmacokinetic tail, not interchangeable numbers. [2][3]

Self-test questions

1. Question: What is the basis for classifying insulins into rapid, short, intermediate and long-acting categories?
1. Different molecular targets for each category
2. Pharmacokinetic differences in onset, peak and duration, since all insulins act on the same receptor
3. Different routes of administration only
4. Different indications for type 1 versus type 2 diabetes
1. Explanation: Every insulin formulation activates the same insulin receptor; the rapid/short/intermediate/long-acting categories describe how quickly each formulation is absorbed and how long it lasts, not a different mechanism. [7]
2. Question: Which insulin formulation is labelled for intravenous administration?
1. Insulin lispro
2. Insulin glargine
3. Regular human insulin
4. NPH insulin
2. Explanation: Regular human insulin can be administered intravenously under close medical supervision with glucose and potassium monitoring; the rapid-acting analogs, glargine, degludec and NPH in the material reviewed here are subcutaneous-only. [5]
3. Question: What must be done with NPH insulin before each dose, and why?
1. Nothing, it is a clear ready-to-inject solution
2. It must be gently rolled and inverted to resuspend the cloudy suspension evenly
3. It must be diluted with saline
4. It must be warmed to body temperature
3. Explanation: NPH is a protamine-containing cloudy suspension that settles between doses; the label instructs gently rolling and inverting the vial or pen, not shaking, to resuspend it evenly before each injection. [4]
4. Question: What distinguishes insulin's contraindication profile from that of the oral glucose-lowering drug classes?
1. Insulin has more absolute contraindications than oral agents
2. Insulin has no absolute contraindication beyond hypersensitivity and active hypoglycemia, because dose is titrated to the individual
3. Insulin cannot be used in renal impairment under any circumstances

4. Insulin and oral agents have identical contraindication profiles

4. Explanation: Because insulin dose has no fixed ceiling and is adjusted to the individual patient's glucose response, it carries no absolute contraindication beyond hypersensitivity and active hypoglycemia in the material reviewed, unlike oral agents with fixed dosing and renal/hepatic thresholds. [7]

5. Question: What is the basal-bolus concept in insulin therapy?

1. Using only one type of insulin for all purposes

2. Pairing a long-acting (basal) insulin for background coverage with a rapid- or short-acting (bolus) insulin dosed before meals

3. Giving insulin only during acute illness

4. Using premixed insulin exclusively

5. Explanation: A basal-bolus regimen combines a long-acting insulin that supplies steady background coverage with a rapid- or short-acting insulin dosed before meals to cover the glucose load from food, approximating normal pancreatic insulin secretion. [7]

6. Question: Which long-acting insulin has the longest labelled duration of glucose-lowering effect?

1. NPH

2. Insulin glargine

3. Insulin degludec

4. Regular human insulin

6. Explanation: Insulin degludec's labelled glucose-lowering effect persists for at least 42 hours after the final dose, with a roughly 25-hour half-life at steady state, longer than glargine's approximately 24-hour duration. [3][2]

Frequently asked questions

1. Question: Why are there so many different types of insulin instead of just one?

1. Answer: Because normal pancreatic insulin secretion has two components - a steady background (basal) level and mealtime (bolus) spikes - and no single formulation's absorption profile matches both. Rapid- and short-acting insulins are engineered or formulated to cover meals quickly, while intermediate- and long-acting insulins are formulated to provide a slower, steadier background level, so combining formulations approximates normal physiology more closely than any one insulin alone. [7]

2. Question: Is insulin more dangerous than oral diabetes medications?

2. Answer: Insulin's dose-limiting risk, hypoglycemia, is described in every insulin product's own label as its most common adverse reaction and can be severe. But 'dangerous' understates the comparison: insulin also has no absolute contraindication beyond hypersensitivity and active hypoglycemia, because the dose is individually titrated, whereas oral agents carry their own distinct contraindications and risks. The safety profile is different in kind, not simply worse or better. [7][2]

3. Question: Why does NPH need to be rolled instead of shaken?

3. Answer: NPH is a suspension, not a true solution - the protamine-insulin complex settles out between doses. The label instructs gently rolling and inverting the vial or pen to resuspend it evenly; vigorous shaking can introduce air bubbles or alter the suspension in ways that affect dose accuracy. [4]

4. Question: What is insulin lipohypertrophy and why does it matter?

4. Answer: Repeated injections into the same small area of skin can cause a localized build-up of fatty tissue at that site. Injecting into lipohypertrophic tissue can make insulin absorption slower and less predictable, which is why rotating injection sites is standard advice for anyone on long-term insulin therapy. [7]

References

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[2] LANTUS (insulin glargine) injection - prescribing information. DailyMed, U.S. National Library of Medicine .

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