Diagnosis and Medical Treatment

Read it before the workbook, or after it as a quick review

What your tests measure, what your medicines do, and what remission means, all through the physiology you already know.

1 What doctors measure and why

Blood glucose is the end result of several systems working together: the liver, pancreas, muscle and fat. Medicine seldom measures those systems directly. It measures their outcome, glucose. That makes sense, because long-term high glucose does much of the damage in diabetes, and risk rises steadily as average glucose rises. Diagnostic thresholds don't mark where the disease begins. They are points on that rising slope where risk is high enough to act, and any fall in glucose, however small, lowers harm.

The main test is **HbA1c**. Red blood cells carry **haemoglobin**, the protein that transports oxygen. Glucose slowly sticks to it by a predictable reaction called **glycation**, and the higher your average glucose, the more haemoglobin is glycated. Red cells live about 90–120 days, so HbA1c reflects your average glucose over roughly the past three months: a record, not a snapshot. It is reported as a percentage or in **mmol/mol**.

Result	Usual meaning
HbA1c 6.5% (48 mmol/mol) or above	Diabetes (confirmed by a repeat test if there are no symptoms)
HbA1c 6.0–6.4% (42–47) in the UK; 5.7–6.4% (39–47) in the US	High risk, or **prediabetes**
Fasting glucose repeatedly 7.0 mmol/L (126 mg/dL) or above	Diabetes

The **oral glucose tolerance test (OGTT)** measures how well you clear a set dose of glucose. HbA1c is the most convenient test because it shows exposure over time rather than one morning.

2 The problem starts well before diagnosis

High glucose usually arrives late. Insulin resistance can build for years while extra insulin keeps glucose near normal (Chapter 3), so by the time glucose climbs, the underlying problem is well established. It may show up as thirst, night-time urination or tiredness, or turn up in tests for something else. Prediabetes is a misleading name: glucose control is already abnormal, and the risk of diabetes and heart disease is already raised. It is not the last healthy state before diabetes. Progression isn't inevitable, though: some people stay stable and some return to normal, especially when the drivers change. Treat it as a chance to act, not a holding pattern. The same applies after diagnosis: left alone, control usually worsens, but you can take a different path.

3 How doctors treat diabetes

In theory, prediabetes triggers active prevention; in practice, it often brings brief advice and another test in a few months. After diagnosis, diet and exercise still count, and medicines may be added. There is no single drug ladder: the choice depends on your glucose, weight, heart and kidney health, other conditions, side effects and cost. Each drug class pulls on a part of the system you met in Chapters 2 and 3:

Class (example)	What it works on
Metformin	The liver's "leaky tap": less glucose released; a common, cheap starting medicine
GLP-1 receptor agonists (semaglutide)	Appetite, stomach emptying and glucose-dependent insulin release; some protect the heart
Dual GIP/GLP-1 agonists (tirzepatide)	Two gut-hormone pathways: often large falls in appetite and weight
SGLT2 inhibitors (empagliflozin)	The kidneys: some glucose leaves in urine; heart and kidney protection
DPP-4 inhibitors (sitagliptin)	Modestly boost the body's own gut hormones
Sulfonylureas (gliclazide)	Force the pancreas to release insulin regardless of glucose; risk of low glucose and weight gain
TZDs (pioglitazone)	Expand safe storage in subcutaneous fat, steering fat away from the liver
Insulin (glargine, aspart)	Replaces insulin the pancreas can no longer supply

GLP-1-based drugs have transformed diabetes and obesity care. They work mainly by reducing appetite, so people eat less without a constant fight. The deficit then drains visceral and ectopic fat, which is the same route to recovery the rest of this book uses. Several also cut heart attacks and strokes, and some protect the kidneys. The trade-offs: stomach and gut side effects are common, gallbladder problems affect a minority, and serious effects are uncommon. Some lost weight is muscle, so protein and resistance training become more important. They are usually long-term, because weight often returns when they stop. Normal glucose on these drugs is an excellent result, but it doesn't count as remission. This book's approach works well alongside them.

4 Benefits, trade-offs and physiology

Lowering high glucose with medicine cuts complication risk, and some drugs protect the heart and kidneys too. But a reading doesn't tell you *why* it improved: two people with the same HbA1c can have very different physiology underneath. Medicine and lifestyle aren't rivals. Medicines control glucose and reduce risk, while changes to food, weight, activity and sleep improve the conditions underneath. The aim is the best health you can safely reach. Relying on medicine while leaving the drivers untouched is a missed opportunity.

5 What remission means

Remission means an HbA1c below 6.5% (48 mmol/mol) for at least three months without glucose-lowering medication. That threshold is the edge of the diabetic range, so a fall from 8.0% to 6.2% counts, even though 6.2% is in the prediabetic range. Lower is still better. This book uses **partial remission** for remission with HbA1c in the prediabetic range, and **full remission** for remission with HbA1c in the normal range. These are descriptive terms, not official categories. In the UK and US, remission can be recorded in your medical notes, so ask your doctor. And remission is not a **cure**: glucose can rise again if the old conditions return, so keep having HbA1c tests and your eye, kidney and foot checks.

JOINING IT UP

Glucose is the reading on the dial and HbA1c its three-month record. Medicines move the reading by pulling on the liver, pancreas, kidneys, appetite or fat storage; draining excess fat changes what lies behind it, and leads to remission.