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- 2023 Dec 14;389(24):2221-2232
- 2023 Dec 14;389(24):2221-2232



Diabetes and Beyond

Guideline Review

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Piedmont

Real change lives here

OUTLINE

Section 1: Screening; diagnosis; glycemia targets; glycemic monitoring

Section 2: Managing comorbidities – obesity; hypertension; dyslipidemia; retinopathy, neuropathy, DM-related DKD/CKD, and CVD

Section 3: Managing prediabetes, T2D, and T1D –choosing glycemic targets, lifestyle interventions, oral/injectable medication (insulin therapy in T1D)

Section 4: Vaccine review in persons with DM – recommendations and CGM review

SECTION 1: SCREENING – Non-Pregnant Adults

Age ≥ 35 without other risk factors

First-degree relative with diabetes – if known T1DM relative or genetic risk – screen for DM1 Abs → refer for TZIELD if 2+ abs positive

History of CVD

Overweight or obese (BMI 25+)

Sedentary lifestyle

Member of an at-risk racial or ethnic group: Asian, African American, Hispanic, Native American (Alaska natives and American pacific islander)

HDL-c < 35 mg/dL (0.9 mmol/L) and/or a triglyceride level > 250 mg/dL (2.82 mmol/L)

IGT, IFG, and/or metabolic syndrome

SCREENING – Pregnancy

Gestational diabetes

Pregnant women 24-27 weeks – **universal screening is cost-effective**

One step approach – 2-hour 75gm glucose load (8 hour/overnight fasting)

Two step approach – 1-hour nonfasted BG post-50g load –
if >130 / to 140 then GDM presumed → confirmatory 3-hour 100gm 3-hour test

SECTION 1: SCREENING – Autoimmune Diabetes (DM1)

ADA recommends screening: Adults and children at risk for autoimmune DM

- Relatives who have type 1 diabetes (1st degree family have 15x greater risk)
- Personal or family history of autoimmune disease - Hashimotos disease, Graves, celiac disease
- Anyone with abnormal glucose at first dysglycemia (e.g. pre-diabetes); especially in those without a family history of type 2 diabetes

Antibodies include:

- Glutamic acid decarboxylase 65 AAB (GADA);
- Insulinoma-associated antigen 2 AAb (IA-2A);
- Insulin AAb (IAA);
- Zinc transporter-8 AAb (AnT8A)

DIAGNOSIS – Non-Pregnant Adults

FPG concentration ≥ 126 mg/dL (after ≥ 8 hours of an overnight fast), *or*

Plasma glucose (PG) concentration ≥ 200 mg/dL 2 hours post 75-g oral glucose load after an overnight fast of at least 8 hours, *or*

Symptoms of hyperglycemia (e.g., polyuria, polydipsia, polyphagia) and a random (non-fasting) PG concentration ≥ 200 mg/dL, *or*

A1C level $\geq 6.5\%$

Diagnosis of DM requires 2 abnormal test results

→ Either same sample or 2 abnormal results on samples drawn on different days.

→ Glucose level ≥ 200 mg/dL in the presence of symptoms for DM confirms the diagnosis of DM.

DIAGNOSIS – Non-Pregnant Adults

Normal	Prediabetes	Diabetes
FPG <100 mg/dL	IFG FPG ≥100 to 125 mg/dL	FPG ≥126 mg/dL
2-h PG <140 mg/dL	IGT 2-h PG ≥140 to 199 mg/dL	2-h PG ≥200 mg/dL Random PG ≥200 mg/dL + symptoms
A1C <5.5%	5.7% to 6.4% For screening of prediabetes ^a	≥6.5% Secondary ^b

Table 4

Glucose Testing and Hemoglobin A1C Interpretation

DIAGNOSIS – Pregnancy

Gestational diabetes TEST: 75gm vs 50gm +/- 3-hour glucose load

2-hour 75gm glucose load (8 hour/overnight fasting)

Glucose $>/$ 92mg/dl is **POS**;

1 hour $>/$ 180mg/dl is **POS**;

2-hour $>/$ 153mg/dl is **POS**

1-hour nonfasted BG post-50g load – if ≥ 130 to 140 then $\rightarrow$
confirmatory 3-hour 100gm 3-hour test

post fasting shows 2 pos tests – fasting ≥ 95 , 1-hour ≥ 180 ,
2-hour $>/155$, 3-hour >1 140

GLYCEMIA TARGETS – Non-Pregnant Adults

A1c <6.5% or below in non-pregnant adults (AACE)

Roughly- FPG < 110 and PPG < 140

Individual targets – TIR, A1c, GMI, FPG and PPG

Get to goal FAST < 3 months - URGENCY

A1c < 7% ADA – same-same

Non-healthy adults – A1c goal 7-8 – limited life, severe hypoglycemia

Pregnant – LOWER GOALS

Inpatient nonpregnant – HIGHER/LOOSER GOALS

GLYCEMIA TARGETS - Pregnancy

Target Ranges:

Fasting: Less than 95 mg/dL (5.3 mmol/L)

1-hour post-meal: Less than 140 mg/dL (7.8 mmol/L)

2-hour post-meal: Less than 120 mg/dL (6.7 mmol/L)

Goals for sensor glucose ranges in pregnancy:

Goal sensor glucose range 63–140 mg/dL (3.5–7.8 mmol/L):

TIR, goal >70% (63-140)

level 1 TBR, goal <4% TBR (<63 mg/dL):

level 2 TBR, goal <1% (<54 mg/dL)

Diabetes Care. 2024;47(8):1257-1275. doi:10.2337/dci24-0032

DKA	A. DKA Diagnostic Criteria	
	Diabetes/hyperglycemia	Glucose ≥ 200 mg/dL (11.1 mmol/L) OR prior history of diabetes
	Ketosis	β -Hydroxybutyrate concentration ≥ 3.0 mmol/L OR urine ketone strip 2+ or greater
	Metabolic Acidosis	pH < 7.3 and/or bicarbonate concentration < 18 mmol/L
HHS	B. HHS Diagnostic Criteria	
	Hyperglycemia	Plasma glucose ≥ 600 mg/dL (33.3 mmol/L)
	Hyperosmolarity	Calculated effective serum osmolality > 300 mOsm/kg (calculated as $[2 \times \text{Na}^+ (\text{mmol/L}) + \text{glucose} (\text{mmol/L})]$), OR total serum osmolality > 320 mOsm/kg $[(2 \times \text{Na}^+ (\text{mmol/L}) + \text{glucose} (\text{mmol/L}) + \text{urea} (\text{mmol/L})]$
	Absence of significant ketonemia	β -Hydroxybutyrate concentration < 3.0 mmol/L OR urine ketone strip less than 2+
	Absence of acidosis	pH ≥ 7.3 and bicarbonate concentration ≥ 15 mmol/L

Figure Legend:

The diagnosis criteria of DKA (A) and HHS (B).

GLYCEMIC MONITORING

A1c – semi-annually in all persons with DM; QUARTERLY if not at glycemia target

-fructosamine or CGM - if sickle cell, thalassemia, anemia, liver, renal disease (esrd)

INSULIN users – CGM or fingerstick minimum BID (ideal QID)

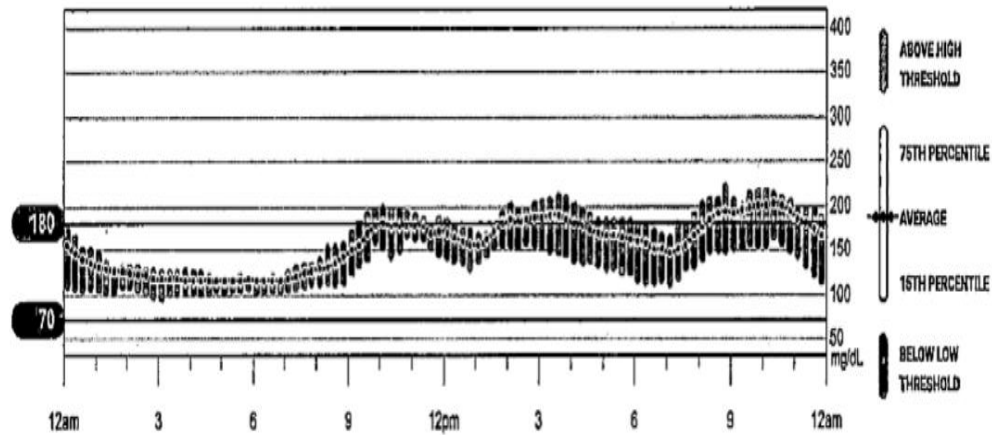
→ **EXPANDED recommendation for CGM to non-insulin**

MORE frequent monitoring:

- uncontrolled
- hypoglycemia or
- mealtime insulin

GLYCEMIC MONITORING - benefits

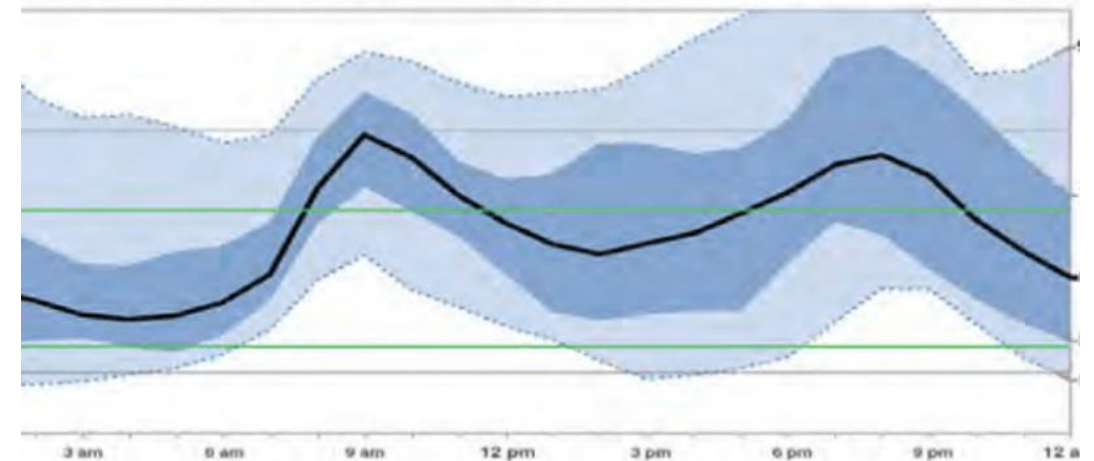
This graph shows your data averaged over 14 days



Feedback on human physiology –
feet on the floor phenomenon

GLUCOSE PROFILE (AGP)

glucose values from the report period, with median (50%) and other percentiles shown as if occurring in a single day



Effects of dietary choices

GLYCEMIC MONITORING

Realtime CGM (rtCGM) recommended

- Type 1 DM (ALL)
- Improves glycemia
- Lowers risk for DKA, hypo events

rtCGM is recommended in all persons with type 2 diabetes

- On insulin
- AT risk for OR experiencing hypoglycemia

TIR > 70%, TBR < 4% (< 70) and < 1% (< 54) goal

SECTION 2: Managing Comorbidities – HYPERTENSION

Goal BP in most T1DM, T2DM, pre-DM is **< 130/80mm/Hg**

To get THERE...start HERE:

- Registered dietician – healthy diet (Mediterranean)
- Weight management – 5-10% loss is goal
- Reduced sodium intake (DASH)
- Regular exercise – frequent!
- CDE for ongoing support, check-ins

HYPERTENSION in DM

IF NOT AT GOAL WITH LIFESTYLE/DIET – goal BP <130/80

Add – medication (after 2-3 months lifestyle)

ACE, ARB first, first, first, and always! (unless allergy etc.)

Add – diuretic, calcium channel blocker, alpha-beta or newer beta-block, mineralocorticoid receptor antagonist

REMEMBER – SGLT-2 class has BP/diuretic effects

Managing Comorbidities – DYSLIPIDEMIA

Over AGE 40 if T1DM, T2DM, pre-DM →

- Fasting lipid panel annually
- Increase frequency if monitoring therapy

LIFESTYLE

- Registered dietitian
- Healthy diet w focus on weight control
- Regular (frequent) exercise
- CDE for support, long-term behavior change

DYSLIPIDEMIA

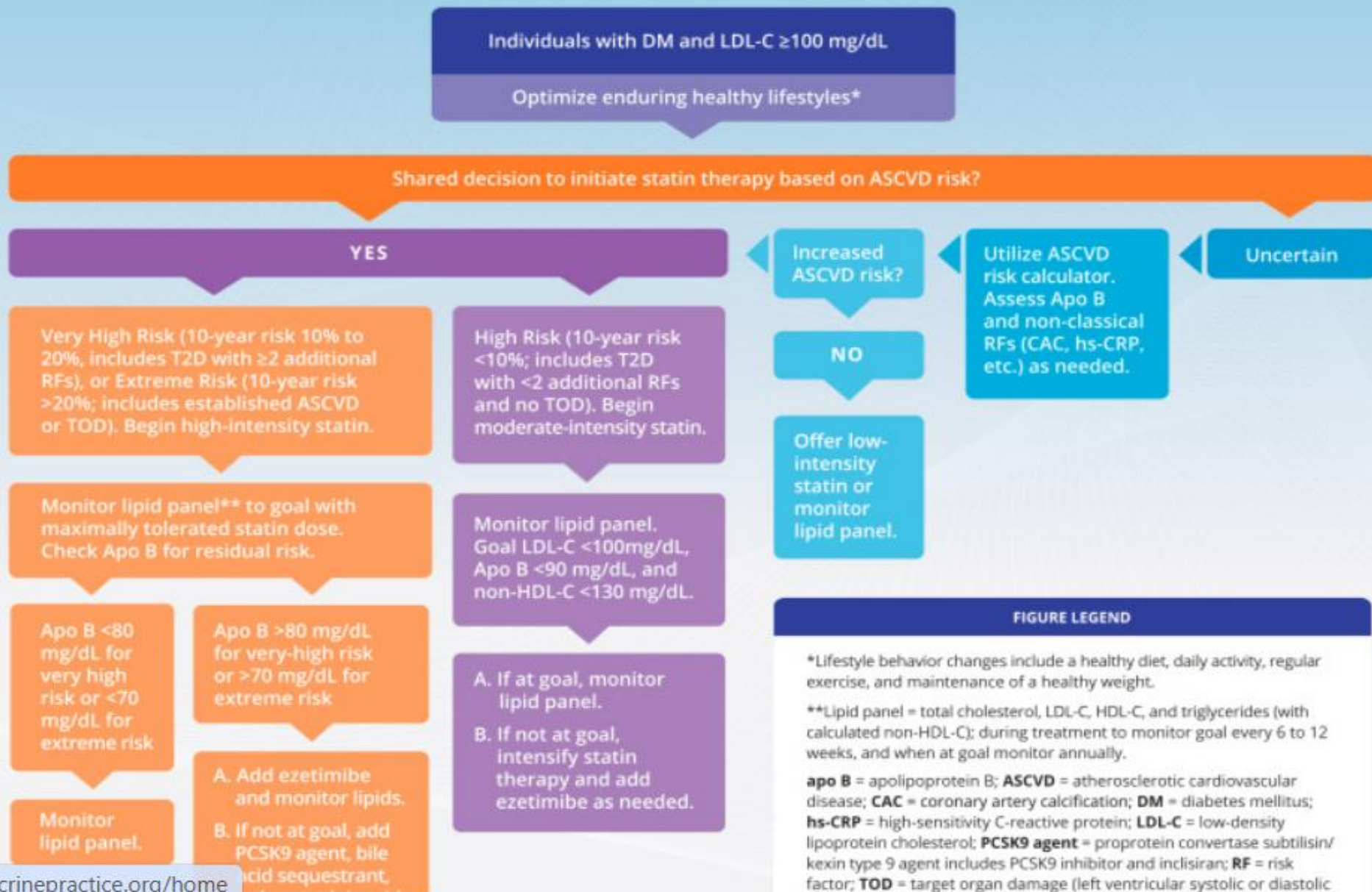
Pre-DM and T2DM with < 2 risk factors and no ASCVD →
→ shared decision-making, assess nontraditional risk factors –
(apoB, Lpa, CAC, c-rp) – **consider statin**

HIGH RISK for ASCVD regardless of DM type → **moderate
intensity statin**

VERY HIGH RISK (DM2 with 2 or more ASCVD risk factors) →
high intensity statin

Risk factors: advancing age, hypertension, CKD stage 3a, cigarette smoking,
family history of premature ASCVD in men <55 years and women <65 years, low
HDL-C, or high non-HDL-C

DECISION TREE FOR TREATING HYPERCHOLESTEROLEMIA IN PERSONS WITH DIABETES MELLITUS



DYSLIPIDEMIA

Treatment targets for persons in **high ASCVD risk category**:

LDL-C <100 mg/dL,
apolipoprotein B (apo B) <90 mg/dL,
non-HDL-C <130 mg/dL

Treatment targets for persons in a **very high risk ASCVD category**:

LDL-C <70 mg/dL,
apo B <80 mg/dL,
non-HDL-C <100 mg/dL

Treatment targets for persons with **extreme risk of ASCVD include**:

LDL-C <55 mg/dL,
apo B <70 mg/dL,
and non-HDL-C <90 mg/dL

Managing Comorbidities - DYSLIPIDEMIA

STATINS – first line-
monitor every 6-12 weeks
increase dose as needed to LDL, apoB, or non-HDL goal based on ASCVD risk

NOT AT GOAL? after maximally tolerated statin → add ZETIA

Still not at goal? - add or substitute → proprotein convertase subtilisin/kexin type 9 (PCSK9)-lowering agent
--OR add bempedoic acid to the maximally tolerated statin
--OR consider adding icosapent ethyl (in persons with triglycerides 135 to 499 mg/dL) for ASCVD risk reduction.

DYSLIPIDEMIA - Hypertriglyceridemia

Triglycerides – In persons with high ASCVD risk or very high ASCVD risk → begin with intensive lifestyle modification + statin therapy – etoh!

On maximally tolerated statin if triglyceride concentrations ≥ 200 mg/dL and HDL-C < 40 mg/dL

- ADD fibrate or high-dose omega-3 fatty acid to achieve the desired apo B or non-HDL-C goal
- Icosapent ethyl can be considered in persons with high or very high ASCVD risk

Risk categories	Risk characteristics	Approximate 10-y risk	Lipid targets	Therapy
High risk	T2D duration <10 y, T1D duration <20 y with <2 additional ASCVD risk factors; no TOD	<10%	LDL-C <100 mg/dL; apo B <90 mg/dL; non-HDL-C <130 mg/dL	Moderate-intensity statin to start, intensify as needed
Very high risk	T2D duration >10 y or T1D >20 y and age >40 y without ASCVD or severe TOD; ≥2 additional traditional ASCVD risk factors	10% to 20%	LDL-C <70 mg/dL; apo B <80 mg/dL; non-HDL-C <100 mg/dL	High-intensity statin, addition of ezetimibe or bempedoic acid to reach lipid targets
Extreme risk	T2D or T1D with established ASCVD or severe TOD: eGFR <45 mL/min/1.73 m ² ; UACR >300 mg/g; ABI <0.9; left ventricular systolic or diastolic dysfunction	>20%	LDL-C <55 mg/dL; apo B <70 mg/dL; non-HDL-C <90 mg/dL	High-intensity statin, addition of ezetimibe, bempedoic acid, and/or PCSK9 agent to reach lipid targets

Table 9
Atherosclerotic Cardiovascular Disease Risk Categories, Characteristics, Lipid Targets, and Therapy^a

Table 10 Drug-Effectiveness for Low-Density Lipoprotein Cholesterol–Lowering Therapy

Drug	Dose			% LDL-C lowering ^{318,324,*}
Statins	Dose intensity (mg/d; po)			
	Low	Mod	High	Typical LDL-C decline based on statin and dose intensity: Low <30% Mod 30% to 45% High ≥50%
Simvastatin	10	20 to 40		
Pravastatin	10 to 20	40 to 80		
Lovastatin	20	40		
Fluvastatin	20 to 40	80 ^b		
Pitavastatin		2 to 4 mg		
Atorvastatin		10 to 20	40 to 80	
Rosuvastatin		5 to 10	20 to 40	

Cholesterol absorption inhibitor

Ezetimibe ^{329,330}	10 mg orally every day	12% to 25% as mono-Rx ⁽³²⁹⁻³³²⁾ ; 25% when added to statin ^{330,331}	Inhibits intestinal and biliary cholesterol absorption, decreasing hepatic stores and increasing LDL-R upregulation	Myalgias, fatigue, URI symptoms, GI symptoms
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Bile acid sequestrants

Colesevelam	625 mg/tab; 3 tabs bid	8% to 16% as mono-Rx	Efficient binding of bile acids, lowering hepatic cholesterol, promoting LDL-R upregulation	GI symptoms, constipation, can bind other drugs; avoid if TG >300mg
Colestipol	1 g/tab; 2 to 6 g/d			
Cholestyramine	4 g/packet; 8 to 16 g/d			

PCSK9i	Initial dose	Max dose		
Alirocumab	75 mg sc every 2 weeks	300 mg sc every 4 weeks	48% to 58% ^c	Decreases PCSK9 levels, leading to reduced hepatic LDL-R degradation and increased expression
Evolocumab	140 mg sc every 2 weeks	420 mg sc every 4 weeks	63% to 71% ^c	

ACL inhibitor					
Bempedoic acid ^a	180 mg orally every day	17% to 18% as mono-Rx. Further lowering when added to statin (+22%) ⁽³³³⁾ or ezetimibe (+13%) ⁽³²⁹⁾	Inhibits ACLY, an upstream enzyme of HMG-CoA reductase	Myalgias, fatigue, URI symptoms, uric acid increase	

PCSK9 siRNA					
Inclisiran ^{a334}	284 mg every 6 months sc Then 284 mg every 6 months	38% to 52%	small interfering RNA directs breakdown of	Injection site reaction, arthralgia	

Managing Comorbidities – DKD and CKD in DM

- Annual assessment of **serum creatinine** to determine the eGFR and urine albumin-to-creatinine ratio (UACR)
- Identify, stage, and monitor progression of DKD (CKD)
- In TYPE 1 DM begin annual DKD assessment 5 years after diagnosis
- In type 2 DM begin annual DKD assessment immediately

Person with CKD in DM MUST BE ADVISED about optimal glycemic control, BP control, lipid control, also smoking cessation to reduce risks of development and progression of CKD and CVD

Managing Comorbidities – DKD and CKD in DM

POS for albuminuria (T1D or T2D) to reduce risk of DKD or CKD in DM progression → RAAS blockade with an **ARB or an ACE inhibitor**

T2D and CKD with eGFR ≥ 20 to for glycemia control + reduce progression of CKD and risk of CVD → **SGLT2i with renal indication**

T2D and DKD or CKD in DM with eGFR ≥ 15 for glycemic control + reduce risk of ASCVD + lower progression of albuminuria → **GLP-1 RA (renal indication)**

T2D and eGFR ≥ 25 , nl serum potassium but POS + albuminuria (UACR ≥ 30 mg/g) despite max tolerable RAS inhibitor → **nonsteroidal mineralocorticoid receptor antagonist (finerenone)** with proven kidney + CVD benefit

Color and number relates to frequency of re-assessment

Guide to Frequency of Monitoring (number of times per year) by GFR and Albuminuria Category				Persistent albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30–300 mg/g 3–30 mg/mmol	>300 mg/g >30mg/mmol
GFR categories (ml/min/1.73 m ²) Description and range	G1	Normal or high	≥90	1 if CKD	1	2
	G2	Mildly decreased	60–89	1 if CKD	1	2
	G3a	Mildly to moderately decreased	45–59	1	2	3
	G3b	Moderately to severely decreased	30–44	2	3	3
	G4	Severely decreased	15–29	3	3	4+
	G5	Kidney failure	<15	4+	4+	4+

Managing Comorbidities – RETINOPATHY

T2D and adult onset T1D → initial dilated and comprehensive eye examination (ophthalmologist or optometrist) at the time of diagnosis or shortly thereafter

Subsequent screening → individualized - based on type and duration of DM, A1C; mean BG, BP, presence and grade of retinopathy

→ In persons with T1D, initial dilated and comprehensive eye examination by an ophthalmologist or optometrist within 5 years of diagnosis under age 18

→ Pregnant women + preexisting T1D or T2D – **eye examinations every trimester during pregnancy** and PP determined by severity of retinopathy during pregnancy

RETINOPATHY

Persons with > mild nonproliferative retinopathy ----> annual EYE examinations at least once a year

Follow-up with eyecare specialists annually –

→ Persons with T1D or T2D who have had a normal ocular examination may be screened every 2 to 3 years

→ Optimal glucose, BP, weight, and lipid control to slow the progression of retinopathy

Managing Comorbidities – NEUROPATHY

Diabetic peripheral neuropathy (DPN) a **clinical diagnosis**

→ A comprehensive differential diagnosis should be considered to rule out nondiabetic neuropathies

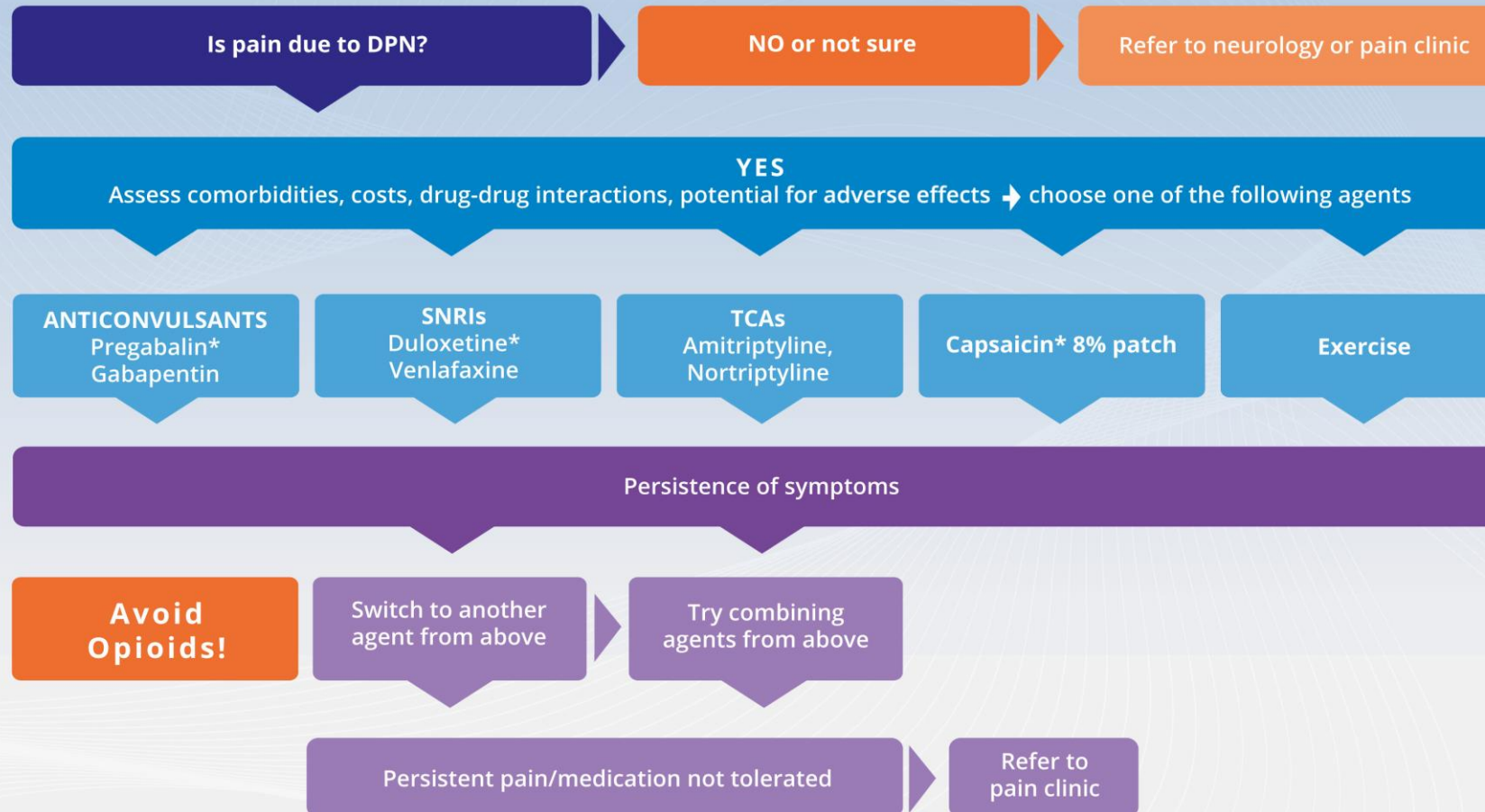
Screening for DPN

→ At diagnosis of T2D

→ Within 5 years of the diagnosis of T1D

→ Subsequently annually or whenever symptoms occur, by performing a clinical history and physical exam

ALGORITHM FOR TREATMENT OF DIABETIC PERIPHERAL NEUROPATHY



*FDA approved for treatment of DSPN

DPN = diabetic peripheral neuropathy; DSPN = distal symmetrical polyneuropathy; FDA = Food and Drug Administration; SNRI = serotonin-norepinephrine reuptake inhibitor; TCA = tricyclic antidepressant

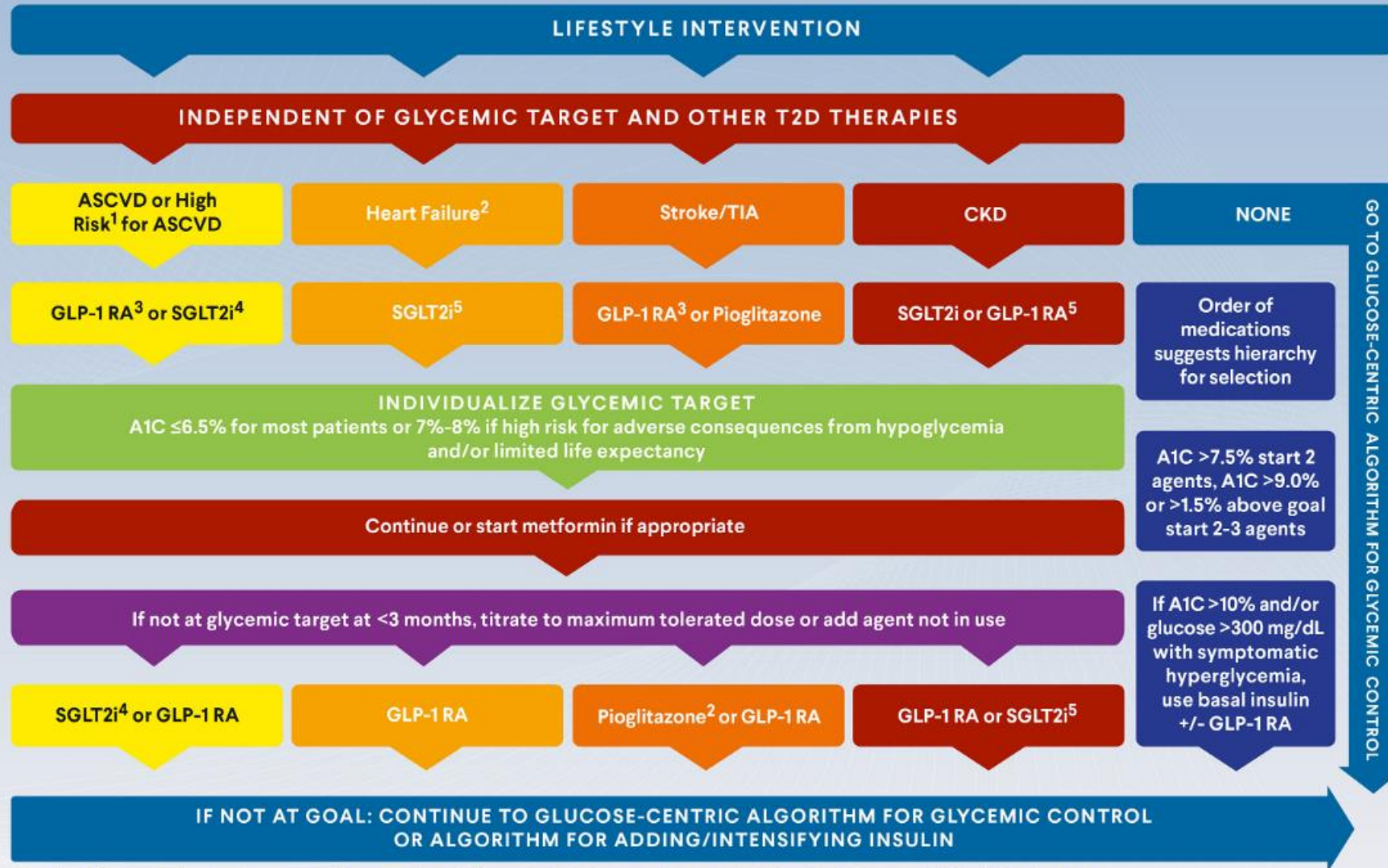
Adapted from Pop-Busui, Boulton, et al, Diabetes Care 2017;40:136-154

Managing Comorbidities –DM- Related CVD

Complication-centric algorithm for glycemia control- ASCVD

- Independent of glucose, what is the ‘best med’ – very rational approach
- Pick ‘the best meds’ first then ‘backfill’
- GLP1-RA and SGLT-2i both shown to lower ASCVD risk – each has distinct effects on varying components of ASCVD – see table

COMPLICATIONS-CENTRIC ALGORITHM FOR GLYCEMIC CONTROL



¹High risk for ASCVD: albuminuria or proteinuria, hypertension and left ventricular (LV) hypertrophy, LV systolic or diastolic dysfunction, ankle-brachial index <0.9.

²TZDs are contraindicated in NYHA Class III/IV HF. ³ASCVD: liraglutide/semaglutide/dulaglutide or Stroke: semaglutide/dulaglutide.

⁴canagliflozin/empagliflozin. ⁵Use SGLT2i or GLP-1 RA with proven benefit.

Managing Comorbidities - OBESITY

Diet – reduced calorie meals

regular CDE contact for support

Weight – 5-10% weight reduction goal

Daily – physical activity

Frequent – exercise

5-10% weight loss for optimal improvement in DM,
BP/HTN, lipids

≥10% weight loss can resolve OSA and NASH

Managing Comorbidities –OBESITY

Weight-loss medication	Dose; escalate as tolerated	Mechanism	Potential side effects	Warnings and contraindications ^a
Approved for short-term therapy (≤ 3 mo)				
Phentermine	Low-dose 15 mg every day; maximum dose 37.5 mg every day (by mouth) ^b	Sympathomimetic amine (decreases appetite); stimulates CNS activity	Restlessness, insomnia, headache, dry mouth, tachycardia, BP elevation	Pregnancy, active coronary artery disease, uncontrolled hypertension, hyperthyroidism, agitated states
Approved for chronic management of obesity				
Orlistat	Treatment dose 120 mg three times a day (by mouth with	Gastrointestinal lipase inhibitor (decreased fat	Fat malabsorption, flatulence, fecal	Pregnancy, fat-soluble vitamin and drug malabsorption (do not use in organ

Managing Comorbidities – OBESITY

Phentermine/Topiramate-ER	Starting dose 3.75 mg/23 mg every day; treatment dose 7.5 mg/46 mg every day; maximum dose 15 mg/92 mg every day (by mouth)	Sympathomimetic amine (decreases appetite)/anticonvulsant, carbonic anhydrase inhibitor, gabaminergic (increases satiety)	Restlessness, insomnia, headache, dry mouth, tachycardia, BP elevation, paresthesia, dysgeusia, mood changes, mental clouding, blurred vision	Pregnancy, glaucoma, hyperthyroidism, metabolic acidosis, urolithiasis
Naltrexone-ER/ Bupropion-ER	8 mg/90 mg tablets; starting dose one tablet every day; treatment dose 2 tablets twice a day (by mouth)	Opioid receptor antagonist (decreases cravings)/dopamine-norepinephrine reuptake inhibitor (decreases appetite)	Nausea, vomiting, diarrhea, constipation, headache, fatigue, insomnia, agitation, mood changes, dry mouth, blurred vision	Pregnancy, seizure risk, uncontrolled hypertension, chronic opioid use

Managing Comorbidities –OBESITY

Naltrexone-ER/ Bupropion-ER	dose one tablet every day; treatment dose 2 tablets twice a day (by mouth)	(decreases cravings)/dopamine-norepinephrine reuptake inhibitor (decreases appetite)	constipation, headache, fatigue, insomnia, agitation, mood changes, dry mouth, blurred vision	risk, uncontrolled hypertension, chronic opioid use
Liraglutide 3 mg	Starting dose 0.6 mg/d; maximum dose 3 mg/d (subcutaneous injection)	Glucagon-like peptide-1 receptor agonist (decreases appetite and delays gastric emptying)	Nausea, vomiting, diarrhea, constipation, headache, fatigue	Pregnancy, medullary thyroid cancer, MEN type 2, tachycardia, acute pancreatitis, acute gallbladder disease
Semaglutide 2.4 mg	Starting dose 0.25 mg/wk; maximum dose 2.4 mg/wk (subcutaneous injection)	Glucagon-like peptide-1 receptor agonist (decreases appetite and delays gastric emptying)	Nausea, vomiting, diarrhea, constipation, headache, fatigue	Pregnancy, medullary thyroid cancer, MEN type 2, tachycardia, acute pancreatitis, acute gallbladder disease, diabetic retinopathy

Question 1:

What two changes were made to the DKA assessment pathway in the 2025 updated diabetes guidelines:

1. Anion gap is emphasized and further categorizes severity of DKA
2. Glucose cutoff for DKA diagnosis changed from 250mg/dl or higher to 200mg/dl or higher
3. Beta-hydroxy buturate (BHB) is recommended to further categorize severity of DKA
4. Glucose cutoff for DKA diagnosis changed from 250mg/dl or higher to 180mg/dl or higher

Question 2:

Which of the following statements is correct with regard to CAD risk and statin recommendations in type 2 diabetes

1. Patients considered low risk should be monitored and only patients with known CAD should be offered statin therapy
2. Low risk patients should be risk stratified with Lpa and CAC, crp and offered statin if risk is increased
3. High risk patients LDL goal is < 100
4. Goal LDL in low-risk patients is < 150

References

1, DKA guidelines - Standards of Care| December 09 2024

16. Diabetes Care in the Hospital: Standards of Care in Diabetes—2025

https://diabetesjournals.org/care/article/48/Supplement_1/S321/157551/16-Diabetes-Care-in-the-Hospital-Standards-of-Care

2. <https://pro.aace.com/clinical-guidance/2023-aace-consensus-statement-comprehensive-type-2-diabetes-management-algorithm>

3. [JGnewslide#1 and #2](#). For the new TZIELD slide:

https://www.tziel dhcp.com/?&utm_source=google&utm_medium=cpc&utm_campaign=Tziel d-HCP-www.tziel dhcp.com_GGL_BRND_Diagnosis-Consideration_AWA_SEA_ALLM_US_EN+KW+-+EN+BR_ALL_P30JS4B&utm_term=tziel d+sanofi&gclid=aw.ds&gad_source=1&gad_campaignid=22386262306&gbraid=0AAAAA p6isEkZaS_AYAq4Cjp07uwJ5HbWw&gclid=CjwKCAjwlaTGBhANEiwAoRgXBaxqvccZaoCuQ0yQ0gVVTQLTyW8XYDbxRZHHTBbEtllQudpu4RGIZBoCnhsQAvD_BwE



Diabetes and Beyond

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Real change lives here

OUTLINE

Section 3: Managing prediabetes, T2D, and T1D –choosing glycemic targets, lifestyle interventions, oral/injectable medication (insulin therapy in T1D)

Section 4: vaccine review in persons with DM – recommendations and CGM review

Section 3: Managing Prediabetes

34.5% of US population meets criteria for prediabetes
(CDC 2020 *National Diabetes Statistics Report*)

Risk of progression to T2DM increased with **higher BMI** and HbA1c value

Prediabetes as ICD Dx code currently does not support coverage of weight loss drugs such as GLP-1 agonists; Rx based on BMI if indicated

Lifestyle modification is the most important preventative intervention

Reduces progression to T2DM and CVD risk factors

Prediabetes has been linked to macular thinning (pre-cursor to retinopathy) and microalbuminuria

JAMA. 2021;326(8):736–743. doi:10.1001/jama.2021.12531

Retina 2022; Mar 1;42(3):442-449. doi: 10.1097/IAE.0000000000003337

According to this chart, I'm not overweight, I'm undertall!



Prediabetes often goes unnoticed

You can have prediabetes for years without symptoms. This means you likely won't know you have prediabetes until serious health problems show up. Talk to your doctor about getting your [blood sugar tested](#) if you have any risk factors, including:

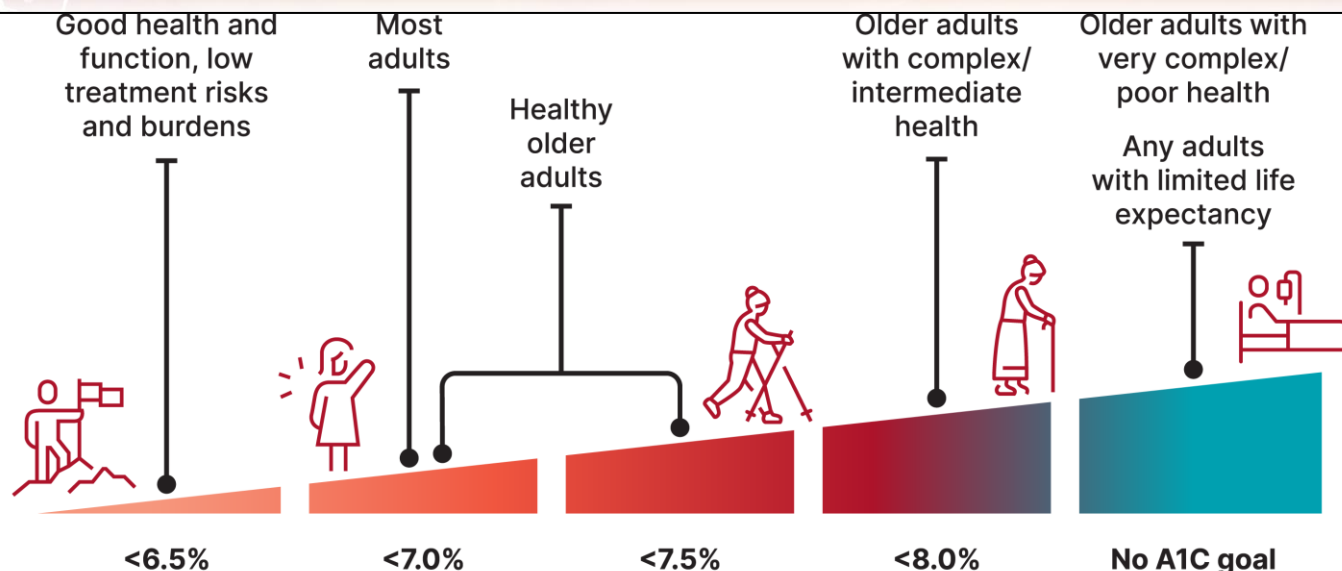
- Having overweight.
- Being 45 years or older.
- Having a parent, brother, or sister with type 2 diabetes.
- Being physically active less than 3 times a week.
- Ever having gestational diabetes (diabetes during pregnancy).
- Giving birth to a baby who weighed more than 9 pounds.

Race and ethnicity are also factors. African American, Hispanic/Latino, American Indian, Alaska Native, Pacific Islander, and some Asian American people are at higher risk.

Summary of Recommendation

Asymptomatic adults aged 35 to 70 years who have overweight or obesity	The USPSTF recommends screening for prediabetes and type 2 diabetes in adults aged 35 to 70 years who have overweight or obesity. Clinicians should offer or refer patients with prediabetes to effective preventive interventions.	B
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ADA 2025 Glycemic Targets for Diabetes



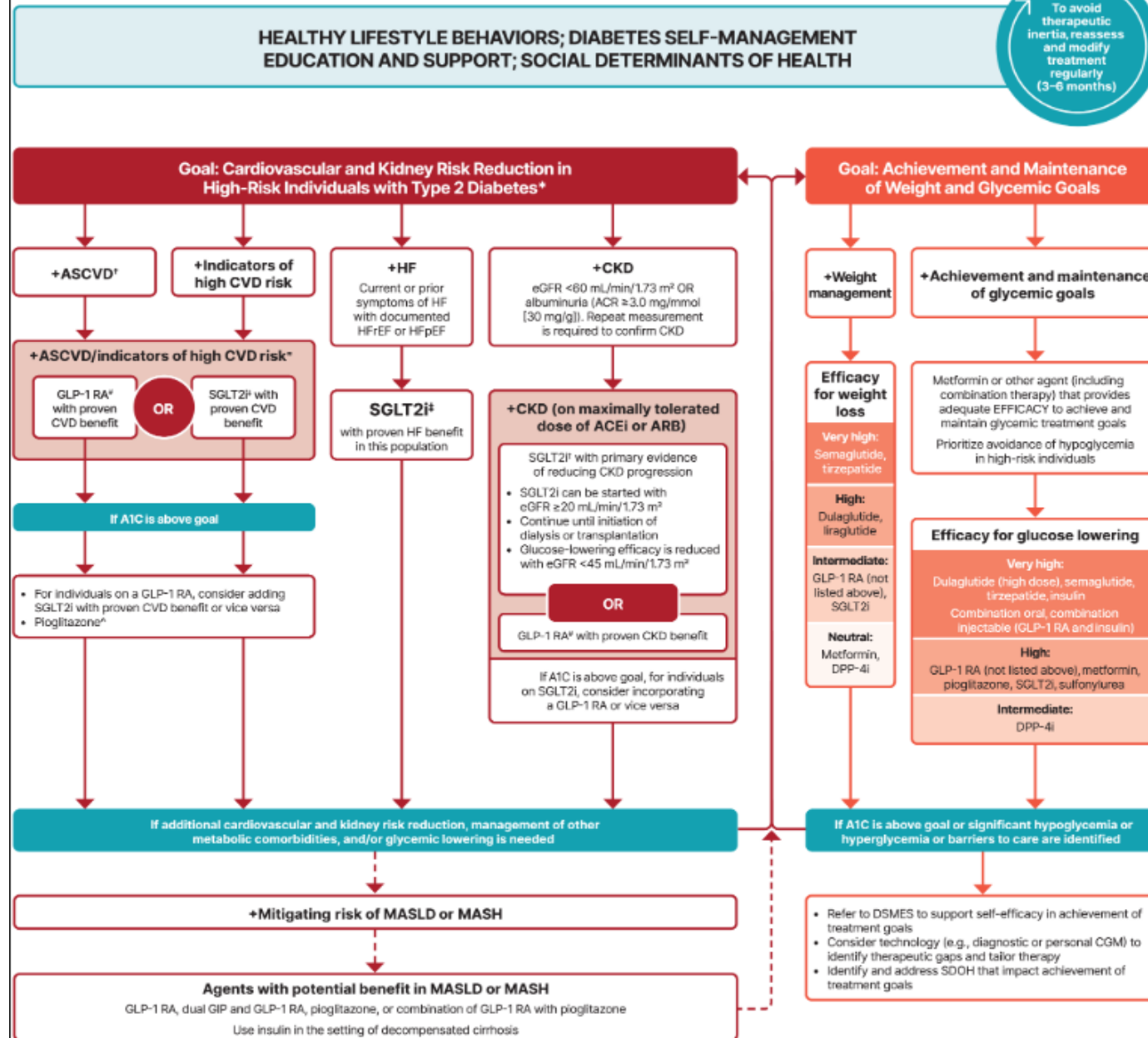
Modifying Factors

Favor more stringent goal	Favor less stringent goal
Short diabetes duration	Long diabetes duration
Low hypoglycemia risk	High hypoglycemia risk
Low treatment risks and burdens	High treatment risks and burdens
Pharmacotherapy with cardiovascular, kidney, weight, or other benefits	Pharmacotherapy without nonglycemic benefits
No cardiovascular complications	Established cardiovascular complications
Few or minor comorbidities	Severe, life-limiting comorbidities

Summary of glycemic goals for many nonpregnant adults with diabetes

HbA1c	<7.0% (<53 mmol/mol)
Pre-prandial capillary plasma glucose	80-130 mg/dL (4.4-7.2 mmol/L)
Peak Postprandial capillary plasma glucose	<180 mg/dL (<10.0 mmol/L)

Use of Glucose-Lowering Medications in the Management of Type 2 Diabetes



Why Does ASCVD Matter?

1990s T2DM drugs approved based on small studies

Early 2000s: Increased CV risk noted with certain drugs

Rosiglitazone (Avandia-GSK) approved 1999 shows increased risk of MI, 13,000 lawsuits against GSK

2008: US FDA issued regulation to ensure CV safety

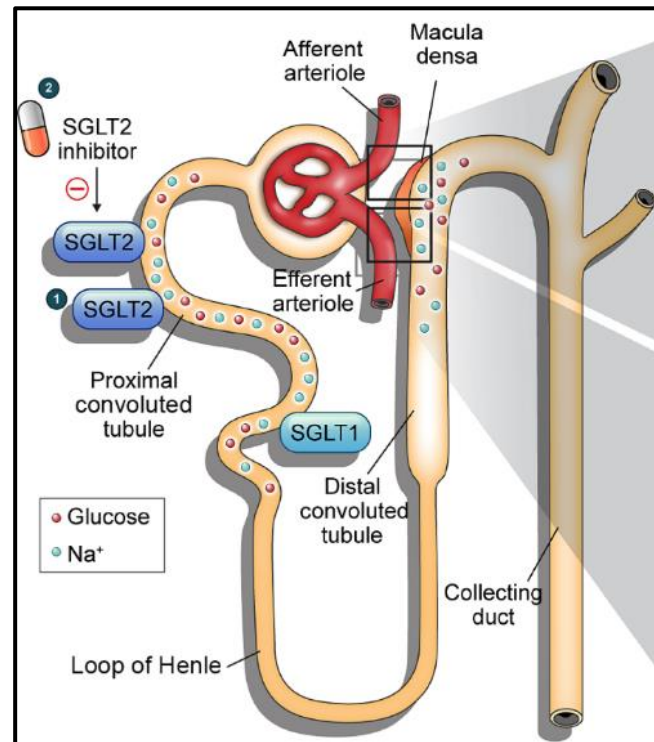
CV outcome trials initiated (placebo non-inferiority trials)



Sodium Glucose CO-Transporter 2 Inhibitors

How they work: Sodium-glucose transport protein 2 (SGLT2) inhibitors act on the SGLT-2 proteins expressed in the renal proximal convoluted tubules to reduce the reabsorption of filtered glucose

❖ **Practical considerations:** Improves fasting hyperglycemia, may start at $\text{eGFR} \geq 20 \text{ ml/min/1.73m}^2$, lower hypoglycemic agents if starting A1c $< 8\%$, may contribute to weight loss



Current FDA approved SGLT-2i

- Dapagliflozin
- Empagliflozin
- Canagliflozin
- Baxagliflozin
- Ertugliflozin

Important SGLT-2i Trials

EMPA-REG : Empagliflozin (Jardiance)

Over a median follow-up of 3.1 years, treatment reduced the composite out- come of MI, stroke by 14% and cardiovascular death by 38%

CANVAS Canagliflozin (Invokana)

Over a median of 3.6 years canagliflozin significantly reduced the composite outcome of cardiovascular death, MI, or stroke versus placebo by 14%

DELIVER III: Dapagliflozin (Farxiga)

Over medium 2.4 years, dapagliflozin reduced composite outcome of renal or cardiovascular death compared to placebo

N Engl J Med 2017;377:644-657

N Engl J Med 2020;383:1436-1446 DOI: 10.1056/NEJMoa2024816

N. Engl. J. Med. 373, 2117–2128 (2015)



SGLT-2i Adverse Events:

Most frequent:

Infections of the genitourinary tract (3.6%- 9.0%)

Most patients experience one episode that responds to standard treatment. Avoid in patients with hx of complicated UTI or Fournier's gangrene

Less frequent:

Volume depletion (~2.7%)

More frequent in older patients taking diuretics

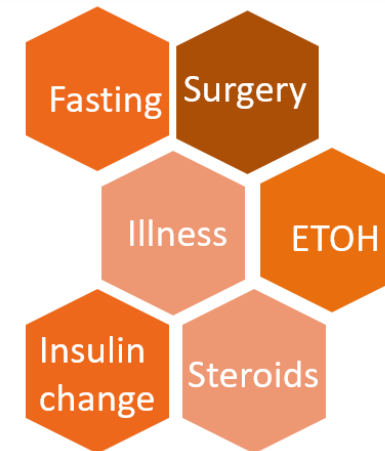
Hypoglycemia (Variable rates)

Mainly occurs in patients taking SU or insulin

Eur J Clin Pharmacol 77, 651–657 (2021).
Drug Des Devel Ther. 2014;8:1335–80.

Serious Side effects

Euglycemic DKA: predisposing factors

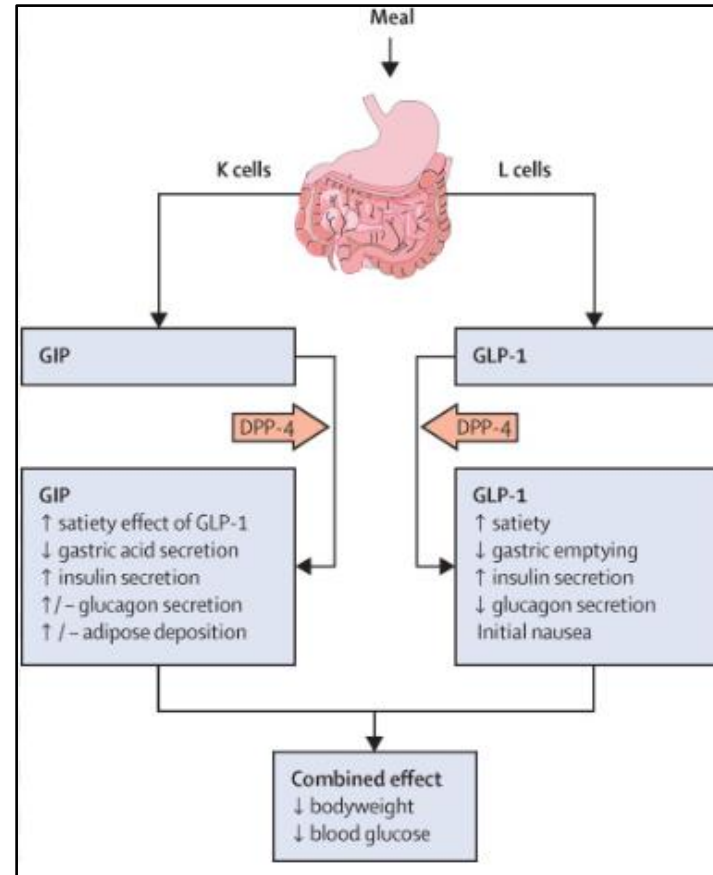


Amputations: CANVAS showed nearly 2x increase in lower limb amputations but not seen in CREDENCE or other SGLT-2i trials. *Meta analysis of 40 cohort studies did NOT show statistically significant increase*
Monitor those with active ulcers/severe PAD closely

Glucagon Like Peptide-1 and GIP Receptor agonists

How they work: GLP-1 and GIP agonists mimic GI peptides that promote oral glucose mediated insulin release from the pancreatic β cells; the “**incretin effect**”, delay gastric emptying and reduce appetite, preserves β cell function

- ❖ **Practical considerations:**
Improves post-prandial hyperglycemia, +weight loss, insulin sensitizer (lower hypoglycemic agents if starting A1c <8%)



FDA approved GLP-1a for T2DM

Exenatide (Byetta/Bydureon)
Liraglutide (Victoza)
Albiglutide (Tanzeum)
Dulaglutide (Trulicity)
Lixisenatide (Soliqua)
Semaglutide (Ozempic, Rybelsus)

GLP-1/GIPa

Tirzepatide (Mounjaro)

Important GLP-1/GIP-Receptor Agonists Trials

SUSTAIN: OZEMPIC (Semaglutide)

Non-fatal MI risk lowered from **3.9% to 2.9 %** and non-fatal CVA lowered from **2.7 % to 1.6%** over 2.1 years

Recent update: *Semaglutide 1 mg prevented decline in kidney function over 3.4 years compared to placebo*

REWIND: Trulicity (Dulaglutide)

Non-fatal MI risk lowered from 4.3% to 4.1 % and non-fatal CVA risk lowered from 3.5% to 2.7% over 5.4 years

SURPASS: Mounjaro (Tirzepatide)

Tirzepatide 5 mg, 10 mg and 15 mg weekly vs. Semaglutide 1 mg weekly showed significant improvement in body weight and A1c over 40 weeks

N Engl J Med 2016;375:1834-44.

N Engl J Med 2024;391:109-121

Lancet 2021; 9,10: 646-648

Lancet 2019; 394: 121-30



GLP-1/GIP Receptor Agonists: Adverse Effects

Most frequent:

Gastrointestinal Side Effects :



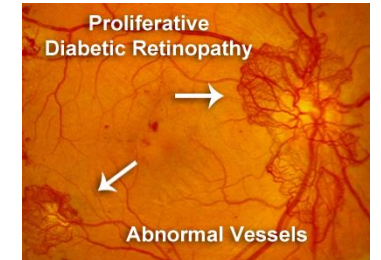
Nausea- 11-24%
Vomiting 5-24%
Diarrhea 9-30%
Constipation 3-24%
Abdominal Pain 6-20%
Dyspepsia 3- 9%

Risk factors: Rapid titration,
higher dose

Less frequent:

Retinopathy (1.3-fold increase, SUSTAIN-6)

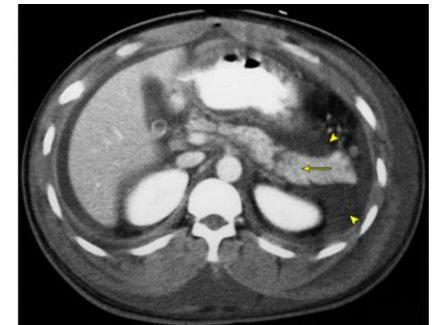
Due to rapid correction of hyperglycemia, potential direct angiogenic effect of semaglutide



Pancreatitis (<1%)

No statistically significant increase pancreatitis episodes vs placebo per REWIND, SURPASS, SUSTAIN-6

Suspect etiology due to GLP-1/GIP agonist induced pancreatic duct hyperplasia



Sarcopenia: more common in elderly, low BMI

N Engl J Med. 2016a;375(19):1834-1844.

Lancet 2019; 394: 121–30

Other Oral Antihyperglycemics

METFORMIN

Pro: cost-effective, improves fasting hyperglycemia, lowers A1c 1-2 %, 1st line

Cons: GI Side effects, weight neutral, avoid in CKD

SULFONYLUREAS (SU)

Pro: cost-effective, effective prandial coverage

Cons: hypoglycemia (Beer's criteria), ineffective in longstanding DM, weight gain, β cell burn out

FYIs: prefer newer agents
Glipizide or Glimepiride,
Avoid Glyburide unless GDM (risk of MI)

Dipeptidyl peptidase-4 inhibitors (DPP-4i)

Pro: better tolerated usually than GLP-1

Cons: costly, only 0.5 A1c reduction, pancreatitis, saxagliptin – R-CHF risk

FYIs: Linagliptin safe in CKD (even ESRD)
Do not use with GLP-1a due to redundancy & pancreatitis risk

Thiazolidinediones (TZDs)

PPAR γ agonist

Pro: cost-effective, improves fasting hyperglycemia, NAFLD

Cons: weight gain, fluid retention (avoid in CHF, CKD), osteoporosis

When Should You Start Insulin?

SEVERE HYPERGLYCEMIA OR “INSULINOPENIA”

Fasting glucose > 250 mg/dL

Random glucose >300 mg/dL

A1c $\geq$ 9.0

Weight loss/Cachexia

Polyuria/Polydipsia

Ketonuria or Ketonemia

ADVANCING COMPLICATIONS

**Moderate to severe
retinopathy**

**Ongoing Diabetic Foot
Ulcer**

Recent MI or CVA

***Advanced liver or kidney
disease***

AT RISK FOR PANCREATIC INSUFFICIENCY

**clinical suspicion for
Type 1 DM**

H/O Cystic Fibrosis

**H/O pancreatectomy or
chronic pancreatitis**

GLUCOTOXICITY: A Temporary Need for Insulin

Severe long-standing hyperglycemia increases mitochondrial stress on beta cell leading to glucotoxicity

- ❑ Impaired insulin production
- ❑ Increased insulin resistance

In Type 2 Diabetes with severe hyperglycemia, **insulin may be temporarily needed** until glucotoxicity resolves and beta cell function improves

Helpful explanation for insulin-hesitancy

Pancreas! What happened to your beta cells?
Don't you need those to make insulin?



You ARE producing insulin ... right?

We have diabetes, don't we?



Initiating Insulin: Basal Dosing Considerations

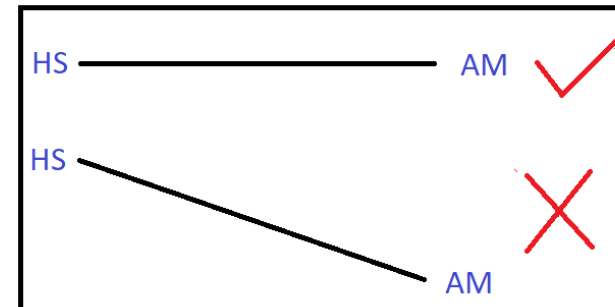
Initiate **BASAL** (long-acting) insulin in order to achieve fasting blood sugar target first

General rule of thumb:

- **0.15 units/kg** in most Type 2
 - For e.g, start 10-12 units once a day in a 70 kg patient
 - titrate up to 0.2 units/kg by first follow up visit to achieve goal fasting blood sugar target

Signs of “overbasalization” insulin

- Basal dose > 0.5 unit/kg
- Fasting hypoglycemia
- Post–preprandial differential
- High bedtime to AM difference



Consider PRANDIAL Coverage

*In cases of **steroid-induced hyperglycemia** or failure to achieve target HbA1c despite **max basal insulin** (not exceeding 0.5 unit/kg) and/or **max tolerable PO metformin/TZD/SGLT-2i**, considering adding/maximizing:*

GLP-1/GIP agonist

especially if BMI in overweight/obese BMI range and/or ASCVD risk factors present

Sulfonylureas

Less effective in those with long-standing T2DM

Prandial insulin

***Discontinue SU to avoid redundant prandial coverage*

Initiating Insulin: Prandial Dosing Considerations

Rapid acting analogue (RAA) insulin preferred

**Humalog/Novolog over regular insulin (Humulin or Novolin)*

***Regimen including prandial AND sliding scale insulin is
proactive and physiological***

**Sliding scale alone without prandial approach is reactive*

Prandial = standing RAA insulin covering carbs on the plate

**may assume average meal 45-60 g for most T2DM*

Sliding scale (SSI) insulin involves use of RAA to correct a high blood glucose above a certain target glucose based on insulin sensitivity factor (ISF)

**1 unit of RAA drops BG how many points? (ISF formula: $1800 \div \text{TDD}$)*

Ideal prandial insulin dosing

*Basal dose is 50% of TDD
Prandial dose is other 50%
in divided doses*

*For e.g. patient on 36 units
Basal ($<0.5 \text{ u/kg}$), add 12
units before each meal +
SSI*

Clinical Vignette: Adding Prandial Insulin

74 yo M, T2DM, CAD s/p CABG, HTN, CKD stage 3b

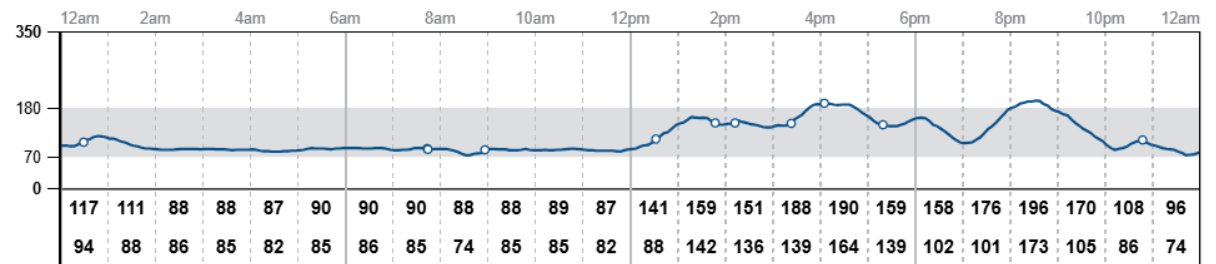
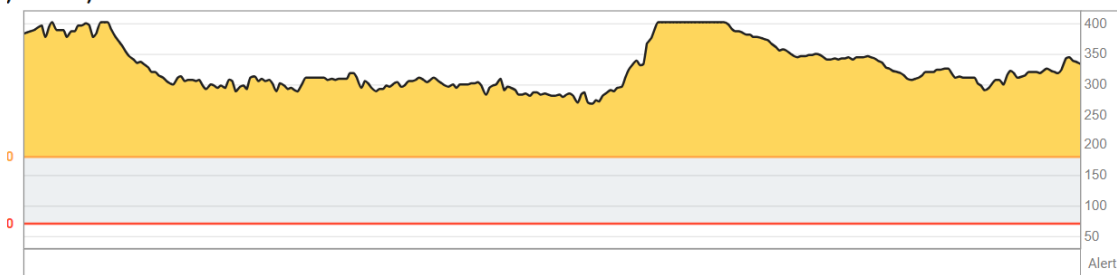
Initial DM Regimen:
Trulicity 1.5 (max tolerable)
Farxiga 10 mg
Glipizide 10 mg XL QAM
Basaglar 25 units QHS

RECOGNIZE NON-COMPLIANCE

- helped set phone reminder for basal insulin & GLP-1a
- stopped SU
- Added prandial insulin with largest meal of the day



Follow up DM Regimen:
Trulicity 1.5 (max tolerable)
Farxiga 10 mg
Basaglar 25 units QHS
Lyumjev 10 AC meals



Initiating Insulin: Premix Insulin

If initiating non-physiologically dosed **pre-mix insulin (70/30 or 75/25 BID)** due to insurance/cost considerations

Use 0.2 to 0.25 units/kg

- *For e.g., 70 kg patient, 0.2 units/kg for this patient is ~ 14-15 units > split 2/3 dose into AM dose and 1/3 dose into PM dose > 10 units QAM, 5 units QPM **BEFORE MEALS***

Distinguishing Between Type 1 and Type 2

- Suspect in **young and/or thin patients with diabetes** presenting with:
 - Severe hyperglycemia
 - Rapid progression of diabetes
 - Ketonuria (>2+ ketones) in the presence of BG >250 mg/dL
- **Type 1 can present at any age**

Distinguishing Between Type 1 and Type 2

- **What labs should you order?**

- C-peptide **WITH** concurrent glucose (in the same lab draw)
C-peptide is measure of endogenous insulin
Do not order insulin level (accounts for exogenous + endogenous)

- Type 1 antibodies- **GAD-65, IA-2, Insulin Antibodies, Zinc transporter Ab (ZnT8 Ab)**

Order and SmartSet Search

diabetes autoimmune

Browse Preference List Facility List

SmartSets, Panels, & Express Lanes (No results found)

After Visit Medications (No results found)

After Visit Procedures

ID	Name	Type	Resulting Agencies	Pref List	Px Code	Cost to...
100876	Diabetes autoimmune profile	Lab	LabCorp, Quest, SCC	PHC AMB EN...	LAB3482	
100876	Diabetes autoimmune profile (Labcorp..	Lab	LabCorp, Quest, SCC	PHC AMB EN...	LAB3482	

If c-peptide is very low or undetectable and/or 1-2 of the Abs is +, suspect Type 1 DM

Clinical Vignette: Case of Missed Type 1 DM

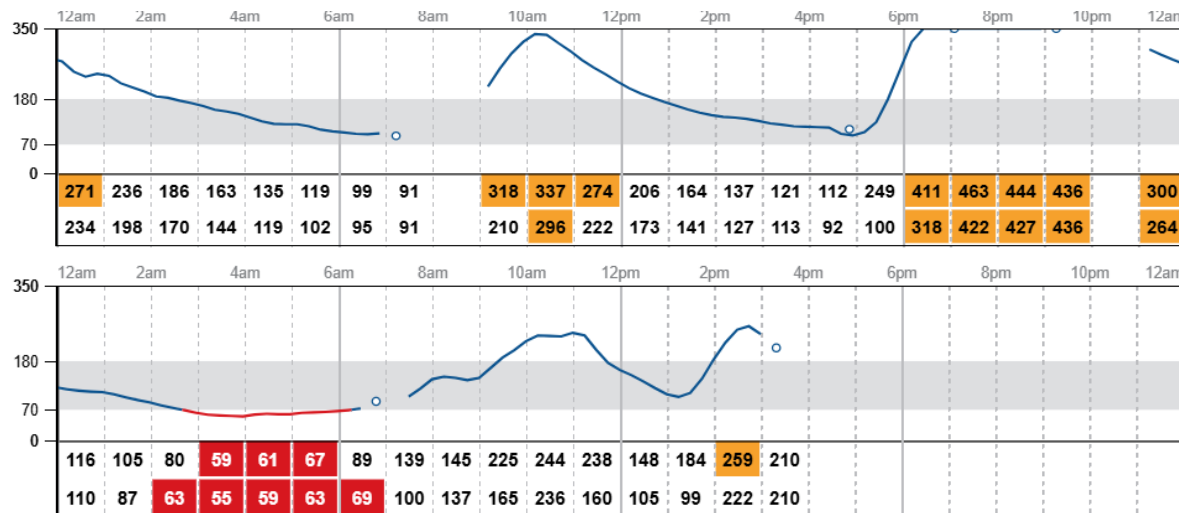
35 yo M, Diabetes Dx 5 years ago & Depression

BMI: 23.5

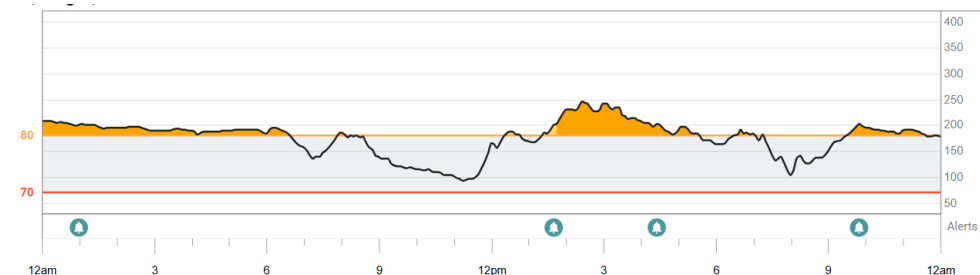
Complications: neuropathy

Fmhx: mother dx DM at young age

Prior meds: SU glipizide reportedly “did not help”



Component	5/11/2022
Latest Ref Rng & Units	
Gad-65	15 (H)
<5 IU/mL	
Insulin AutoAb	<0.4
<0.4 U/mL	
IA-2 ANTIBODY	<5.4
<5.4 U/mL	
Glucose	123 (H)
65 - 99 mg/dL	
C-Peptide	0.31 (L)
0.80 - 3.85 ng/mL	



Initial DM Regimen:
Metformin 1000 mg BID
Victoza 1.2 mg daily
Tresiba 10 QHS



Follow-up DM Regimen:
Tresiba 8 QHS
Humalog 4-6 AC + 1:70>150

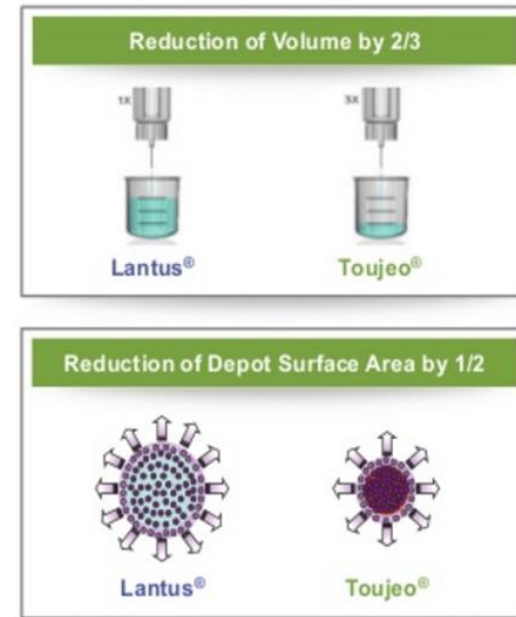
Initiating Insulin: Examine Injection Site

Improper insulin injection practices are common reasons for uncontrolled diabetes. **Provide counseling** on proper injection technique



Rotate sites!

Avoid hypertrophied sites!!!



Consider switching basal to **concentrated U200 Tresiba or U300 Toujeo** if using >40-50 units of basal to reduce insulin volume/improve absorption

Initiating Insulin: Ask the BURNING Questions

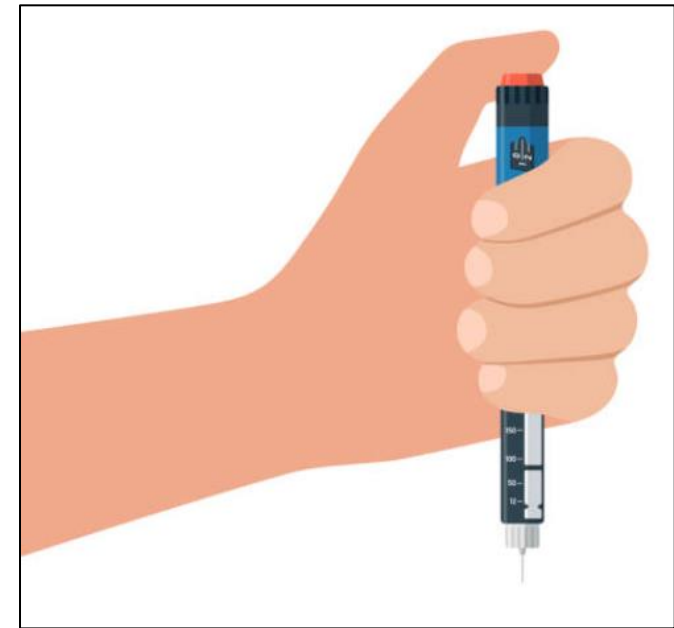
Does it **burn** when you inject?

*Consider switching to **longer pen needle** and **larger gauge** (pts with obesity), switch to **smaller needle** (pts with low BMI)*

“Have you removed **the 2 caps** that go over the needle?”

“Show me how you **dial** your insulin”

“Are you **avoiding hardened areas** and **rotating sites**?”



HYPOGLYCEMIA in Diabetes: The Rate Limiting Step

LEVEL 1 HYPOGLYCEMIA

Glucose concentration of
54–69 mg/dL (3.0–3.8 mmol/L)

LEVEL 2 HYPOGLYCEMIA

Glucose concentration **<54 mg/dL (<3.0 mmol/L)**, which is typically the threshold for **neuroglycopenic symptoms**

LEVEL 3 HYPOGLYCEMIA

clinical event characterized by **altered mental and/or physical functioning that requires assistance from another person for recovery**

Avoiding Hypoglycemia

Avoid stringent HbA1c control when using hypoglycemic agents such as SU or insulin.

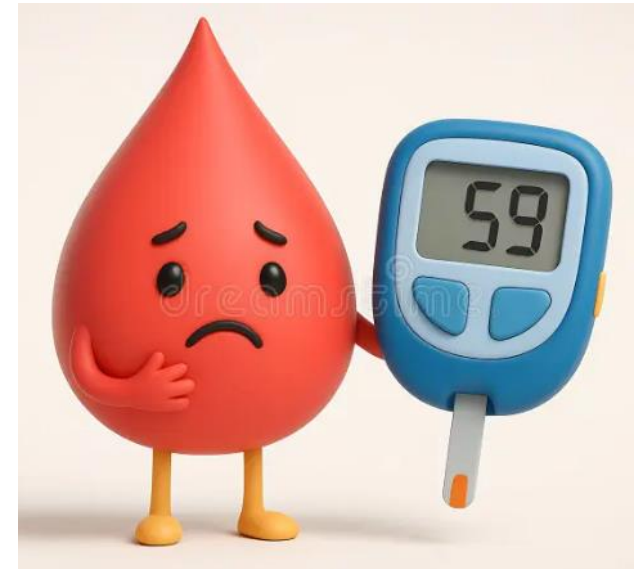
Set HbA1c goal >6.5, in elderly >7.0

Avoid redundancy

Discontinue SU if initiating pre-mix insulin or rapid acting insulin

Add CGM when using hypoglycemic agents

Reduce dose of hypoglycemic agents such as SU and insulin by 10-15% if initial A1c < 8 when initiating insulin sensitizers (GLP-1 α /SGLT-2i/TZD/DPP-4i/metformin)



“Fear of lows” is a common cause of non-compliance

Available Glucagon Formulations



Clinical Vignette: Hypoglycemia

68 yo F, T2DM with nephropathy, HTN, Osteopenia

BMI: 25

Notes mention “*does not monitor blood sugar at home*”

Diabetes regimen was not adjusted despite multiple HbA1c readings <6.5

Diabetes Regimen:

Metformin 500 mg XR QAM

Glimeperide 2 mg QAM

In the setting of low PO intake, patient experienced a **level 3 hypoglycemia event 2/2025 with loss of consciousness, required overnight hospital stay**
Did not have glucagon at home, lives alone

Hemoglobin A1c

Status: Final result Next appt: 09/24/2025 at 02:00 PM in Cardiology

Test Result Released: No

0 Result Notes | 1 HM Topic

Component	2/23/25 0849	2/7/25 0934	5/15/24 1547	7/18/23 1632	3/7/22 1412	8/11/21 1602
Ref Range & Units						
 Hemoglobin A1C	5.6	5.8 ▲ R, CM	6.1 ▲ R, CM	5.8 ! R	5.8 ▲ R, CM	5.4 R, CM
4.0 - 6.0 %						

Benefits of Continuous Glucose Monitors

“CGM is essential for creating the ambulatory glucose profile (AGP) and providing data on time in range, percentage of time spent above and below range, and variability. Access to CGM devices should be considered from the outset of the diagnosis of diabetes that requires insulin management. This allows for close tracking of glucose levels with adjustments of insulin dosing and lifestyle modifications and removes the burden of frequent SMBG monitoring. Interruption of access to CGM is associated with a worsening of outcomes; therefore, it is important for individuals on CGM to have consistent access to the devices.”

-Standards of Medical Care in Diabetes 2021

Improved glycemic control

Offers more dynamic and precise glucose monitoring

Improves compliance

Reduced hypoglycemia

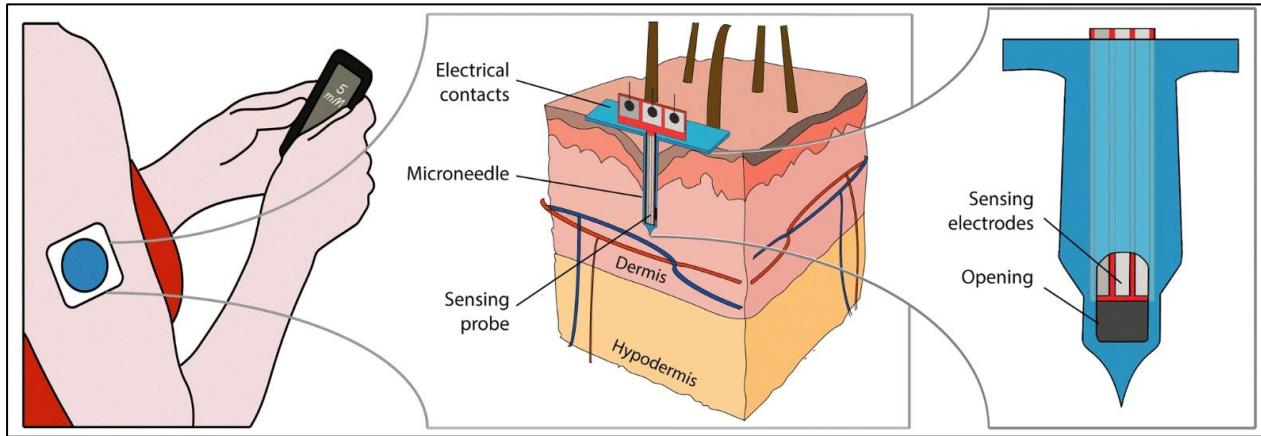
Allows for timely interventions

Less “fear of lows”

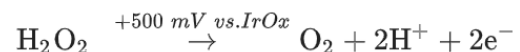
Enhanced quality of life

Reduced physical discomfort compared to fingersticks

Continuous Glucose Monitor: How Do They Work?



Glucose in the Interstitial Fluid is catalyzed by Glucose Oxidase in the sensor to Hydrogen Peroxide (H_2O_2). An electrical current in proportion to amount of H_2O_2 generates electrical current, allows transmitter to convert signal to a glucose displayed in mg/dL on receiver.



CGM Value vs Blood Glucose Monitor (BGM) value:

10-15 point “discrepancy” between BGM and CGM values

Values are separated by time and space

15-20-minute lag time to allow for glucose to diffuse from capillary space to interstitial fluid, especially during rapid glucose changes

If symptoms do not match CGM readings, may calibrate with fingerstick (Dexcom only)

Clinical Vignette: Impact of CGM

60 yo F, T2DM Dx 25 years ago, HPLD, HTN

BMI: 34.11

Hypo awareness: unaware, level 3 with LOC

Initial DM Regimen:
Humalog 15 units AC meals
Lantus 55 units QHS
Victoza 0.6 mg daily

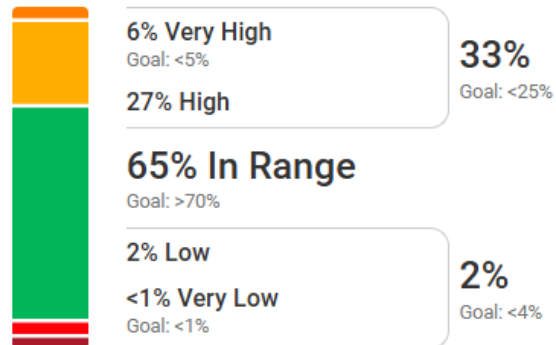


Follow up DM Regimen:
Humalog 10 units AC meals
Lantus 35 units QHS
Ozempic 0.5 mg weekly
Farxiga 10 mg

HbA1c before and after CGM and med changes

Time in Ranges Goals for Type 1 and Type 2 Diabetes

Each 5% increase in the Target Range is clinically beneficial.
Each 1% time in range = about 15 minutes per day



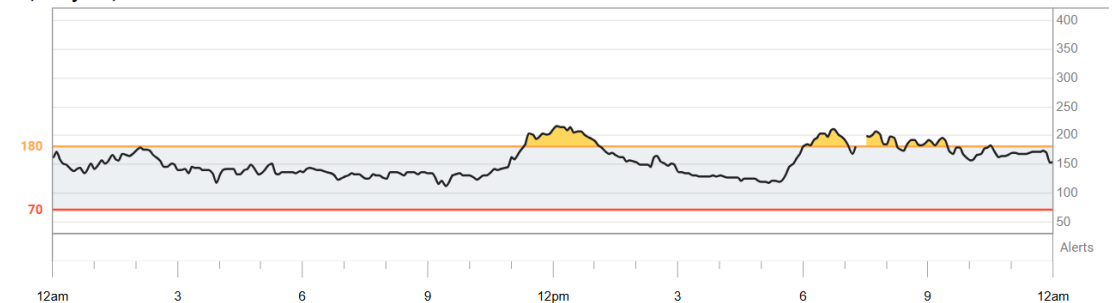
Target Range: 70-180 mg/dL
Very High: Above 250 mg/dL
Very Low: Below 54 mg/dL

Glucose Metrics

Average Glucose Goal: <154 mg/dL	162 mg/dL
GMI Goal: <7%	7.2%
Coefficient of Variation Goal: <36%	31.6%
Time CGM Active	99.1%

	Latest Ref Rng & Units	2/3/2025	5/28/2025
Hemoglobin A1C	4 - 5.5 %	10.4 !	6.4 !

Sat, May 24, 2025

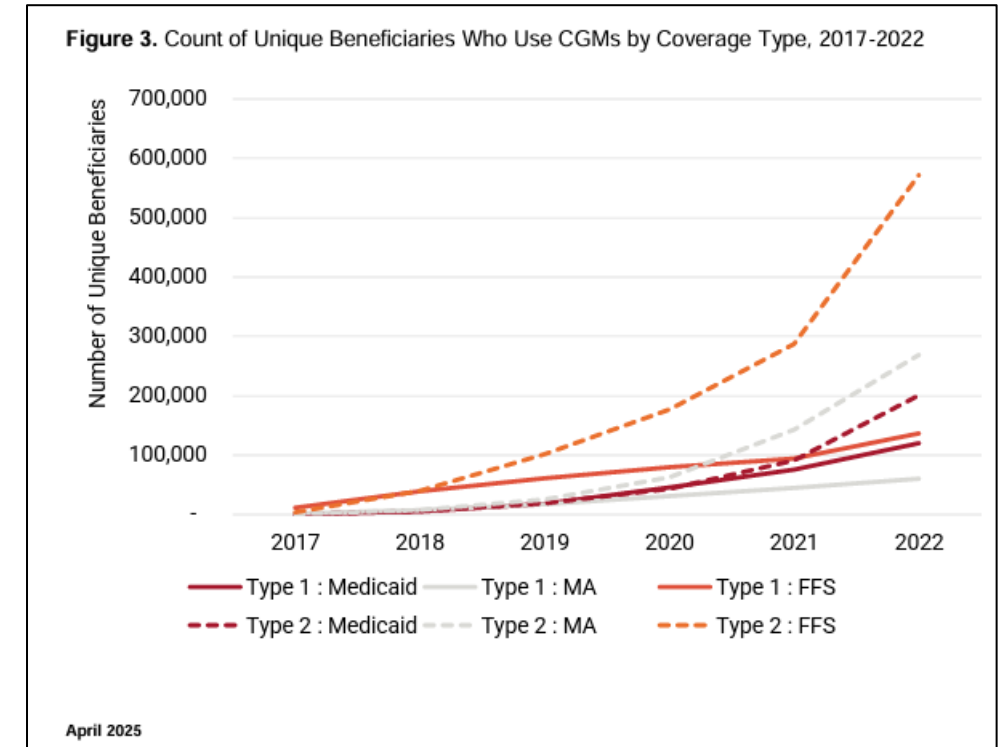


CGMs: Coverage

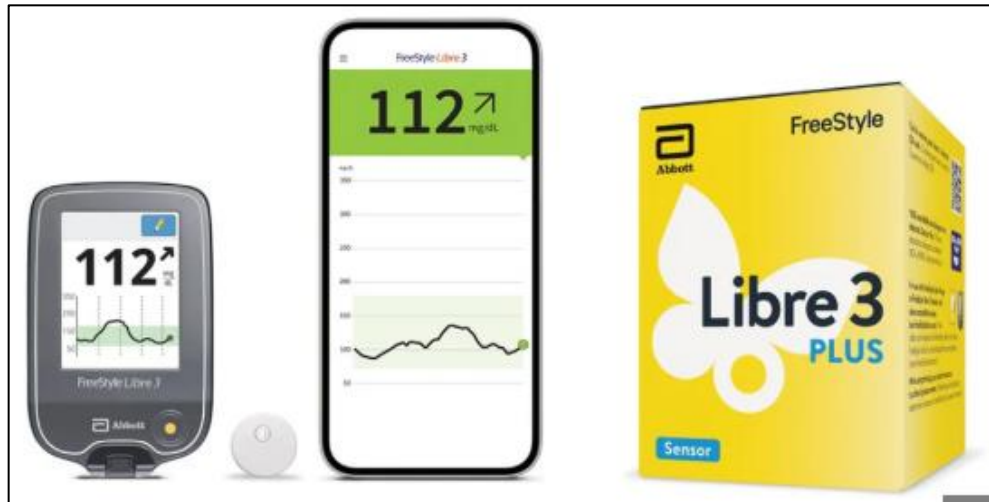
CGMs are now covered by most commercial insurance plans (with or without insulin use) and Medicare/Medicaid (with insulin)

Necessary documentation for Medicare/Medicaid:

1. Beneficiary has documented Type 1 or Type 2 Diabetes **and** displays sufficient training in operating CGM
2. CGM is used in accordance with FDA guidelines
3. Beneficiary is **insulin-treated** or has a history of **problematic hypoglycemia** (level 2 or 3)
4. Beneficiary had visit with provider within 6 months



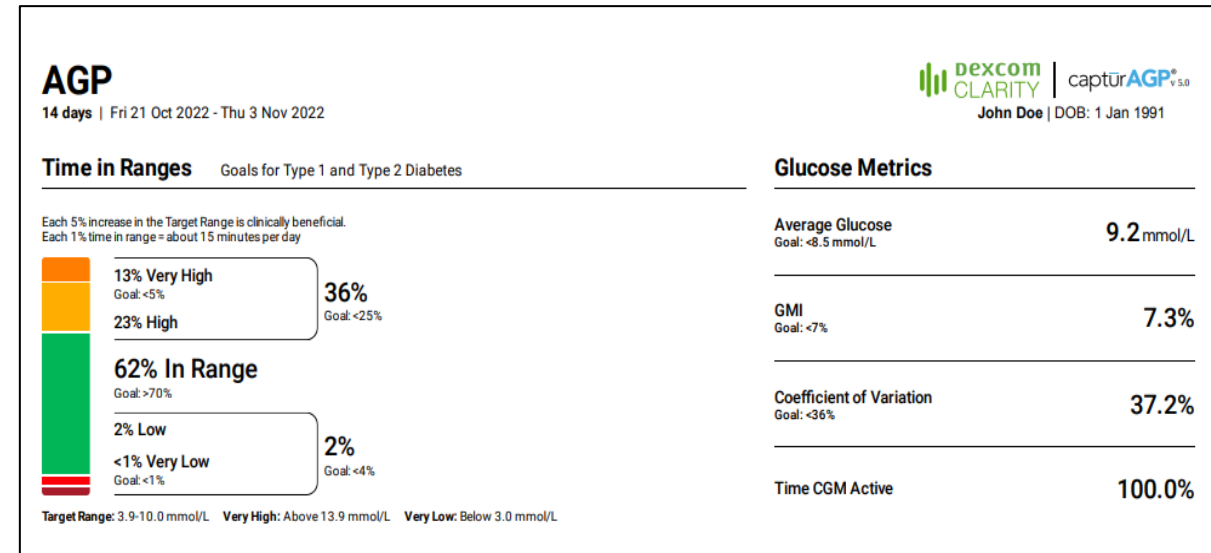
Continuous Glucose Monitors: Newest Iterations



Types of CGMs

	Freestyle Libre 2 Plus	Freestyle Libre 3 Plus	Dexcom G6	Dexcom G7
Duration of wear	15 days	15 days	10 days	10 days
Calibration needed?	No	no	No	no
Alarms	Low <70mg/dL and high >180 mg/dL	Low <70mg/dL and high >180 mg/dL	Low and high, predictive low/high, rate of change	Low and high, predictive low/high, rate of change
Insulin pump integration	Yes, with Omnipod 5, Tandem Mobi	No	Yes with Tandem Control IQ and Mobi, Omnipod 5	Yes with Tandem Control IQ, Mobi and Omnipod 5
Approved for use in pregnancy?	Yes	Yes	No	Yes
Share Features for provider	Libreview	Libreview	Dexcom Clarity	Dexcom Clarity
Method of insertion	Self-inserted	Self-inserted	Self-inserted	Self-inserted

CGMs: Ambulatory Glucose Profile (AGP)



Goal of therapy is to achieve TIR over 85% and 0 % hypoglycemia.

CGM Time In Range (TIR) is The New HbA1c

Limitations of Hemoglobin A1c

3-month glycated hemoglobin percentage

Less precise

Fails to identify hypoglycemia

contributes to hypoglycemia unawareness

Fails to identify hyperglycemic excursions

contributes to micro- and macrovascular complications

Anemia can cause false lows (CKD, elderly)

Time In Range

Estimation of time spent between **70-180mg/dL** gathered from CGM values measured every 5 mins

More precise

More nuanced

Provides shorter term overview, more recent trends

VACCINES FOR ADULTS WITH TYPE 2 DIABETES

VACCINE	RECOMMENDED AGES	SCHEDULE
COVID-19	Age ≥ 65 or 19-64 with underlying conditions such as Diabetes	One dose annually for those eligible
Hepatitis B	People <60 years of age People ≥ 60 years of age based on clinician's risk assessment	—
Influenza	Recommended for all 6 months of age and older. The live attenuated (nasal spray) vaccine should not be used.	Annual
Pneumonia (older vaccine: PPSV23)	19–64 years of age ≥ 65 years of age	• If received PCV15, follow with PPSV23 after ≥ 1 year • PPSV23 is not indicated after PCV20 or 21 • Adults who received only PPSV23 may receive PCV15 or PCV20 ≥ 1 year after their last dose • One dose is recommended for those who previously received PCV13 • If PCV15 was used, follow with PPSV23 ≥ 1 year later • PPSV23 is not indicated after PCV20
Pneumonia (newer vaccines: PCV 15, PCV 20, PCV21)	Adults 19–49 years of age with an immunocompromising condition (e.g., chronic renal failure, cochlear implant, or cerebrospinal fluid leak) or ALL adults 50 and over	One dose of PCV21 is recommended by the CDC
RSV	Adults ≥ 50 years of age with diabetes or other underlying high-risk complications	May receive a single dose of an RSV vaccine
Tetanus, diphtheria, pertussis (Tdap)	• All adults • Pregnant individuals should have an extra dose	Booster every 10 years
Zoster	≥ 50 years of age	Two doses, even if previously vaccinated

THANK YOU



Question 3:

What is the recommended Time In Range in mg/dL for all FDA approved Continuous Glucose Monitors in use for Diabetes?

1. 50-150 mg/dL
2. 70-150 mg/dL
3. 70-180 mg/dL
4. 80-250 mg/dL

Question 4:

Which vaccine is recommended for patients with Type 2 Diabetes over the age of 50?

1. Zoster and Hep B
2. Zoster, Influenza, PCV-21, RSV, Covid-19, TDAP
3. Flu, PCV-21, RSV
4. None of the above

References

1. Slide 34: <https://diabetes.org/sites/default/files/2025-08/ADA-CGM-Covg-Report-Patient-and-HCP-Experience-of-Access-and-Choice-2025-final.pdf>
2. Vaccine slide 39 <https://professional.diabetes.org/sites/dpro/files/2024-04/sri-protectpeoplewithdiabetes-pro.pdf>